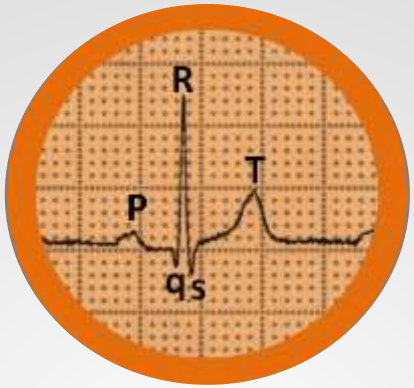


VT Analysis on ECG Made Simple

DR. D.P Khaitan

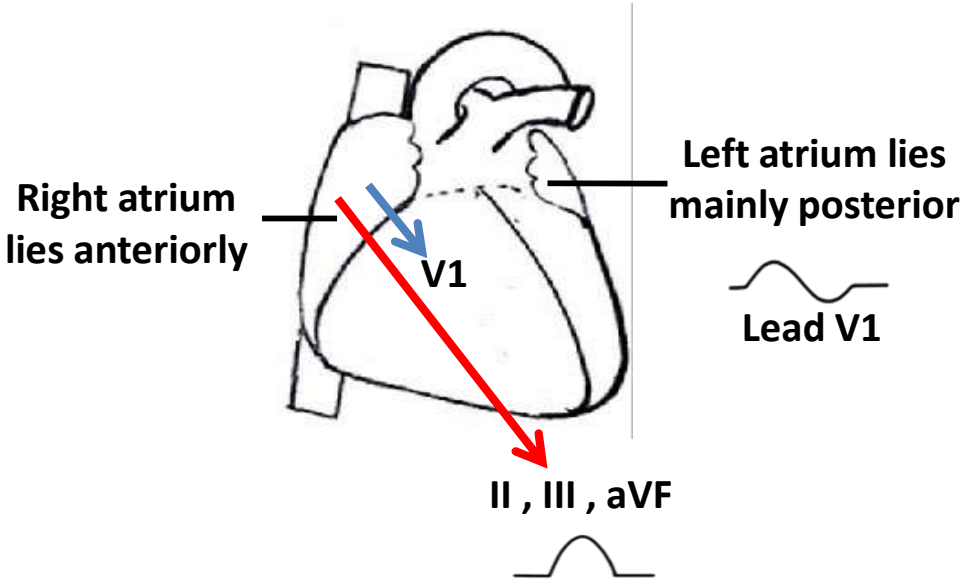
M.D (Medicine) F.C.G.P(IND) F.I.A.M.S (Medicine) FICP FICCMD FIACM

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

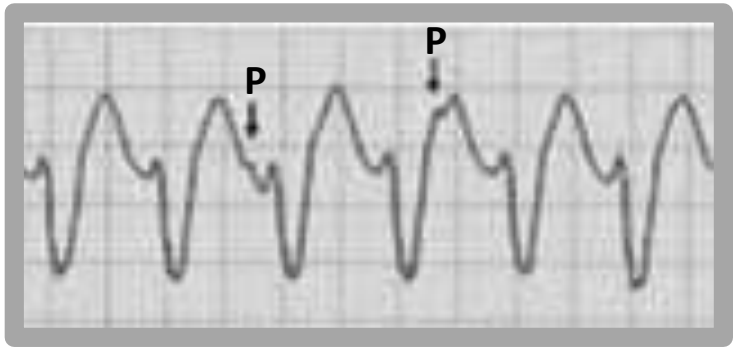
P amidst AV Dissociation

- Look for P waves marching through at their own regular rate (often ~60–100 bpm) — even though QRS are wide and fast– no fixed relationship
- Check inferior leads (II, III, aVF) / V1 , where P-waves often stand out best (apply "Haystack principle" described by Dr. Marriot)

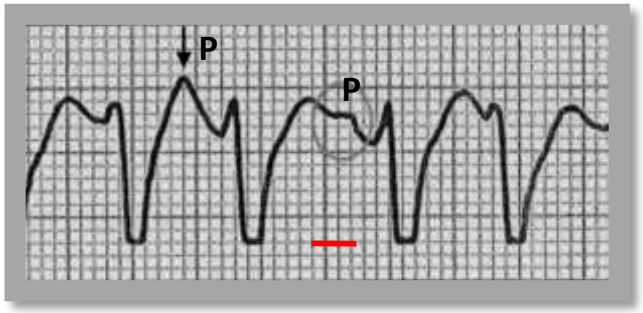


- Indirect evidence : capture / fusion beats
- Even group beating or occasional slight cycle length changes can hint at atrial influence.

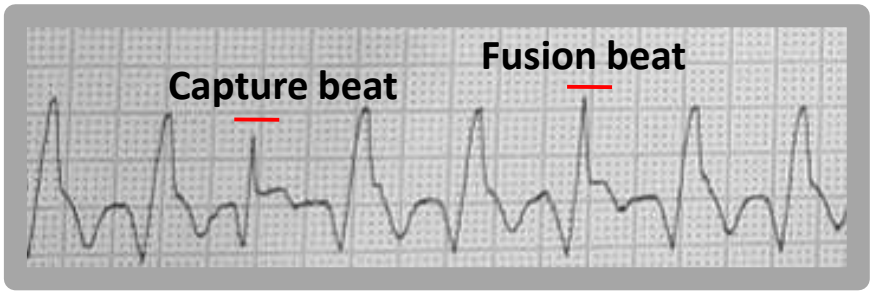
Spot P in VT: Direct and indirect evidence



