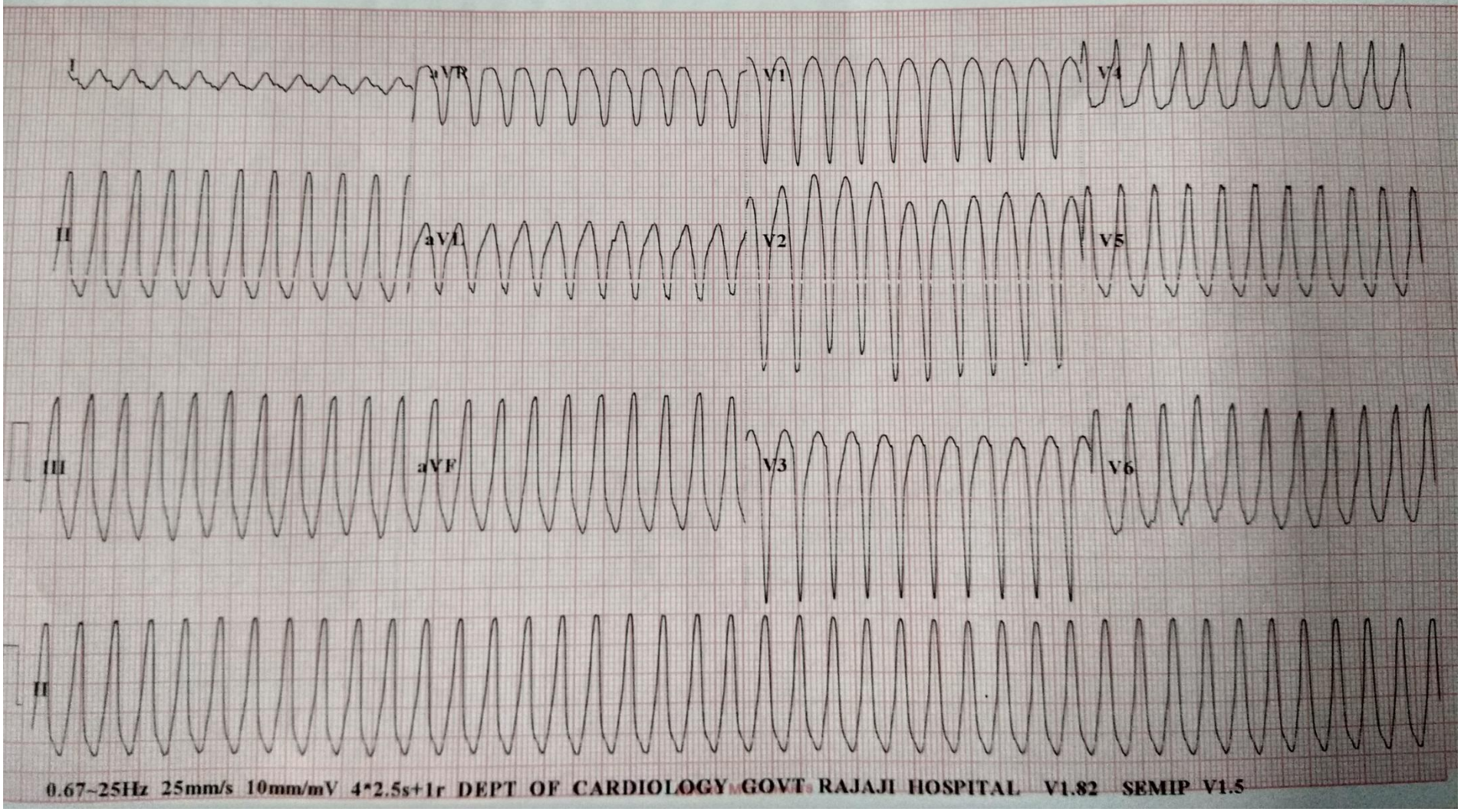
Wide Complex Tachycardia

26.3.18

Name :
Age : Years Gender : Male
Height . cm Weight . kg

HR	: 248	BPM	
P Dur	: 0	ms	
PR int	: 0	ms	
QRS Dur	: 199	ms	
QT/QTC int	: 263/535	ms	
P/QRS/T axis	: 0/90/269	°	
RV5/SV1 amp	: 1.303/1.535	mV	
RV5+SV1 amp	: 2.838	mV	
RV6/SV2 amp	: 1.342/2.254	mV	

Report Confirmed by:



Age	: Years	Gender	: Male	PR int	: 123	ms
Height	: cm	Weight	: kg	QRS Dur	: 96	ms
				QT/QTC int	: 304/434	ms
				RV5+SV1 amp	: 2.960	mV
				RV6/SV2 amp	: 0.940/1.458	mV

Report Confirmed by:

