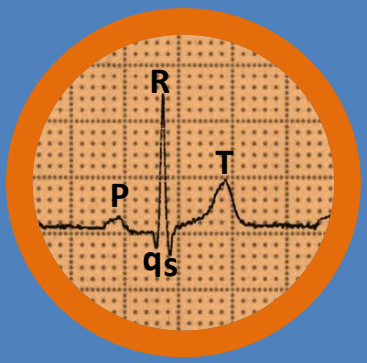

ECG Review : Ventricular Arrhythmia (Part-1)

(For Academic Purpose only)

Dr. DURGA PRASAD KHAITAN

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

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

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Vents race wild in electric gale ,
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Knowledge and skill in the field of electrocardiography are constantly changing with the new researches and understanding.

With humble words I wish to say that 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

BRUGADA ALGORITHM ON ECG : A PYHSIOLOGY-DRIVEN CONCEPT

ECG

BRUGADA ALGORITHM ON ECG : A PHYSIOLOGY-DRIVEN CONCEPT

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OUTLINE

Brugada Algorithm on ECG : A Physiology-Driven Concept

A Narrative Review

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Understanding through conceptual insight builds a mental map of reality and cultivates a critical approach to think over. Concepts are fundamental to learning and the construction of knowledge both. The concept of ventricular tachycardia (VT) began to take shape with the pioneer working of the Brugada et al. — P. Brugada, J. Brugada, L. Mont, J. Smeets, and E.W. Andries — they introduced a new approach to the differential diagnosis of regular tachycardia with a wide QRS complex and proposed four steps diagnostic algorithm for the purpose. Prior to this, such a mechanistic algorithm was not prevailing, and their work profoundly fascinated and reshaped the field of cardiology.

- ☐ **Normally the cardiac activation starts from the SA node and afterwards, it spreads downwards bi-directionally through HIS-Purkinje system , the resultant wavefronts are expressed sequentially across V1-V6 , with a mid-transition inscription of RS (V3/V4).**
- ☐ **In VT the story of electrical journey starts de novo from a single ventricular focus , the resultant depolarization wavefront travels in one dominant direction , passing slowly through muscle-to-muscle domain.**

VT is favoured when the normal physiological sequence of ventricular activation is lost or distorted. Based on this principle, the Brugada algorithm converts ventricular conduction physiology into a simple four-step bedside diagnostic tool.

1. Introduction (Keypoints)

- In year 1991 , the Brugada algorithm came into existence when Brugada et al. published their article titled as **"A new approach to the differential of regular tachycardia with a wide QRS complex"** , published in the journal **'Circulation'.** Before such publication wide QRS complex tachycardia was remaining a diagnostic chaos. Such diagnostic dilemma seemed to be the result of improper interpretation of existing criteria at that time. The cardiologists of that era at times mislabelled VT as SVT by making their observation exclusively based on morphological criteria (such as QRS width , associated bundle branch block , axis determination , etc.). This approach was associated with a higher mortality rate which proved to be a driving force to search out a better diagnostic alternative.
- This was the environment prevailing during that era which compelled the minds of Brugada et al. to search out a new ECG based bedsite diagnostic algorithm for the purpose. This entire group investigated the reasons for the failure of existing criteria and put a new criteria and incorporated them in stepwise approach that provided better

sensitivity and specificity to have a more correct answer. This new stepwise algorithm prevented the prevailing diagnostic mistakes to a greater extent.

- Prior to entailing different steps of Brugada algorithm , there is a pre-requisite need to understand the normal sequential activation of wavefronts running across V1-V6 with RS inscription in the midway of its precordial journey , illustrated as below :

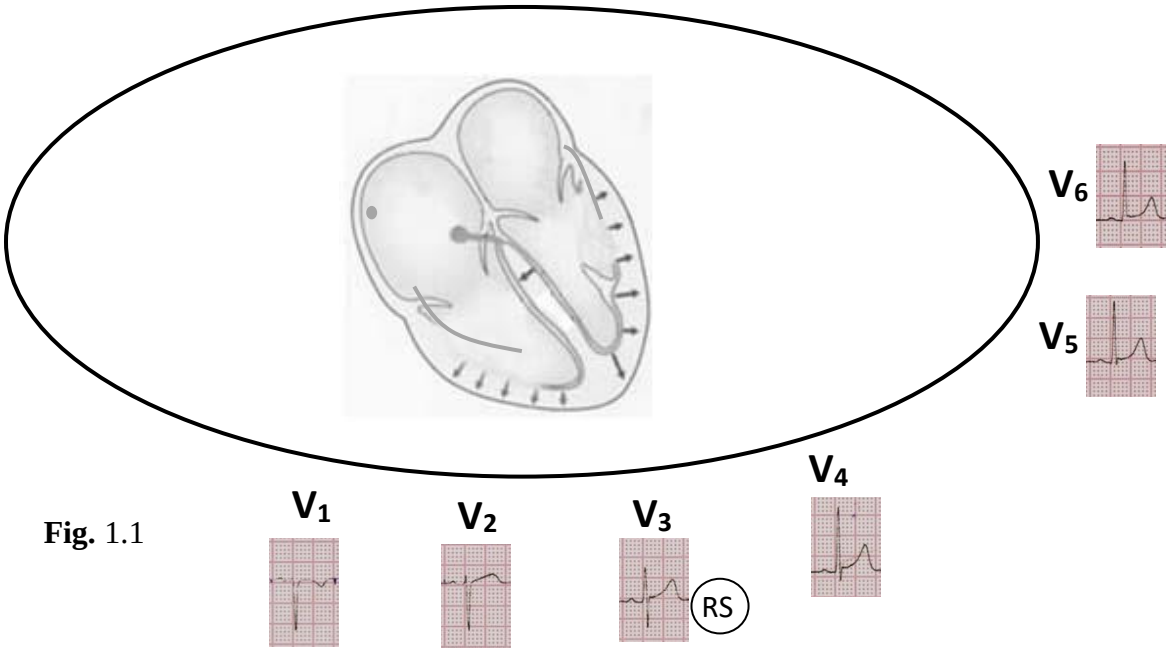


Fig. 1.1

Eyes of Precordial Leads

A remarkable point is to be noted here :

Any precordial lead from V1 to V6 records the electrical potential from the small area of the underlying myocardium – all the concerned net electrical events are viewed from that selected precordial lead.

The following steps illlustrate how His-Purkinje based ventricular activation occurs in a sequential manner across V1-V6 **(with Brugada alogirithm this sequential activation is lacking)**

Initial activation uses His-Purkinje system

↓

Septum activation occurs from left to right (septal wave r/q)

↓

Depolarization spreads downwards bidirectionally (synchronized ventricular activation)

↓

Sequential spread of wavefront across V1-V6

rS (V1-V2) → **RS** pattern V3/V4 → qR (V5-V6)

- **Electrophysiological basis of this newer diagnostic algorithm**.
  If this concept is understood properly , every step of Brugada algorithm might be understood with a more precision accuracy.
  "In VT the story of electrical journey starts de novo from a single ventricular focus. The resultant depolarization wavefront travels in one dominant direction , passing slowly through muscle-to-muscle domain".
  This breakthrough of depolarization pattern in a way as described is the diagnostic foundation of VT.

2. Spatial layout of Brugada algorithm

Brugada et al. laid down four steps oriented diagnostic algorithm in context with regular tachycardia with a wide QRS complex. The following sketch is the self-explanatory :

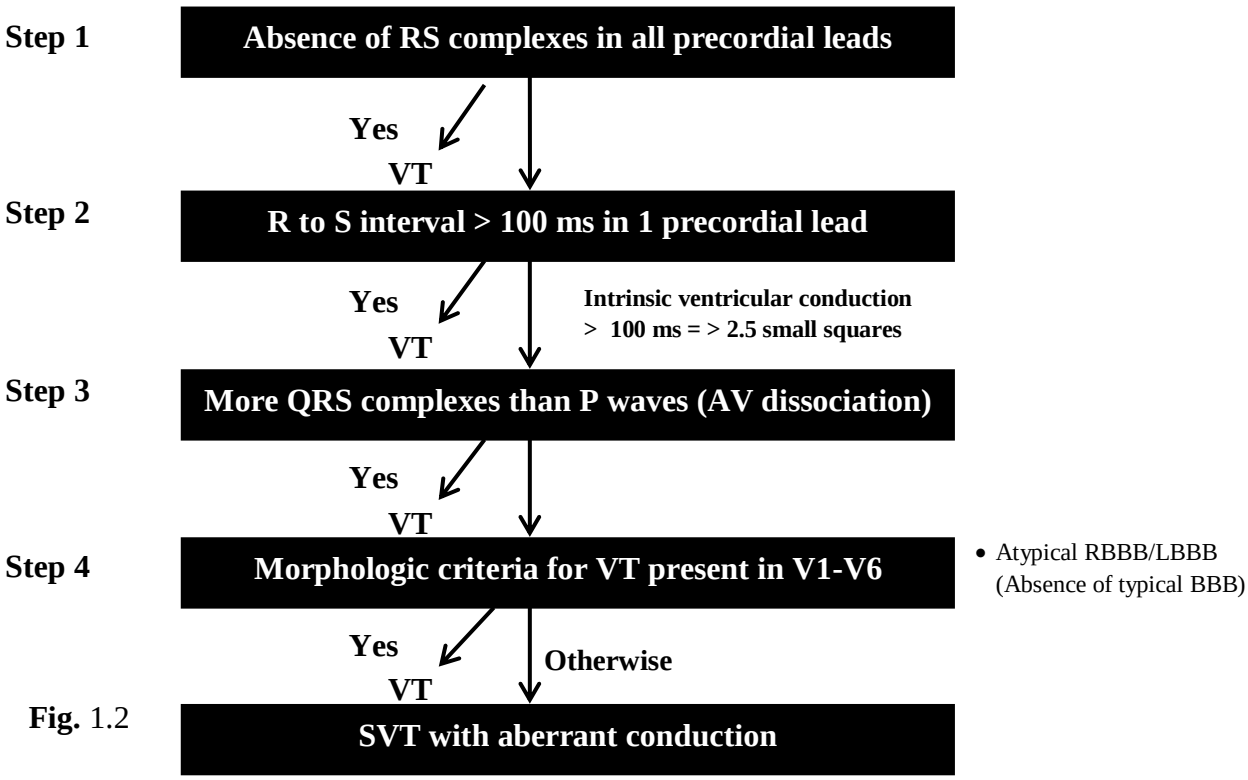


Fig. 1.2

NB : How Brugada validated this algorithm :

- Studied well-characterized EP-proven cases
- Compared algorithm vs expert cardiologist-opinion
- Sensitivity for VT ~ 98%
- Far superior to isolated criteria

This would be worthwhile to mention here that each step of this Brugada algorithm represents the integrated approach one after the other , proving the same fact that the resultant repolarization wavefront travels in one dominant direction.

3. A conceptual bridge between electrophysiology and bedside ECG interpretation of Brugada Algorithm

Brugada et al. established a four-step diagnostic algorithm for assessing regular tachycardia with a wide QRS complex. It would be beneficial initially just to laying down each step, its electrophysiological basis, and the corresponding sensitivity and specificity.

	Step content	**Physiological justification for VT**	**Sensitivity / Specificity**
Step 1	Absence of RS complexes in all precordial leads (V1–V6)	This reflects loss of early bidirectional ventricular activation due to myocardial-origin of VT	Sensitivity ≈ 21–25%; Specificity ≈ 98–100%
Step 2	RS interval > 100 ms in any precordial lead	Prolonged initial-to-nadir ventricular activation reflects slow muscle-to-muscle conduction and delayed ventricular mass recruitment, inconsistent with rapid Purkinje-mediated activation	Sensitivity ≈ 60–70%; Specificity ≈ 90–95%
Step 3	Presence of AV dissociation	Independent atrial and ventricular activation confirms ventricular origin of tachycardia, excluding supraventricular mechanisms	Sensitivity ≈ 20–30%; Specificity ≈ ~100%
Step 4	Morphological criteria for VT (RBBB or LBBB patterns) Observer variability	This step is based on the fact that VT does not use the normal septal conduction pathway , so its wide QRS fails to follow the typical RBBB or LBBB patterns seen in SVT with aberrancy.	Sensitivity ≈ 60–80%; Specificity ≈ 70–90%

NB :

- The Brugada algorithm was intentionally structured to prioritize highly specific mechanistic indicators of ventricular origin before resorting to morphology-based criteria, thereby minimizing false-positive diagnosis of VT / SVT both.
- The most important truth (often missed) is :
  - Specificity drops mainly at step 4 and that's why it should never be used alone.
  - **The step 4 is somewhat difficult to be analysed even by the experts.**
- Every step is directed towards the mechanistic laying out but with different manifestation .
- Therefore , this algorithm should be viewed as sculpt directed towards the mechanism – driven narrative. Such a mechanistic diagnostic algorithm is the utmost need amongst the clinicians .

4. Descending into the depth of Brugada algorithm for conceptual clarity

Physiological basis : The Brugada algorithm translates ventricular conduction physiology into a practical bedside diagnostic sequence.

STEP 1: Absence of RS complexes in all precordial leads (V1-V6)

This reflects loss of early bidirectional ventricular activation due to myocardium-origin of VT. At this stage though there is absence of RS complexes throughout the chest leads , there is appearance of some altered ECG patterns in the support of VT. **Here many clinicians face diagnostic dilemma by asking why these peculiar ECG patterns are coming into play.**

❒ **The Answer is : Altered ECG patterns as like R , QS , QR, or rS may appear as per the location of ventricular focus in the myocardium , as illustrated below :**

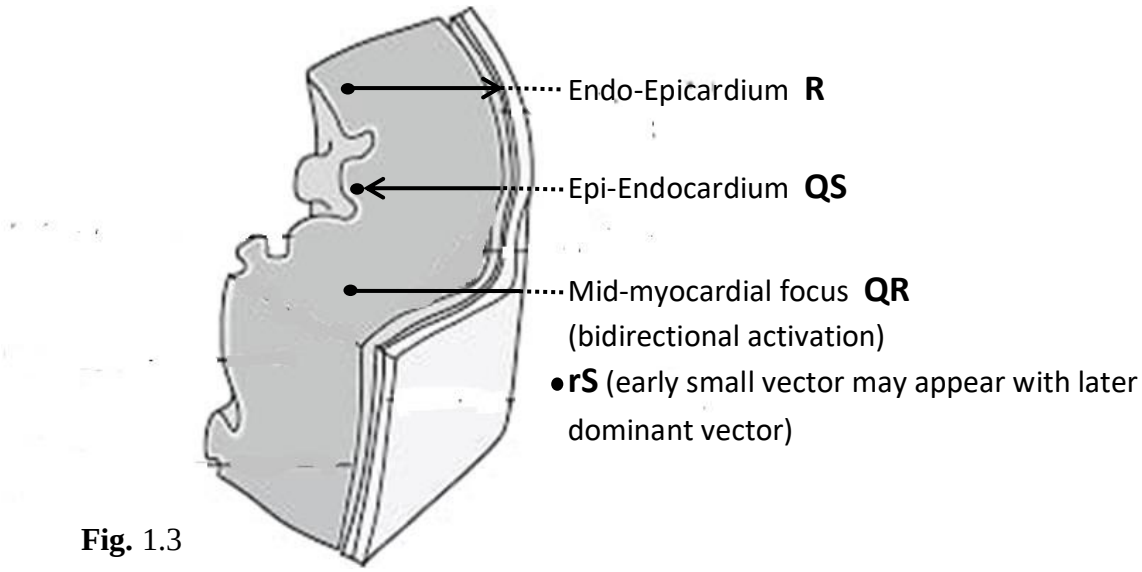


Fig. 1.3

❒ During ventricular tachycardia, a positive deflection is recorded in the precordial leads when the activation wavefront propagates toward the exploring electrode, typically from endocardium to epicardium. Conversely, a negative deflection occurs when activation spreads away from the exploring electrode, as in epicardial-to-endocardial propagation. When the VT focus arises from the mid-myocardium, ventricular activation occurs bidirectionally, producing a more complex and often balanced QRS morphology.

❒ **Some tracings prioritize urgent cardioversion in unstable patients ,** as illustrated below

- Negative concordance: QS or predominantly negative QRS throughout in V1-V6 , originating from the posteriorly situated left ventricle .
- Positive concordance : Monophasic R waves in all precordials, typical of apical septal VT focus.
- QR complexes in all precordial leads is highly specific for VT.
  (This implies ventricular activation not through normal His–Purkinje system).

