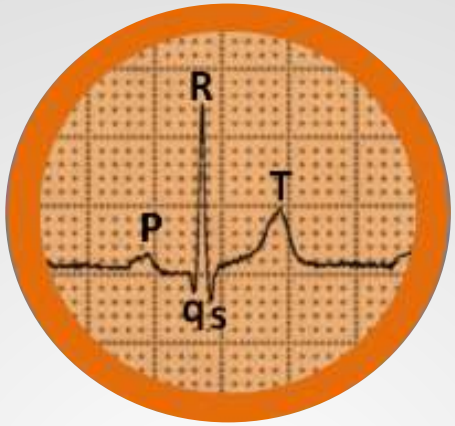

A PRACTICAL APPROACH TO VT ANALYSIS ON ECG

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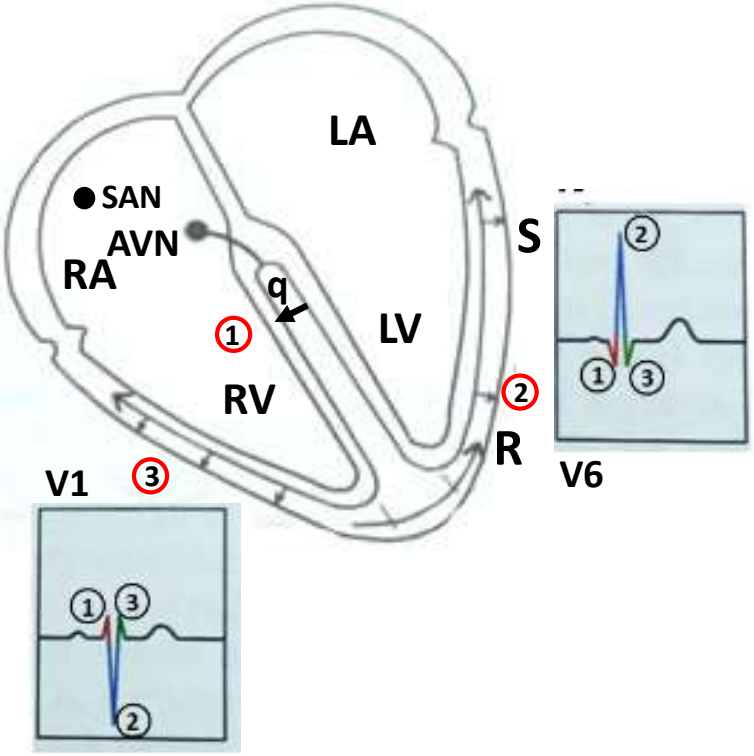
Gayajee , Bharat

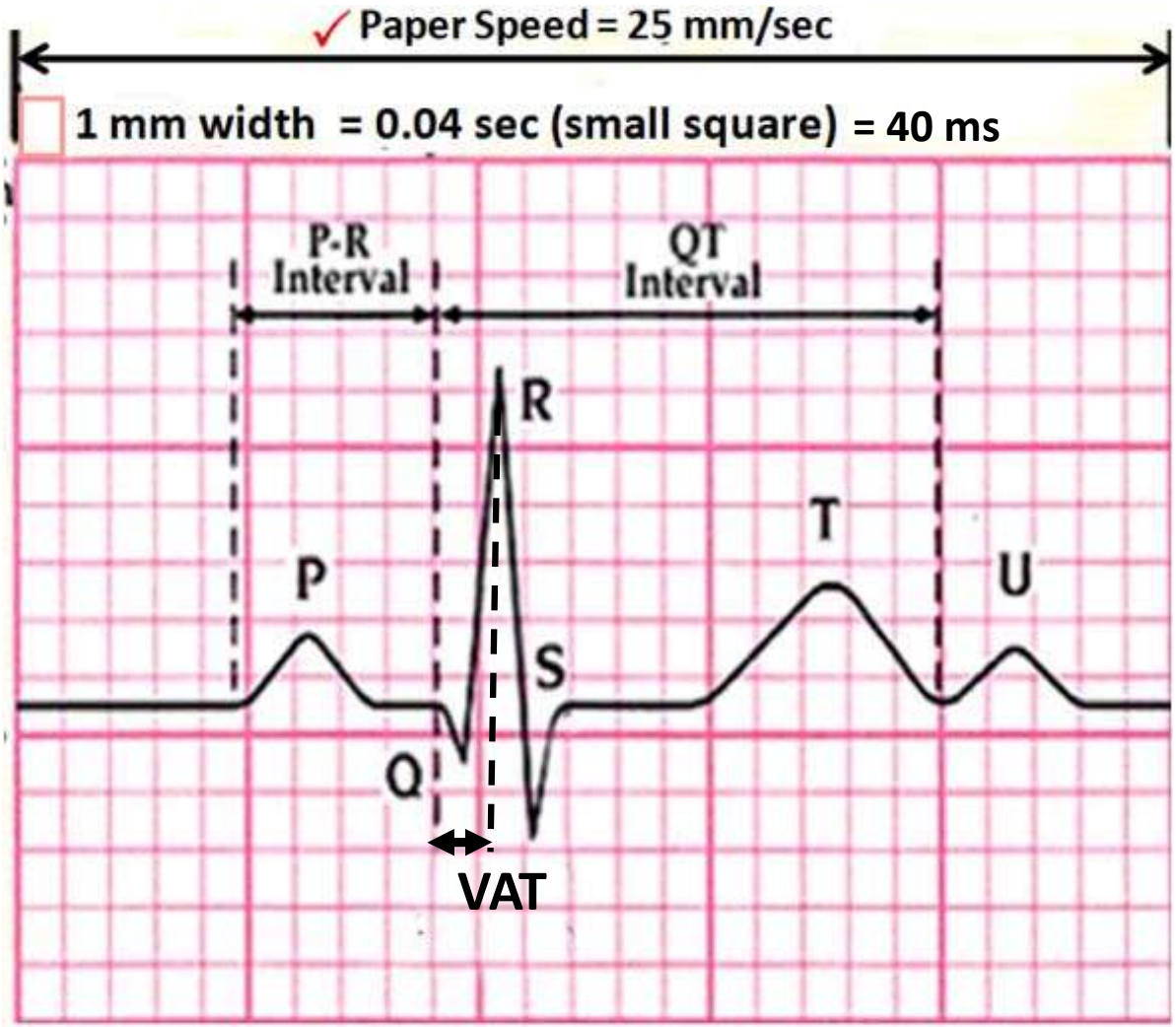
Normal ECG

- Positive wave towards the flow of current
- Negative wave away from the flow of current



- Purkinje-mediated conduction
- Sequential ventricular activation

HIS PURKINJE SYSTEM JOURNEY



In a normal ECG, one small square (40 ms) accommodates septal activation and ventricular activation time ; crossing this limit implies loss of Purkinje-mediated conduction.

