Differential Diagnosis and Treatment of Wide Complex Tachycardia

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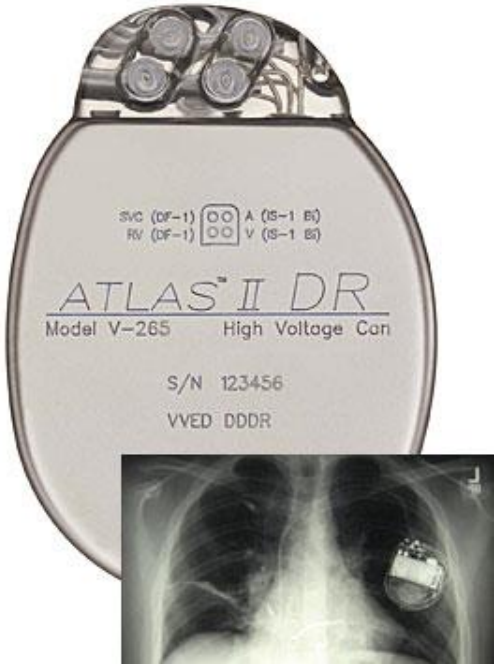
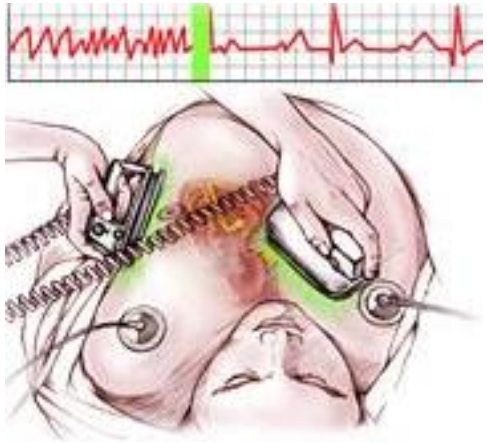
When the diagnosis of a WCT is uncertain

the patient be treated as if the rhythm is VT

Treatment of WCT





Definitions

- Wide Complex Tachycardia(WCT)-a rhythm with QRS duration ≥ 120 ms and heart rate > 100 beats/min
- Ventricular tachycardia - WCT originating below the level of His bundle
- SVT - tachycardia dependent on participation of structures at or above the level of His bundle

General Approaches to WCT

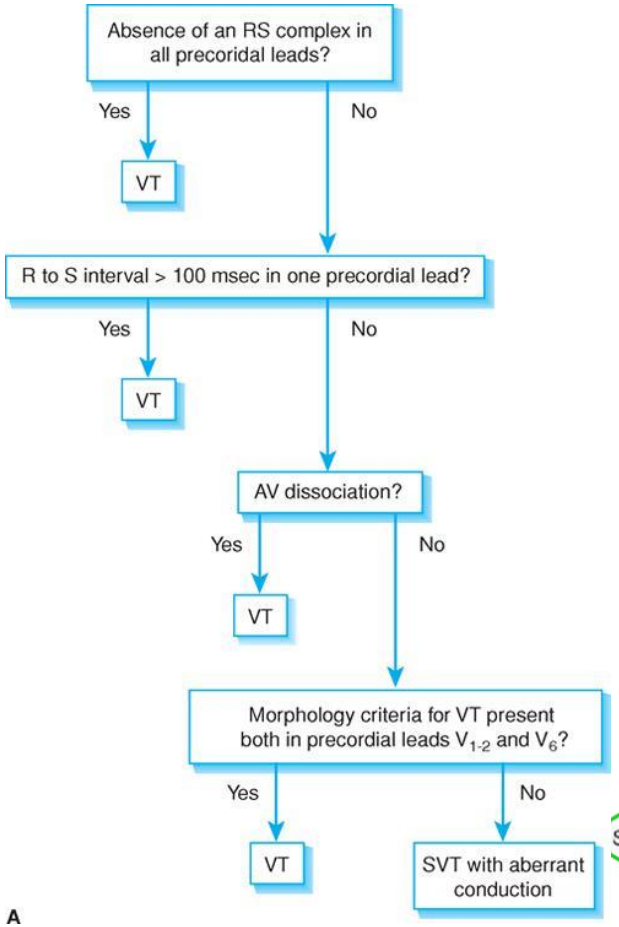
- **Clinical Characteristics** of the patient
  - Absence of structural heart disease makes SVT more likely.
    : But idiopathic VT can be seen!
  - History of structural heart disease makes VT more likely

- Classic RBBB or LBBB morphology argues STRONGLY for SVT with aberrancy

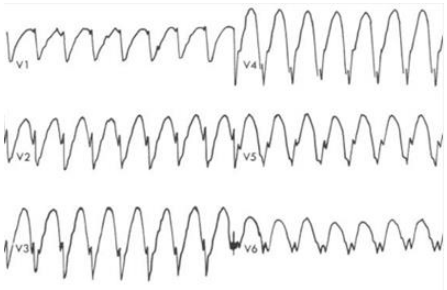
- Features suggestive of VT:
  - **QRS Morphology** not consistent with classic BBB
  - VA dissociation
  - Capture and fusion complexes

- **Brugada Algorithms**

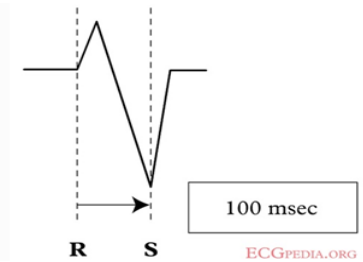
Brugada algorithm



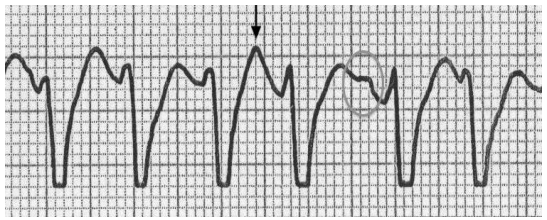
A



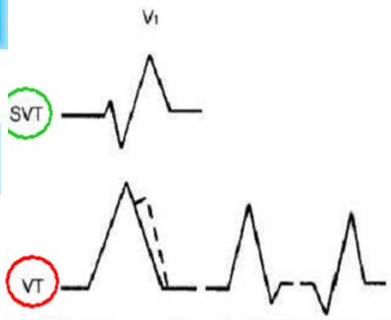
Absence of RS complex in all precordial leads



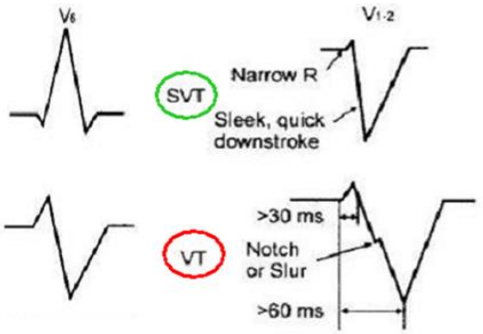
R to S interval>100ms in one precordial lead