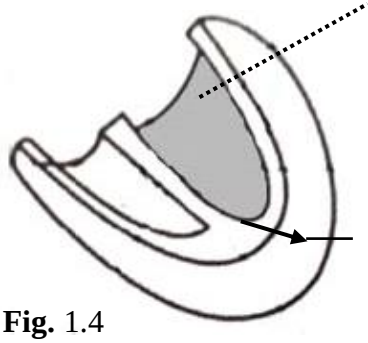


Fig. 1.4

In nutshell , absence of an RS complex in all precordial leads (V1-V6) in wide-complex tachycardia indicates ventricular tachycardia (VT) as the first step of the Brugada algorithm, with patterns like R, QS, QR, , or rS confirming this.

STEP 2

❒ **VT Conduction Physiology** : Normal QRS uses rapid His-Purkinje fibers for sequential endo- to epicardial activation; VT originates focally (e.g., scar), propagating cell-to-cell with phase delays—journey "start" at exit site, "dropout" at boundaries causing notched/slurred S nadir.

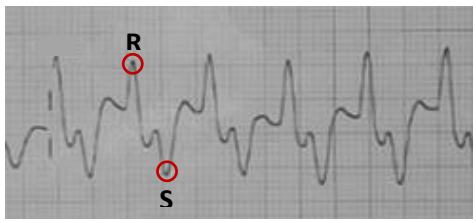


Fig. 1.5

❒ **Quantitative components :** RS interval from R-wave onset to S-wave nadir exceeding 100 ms in any precordial lead indicates VT due to delayed activation.
R-wave peak time ≥40 ms plus ≥60 ms to S-nadir , combination of both = > 100 ms

❒ **RS interval may be falsely prolonged in:**

- Severe hyperkalemia
- Sodium-channel blocker toxicity
- Massive ventricular hypertrophy

Hence : Always integrate with clinical context

STEP 3

❒ **AV dissociation means :**

- Atria and ventricles depolarize independently
- Ventricular rate ≥ atrial rate
- P waves may march through QRS , appear before, within, or after QRS or it may be visualized over the tip of T-wave

❒ **Site of visualization of P-wave**

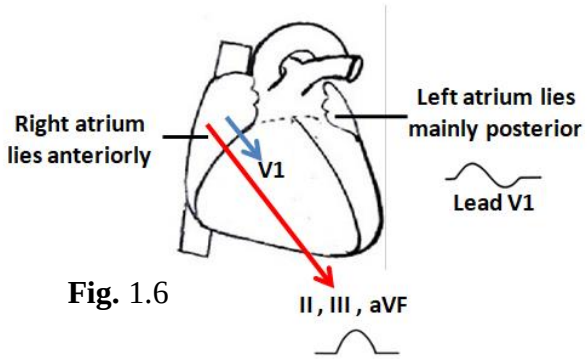


Fig. 1.6

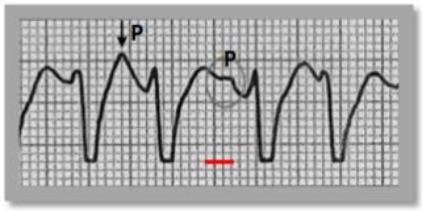


Fig. 1.7

Atria and ventricles depolarize independently

Check inferior leads (II, III , aVF) / V1 , where P-wave often stand out best to be visualized.

❒ Capture / Fusion beat : indirect evidence of 'P' in AV dissociation

- Capture beats (normal-looking narrow QRS within wide tachycardia)
- Fusion beats (hybrid morphology)

Fig. 1.8

❒ Absence of AV dissociation does not exclude VT. Presence confirms VT.

STEP 4 :

❒ **The basic anatomical and physiological part**

- Since the right ventricle is situated on front and the left ventricle lies somewhat deeper, ventricular activation in VT progresses obliquely across the chest leads. This depth-dependent delay explains the atypical and non-physiological bundle branch block morphology seen in VT.

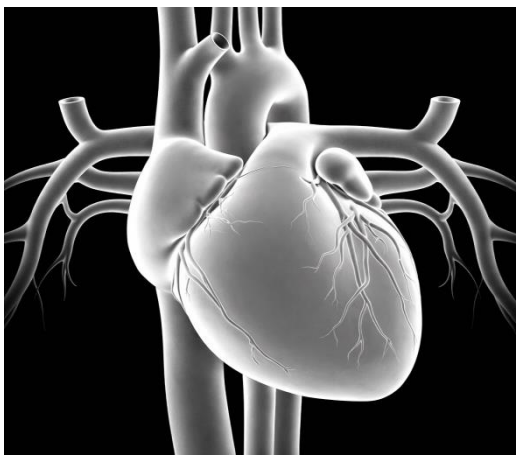


Fig. 1.9

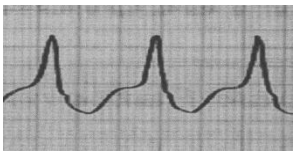
- The electrical activation fields in between two ventricles partially overlap within the precordial projection , particularly over lead V1. During ventricular tachycardia, abnormal propagation across this overlapping electrical zone produces fused and slurred QRS components, accounting for the atypical bundle branch block patterns seen on the ECG.

- If the current flows towards the exploring lead , it records positive deflection and when the current flows away from the exploring electrodes , it records negative deflection.

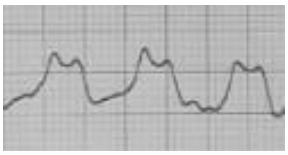
❒ **Right bundle branch block (RBBB) - like pattern**

The lead V1 records monophasic R, , taller R than R' (Left rabbit ear) or qR while the opposite counterpart V6 records more or less mirror image.

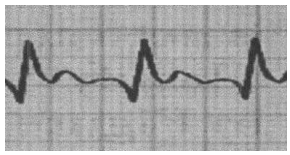
With RBBB current flows from LV to RV.



'Monophasic R'



'Left rabbit ear'



Biphasic qR

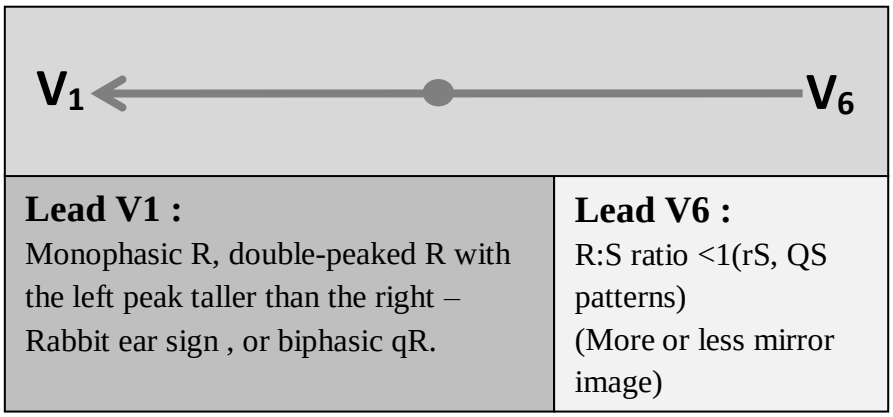


Fig. 2.0

NB :

- **V1** ← ● → **V6**
  - This symbol points toward 'overlap zone' (the electrical actication fields in between two ventricles partially overlap within the precordial projection , particularly over lead V1 , accounting for the atypical RBBB-like pattern , as illustrated above.
- QRS duration > 140 ms with RBBB-like pattern suggests VT.

❒ **Left bundle branch block (LBBB) - like pattern**

The lead V1 records smooth initial R>40ms with RS nadir>60ms and the opposite counterpart V6 records QS , QR or qR pattern.

With LBBB current flows from RV to LV (QRS duration > 160 ms).

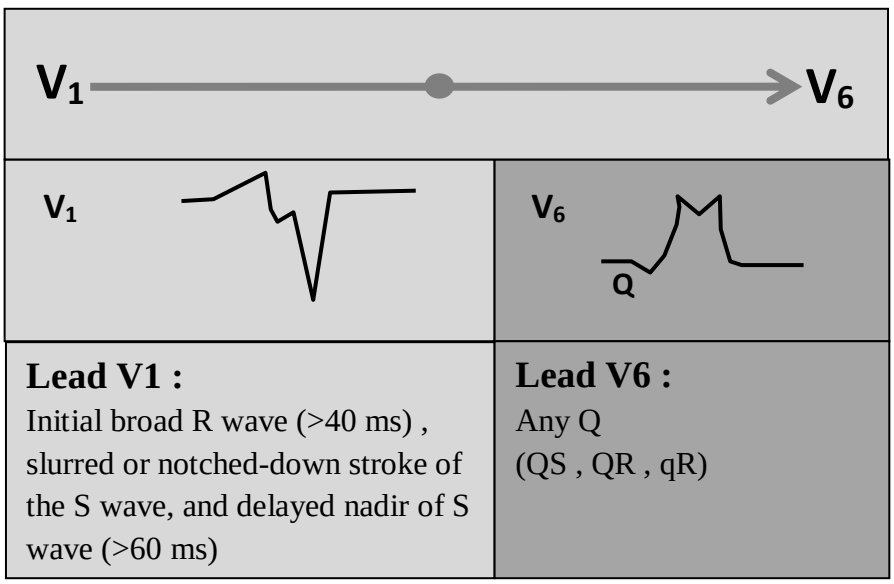
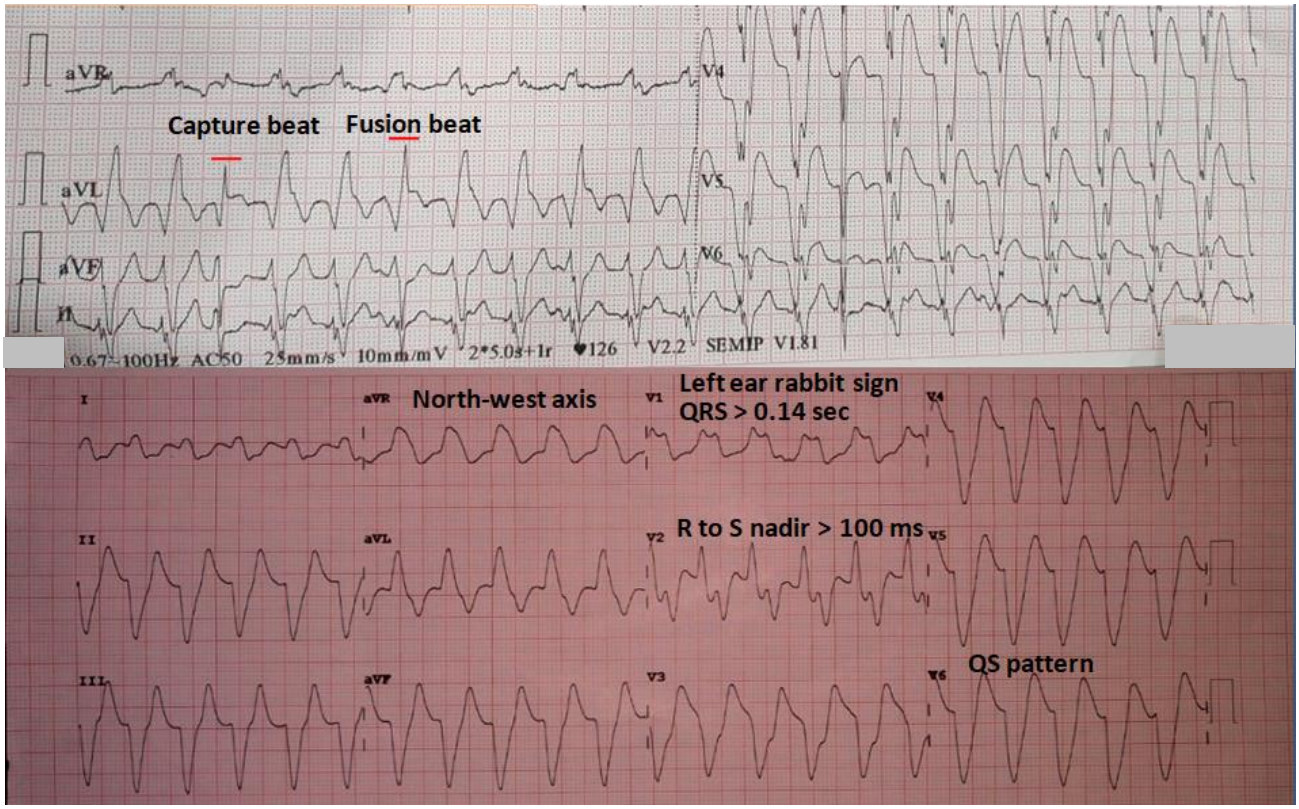


Fig. 2.1

5. ECG illustration : self explanatory



6. Take-Home Message

☐ In year 1991 , the Brugada algorithm came into existence when Brugada et al. published their article titled as **"A new approach to the differential of regular tachycardia with a wide QRS complex"** ,

☐ **A Phsyiology- Driven Concept**

- This algorithm interrogates how the ventricular activation begins in myocardium and how does it spread across the precordial leads.
- In VT the story of electrical journey starts de novo from a single ventricular focus , the resultant depolarization wavefront travels in one dominant direction , passing slowly through muscle-to-muscle domain.
- This Brugada algorithm thus translates ventricular conduction physiology through precordial leads . Any precordial lead from V1 to V6 records the electrical potential from the small area of the underlying myocardium – all the concerned net electrical events are viewed from that selected precordial lead.

☐ **Brugada Algorithm Overview**

The Brugada algorithm sequentially evaluates four steps: absence of RS complex in precordial leads (step 1), RS interval >100 ms in precordials (step 2), AV dissociation (step 3), and QRS morphology in V1/V6 (step 4).

Step 3 checks the full 12-lead ECG for AV dissociation, fusion beats, or capture beats anywhere, not limiting to precordials, to achieve high specificity (~100%) for VT.

- **Sensitivity vs Specificity**
  Brugada excels in overall sensitivity (around 90%) with moderate specificity (60-90%). It performs well with RBBB-like patterns but not so with LBBB like morphology and it may also may falter in idiopathic VT or pre-existing bundle branch block (The step 4 is somewhat difficult to be analysed even by the experts).

- **Clinical application**
  This criterion does not stand 100% true scanner for VT analysis.
  Select Vereckei as a helping hand if the rhythm shows LBBB-like morphology, suspected idiopathic VT or any doubt exists in the interpretation of Brugada algorithm.

- **Use the Brugada algorithm** as the primary approach for differentiating wide QRS complex tachycardia (WCT) into ventricular tachycardia (VT) versus supraventricular tachycardia (SVT) with aberrancy, especially when a comprehensive, multi-lead assessment across precordial leads (V1-V6) is feasible in stable patients.

7. References

All the articles with the references mentioned against the internet links are retrieved from therein.

---

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   Oct 8, 2024
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   Authors: Federico Moccetti, Pedro Brugada, Gregorio Tersalvi, Mrinal Yadava, Yllka Latifi, Benjamin Berte, Richard Kobza, Christian Sticherling, and Tobias Reichli
   22 Feb , 2023
   https://www.jacc.org/doi/10.1016/j.jacep.2022.12.017

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   András Vereckei
   Aug 2014
   https://pmc.ncbi.nlm.nih.gov/articles/PMC4040878/

NB : Consulted and discussed with ChatGPT whenever needed

VERECKEI CRITERIA THROUGH LEAD aVR : A TWO PATHWAY (VT/SVT-A) DIAGNOSTIC APPROACH

ECG

VERECKEI CRITERIA THROUGH LEAD aVR : A TWO PATHWAY (VT/SVT-A) DIAGNOSTIC APPROACH

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Vereckei Criteria through Lead aVR : A two pathway (VT/SVT-A) Diagnostic approach

A Narrative Review

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ECG reads the rhythmic and sequential transmission of impulses from SA node through the AV-HIS-Purkinje hierarchy to the ventricular myocytes , demonstrating the physiological continuity of the atrial-ventricular conduction system. In this hierarchy SA node dominates and works as a pacemaker. But the reverse occurs when ventricular activation comes first rather than as a terminal sequence. Here ventricular system overpowers the atrial system of conduction. This happens during VT , a dreadful condition to the life , precipitating hemodynamic collapse. There are many ECG criteria that seek to unravel this puzzling domain of VT.