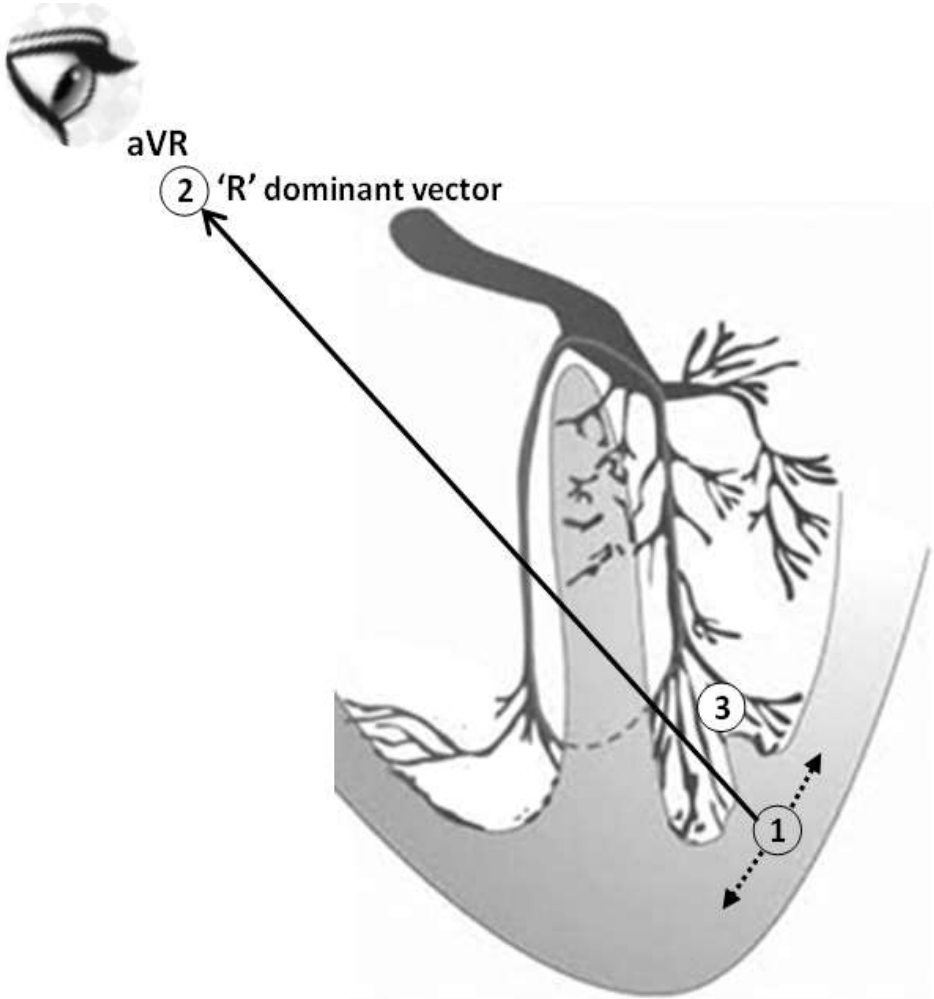
Ventricular tachycardia

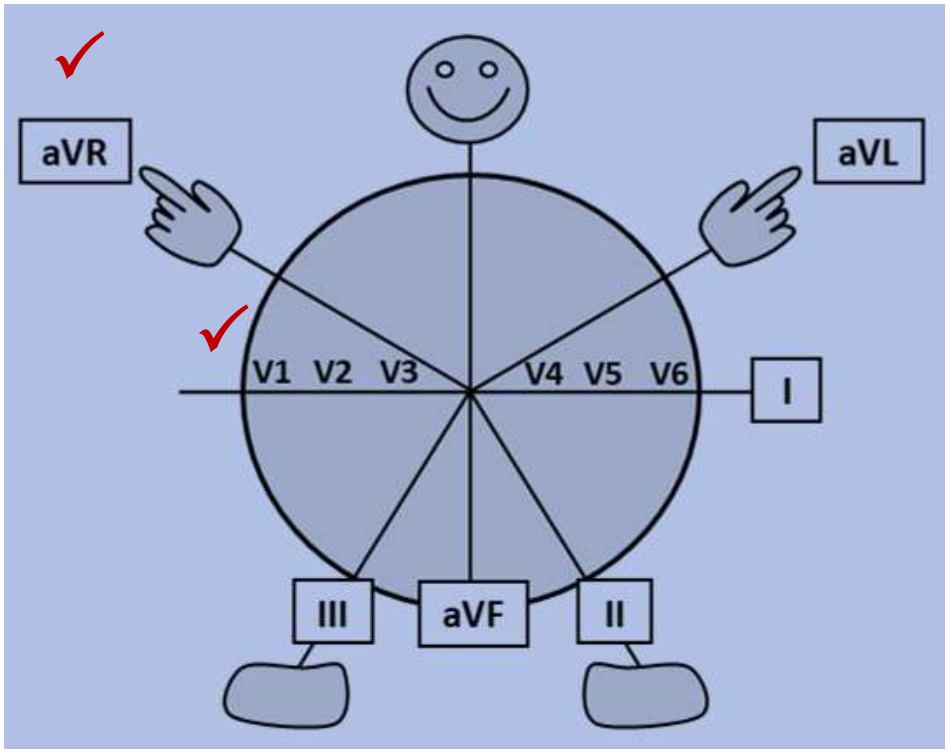
- VT = a rapid (>100 bpm), independent, wide-QRS rhythm driven by the ventricles, not the atria (the ventricles act as the pacemaker)
- The QRS is wide (>120 ms), often bizarre in morphology
- The atria and ventricles are dissociated (AV dissociation) : no fixed AV relationship
- Mechanism: reentry, triggered activity or enhanced automaticity

Electrophysiological basis in VT : Reverse transmission

Reverse transmission — first through ventricular myocytes and then through the His-Purkinje system — is sufficient to identify VT, irrespective of whether Vereckei, Brugada, or any other criteria are used. Different leads merely display different morphological expressions of the same principle.

