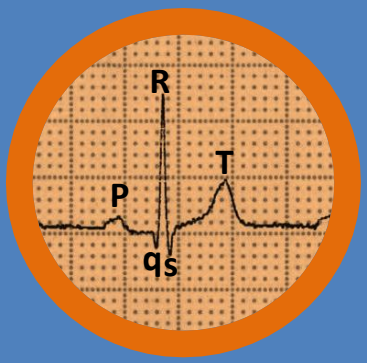

ECG Review : Ventricular Arrhythmia (Part-2)

(For Academic Purpose only)

Dr. DURGA PRASAD KHAITAN

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Vents race wild in electric gale ,
Beats run free with a frantic tale …..

ECG Review : Ventricular Arrhythmia (Part-2)

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Vents race wild in electric gale ,
Beats run free with a frantic tale …..

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 articles of my wirte-up on Ventricular arrhythmias are being covered in 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 ALL THE
FELLOW COLLEAGUES

Index

PAPILLARY MUSCLE VT : UNDERSTANDING THE ECG THROUGH ITS BIDIRECTIONAL SPREAD

ECG

PAPILLARY MUSCLE VT : UNDERSTANDING THE ECG THROUGH ITS BIDIRECTIONAL SPREAD

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OUTLINE

Introduction
Papillary muscle ventricular tachycardia (PM-VT) is a distinct and increasingly recognized subtype of idiopathic ventricular arrhythmia arising from the papillary muscles of either the left or right ventricle.

Anatomy of Papillary Muscles
- Left Ventricular Papillary Muscles : Two in number
- Right Ventricular Papillary Muscles : Three in number
- Purkinje-myocardial junction (PMJ)

Electrophysiology-mechanism
This decreased Purkinje-ventricular coupling is the soul essential of PM-VT.

ECG Features
- ☐ Recording over vertical (frontal) plane
- ☐ Recording over horizontal (precordial) plane

Take-Home Message

References

Papillary Muscle VT : Understanding the ECG through its Bidirectional Spread

A Narrative Review

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In Geometry , a focal point of spread may generate multidirectional exits, distinctly different from a linear or sequential propagation. When viewed on the ECG through the vertical plane, this manifests as bidirectional spread (forward and backward). A similar phenomenon is appreciated in the horizontal plane, where the electrical forces are recorded as left-to-right or right-to-left dispersion.

Papillary muscle ventricular tachycardia (PM-VT) exemplifies this concept of spatial geometric vectors. It reflects electrical activation arising from a single focal site with preferential bidirectional dispersion, rather than conduction along a fixed, orderly activation pathway. In essence, this form of VT is not merely an arrhythmia, but a geometric language of the heart—teaching the observer to interpret direction before morphology.

- ☐ **Papillary muscle VT represents bidirectional electrical dispersion from a focal site of excitation, rather than sequential activation along a defined myocardial pathway.**
- ☐ **It is best appreciated as a localized intracavitary focal propagation, where the direction of electrical forces predominates over temporal sequence.**

In papillary VT, electrical impulses originate from a focal source but propagate through the surrounding myocardium with variable directional dominance. On the surface ECG, this behavior is decoded across both vertical and horizontal planes.

1. Introduction (Keypoints)

- Papillary muscle ventricular tachycardia (PM-VT) is a distinct and increasingly recognized subtype of idiopathic ventricular arrhythmia arising from the papillary muscles of either the left or right ventricle. Advances in intracardiac imaging and electroanatomic mapping have identified papillary muscles as important arrhythmogenic substrates. Its presentation includes isolated premature ventricular contractions (PVCs), nonsustained ventricular tachycardia (VT), and sustained recurrent VT. In addition, PVCs arising from the PMs may play a role as triggers of ventricular fibrillation (VF).
- This VT may occur in both structurally normal and abnormal hearts. PM-VT poses unique diagnostic and therapeutic challenges due to papillary muscle complex anatomy, constant motion, deep intramyocardial foci, and multiple exit sites.
- The earliest formal description of this entity as a separate clinical and electrophysiological syndrome was published in April 2008, when researchers identified seven patients whose ventricular tachycardia or premature ventricular

contractions (PVCs) originated from the posterior papillary muscle, with characteristic ECG features and focal rather than reentrant mechanisms.

- This review provides a comprehensive overview of the anatomy, electrophysiology, and ECG features for both left and right ventricular papillary muscle VT. Preprocedural ECG helps in deciding the site of papillary muscle VT ablation.

2. Anatomy of Papillary Muscles

The PMs are an integral part of the mitral valve (MV) and the tricuspid valve (TV) apparatus. Because of their highly variable and complex anatomy and independent motion during the cardiac cycle, catheter ablation is challenging, with lower procedural success and higher recurrence rates compared with other locations.

Left Ventricular Papillary Muscles : Two in number

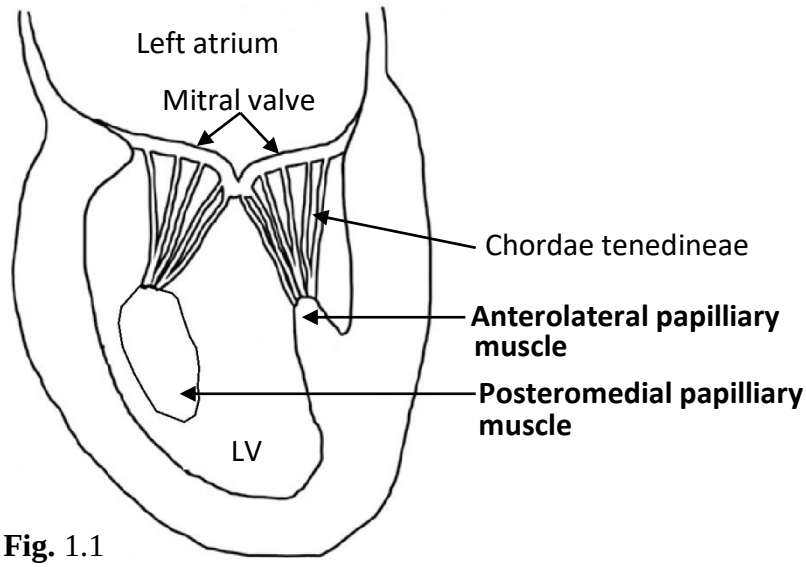


Fig. 1.1

Left LV-Papillary muscle	Characteristics
1. Anterolateral Papillary Muscle (ALPM)	• Usually single-headed • Arises from anterolateral LV wall • Chordae attach to both mitral leaflets • Dual blood supply (LAD and LCX) • Less prone to ischemia
2. Posteromedial Papillary Muscle (PMPM)	• Often multi-headed • Arises from inferior LV wall • Chordae attach to both mitral leaflets • Single blood supply (PDA) • More ischemia-prone

Right Ventricular Papillary Muscles : Three in number

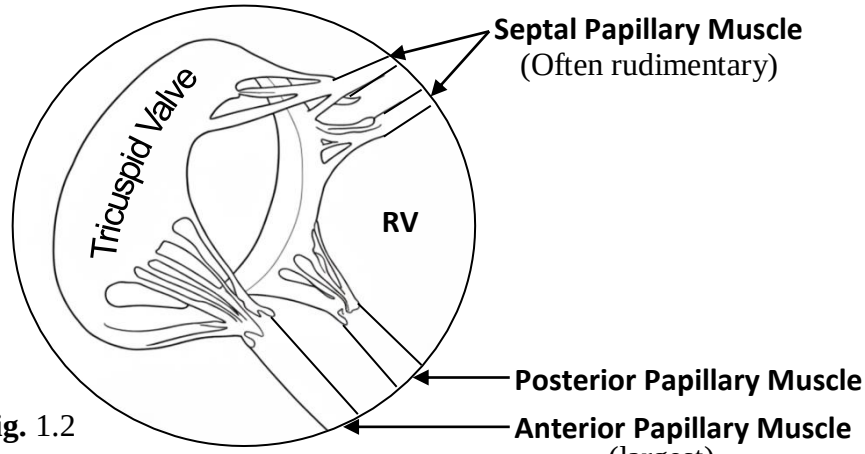


Fig. 1.2

Right RV-Papillary muscle	Characteristics
1. Anterior Papillary Muscle (largest)	• Arises from RV free wall • Supplies chordae to anterior & posterior tricuspid leaflets
2. Posterior Papillary Muscle	• Smaller • Arises from inferior wall • Chordae to posterior leaflet
3. Septal papillary muscle	• The septal papillary muscle can be more than one or even absent. This variability is actually one of the reasons it is called rudimentary • The septal papillary muscle often appears as several tiny projections rather than one proper muscle

Purkinje-myocardial junction (PMJ) :

- ❒ This junction often consists of specialized transitional zones that act as an intermediate, high-resistance interface between Purkinje fibers (PF) and the working ventricular myocytes.
- ❒ The Purkinje–myocyte junction functions as a physiological safety zone by regulating impulse transfer from the conduction system to the working myocardium through an appropriate balance between depolarizing current and myocardial electrical load.
- ❒ However, reduced efficiency of this safety barrier, i.e. decreased Purkinje–ventricular coupling, increases the number of breakthrough sites, resulting in a more complex and chaotic excitation pattern that facilitates PM-VT.

  NB : The left ventricular posteromedial papillary muscle is the most common substrate for papillary muscle VT.

3. Electrophysiology-mechanism

- This decreased Purkinje-ventricular coupling is the soul essential of PM-VT.
- Decreased Purkinje–ventricular coupling at the papillary muscle means poor electrical integration between the Purkinje network and papillary myocardium, creating slow, fragmented conduction → Electrically semi-isolated Island of disarrayed myocytes.
- This electrically semi-isolated island of densely packed, disarrayed papillary myocytes can itself become an ectopic focus of excitation, even in the absence of structural heart disease. Such a milieu provides the perfect substrate for arrhythmogenesis—either through delayed afterdepolarization (DAD)–mediated triggered activity or through micro-reentry facilitated by muscular fiber disarray. Furthermore, if adjacent Purkinje fibers become involved, abnormal automaticity may further amplify the arrhythmic potential.
- Acute anxiety, emotional stress, and physical exertion can precipitate papillary muscle VT by inducing a catecholamine surge, which increases intracellular calcium loading and enhances triggered activity within the electrically vulnerable papillary muscle substrate.
- The presence of myocardial scar may also induce PM-VT through a reentrant arrhythmia, which may also be influenced by catecholamine surge.