BUT

- ☐ **Clinicians find lead aVR quite promising for identifying the direction of earliest activation—whether it travels through ventricular myocytes (VT) or through His-Purkinje system with aberrancy (SVT-A).**
- ☐ **Vereckei criteria shift the answer towards the mechanism : focussing on the initial ventricular activation rather than terminal QRS morphology.**

The challenge of differentiating ventricular tachycardia (VT) from supraventricular tachycardia with aberrancy (SVT-A) has occupied clinicians for decades – lead aVR proves to be compass and Verecki criteria turns to be decision nodding.

1. Introduction (Keypoints)

- The lead aVR views the upper (basal) interventricular septum , the point from where the impulses are directed downward towards the ventricular system. This lead also receives reciprocal information being transferred from the oppositely situated left ventricle (LV).

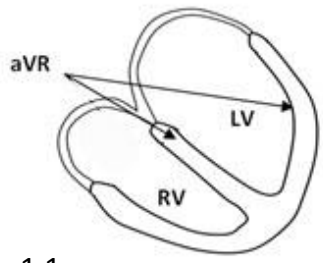

Fig. 1.1

The Vereckei criteria shift the diagnostic focus from morphology-based patterns to a mechanism-based assessment, clarifying whether the impulse enters the ventricles through the His–Purkinje system (as in SVT with aberrancy) or activates the ventricles retrogradely via myocardial conduction (as in VT)

- The criteria based on the aVR algorithm were developed to differentiate VT from SVT with aberrancy, rather than to diagnose VT in isolation.

- In both the situations , VT vs SVT with aberrancy , the resultant ventricular QRS complex is widened and uncertainty exists in distinguishing between these two. The lead aVR sees what other criteria overlook – the direction of initial activation that reveals VT. In such a chaos of wide complex tachycardia , the lead aVR is the compass – the Verecki criteria shift this into the decisive reality.
- **In nutshell to say** , the Vereckei criteria represent a paradigm shift. Rather than evaluating how the ventricles complete depolarization, this algorithm analyzes how they begin. Because the origin of activation determines the initial deflection in lead aVR, this single lead becomes the window into the true site of impulse generation.
- The conceptual ideation : a decisive reality
  " If VT originates from outside the His–Purkinje system, the initial phase of ventricular depolarization should be fundamentally different from that of SVT with aberrancy".

2. How Vereckei criteria came into existence

Before the Vereckei criteria , the most well-known and widely used diagnostic tool was the Brugada algorithm , introduced in 1991. However , the Brugada critera to diagnose VT were somewhat more complex to be used in the hours of emergency – multiple algorithmic steps were needed to evaluate its integrity fully , requiring a considerable expertise to use them clinically.

Dr. Andras Vereckei , a Hungarian cardiologist from Semmelweis University (Budapest), published in 2008 a simpler , more objective algorithm by using lead aVR alone. Most possibly the following conceptual ideation was in his mind :

If VT originates from outside the His–Purkinje system, the initial phase of ventricular depolarization should be fundamentally different from that of SVT with aberrancy.

Vereckei criteria as a whole is considered a very important diagnostic tool to distinguish between ventricular tachycardia and supraventricular tachycardia with aberrancy.

3. Electrophysiological Triad as registered through lead aVR

In ventricular tachycardia, the electrical wavefront involves the ventricles with three distinct electrophysiological phases, which are registered through lead aVR :

- ☐ **Initial Phase** : Slow "muscle-to-muscle" activation (Vi= **initial ventricular activation velocity**)
  Lead aVR inscribes this as a broad initial q or r wave lasting ≥ 40 ms
- ☐ **Middle phase** : Here R-wave denotes the dominant vector
  As the re-entry wave continues around the circuit, it recruits a large myocardial mass , which generates usually a dominant R wave on lead aVR.
- ☐ **Terminal phase** : Possible Purkinje takeover or completion of the loop (Vt)
  Late in the cycle, if the impulse enters surviving Purkinje fibers, the terminal activation becomes faster . Here Vt points toward terminal ventricular activation velocity (last 40 ms)

NB :

- **Vi/Vt consideration**

Vi — Initial Ventricular Activation (first 40 ms)	**Vt—Terminal Ventricular Activation (last 40 ms)**
• Slow muscle-to-muscle conduction • "Lazy move" • Less vertical excursion • Often slurred, stepped, or notched • Represents intramyocardial activation **Measured as vertical voltage change (mV) during the first 40 ms (one small square)**	• Fast Purkinje-mediated conduction • "Active move" • More vertical excursion • Smooth and steep • Represents purkinje induced ventricular activation **Measured as vertical voltage change (mV) during the last 40 ms (one small square)**

In VT (Ventricular tachycardia) , the first initial activation is from the intramyocardial zone causing spread of impulses from muscle to muscle → then to the purkinje front. The quantitative measurement is as follows :

Measure Vi / Vt ratio Vi / Vt ≤ 1 → VT	But when Vi/Vt ratio > 1 it points toward supraventricular tachycardia with aberrancy

Let us explain three steps electrophysiology in context with Vereckei Criteria (VT) with the following self-explanatory sketch :

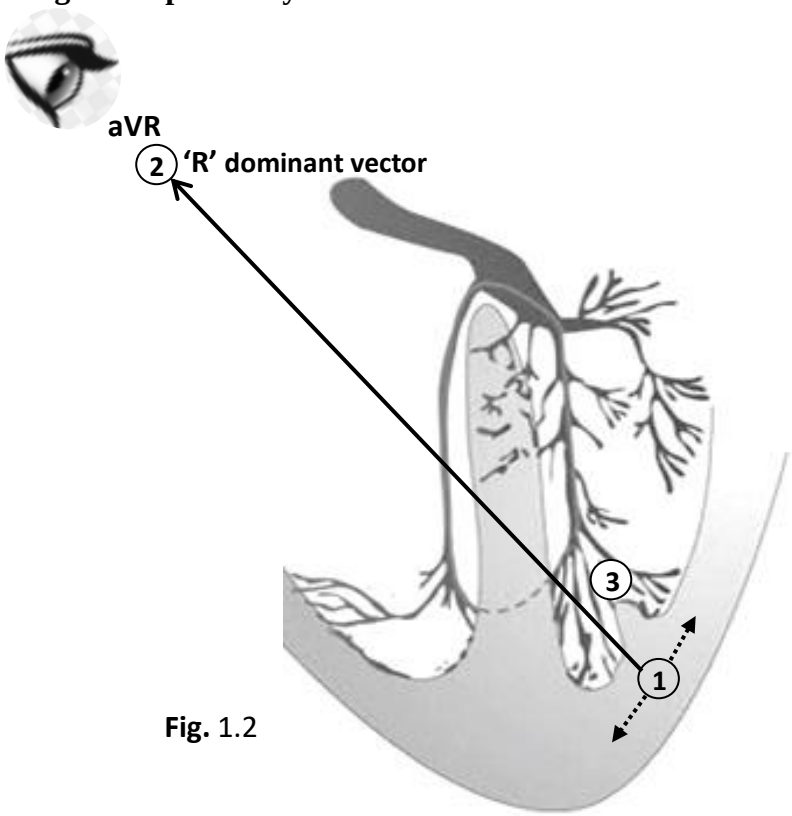

Fig. 1.2

Electrophysiological based three steps of Vereckei Criteria

1. Slow "muscle-to-muscle" activation
2. Dominant vector (R-wave) – recruiting a large myocardial mass
3. Purkinje takeover or completion of the loop via the non-surviving purkinje zone

4. Electrophysiological concept Vi/Vt

The key electrophysiological concept Vi/Vt is illustrated here through lead aVR.

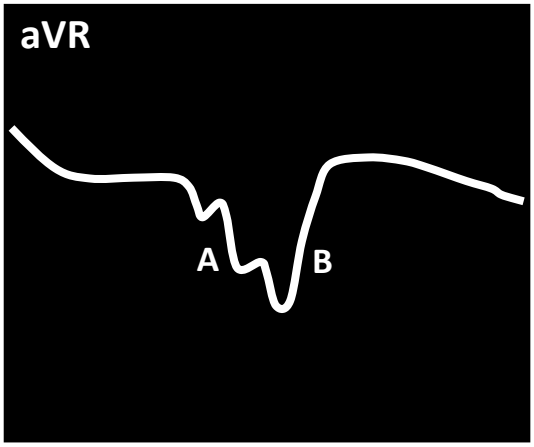

Fig. 1.3

NB :
Vereckei's Vi/Vt measurement does not depend on the dominant R at all. It depends only on the first 40 ms (whatever shape that is) , the last 40 ms (whatever shape that is).
Only two slopes for Vi and Vt - not the big R for the purpose. That's why , vector R is not illustrated in this sketch – only the descending limb and ascending limbs accordingly.

A Initial slope (Vi) = slow "muscle-to-muscle" activation (notched downslope)
This notched downslope needs a distinct explanation
B Terminal slope (Vt) = Fast purkinje induced ventricular activation

The electrophysiological "essentials of Notch" – Heterogeneous background :
In VT, the terminal part of in-circuit ventricular activation often encounters heterogeneous conduction background , as follows :

- Areas of slow muscle-to-muscle conduction
- Areas of scar-border reentry
- Areas of fast Purkinje recruitment

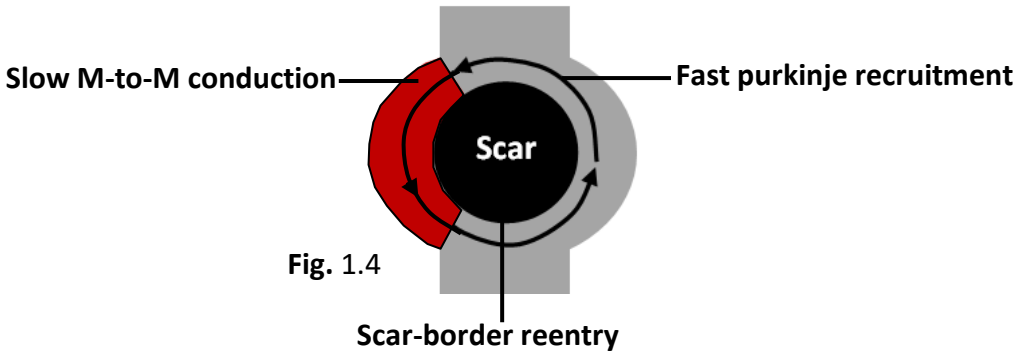

Fig. 1.4

These zones do not blend smoothly due to the prevailing heterogeneous inconsistency And therefore ,

The depolarization speed is suddenly changed during the latter half of QRS → which produces a notch.

- The 'notch' on the descending limb of the QRS is the morphological footprint of the electrophysiological switch from **slow initial activation to fast terminal conduction.**
- **Visual mental model** to understand the concept of notch : **VT**

Slow muscle ⟶ transitional heterogeneity ⟶ fast Purkinje
(Notch)

- **The reverse scenario with SVT-A** : negative wide S-wave with slurring.

5. Vereckei aVR Algorithm : Its clinical application

- ☐ Though the underlying electrophysiology rests on 3 fundamental components , the algorithm is interpreted through 4 decisive steps.
- ☐ Connecting the three electrophysiological triad with four steps sequences ———————— Initial dominant R-wave (step 1) → Initial q or r wave (step 2) → notched down stroke with negative QRS (step 3) → Vi/Vt (step 4).

- ☐ **The Real Logic Behind the Four Steps of Vereckei Criteria :**

  The electrophysiology of VT is a three-phase process — slow initial muscle-to-muscle activation, a dominant middle depolarization wave, and a late faster Purkinje-mediated spread.
  But because lead aVR views these events from a distant and opposite angle, it doesn't see all three phases clearly at once.
  Therefore, the algorithm breaks the triad into four interrogative steps — three visual probes and the remaining one as quantitative calculation — to obtain a final decisive distinction between VT and SVT with aberrancy.

- ☐ **Vereckei aVR algorithm (VT) :**

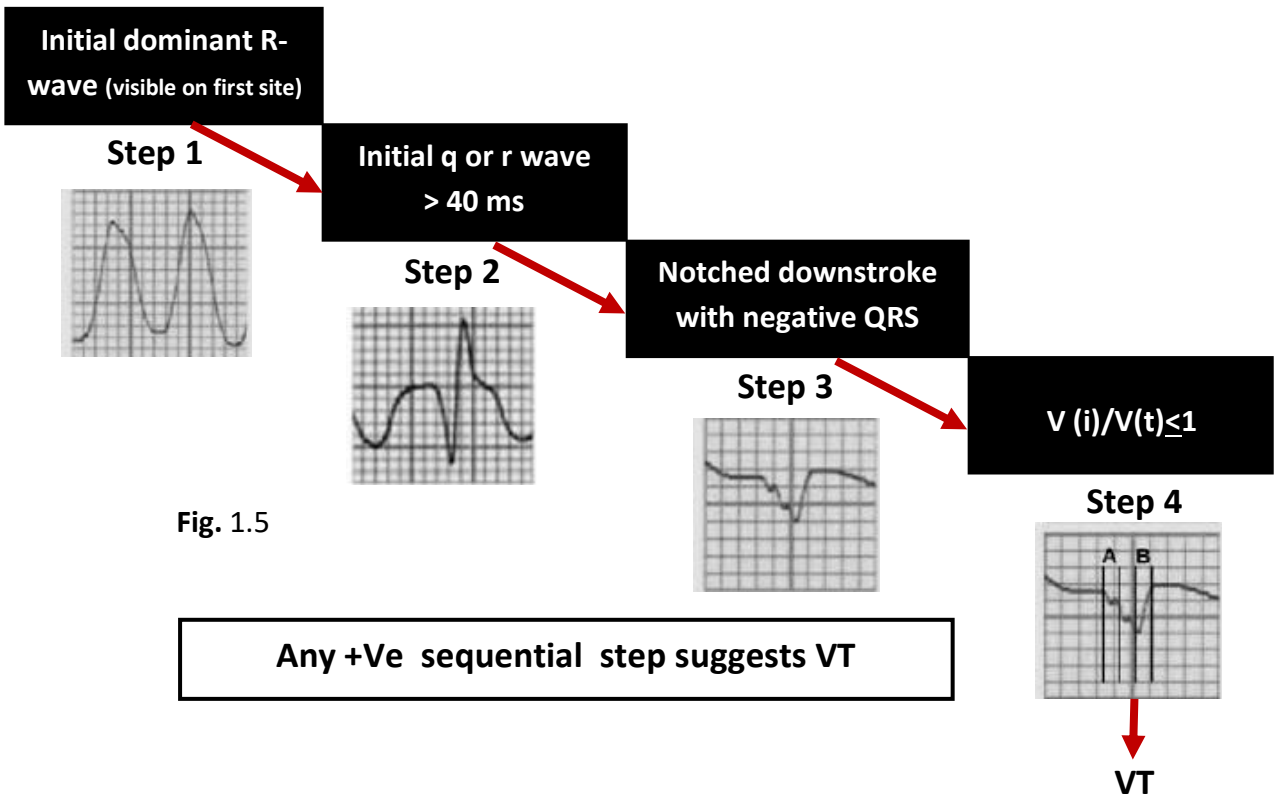

Fig. 1.5

Step 4 (Quantitative calculation)

Measure Vi / Vt ratio

Vi = initial 40 ms QRS amplitude

Vt = terminal 40 ms QRS amplitude

Vi / Vt ≤ 1 → VT

☐ **Reversal of the VT pattern :**

When the electrical signatures of VT disappear and reverse, the pattern strongly supports SVT with aberrancy

Electrophysiological interpretation

The atrial impulse travels down the His–Purkinje network, say with a bundle branch block:

- The initial 40 ms (Vi) reflects rapid purkinje activation and remains narrow
- Terminal slow muscle-to-muscle conduction (Vt)
- Therefore: Vi /Vt >1

ECG wave form through lead aVR

The narrow Vi component merges with the second broader Vt component – the whole scenario is translated into negative wide S-wave with distal slurring . Here since the dominant depolarization vector moves away from lead aVR , a deep negative QRS is inscribed on this lead.