26/

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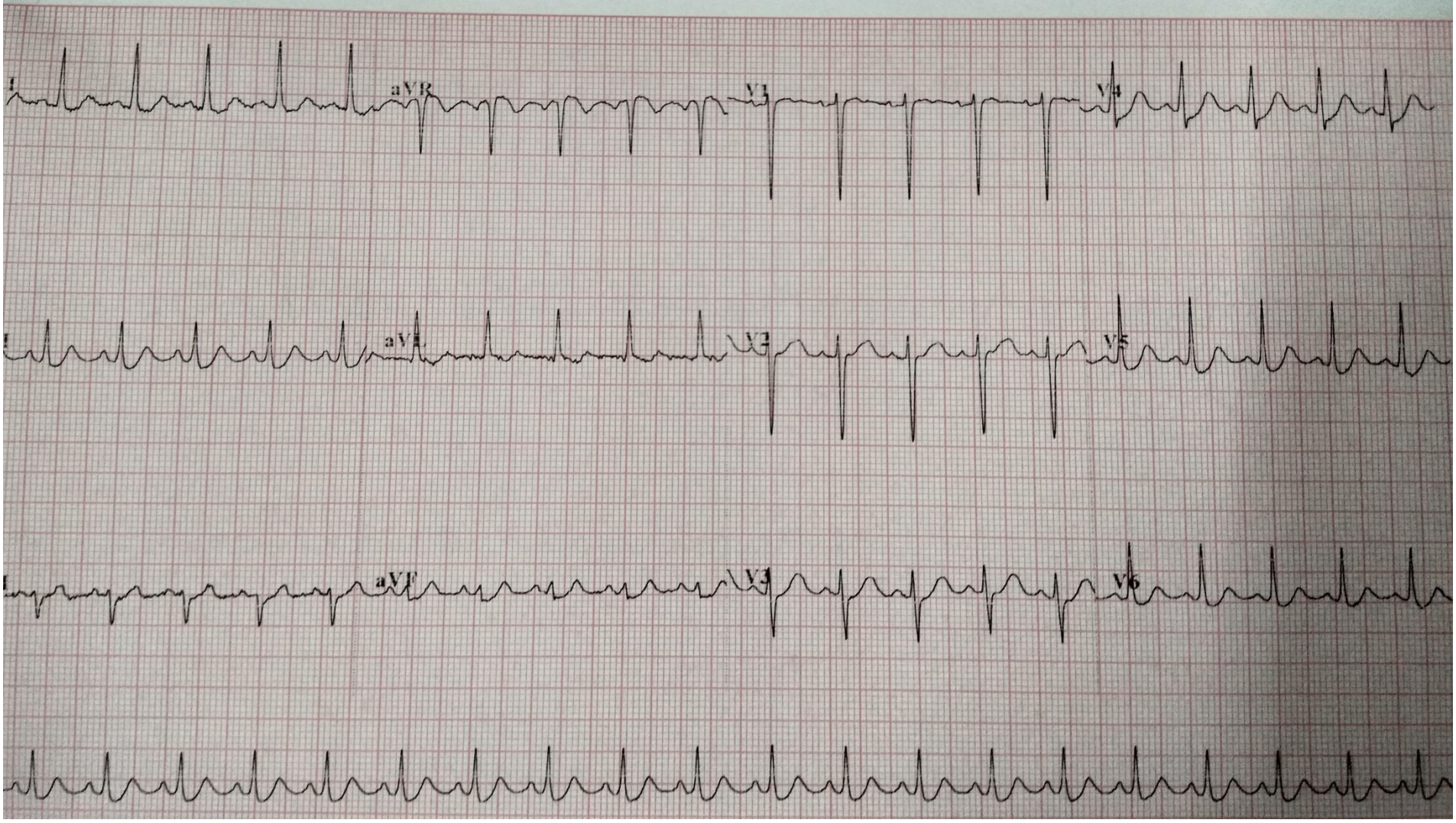


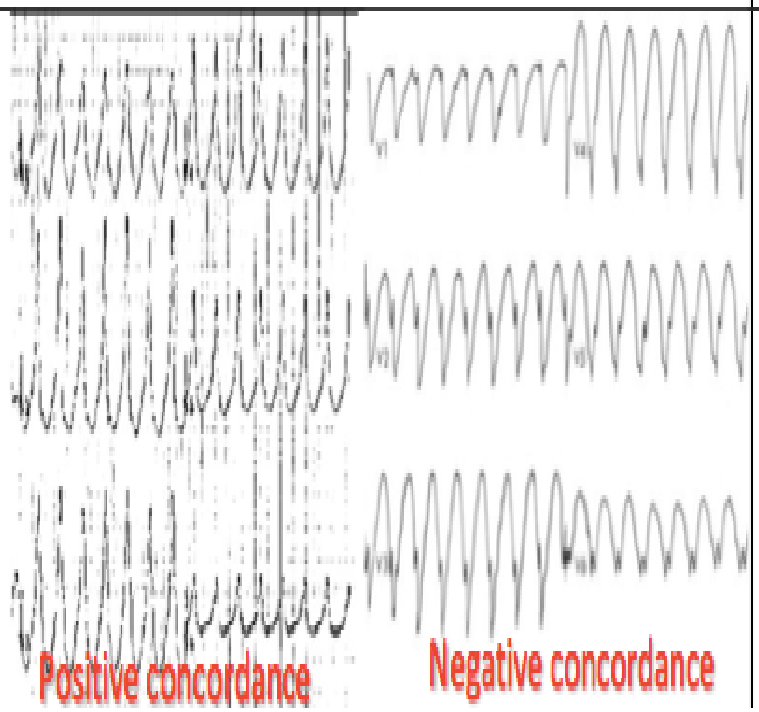
Causes of Wide Complex Tachycardia

- Ventricular Tachycardia
- SVT with Bundle Branch Block
- SVT with Accessory Pathway

Differentiating VT from SVT

BRUGADA CRITERIA

	YES
Absence of RS complex in V1 – V6	VT
RS complex duration > 100 ms	VT
AV dissociation	VT
Morphology criteria	VT

1	**Absent RS complex** in any precordial lead (ie, precordial leads all monophasic R-waves or all S-waves). **≡positive or negative concordance in chest leads**		21% sensitive, 100% specific