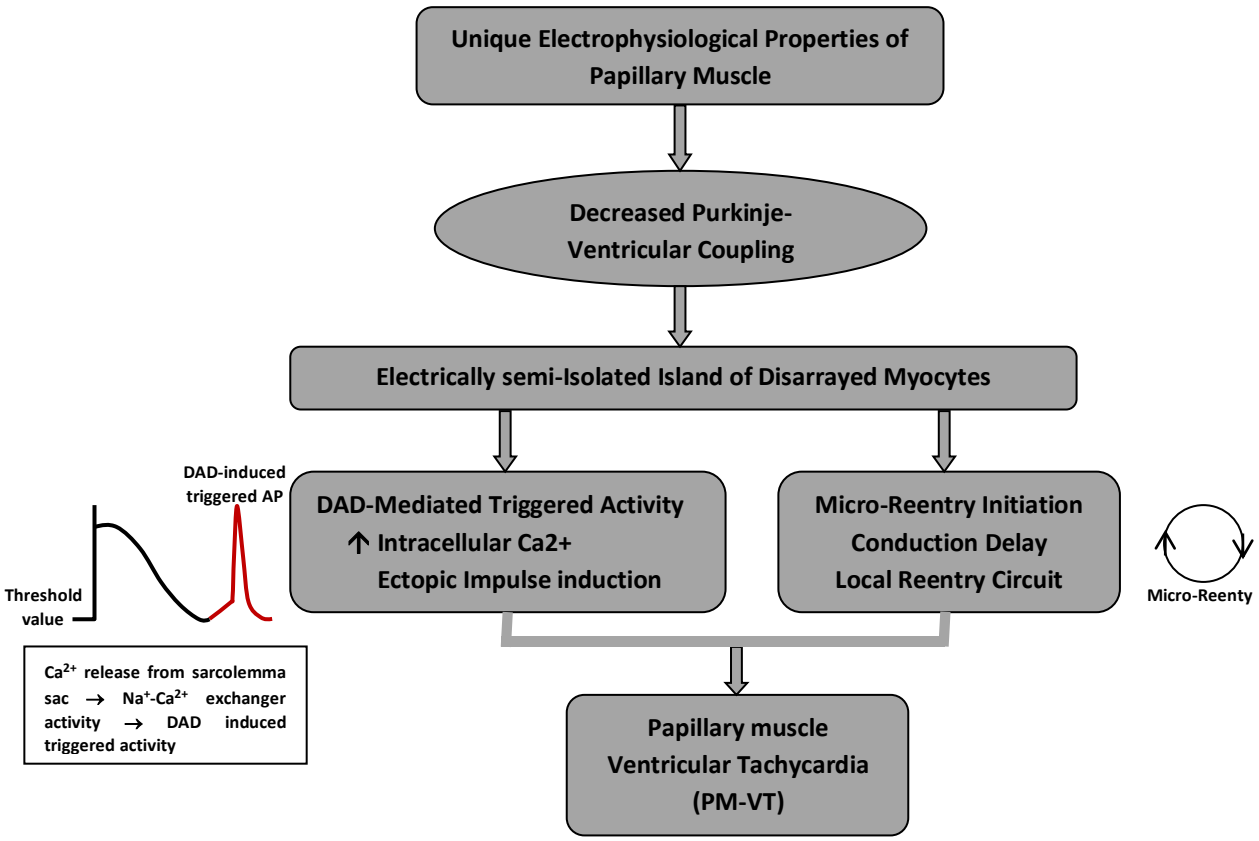


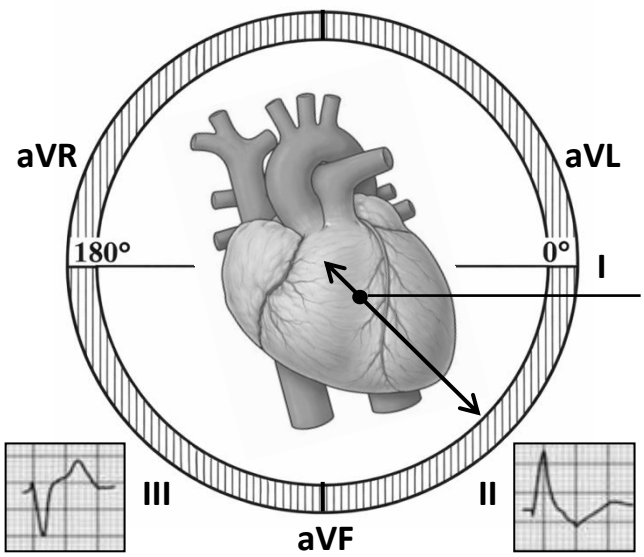
Fig. 1.3

4. ECG Features

Basis :

- Papillary muscles are intracavitary projections of specialized myocardial tissue, PM-VT may exhibit the following fundamental characteristics:
  - The focal point of activation is relatively deep with multiple potential exit sites, resulting in subsequent propagation to the extracavitary ventricular myocardium and producing a significant conduction delay
  - There is no fixed or uniform vectorial direction of activation; instead, the electrical activity arises from a single focal site with preferential bidirectional dispersion, rather than a linear or sequential activation pathway

- **Recording over vertical (frontal) plane :**
  - On the vertical plane, although the individual muscle exhibits a bidirectional axis, the dominant vector is determined by the direction toward which it is predominantly oriented.



Focal point of activation with bidirectional axis , but dominant towards lead II

Dominant vector directed inferior and leftward but away from the rightward orientation of lead III (see Group 1)

Fig. 1.4

Discordance between lead II and III in papillary muscle VT provides important localizing clues.

Group 1		
Dominant vector directed inferior and leftward but away from the rightward orientation of lead III	Lead II positive and lead III negative	Favours a septal origin, including LV posteromedial papillary or RV septal papillary muscle
Group 2		
Dominant vector directed → inferior and rightward	Lead II negative and lead III positive	Left anterolateral papillary muscle

This needs to be mentioned here that Ventricular tachycardias arising from right ventricular anterior and posterior papillary muscles typically demonstrate a superior frontal axis, reflecting upward propagation of activation away from inferior leads.

- **Recording over horizonatal (precordial) plane :**
  - Precordial leads are helpful in determining the QRS morphology in lead V1 in the context of left- or right-ventricular papillary muscle origin and in identifying the corresponding transition zone.

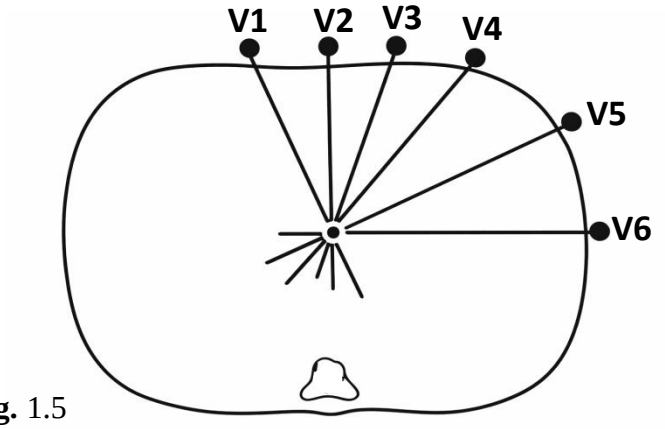
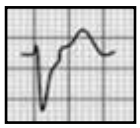
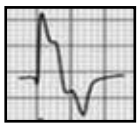


Fig. 1.5

The initial focal point of activation in left-ventricular papillary muscle VT is directed posteriorly and leftward, whereas in right-sided papillary muscle VT it is directed anteriorly and rightward. This difference reflects the anatomical relationship of the ventricles, with the right ventricle positioned anteriorly and the left ventricle lying deeper and more posterior.

LEFT PAPILLARY VT	RIGHT PAPILLARY VT
• V1–V3 → QS / deep S • R wave appears very late • Transition → V5–V6 With R/S ration <1  **V1**	• V1 → R, rSR′, Rs, or qR (often prominent R) • Transition → somewhat earlier (still late)  **V1**

Pertinent points :

- Late precordial transition in left papillary muscle VT occurs because electrical activation spreads centrifugally from a deep intracavitary focus toward the lateral precordial leads, with an R/S ratio < 1 persisting up to lead V6. This delayed transition results in QS complexes or deep S waves in leads V1–V3, creating an apparent right bundle branch block–like pattern when assessed from the terminal forces in the lateral leads. However, this does not represent a true right bundle branch conduction delay

- In right papillary muscle VT, the thinner right ventricular wall allows a relatively earlier precordial transition compared with left papillary muscle VT, although the transition remains delayed overall. QRS morphology in lead V1 is highly variable and depends on the specific papillary muscle involved and the direction of initial electrical activation.

5. Take-Home Message

- Papillary muscle VT represents a unique arrhythmogenic substrate arising from both left and right ventricles.
- The left ventricular posteromedial papillary muscle is the most common substrate for papillary muscle VT.
- Purkinje myocardial junction (PMJ) consists of specialized transitional zones that act as an intermediate, high-resistance interface between Purkinje fibers (PF) and the working ventricular myocytes. Reduced efficiency of this interface barrier , i.e. decreased purkinje ventricular coupling increases the number of breakthrough sites resulting in a more complex and chaotic excitation pattern that facilitates PM-VT.
- The focal point of activation is relatively deep with multiple potential exit sites, resulting in subsequent propagation to the extracavitary ventricular myocardium and producing a significant conduction delay
- There is no fixed or uniform vectorial direction of activation; instead, the electrical activity arises from a single focal site with preferential bidirectional dispersion, rather than a linear or sequential activation pathway
- For strong ECG suspicion of papillary muscle VT, three key principles are crucial:
  i. Frontal plane analysis focusing on lead II–III discordance to determine leftward or rightward vector dominance
  ii. Extremely late precordial transition with R/S <1 even in V6 for left papillary muscle VT
  iii. Markedly wide, slurred QRS complexes (>160 ms) for right papillary muscle VT. Other ECG findings are often misleading and should not be relied upon in isolation.
- Recognition of characteristic ECG patterns, use of intracardiac echocardiography, and targeted ablation of Purkinje potentials are essential for procedural success. With modern techniques, catheter ablation offers excellent outcomes, though recurrence remains a challenge due to deep intramuscular origins and multiple exit sites.

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Subhi J. Al'Aref, MD, James E. Ip, MD, Steven M. Markowitz, MD, Christopher F. Liu, MD, George Thomas, MD, Daniel Frenkel, MD, Nikhil C. Panda, MD, Jonathan W. Weinsaft, MD, Bruce B. Lerman, MD, and Jim W. Cheung, MD , 2015
https://www.ahajournals.org/doi/10.1161/circep.114.002619

NB : Consulted and discussed with ChatGPT whenever needed

SITE OF ORIGIN IN VENTRICULAR TACHYCARDIA : ECG PERSPECTIVE

ECG

SITE OF ORIGIN IN VENTRICULAR TACHYCARDIA : ECG PERSPECTIVE

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OUTLINE

Introduction
The ECG serves as a non-invasive electrophysiologic map, enabling clinicians to identify the anatomical origin of VT, guide management, assess prognosis, and plan catheter ablation when a necessity arises.

Fundamental Electrophysiological Basis
The QRS complex points away from the site of origin and toward the last activated region. This single principle governs all ECG localization of VT

How to determine the site of Ventricular Tachycardia on ECG

- V1 (RBBB/LBBB) → Axis → Transition → Width → Concordance → Site of VT Origin
- A few words about 'Epicardial vs Endocardial origin'

Illustration by 12-lead surface ECG

Take-Home Message

References

Site of Origin in Ventricular Tachycardia : ECG Perspective

A Narrative Review

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Geographical mapping translates complex spatial data into understandable visuals , acting as a crucial tool for navigating how to reach to a particular place. It provides a path to arrive at the point of destination at a glance. A map is not the territory it represents , but if accurate , it maintains a point-to-point structural correspondence with the territory, which accounts for its usefulness.

In a similar way , 12-lead surface ECG helps to localize the site of ventricular tachycardia (VT) , thereby enabling clinicians to guide its management.

- ☐ **In ventricular tachycardia, the impulse writes its autobiography in the form of the QRS complex, and the ECG allows us to read the story of its origin.**
- ☐ **The ECG serves as a non-invasive electrophysiologic map, enabling clinicians to identify the anatomical origin of VT, guide management, assess prognosis, and plan catheter ablation when a necessity arises.**

Thus, the ECG functions as a non-invasive electrophysiologic mapping tool that helps localize the site of origin in ventricular tachycardia, directs therapeutic strategy, aids prognostic assessment, and facilitates procedural planning when intervention is required.

1. Introduction (keypoints)

- Ventricular tachycardia (VT) represents an abnormal rapid rhythm arising from the ventricular myocardium, where electrical activation proceeds outside the normal His–Purkinje conduction system. As a result, ventricular depolarization follows an altered and slower pathway, producing characteristic changes in QRS morphology.
- The shape, axis, and pattern of the QRS complex during VT are not random phenomena. Rather, they reflect the direction of impulse propagation as it spreads away from its site of origin. Since electrical activation moves centrifugally from its source, the surface ECG provides valuable spatial information regarding the location of the arrhythmic focus.
- This principle forms the fundamental basis of ECG localization of VT. By careful analysis of bundle branch block morphology, frontal plane axis, precordial transition, and concordance patterns, one can determine whether the tachycardia originates from
  - Right ventricle or left ventricle
  - Septum or free wall
  - Apex or base
  - Outflow tract or inflow region

- Understanding this relationship between electrical morphology and myocardial origin transforms the ECG from a simple recording tool into a powerful instrument for VT localization.