6. Limitations of the Vereckei (aVR) criteria

Vereckei criteria as a whole is considered a very important diagnostic tool to distinguish between ventricular tachycardia and supraventricular tachycardia with aberrancy. But it does not cover all the essentials to diagnose these two entities. At times Vereckei criteria does not produce clear cut answer to the problem. Some of the important conditions are enumerated below as a part of limitations :

⇒ VT origin may not be directed toward aVR
  - VT from posterior / basal / lateral LV or RVOT/LVOT may not generate a dominant initial vector toward aVR

⇒ Fascicular VT (narrow QRS)

⇒ Extremely wide RBBB/LBBB aberrancy

⇒ Extreme clockwise or counter-clockwise rotation alters the aVR perspective , reducing reliability.

⇒ Hypertrophy / fibrosis/prior MI
  - Slowed conduction in diseased myocardium during SVT can imitate VT-like patterns

⇒ Vi/Vt ratio requires careful measurement

⇒ Single-lead dependence
  - Entire algorithm depends on one lead (aVR) → anatomical variations easily distort interpretation.

7. Take-Home Message

- The lead aVR views the upper (basal) interventricular septum , the point from where the impulses are directed downward towards the ventricular system. This lead also receives reciprocal information being transferred from the oppositely situated left ventricle (LV).
- The Vereckei principle is based on early slow conduction (Vi), a global depolarization vector (R) frequently directed toward aVR, and terminal fast conduction (Vt).
  In brief :
  one perfect single-line summary of the 3-steps physiology narrates the "Slow first (Vi), prominent middle (dominant vector R), and fast last (Vt) as the sequential signature of VT".
- **Vi/Vt consideration**

  In VT (Ventricular tachycardia) , the first initial activation is from the intramyocardial zone causing spread of impulses from muscle to muscle → then to the purkinje front. The quantitative measurement is as follows :

Measure Vi / Vt ratio Vi / Vt ≤ 1 → VT (by amplitude measurement)	But when Vi/Vt ratio > 1 it points toward supraventricular tachycardia with aberrancy

- The electrophysiological "essentials of Notch" – Heterogeneous background :
  In VT, the terminal part of in-circuit ventricular activation often encounters heterogeneous conduction background → The depolarization speed is suddenly changed during the latter half of QRS → which produces a notch.
- Because lead aVR views these events from a distant and opposite angle, it doesn't see all three phases clearly at once.
  Therefore, the algorithm breaks the triad into four interrogative steps — three visual probes and the remaining one as quantitative calculation — to obtain a final decisive distinction between VT and SVT with aberrancy.
  Four step sequences :
  Initial dominant R-wave (step 1) → Initial q or r wave (step 2) → notched down stroke with negative QRS (step 3) → Vi/Vt (step 4).

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András Vereckei, MD* vereckei@kut.sote.hu · Gábor Duray, MD† · Gábor Szénási, PhD‡ · Gregory T. Altemose, MD, FHRS¶ · John M. Miller, MD, FHRS
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NB : Consulted and discussed with ChatGPT whenever needed

UNDERSTANDING THE BASEL ALGORITHM : A SIMPLIFIED CLINICAL-ECG APPRAOCH TO WIDE COMPLEX TACHYCARDIA

ECG

UNDERSTANDING THE BASEL ALGORITHM : A SIMPLIFIED CLINICAL-ECG APPROACH TO WIDE COMPLEX TACHYCARDIA

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OUTLINE

Understanding the Basel Algorithm:
A Simplified Clinical-ECG Appraoch to Wide Complex Tachycardia

A Narrative Review

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Ancient fables often reveal truths that modern science later confirms. The timeless story of the tortoise and the hare, though simple in form, carries a lesson that mirrors the physiology of ventricular conduction.

Slow conduction in ventricular tachycardia (VT), like the steady pace of the tortoise, exposes the roughness of the path it travels—the myocardium—rather than the rapid, well-paved His–Purkinje network that the hare relies on by speeding ahead. In this sense, the tortoise reveals more about the road than the hare ever could.

Such an understanding aligns clinical intuition with a mental map in which the concept of 'Wide QRS Complex Tachycardia' is truly and accurately embedded.

- **The Basel Algorithm is a time-based assessment of ventricular activation, judged by 'Time to First Peak' , which reflects myocardial conduction time.**
- **This algorithm integrates cardiac clinical history with Time to First Peak analysis, expressed through leads II and aVR , providing an allied and practical diagnostic framework.**

This treatise points toward an easy-to-use algorithm for differential of wide QRS complex tachycardia using the leads II and aVR.

.

1. Introduction (Keypoints)

- This article does not propose a new diagnostic algorithm but aims to provide a physiological and clinically integrated interpretation of the Basel Algorithm as originally described by **Moccetti et al**. This **Basel Algorithm** takes its name from Basel, Switzerland, the city and academic center where the study was conducted (Division of Cardiology, Department of Medicine, University Hospital Basel, University of Basel, Basel, Switzerland).
  Importantly, the term "Basel" carries no anatomical, electrophysiological, or 'basal ventricular' implication, and its use is purely geographic, consistent with historical conventions in clinical electrocardiography.

- **Basel algorithm** (differential of wide complex tachycardia**)** , VT is diagnosed in the presence of at least 2 of the following criteria : 1) Structural heart disease ; 2) lead II time to first peak > 40 ms ; and 3) lead aVR time to first peak > 40 ms ; failing to which is the SVT.
  'Time to First Peak' extends from the onset of the QRS complex to the peak of the first major deflection. This can be expressed as a QRS complex starting with an r-or R-wave >40 ms , or beginning with a q-, Q-, or QS-wave > 40 ms.

This reflects the speed of initial ventricular activation and thus serves as a mechanistic marker of early ventricular depolarization

- Moccetti et al. recognized that identifying the onset and peak of the QRS complex can at times be challenging in a given lead, particularly in lead aVR. Therefore, in their proposed Basel algorithm, they recommended integrating two limb leads (II and aVR), which likely helps delineate the 'Time to First Peak' when measurements from a single lead are difficult to rely upon.
- Its diagnostic performance in clinical set up of 'Structural Heart Disease' was found to be par excellence.

2. The Novel Basel Algorithm

☐ Graphical Representation of this Novel Basal Algorithm ,as illustrated below :

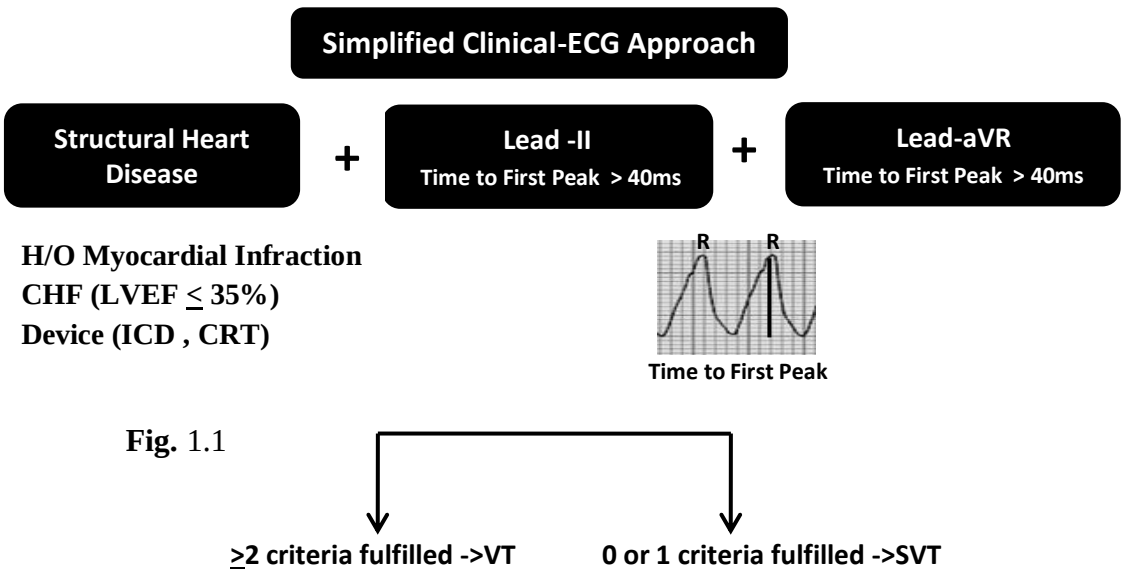

Fig. 1.1

For the algorithm , a cutoff 40 ms was chosen to facilitate user-friendly application in clinical set up (ROC derived optimal cutoffs were 51 ms for lead II time to peak and 46 ms for lead aVR time to peak)

☐ This algorithm is based on the clinical and ECG criteria both – it allows for rapid and accurate differential diagnosis of wide complex tachycardia (median 36 seconds vs 105 seconds for the Brugada algorithm and 50 seconds for the Vereckei algorithm). A shorter time scale with such a simplified approach makes this algorithm more suitable in emergency medicine , cardiology , or as a tool in primary care set up.

☐ The presence of structural heart disease as a conduction substrate is an important part of this algorithm – it defines this as the pre-test probability of VT. The measurement of 'Time to First Peak' exceeding one small ECG square (>40 ms) serves as an immediate alert to the attending clinician.

3. Electrophysiological Perspective

Moccetti et al. introduced this Basel Algorithm as a clinically sound and time-efficient approach to wide complex tachycardia. The following section aims to provide an electrophysiological perspective that complements their original framework, without modifying the diagnostic criteria.

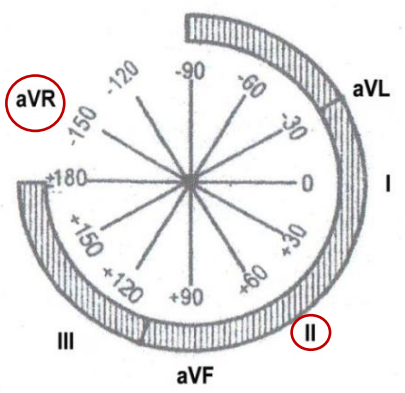

Fig. 1.2

❒ Two limb leads , namely II and aVR , on the frontal plane are used for 'Time to First Peak' measurement :

The frontal plane primarily records the net ventricular depolarization vector – the electrical axis, this predicts the accompanying waveforms polarity whether positive or negative, within or beyond 90^0 in context with the vector. **Early septal activation, being brief, low in mass, and oriented anteriorly, is minimally expressed in limb leads** and is best appreciated in the horizontal plane through the precordial leads.

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

❒ The diagnostic logic of the Basel Algorithm aligns with the electrophysiological behavior of ventricular tachycardia, in which early activation reflects global myocardial conduction rather than rapid Purkinje-mediated spread.

❒ Leads II and aVR are oriented in nearly opposite directions within the frontal plane, together surveying a wide cardiac field of ventricular activation. Lead II predominantly reflects inferior–apical depolarization, whereas lead aVR faces the basal–septal–apical regions. This complementary orientation allows effective assessment of early ventricular myocardial activation using time-based criteria.

❒ In the Basel algorithm, absence of ventricular tachycardia criteria leads to classification as supraventricular tachycardia (SVT). From an electrophysiological perspective, this implies preservation of rapid His–Purkinje–mediated ventricular activation, irrespective of whether bundle branch block or rate-related conduction delay is present.

In essence, wide-complex tachycardia represents a contest between two fundamentally different modes of ventricular activation: slow, stepwise myocardial propagation versus rapid, organized conduction through the His–Purkinje network. The Basel algorithm implicitly exploits this distinction by focusing on time-dependent and direction-dependent features captured in the frontal plane. Delayed initial activation and prolonged ventricular engagement reflect myocardial origin. Conversely, when Purkinje-mediated conduction is preserved, ventricular activation remains swift and coordinated despite surface QRS widening.

4. Illustration by ECG

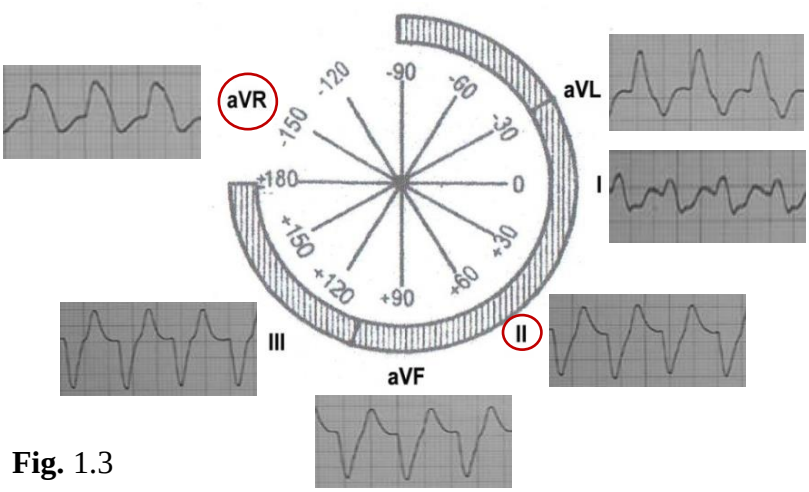

Fig. 1.3

Frontal plane : Broad Complex Tachycardia with 'Time to first peak' >40 ms (> one small ECG square) in leads II and aVR proves this to be ventricular tachycardia (VT) based on the basel algorithm.

The accompanying waves on the horizontal plane (precordial leads) are laid down as below :

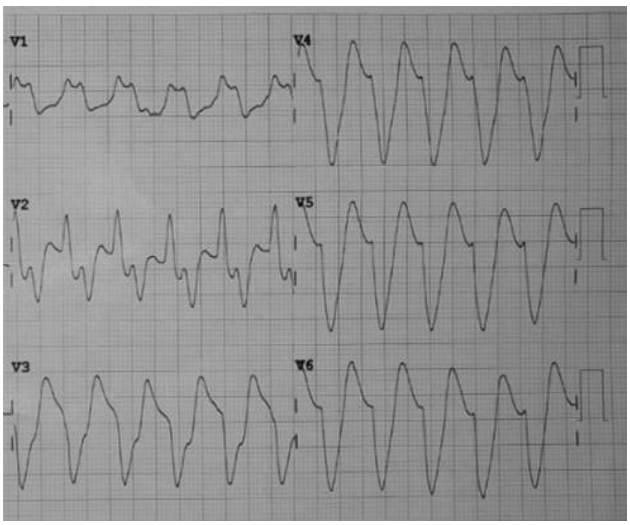

Fig. 1.4

5. Take-Home Message

- The Basel Algorithm offers a novel simple , reproducible , sensitive algorithm based on clinical and ECG parameters using two leads (II and aVR) for differentiating wide complex tachycardia.
- **Basel algorithm** (differential of wide complex tachycardia**)** , VT is diagnosed in the presence of at least 2 of the following criteria : 1) Structural Heart Disease ; 2) lead II time to first peak > 40 ms ; and 3) lead aVR time to first peak > 40 ms ; failing to which is the SVT.

- Structural heart disease significantly increases the pre-test probability of ventricular tachycardia and should always be considered alongside ECG findings.
- As reported by Moccetti et al. , the Basel algorithm showed a sensitivity , specificity, and accuracy of 92% , 89%, and 91%, respectively, in the derivation cohort and 93% , 90% and 93% respectively, in the cohort. .
- The clinical applicability of the Basel algorithm exhibited a similar diagnostic accuracy compared with the Brugada Algorithm but superiority compared with Vereckei algorithm. In compared to Vereckei criteria (based on a single lead aVR) , this algorithm is based on two lead system (II and aVR) to arrive at the diagnosis of WCT primarily determined by R-wave parameters.
- This algorithm allows for rapid and accurate differential diagnosis of wide complex tachycardia (median 36 seconds vs 105 seconds for the Brugada algorithm and 50 seconds for the Vereckei algorithm). A shorter time scale with a simplified approach makes this algorithm more suitable in emergency medicine , cardiology , or as a tool in primary care set up.

6. Reference

Simplified Integrated Clinical and Electrocardiographic Algorithm for Differentiation of Wide QRS Complex Tachycardia
The Basel Algorithm
Federico Moccetti, MD, Mrinal Yadava, MD, Yllka Latifi, MD, Ivo Strebel, PHD, Nikola Pavlovic, MD, PHD, Sven Knecht, DSc, Babken Asatryan, MD, PHD, Beat Schaer, MD, Michael Kühne, MD, Charles A. Henrikson, MD, Frank-Peter Stephan, MD, Stefan Osswald, MD, Christian Sticherling, MD, Tobias Reichlin, MD
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A HOLISTIC APPROACH TO THE DIAGNOSIS OF VENTRICULAR TACHYCARIDA USING BRUGADA , VERECKEI AND BASEL ALGORITHM

ECG

A HOLISTIC APPROACH TO THE DIAGNOSIS OF VENTRICULAR TACHYCARDIA USING BRUGADA, VERECKEI AND BASEL ALGORITHMS

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OUTLINE

Introduction
This article deals with a holistic diagnostic approach integrating these algorithms with clinical assessment and supplementary ECG features.

