Ventricular tachycardia in non-ischemic patients

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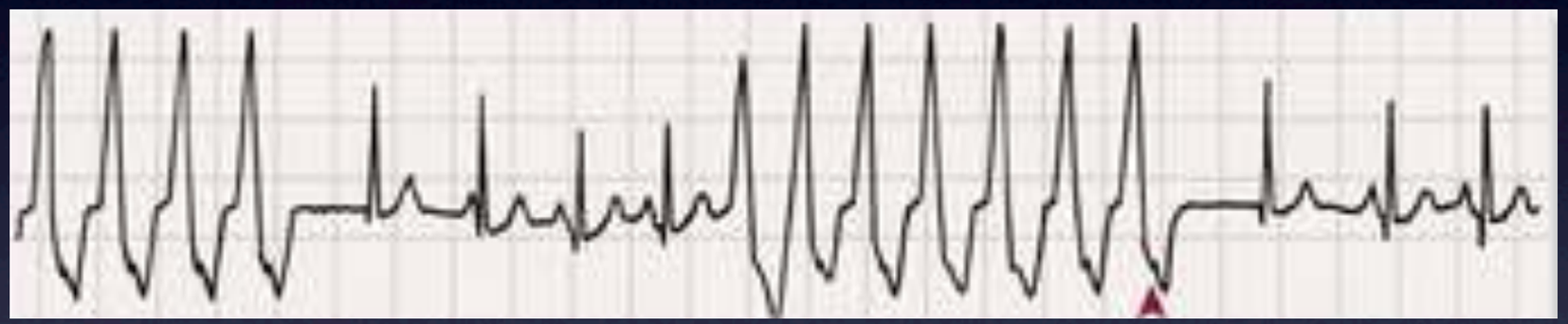
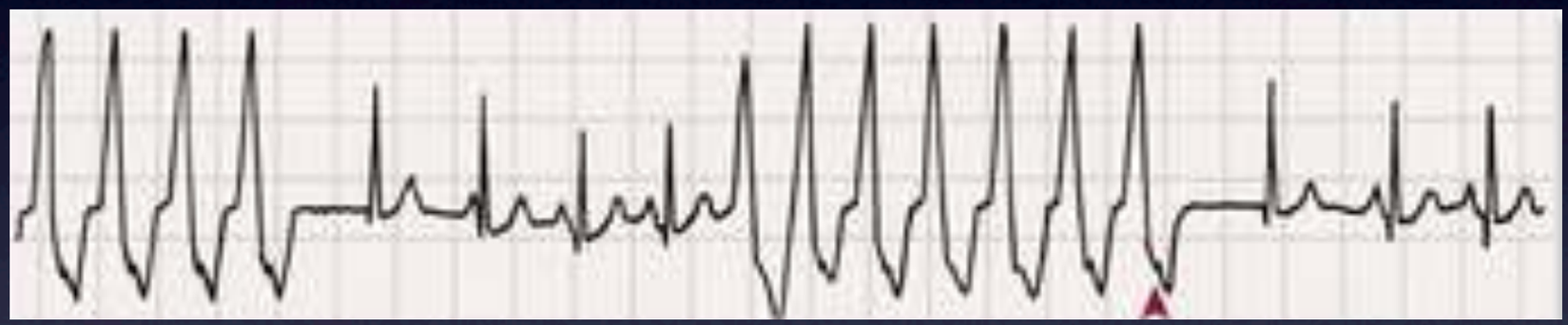
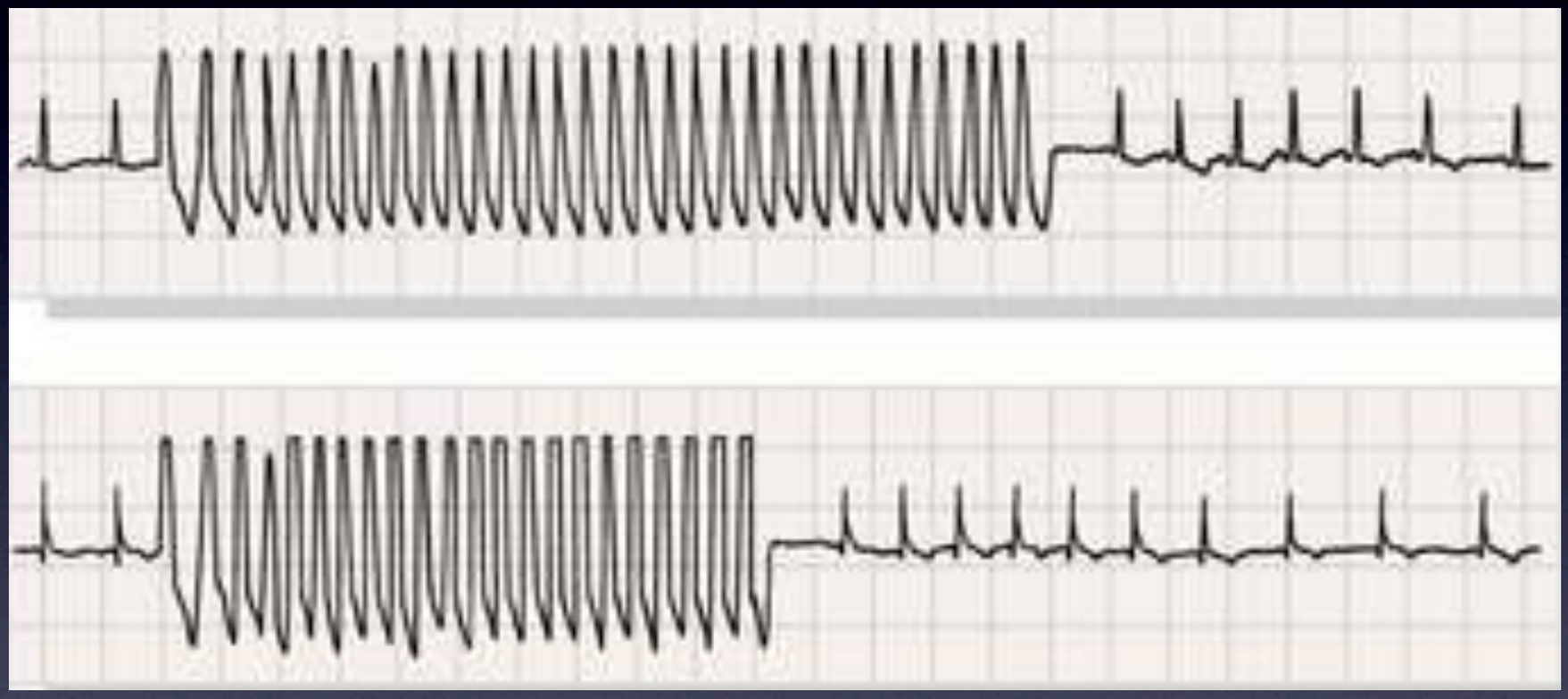
6/10/13