2. Fundamental Electrophysiological Basis

☐ In ventricular tachycardia, the electrical impulse originates from an abnormal focus within the ventricular myocardium rather than the His–Purkinje system. From this point, depolarization spreads slowly from cell to cell.

Normal Ventricular Activation	Ventricular Tachycardia Activation
• Activation begins in the His bundle	Activation begins outside His–Purkinje system
• Rapidly spreads through right and left bundle branches	Impulse spreads slowly through myocardium
• Purkinje network distributes impulse uniformly	Abnormal focus in ventricular myocardium spreads from cell to cell.
• Ventricles activated synchronously , producing narrow QRS complex	Producing wide, abnormal QRS complexes ≥ 120 ms.

☐ The QRS complex points away from the site of origin and toward the last activated point.

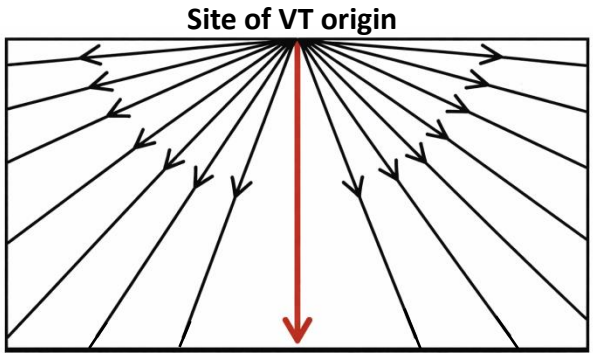


Fig . 1.1 **Last mean activated ventricular point (Illustrated by pointed red arrow)**

This single principle governs all ECG localization of VT

If impulse begins in:

Right ventricle → activation moves toward left ventricle → LBBB morphology

Left ventricle → activation moves toward right ventricle → RBBB morphology

Thus, QRS morphology becomes a direct expression of impulse origin.

3. How to determine the Site of Ventricular Tachycardia on ECG

This becomes easier to follow the steps in determining the site of VT origin if the following sketch is used for the purpose :

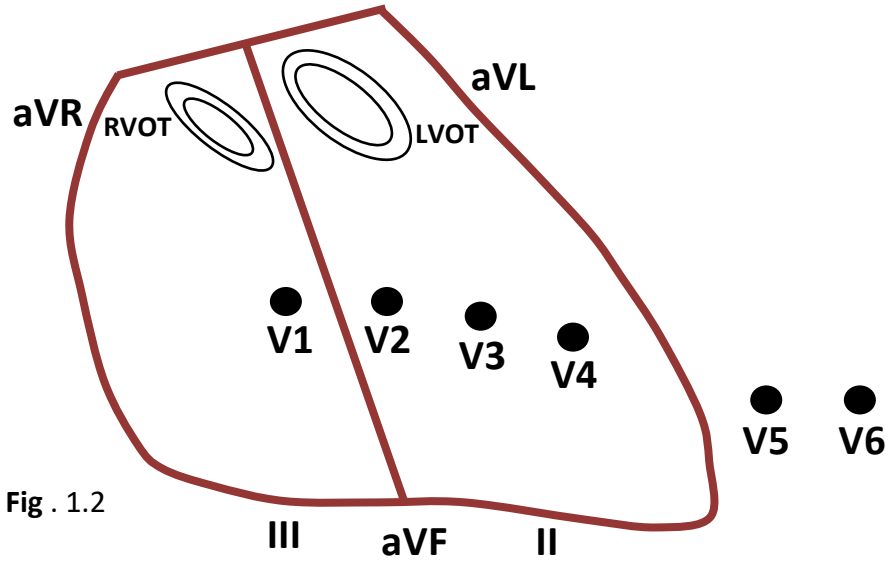


Fig . 1.2

- The morphology of ventricular tachycardia is a direct electrocardiographic projection of its anatomical origin, transforming the surface ECG into a non-invasive map of VT localization.
- Localization of VT follows a logical sequence: morphology defines the ventricle, axis defines the level, transition defines the surface, width defines the depth and concordance defines precise regional localization

---

Stepwise Approach to Localization Site with the following plan :

Lead V1 → Right or Left ventricle origin

Axis → Superior or Inferior origin

Transition → Early or Late transition

QRS width → Septal or Free wall origin

Concordance → Defines regional localization

STEP 1 Bundle Branch Block Morphology: Identifies Right vs Left Ventricular Origin

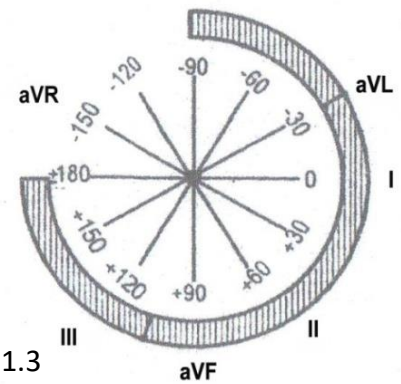
A.

RBBB-like morphology	**Interpretation**	**Common site**
V1: predominantly positive QRS (R wave) V6: predominantly negative or deep S wave	Impulse starts in left ventricle and moves toward right ventricle.	LV septum LV free wall (LV posterior fascicle - fascicular VT)

B.

LBBB-like morphology	Interpretation	Common site
V1: predominantly negative QRS (QS or rS) V6: predominantly positive QRS (Instead Q/QS may be seen)	Impulse starts in right ventricle and moves toward left ventricle.	RV outflow tract (most common in children and young adults) RV free wall RV apex

STEP 2 The site of VT origin lies opposite to the direction of electrical axis.
(Positive polarity towards the direction of electrical axis and negative polarity opposite to the direction of electrical axis).

Hexaxial lead system	Inferior leads (II, III , aVF)
 **Fig** . 1.3	The orientation of the electrical axis is best reflected through inferior leads II , III and aVF. • Predominantly positive deflection in inferior leads→ VT origin from superior portion of the ventricles i.e. the outflow tracts (right/left) – RVOT/LVOT. • Predominantly negative deflection in inferior leads → VT origin from the inferior aspect of the either ventricle, apical VT and fascicular VT – (left posterior)

STEP 3 Precordial transition is an ECG parameter that can be easily approached through 12 lead ECG and it is **defined as the precordial lead in which the R-wave equal or exceeding the S-wave in amplitude**. Normally precordial transition zone exists at V3/V4 but with VT it is either shifted earlier or later.

Early transition :

- Definition : Transition occurs before V3 → i.e., in V1 or V2
- Mechanism : Electrical wavefront is moving anteriorly toward V1–V2, so those leads become positive early (LV→RV)
- Clinical correlation :
  Left ventricular origin, especially LVOT VT (activation moves toward right precordial leads from posteriorly situtated left ventricle)

Late transition :

- Definition : Transition occurs after V4 → i.e., in V5 or V6
  (QRS may remain predominantly negative in V1-V4)
- Mechanism : Activation moves away from anterior chest leads → so they remain negative longer (RV→LV)
- Clinical correlation :
  Right ventricular origin, especially RVOT VT (activation moves towards the posteriorly situated left ventricle).

STEP 4 QRS width determines Septal vs Free Wall Origin

Septal origin	**Free Wall Origin**
• Moderately wide QRS (140–160 ms) • Reason: Septum close to conduction system (Impulse quickly enters conduction network)	Very wide QRS (>160 ms) Reason: Slow muscle-to-muscle conduction

STEP 5 Concordance in Precordial leads

- **Definition** : All chest leads (V1-V6) with QRS complexes that are either uniformly dominantly positive (positive concordance) or uniformly dominatly negative – QS / rS pattern throughout (negative concordance).
  No R/S progression. No transition-just uniform polarity.
- **Positive concordance** : All precordial leads show uniformly dominant R-waves. This means the depolarization wavefront is moving toward the anterior chest wall. This implies that activation is arising from posterior region , often from posterobasal LV OR Inferior/posterior LV wall.
- **Negative concordance** : All precordial leads show uniformly QS or predominantly negative complexes with rS pattern throughout. This means the depolarization wavefront is moving away from anterior chest wall. This implies that impulse is arising from anterior LV wall , apical region , and possibly epicardial anterior surface.
  This negative concordance pattern is generally more common than positive concordance in ventricular tachycardia. Negative concordance is particularly frequent in structural heart disease , depending on the site of origin.

In addition , a few words about **'Epicardial vs Endocardial Origin'**

- Epicardial VT shows delayed activation as ECG features , very wide QRS, slurred initial QRS with slow onset (because impulse travels from epicardium to endocardium)
- Seen in ARVC (Arrhythmogenic right ventricular cardiomyopathy) , at times in others cardiomyopathy, and scar VT if nearby epicardium.

Final concluding touch

As a final concluding touch the following localizing schedule may be summarized as follows : :

V1 (RBBB/LBBB) → Axis → Transition → Width → Concordance → Site of VT Origin.

4. Illustration by 12-lead surface ECG

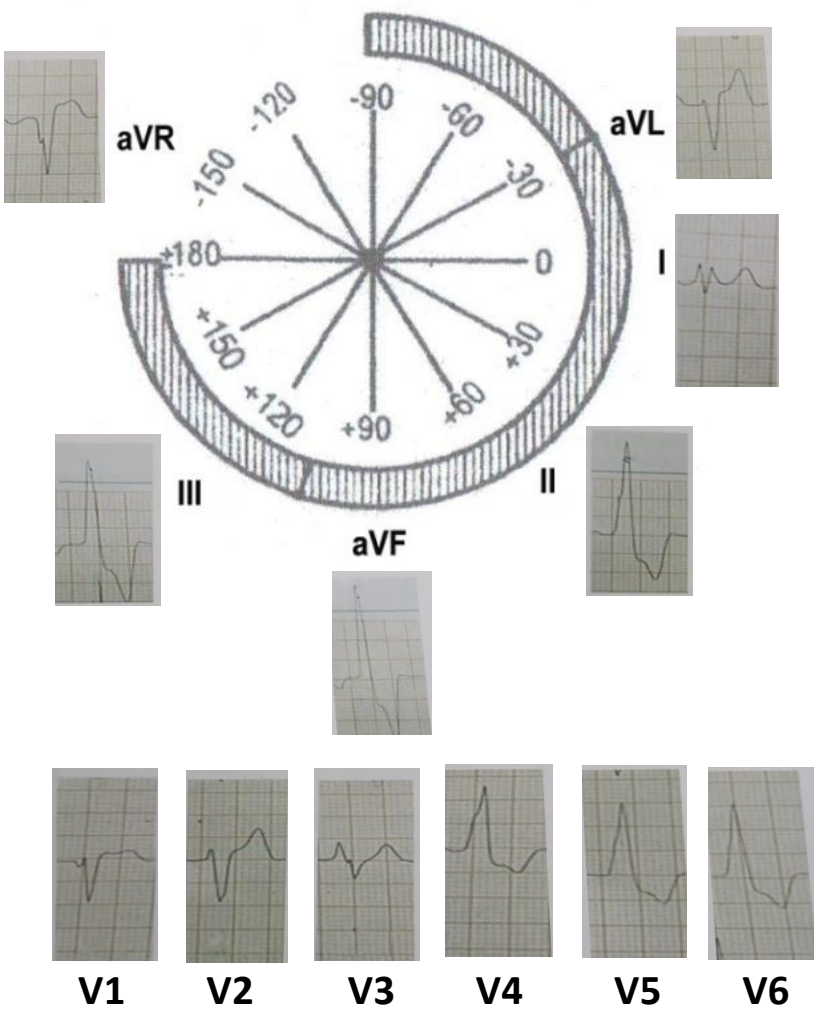


Fig . 1.4

Findings :

- LBBB-like morphology (site of origin in right ventricle) :
  V1 with a predominantly negative QRS complex (rS pattern) and V6 with a predominantly positive QRS complex — consistent with activation spreading from right to left.
- Predominantly positive deflection in inferior leads (II, III, aVF) →
  Suggests VT origin from the superior portion of the right ventricle, most commonly the Right Ventricular Outflow Tract (RVOT).
- Late precordial transition (around V4 or beyond).
- Wide QRS complex, consistent with ventricular origin.
- Absence of positive or negative concordance in the precordial leads

Comments :

The accumulated electrocardiographic evidence strongly suggests an origin from the Right Ventricular Outflow Tract (RVOT), consistent with idiopathic RVOT VT typically seen in younger individuals without structural heart disease.