Overview of Brugada , Vereckei , and Basel Algorithm
Each discussed with diagnostic parameters, merits and limitations

A Proposed Holistic diagnostic framework
Key predictors of VT are :

- ⇒ Clinical scenario
- ⇒ Stewise Integrated Approach
- ⇒ Supporting traditional ECG features

ECG explanation (based on holistic diagnostic framework)

Take-Home Message

References

A Holistic Approach to the Diagnosis of Ventricular Tachycardia Using Brugada, Vereckei and Basel Algorithms

A Narrative Review

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A holistic approach seeks to view a situation in its entirety, recognizing that all its components are interconnected and mutually influential, rather than analysing isolated element. It emphasizes a comprehensive and integrated understanding in order to arrive at an appropriate solution. In essence, it involves a unified appraisal of the whole scenario to appreciate unity in diversity.

A similar holistic step is essential when a clinician analyses an ECG with a strong suspicion of ventricular tachycardia (VT).

- ☐ **Misinterpretation of ventricular tachycardia as a supraventricular rhythm may lead to catastrophic clinical consequences.**
- ☐ **Diagnostic algorithm facilitates decision-making but is never a substitute of bedside clinical judgement.**

Every ECG narrates a story through its waveforms; however, accurate identification of ventricular tachycardia is best achieved by integrating ECG algorithms with global ECG interpretation and clinical context, thereby ensuring a more reliable and comprehensive diagnostic approach.

1. Introduction (Keypoints)

- A tachyarrhythmia with a QRS duration ≥120 ms is defined as Wide-complex tachycardia. It is frequently encountered in emergency setup and cardiology practice. The primary diagnostic challenge lies in distinguishing ventricular tachycardia from supraventricular tachycardia with aberrant conduction. Studies reveal that the majority of WCTs in adults are ventricular in origin, particularly in patients with structural heart disease. Despite this, misdiagnosis remains common due to overlapping electrocardiographic patterns.
- Accurate differentiation between ventricular tachycardia (VT) and supraventricular tachycardia with aberrant conduction (SVT-A) is crucial, as management strategies differ significantly. VT requires antiarrhythmic approach or cardioversion, whereas SVT may respond to vagal maneuvers or parental adenosine. Administering AV nodal blocking agents to VT patients can precipitate cardiovascular collapse.
- Over the past three decades, several electrocardiographic algorithms have emerged out to improve diagnostic accuracy, including the Brugada, Vereckei, and Basel algorithms. Each algorithm has distinct advantages and limitations. This article deals with a holistic diagnostic approach integrating these algorithms with clinical assessment and

supplementary ECG features. Such integration imparts diagnostic precision, enhances clinician confidence, and improves patient outcomes.

- Historically, clinicians rely on basic ECG signs such as AV dissociation, fusion beats, and QRS oriented morphological changes. However, these features are either subtle or difficult to be interpreted. To improve diagnostic accuracy, structured ECG algorithms came into existence. The most notable ones are :
  - Brugada Algorithm (1991)
  - Vereckei Algorithm (2008)
  - Basel Algorithm (2022) – as described by Moccetti et al.

  While each demonstrates high diagnostic accuracy, none is perfect. A holistic approach integrating multiple algorithms in the background of clinical context offers superior reliability.

- As a component of holistic approach, structural heart disease is the strongest preprobality of VT , such as Ischemic heart disease , including prior MI, CHF (LVEF≤ 35%) , Device (ICD , CRT) , etc. A person exceeding 35 years of age is rather more vulnerable to have VT.

- In essence, a holistic approach integrates multiple electrocardiographic algorithms with the clinical context to improve the accuracy of ventricular tachycardia (VT) diagnosis. At this juncture, it is important to emphasize that distinguishing VT from supraventricular tachycardia with aberrancy (SVT-A) directly influences the choice of appropriate management strategies. Early and accurate identification of VT may also help prevent adverse cardiac remodeling. Furthermore, differentiating VT from SVT-A alerts clinicians to the potential risk of future recurrences. Timely and precise arrhythmia diagnosis may therefore interrupt the progression of associated pathophysiological changes before overt structural cardiac damage becomes evident.

Why a holistic approach needed to diagnose VT ?

A brief consideration includes :

- Overlapping electrocardiographic patterns lead to a diagnostic challenge in distinguishing VT from SVT-A.
- Misdiagnosis can result in misdirected therapy , leading to hemodynamic compromise or even death.
- A holistic diagnostic approach integrates the different algorithms in the background of clinical scenario and added supplementary ECG features. The whole workout leads to a conclusive step with superior reliability.

Therefore, a holistic approach that synthesizes established ECG algorithms with sound clinical judgment provides the most reliable pathway for the accurate diagnosis of ventricular tachycardia.

2. Overview of Brugada , Vereckei , and Basel algorithms

Algorithm-Based characteristic of each group is the basic need of this multimodal holistic approach in the diagnosis of ventricular tachycardia vs Supraventricular tachycardia : A Revisiting of Brugada , Vereckei and Basal Algorithms in context with wide complex tachycardia.

❒ **Brugada Algorithm** : Introduced in 1991

Each state in succession points toward VT, otherwise SVT-A	Merits	Limitations
• Absence of RS complexes in all precordial leads • RS interval > 100 ms • AV dissociation • Morphological criteria in V1–V6	Sytemic stepwise approach High sensitivity for VT~ 98%	Complex for rapid bedside use Requires experience to interpret morphology Less reliable in pre-existing bundle branch block

❒ **Vereckei Algorithm** : Introduced in 2008

Key Focus on lead aVR	Merits	Limitations
• Initial R wave in aVR • Initial r or q wave duration > 40 ms • Notching on the initial downstroke • Vi/Vt ratio (initial vs terminal ventricular activation) Vi / Vt ≤ 1 → VT , otherwise Vi/Vt > 1→ SVT-A (measurement amplitudewise)	Uses a single lead (aVR) Easier application in emergencies Good diagnostic accuracy	Requires precise measurement Less familiar to many clinicians Vi/Vt calculation may be challenging

❒ **Basal Algorithm** : Introduced in 2022 , described by Moccetti et al.

VT diagnosed in the presence of at least two of the followings	Merits	Limitations
• Structural heart disease • Lead II time to first peak > 40 ms • Lead aVR time to first peak > 40 ms Failing to this is the SVT-A.	Faster decision-making User-friendly High diagnostic accuracy	Limited widespread validation Less exposure in standard teaching

Advantages of the Holistic Approach

- Reduces misdiagnosis
- Enhances clinician confidence
- Balances sensitivity and specificity
- Adaptable to emergency settings
- Improves patient safety

Clinical Implication :
When in doubt, VT should be presumed and treated accordingly. Overreliance on one algorithm can be misleading. Combining ECG algorithms with patient history provides the safest approach.

3. A Proposed Holistic diagnostic framework

Key predictors of VT are :

⇒ **Clinical Scenario**
Diagnostic algorithm facilitates decision-making but is never a substitute of bedside clinical judgment. Structural heart disease is the strongest preprobality of VT , such as Ischemic heart disease , including prior MI, CHF (LVEF≤ 35%) , Device (ICD , CRT) , etc. Individuals aged over 35 years are more vulnerable to ventricular tachycardia.

⇒ **Stepwise Integrated Approach**
- Apply Brugada for structural analysis (high sensitivity for VT ~ 98%)
- Confirm with Vereckei when morphology is unclear
- Rapid check with Basel

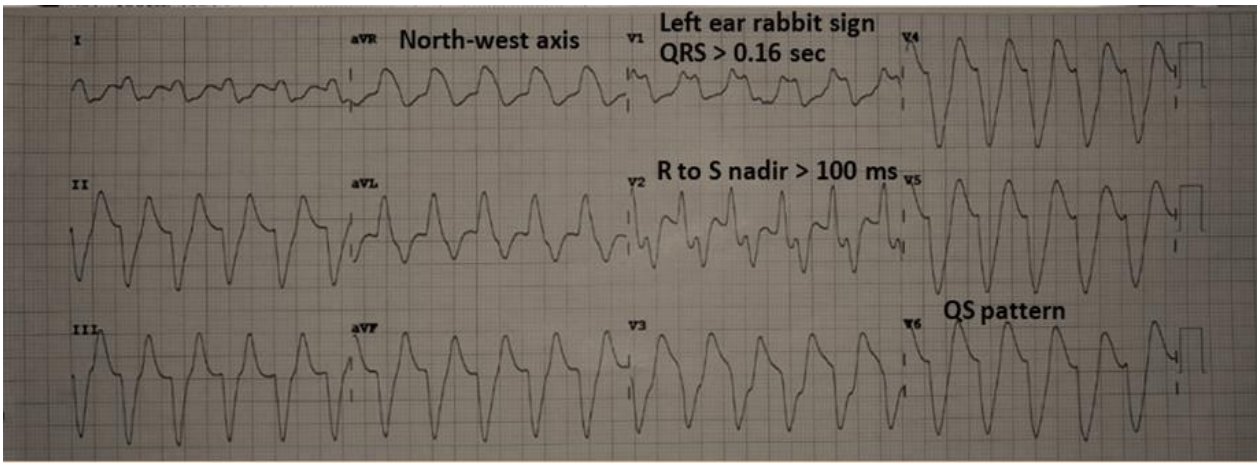
⇒ **Supporting traditional ECG features**
- AV dissociation : Capture beats , Fusion beats
- QRS duration > 160 ms
- Extreme axis deviation
- Precordial concordance
- Initial R in aVR

While highly specific, these signs have low sensitivity and are often absent.

NB : In a setup of emergency one has to proceed through some emergent steps :

- Assess stability
- If unstable → cardioversion
- If stable:
  - History
  - ECG analysis
  - Apply algorithms
- Treat as VT if uncertain

4. ECG explanation (based on holistic diagnostic framework)



Holistic diagnostic framework (in support of VT)

- Clinical scenario : No history available
- Stepwise Integrated approach

  As per Brugada algorithm

  - No absence of RS complexes in all precordial leads : non-conclusive
  - ✓ RS interval > 100 ms : yes
  - No AV dissociation
  - ✓ Morphological criteria Lead V1 : Left ear rabbit sign plus Lead V6 QS present = Atypical RBBB

  Confirmation with Vereckei : lead aVR with prominent R

  Rapid checkup with Basel : leads II and aVR time to first peak > 40 ms.

- Supporting traditional ECG features
  - QRS duration > 160 ms (V1)
  - Axis deviation (north-west axis)

5. Take-Home Message

- ☐ Accurate differentiation between VT and SVT with aberrancy remains challenging. No single algorithm is perfect. The Brugada, Vereckei, and Basel algorithms each offer unique diagnostic strengths.
- ☐ A holistic approach integrating these algorithms with clinical assessment and traditional ECG signs significantly improves diagnostic accuracy.
- ☐ Clinicians should prioritize patient safety by assuming VT when uncertainty exists.
- ☐ Early and accurate diagnosis prevents inappropriate therapy and reduces mortality. Education and training should emphasize integrated algorithm-based learning rather than reliance on a single method

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NB : Consulted and discussed with ChatGPT whenever needed

MONOMORPHIC VENTRICULAR TACHYCARDIA : ORDER WITH ELECTRICAL INSTABILITY

ECG

MONOMORPHIC VENTRICULAR TACHYCARDIA : ORDER WITHIN ELECTRICAL INSTABILITY

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OUTLINE

Monomorphic Ventricular Tachycardia : Order within Electrical Instability

A Narrative Review

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A car moving at a fixed speed along an even road lined with regularly spaced speed breakers demands constant corrective effort from the driver; with every jolt, both driver and passengers remain acutely aware that sustained vigilance alone prevents catastrophe.
Monomorphic ventricular tachycardia may be likened to such a car compelled to travel repeatedly along the same straight road, encountering identical speed breakers at fixed intervals—predictable in pattern, yet potentially dangerous in consequence.

- **Monomorphic ventricular tachycardia is sustained by a stable re-entrant circuit or fixed ventricular focus, producing repetitive activation along an identical electrical pathway.**
- **Its hallmark on ECG is beat-to-beat uniformity of QRS morphology and cycle length, reflecting the constancy of ventricular activation.**

This form of ventricular tachycardia represents a life-threatening emergency and requires prompt recognition and appropriate management by the clinician.

1. Introduction (keypoints)

- Monomorphic ventricular tachycardia (MMVT) represents a paradox in clinical electrophysiology. Despite arising from a profoundly abnormal myocardial substrate, it manifests with striking regularity in rate and QRS morphology. This apparent order often masks the seriousness of the underlying pathology. Understanding monomorphic VT therefore requires moving beyond surface ECG patterns to the deeper mechanisms that sustain such organized electrical instability.

- This VT is defined as :
  - A ventricular tachycardia with uniform QRS morphology (usually ≥120 ms) from beat to beat
  - Regular or near-regular cycle length
  - Heart rate usually >100 bpm
  - Sustained (≥30 seconds) or non-sustained (<30 seconds)

  The uniformity of morphology reflects activation through a single stable ventricular circuit or focus.

- This morphological variant of Ventricular tachycardia in most cases at a rate > 120 bpm , and it is worthwhile to mention here that ventricular tachycardia with a rate between 100 and 120 bpm is often referred to as a “slow ventricular tachycardia”.

(Ventricular tachycardia with a rate >200–250 bpm may merge into the entity known as ventricular flutter, characterized by very rapid, sinusoidal ventricular activity).

- Further to say , monomorphic VT commonly arises from a single dominant re-entrant circuit or focal source within the ventricle, and localization of its site provides valuable diagnostic and therapeutic information to the clinician.
- Sometimes the source of monomorphic VT involves the Purkinje system with its highly arrhythmogenic nature. Such Purkinje-related VT is popularly known as 'Fascicular VT.' This is one of the most common forms of idiopathic VT, most commonly originating near the left posterior fascicle of the left bundle branch. It occurs mostly in young healthy persons, predominantly males. It is worthwhile to mention here that this VT often presents as a relatively narrow-complex tachycardia, with QRS duration usually in the range of 120–145 ms.
- The Repetitive monomorphic VT may also originate from the ventricular outflow tracts, most commonly the right ventricular outflow tract (RVOT). This form of VT is typically idiopathic and is often triggered by sympathetic stimulation, such as exercise or emotional stress, producing recurrent paroxysmal episodes.

2. Electrophysiological mechanism

☐ **Most monomorphic VT is a scar-driven re-entry.**
DAD and automaticity are exceptions rather than the rule.

☐ **Re-entry :** The Dominant Mechanism

The vast majority of monomorphic VT is caused by re-entrant circuits , typically involving :

- Area of slow conduction
- Unidirectional block
- Electrically inert scar tissue acting more or less as anatomical circular boundary

A Normally supraventricular impulse coming down through Purkinje-myocyte unit is rather blocked at this slow conduction pathway (illustrated as a brick coloured zone over this sideway sketch).

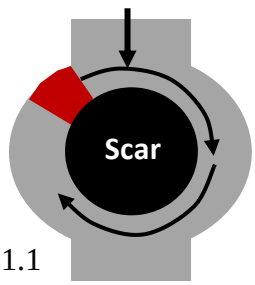


Fig. 1.1

B PVC in its progression round the circle can penetrate slow conduction pathway when it is in non-refractory state → **reentry circuit** induction

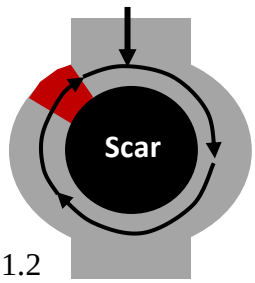
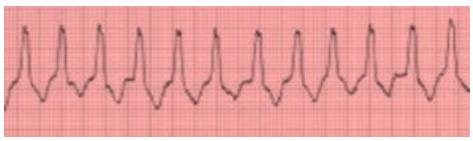


Fig. 1.2

Fig. 1.3



A run of MVT

Common substrates include **'Structural Heart Diseases'** induced scar , such as :

- Post-myocardial infarction ventricular scar
- Fibrosis in cardiomyopathies
- Surgical scars in congenital heart disease

3. When to Suspect Monomorphic VT on ECG

One should suspect a regular wide-complex tachycardia as monomorphic VT , especially in the appropriate clinical setting of the history of structural heart disease (assuming the presence of scar tissue therein).
This is the **'First Red Flag'**.
Regular wide QRS tachycardia (>120 ms) at the rate of > 100 bpm.
(At least 3 or more identical consecutive beats of wide QRS complexes).