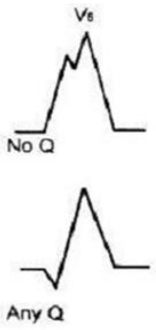


AV dissociation



RBBB



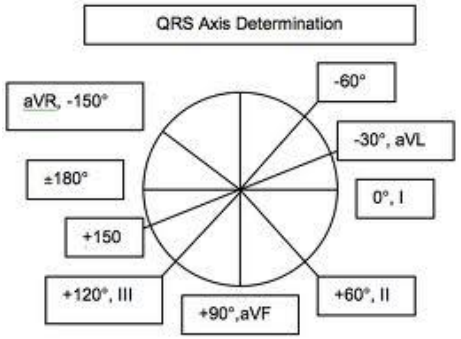


LBBB

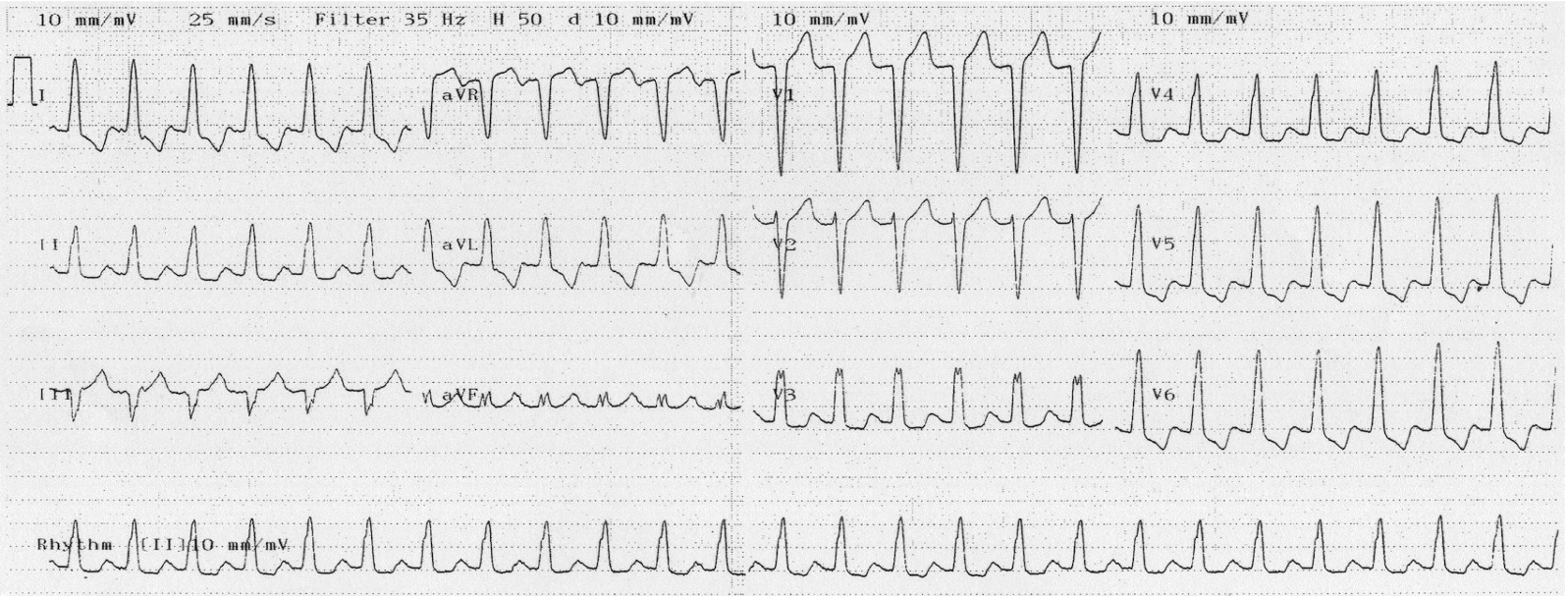
Morphology criteria

ECG distinction of VT from SVT with aberrancy

	Favors VT	Favors SVT with aberrancy
Duration	RBBB : QRS > 0.14 sec LBBB : QRS > 0.16 sec	< 0.14 sec < 0.16 sec
Axis	QRS axis -90°to± 180°	Normal



SVT with aberrancy

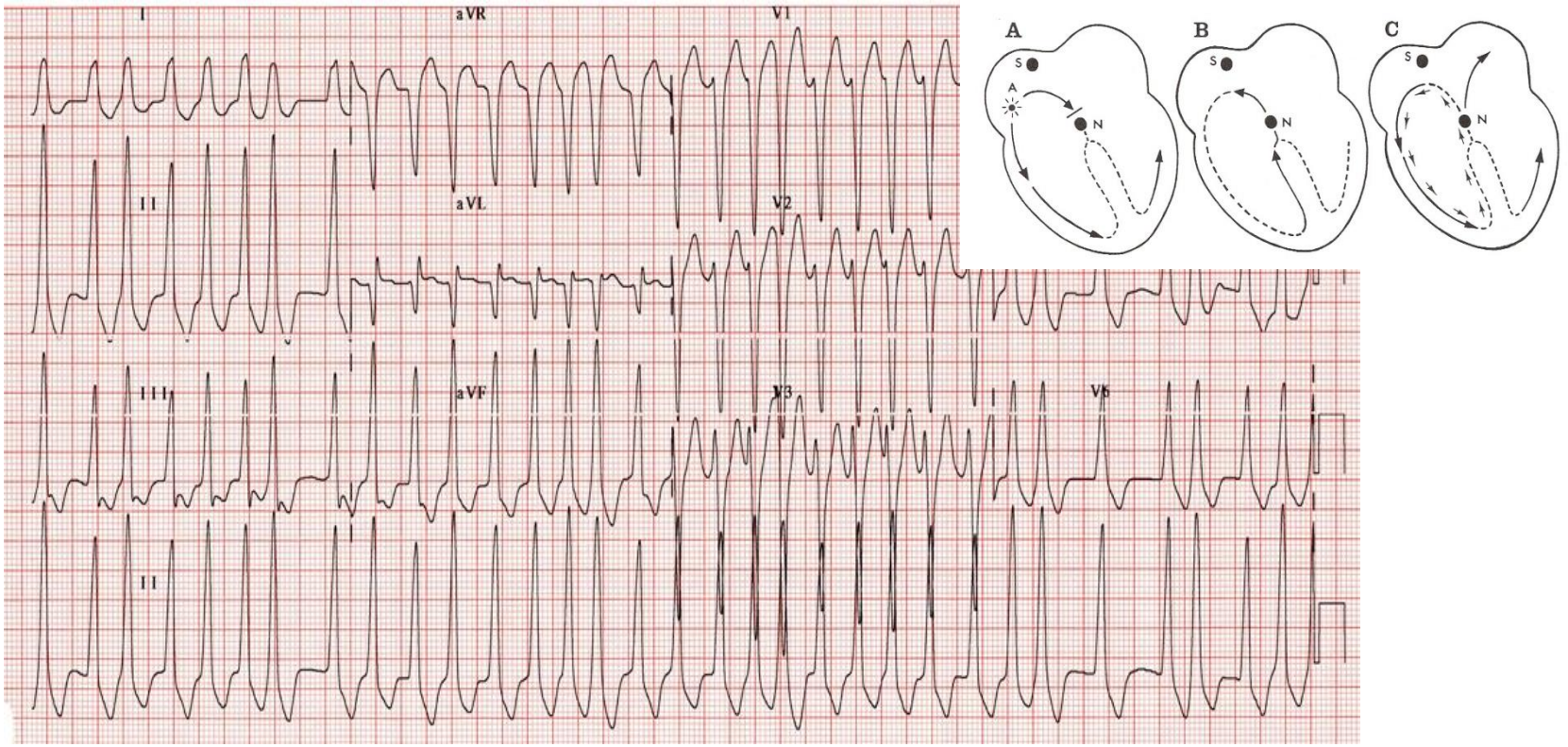


- Conduction to the ventricles via the His-Purkinje system, but with an abnormality
  - Right Bundle Branch Block (RBBB)
  - Left Bundle Branch Block (LBBB)
  - Intraventricular Conduction Delay (IVCD)
- These can be
  - Pre-existing BBB (helpful clue)
  - SVT-associated

- Any SVT can be conducted with aberrancy:
  - Sinus Tachycardia
  - Atrial tachycardia
  - Atrial flutter
  - Atrioventricular nodal reentrant tachycardia
  - Junctional Tachycardia
  - Atrioventricular Reentrant Tachycardia

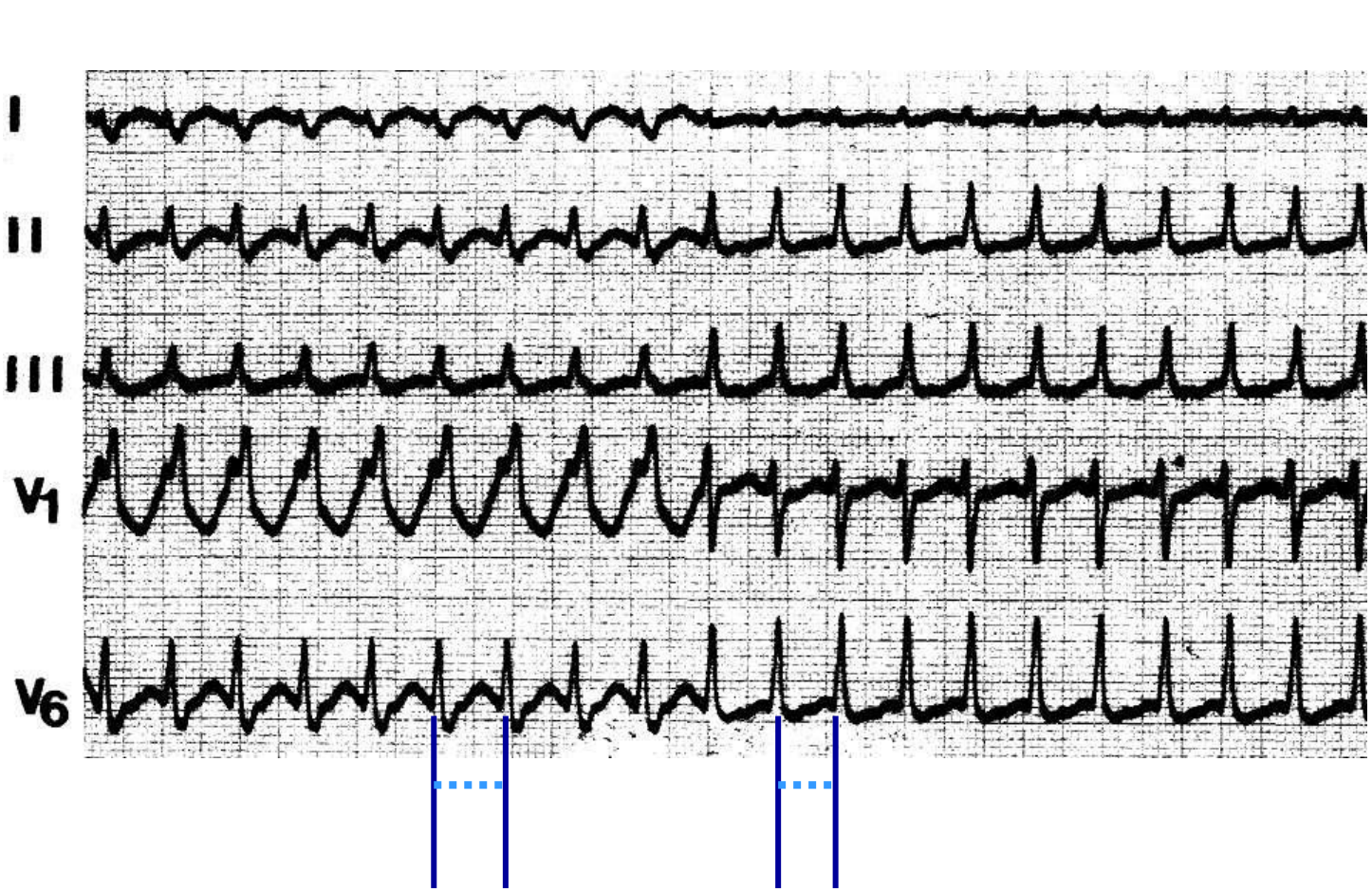
WPW syndrome

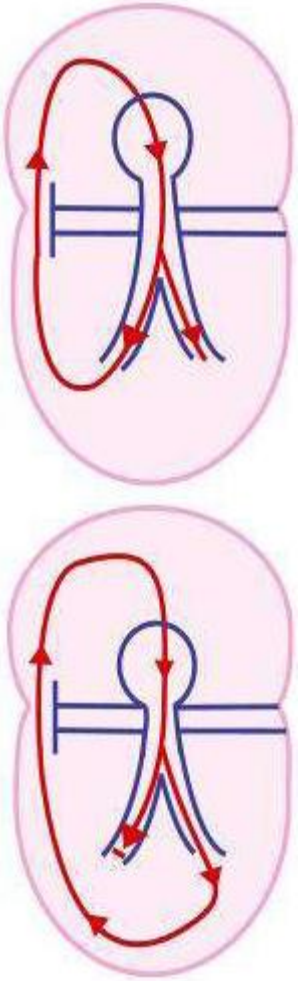
(Antegrade conduction via accessory)