P/QRS – no fixed relationship

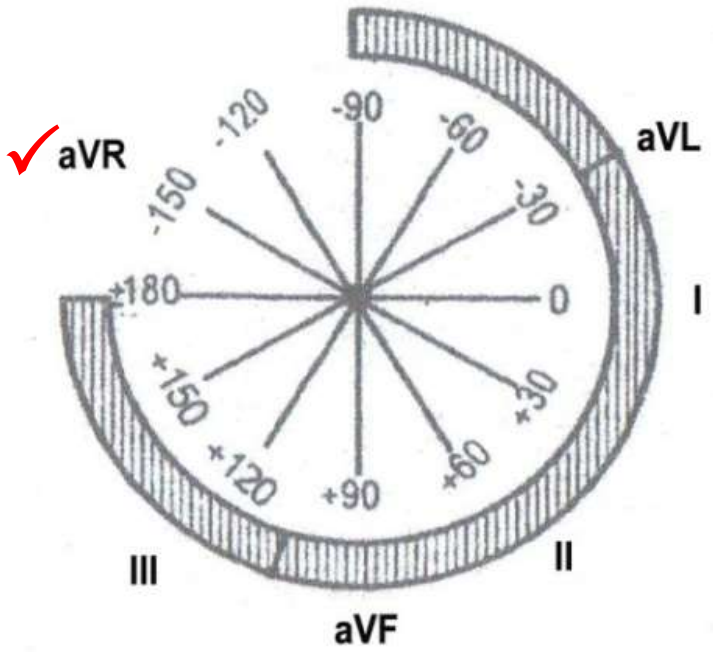


Occasional a slight cycle length change can hint atrial influence

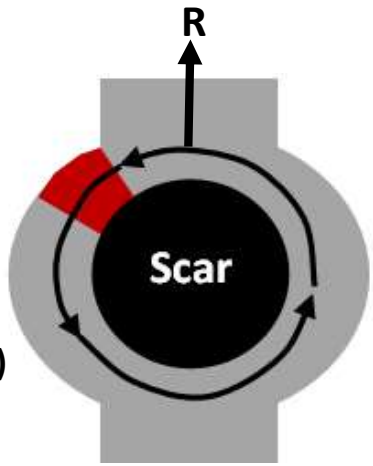


Capture / fusion beat : indirect evidence of ‘P’ in AV dissociation

Conceptual Basis of Vereckei-Criteria



Initial Vi
A small , broad negative deflection (initial q or rS) due to fragmented conduction



Terminal Vt
Higher-amplitude +ve wave compared to Vi

High looking bird's-eye view (vertical plane) with more coverage :

- **aVR captures initial , middle and terminal conduction phases**

Initial: slow muscle-to-muscle (Vi)
Middle: dominant vector (R wave)
Terminal: possibly faster Purkinje or completing wavefront (Vt)

- **Vereckei criteria – precision tool , sensitive to subtle vertical shift :**

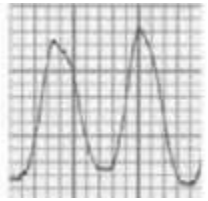
All the components of this triphasic phase may not be present simultaneously , any +ve sequntial step suggests VT.

Vereckei aVR Algorithm

- 'R-r/q-N-V'
- Zoom lead aVR for the purpose

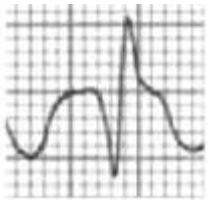
Initial dominant R wave

Step 1



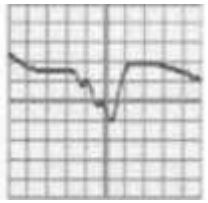
Initial r or q wave > 40 ms

Step 2



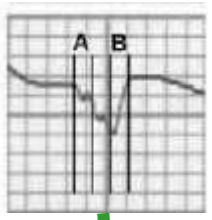
Notched downstroke of negative QRS

Step 3



V(i)/V(t)≤1

Step 4

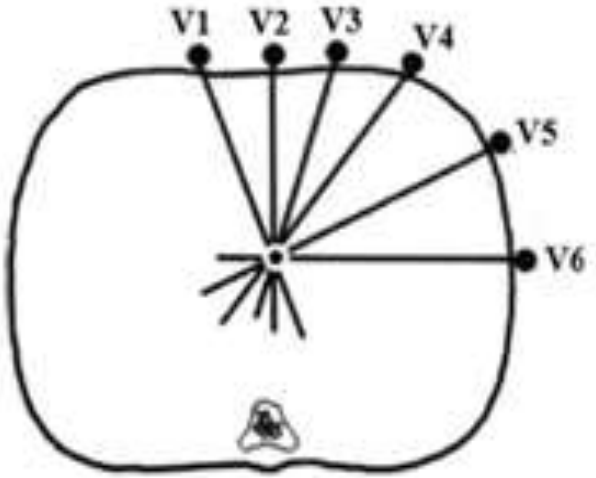


VT

Any +Ve sequential step suggests VT

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

Conceptual Basis of Brugada criteria



The dominant horizontal deflection comes from the main bulk depolarization (middle phase) , not from the starting or ending activation forces

Horizontal transmural flow with a restricted coverage

- Focuses mainly on the dominant depolarization wave (muscle-to-muscle conduction)
- Fine initial and terminal deflections are nullified

Brugada Algorithm for 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

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

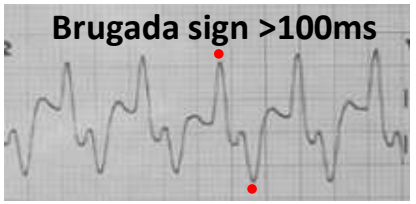
Yes → VT

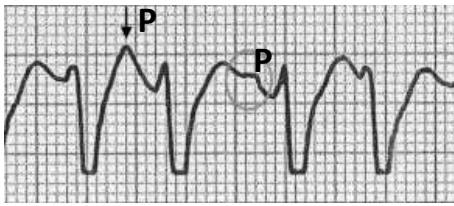
Step 4 Morphologic criteria for VT present in V1-V6

- Atypical RBBB/ LBBB
- Total +Ve /-Ve QRS concordance

Yes → VT

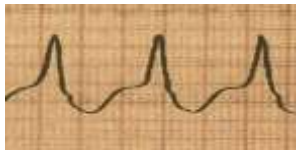
SVT with aberrant conduction



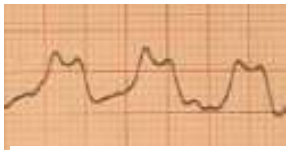


Atypical RBBB pattern with VT

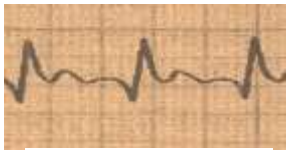
Simultaneous flow across V1 and V6 : through muscle to muscle transmission