4. Spatial layout in favour of monomorphic VT as per Brugada algorithm

Brugada et al. laid down four steps oriented diagnostic algorithm in context with regular tachycardia with a wide QRS complex. The following sketch is the self-explanatory :

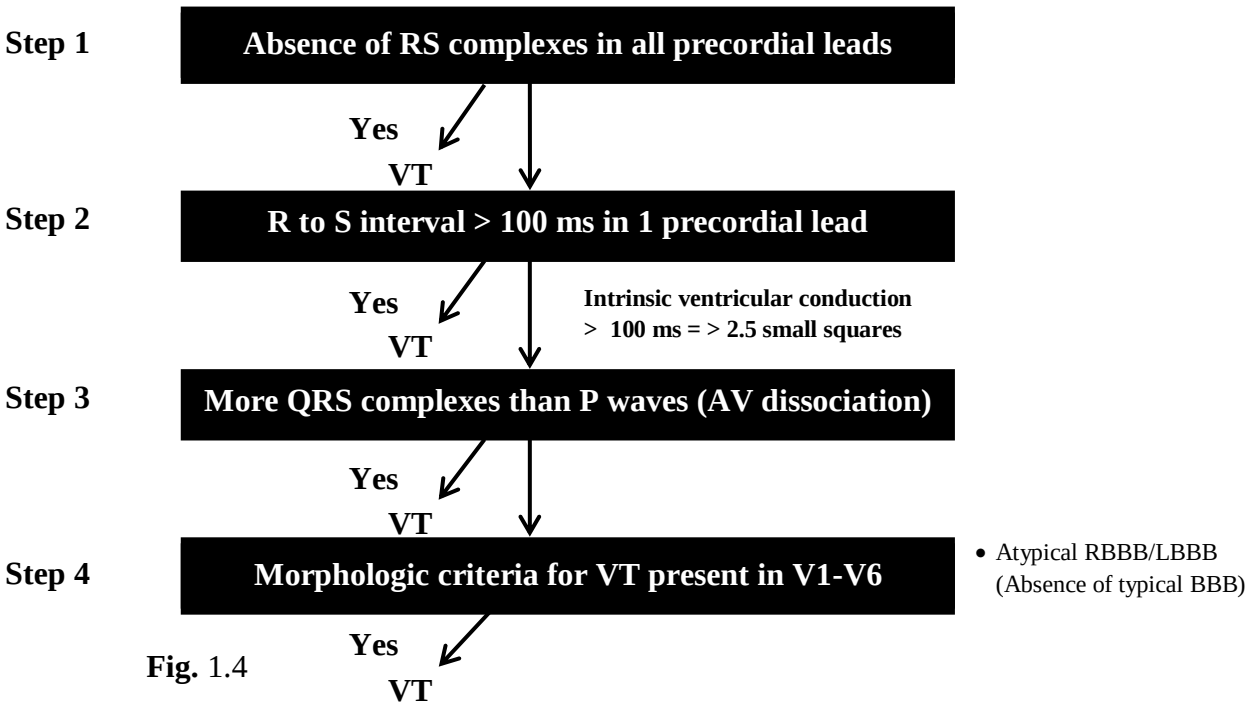


Fig. 1.4

This would be worthwhile to mention here that each step of this Brugada algorithm represents the integrated approach one after the other , proving the same fact that the resultant repolarization wavefront travels in one dominant direction.

Step 1

Since MMVT arises from a single point of source on the ventricle , there is no question of the presence of transition zone anywhere over the precordial leads. In other words, there is complete **'absence of RS complexes in all precordial leads'.**

Some tracing patterns prioritize the presence of MMVT on ECG :

- Negative concordance: QS or predominantly negative QRS throughout in V1-V6 , originating from the posteriorly situated left ventricle .
- Positive concordance : Monophasic R waves in all precordials, typical of apical septal VT focus.

NB : QR complexes in all precordial leads is highly specific for VT.
(This implies ventricular activation not through normal His–Purkinje system).

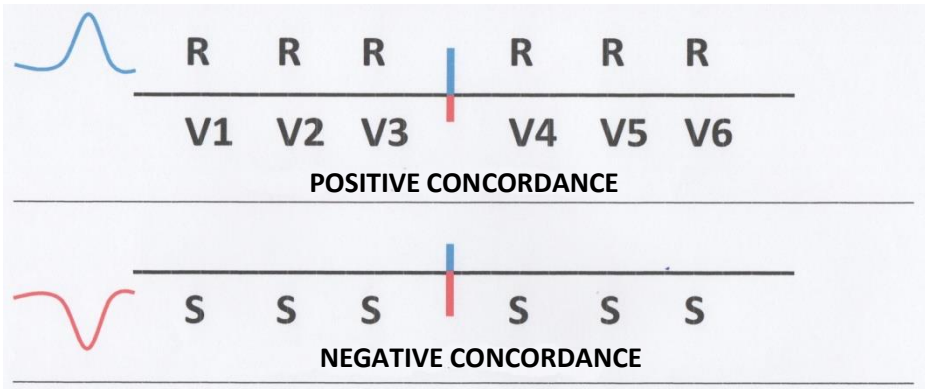


Fig. 1.5

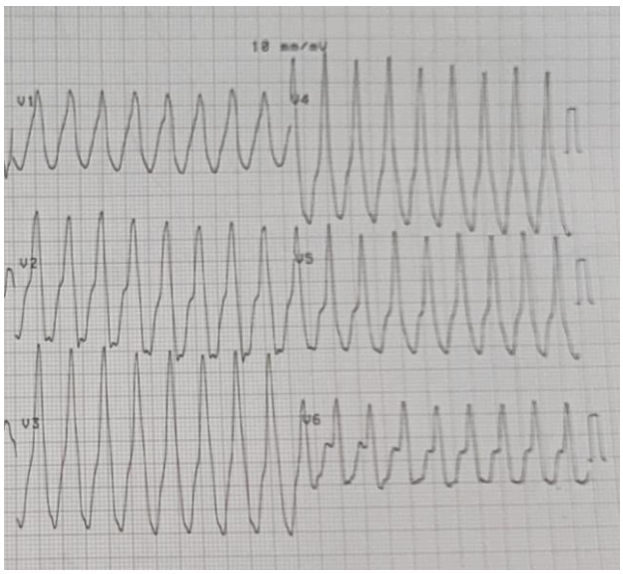


Fig. 1.6

Positive concordance from V1 to V6

Step 2

- **VT Conduction Physiology** : Normal QRS uses rapid His-Purkinje fibers for sequential endo- to epicardial activation; VT originates focally (e.g., scar),

propagating cell-to-cell with phase delays—journey "start" at exit site, "dropout" at boundaries causing notched/slurred S nadir.

- **Quantitative components :** RS interval from R-wave onset to S-wave nadir exceeding 100 ms in any precordial lead indicates VT due to delayed activation.
  R-wave peak time ≥40 ms plus ≥60 ms to S-nadir , combination of both = > 100 ms.

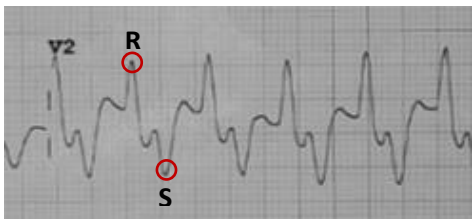


Fig. 1.7

Step 3

- **AV dissociation means :**
  - Atria and ventricles depolarize independently
  - Ventricular rate ≥ atrial rate
  - P waves may march through QRS , appear before, within, or after QRS or it may be visualized over the tip of T-wave

- **Site of visualization of P-wave**

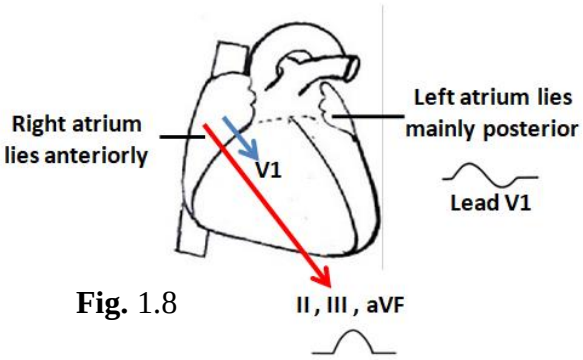
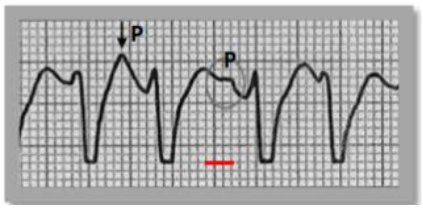


Fig. 1.8



Atria and ventricles depolarize independently

Fig. 1.9

Check inferior leads (II, III , aVF) / V1 , where P-wave often stand out best to be visualized.

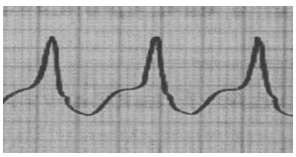
- Capture / Fusion beat : indirect evidence of 'P' in AV dissociation
  - Capture beats (normal-looking narrow QRS within wide tachycardia)
  - Fusion beats (hybrid morphology)

Fig. 2.0

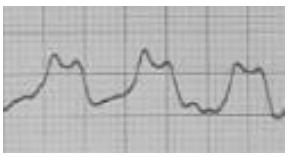
- Absence of AV dissociation does not exclude VT. Presence confirms VT.

Step 4

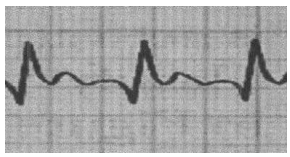
- **Right bundle branch block (RBBB) - like pattern**
  The lead V1 records monophasic R, , taller R than R' (Left rabbit ear) or qR while the opposite counterpart V6 records more or less mirror image.
  With RBBB current flows from LV to RV.



'Monophasic R'



'Left rabbit ear'



Biphasic qR

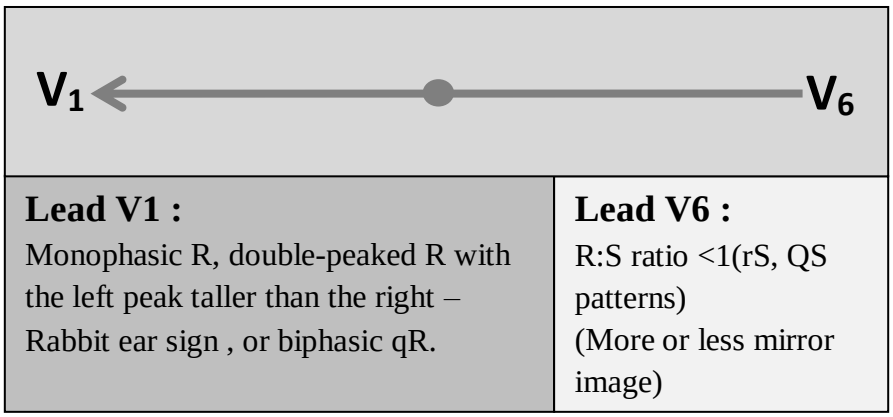


Fig. 2.1

NB :

- **V1 ← ● → V6**
  - This symbol points toward 'overlap zone' (the electrical actication fields in between two ventricles partially overlap within the precordial projection , particularly over lead V1 , accounting for the atypical RBBB-like pattern , as illustrated above.
- QRS duration > 140 ms with RBBB-like pattern suggests VT.
- **Left bundle branch block (LBBB) - like pattern**
  The lead V1 records smooth initial R>40ms with RS nadir>60ms and the opposite counterpart V6 records QS , QR or qR pattern.
  With LBBB current flows from RV to LV (QRS duration > 160 ms).

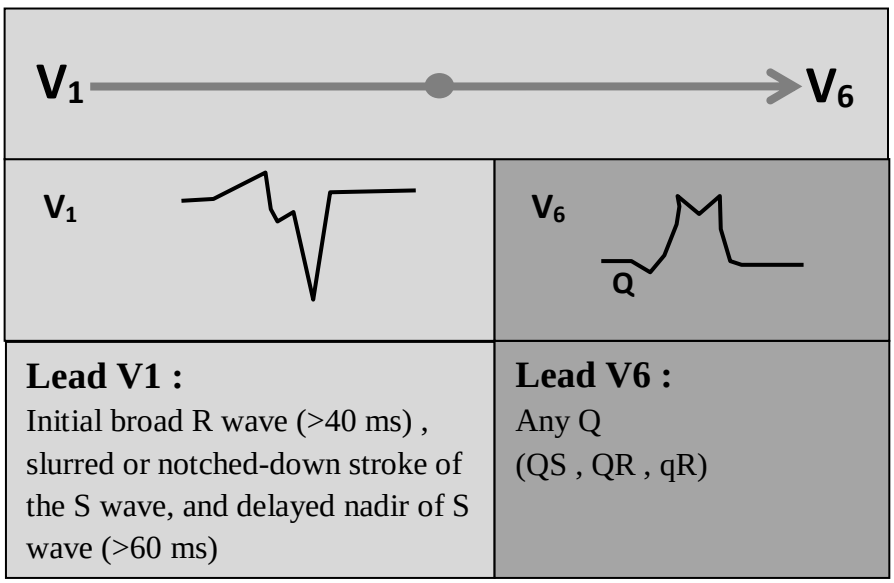


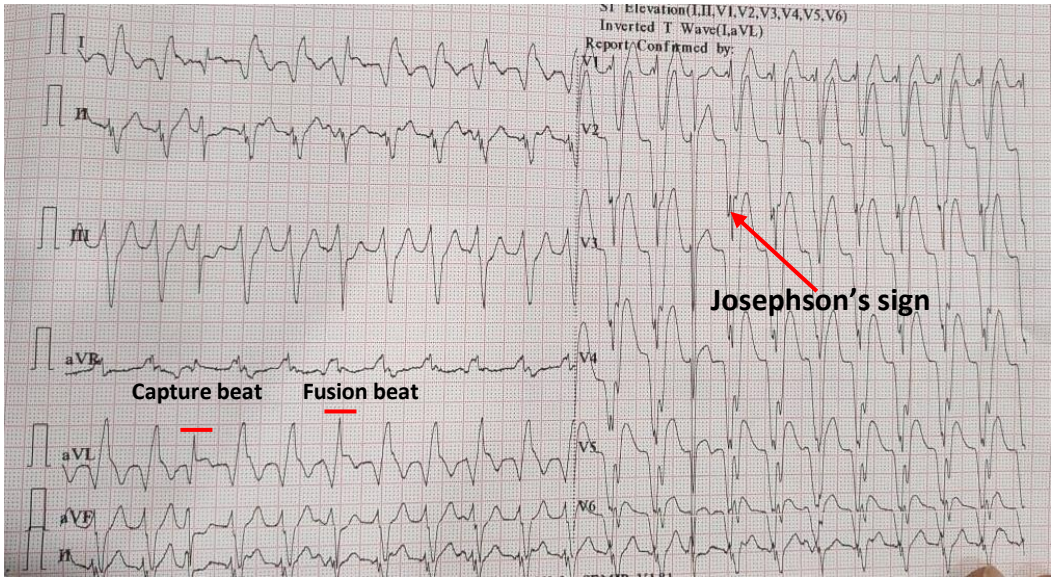
Fig. 2.1

Miscellaneous QRS axis mostly directed towards north-west zone (-90^0 to -180^0)

5. Illustration by ECGs

ECG no. 1

History : 50 years male with sudden onset of palpitation in the background of CAD

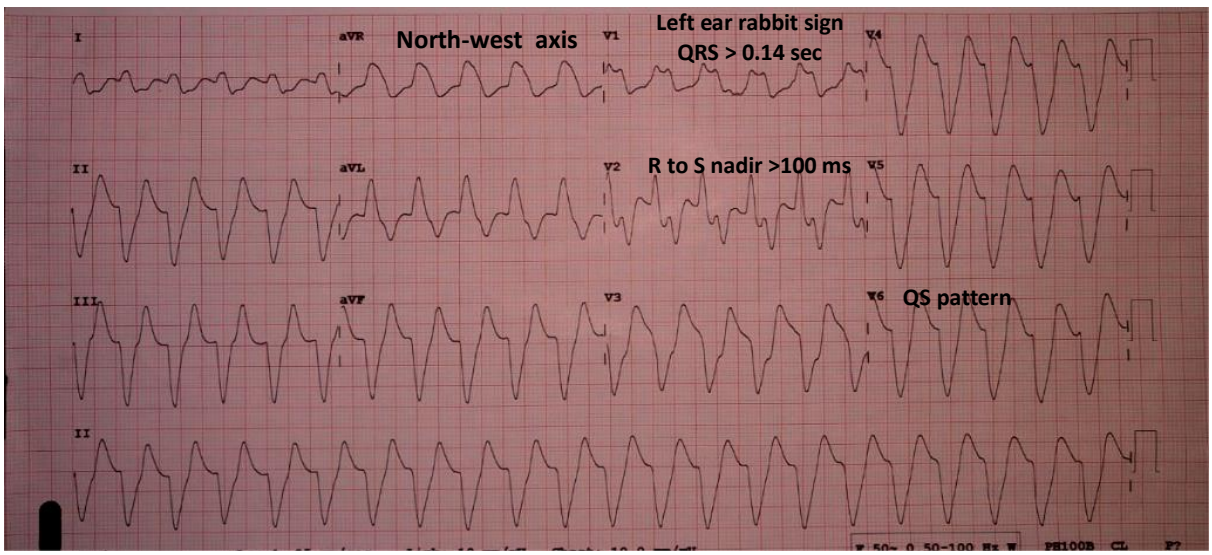


Findings :

- Wide complex regular tachycardia (180 ms) with the rate of 125 bpm.
- The presence of capture beat and fusion beat on third and sixth complexes respectively (please see over aVL , as marked by red circles).
- Josephson sign – Notching near the nadir of the S-wave.