5. Take-Home Message

- ☐ Ventricular tachycardia is not a random electrical disturbance. It is a structured and directional propagation originating from a fixed anatomical source. The ECG provides a projection of this propagation.
- ☐ The ECG serves as a non-invasive electrophysiological map. By analyzing QRS morphology, axis, transition, concordance pattern and width, the clinician can determine the anatomical origin of ventricular tachycardia. This transforms ECG interpretation from pattern recognition into spatial diagnosis.
- ☐ Integrated Interpretation Strategy (stepwise analysis) :

  Step 1: Examine V1
  → Right vs Left ventricle

  Step 2: Examine axis
  → Superior vs Inferior origin

  Step 3: Examine transition
  → Early vs Late

  Step 4: Examine QRS width
  → Septal vs Free wall

  Step 5: Examine concordance
  → Defines regional origin

- ☐ As a closing line of message , it would be worthwhile to mention that the impulse may arise silently within the depths of the myocardium, but its journey leaves a visible signature, and the ECG teaches us how to trace it back to its source

6. References

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   John L. Sapp, MD john.sapp@nshealth.ca, Meir Bar-Tal, MSc, Adam J. Howes, MD, Jonathan E. Toma, MD, Ahmed El-Damaty, MD, James W. Warren, BSc, Paul J. MacInnis, BSc , 2017
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NB : Consulted and discussed with ChatGPT whenever needed

PSEUDO VENTRICULAR TACHYCARDIA : ‘AN UNREAL MAY BE TREAED ASS REAL’

ECG

PSEUDO VENTRICULAR TACHYCARDIA : 'AN UNREAL MAY BE TREATED AS REAL'

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OUTLINE

Introduction

Artifacts on ECG can be misdiagnosed as VT and this possibility should be considered to avoid unnecessary diagnostic and therapeutic interventions.

Pseudo ventricular tachycardia and its mechanism

- ☐ The skin-electrode interface needs a special note as it is the largest source of interference induced artifacts.
- ☐ These artifacts producing noises are difficult to be filtered to the desired level , due to considerable overlapping of its frequency scale to that of the frequency scale of ECG signals.

Electrocardiographic approach to identify Pseudo-ventricular tachycardia

There are certain criteria , laid down for the identification of Pseudo-ventricular tachycardia induced by motion artifacts : **the sinus sign , the spike sign and the notch sign**

Putting all together on ECG

Take-Home Message

References

Pseudo ventricular tachycardia : 'An unreal may be treated as real'

A Narrative Review

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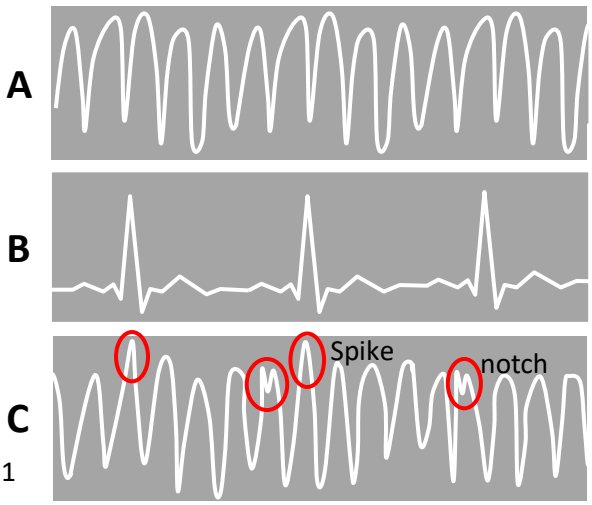
Artifacts are not the product of intrinsic cardiac electrical activity; rather, they are fabricated signals arising from external sources. For example, bodily movements during ECG recording may occur simultaneously with genuine cardiac depolarization and repolarization, thereby distorting the true P–QRS–T complexes.
These signals are trivial in origin yet deceptive in appearance, often mimicking real arrhythmias despite having no true cardiac basis.

- ☐ **External energy—mechanical or electrical—entering the recording system from outside the heart and not adequately filtered by the ECG machine is transformed into artifacts. Their recording is superfluous and misleading in nature**
- ☐ **The most common electrocardiographic artifacts are produced by mechanical factors such as patient movement. At times, these artifacts may closely simulate a run of VT (ventricular tachycardia), and even experienced clinicians may misinterpret them as true VT, leading to unnecessary investigations, unwarranted therapeutic interventions, and potential patient harm.**

Artifacts on ECG serve as important learning lessons for every clinician. One must cultivate careful analytical skills and mental alertness to recognize them; otherwise, the unreal may be mistaken for the real.

1. Introduction (keypoints)

- ○ Ventricular tachycardia (VT) is a life-threatening arrhythmia associated with a significant risk of sudden cardiac death; therefore, it warrants prompt evaluation and appropriate management. However, ECG artifacts may be mistaken for VT, and this possibility must always be considered to prevent unnecessary diagnostic procedures and inappropriate therapeutic interventions.
- ○ The most common cause of electrocardiographic artifacts is bodily movement - they may superimposed on normal ECG tracings, with distorting its signal P–QRS–T.



Bodily movements travelling as waves

↓ **Superimposed on**

ECG tracings : P-QRS-T

↓

Distortion of some QRS signals as spikes and notches – the entire run mimicking as VT (Pseudo-ventricular tachycardia)

Fig. 1.1

- One has to be very precautious to diagnose whether it is an artifact or true ventricular tachycardia. **It is a clinical dictum that the possibility of artifact as a cause of ECG rhythm disturbances should always be considered in otherwise asymptomatic patient who is hemodynamically stable**. ECG interpretation should always be correlated with the clinical history , keeping a keen eyes inspection over the bodily movements if any. We should treat the patient , not the ECG.

2. Pseudo ventricular tachycardia and its mechanism

☐ **An interlinked pathway for recording the ECG signals originating from the heart** : While recording an ECG, the system may be influenced by re-entrant or external energy (e.g., bodily movement) arising outside the cardiac source. Such external interference can affect the signal at any point along the recording pathway, as illustrated below :

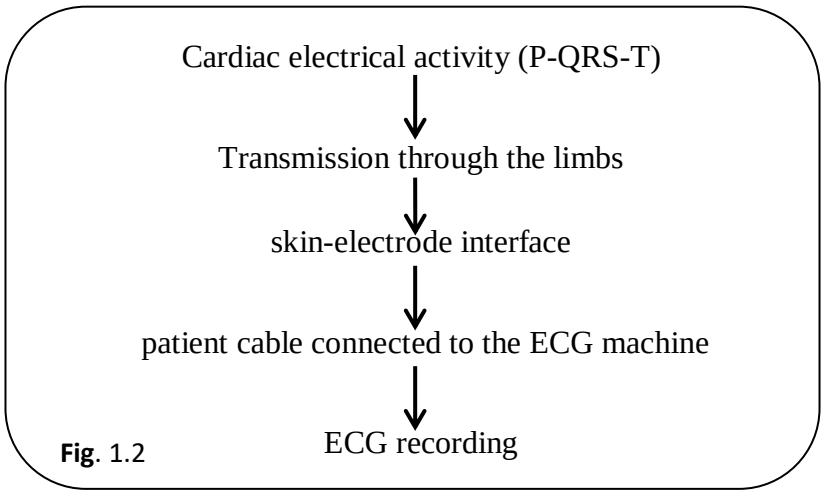


Fig. 1.2

Any disturbance occurring anywhere along this chain can modify the ECG tracings. The alteration may result either from a true cardiac arrhythmia (e.g., ventricular tachycardia) or from mechanical movements/electrical interference arising externally at any level in this continuous pathway. The following factors may be responsible for such changes :

- **Bodily movements**
- **Skin-electrode interface disturbance :** Either the electrodes may be loosely applied to the limbs or the underneath jelly may be either more or less in quantum – all these may produce unwanted muscular movements at this interface.
- **Any sort of external interference** : nearby mobile phones or external electricity in vicinity.

  The skin-electrode interface needs a special note as it is the largest source of interference induced artifacts. The artifacts produced from its component are magnified either by the patient movement or respiratory variation.

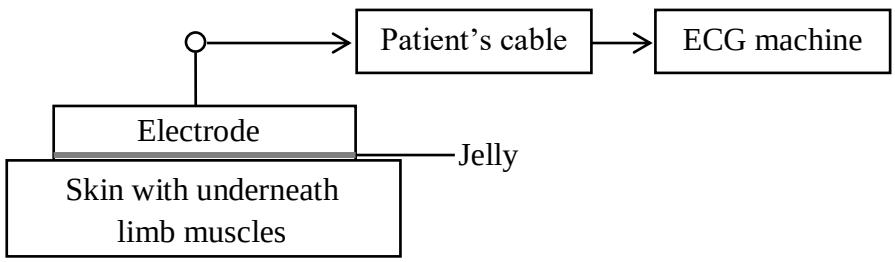


Fig. 1.3

Skin-electrode interface

☐ ECG filter and artifacts

This would be ideal to obtain ECG signals (P-QRS-T) without being mixed with artifacts. ECG filters try to eliminate or remove unwanted noisy interference coming from sources other than the cardiac origin. These artifacts producing noise are difficult to be filtered to the desired level due to considerable overlap of their frequency scale with that of ECG signals. There is a need to choose the appropriate filter to accommodate ECG waves within the frame of recording but this remains a challenge – frequency scale of artifacts and ECG waves are very close to each other. That's why , inspite of choosing the appropriate filter as per need , there remains the possibility of body movement – induced waves being mixed with ECG waves.

It would be appropriate to mention here that the frequency of any such signal whether coming from normal ECG or artifacts, is measured as Hertz (Hz) unit. A frequency of 1 Hz means one signal repetitions per second. The heart normally produces electrical activity at the rate of 60 to 90 beats per minute. It can be said that with a fundamental frequency of 1 Hz at this heart rate, all ECG signals (P-QRS-T) would be recorded at or above this frequency. The frequency scale of ECG signals and that of artifacts producing signals (muscles activity) may run more or less within the same frequency framework.

QRS frequency is 10-50 Hz (considering the heart rate in between 40-300 bpm)	**Frequency of muscle artifacts is 10-500 Hz but do not follow a predictable pattern – they contain power across wide range of frequencies, including the 10-50 Hz range.**

Ref :
UNDERSTANDING ECG FILTERING
PUBLISHED: MARCH 10, 2014
AUTHOR: Christopher Watford ; Retrieved from :
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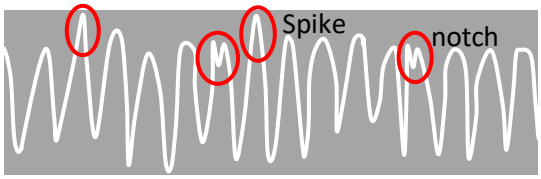
Since the frequency of the QRS wave is often more or less equivalent to the frequency of muscles-induced artifacts , ECG filters may allow both frequency components to be recorded on the ECG simultaneously , mimicking ventricular tachycardia – **termed as Pseudo Ventricular tachycardia**. Even with an increase in heart rate , the QRS complex continues to show higher amplitude , and the same is true for muscles artifact signals , which also exhibit relatively higher amplitudes. Therefore , both may be recorded as having higher amplitudes on the ECG , while the lower-amplitude P and T signals may become buried within the crowded artifacts.