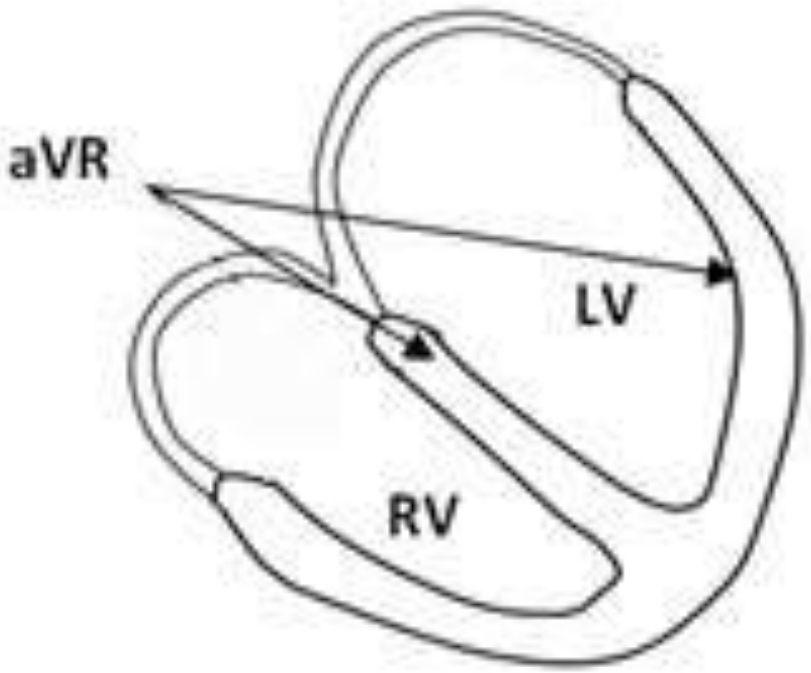
HOW VT IS REGISTERED THROUGH LIMB LEADS / CHEST LEADS





- Reverse transmission — first through ventricular myocytes and then through the His-Purkinje system
- The frontal plane best detects the electrophysiological principle of VT, > while the horizontal plane refines, confirms, and localizes it

HOW VT IS EVOLVED



Mechanism decides

- Muscle to Muscle (Vi) → then to the purkinje front (Vt)
- Frontal QRS axis – mostly north-west axis
- Novel Basal Algorithm – RWPT > 40 ms
  At least 2 criteria (structural heart disease + lead II + lead aVR)

Morphology supports

- VT breaks the normal sequence of ventricular activation.
  Loss of orderly V1→V6 progression = VT , as in the case of Brugada algorithm

Mathematics refines – physiological rules

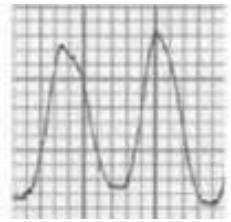
- One small square = 40 ms
- Vi /Vt ratio < 1 → VT
- RWPT > 40 ms → VT

Vereckei aVR Algorithm

- QRS is a dynamic process, not a static shape

Initial dominant R wave

Step 1



Initial r or q wave > 40 ms

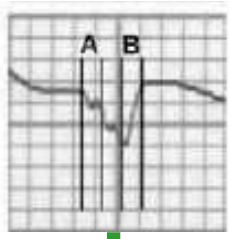
Step 2

Notched downstroke of negative QRS

Step 3

V(i)/V(t)≤1

Step 4



VT

Any +Ve sequential step suggests VT

Vi= Voltage amplitude in first 40 ms of QRS
Vt = Voltage amplitude in last 40 ms of QRS
Vi/Vt ≤ 1

Brugada Algorithm for VT : Loss of orderly V1→V6 progression = VT

Step 1 Absence of RS complexes in all precordial leads

Yes → VT

Step 2 R to S interval > 100 msec in 1 precordial lead

Yes → VT

Intrinsic ventricular conduction
100 ms = 2.5 small square

Step 3 More QRS complexes than P waves (AV dissociation)

Yes → VT

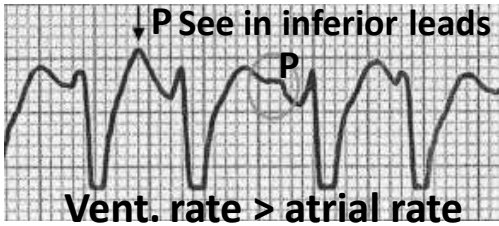
Step 4 Morphologic criteria for VT present in V1-V6