ECG no. 2

History : 55 years male presenting as palpitation of sudden onset since one hour. No background history is available.



Findings : Illustrated on ECG tracings (self-explanatory)

5. Take-Home Message

- ☐ Monomorphic ventricular tachycardia teaches a crucial lesson in cardiology: order on the ECG does not always imply order within the myocardium. Beneath its rhythmic uniformity lies a vulnerable heart , with a tendency to repeat such recurrences in future as well.
- ☐ Reentry is the predominant mechanism
- ☐ Important clues :
  - ➢ A wide complex tachycardia (>120 ms) , defined as three or more identical consecutive beats of ventricular origin at a rate of >100 bpm
  - ➢ Positive or negative concordance (either totally positive or totally negative waves over the precordial leads strongly suggests VT).
  - ➢ MMVT is AV dissociation type of arrhythmia wherein atrial and ventricular activities are independent of each other – termed as AV dissociation (atrial activity and ventricular activity occur independent of each other)
  - ➢ Capture beat / fusion beat , as discussed before
  - ➢ QRS axis determination mostly directed towards north-west zone (-90^0 to -180^0)
  - ➢ Block pattern morphology –
    - Either atypical RBBB or LBBB pattern
    - QR in V6 in the presence of RBBB / LBBB
- ☐ Important predictors of adverse outcome include:
  - Presence of structural heart disease
  - Reduced left ventricular ejection fraction
  - Sustained VT
  - History of syncope or cardiac arrest
  - Monomorphic VT in a scarred ventricle is a marker of fixed arrhythmogenic substrate and carries significant prognostic weight.

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NB : Consulted and discussed with ChatGPT whenever needed

POLYMORPHIC VT (PMVT) AND TORSADES de POINTES (TdP) – A BRIEF OVERVIEW

ECG

POLYMORPHIC VT (PMVT) AND TORSADES de POINTES (TdP) – A BRIEF OVERVIEW

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OUTLINE

Introduction

- PVT with normal baseline QT interval
- PVT with prolonged baseline QT interval – **'Torsades de pointes'**

Electrophysiologic basis

The fundamental abnormality in polymorphic VT, particularly torsades de pointes, lies in disordered ventricular repolarization.

PMVT vs Torsades de pointes Core Mechanistic Differences : Overview

Main differentiating points

- PMT (Normal QT) DAD mediated short coupled VPC (R-on-T)
- Torsades de pointes (prolonged QT) EAD mediated pause-dependent (short-long-short cycle)

Take-Home Message

References

Polymorphic VT (PMVT) and Torsades de Pointes (TdP) – A brief overview

A Narrative Review

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Some road journeys are difficult because the ground beneath is uneven and unpredictable, demanding constant effort. Other journeys may feel smoother, yet the path itself keeps changing direction, slowly disorienting the traveller. The ventricle can become the site of such electrical road signals, where the rhythm follows an uncertain and shifting course.

Polymorphic ventricular tachycardia (PMVT) resembles a journey troubled by an irregular surface, disrupting each step. Torsades de Pointes (TdP), on the other hand, resembles a journey in which the path twists continuously, leading to loss of direction despite an apparently smooth progression.

- **In polymorphic VT, the changing QRS morphology mirrors constantly shifting activation wavefronts, with a continuously varying electrical axis and no predictable pattern. Here, the QT interval remains within normal limits.**
- **In Torsades de Pointes, the journey reflects oscillating dispersion of ventricular repolarization, resulting in a characteristic cycling, sinusoidal "twisting" of QRS complexes around the baseline. Here, the QT interval is prolonged.**

Polymorphic VT and Torsades de Pointes remind clinicians that rhythm depends not only on impulse generation, but also on how orderly propagation and repolarization are maintained within the ventricular myocardium.

1. Introduction (keypoints)

- **Polymorphic ventricular tachycardia (PMVT)** is a ventricular arrhythmia characterized by continuously changing QRS morphology and axis on the ECG, reflecting significant electrical instability within the ventricles. A distinct subtype is **torsades de pointes (TdP)**, which occupies a unique position because it is specifically associated with abnormalities of ventricular repolarization, typically occurring in the setting of **QT interval prolongation.**

- **PVT with normal baseline QT interval**
  This is a type of ventricular tachycardia which gets manifested as polymorphic QRS complexes, which are having variable amplitude and duration. The most common causative factor for PMVT is myocardial ischemia / infarction. At times it may be associated with some other structural heart diseases.

- **PVT with prolonged baseline QT interval – known as Torsades de Pointes**.
  This also shows the variable nature of QRS complexes as those of PMVT but in addition , the cyclic twisting of these polymorphic waves round the isoelectric baseline is witnessed as the main companion of this subtype.

Torsades de Pointes is a French word which means 'Twisting of Points'. This pattern is usually seen with clinical conditions causing prolongation of QT interval – as with congenital or acquired causes , such as hypokaleamia or with the use of some specific drugs .

- Torsades de pointes (TdP) may terminate spontaneously, but if it persists for more than a few seconds, it can lead to transient cerebral hypoperfusion and syncope. In some cases, TdP may degenerate into ventricular fibrillation (VF), resulting in sudden cardiac arrest.
  Similarly, polymorphic ventricular tachycardia (PMVT) may also be self-terminating or sustained. When sustained, it often produces severe hemodynamic instability and may rapidly deteriorate into VF, particularly when it occurs in the setting of acute myocardial ischemia or structural heart disease.
- **Importantly to mention here that not all polymorphic VT is torsades, but all torsades is polymorphic VT.** Polymorphic ventricular tachycardia and torsades de pointes represent the ECG expression of a ventricle that has lost uniformity in repolarization. It reminds clinicians that electrical stability depends not only on impulse generation and conduction, but equally on orderly recovery. Recognition of the underlying mechanism is essential, as timely correction of repolarization abnormalities can be lifesaving.

2. Electrophysiologic basis

The fundamental abnormality in polymorphic VT, particularly torsades de pointes, lies in disordered ventricular repolarization.

❒ Electrophysiologic mechanism in PMVT

- PMVT is often induced by phase 4–dependent delayed afterdepolarizations (DADs).
- Continuous influx of Ca^{2+} may induce DAD dependent triggered activity.
- Tachycardia and sympathetic stress favour its occurrence
- These DAD-mediated beats are often short-coupled
- A short-coupled VPC falling on the vulnerable period can produce R-on-T, precipitating PMVT

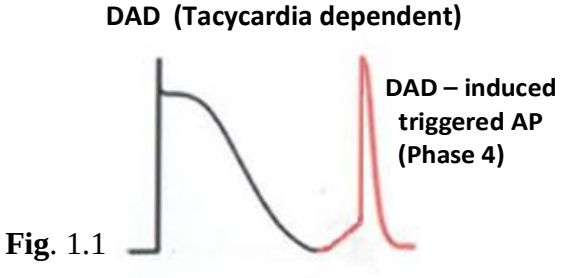

Fig. 1.1

- Clinical entities in association :
  - Acute myocardial ischemia / infarction
  - Reperfusion arrhythmias
  - Severe myocardial irritability

- **The resultant ECG**

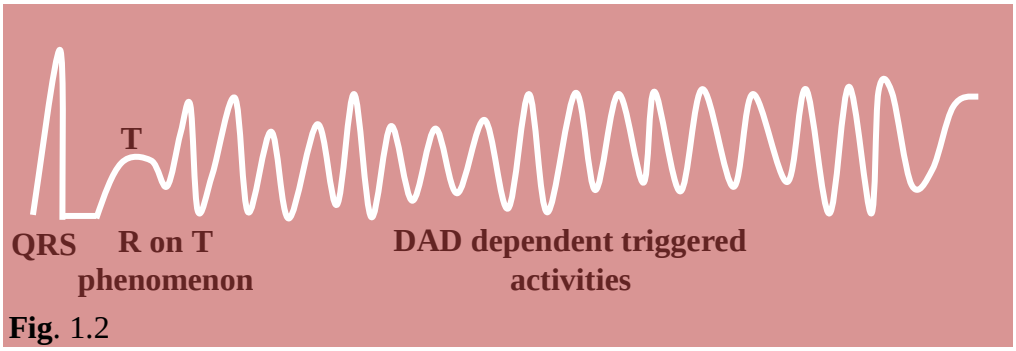

Fig. 1.2

Polymorphic Ventricular Tachycardia (PMVT) with normal baseline QT interval

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❒ Electrophysiologic mechanism in TdP

- TdP is triggered by phase 2-3 dependent early afterdepolarizations (EADs).
- **"Short-Long-Short"** cycle sequence **:**
  Short-coupled PVC→ compensatory pause(long pause) →Pause-dependent QT ↑ → 2nd PVC triggered ventricular activity (small pause between 2nd PVC and the start of TdP) → **TdP**
- Clinical settings :
  - Congenital long QT syndromes
  - Drug-induced QT prolongation (antiarrhythmics, psychotropics, antibiotics , etc)
  - Electrolyte abnormalities (hypokalemia, hypomagnesemia)
  - Severe bradycardia or AV block

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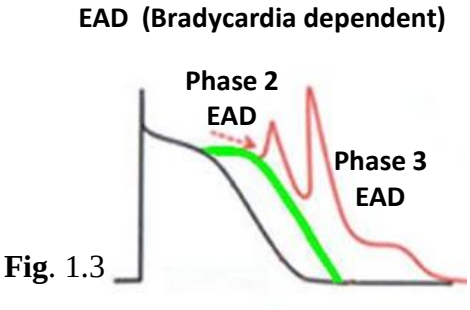
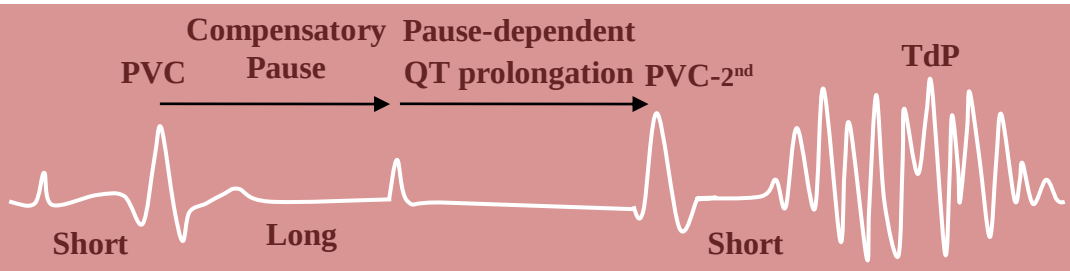


Fig. 1.3

- **The resultant ECG**



NB : Pause-dependent QT prolongation occurs because longer cycle lengths reduce repolarizing potassium currents and allow excessive action-potential prolongation , promoting EADs that trigger TdP.

Fig. 1.4

Classical Polymorphic Ventricular Tachycardia (PVT) with prolonged baseline QT interval known as Torsades de Pointes

Electrocardiographic tips (concerned with TdP) - an important consideration

Increasing data indicate that the electrocardiographic findings of the TdP are insufficient to make an accurate diagnosis in all cases. This would be worthwhile to mention here that TdP can terminate itself with progressive prolongation of the cycle length , or ending with a return to the basal rhythm , or to a new episode of Torsades de pointes or to the episode of ventricular fibrillation.

- During short run of TdP or on single lead recording the characteristic 'twisting morphology' may not be apparent .
- Ventricular bigemini pattern in the background of long QT interval may herald imminent TdP.
- TdP with higher heart rates > 220 beats / min are having longer duration with more vulnerability to degenerate into VF.
- Important to note that the presence of abnormally giant shaped 'T-U' waves may precede TdP in the place of long QT interval .
- Prolonged QT interval might not be visible in the setup of ECG findings. Basal ECG may be needed for the purpose.
- The twisting pattern of TdP virtually stamps the diagnosis of TdP

❒ Events summary

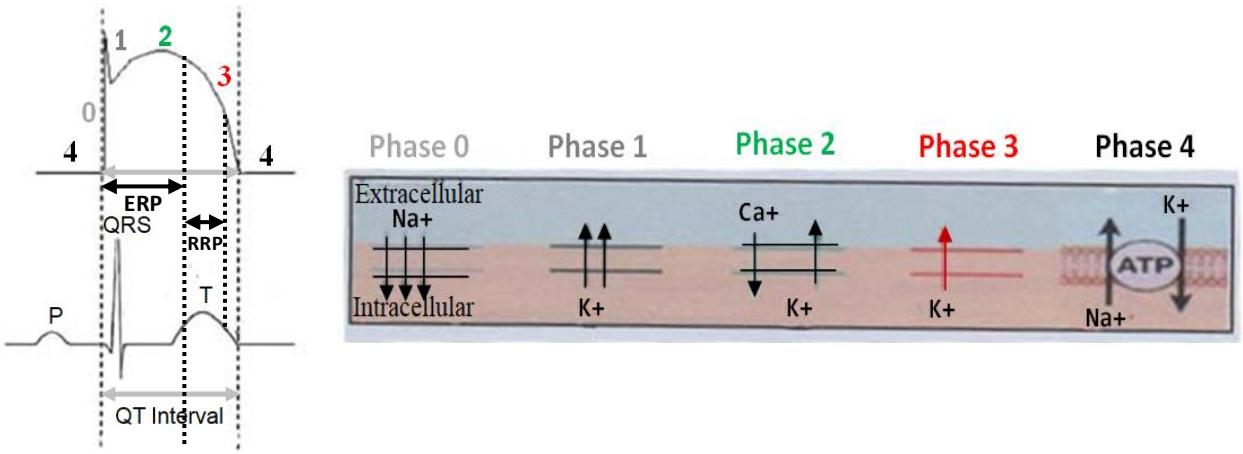


Fig. 1.5

- **Early afterdepolarization (EAD)** includes the terminal part of phase 2 or phase 3 of cardiac action potential . This is very vulnerable period for such afterdepolarization dependent triggered activites → Torsades de pointes (TdP).
- **Delayed afterdepolarization (DAD)** – This includes phase 4 induced afterdepolarization which plays its role in Polymorphic Ventricular Tachycardia (PMVT).