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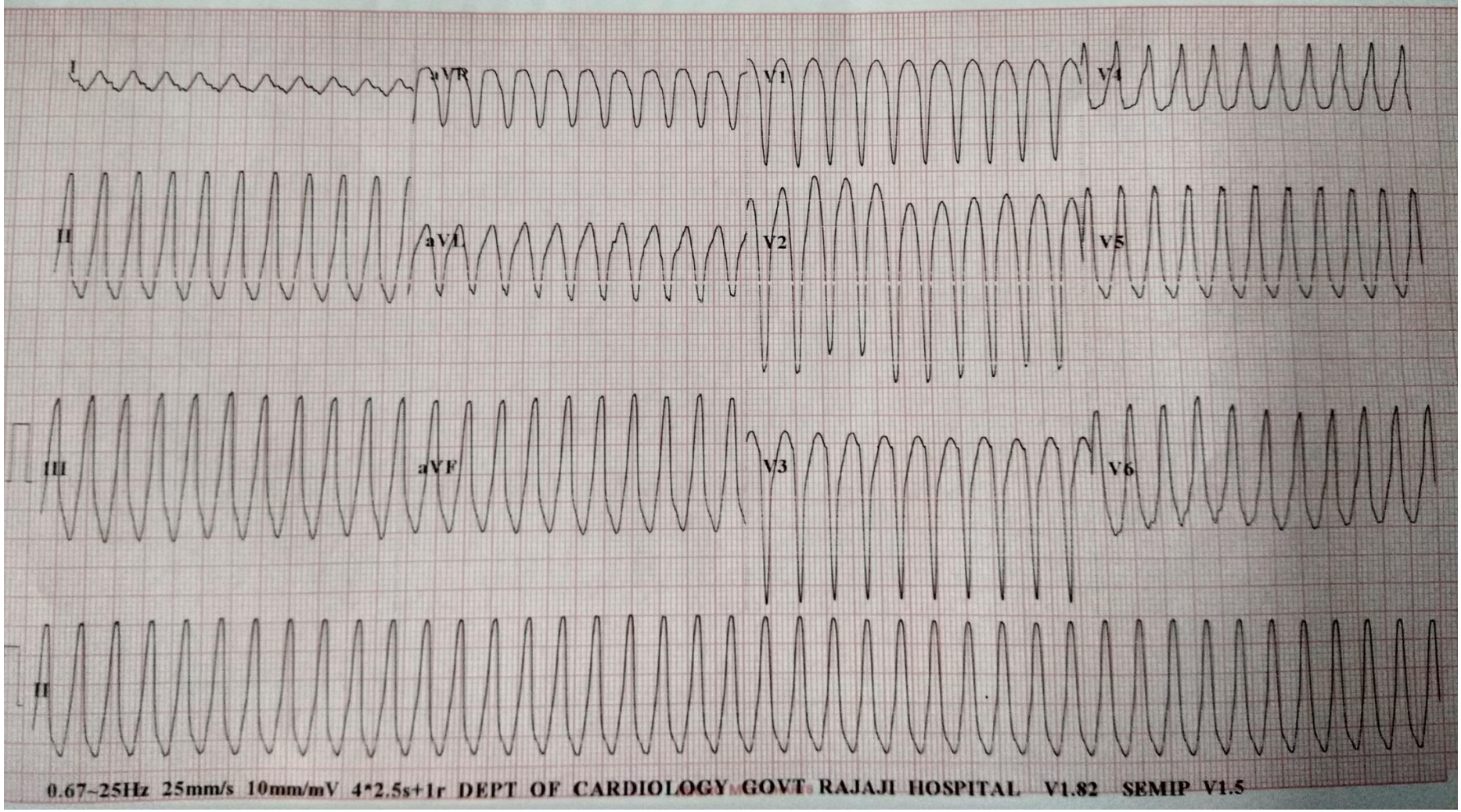
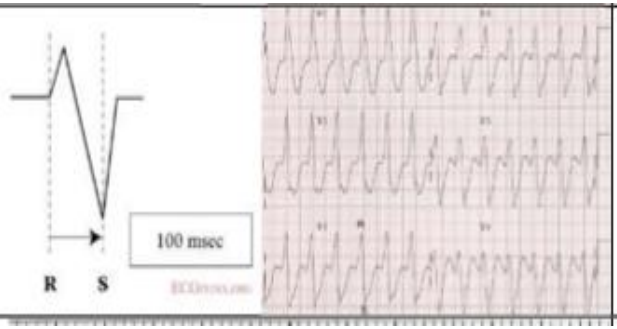
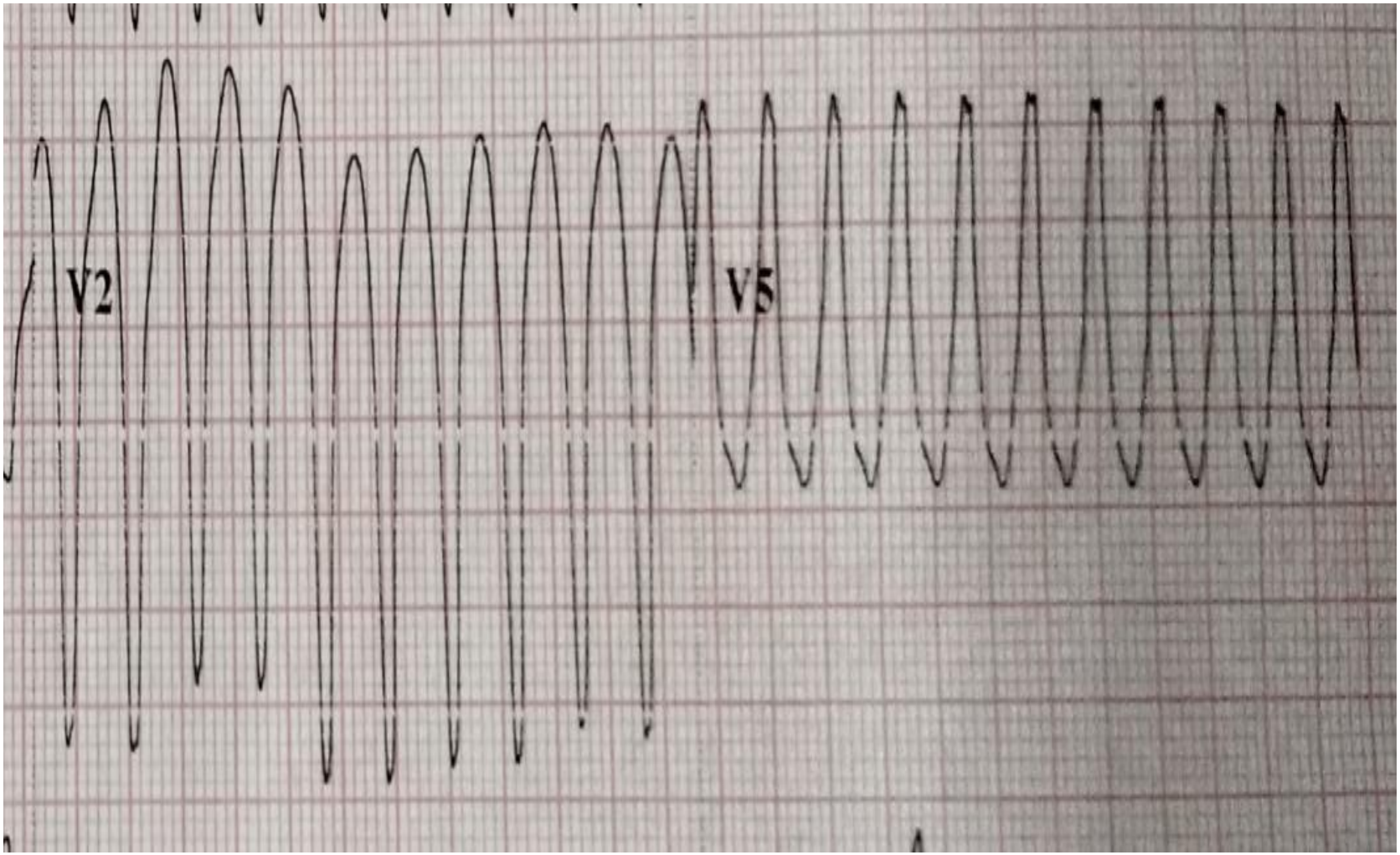