- Any SVT with antegrade conduction down an accessory pathway (WPW syndrome) will produce a wide QRS.
  - Slow myocyte-to-myocyte conduction arising from the ventricular insertion of the pathway
  - QRS morphology during tachycardia will look a lot like VT!

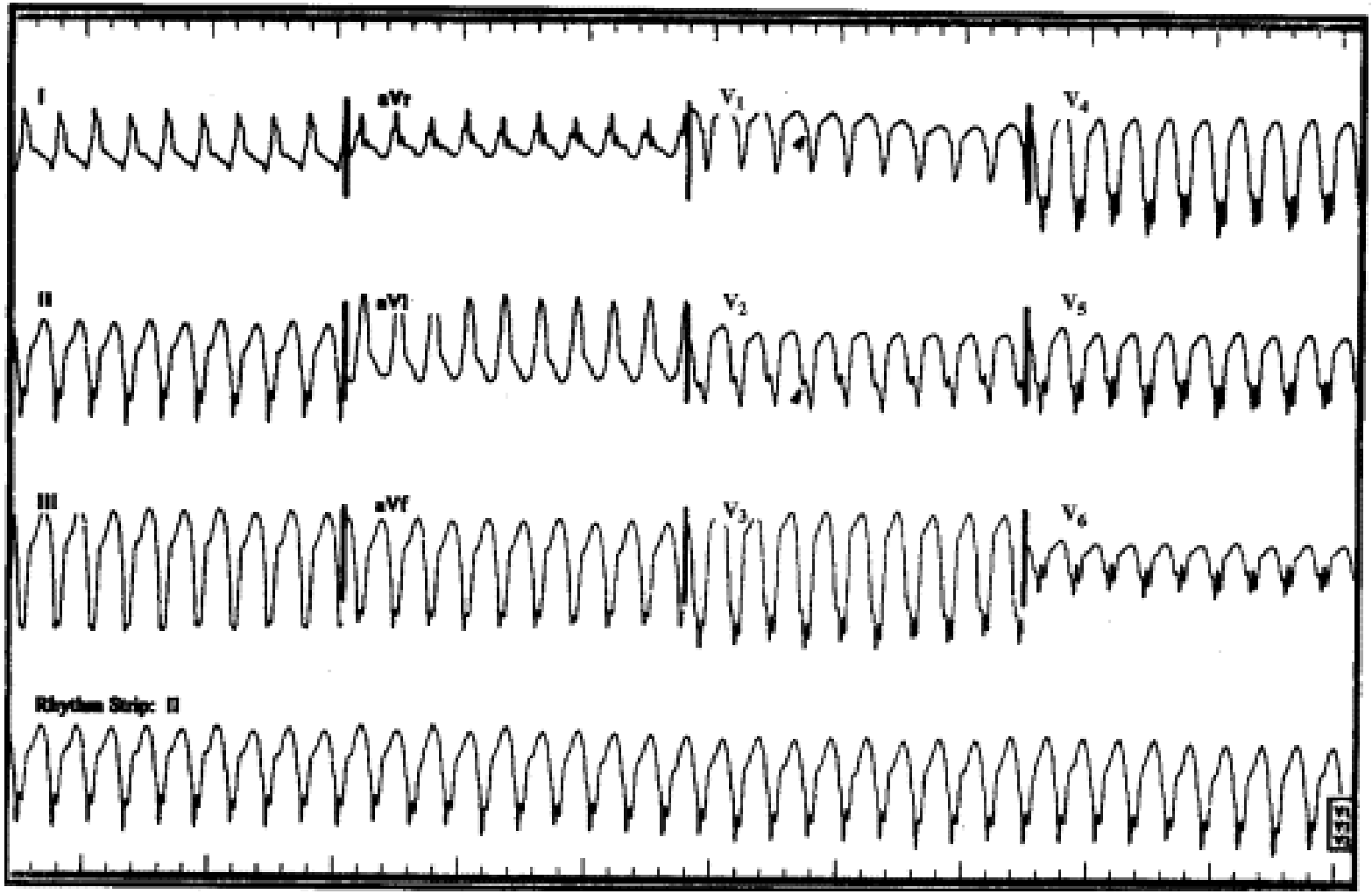
SVT vs VT

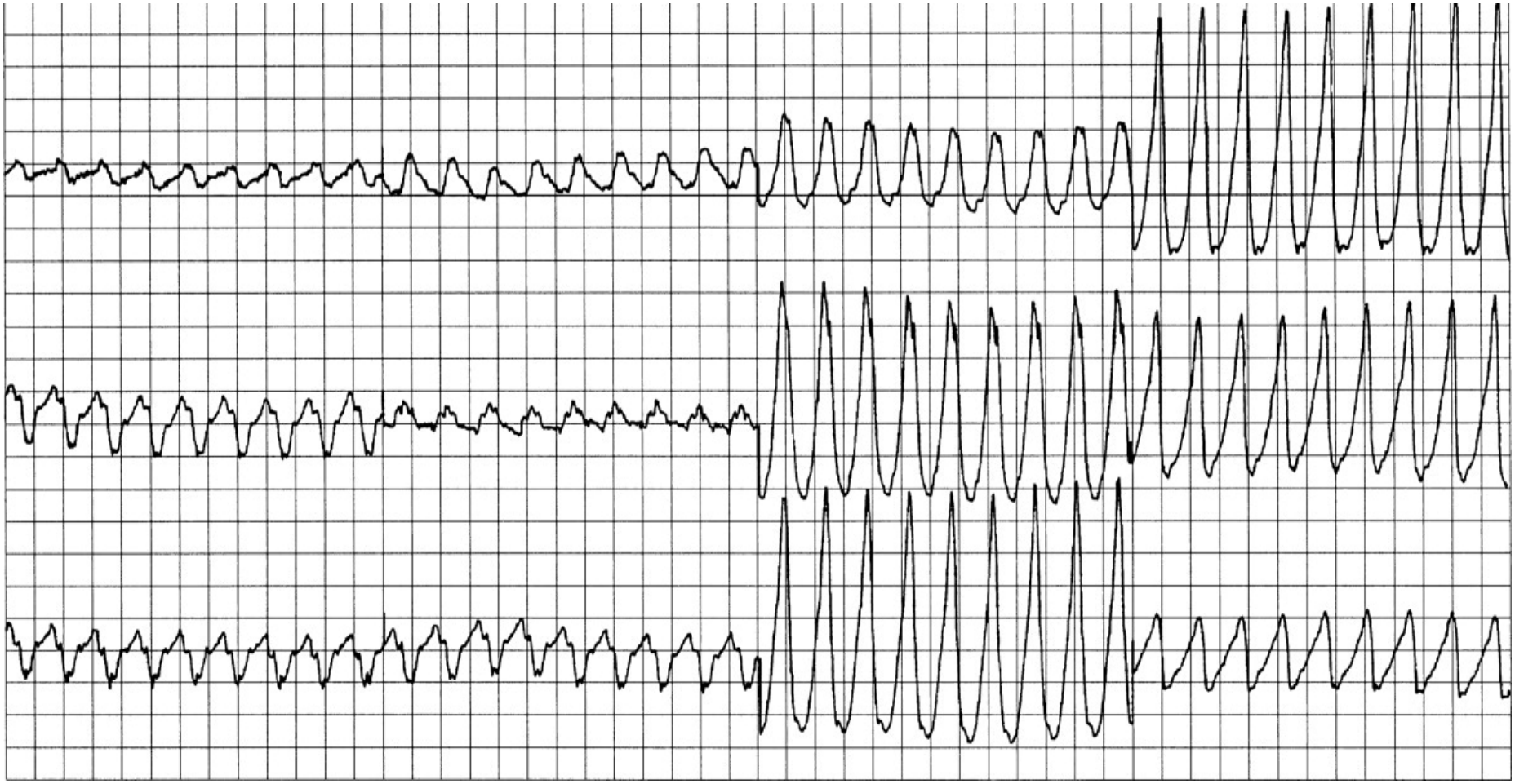




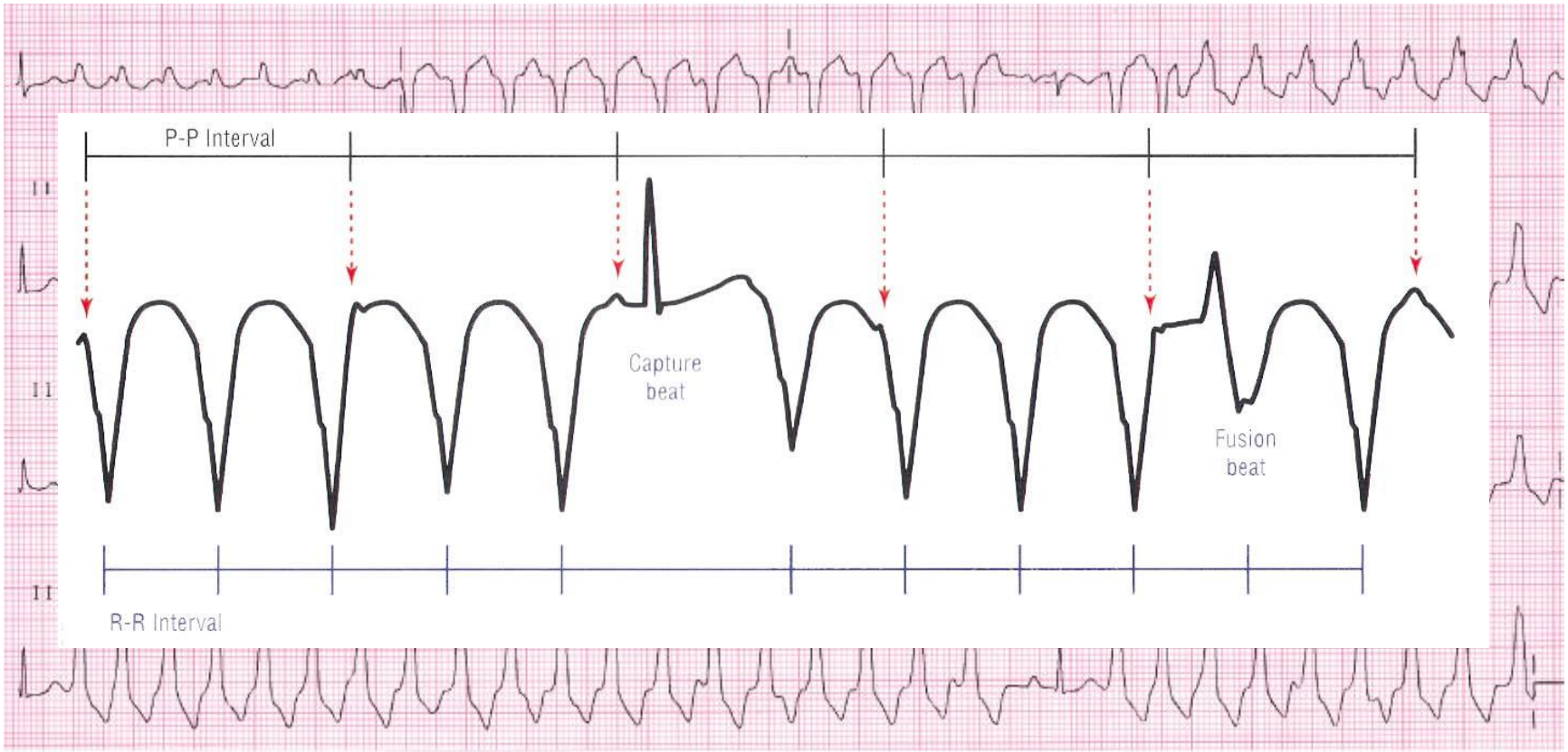
Coumel's law

Absence of RS complex in all precordial leads

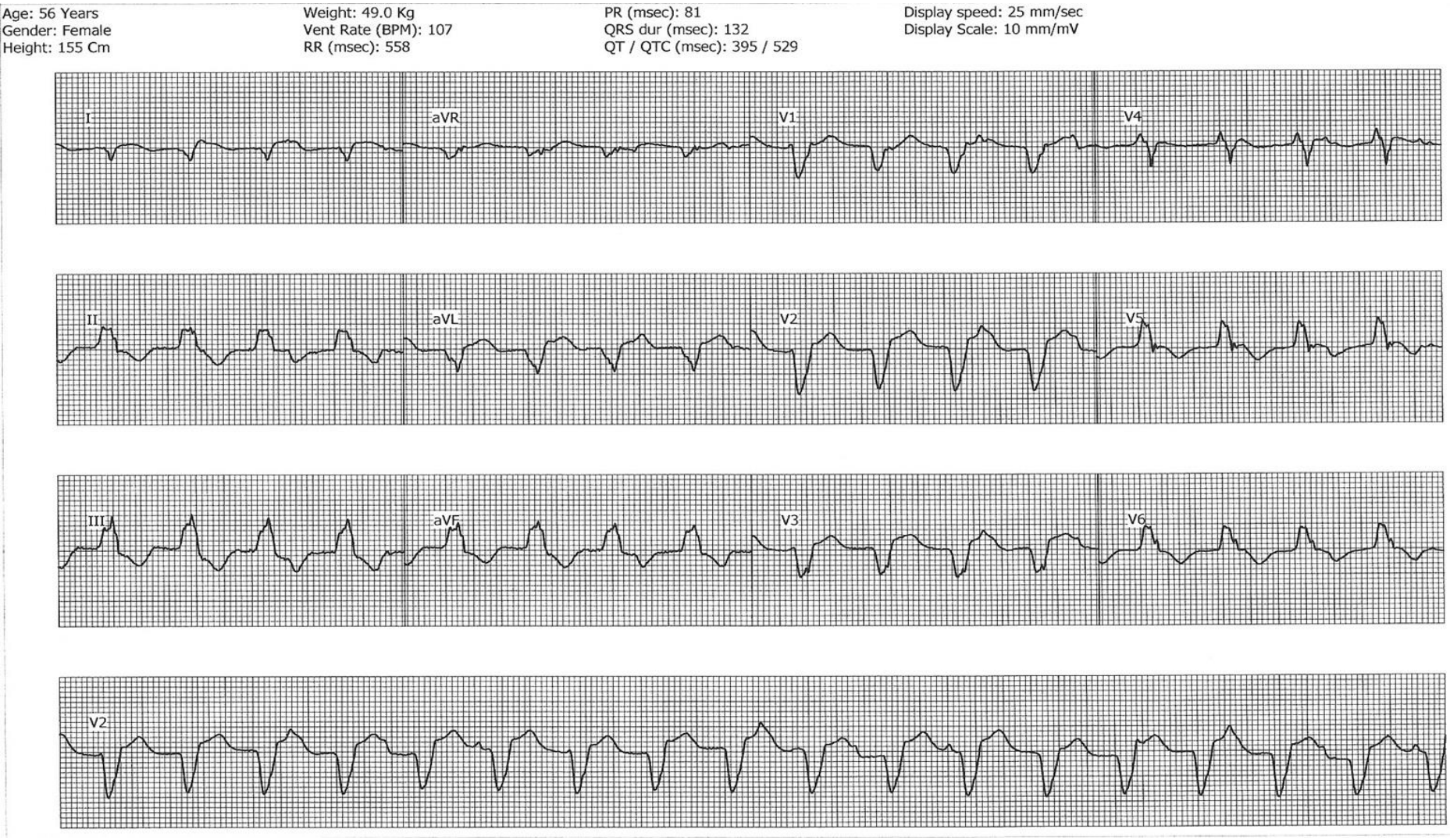




Capture or Fusion beat



AV dissociation

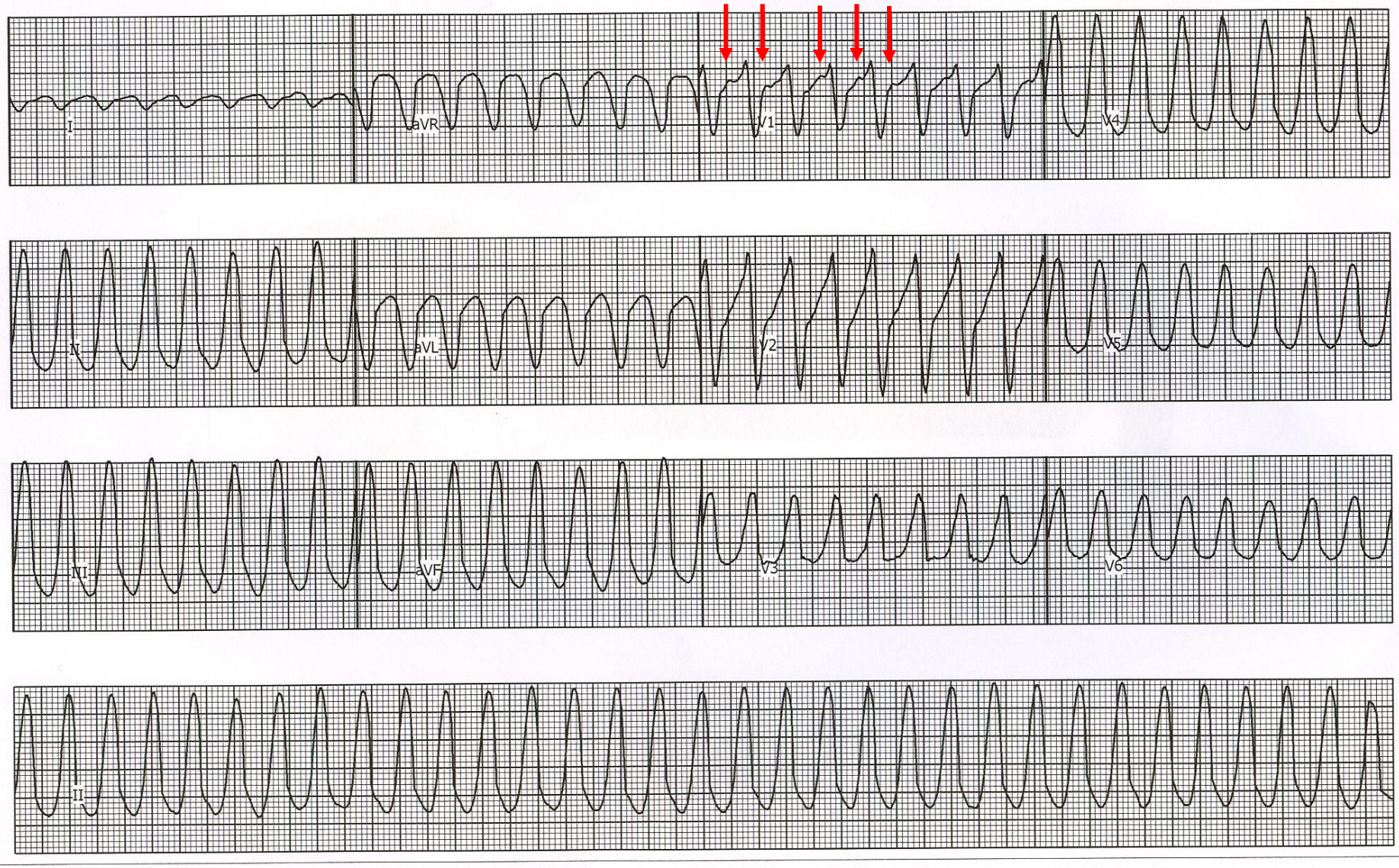


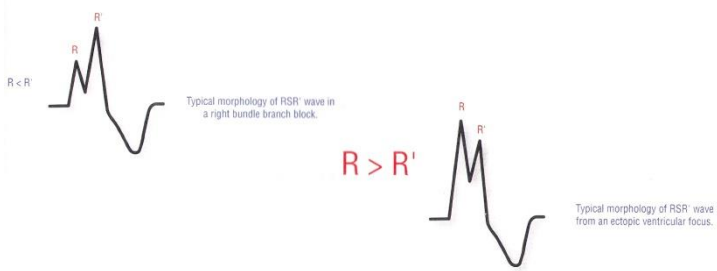
Age: 57 Years
Gender: Male
Height: 170 Cm

Weight: 76.0 Kg
Vent Rate (BPM): 196
RR (msec): 305

PR (msec): 111
QRS dur (msec): 145
QT / QTC (msec): 308 / 558

Display speed: 25 mm/sec