3. PMVT vs Torsades de pointes Core Mechanistic Differences : Overview

Feature	PMVT	Torsades de Pointes
• QT interval	Normal	Prolonged
• Primary instability	Depolarization-unstable with preserved recovery	Repolarization-unstable with delayed recovery
• Key triggered	Short-coupled VPC (R-on-T)	Pause-dependent (short-long-short cycle)
• Afterdepolarization	DAD-mediated	EAD-mediated
• Rate dependency	Tachycardia/stress	Bradycardia/pauses
• QRS behaviour	Irregular , chaotic change	Cyclic “twisting”
• Core mechanism	Trigger driven	Substrate driven

4. Take-Home Message

- ☐ Polymorphic VT is a manifestation of electrical instability, not a single disease entity ; its mechanism is dependent on delayed afterdepolarization (tachycardia dependent) in the presence of normal QT interval.
  Short-coupled PVCs and Purkinje triggers play an increasing role in modern understanding of polymorphic VT (R on T phenomenon).
  This entity may occur in association with :
  - Acute myocardial ischemia / infarction
  - Reperfusion arrhythmias
  - Severe myocardial irritability

- ☐ Torsades de pointes is a specific form of polymorphic VT that is : QT-prolongation related , Pause-dependent (‘short-long-short’ cycle) , Primarily mediated by Early Afterdepolarizations (EADs).
  This entity may occur in association with :
  - Congenital long QT syndromes
  - Drug-induced QT prolongation (antiarrhythmics, psychotropics, antibiotics)
  - Electrolyte abnormalities (hypokalemia, hypomagnesemia)
  - Severe bradycardia or AV block

- ☐ Polymorphic ventricular tachycardia and torsades de pointes represent the ECG expression of a ventricle that has lost uniformity in repolarization. They remind clinicians that electrical stability depends not only on impulse generation and conduction, but equally on orderly recovery. Recognition of the underlying

mechanism is essential, as timely correction of repolarization abnormalities can be lifesaving.

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NB : Consulted and discussed with ChatGPT whenever needed

NONSUSTAINED VT AS A WINDOW INTO VENTRICULAR ELECTRICAL INSTABILITY

ECG

NONSUSTAINED VT AS A WINDOW INTO VENTRICULAR ELECTRICAL INSTABILITY

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OUTLINE

Introduction
Nonsustained ventricular tachycardia is defined as a sequence of three or more consecutive ventricular complexes at the rate of >100 bpm , lasting less than 30 seconds , and resolving spontaneously.

An easy way to understand the concept of NSVT
NSVT is like a short-circuit spark – both are brief electrical instabilities that disrupt normal function , signaling underlying issues that demand attention

Electrophysiological Mechanism
- ☐ Reentry
- ☐ Triggered Activity
  - Early afterdepolarization (EAD)
  - Delayed afterdepolarization (DAD)
- ☐ Abnormal automaticity

ECG characteristics and their clinical significance in nonsustained VT

Take-Home Message

References

Nonsustained VT as a Window into Ventricular Electrical Instability

A Narrative Review

© **DR. D.P. KHAITAN**
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Electrical instability in the heart may be a mis-happening that disrupts and transforms an ongoing rhythmic pattern into a chaotic electrical outburst. This phenomenon may appear as nonsustained ventricular tachycardia (NSVT) on the ECG.

This nonsustained ventricular tachycardia requires a vulnerable myocardial substrate, a triggering impulse, and temporal instability for its initiation and brief persistence. Its spontaneous termination does not imply benignity; rather, it reflects transient recovery of refractoriness or interruption of re-entrant or triggered pathways. The true significance of NSVT lies not in its brevity, but in the forces that allowed it to occur.

- ☐ **This NSVT is nature's warning whisper—quiet enough to end on its own, yet clear enough to demand attention.**
- ☐ **To ignore this arrhythmia is to miss a message; to misunderstand its language. The art lies in listening carefully to what the ventricle is trying to say.**

Nonsustained ventricular tachycardia is neither a trivial curiosity nor an automatic harbinger of catastrophe. It is a brief disclosure of ventricular vulnerability, offering clinicians a window into the heart's electrical health.

1. Introduction (Keypoints)

- ○ Ventricular tachycardia has traditionally been viewed through the prism of duration and hemodynamic consequence. Among its forms, nonsustained ventricular tachycardia (NSVT) occupies a unique and often misunderstood position. Defined by its self-terminating nature, NSVT may appear deceptively benign. Yet, in clinical practice, it often represents the earliest electrical manifestation of ventricular instability.
- ○ Nonsustained ventricular tachycardia is defined as a sequence of three or more consecutive ventricular complexes at the rate of >100 bpm , lasting less than 30 seconds , and resolving spontaneously.

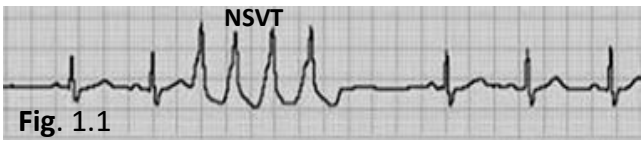


Fig. 1.1

This strip shows a run of NSVT - the R-wave of a PVC coinciding with the T-wave of the previous beat. This initiates the cadence of NSVT , fulfilling the criteria of its definition.

- NSVT is usually harmless but it can be a harbinger of more serious sustained ventricular arrhythmia with associated risk of sudden death. In individuals with underlying heart disease, structural changes such as fibrosis enhances its susceptibility to conduction block and reentrant circuits. This makes the heart more prone to NSVT when the R-on-T phenomenon occurs.

- It may occur in the absence of any underlying cardiac disorder but it may be associated with structural heart diseases whether congenital or acquired , ageing heart, metabolic problems , including drugs toxicity , electrolyte imbalances , etc.
  Always look for changes in life style : physical or mental stress , lack of sleep , excessive consumption of caffeine or alcohol intake.
  An attention should be directed towards sleep apnea if present.

- **In professional athletes** NSVT is considered a part of the "athlete's heart syndrome" having no adverse progostic significance , provided conditions such as hypertrophic cardiomyopathy (HCM) , early repolarization syndrome and genetic channelopathies are excluded.

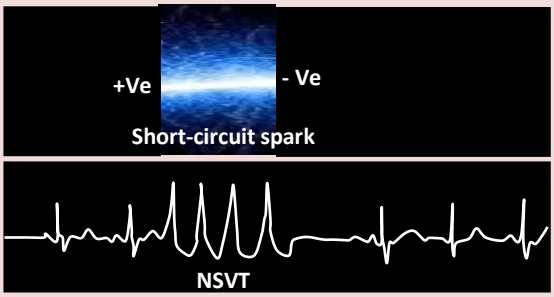
- NSVT cannot be prevented , instead life style changes that support heart health should be given due attention. And some heart conditions that predispose to NSVT should be tackled properly.

- Prognosis of NSVT is dependent upon the presence or absence of cardiac disease. Left ventricular dysfunction in post myocardial infarction patients remains the most important consideration for overall mortality and risk for sudden cardiac death.

- No increase in mortality has been documented in patients without cardiac disease in association.

2. An easy way to understand the concept of NSVT

Just for easier understanding , NSVT can be compared with home electricity with a short-circuit spark , caused by a misconnection between positive and negative wires , appearing as a phenomenon of electrical arc. This flow with electrical arc occurs because of the potential differences in between these two wires. Once the connection is fully broken , the sparks ceases. The same phenomenon more or less akin to electrical spark occurs with NSVT when there is a potential difference in vicinity



Be clear about the fact that NSVT is not the carbon copy of short-circuit spark in true sense but it can be said that NSVT is like a short-circuit spark – both are brief electrical instabilities that disrupt normal function , signaling underlying issues that demand attention

Fig. 1.2

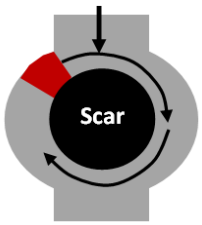
3. Electrophysiological Mechanisms

NSVT arises from the same fundamental mechanisms responsible for sustained ventricular arrhythmias. What distinguishes it is failure of perpetuation, not lack of arrhythmogenic potential. It is not defined by how long it lasts, but by what allows it to begin. The mechanisms involved are :

- ☐ Reentry
- ☐ Triggered Activity
  - Early afterdepolarization (EAD)
  - Delayed afterdepolarization (DAD)
- ☐ Abnormal automaticity

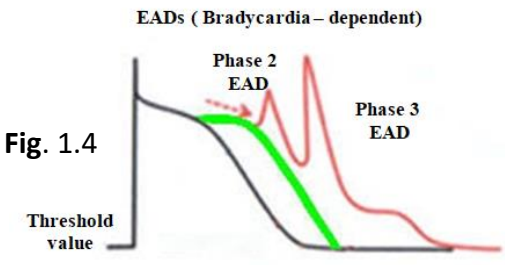
REENTRY

It is the most common mechanism in structural heart disease . NSVT occurs when the reentrant circuit is formed transiently but collapses due to refractoriness or conduction failure. It has a progonostic significance.

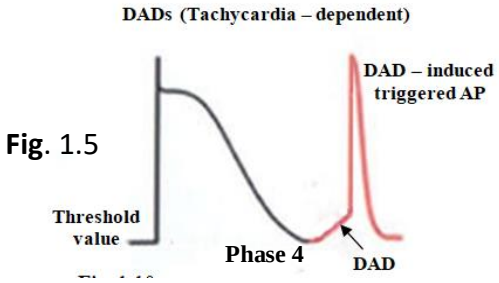
Type	Mechanism	Pathophysiological substrate
Reentry	 **Fig**. 1.3 Requires • Conduction heterogeneity (e.g. scar) • Unidirectional block (indicated by a red column) • Critical circuit length	Structural heart disease • Prior myocardial infarction • Cardiomyopathy • Ventricular fibrosis or scar

TRIGGERED ACTIVITY

Early afterdepolarization (EAD) : The vulnerable repolarization period is either earlier during the phase 2 and/or phase 3 of the cardiac action potential where the relative refractory period may allow the entry of early depolarization potential.

Type	Mechanism	Pathophysiological substrate
Early afterdepolarization (EAD)	 **Fig**. 1.4	• Long QT syndromes • Bradycardia-dependent arrhythmias

Dealyed afterdepolarization (DAD) : it may fall after full repolarization resulting in the triggered activity as delayed afterdepolarization. In contrast to EADs, DADs are always induced at relatively rapid rates.

Type	Mechanism	Pathophysiological substrate
Delayed afterdepolarization (DAD)	 **Fig**. 1.5	• Catecholamine excess • Ischemia • Digitalis toxicity

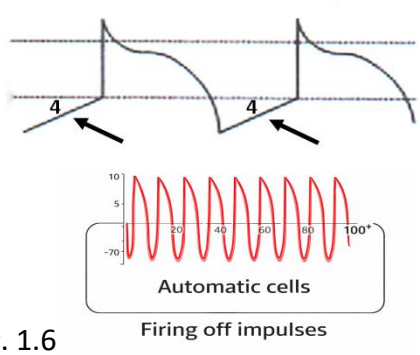
NB : When EAD / DAD amplitude is sufficient enough to bring the membrane to its threshold potential, a spontaneous action potential, referred to as a triggered activity is noted. These triggered activities may give rise to NSVT.

Comparison in between early afterdepolarization and delayed afterdepolarization induced triggered activity

Characteristics	Early afterdepolarization - Induced triggered activity	Delayed afterdepolarization - Induced triggered activity
Site	The plateau phase 2 and/or phase 3 of the action potential	Phase 4 of the action potential
Basic mechanism	Slowing of the repolarization rate associated with one or more triggered beats leading to QTc prolongation (due to high membrane resistance during this phase of relative refractory period is usually not capable of inducing tachycardia directly)→ Dispersion of ventricular repolarization → NSVT	As the cycle length decreases, the amplitude and the rate of DADs enhances → NSVT
Rate dependency	Bradycardia – dependent	Tachycardia – dependent
Electrophysiology	Through K+ Current	Burst of Ca++ release from sarcolemma sac with activating electrogenic Na +-Ca++ exchanger activity

ABNORMAL AUTOMATICITY

Automaticity is the inherent quality of certain group of cardiac cells, capable of firing the impulses spontaneously without any provocation, as a result of diastolic depolarization during phase 4 of the action potential, which is responsible for bringing the membrane potential quickly to threshold value

Type	Mechanism	Pathophysiological substrate
Abnormal Automaticity	 **Fig**. 1.6	• Acute ischemia • Myocarditis • Electrolyte imbalance

NB : Mechanism of Nonsustained VT in a Structurally Normal Heart

- Nonsustained VT in a normal heart is most commonly due to triggered activity or focal automaticity rather than reentry.
- Apparently Normal Hearts :
  - RVOT / LVOT myocardium
  - Fascicular VT
  - Purkinje-related arrhythmias often benign
- May also be encountered with Systemic and Metabolic condition :
  - Electrolyte disturbances (K^+, Mg^{2+})
  - Sepsis
  - Hypoxia
  - Acid–base disorders
- These arrhythmias are usually catecholamine-sensitive, pause-dependent, or exercise-related, and often terminate spontaneously due to lack of sustaining substrate.

4. ECG characteristics and their clinical significance in nonsustained VT

In NSVT, the ECG is not merely diagnostic—it is prognostic as well

⇒ **Rate of NSVT (Ventricular Rate)**
Faster NSVT runs indicate greater electrical instability. High rates shorten diastole, increase dispersion of refractoriness, and promote wavefront fragmentation. Fast NSVT is more likely to degenerate into sustained VT or VF.

Clinical implication: Rate acts as a stress test of ventricular electrophysiology.

⇒ **Coupling interval**

Short coupling interval PVCs with NSVT runs are more dangerous. It allows impulses to fall during the vulnerable phase of repolarization (R-on-T phenomenon) and is commonly commonly associated with Purkinje-mediated arrhythmias and idiopathic VF.

Clinical implication : Timing is often more important than morphology.

⇒ **Monomorphic NSVT**

It suggests a stable focus or circuit. Risk depends on underlying substrate (benign in normal hearts, malignant in scarred myocardium)

Clinical implication: Electrical chaos is more dangerous than electrical organization.

⇒ **Electrical Axis of NSVT**

Superior axis : Origin from RVOT/LVOT **,** usually benign, catecholamine-sensitive

Inferior axis : Origin from inferior LV, apical scar - higher arrhythmic risk

Clinical implication: Axis localizes origin and stratifies risk.

⇒ **Site of Origin Suggested by ECG**

Outflow tract NSVT : Superior axis, LBBB/RBBB pattern , Usually benign

Purkinje-related NSVT : Narrow QRS, short coupling interval , May trigger polymorphic VT or VF

Scar-related NSVT : Fixed morphology, often inferior axis **, s**trong predictor of malignant arrhythmias

⇒ **Exercise and Recovery Phase Behavior**

NSVT during exercise : Often catecholamine-mediated , frequently benign in structurally normal hearts

NSVT during recovery phase : Indicates impaired autonomic and repolarization reserve **.** stronger predictor of mortality

Clinical implication: Recovery unmasks vulnerability; exercise applies stress.

⇒ **ECG Context Over Duration**

Duration (<30 s) alone is misleading Prognosis depends on rate , coupling interval, morphology, axis, triggering circumstances.

Clinical implication: NSVT should be interpreted as a signal, not a standalone diagnosis.

5. Take-Home Message

- ☐ Nonsustained ventricular tachycardia is not defined by its brevity, but by the vulnerability it reveals.
- ☐ NSVT represents a transient loss of ventricular electrical stability that terminates spontaneously—not necessarily because it is benign, but because conditions for perpetuation are momentarily incomplete.
- ☐ The clinical significance of NSVT depends entirely on context
  - In structurally abnormal hearts, it is often a marker of increased arrhythmic risk.
  - In structurally normal hearts, it is frequently benign but still demands thoughtful evaluation.
- ☐ NSVT cannot be prevented , life style changes that support heart health should be given due attention. And some heart conditions that predispose to NSVT should be tackled properly.
- ☐ Judge the nature of NSVT if there is underlying heart disease , treat this. This would be worthwhile to mention here that frequent ventricular ectopy during recovery after exercise is a better predictor of an increase risk of death than ventricular ectopy occuring only during exercise. This is essential to follow up the patients and modify risk factors if they are present therein.
- ☐ In nonsustained ventricular tachycardia, the ECG does not merely record the rhythm—it reveals the vulnerability of the ventricle as well.
- ☐ Further evaluation of NSVT should be individualized and guided by clinical context , with the primary aim of identifying the underlying causative factor.

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NB : Consulted and discussed with ChatGPT whenever needed

Personal Notes :