- Atypical RBBB/ LBBB (Absence of typical BBB)
- Total +Ve /-Ve QRS concordance

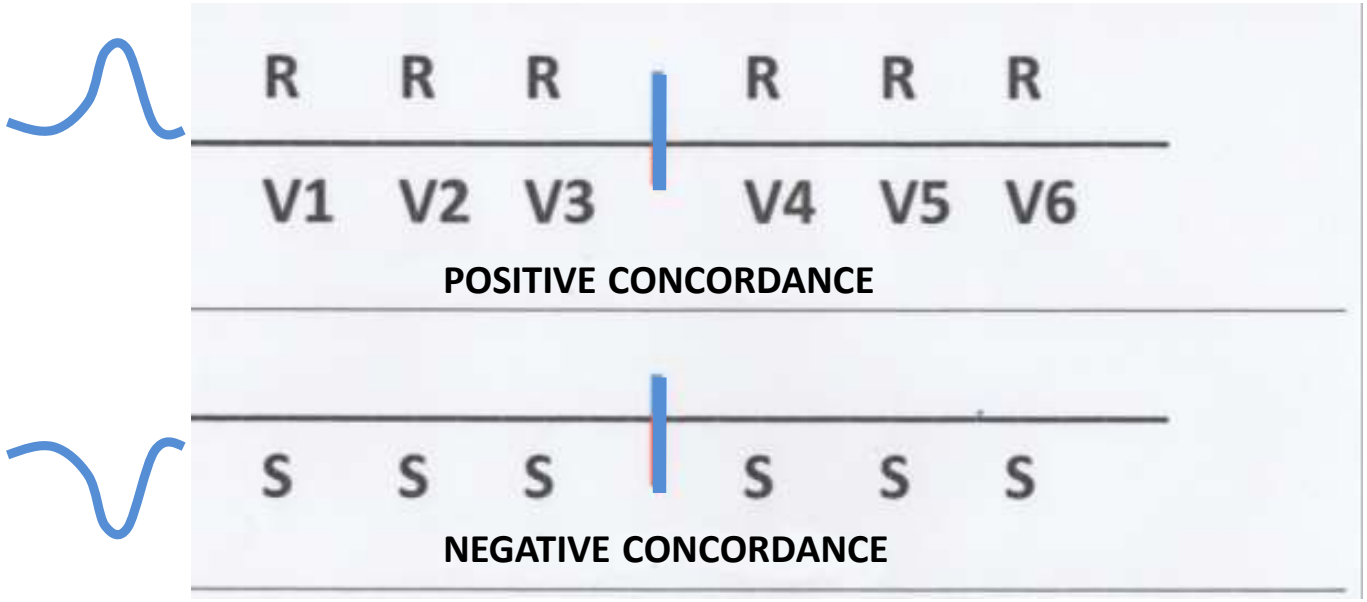
Yes → VT

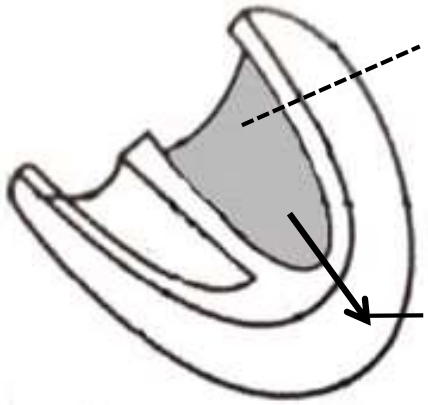
SVT with aberrant conduction

Unidirectional ventricular activation → Either only R OR QS



Chest leads concordance





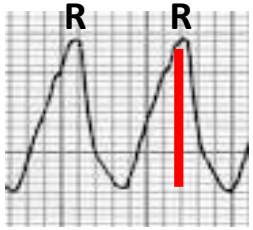
Positive concordance = Origin of VT from posterobasal left ventricle
Negative concordance = Origin of VT from anteroapical left ventricle

The Novel Basal Algorithm (VT)

BASIS = Muscle-to-Muscle conduction (RWPT)

Structural Heart Disease + **Lead -II** RWPT >40ms + **Lead-aVR** RWPT >40ms

- H/O Myocardial Infraction
- CHF (LVEF < 35%)
- Device (ICD , CRT)