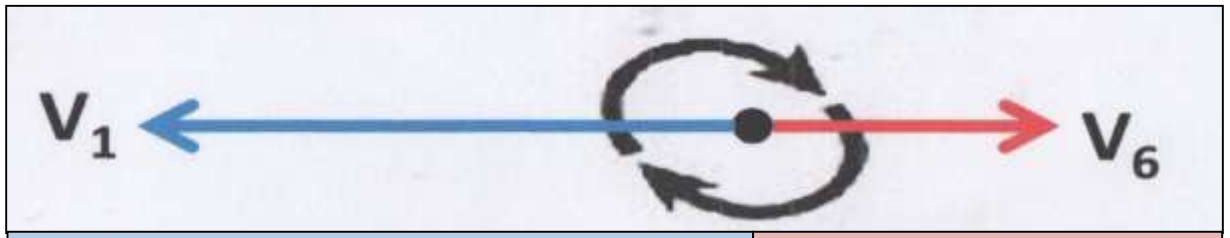
'Monophasic R'



'Left rabbit ear'



Biphasic qR



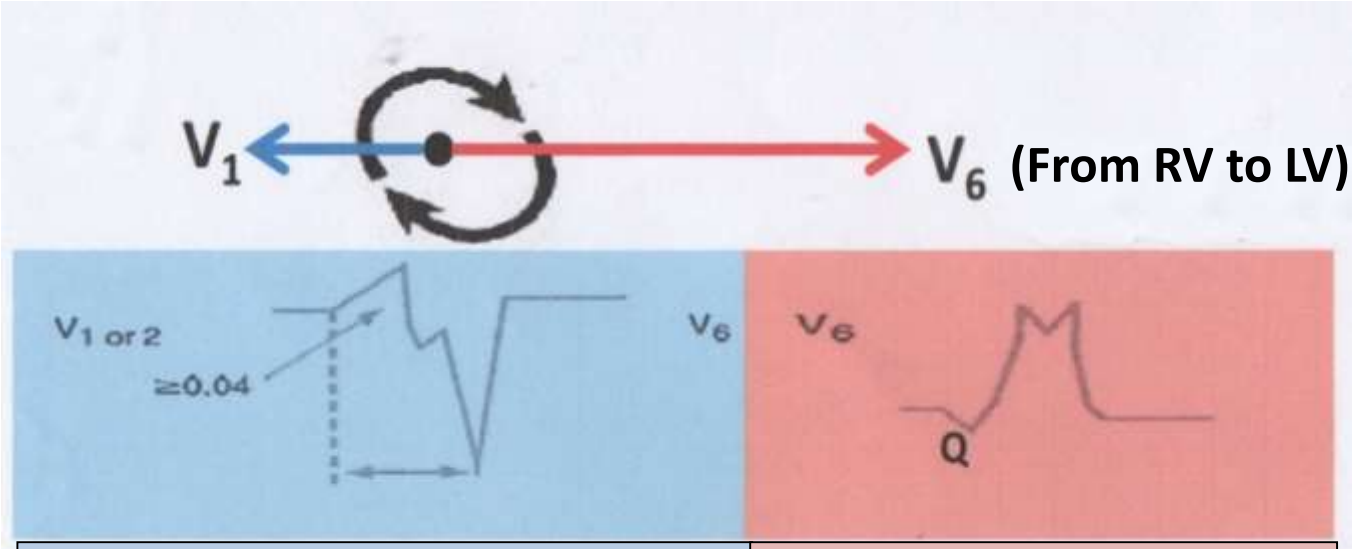
From LV to RV

Lead V1 :	**Lead V6 :**
Monophasic R, Rsr', biphasic qR – double-peaked R with the left peak taller than the right – Rabbit ear sign	R:S ratio <1 , rS, QS patterns (More or less mirror image)

A QRS duration > 140ms with RBBB pattern suggests VT

Atypical LBBB pattern with VT

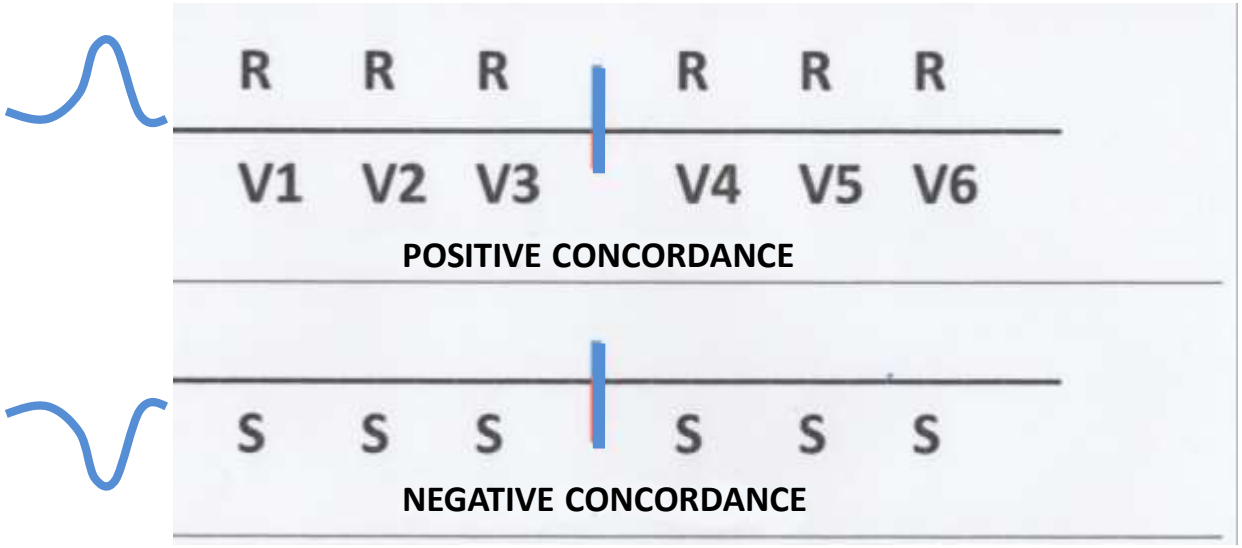
Simultaneous flow across V6 and V1 : through muscle to muscle transmission