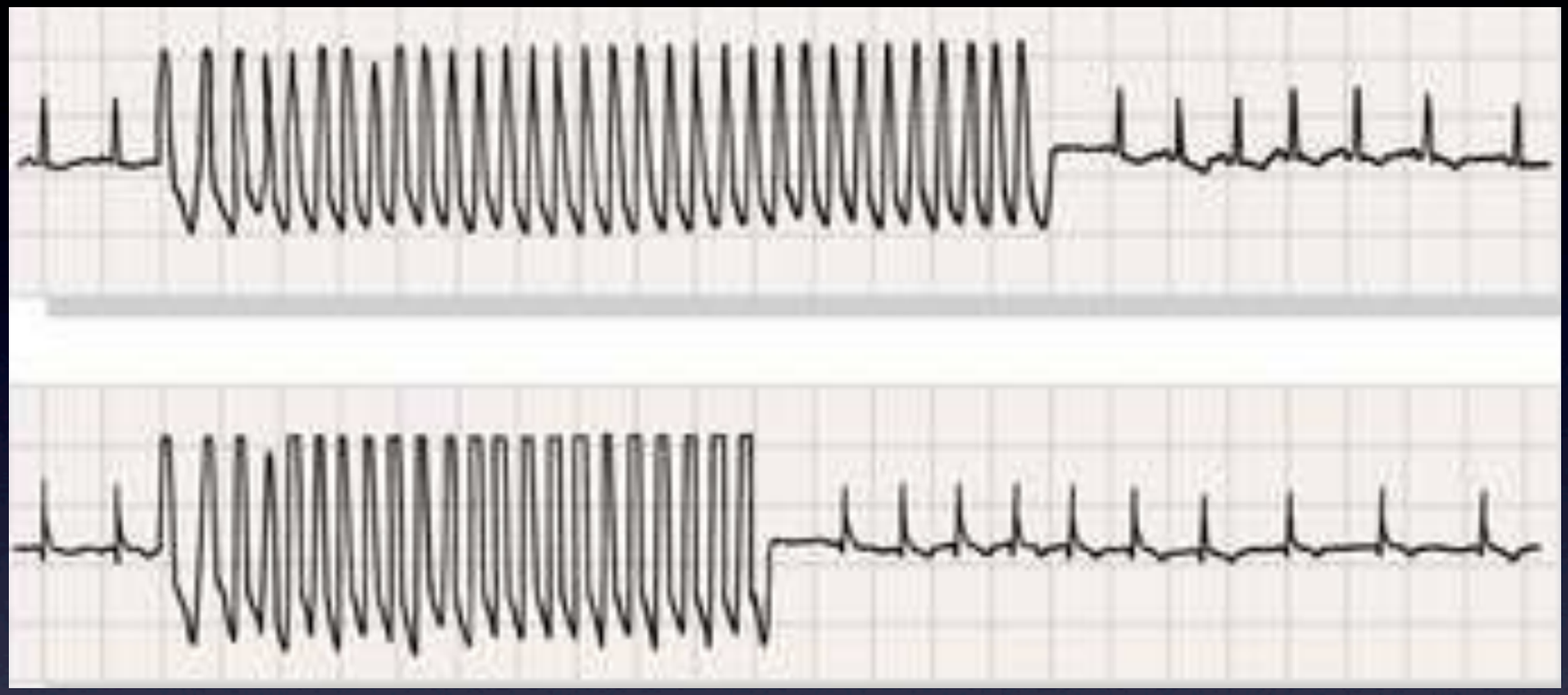


Repetitive monomorphic VT



Retrograde
atrial capture





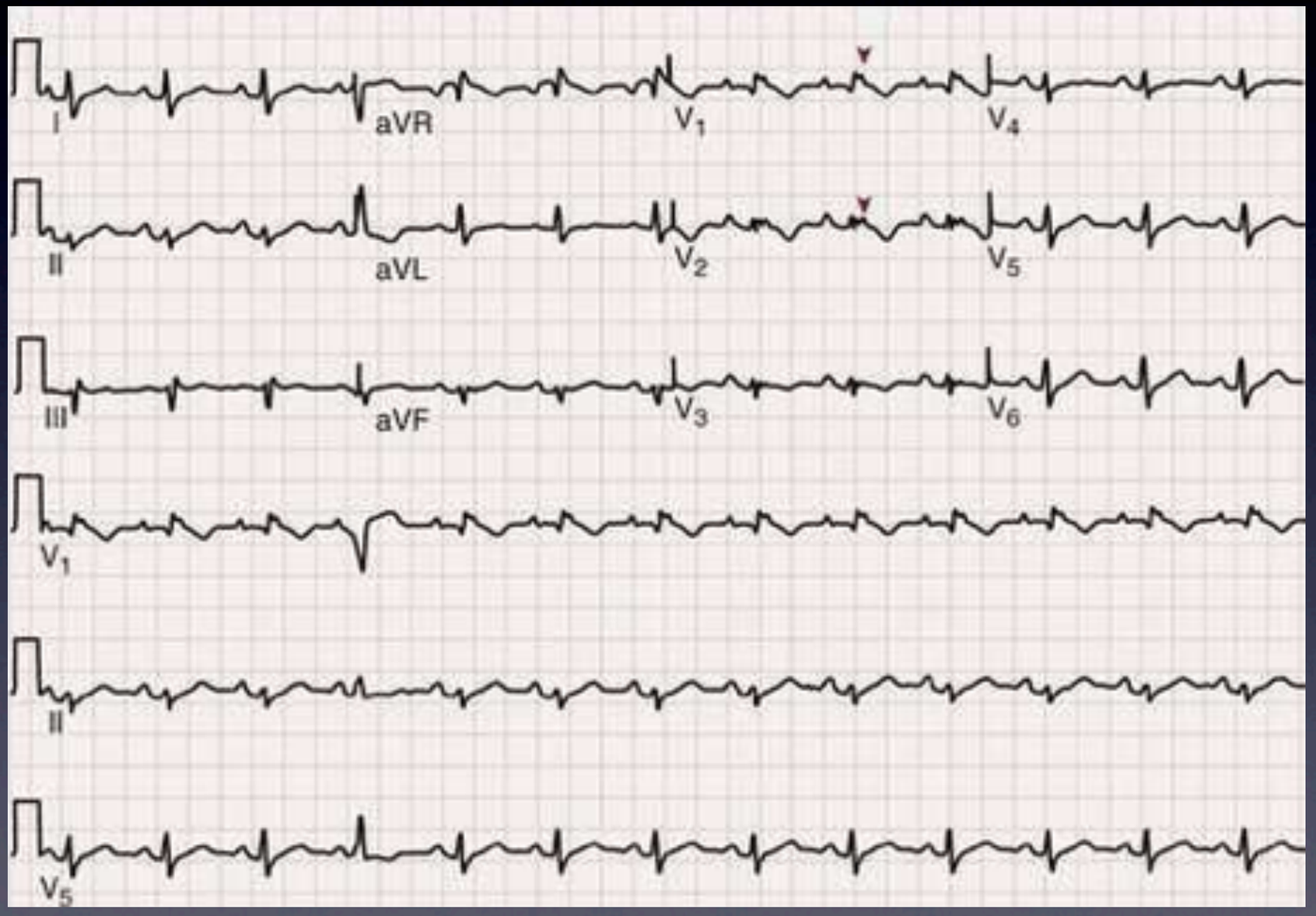
rapid VT ~ 260 bpm with compensatory sympathetic stimulation, sinus tachycardia and unstable sinus pacemaker