0.67~25Hz 25mm/s 10mm/mV 4*2.5s+1r DEPT OF CARDIOLOGY GOVT RAJAJI HOSPITAL V1.82 SEMIP V1.5

2	If any RS complex in V1-6, check if **RS>100ms** in one precordial lead		21% sensitive, 100% specific


V2
V5

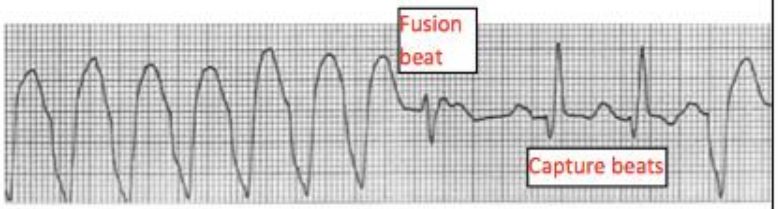
3

If RS<100ms,

Check for any **AV dissociation** (notching of QRS at various points, due to superimposed P-wave)

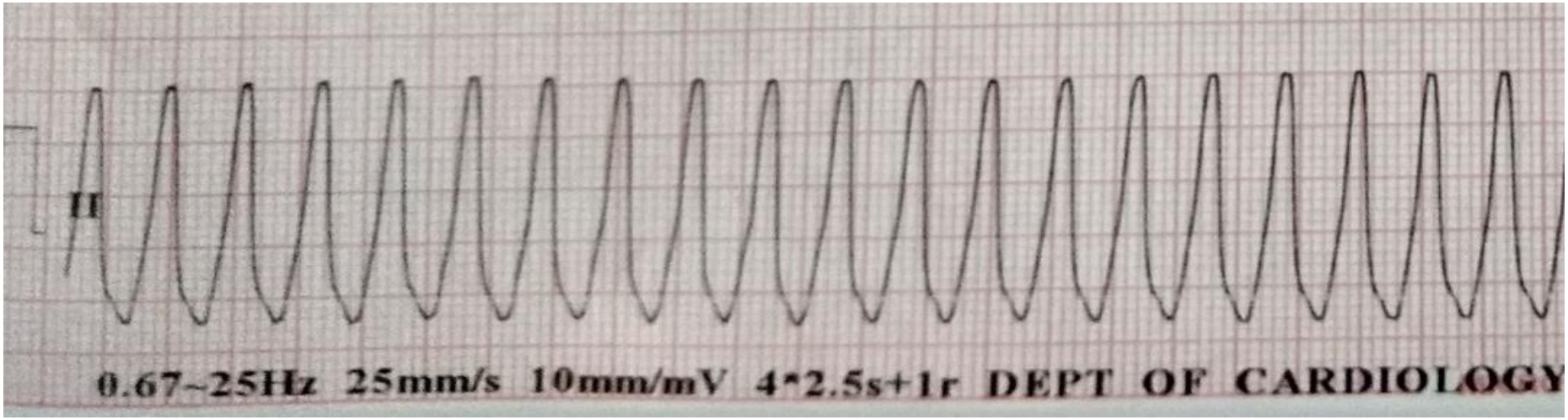
Secondary signs of AV dissociation:

Fusion beats- SV impulse reaches AV node at same time as ventricular impulse



Capture beats- SV impulse wins. Normal single QRS amidst broad complexes.

82% sensitive, 98% specific


II
0.67~25Hz 25mm/s 10mm/mV 4*2.5s+1r DEPT OF CARDIOLOGY

4

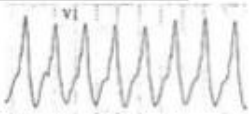
If no signs of AV dissociation,
Check for characteristic features of VT in V1-2,6

98% sensitive,
97% specific

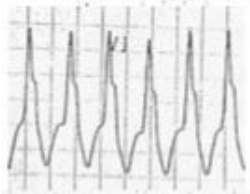
In V1, if **dominant R-wave**, look for **RBBB** morphology

<u>3 patterns in V1-2 indicative of VT:</u>

- Smooth monophasic R wave



- **Taller Lt Rabbit's Ear** (Marriott's sign)

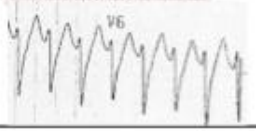


- qR complex V1

<u>2 patterns in V6 indicative of VT:</u>

- QS complex (no R wave)

- rS (indicates VT if LAD present)



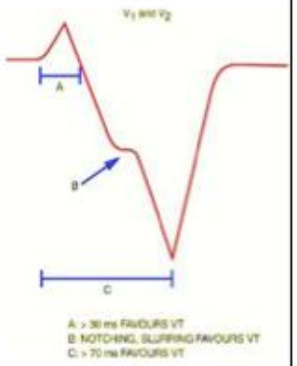
In V1, if **dominant S-wave**, look for **LBBB** morphology