Display Scale: 10 mm/mV





I aVR V1 V4

II aVL V2 V5

III aVF V3 V6

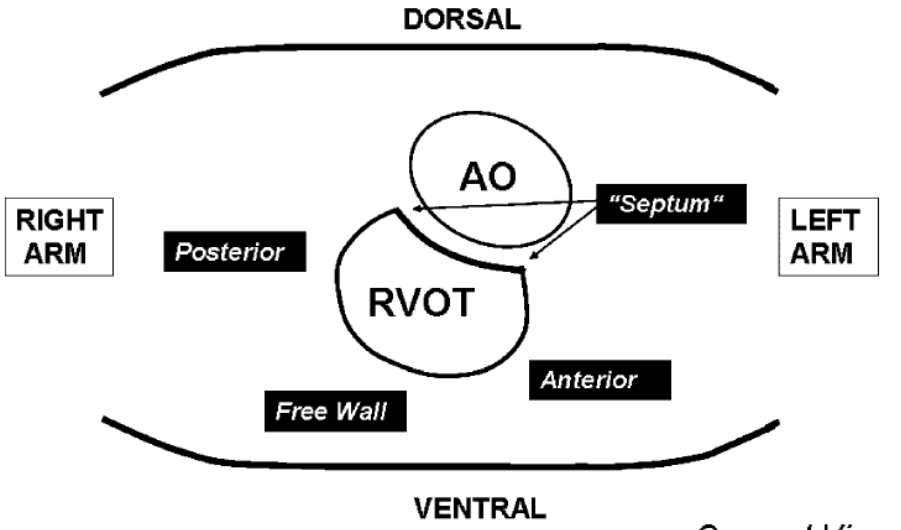
II

VT

Idiopathic monomorphic VT

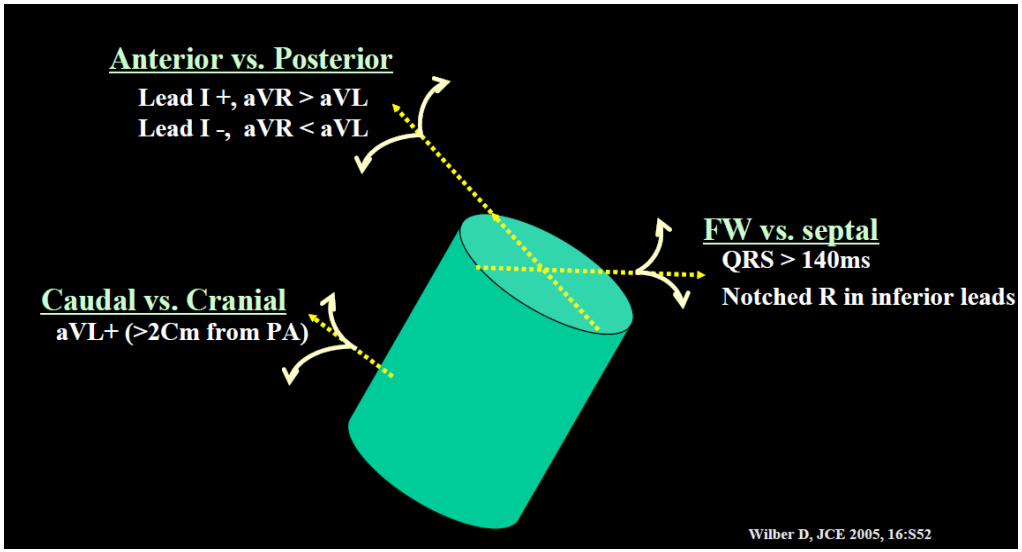
	Adenosine-sensitive	Verapamil-sensitive	Propranolol-sensitive
Mech	*(Triggered activity)* 1) Exercise-induced 2) Repetitive monomorphic	*(Fascicular reentry)* Fascicular	*(Automaticity)* 1) Exercise-induced 2) Incessant
Induction	PES c/s cathecholamine	PES c/s cathecholamine	Cathecholamine
ECG	LBBB with inferior axis RBBB with inferior axis	RBBB with superior axis RBBB with rt inferior axis	RBBB, LBBB, Polymorphic
Origin	RVOT/LVOT (1)	Lt posterior fascicle Lt anterior fascicle (2)	RV/LV

RVOT VT



J Cardiovasc Electrophysiol, Vol. 16, pp. S52-S58, Suppl. 1, September 2005

- No evidence of underlying structural heart disease