Dilated Cardiomyopathy

- ICD indicated if life threatening
- bundle branch reentry in some ~ ablation

Hypertrophic Cardiomyopathy

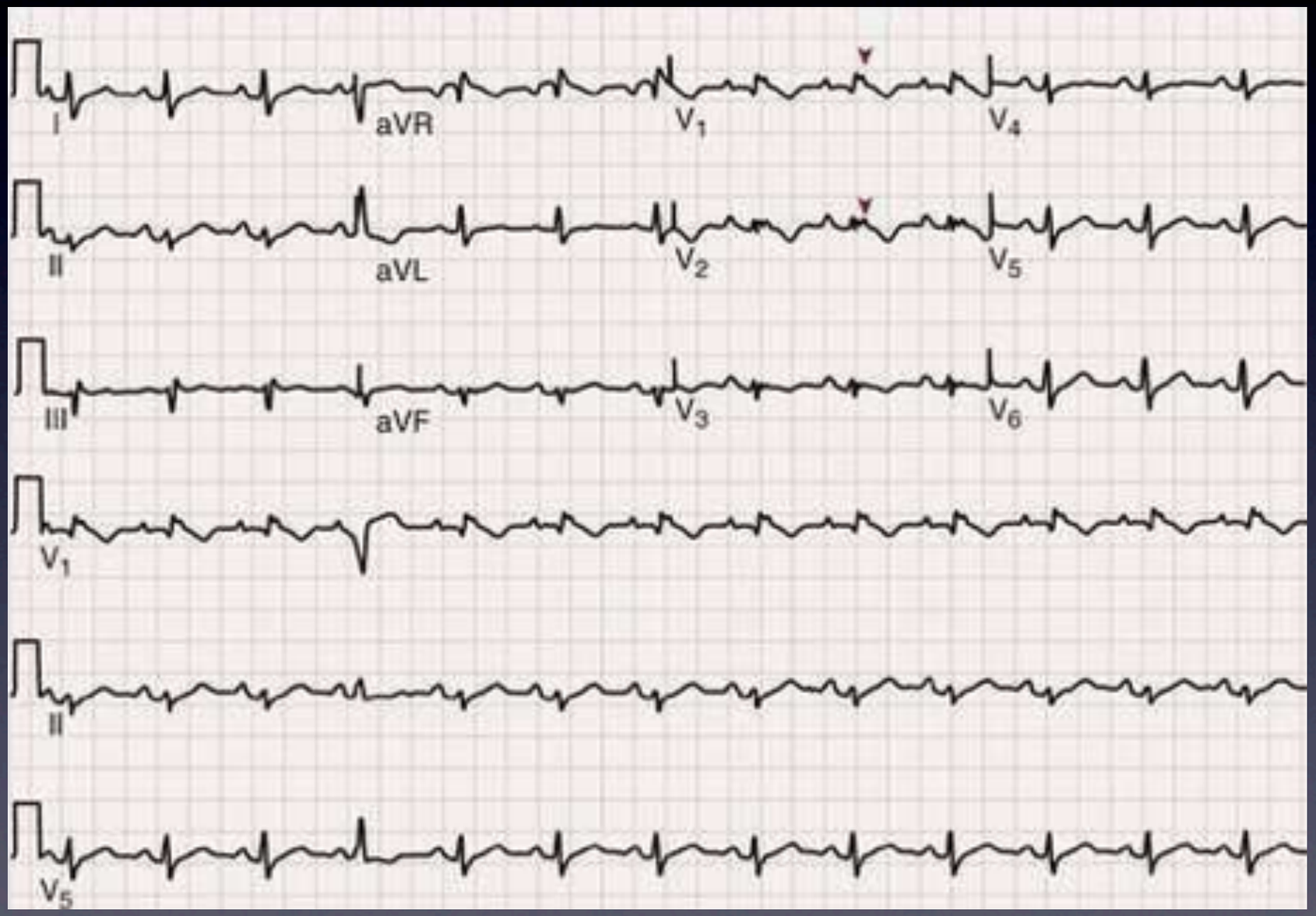
- syncope
- family history of sudden death in first-degree relatives
- septal thickness > 3 cm
- NSVT on holter
- Consider ICD in these patients
- alcohol ablation increases arrhythmia risk