<u>3 features in V1-2 indicative of VT:</u>

A. Initial R wave >30ms

B. **Notching/Slurring of S wave (Josephson's sign)**

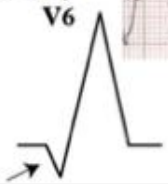
C. RS>70ms



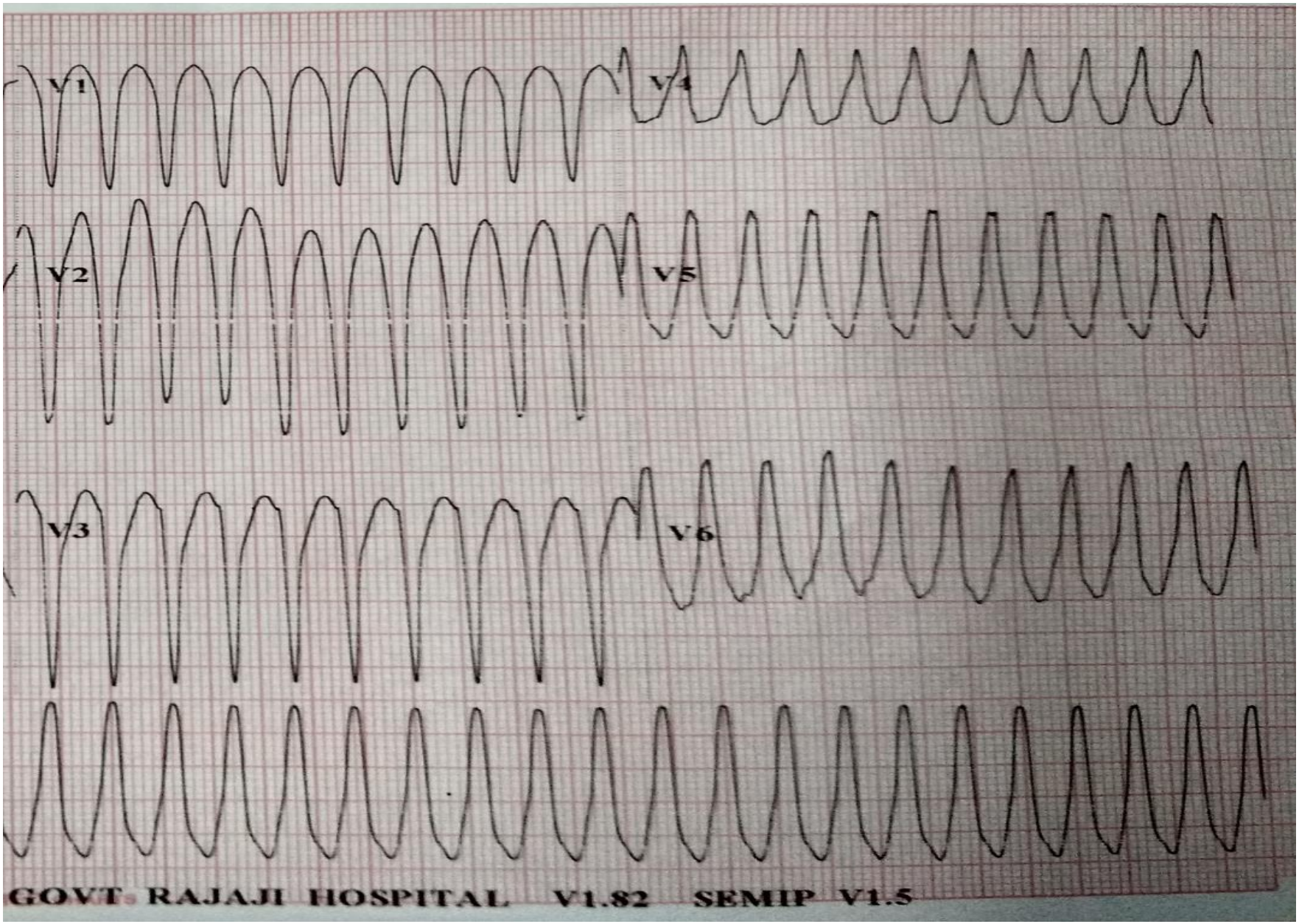
<u>2 patterns in V6 indicative of VT:</u>

- QS complex (same as for RBBB)