- Patients with symptoms not readily treated with medications are candidates for ablation.
- An ECG showing PVCs or VT can suggest the likely region of origin of the arrhythmia to assist in mapping.
- Mapping based on earliest activation



	Sensitivity	Specificity	PPV
Free wall versus septal sites			
QRS duration ≥140 msec	0.74	0.93	0.88
R-wave notching in inferior leads	0.79	0.99	0.94
Lead V3 R/S ratio ≤1	1.00	0.74	0.73
Anterior (leftward) versus Posterior (rightward) sites			
Negative or isoelectric QRS in lead I	0.96	0.67	0.77
Caudal (>2 cm from PV) versus Cranial Sites			
Isoelectric or positive QRS in lead avL	0.86	1.00	1.00

PPV = positive predictive value; PV = pulmonary valve; RVOT = right ventricular outflow tract.

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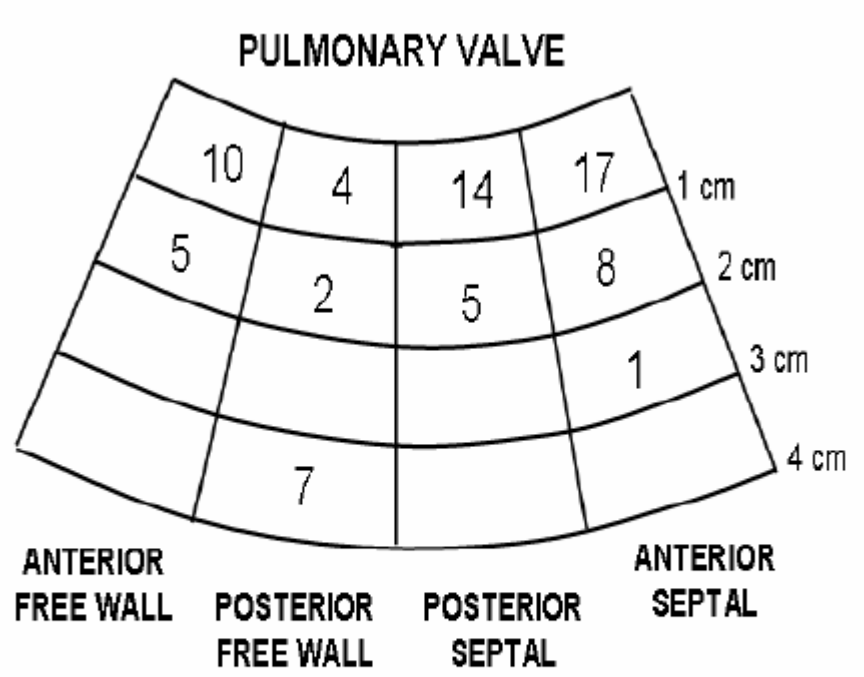
Localization