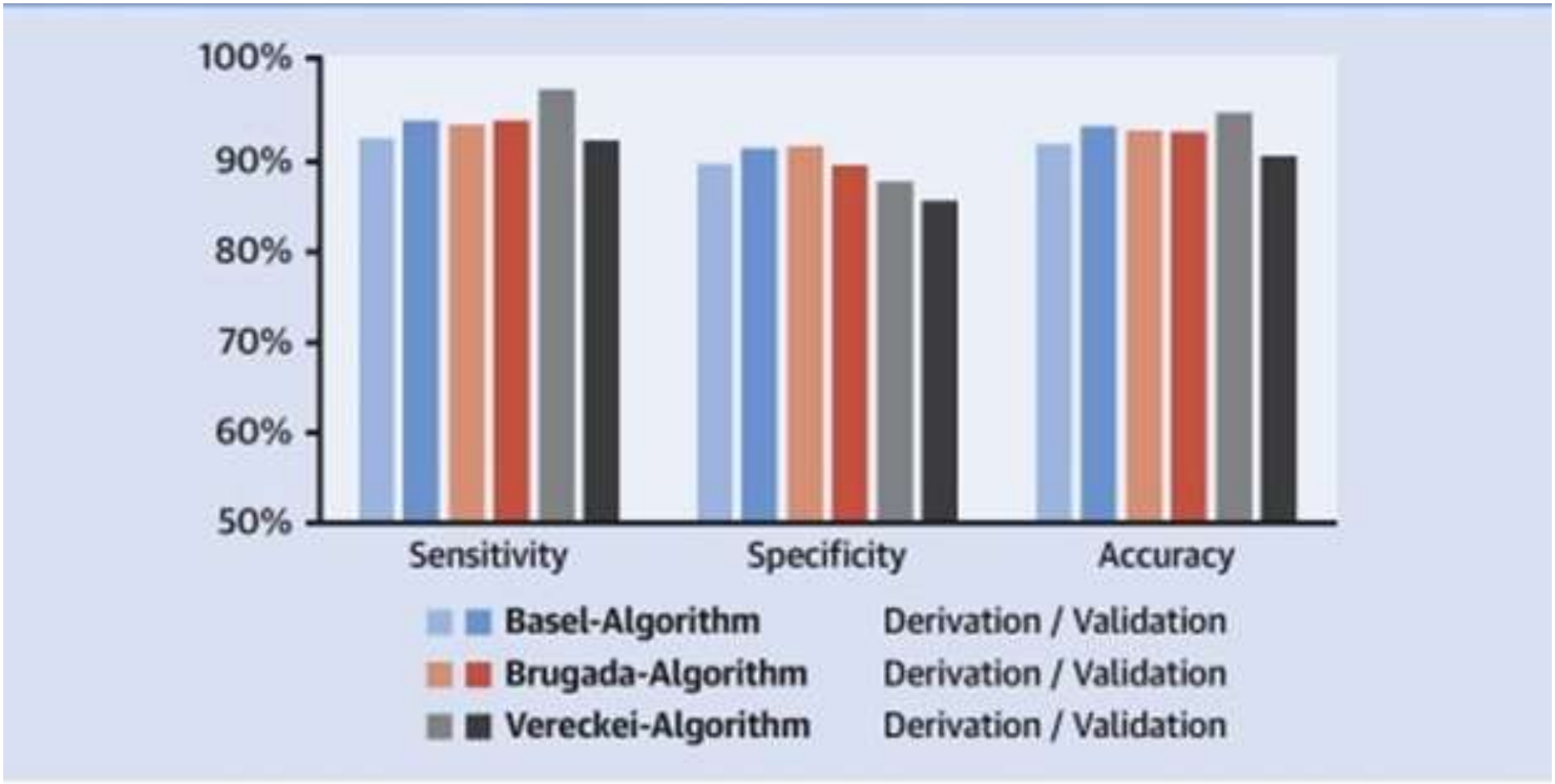
RWPT = R-wave peak time

≥2 criteria fulfilled ->VT

0 or 1 criteria fulfilled ->SVT

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

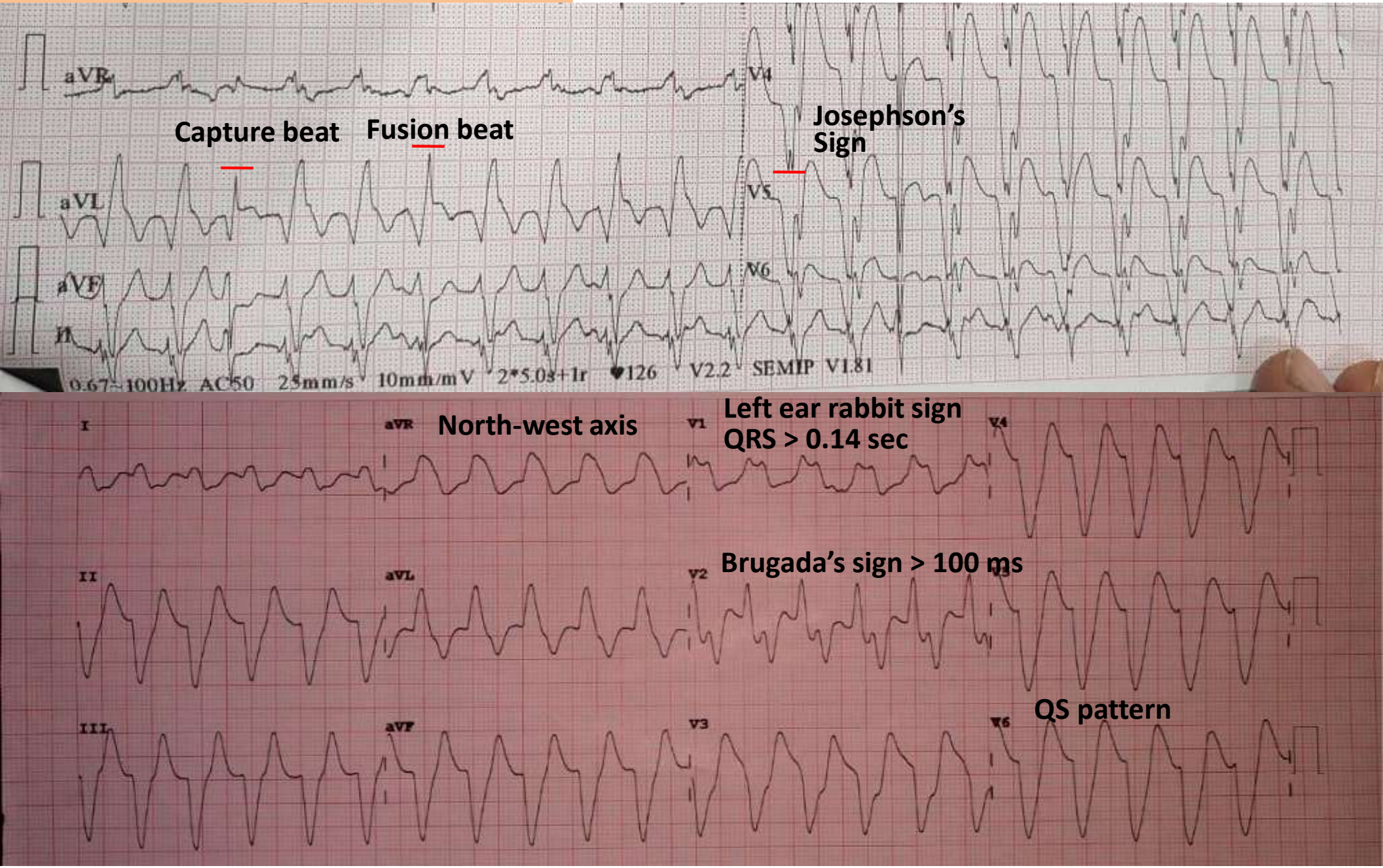
Comparision of Algorithm Performance



Ref : Moccetti F, et al. J Am coll Cardiol EP. 2022; 8 (7):831-839

Ventricular tachycardia - VT

AV dissociation with faster ventricular rate of ventricular origin



NB : ✓+Ve or –Ve concordance throughout the chest lead , i.e. with leads V1-6 so entirely positive (R) or entirely negative (QRS complexes) , with no RS complexes seen