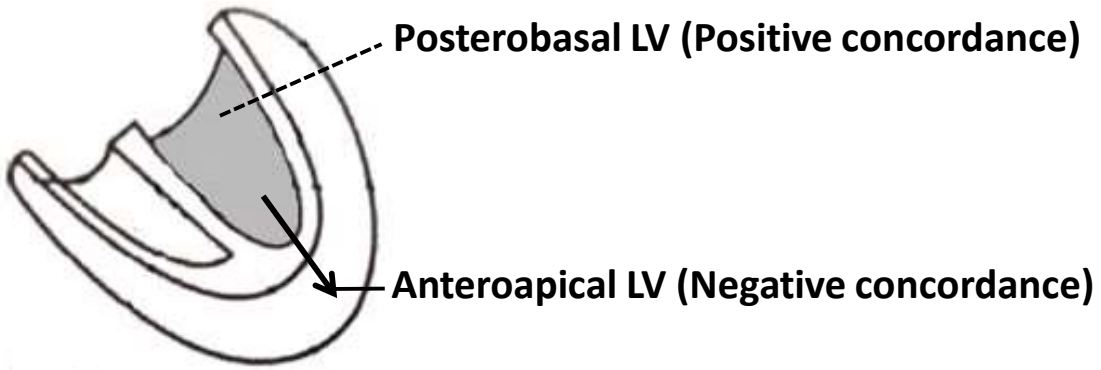


Lead V1/V2 :	**Lead V6 :**
Initial broad R wave (>40 ms) slurred or notched-down stroke of the S wave, and delayed nadir of S wave (>60 ms)	Any Q or QS

A QRS duration > 160ms with LBBB pattern suggests VT

Chest leads concordance





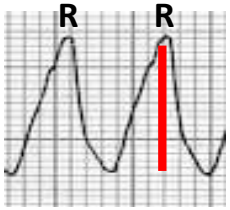
Positive concordance = Origin of VT from posterobasal left ventricle
Negative concordance = Origin of VT from anteroapical left ventricle

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)



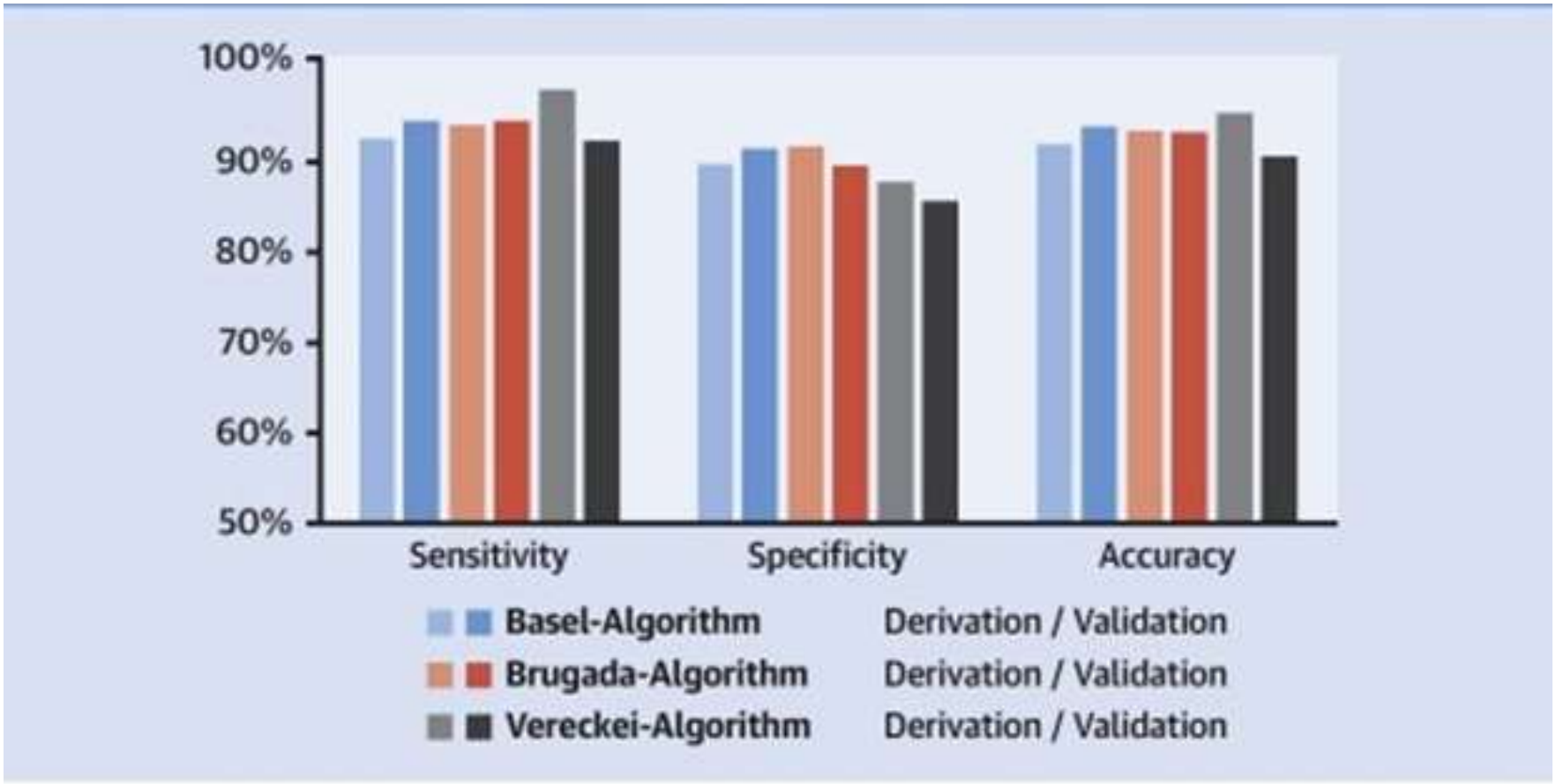
RWPT = R-wave peak time

≥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

Mastercard in Assessing VT

Vertical 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
- Opposing QRS in limb leads

  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)

Horizontal Plane

- 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

  Atypical RBBB Rabbit ear sign , Monophasic R , Biphasic qR (V1) + R:S ratio <1 , rS, QS (V6)