(QRS: Septal vs Free wall)



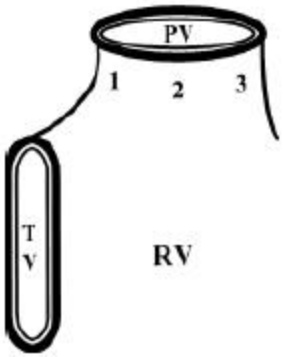
- QRS duration ≥ 140 msec
- QRS notching in inferior leads
- Lead V3 R/S ratio ≤ 1

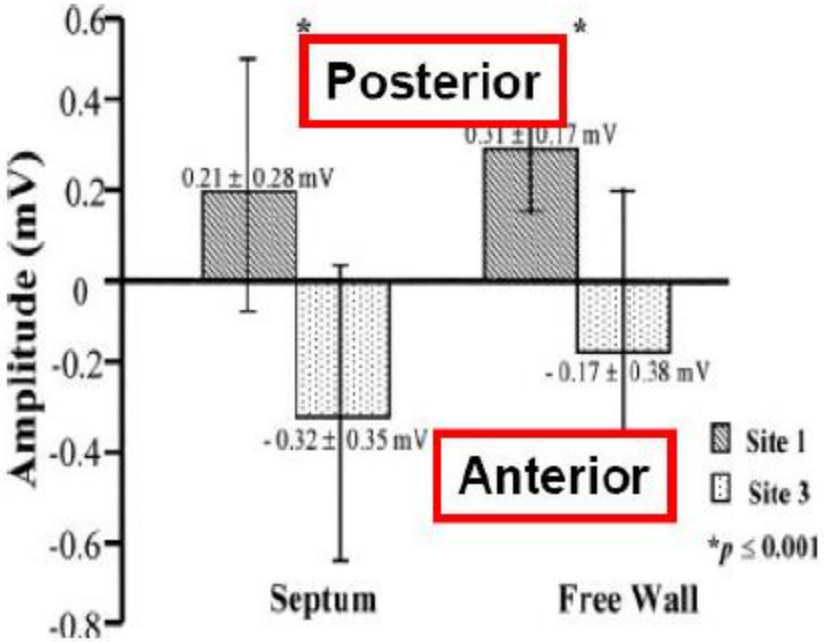
•Majority arise 1-2 cm below the PV
•20-30 % are free wall

Dixit et al, JCE 2003;14:1
Joshi et al, JCE 2005;16suppl:S52

Localization

(Lead I : Anterior vs Posterior)

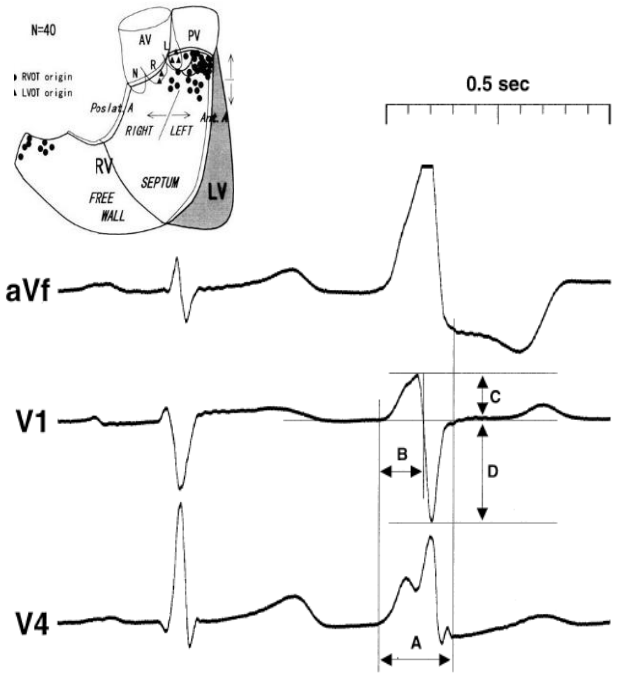




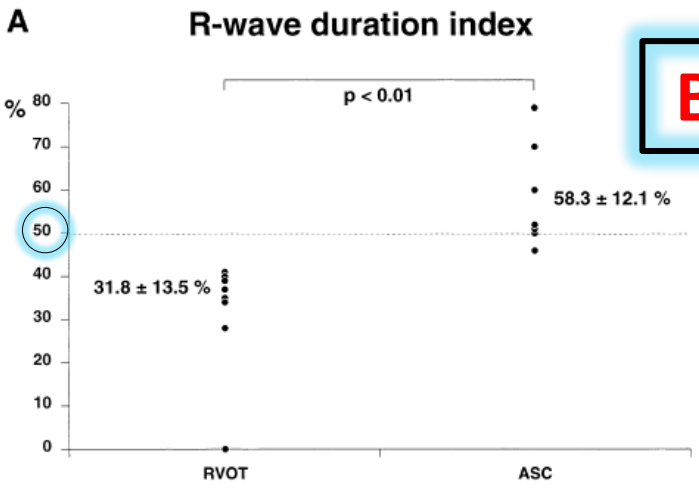
(J Cardiovasc Electrophysiol, Vol. 14, pp. 1-7, January 2003

Monomorphic ventricular tachycardia with LBBB morphology and an inferior axis

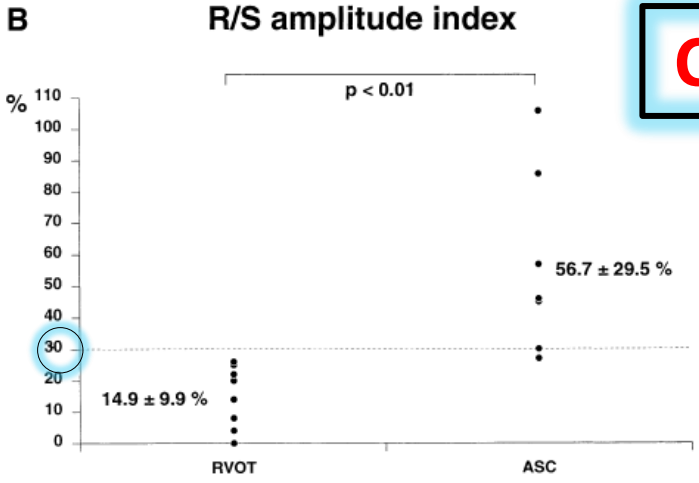
RVOT vs Aortic Sinus Cusp origin



A Total QRS duration(ms)
B R-wave duration (ms)
C R-wave amplitude (mV)
D S-wave amplitude (mV)



B/A



C/D

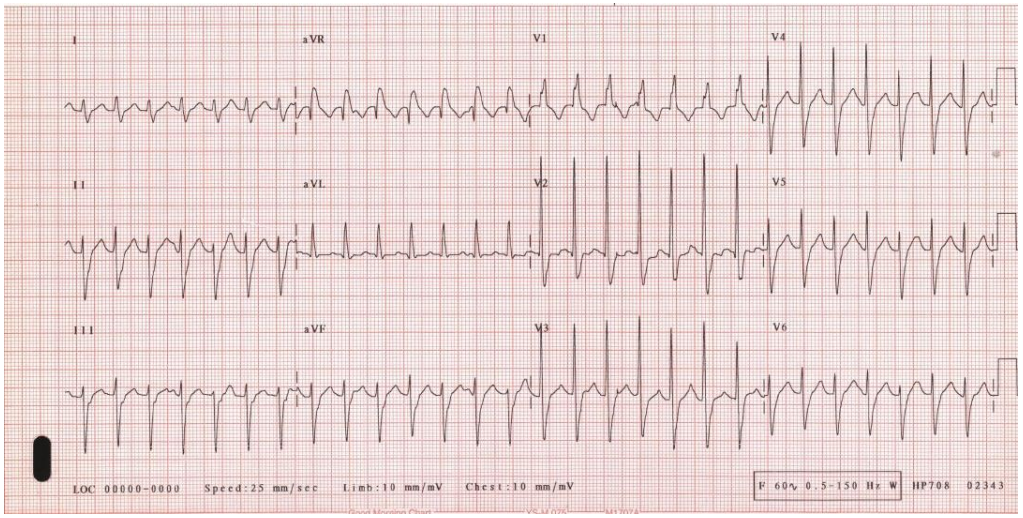
1) total QRS duration
2) R-wave duration in leads V1 and V2
3) **R-wave duration index**, calculated as a percentage by dividing the QRS complex duration by the longer R-wave duration in lead V1 or V2
4) R/S-wave amplitude ratio in leads V1 and V2, measured from the QRS complex peak or nadir to the isoelectric line, expressed as a percentage
5) **R/S-wave amplitude index**, calculated from the greater percentage of the R/S-wave amplitude ratio in lead V1 or V2.