Mastercard in Assessing VT : Holistic approach

Vertical plane	Horizontal Plane
❑ Vereckei aVR algorithm : • Initial dominant R-wave • Initial r or q wave > 40 ms • Notched downstroke of negative QRS • V(i)/V(t)<1 AV +Ve sequential Step suggests VT • N-W axis / extreme axis deviation ❑ The Novel Basal Algorithm • Structural heart disease + Lead II RWPT >40ms +Lead-aVR + RWPT >40ms • ≥2 criteria fulfilled ->VT (0 or 1 criteria fulfilled ->SVT)	❑ Brugada algorithm for VT (stepwise) • Absence of RS complexes in all precordial leads • R to S interval > 100 msec in 1 precordial lead • More QRS complexes than P waves (AV dissociation) • Morphologic criteria for VT present in V1-V6 (absence of typical BBB , total +Ve /-Ve QRS concordance) ❑ If VT cannot be confidently excluded, treat as VT. This is not dogma — it is patient safety ❑ VT prefers a diseased heart. Clinical background matters as much as ECG.

Clinical Context in VT

- **Does the heart have a reason to generate VT?**
  - Age > 35
  - Structural heart disease
  - Prior MI
  - Cardiomyopathy
  - ICD/CRT presence
  - Hemodynamic instability

NB :

VT prefers a diseased heart.

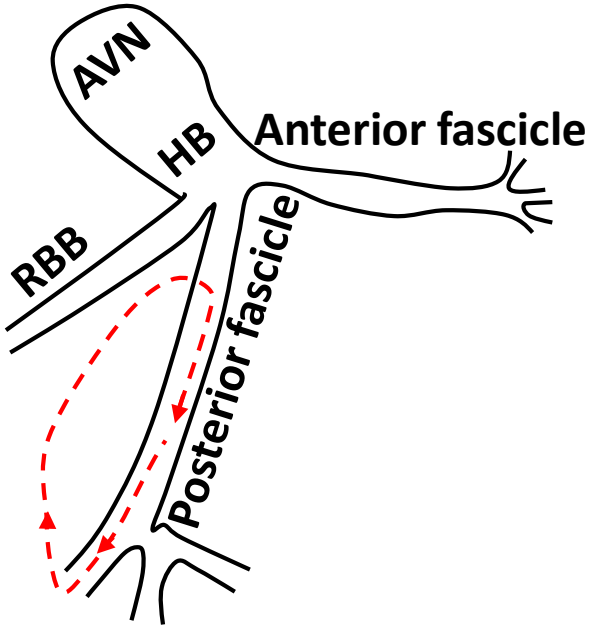
Clinical background matters as much as ECG.

Classification of VT

ECG Findings	Classification
3 or more PVCs in a row, <30 sec in duration	Non sustained VT (NSVT)
VT lasting >30 sec and/or causing hemodynamic instability	Sustained VT
VT with stable QRS morphology from a single focus within the ventricles.	Monomorphic VT
VT with variable QRS morphology originating from different sides of the ventricles	Polymorphic VT
Polymorphic VT occurring in people with prolonged QTc interval with 'twisting around an axis'.	Torsades de pointes

Rate dependent subsets of VT

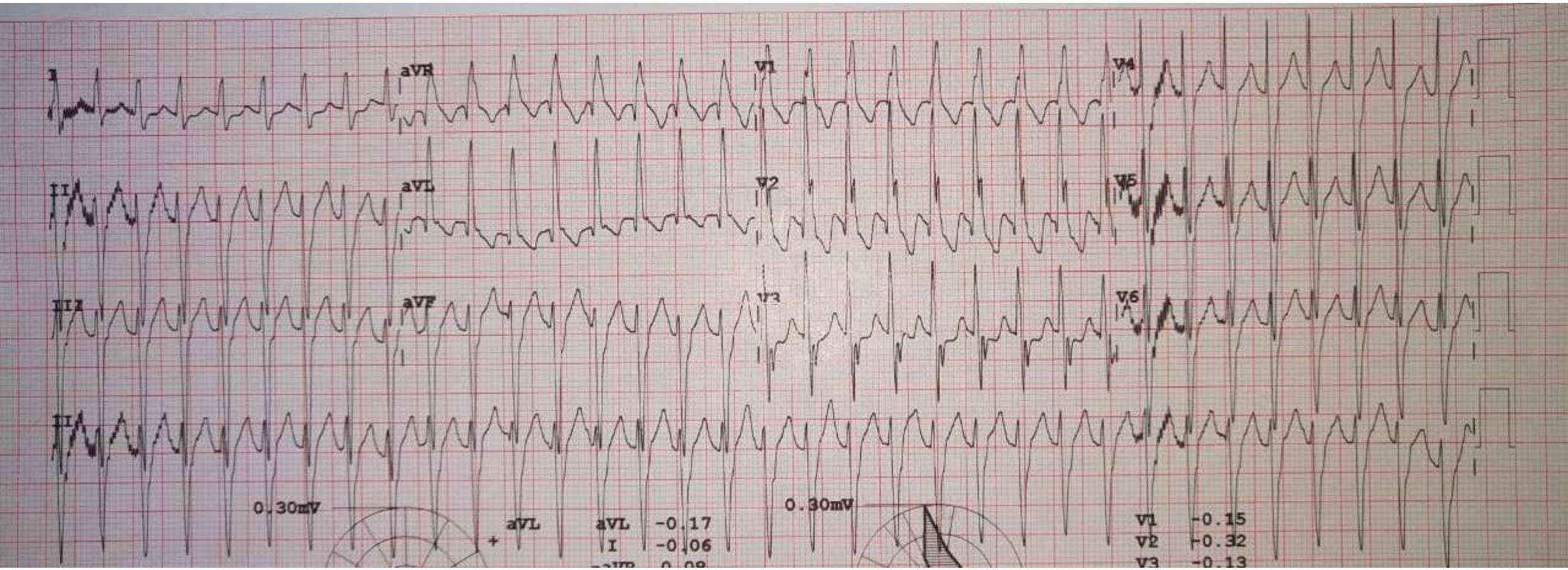
- 100–120 bpm → classic “slow VT” → large, slow circuits on diseased tissue (Diseased substrate limits speed → slower cycle) : Post-infarct scar VT, dilated cardiomyopathy, AIVR
- >150–200 bpm → typical fast monomorphic
- VT>200 bpm → Fast VT → small, fast circuits on normal tissue : Triggered by catecholamines (e.g., RVOT VT , CPVT) or tight reentry
  (fascicular VT, BBRVT , Narrower or with less broader QRS compared to slow VT)



- **Posterior fascicular VT (Narrow complex) :**

  RBBB pattern + Left axis deviation , arising close to the left posterior fascicle

  It occurs in young healthy person mostly in males. The episodes may either arise at rest or may be triggered by exercise, stress and beta agonists. The mechanism is re-entrant tachycardia.