Artifacts signals distort the QRS signals , appearing as spikes or notches amidst the noisy spells.
(Smaller P and T signals are buried within.)

Fig. 1.4

3. Electrocardiographic approach to identify Pseudo-ventricular tachycardia

Basics

This is essential to review the nature of different leads concerned with ECG recording. It would be appropriate to mention here that the tracings on ECG are recorded by applying limb and chest electrodes at its appropriate places.

Leads	Negative Electrode	Positive Electrode
Bipolar I II III	Right Arm Right Arm Left Arm	Left Arm Left Leg Left Leg
Unipolar aVR aVL aVF	*Central Terminal	Right Arm Left Arm Left Leg
Chest leads	*Central Terminal	V1-V6 on different specified spots on chest wall

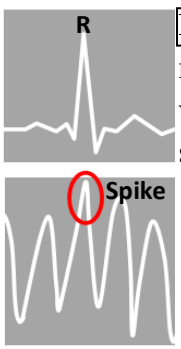
***Central terminal** is formed by joining the right arm , left arm and left leg electrodes together - this comes to 0 (Zero) potential. The current flows from negative electrode towards positive electrode due to the potential difference in between two.

Main criteria for the identification of Pseudo-ventricular tachycardia induced by motion artifacts : the sinus sign , the spike sign and the notch sign.

☐ **Sinus sign** : One of the frontal leads (leads I , II and III) may present with sinus rhythm with the preservation of normal P-QRS-T waves. The reason behind this is that one of the upper limb electrodes is free of movement artifact. This sign was introduced by Huang et.al.

Explain: If the left arm is free from movement , the lead III (left arm-left leg) would be showing sinus rhythm free from motion artifacts. And the remaining leads I and II would be showing artifacts because the right arm having motion artifacts is common to these two leads. The augmented unipolar leads aVR, aVL and aVF would also be showing motion artifacts because the right arm is one of the components of central terminal.

☐ **Spike sign** :



Explain **:** The superimposition of **R** (Q**R**S) on the top of simultaneously running taller artifact wave – giving this the appearance of a taller wave having spine on its top, as illustrated below with the corresponding sketch.

If one finds a wave of higher amplitude with spike on its top amidst the waves of motion artifacts , this is considered as 'spike sign' to be positive.

Fig. 1.5

☐ **Notch sign** :

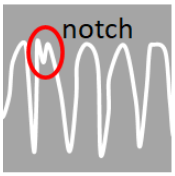


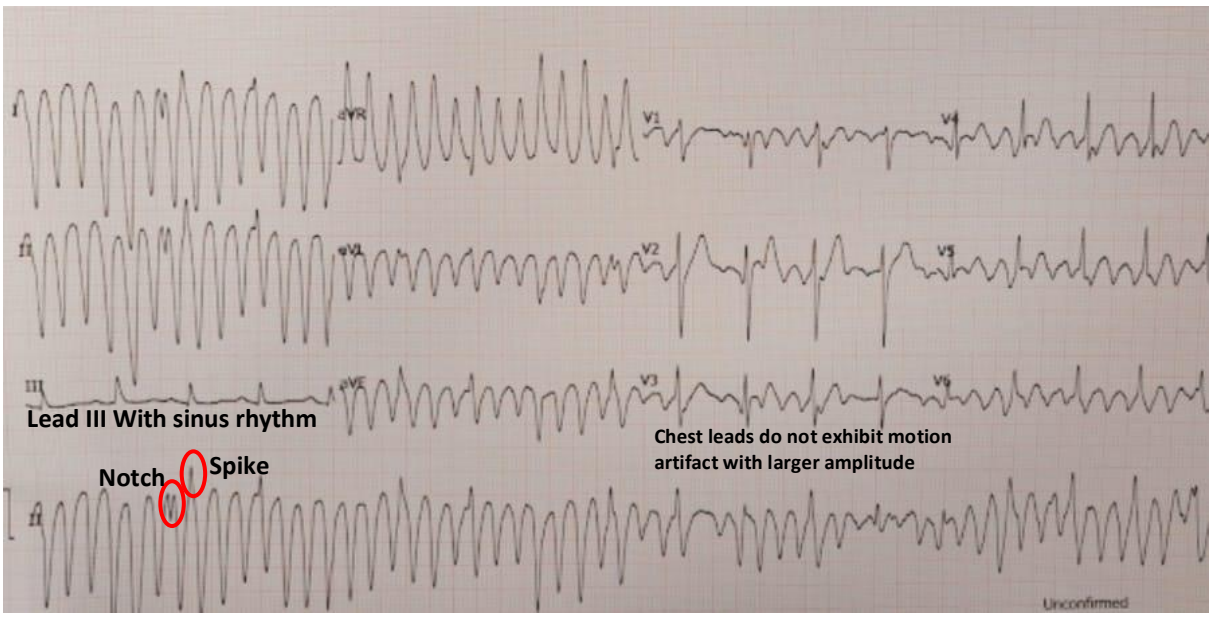
Fig. 1.6

Explain : The intersection of two waves running very closer to each other may create a visible notch in between (Firstly pointed out by Littmann and Monroe). ECG machine contains some ability to reject some noises even without a filter – eliminating a small region of frequencies on the top of these two simultaneously running waves, appearing as a notch.

If one finds such a notch amidst the waves of motion artifacts , this is considered as 'notch sign' to be present.

Chest leads do not exhibit motion artifacts having larger amplitudes – rather artifacts with smaller amplitudes come in view. This is due to the fact that respiratory-motion excursion over the chest wall is having 0.12-0.50 Hz range (e.g. 8-30 bpm) , thus eliminating the possibility of artifacts having higher amplitude.

4. Putting all together on ECG



Source : CME INDIA on 22.10.2022 by Dr. N.K Singh , Consultant Physician & Diabetologist , Director , Diabetes and Heart Research Centre, Dhanbad , Editor , www.cmeindia.in and Editor-in-Chief , IJCCP

73 years hypertensive , no history of chest pain , no breathlessness

Discussion : This ECG reveals right upper arm as the cause of artifact.

- Lead III (left arm + left leg) is showing sinus rhythm with the preservation of P-QRS-T signals as such , thereby indicating the absence of motion artifacts over left arm and left leg.
- Since lead I (left arm + right arm) and lead II (right arm + left leg) both are exhibiting motion artifacts – thereby , indicating involvement of the right arm with motion artifacts being intermingled with spikes and notches .

- Augmented leads (aVR , aVL , and aVF) also exhibit the evidence of artifacts , since the right arm is one of the components of central terminal ,being formed by the combination of all these three.
- Chest leads do not exhibit motion artifacts with larger amplitudes – rather recording artifacts with smaller amplitudes. This is due to the fact that chest wall is having 0.12-0.50 Hz (e.g. 8-30 bpm).

Comments

The waves-pattern in this ECG resembles very much with ventricular tachycardia, but this is not so. That's why , this is termed as Pseudo ventricular tachycardia (an unreal may be treated as real).

5. Take-Home Message

- ☐ Ventricular tachycardia (VT) is a life-threatening arrhythmia associated with a significant risk of sudden cardiac death; therefore, it warrants prompt evaluation and appropriate management. However, ECG artifacts may be mistaken for VT, and this possibility must always be considered to prevent unnecessary diagnostic procedures and inappropriate therapeutic interventions.
- ☐ The most likely cause of electrocardiographic artifact is bodily movement – superimposed on normal ECG tracings with distorting of its signal P-QRS-T , distorted QRS complexes appearing as spikes and notches amidst the noisy spells (smaller P and T signals are buried within).
- ☐ There are certain criteria , laid down for the identification of Pseudo-ventricular tachycardia induced by motion artifacts : **the sinus sign , the spike sign and the notch sign**.
- ☐ It is a clinical dictum that the possibility of artifact as a cause of ECG rhythm disturbances should always be considered in otherwise asymptomatic patient who is hemodynamically stable.

If one notices spikes and notches amidst the crowd of noisy artifacts over the ECG tracings , the presence of pseudo ventricular tachycardia should strongly be suspected therein.

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NB : Consulted and discussed with ChatGPT whenever needed

PSEUDO P- A LAND OF DIAGNOSTIC PITFALL IN VT (P-PREOCCUPATION SYNDROME)

ECG

PSEUDO P – A LAND OF DIAGNOSTIC PITFALL IN VT (P-PREOCCUPATION SYNDROME)

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OUTLINE

Introduction

Any P-like wave that lies adjacent to its accompanying QRS complex may actually emerge as, or represent, a part of the QRS complex itself. This can easily mislead a clinician whose mind suffers from "P-preoccupation syndrome," for whom any wave that resembles a 'P' is assumed to be nothing but a true P-wave.

Mechanism of pseudo P - a possible speculation

Under these circumstances, the P and QRS complexes as a whole should be carefully scrutinized in every ECG lead, especially in the precordial leads, and equal emphasis should be placed on analyzing any preceding P-like wave for this purpose.

Discussion

- QRS complexes should be analyzed first as it gives maximum information and state of ventricular behavior.
- Cherchez le P
- Haystack principle
- The presence of extra ECG findings in favour of VT

Take-Home Message

References

Pseudo P – A land of diagnostic pitfall in VT (P-preoccupation syndrome)

A Narrative Review

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"First romance , and first love , is something so special to all of us , both emotionally and physically , that it touches our lives and enriches them forever **"**

Rosemary Rogers (Srilankan Author of historical romance novels : **1932-2019**)

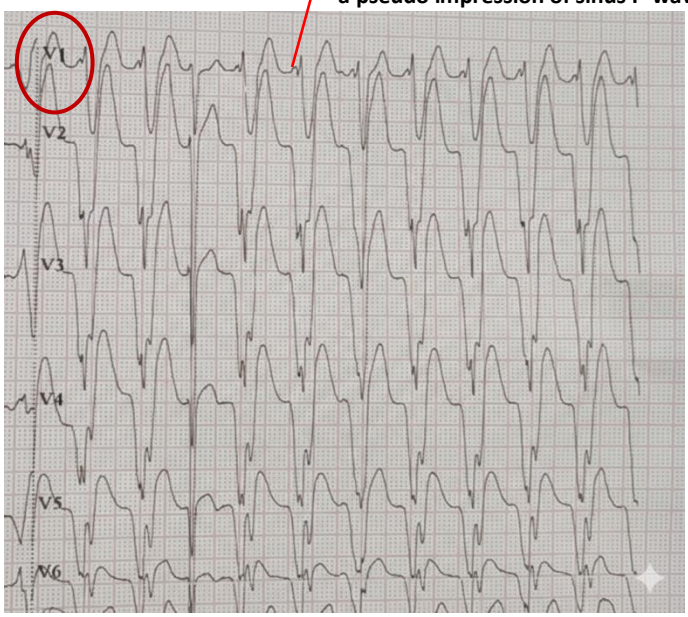
Truly said—none can forget the first love. In much the same way, the P-wave is the "first love" for ECG interpreters, and its impression can never be wiped away from their minds. Whenever a clinician encounters a wave simulating a 'P' on an ECG, the pre-biased mind instinctively interprets it as a sinus P-wave. This phenomenon may aptly be termed as "P-preoccupation syndrome.