J Am Coll Cardiol 2002;39:500–8)

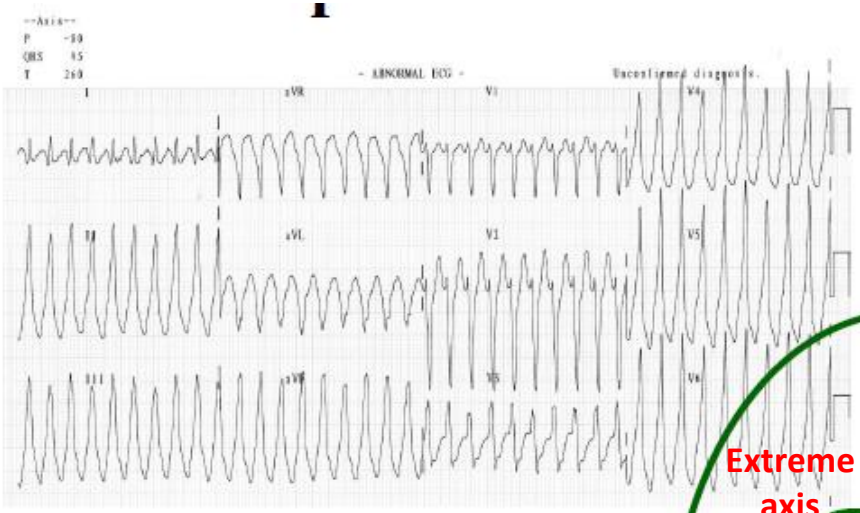
Fascicular VT

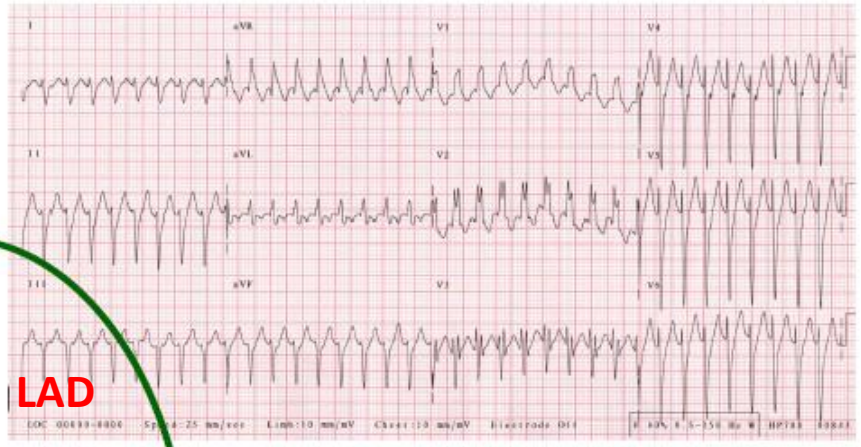
Idiopathic left ventricular tachycardia
- structurally normal heart
- Right bundle branch block+Left axis deviation
- verapamil-sensitive
- good longterm prognosis

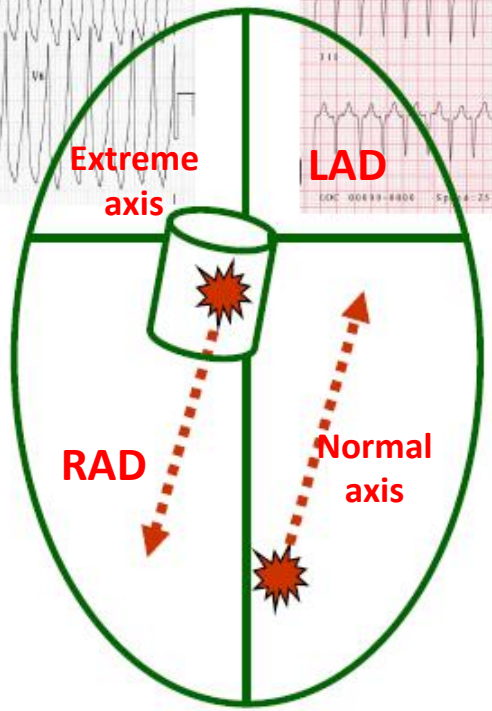
Mechanism of ILVT
- Triggered activity
- microreentry
- Purkinje reentry



Anatomic extent of the reentry circuit in ILVT has not been defined.







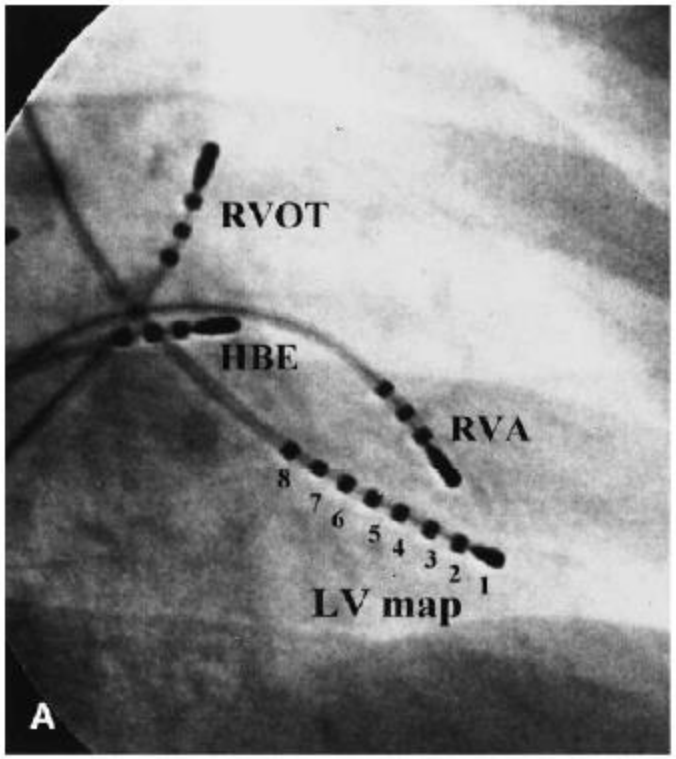
LBBB

RAD

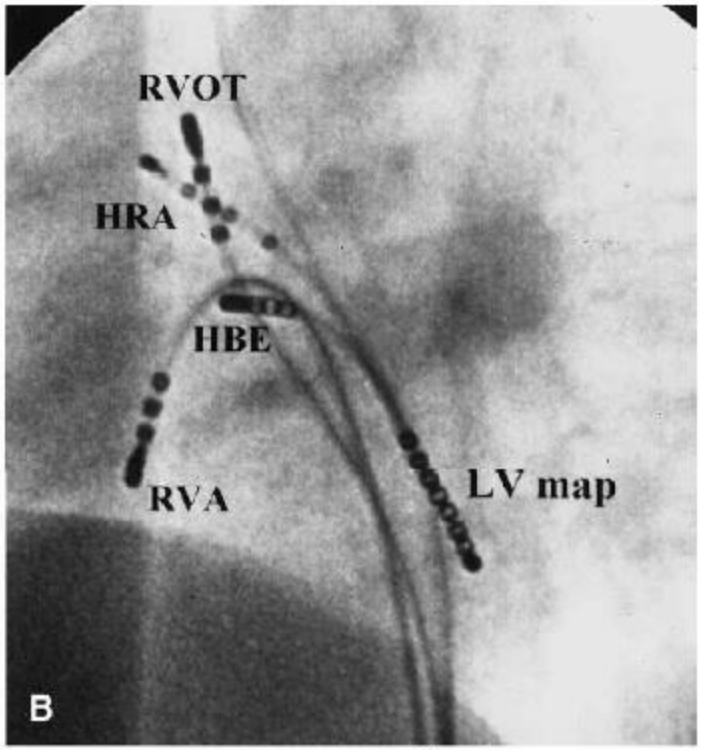
RBBB

LAD

(Sup axis deviation)



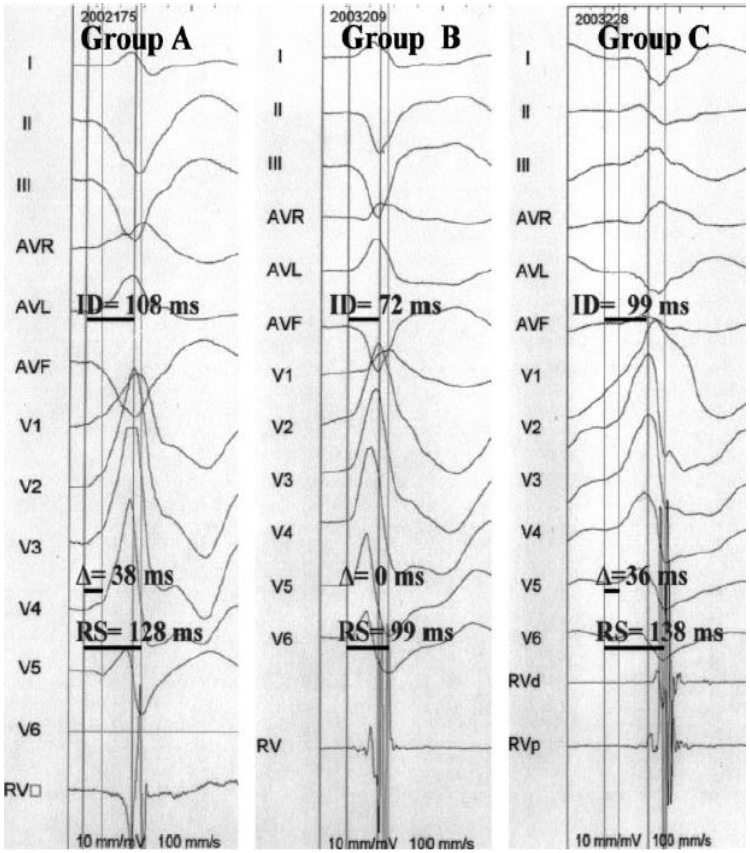
RAO 30°



LAO 60°

PACE 2001; 24:333–344

Epicardial VT



A. pseudodelta wave ≥ 34 ms

B. The intrinsicoid deflection time ≥ 85 ms

C. RS complex duration ≥ 121 ms

Pseudodelta Wave
from the earliest ventricular activation (from the stimulation artifact in paced patients) to **the earliest fast deflection in any precordial lead**