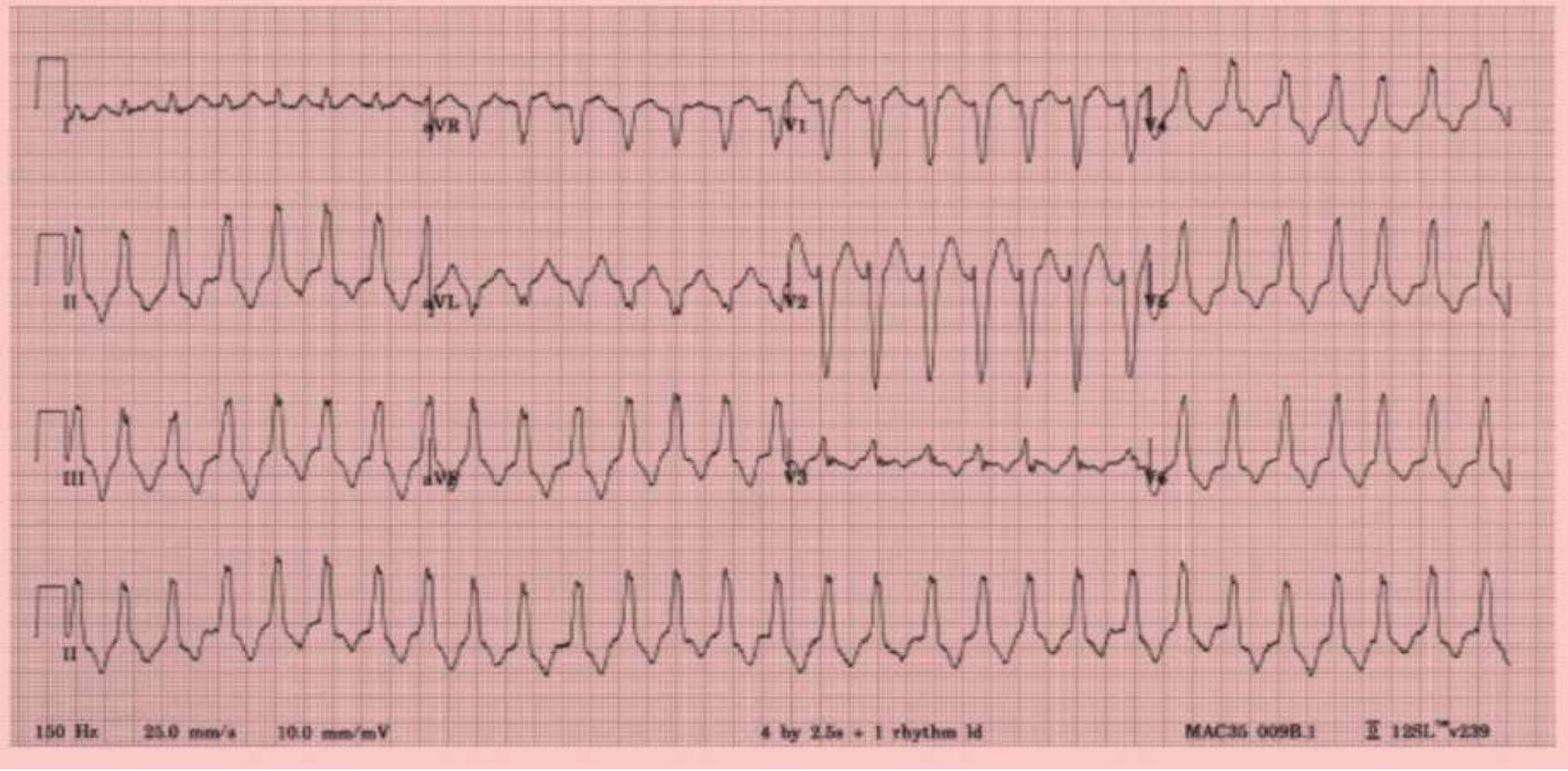


❑ RVOT-VT (Idiopathic , related to triggered activity – delayed afterdepolarizations , sensitive to catecholamines)