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5

ARVD

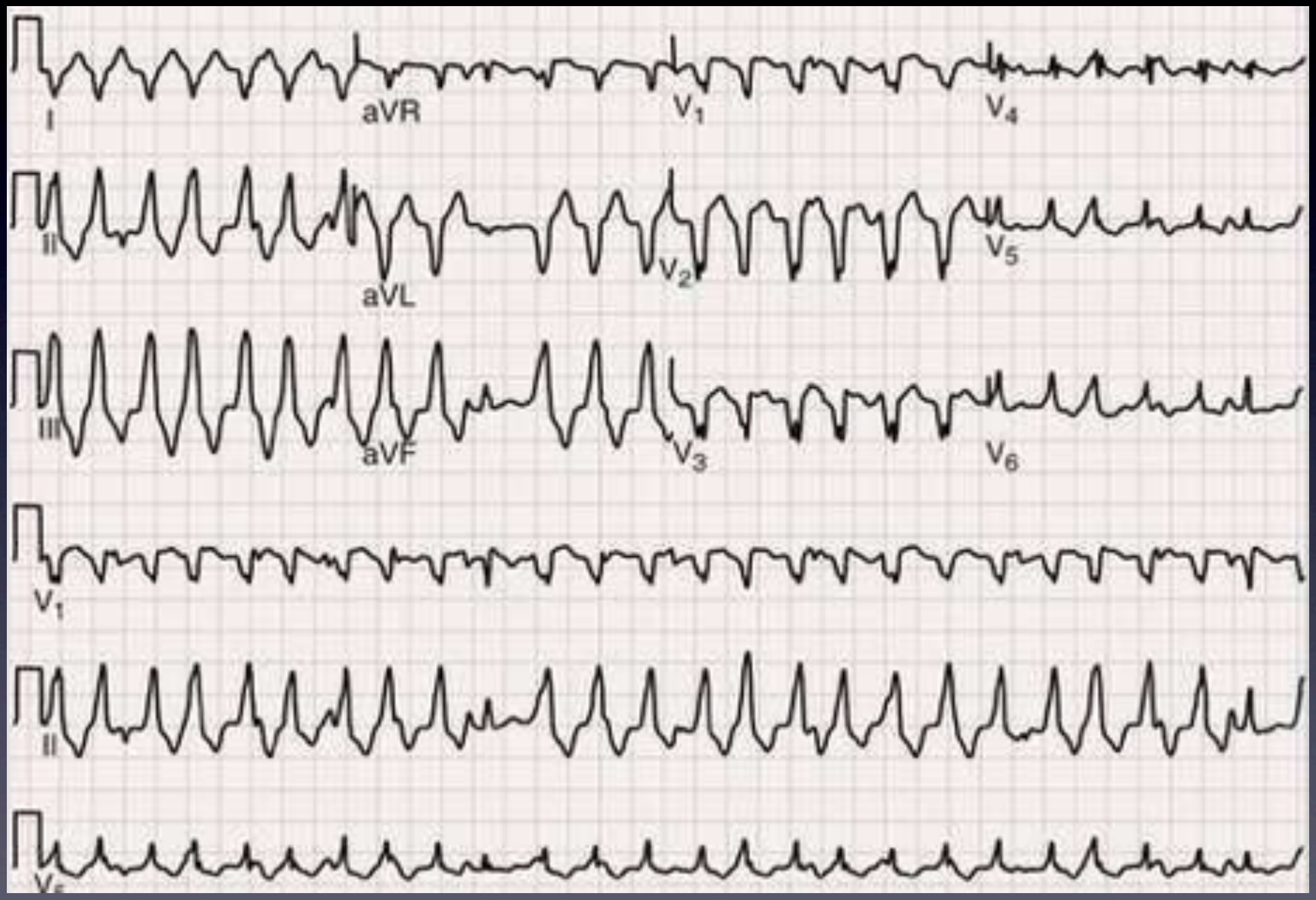
incomplete RBBB

inverted t wave in V1-V3



Epsilon wave

RAD



LBBB

Arrhythmogenic right ventricular cardiomyopathy

- VT with LBBB contour
- Right axis deviation
- t waves inverted over the right precordial leads
- reentry
- SVT can occur
- exercise can induce VT

- Cardiomyopathy ~ RV wall hypokinesis
- Genetic abnormalities of desmosomal proteins, ryanodine receptor and transforming growth factor beta-3

- children or young adults
- tachycardia or asymptomatic RV enlargement
- Male predominance

- Fibrofatty infiltration
- Fatty degeneration: RV inflow and outflow tract and apex
- Rx: RF ablation or ICD

Tetralogy of Fallot

- seen in patients with repair
- reentry at the site of previous surgery in the RVOT
- Rx: resection or ablation

Catecholaminergic polymorphic ventricular tachycardia

- Children and adolescents
- No overt structural heart disease
- Syncope or aborted sudden death
- Reproducible stress induced VT