  Atypical LBBB Q wave or initial q wave in V6 with LBBB pattern

  Total +Ve /-Ve QRS concordance

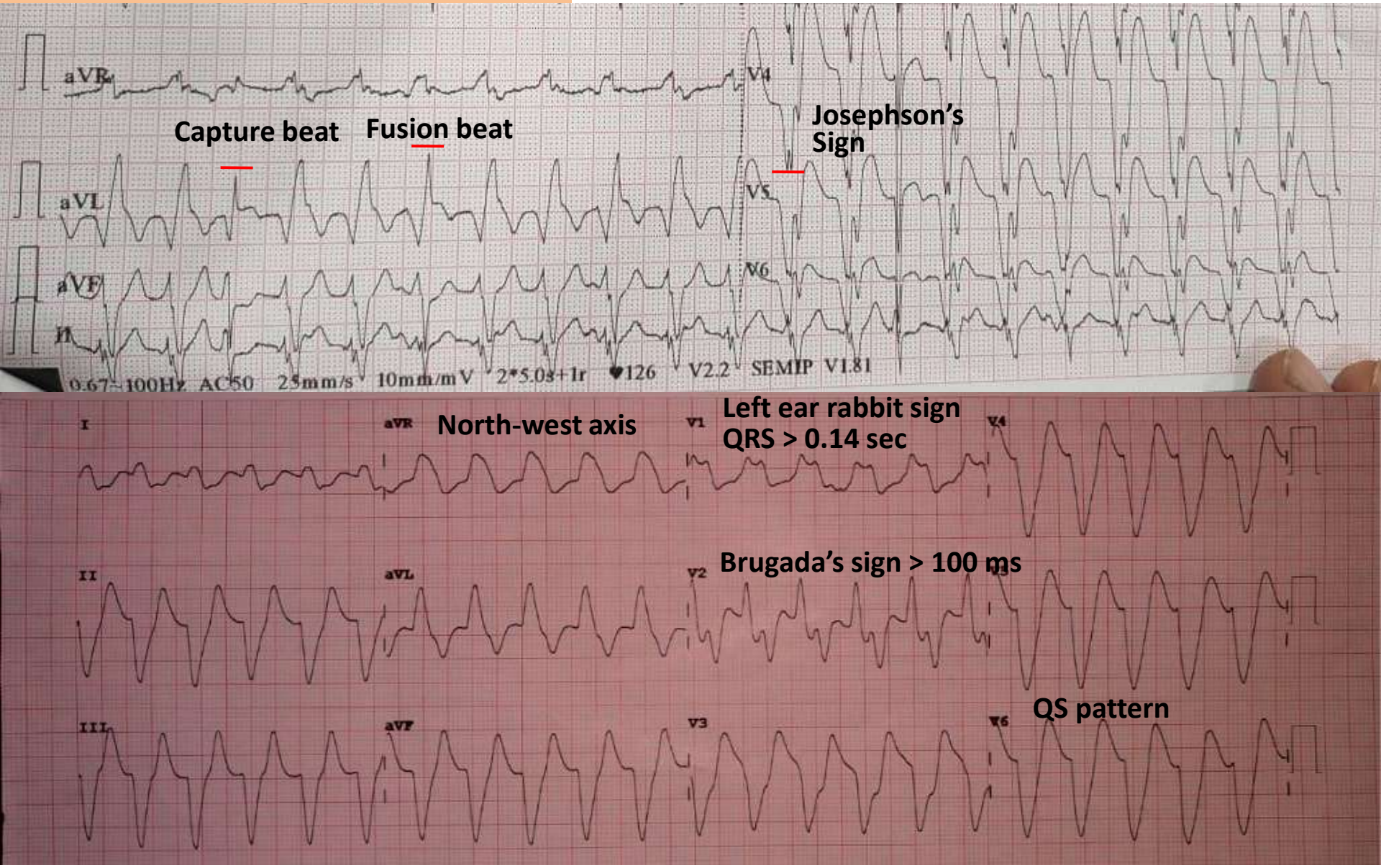
If in doubt , treat as VT

More likelihood of VT with the following consideration :

- **Age >35 (positive predictive value of 85%)**
- **Structural heart disease like coronary artery disease, congestive heart failure, cardiomyopathy etc.**
- **Family history of the sudden cardiac death.**

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

Miscellaneous slides

Mechanism of VT

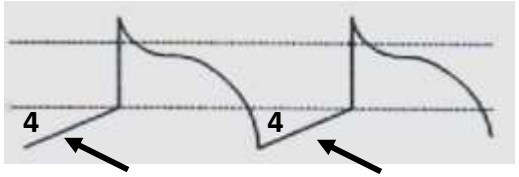
VT arises distal to the bifurcation of the HIS bundle in the specialized conduction system , ventricular muscle , or combination of both

1. **Reentry (most common)**
   Scar-related , Post-MI VT

2. **Triggered activity (Please see the next skide for the details)**

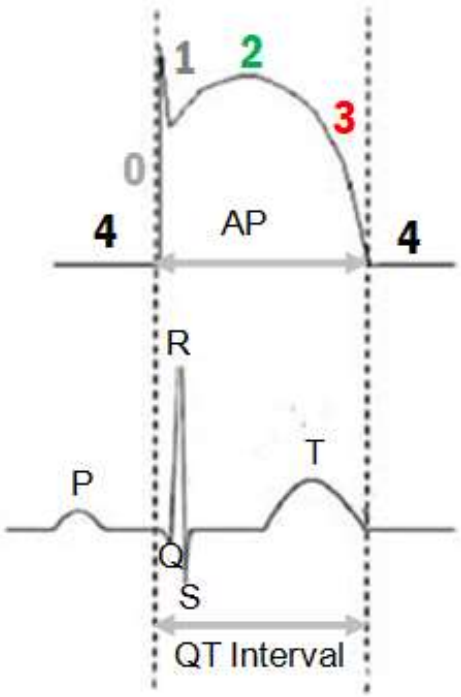
Early aferdepoalrization	Delayed afterdepolarization
• Occur during phase 2 or 3 of the action potential • More likely at slow heart rates and with prolonged QT • Leads to : torsades de pointes , polymorphic VT	• Occur during phase 4 (after full repolarization) • Linked to high intracellular calcium • Seen in : digitalis toxicity , catecholamine excess , ischemia (monomorphic VT)