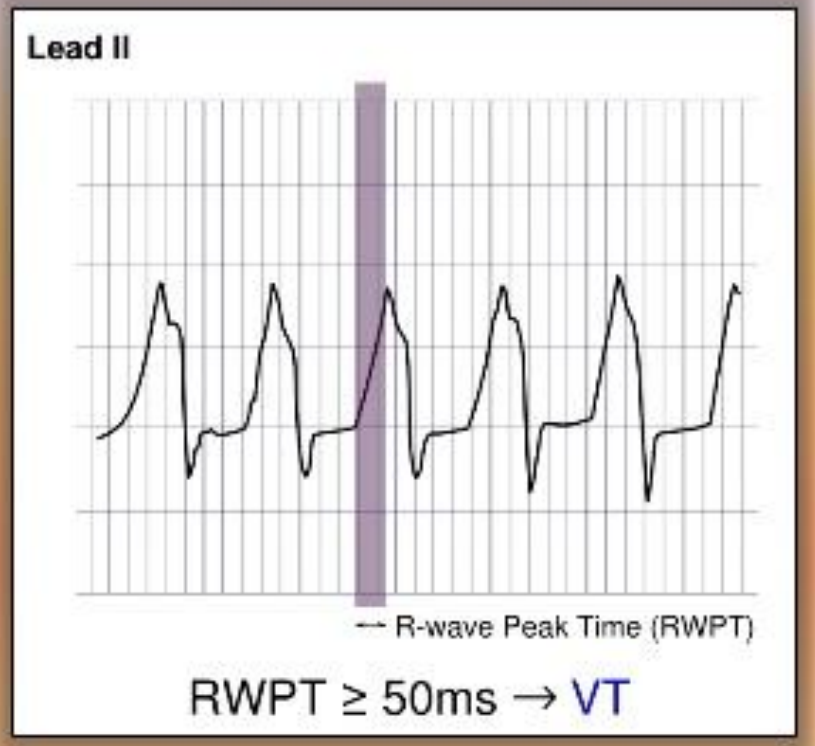
- qR



Francis Lam

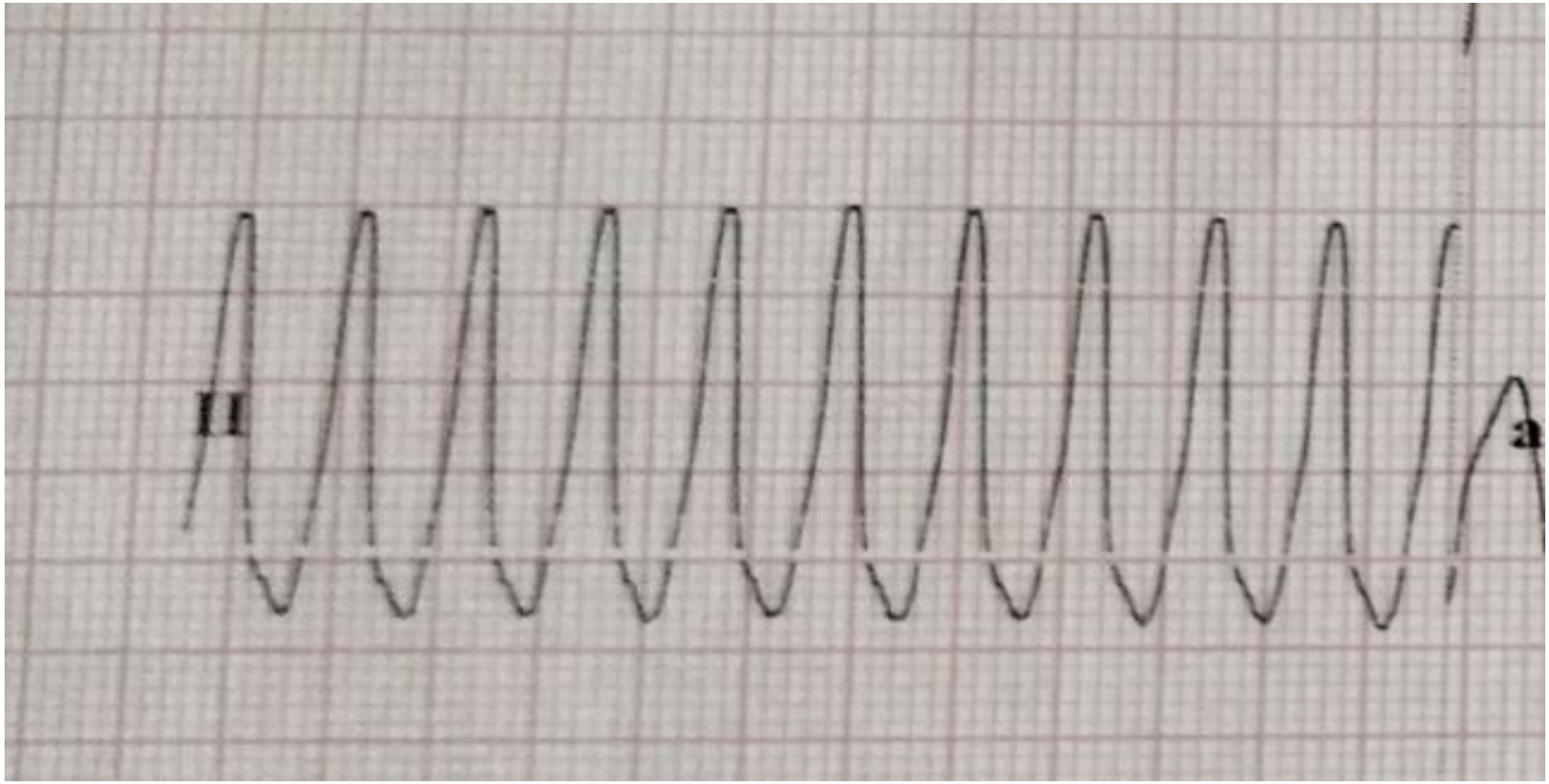

V1
V4
V2
V5
V3
V6
GOVT. RAJAJI HOSPITAL V1.82 SEMIP V1.5

Ultrasimple Brugada criterion: RW to peak Time (RWPT)

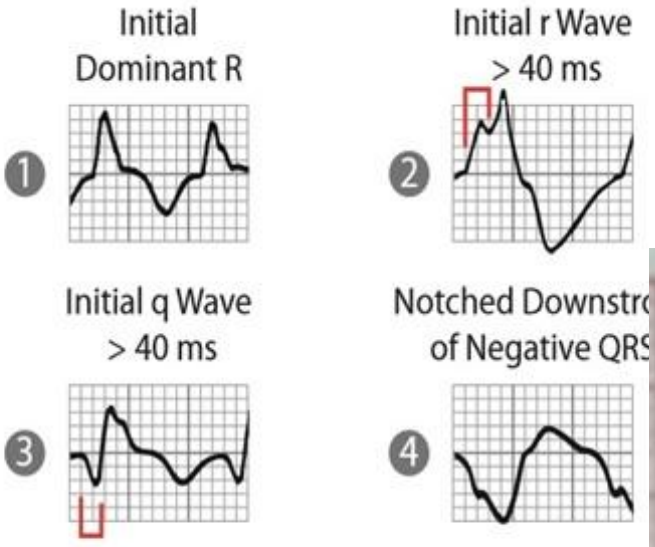


Sensitivity 60%, specificity 82.7%.