- Bidirectional
- QT interval is normal
- Positive family history in 30%

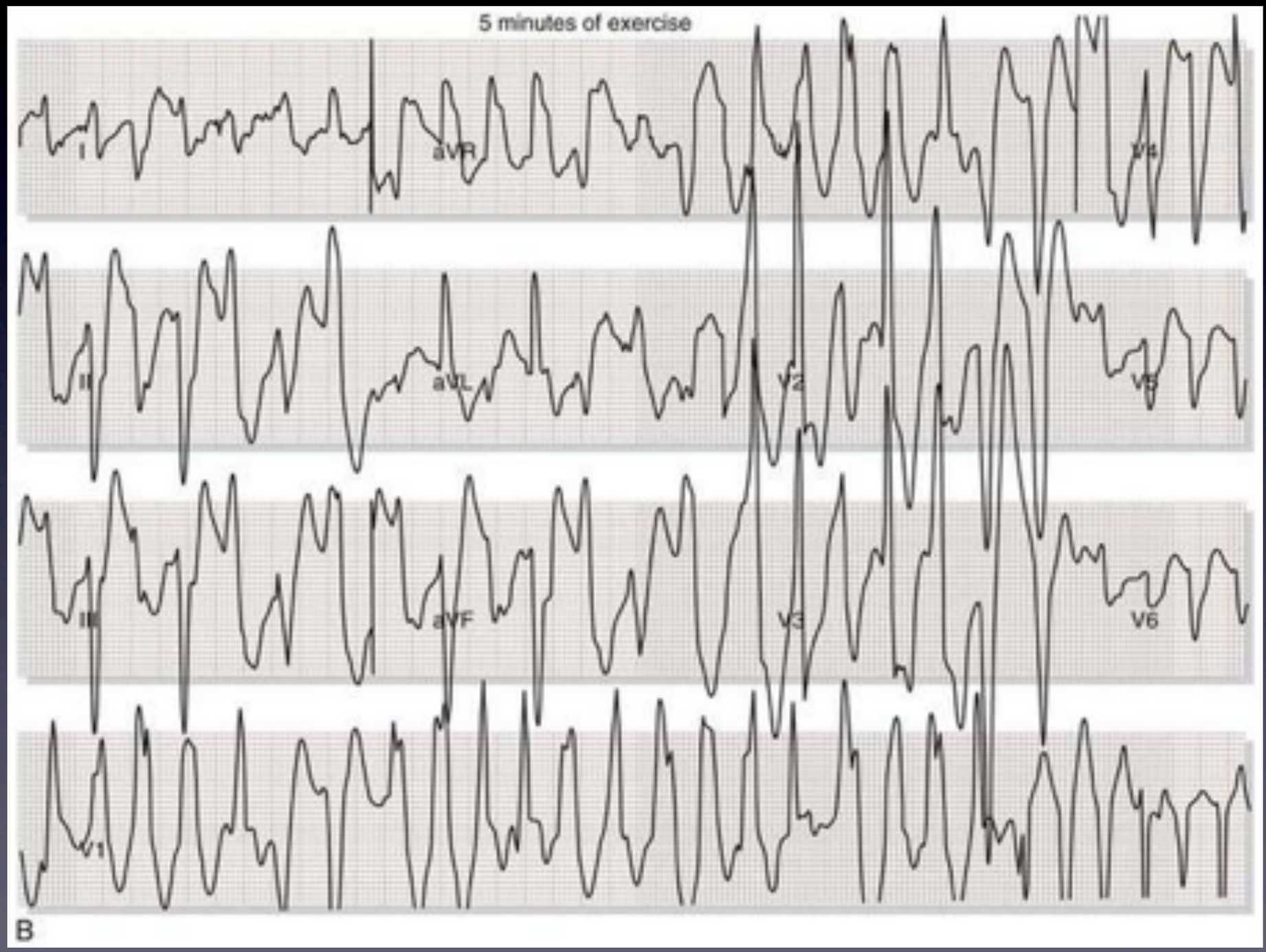
- mutations of the ryanodine receptor gene ~ AD form accounts for 50% of cases
- mutations in the calsequestrin gene ~ AR form accounts for 2% of the cases
- Rx: beta-blocker, ICD, sympathectomy, ? flecainide in ryanodine receptor mediated, avoid vigorous exercise