Intrinsicoid Deflection Time
from the earliest ventricular activation (from the stimulation artifact in paced patients) to **the peak of the R wave in V2**

Shortest RS Complex
From the earliest ventricular activation (from the stimulation artifact in paced patients) **to the nadir of the first S wave in any precordial lead**

Circulation. 2004;109:1842-1847

Idiopathic Epicardial Left Ventricular Tachycardia Originating Remote From the Sinus of Valsalva

Electrophysiological Characteristics, Catheter Ablation, and Identification From the 12-Lead Electrocardiogram

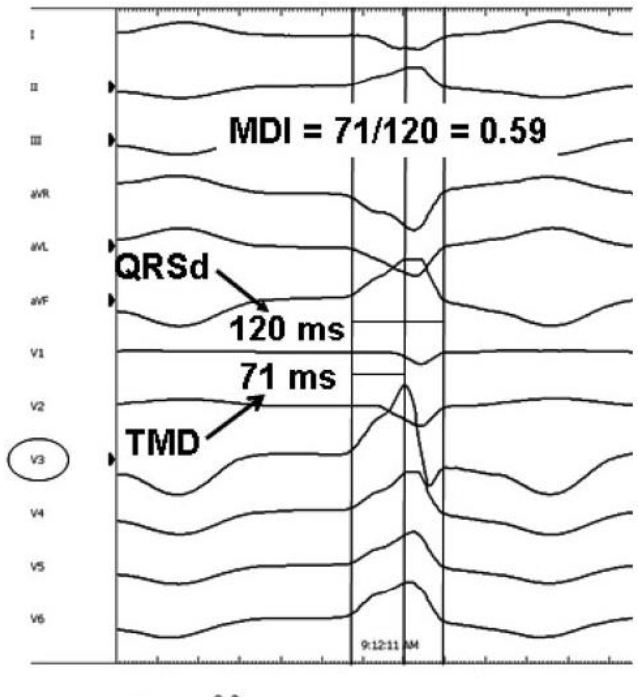
David V. Daniels, MD; Yen-Yu Lu, MD; Joseph B. Morton, MD; Peter A. Santucci, MD; Joseph G. Akar, MD, PhD; Alex Green, MD; David J. Wilber, MD

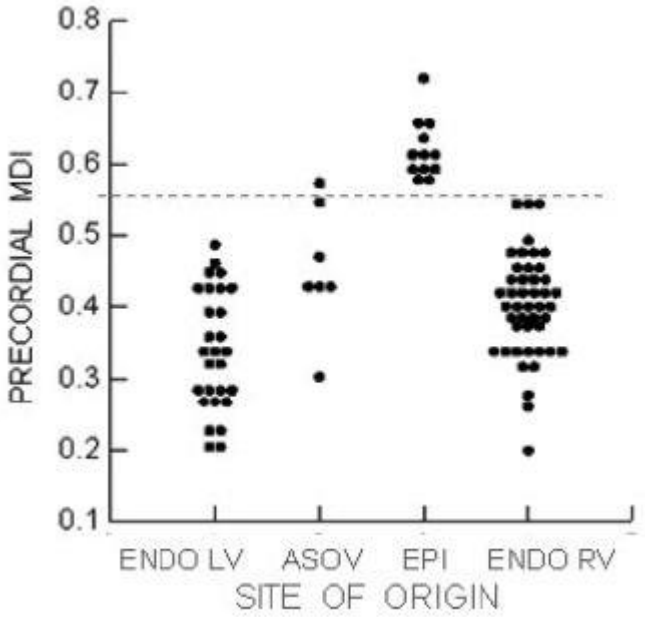
Background—Despite the success of catheter ablation for treatment of idiopathic ventricular tachycardia (VT), occasional patients have been reported in whom VT could not be ablated from the right or left ventricular endocardium or from the aortic sinus of Valsalva (ASOV).

Methods and Results—In 12 of 138 patients (9%) with idiopathic VT referred for ablation, an epicardial left ventricular site of origin was identified >10 mm from the ASOV. Coronary venous mapping demonstrated epicardial preceding endocardial activation by >10 ms (41±7 versus 15±11 ms before QRS onset; $P<0.001$). VT induction was facilitated by catecholamines and terminated by adenosine. Ablation through the coronary veins or via percutaneous transpericardial catheterization was successful in 9 patients; 2 required direct surgical ablation as a result of anatomic constraints. No ECG pattern was specific for epicardial VT. However, slowed initial precordial QRS activation, as quantified by a novel metric, the maximum deflection index, was more useful. A delayed precordial maximum deflection index ≥0.55 identified epicardial VT remote from the ASOV with a sensitivity of 100% and a specificity of 98.7% relative to all other sites of origin ($P<0.001$).

Conclusions—Although clinically underrecognized, idiopathic VT may originate from the perivascular sites on the left ventricular epicardium. The mechanism is consistent with triggered activity. It is amenable to ablation by transvenous or transpericardial approaches, although technical challenges remain. Recognition of a prolonged precordial maximum deflection index and early use of transvenous epicardial mapping are critical to avoid protracted and unsuccessful ablation elsewhere in the ventricles. (***Circulation*. 2006;113:1659-1666.**)

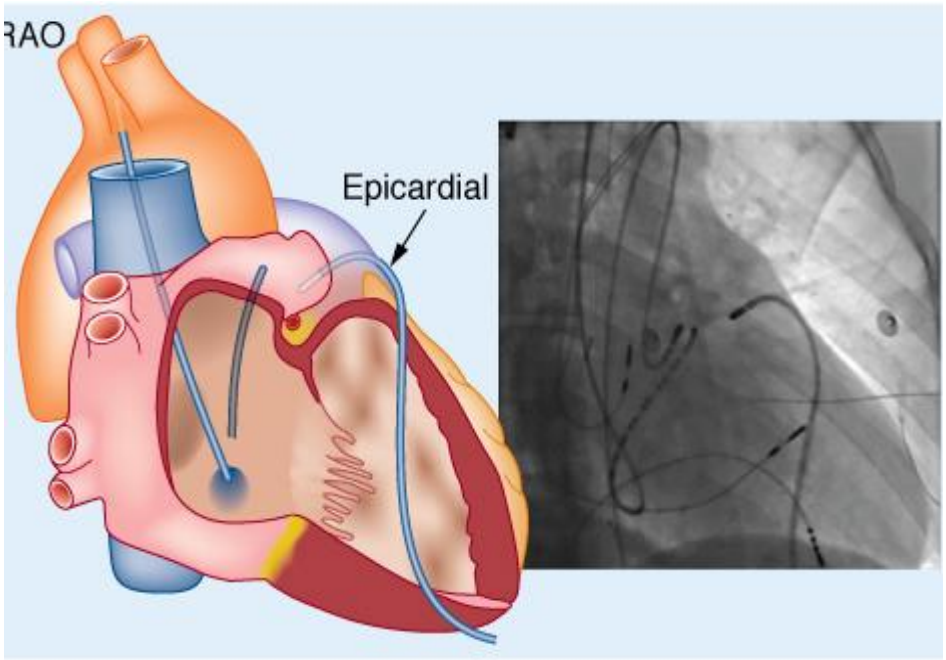
Precordial MDI >0.55 reliably identified EPI VT



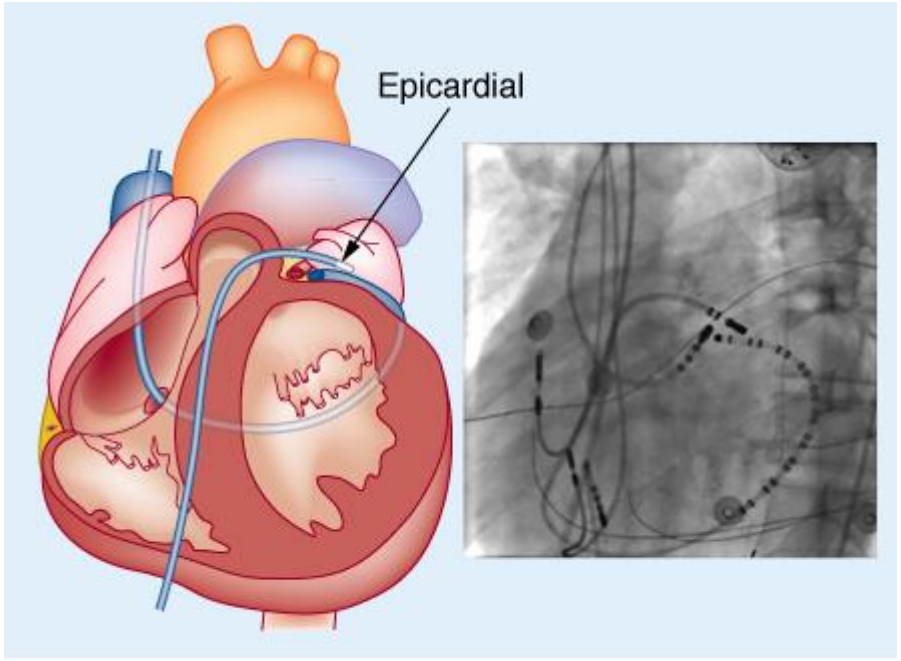


MDI : the maximum deflection index
TMD: time to maximum deflection in precordial lead

Circulation. 2006;113:1659-1666



(A)



(B)

Take-home massages

- When the diagnosis of a WCT is **uncertain**
  The patient be treated as if the rhythm is **VT**

- Several strategies or algorithms based upon

  ECG features