- ☐ **Any P-like wave that lies adjacent to its accompanying QRS complex may actually emerge as, or represent, a part of the QRS complex itself. This can easily mislead a clinician whose mind suffers from "P-preoccupation syndrome," for whom any wave that resembles a 'P' is assumed to be nothing but a true P-wave.**
- ☐ **This becomes essential to unmask such an illusive P-wave , which may otherwise lead to an incorrect diagnosis of SVT.**

Whenever a clinician is faced with this problem of a deceptive P-like deflection—almost stitched to the following QRS complex—one must be prepared for a meticulous and comprehensive analysis in order to arrive at a precise diagnosis.

1. Introduction (keypoints)

- **The term Pre-occupation syndrome** was first coined by Dr. Henry J. L. Marriott, the author of the landmark book 'Practical Electrocardiography'. This term refers to a preconceived bias on the part of the ECG interpreter, wherein any P-like wave is readily assumed to be a sinus P wave.
- In certain situations, when a clinician is presented with a strip of precordial leads, the tracing may be misinterpreted as supraventricular tachycardia. Such confusion arises when P-like activity appears in very close proximity to the QRS complexes.
- At this juncture there exists a diagnostic dilemma.



A case of VT with pseudo P in lead V1 : please zoom and see

Fig. 1.1

2. Mechanism of pseudo P : a possible speculation

Under the prevailing circumstances P and QRS complexes as a whole should be scrutinised in every lead of ECG , specially with precordial leads and equal emphasis should be imparted to the analysis of preceding P-like wave as well.

If V1 (from the ECG strip as posted below) is carefully scrutinised , there seems an initial wave simulating P-wave , almost stitched together with its accompanying QRS complex - this encircled like this as **1** on the ECG tracings , as posted below and the terminal component of adjacent QRS is marked as negative wave **2** for the purpose of scrutinizing the complex as a whole.

If these two components of V1 - **1** and **2** are considered together as integrated QRS complex , this seems to be a case of negative concordance on ECG (V1-V6) in favour of VT. It is well known fact that negative concordance in VT arises from a non-septal free wall of left ventricle or its apical territory , typically corresponding with the ventricular activation site in this case (ECG tracings V1-V6 are posted below).

✓ **ECG** : 50 years male presents with palpitation in the background of recent CAD with EF 20-25% on cardiac echo.

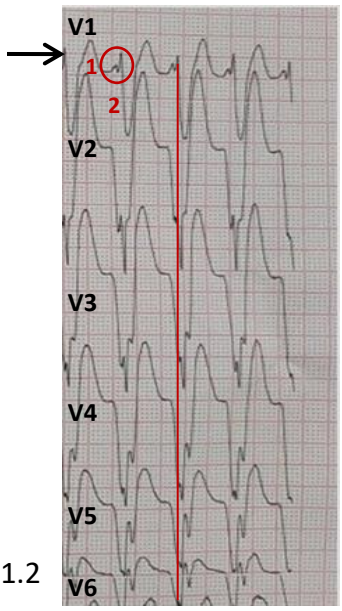


Fig. 1.2

If the P-like wave in V1 is traced downward with a vertical line , it may be seen as notches occupying the corresponding negative QRS complexes over V2-V6. This also proves that P-like wave is a part of the QRS complex – not P-wave separately (Pseudo P-wave).

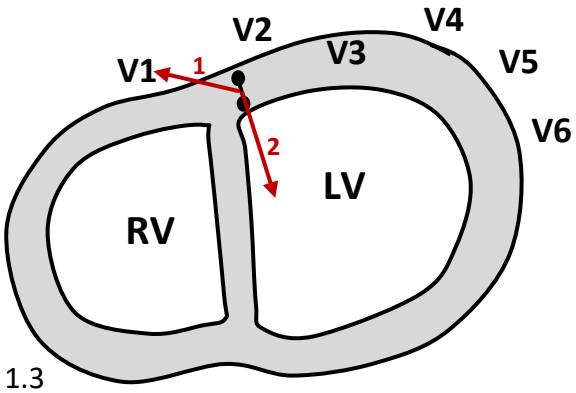


Fig. 1.3

There seems to be a simultaneous epicardial and endocardial exit routes involvement , as illustrated by the above diagram

1 = Epicardial exit of ventricular activation site (Posterior to anterior direction) Positive deflection

2 = Endocardial exit of ventricular activation site (Anterior to posterior direction) Negative deflection

Any exploring electrode placed towards the flow of current records positive deflection and negative deflection if away from the flow of current

Ref :

Simultaneous Endocardial and Epicardial Delineation of 3D Reentrant Ventricular Tachycardia
Author : Roderick Tung et.al
https://pubmed.ncbi.nlm.nih.gov/32130924/

3. Discussion

The following points should be considered in this context :

- A clinician should be very cautious in delineating 'P-QRS' , specially in a situation of VT when both complexes are stitched together without any gap in between , as shown in lead V1 of the following ECG. If this conjoint complex (V1) is traced downward with a vertical line , this P-like can also be seen as notches occupying the corresponding QRS complexes over V2-V6 with increment in its width (>0.12 sec). This also proves that P-like wave is a part of the QRS complex – not P-wave separately. **Therefore , QRS complex should be analysed first as it gives the maximum information and state of ventricular behaviour.**

- With no doubt, morphology of QRS complex while analysing VT plays a major role. This may not be conclusive , then , P-wave should be analysed what is called as "**Cherchez le P**" by Marriott (By dictionary Cherchez means 'to look for'). As discussed these P-like wave in V1 is the part of the QRS complex.

- Sometimes **'Haystack principle'** offers a great help under the situation. Here , this wording means to search a needle in a haystack , smallest haystack should be searched out for its presence. In case of P-like wave in VT a lead with flattest deflection should be focused , wherein a separate P from QRS complex might be observed. Here the aVR is the lead with flattest deflection ,which does not show a clear cut separate P (the concerned ECG is posted below).

- The present ECG in question is to be seen carefully , because it also shares **the other findings of VT as well** - **capture beat** and **fusion beat** on 3rd and 6th complexes respectively on frontal plane , as encircled below and see lead aVR for **Northwest axis.** (NW).

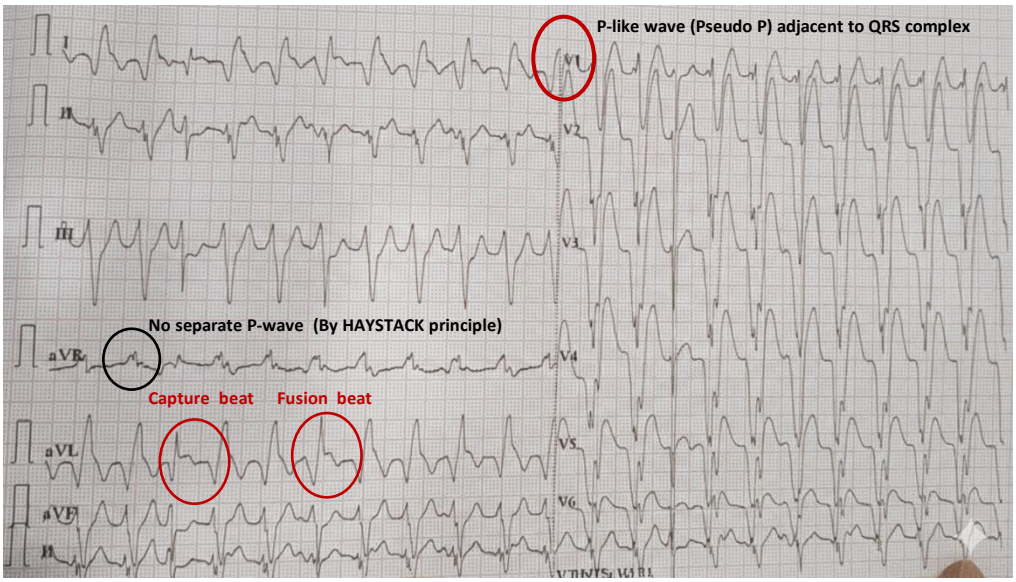


Fig. 1.4

- **Capture beat** occurs when an occasional atrial impulse reaches the ventricle when it is not in refractory state , leading to the capture of the sinus beat.
- **Fusion beat** occurs when a ventricular and supraventricular impulses simultaneously activate the ventricle , leading to fusion of both the complexes (with an intermediate morphology to look at).

4. Take-Home Message

- The identification of Pseudo P-wave as a part of QRS complex is of much significance while analysing the rhythm pattern of VT. Such a diagnosis on ECG may be missed at the first glance but a systemic approach is always fruitful in such a situation. The detailed and careful analysis of conjoint complex of 'P-QRS' in precordial leads makes the pathway very easy to be understood in favour of VT.
- Truly to say, the QRS complexes , especially over precordial leads should be analysed first because it gives the maximum information about the ventricular runway.
- This would be better to analyse the QRS complexes in conjunction with "Cherchez le P" – a term coined by Marriot (By dictionary Cherchez means 'to look for').
- It would be appropriate to quote the saying of a wise men "when you hear hoofbeats , look for horses , not zebras" , meaning thereby widened QRS wave should be considered first in VT if it is found in conjunction with P-like wave.

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PARASYSTOLE : MATHEMATICAL AND PHYSIOLOGICAL INSIGHTS FROM SURFACE ECG ANALYSIS

ECG

PARASYSTOLE :
MATHEMATICAL AND PHYSIOLGOICAL INSIGHTS FROM SURFACE ECG ANALYSIS

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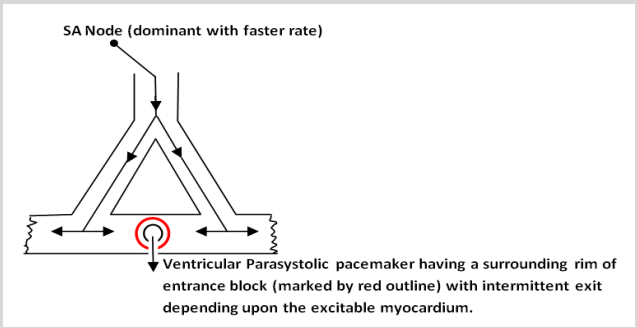
OUTLINE

Introduction
When a sinus pacemaker and a protected independent pacemaker coexist , a characteristic arrhythmia emerges - known as 'Parasystole'.

Epidemiology
Incidence , Age / sex , Race , Risk factors

Natural history

Mechanism – its electrophysiology



Typical ECG findings in parasystole

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

Take-Home Message

References

Parasystole :
Mathematical and Physiological Insights from Surface ECG Analysis

A Narrative Review

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Albert Einstein viewed mathematics as a beautiful, logical language for understanding nature, famously referring to pure mathematics as "the poetry of logical ideas".
The laws of mathematics are certain, and they assist in unveiling underlying reality—often providing a crucial clue in problem-solving.
The physiological insight of parasystole can be appreciated with greater clarity when one moves beyond sole dependence on surface ECG tracings. Parasystole, therefore, may be revisited through the combined laws of physiology, mathematics.

- ☐ **Parasystole represents a unique arrhythmic phenomenon in which an ectopic ventricular focus functions independently of the dominant rhythm, protected by entrance block and governed by its own intrinsic timing.**
- ☐ **An ECG pattern described as "fixed inter-ectopic intervals with variable coupling" clearly calls mathematics into action. This observation guides the clinician toward a more judicious step—marking the parasystolic cycle length with calipers.**

Thus, parasystole is ultimately revealed on the surface ECG through the combined lenses of physiology and mathematics.