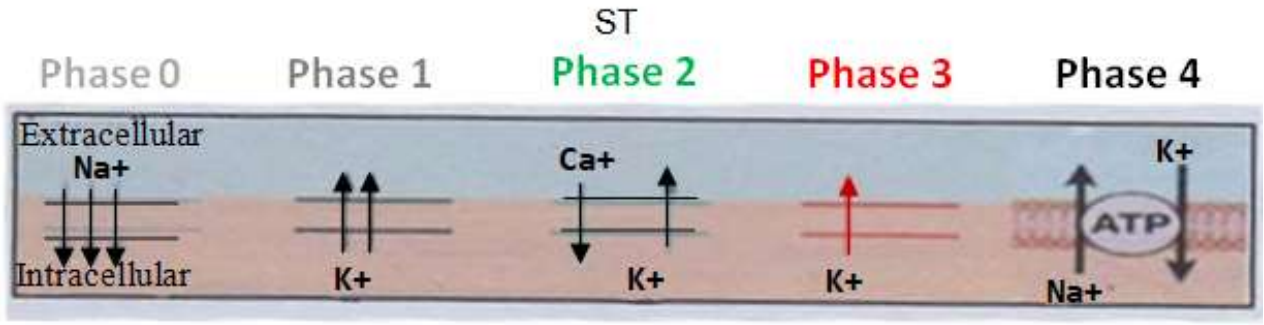
3. **Enhanced automaticity**

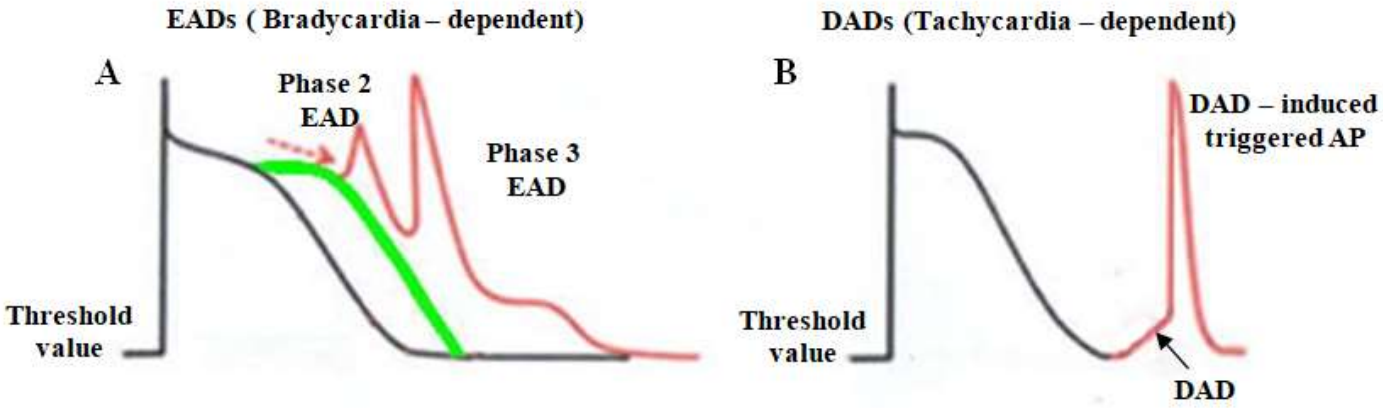


Ischemia , hypoxia , electrolyte abnormilities hypo-K/ Mg , Catecholamine assess , certain drugs and toxins

Afterdepolarization : EAD and DAD







Polymorphic VT

through K+ Current

Monomorphic VT

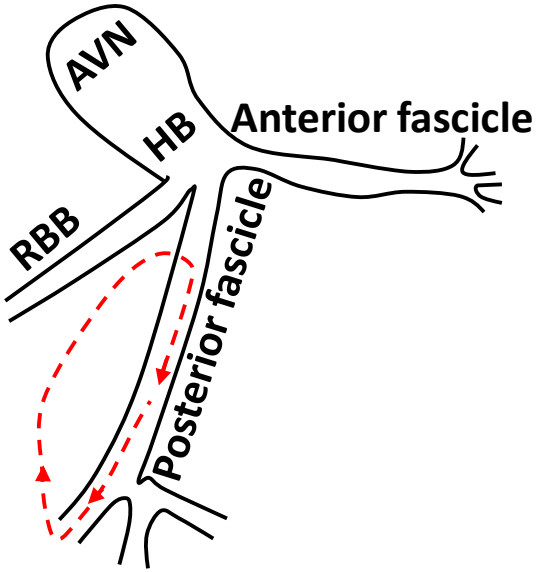
Ca++ release from sarcolemma →Na +-Ca++ exchange