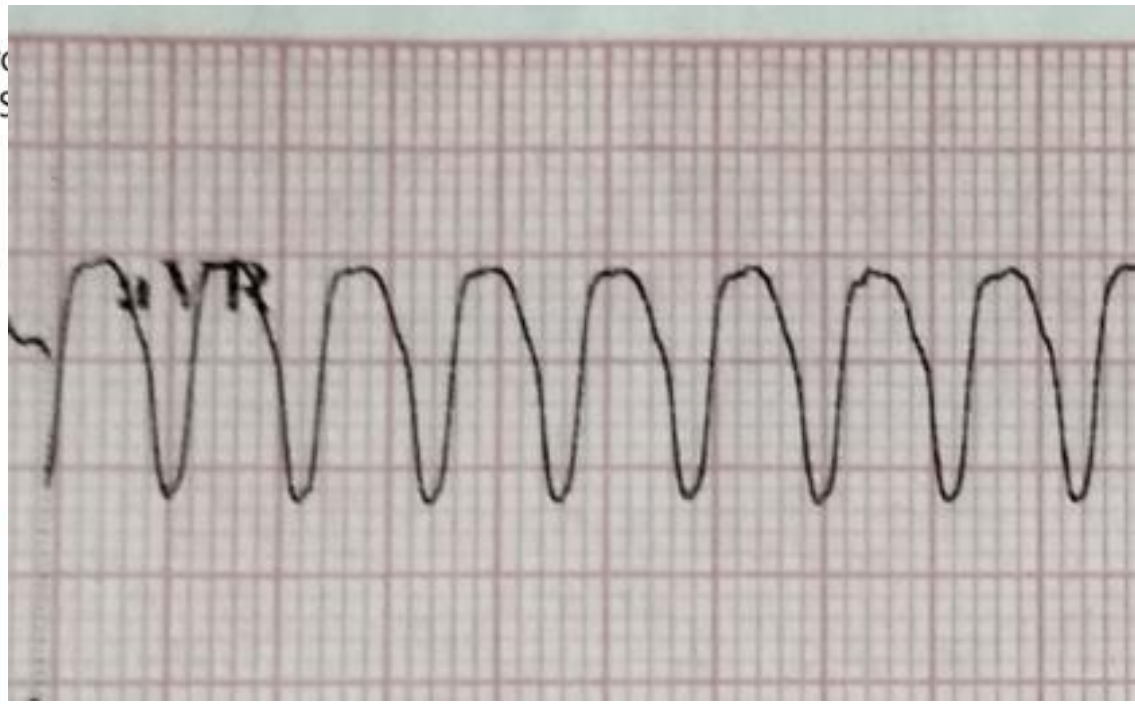
Pava LF, Perafán P, Badiel M, Arango JJ, Mont L, Morillo CA, and Brugada J. R-wave peak time at DII: a new criterion for differentiating between wide complex QRS tachycardias. Heart Rhythm 2010 Jul; 7(7) 922-6. doi:10.1016/j.hrthm.2010.03.001 pmid:20215043