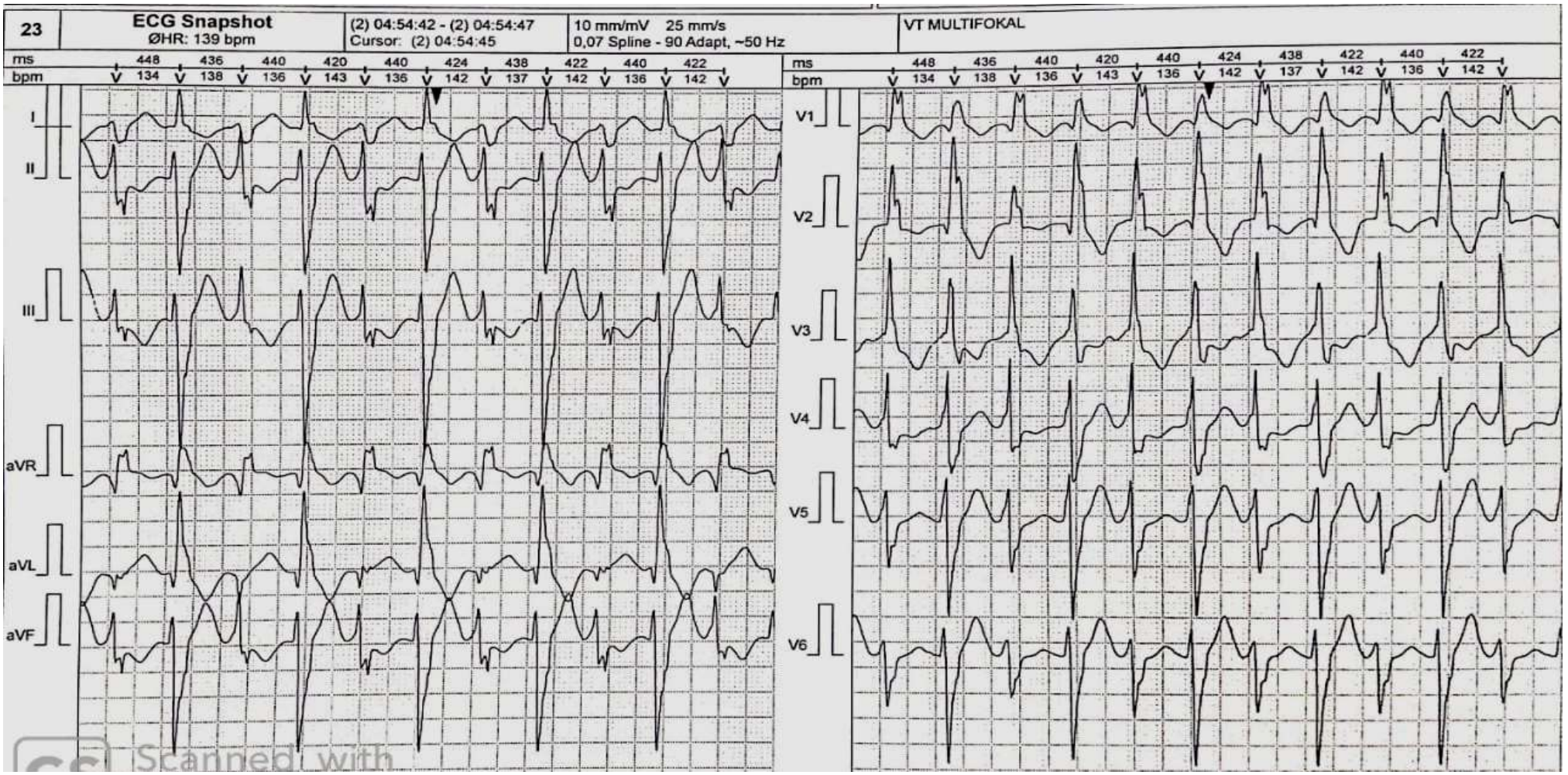
Arising from right ventricular outflow tract which is an infundibular extension of the ventricular cavity , connecting it to the pulmonary artery

ECG findings :

- **Relatively narrow QRS with somewhat higher rate**
- **Axis QRS + 110^0 (or may be inferiorly directed)**
- **Left bundle branch morphology**
- **Earlier transition zone , often in V3-V4**

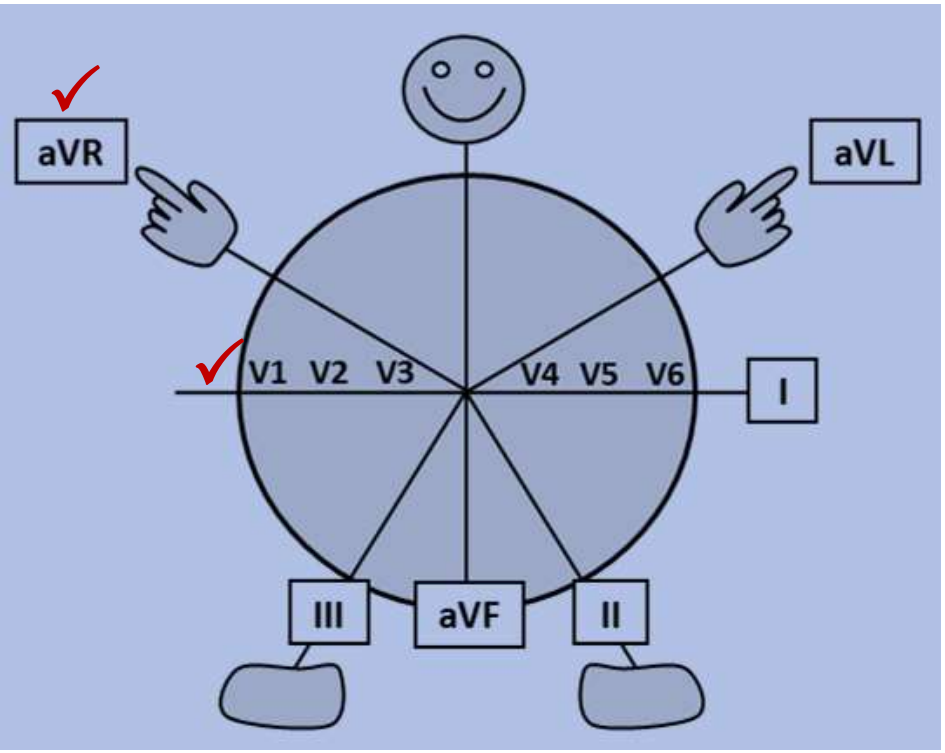


ECG showing CPVT



A female 24 years old with history of palpitation – CPVT - Bidirectional VT (Two alternative ventricular foci or alternating triggered activity between → The left fascicular and the right ventricular purkinje system)

(Source : Global Heart Rhythm Form by Dr. Fera Hidayati – Famous Cardiologist of Indonesia , posted on 18th Feb..2020)



Concluding remark

VT analysis isn't about memorizing patterns — one should be acquainted with the heart's language with clarity and so saving lives with confidence. The flow of electrical current through both the vertical and horizontal planes makes VT analysis on ECG truly simple and precise.


Thanks