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

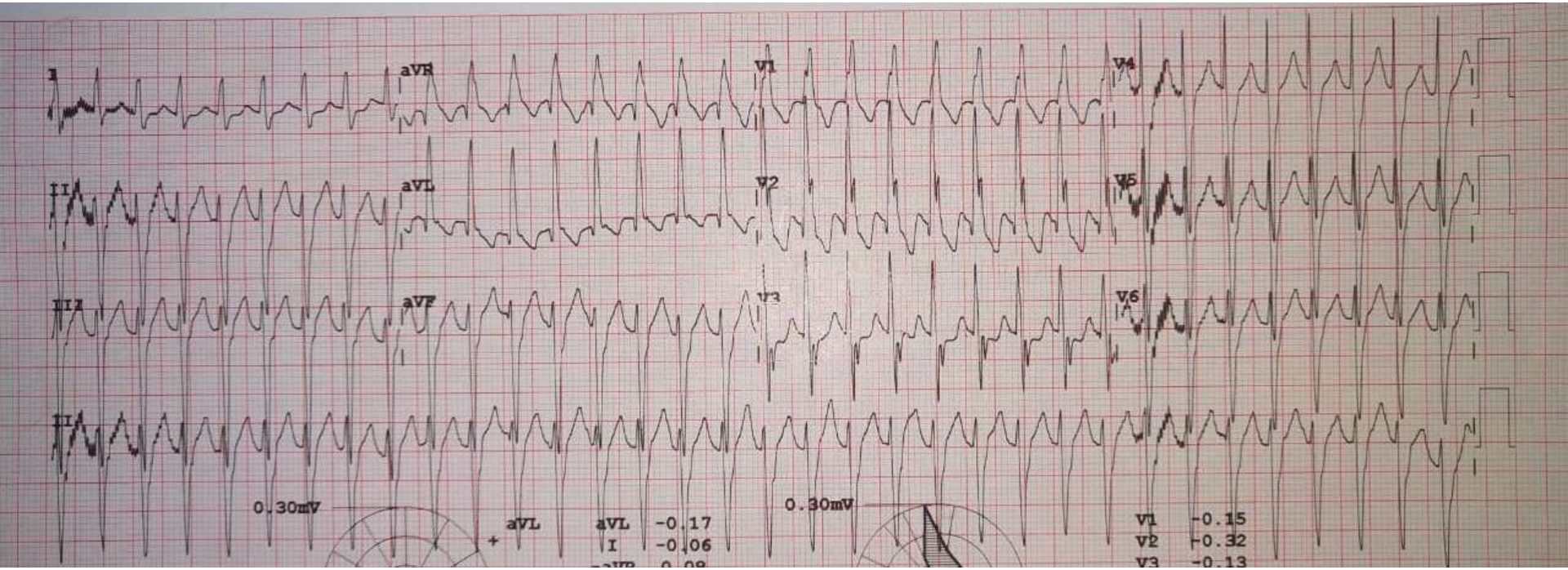
- 100–120 bpm → classic “slow VT” → large, slow circuits on diseased tissue (Diseased substrate limits speed → slower cycle) : Post-infarct scar VT, dilated cardiomyopathy, AIVR
- >150–200 bpm → typical fast monomorphic
- VT>200 bpm → Fast VT → small, fast circuits on normal tissue : Triggered by catecholamines (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.

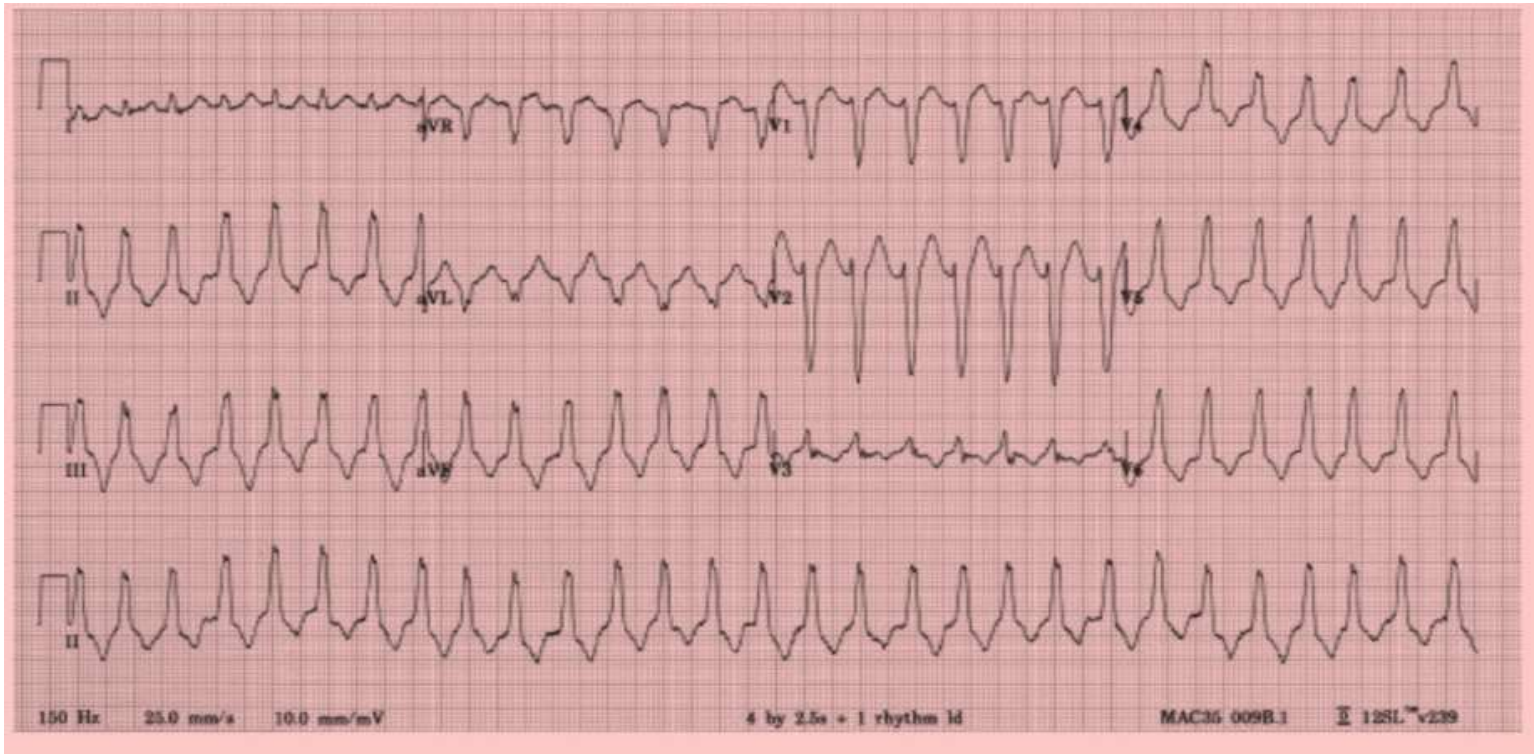


❑ RVOT-VT (Idiopathic , related to triggered activity – delayed afterdepolarizations , sensitive to catecholamines)