1. Introduction (Keypoints)

- Parasystole is a rare arrhythmia that appears on the ECG as a characteristic dual rhythm, generated by two separate autonomous pacemaker foci with different inherent rates. One focus is the sinoatrial (SA) node, the normal dominant pacemaker, while the other is a secondary focus—most commonly located in the ventricle, but it may also arise from the atrioventricular (AV) node or the atria.
- **NORMALLY**, impulse initiation at the SA node dominates over all other subsidiary pacemakers because of its relatively faster discharge rate, achieved through the mechanism of overdrive suppression. Occasionally, however, an independent pacemaker center acquires the ability to fire at its own intrinsic autonomous rate, being protected by an entrance block in its surrounding tissue from impulses originating in the dominant SA node. In other words, this ectopic focus becomes electrically insulated from the dominant rhythm.
- **When a sinus pacemaker and a protected independent pacemaker coexist , a characteristic arrhythmia emerges - known as 'Parasystole'.**
- Parasystole was first observed and reported by **Schamroth in 1967**. He described parasystole as a dual rhythm entity in which the an independent secondary pacemaker

is formed and protected from the effect of the dominant pacemaker SA node : this protection is the essential requisite of this arrhythmia. This protection is considered to lie within the immediate vicinity of the parasystolic focus. Here , SA node – dominant pacemaker hierarchy is not able to suppress this secondary independent pacemaker , known as parasystole , this ectopic focus fires its own intrinsic rate.

- This parasystolic focus is continuously firing , but one can witness this only when its impulse finds excitable myocardium. So, parasystole is not intermittent firing , rather it is a continuous firing with intermittent expression on ECG.

- Parasystole becomes manifest when:
  - Sinus rate slows (sleep, ischemia, drugs)
  - Conduction delays get increased
  - Myocardial refractoriness changes

- Parasystole is often benign , rarely degenerates into malignant arrhythmia by itself

- **In nutshell**, Parasystole is the coexistence of two independent pacemakers, where the ectopic focus is protected from sinus impulses by entrance block and expresses itself whenever the myocardium is excitable

2. Epidemiology

- **Incidence** : This is a very rare arrhythmia with the incidence of 0.13 percent of all the electrocardiograms recorded in a general hospital.

- **Age / sex** : Patients of all age groups may have this arrhythmia but it is commonly encountered in older patients , usually more than 60 years of age.
  Males are more commonly affected , compared to females with an approximate ratio of 2:1.

- **Race** : No race is immune to parasystole.

- **Risk Factors**
  - The most common association with this arrhythmia had been found with atherosclerotic heart disease and / or hypertensive cardiovascular disease (60 percent) and surprisingly half of these patients were found to have congestive heart failure in addition. Other acquired and congenital heart diseases were also observed to be associated with this arrhythmia.
    (The association of parasystole with heart diseases is not definitely proven and such arrhythmia is mostly benign and self limited)
  - Some cases (14 percent) were found to be idiopathic in nature without any evidence of heart disease.
  - Since the main requisite of this arrhythmia is the presence of a secondary pacemaker in heart , any cardiac arrhythmia might work as a rich factor.
  - Sometimes its occurrence is seen with digitalis toxicity.

3. Natural history

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

4. Parasystole and its electrophysiology

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

(i) **A concept** : Parasystole represents a protected automatic focus firing at a fixed intrinsic rate, intermittently expressing itself among sinus beats with variable coupling but constant inter-ectopic timing.
It would be worthwhile to visualize the following simple sketch to understand the electrophysiology of parasystole in depth :

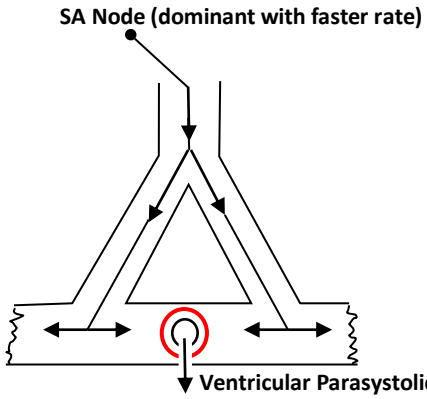

Fig. 1.1 **Ventricular Parasystolic pacemaker having a surrounding rim of entrance block (marked by red outline) with intermittent exit depending upon the excitable myocardium.**

(ii) **Entrance block : ** The parasystolic ventricular pacemaker is protected from the overdriven jolt of the dominant SA node and this happens by its entrance block – the dominant impulses of the sinus origin fail to enter into the domain the parasystolic pacemaker.
Such entrance block around the ventricular pacemaker is unidirectional – its exit allows the activity from this secondary pacemaker to proceed onwards , provided the myocardium in vicinity is non-refractory. .

(iii) **Exit block VS Entrance block :**

Property	Entrance block	Exit block
• Function	Protects focus from suppression	Limits expression
• Effect on timing	Preserves fixed cycle	Does not change cycle
• ECG role	Allows parasystole	Makes it intermittent

Most parasystolic foci have :
Complete entrance block
Partial exit block
That's why, one observes variable coupling interval with fixed inter-ectopic intervals.
Parasystolic rhythm is not a continuous one.

NB : The entrance block occurs because the tissue surrounding the ectopic pacemaker is functionally or anatomically refractory, so incoming sinus impulses cannot depolarize it.

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

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

(vi) **Fusion beat :** Occasional discharge from both the pacemakers (sinus and parasystole) do occur concurrently resulting in the fusion of both the impulses , and hence having a configuration in between these two. This is a surer point to indicate that two foci of simultaneous running rhythm are in operation.

(vii) **Modulated parasystole** : There may be at times an electronically active zone surrounding the ectopic parasystolic site , which can allow the rhythmic activity of the surrounding tissue to have its impact over the periodicity of the parasystolic discharge rate. In other words , the barrier of the entrance block may not be complete. Subthreshold inputs from this surrounding tissue can cross the entrance barrier to depolarize the myocardium. This modulation phenomenon may alter the timing of parasystolic stimulation through the mechanism of delay or acceleration.

(viii) **Parasystole of all types begin and end spontaneously**.

5. Typical ECG Findings in Parasystole

Typical ECG findings are illustrated by the following strip of standard lead II.

Fig. 1.2

A = Sinus beat

B = Parasystolic ventricular beat

C = Fusion beat (resulting from simultaneous activation by sinus and parasystolic impulses)

A–B = Variable 'coupling intervals' between sinus beats and parasystolic ventricular beats , a hallmark of parasystole reflecting electrical independence of the both ectopic foci.

C = Fusion beat , indicating simultaneous depolarization of the ventricles by both the sinus impulse and the parasystolic focus.

NB : The longest interectopic interval is almost exact multiple of the shortest basal interectopic interval , supporting the presence of an independent parasystolic rhythm. Accurate assessment of coupling interval variability and interectopic intervals requires caliper measurement.

The coexistence of variable coupling intervals, fusion beats, and mathematically related interectopic intervals constitutes electrocardiographic proof of parasystole.

6. Take-Home Message

- ☐ Parasystole is characterized by the coexistence of these two independent automatic pacemakers with distinct intrinsic rates, protected by entrance block. Their impulses are expressed only when an exit pathway is available and the surrounding myocardium is excitable at the moment of impulse generation.
- ☐ The fixed inter-ectopic interval is the most reliable ECG clue. The presence of variable coupling interval is also the cardinal sign.
- ☐ Fusion beats are physiological evidence of dual rhythm coexistence.
- ☐ The parasystolic pacemaker may be located anywhere in the heart but is commonly situated in the ventricles , less commonly in the AV node and rarely in the atria.

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

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REPOLARIZATION HETEROGENETIY EXPRESSED THROUGH THE T-WAVE : LOSS OF TRANSMURAL REPOLARIZATION SEQUENCE

ECG

REPOLARIZATION HETEROGENEITY EXPRESSED THROUGH THE T-WAVE : LOSS OF TRANSMURAL REPOLARIZATION SEQUENCE

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OUTLINE

Repolarization Heterogeneity expressed through the T-wave : loss of transmural repolarization sequence

A Narrative Review

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A coherent set of steps is essential in life to achieve its goal. When it is broken down into incoherent steps, the entire aim in life is collapsed to failure. A creative step turns into a destructive step with a fragmented turn. Thus comedy turns into tragedy.

The heart is gifted with two steps during each cardiac cycle , namely depolarization and repolarization. Repolarization process is inherently different from that of depolarization.

- ☐ **Repolarization is the electrical recovery phase of ventricular myocytes aiming at to resume its resting polarity without creating chaos of voltage gradient across them.**
- ☐ **But the repolarization heterogeneity is an unwanted guest – it is the electrophysiological soil from which malignant arrhythmias germinate.**

The T-wave heterogeneity in this context signifies underlying loss of orderly repolarization in myocardial layers, associated with local voltage gradient – a substrate for arrhythmias by promoting re-entrant circuits.

1. Introduction (keypoints)

- Cardiac repolarization is a critical physiological process that resets myocardial electrical events to prepare for the next contraction. T-wave is a very important member of this repolarization process.
- Under normal physiological conditions, an uniform and disciplined myocardial cells repolarization occurs in a sequential manner , epicardial cells first followed by endocardial cells and lastly the M-cells. This cellular anatomical integrity as such is expressed through the T-wave.
- Normally the action potential durations amongst these three cellular elements differ, the gap between successive layers is extremely short generating practically no voltage gradient on the surface ECG. As a result a normal T appears as a smooth positive wave with a slight asymmetry.
- Repolarization heterogeneity expressed through T-wave is a state of non-uniform recovery of group of cells such as epicardium , endocardium and M-cells resulting in voltage gradient across them, acting as the culprit architect of electrical instability. This phenomenon accounts for ventricular arrhythmias.

- Multiple studies have emerged out showing the direct correlation of T-wave with spacial and temporal dispersion of repolarization across the myocardium. Further study has also added that T-wave changes capture non-linear and dynamic repolarization process vividly by providing a pivotal insight beyond traditional QT interval measures.
- The T-wave corresponds to repolarization (phase 3), where heterogeneity may be the maximum during its altered electrophysiological journey.

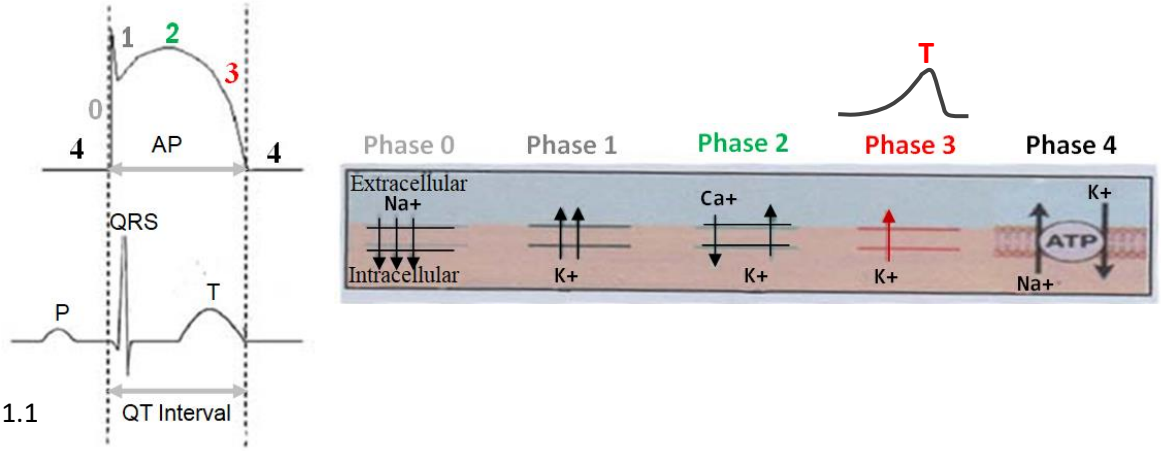

Fig. 1.1

2. A brief review of sequential myocardial layers repolarization , reflected through the T-wave

This would be worthwhile to mention in this context that studies in vitro revealed three types of myocardial cells during repolarization phase 3.

Epicardial cells, Endcoardial cells and M-Cells. Endocardial and M-cells are having the longer Action Potential Duration (APD) while Epicardial cells are having the shortest APD and that's why , epicardial cells are repolarized earlier, followed by those of Endocardial and M-cells respectively.