II
a

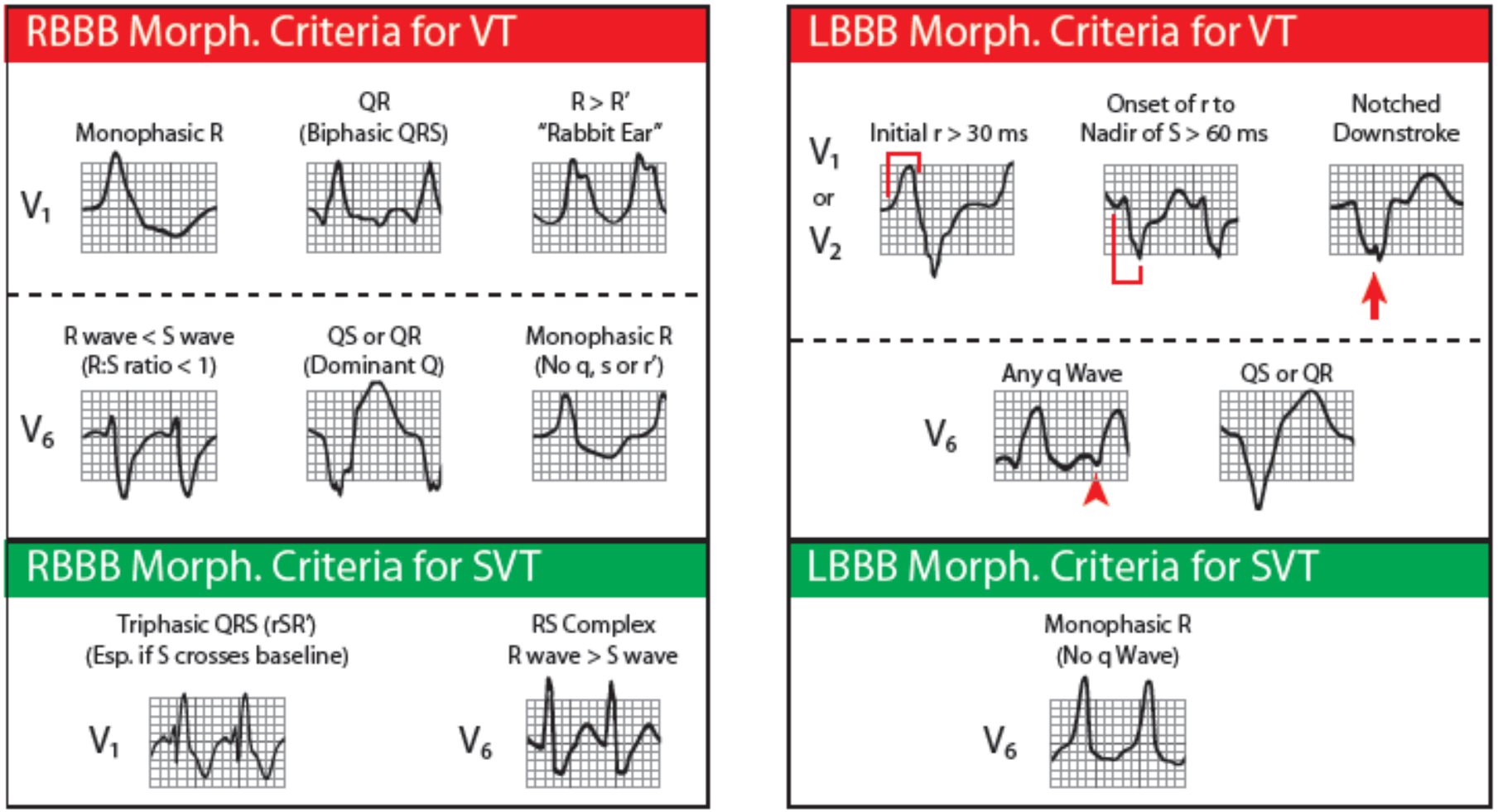
Vereckei aVR algorithm



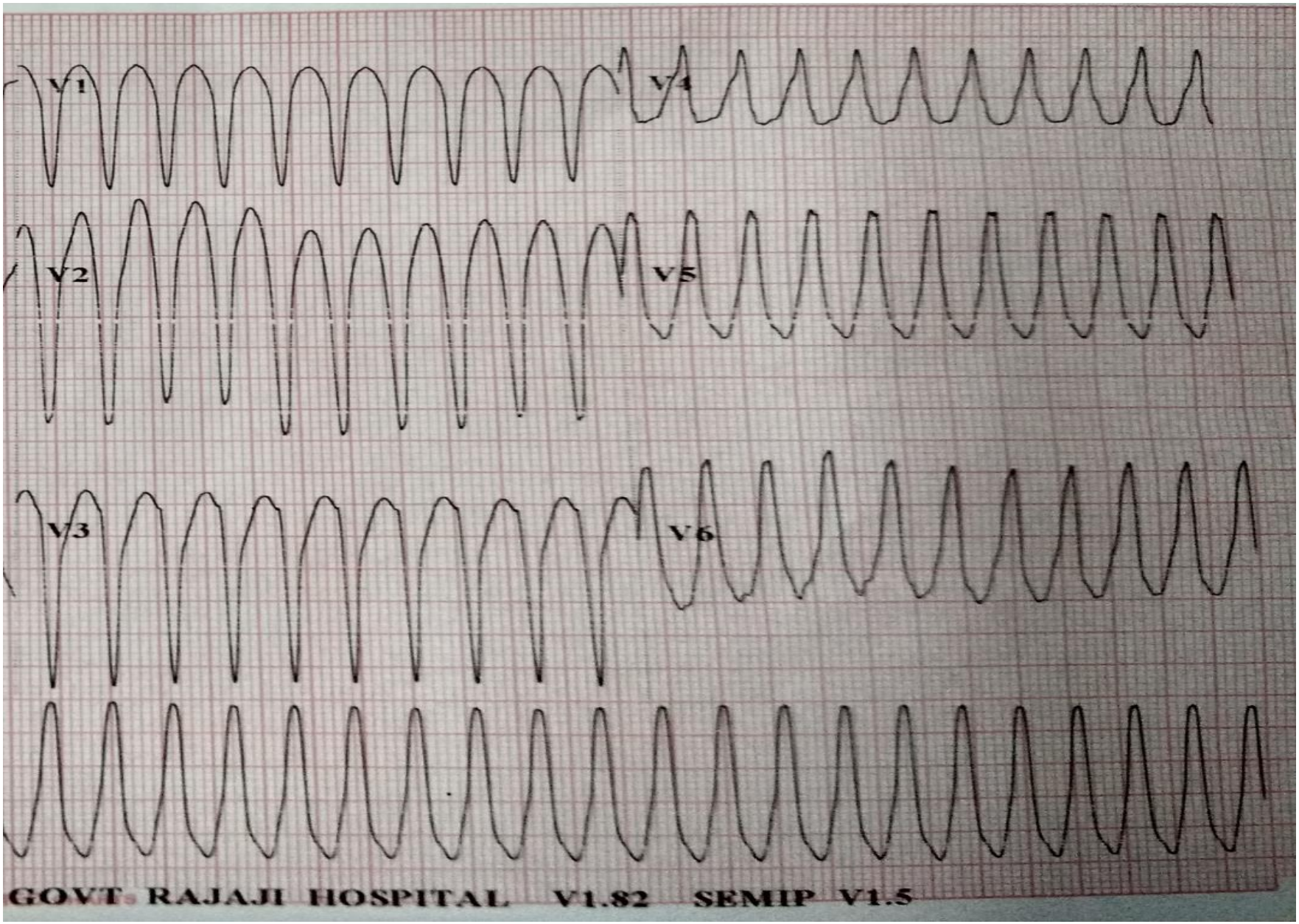
Any of the above in lead aVR ➜ VT
None of the above in lead aVR ➜ SVT



Griffith Algorithm



AV = atrioventricular; aVR = augmented vector right; LBBB = left bundle branch block; RBBB = right bundle branch block; SVT = su


V1
V4
V2
V5
V3
V6
GOVT. RAJAJI HOSPITAL V1.82 SEMIP V1.5

Mechanisms

- Reentry
- Triggered Activity
- Abnormal Automaticity

Causes

- Ischemic Heart Disease
- Structural Heart Disease:
  - Hypertrophic Cardiomyopathy
  - Other Cardiomyopathies
  - ARVD
- Congenital Heart Diseases (TOF) and their surgical scars

Causes contd...

- Inherited Channelopathies:
  - Long QT syndrome
  - Short QT syndrome
  - Brugada syndrome
  - Catecholaminergic VT
- Acquired Channelopathies:
  - Drugs that prolong QT interval

Causes contd...

- Eletrolyte imbalances: hypokalemia, hypomagnesemia
- Sympathomimetic agents
- Digitalis toxicity
- Systemic illness causing cardiomyopathy or scar: SLE, Amyloidosis etc

Causes in <35yrs of age

- Hypertrophic Cardiomyopathy
- ARVD
- Myocarditis
- Long QT syndrome
- Congenital Coronary artery abnormalities

Treatment

- **Unstable Patients**: (Chest pain, Dyspnea, Hypotension, Altered level of consciousness)
  - **Monomorphic VT** → Synchronised DC cardioversion starting at 100J monophasic
  - **Polymorphic VT** → Defibrillation

Treatment in Stable patients

- Monomorphic VT and normal LV fn:
  - Procainamide
  - Sotalol
  - Lidocaine
- Monomorphic VT with impaired LV fn:
  - Amiodarone
  - Lidocaine
  - If drug therapy fails – synchronised cardioversion

Treatment in Stable patients

- Polymorphic VT:
  - Terminates on its own, but recurs. Sinus rhythm ecg analysed for prolonged QT interval
  - Normal QT interval treated as above
  - Prolonged QT interval
    - Magnesium sulphate
    - Isoproterenol
    - Pacing
    - Phenytoin, Lidocaine may also help
    - Procainamide and Amiodarone are **contraindicated**

Thank You