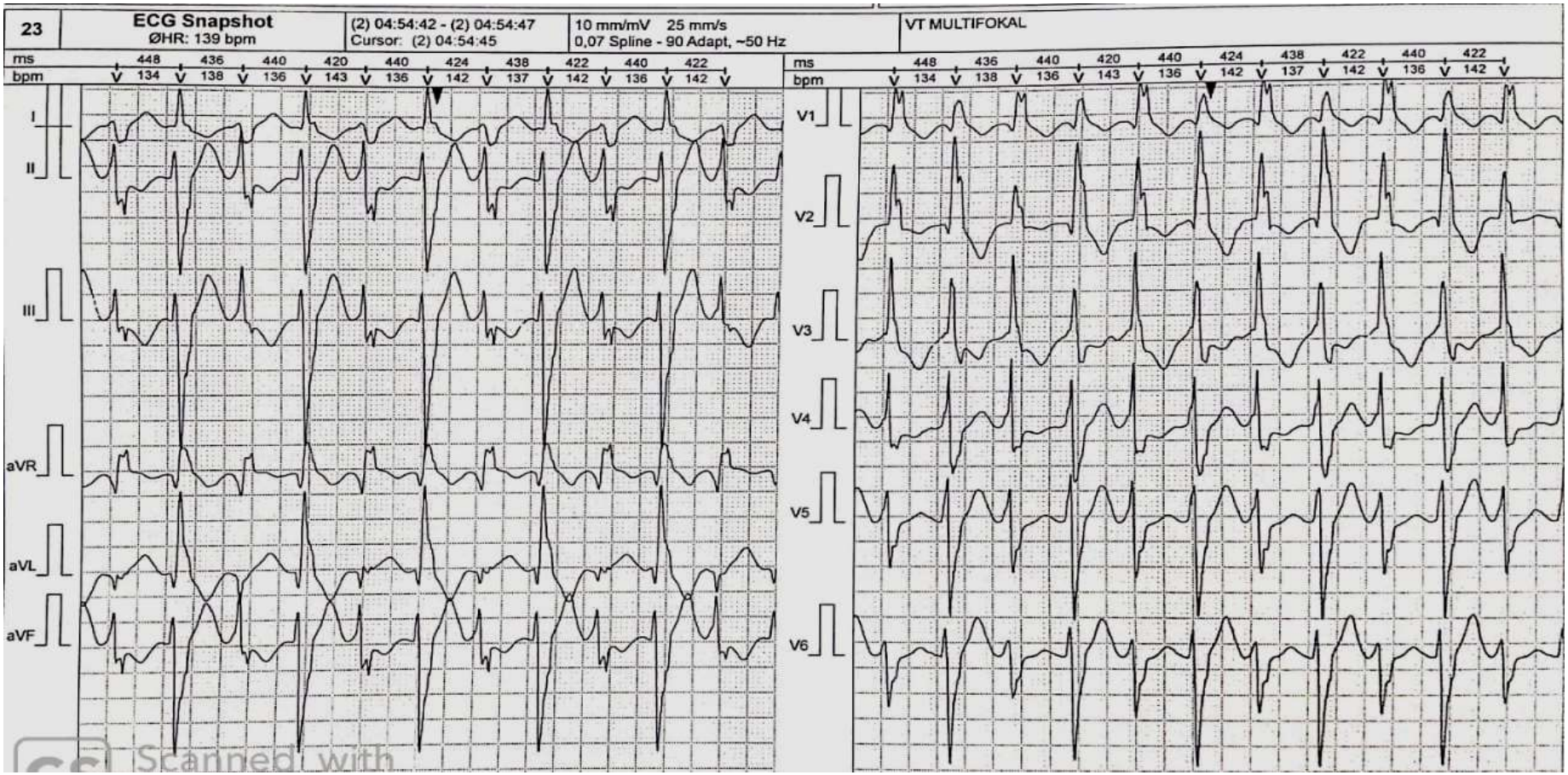
Arising from right ventricular outflow tract which is an infundibular extension of the ventricular cavity , connecting it to the pulmonary artery

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**

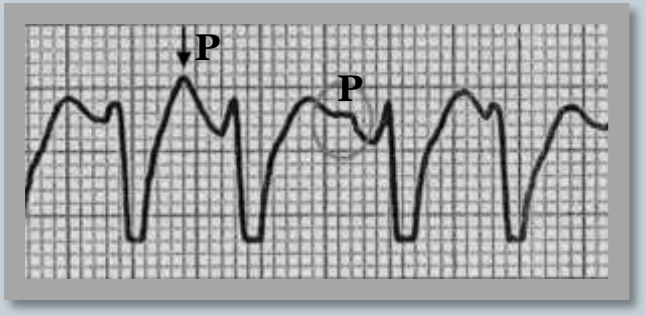


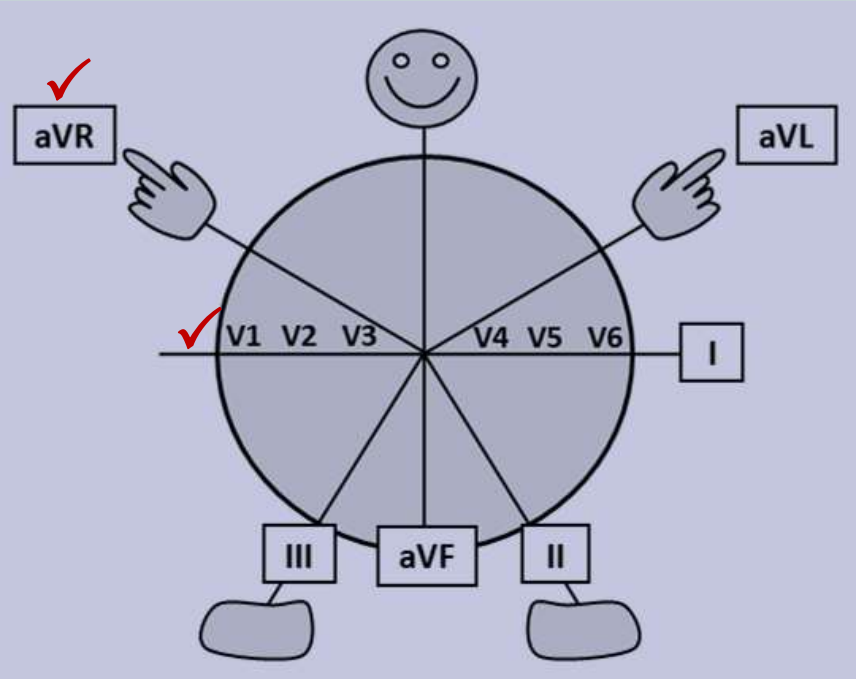
ECG showing CPVT



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