Epicardial cells →Endocardial cells →M-cells

This orchestral temporal sequence produces upright T-wave on surface electrocardiogram (ECG).

Such sequential synchronization of epi-endo-M cells is a crucial step to prevent repolarization heterogeneity to the extent of reaching arrhythmogenic level.

Sicouri and Antzrelevitch associates described a subpopulation of cells in the deep subepicardium that displayed distinct electrophysiological properties. These mid-myocardial cells (i.e.. **M cells**) exhibited the longer action potential duration (APD).

Experiments in vivo do not confirm the presence of M-cells but it has been shown that APD recording in vivo is always significantly less than those recorded in vitro – a question mark ? to the completely being absence of M-cells in vivo , signifying the presence of these cells in vivo in subquantum population.

The steps of sequential repolarization are illustrated , as below :

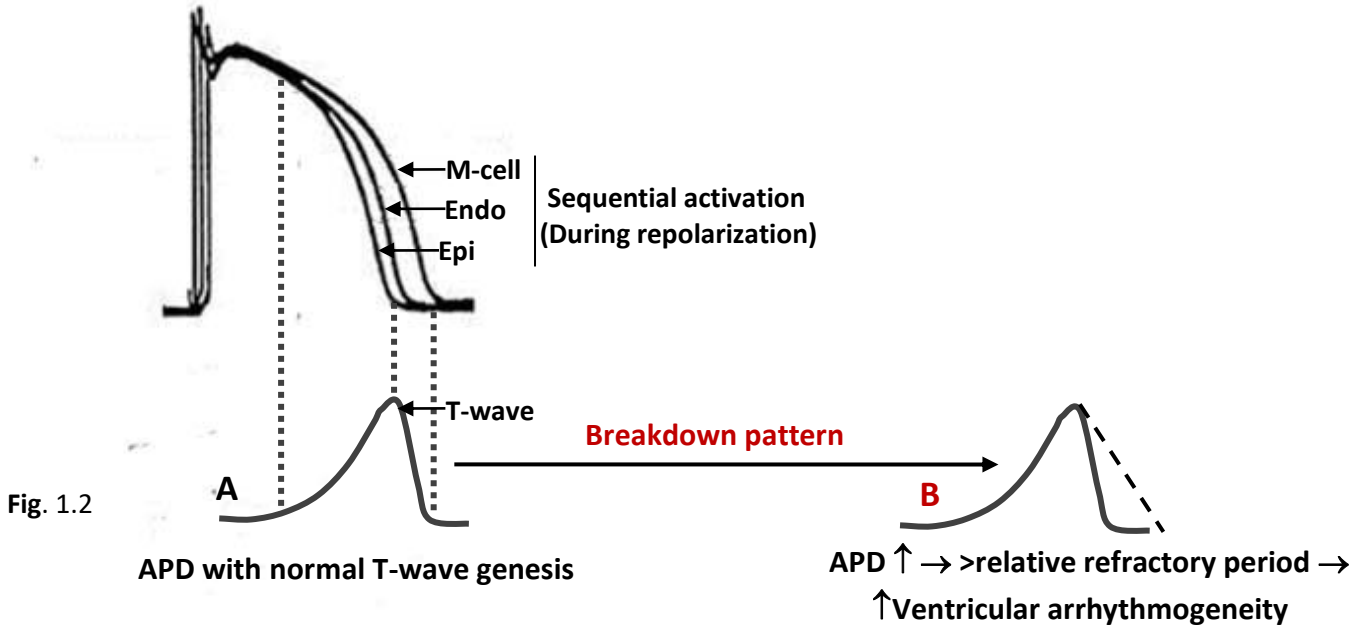

Fig. 1.2

It has been shown that there exists anatomically a zonal differences with sequential activation over the different parts of the ventricle during repolarization – apicobasal , anteroposterior and left to right. This makes the heart capable to perform its function smoothly and coherently starting from apex to the base.

These T-wave changes with underlying loss of orderly repolarization in myocardial layers create a substrate for arrhythmias through local voltage gradient.

Discussing repolarization heterogeneity dependent upon T-wave provides a powerful , non-invasive clinical tool to assess arrhythmia risk and thereby myocardial electrical remodelling as well.

This entire episode can be summarized as below :

- **Alteration in transmural cellular expression-pattern (Epicardial-Endocardial-M-cells)**
- **A change over the pattern of temporal spread (apicobasal , anteroposterior and from L to R)**

↓

Altered expression
(either singly or in combination)

↓

Incoherent repolarization heterogeneity (ventriuclar repolarization dispersion) leading to life threatening ventricular arrhythmias and sudden cardiac death

3. Electrophysiological Shift : How Heterogeneity comes in action

The loss of sequential myocardial repolarization is fundamentally caused by several mechanisms :

A. Endocardial injury (ischemia / strain)

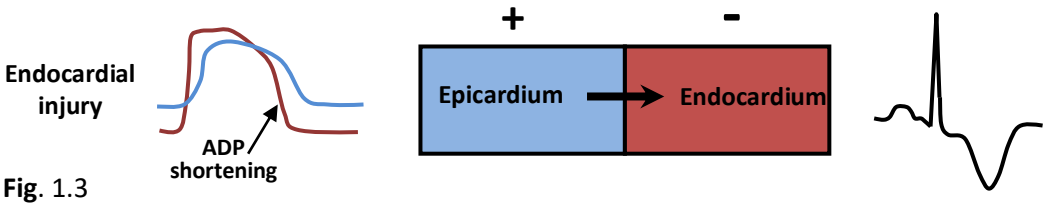

Fig. 1.3

- Endocardium suffers prolonged depolarization
- Subsequent repolarization becomes relatively shorter compared to epicardium
- The resultant volatage gradient causes the flow of current from epicardium to endocardium causing ST depression and symmeterical T-wave inversion.

B. Epicardium injury (ischemia / inflammation)

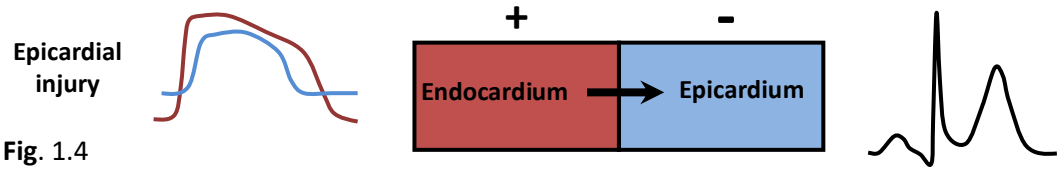

Fig. 1.4

- Epicardium repolarizes somewhat later
- Endocardial repolarization precedes it
- The resultant voltage gradient causes the flow of current from endocardium to epicardium causing ST elevation with upright peaked or hyperacute T-wave.

C. M-cell augmentation

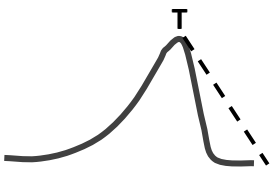

Fig. 1.5

M-cell augmentation occurs because the efflux of potassium ions during repolarization is delayed , leading to prolonged repolarization. This prolonged APD in M-cells creates an electrical gradient across the ventricular wall. Some QT prolonging drugs , electrolyte imbalances (particularly potassium) or congenital channelopathy may prolong ADP in M-cells and so there is exaggerated transmural dispersion which may be expressed through T-wave alternans , long QT related changes or Torsades de pointes.

- **T-wave alternans**

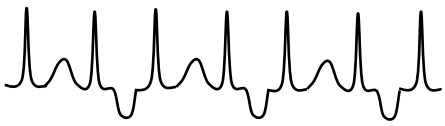

Fig. 1.6

This can be used as a ECG marker to predict the malignant arrhythmias and sudden cardiac death (SCD)

- **Torsades de pointes**

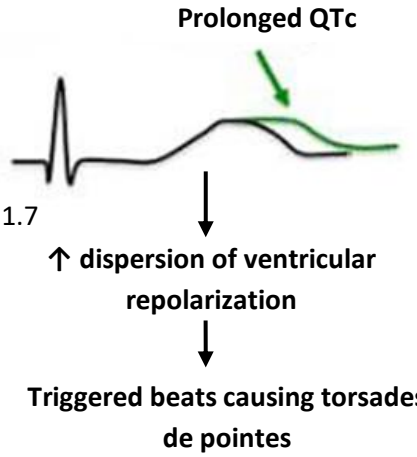

Fig. 1.7

QTc prolongation may result in enhanced repolarization dispersion , triggered beats falling upon this may ensue Torsades de pointes.

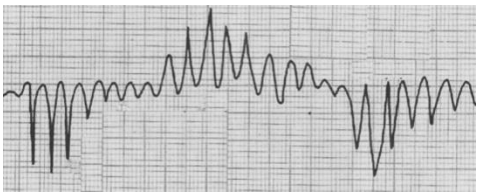

Fig. 1.8

4. Repolarization heterogeneity expressed through the different shapes of T-wave on ECG

- T-wave inversion → localized endocardial injury
- Upright T-wave with ST segment elevation → localized epicardial injury
- Tall and peaked T as in hyperkalemia
- Biphasic T (expresses dynamic reperfusion as with Wellens' syndrome)
- T-wave alternans with beat-to-beat repolarization variation (It predicts the malignant arrhythmias ± sudden cardiac death)
- Prolonged T-peak – T-end interval (Tp – Te) : It parallels with prolonged QT interval.

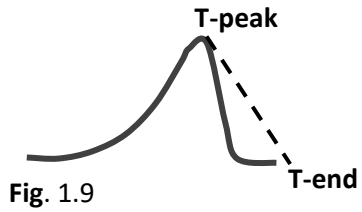

Fig. 1.9

The normal range for the Tp-Te interval (T wave peak to T wave end) on ECG in healthy adults is typically between 50 and 90 milliseconds (ms), with a median value around 70 ms.

- Discordant T-waves is a T-wave that points to the opposite direction to that of corresponding QRS complex. It is a measure of electrical difference between depolarization and repolarization.

5. Clinical usefulness

Repolarization heterogeneity as reflected through the different shapes of T holds definite diagnostic and prognostic value.

➢ **Earlier pointer to ischemia**
Changes in T-wave morphology often precedes ST deviation, making them useful for early diagnosis.

- **Assessement with LVH and cardiomyopathy**
  Here the changes reflect increased wall stress and fibrosis burden. And the concerened lateral leads show down-sloping ST depression and inverted T-waves.

- **Electrolyte disorders**
  - Hyperkalemia : Tall peaked T
  - Hypokalemia : Flat T with U wave prominency

- **Risk stratification in Channelopathy**
  Tp – Te measurement in the identification of impending Torsades risk , even when QTc is borderline.

- **Long congential QT syndrome**
  Broad-based T (LQT1) , low amplitude and bifid T (LQT2) , long ST segment with late T (LQT3).

Numerous additional clinical implications are associated with recognizing different T-wave contours that reflect repolarization heterogeneity.

6. Take-Home Message

- ☐ Under normal physiological conditions, an uniform and disciplined myocardial cells repolarization occurs in a sequential manner , epicardial cells first followed by endocardial cells and lastly the M-cells. This cellular anatomical integrity as such is expressed through the T-wave.
- ☐ Alteration in this transmural cellular expression pattern (epicardial-endocardial-M cells) may bring incoherent repolarization , termed as Repolarization Heterogeneity.
- ☐ Repolarization heterogeneity is a fundamental electrophysiological phenomenon that find its distinct surface expression through the T-wave.
- ☐ Careful observation of T-wave morphology provides a powerful , non-invasive clinical tool to assess arrhythmia risk.
- ☐ These T-wave changes with underlying loss of orderly repolarization in myocardial layers create a substrate for arrhythmias through local voltage gradient.

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NB : Consulted and discussed with ChatGPT whenever needed

Personal Notes :