Exercise response

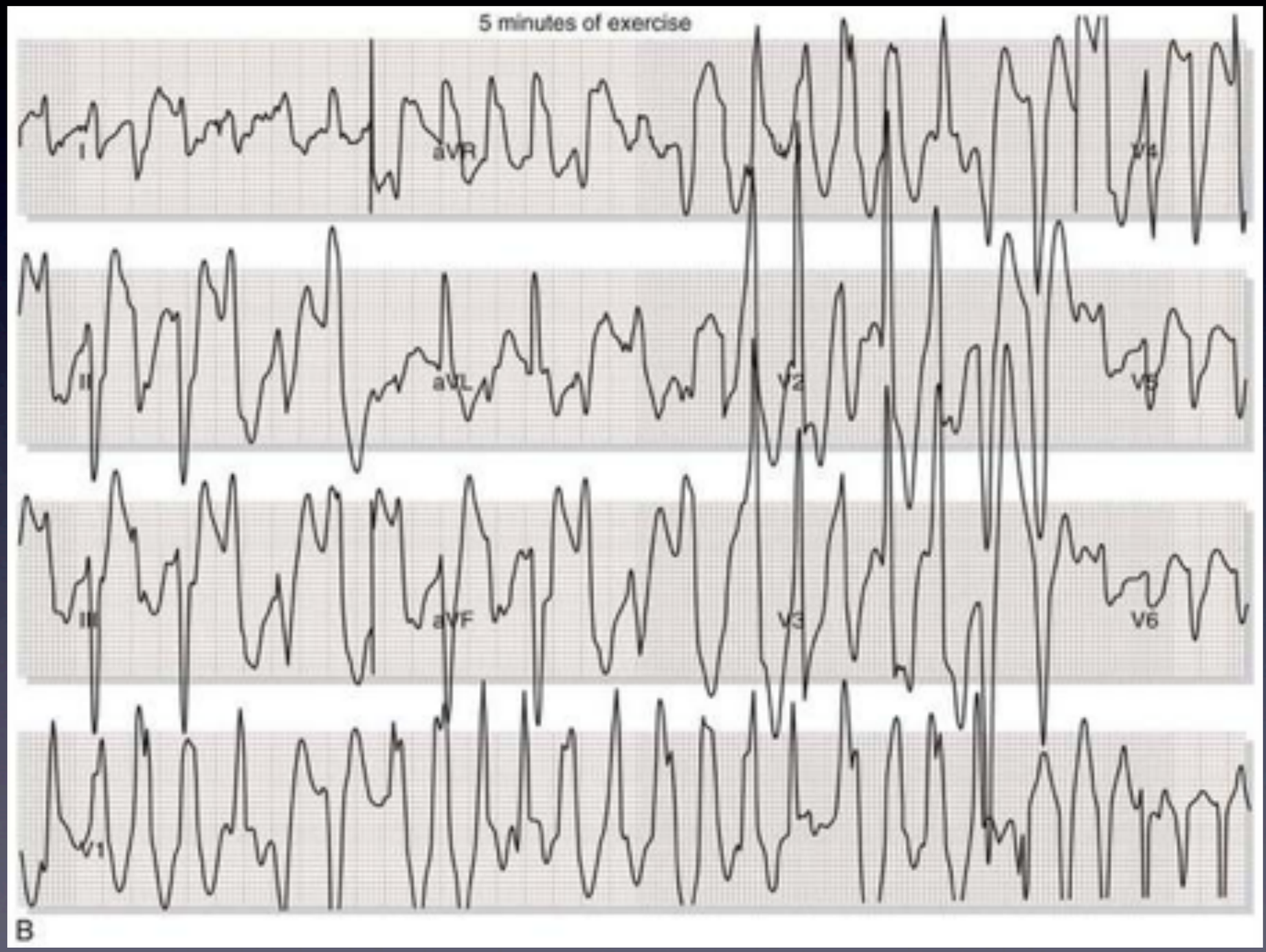
sinus tachycardia

PVC's

salvoes of monomorphic VT


5 minutes of exercise
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
B

Bidirectional VT



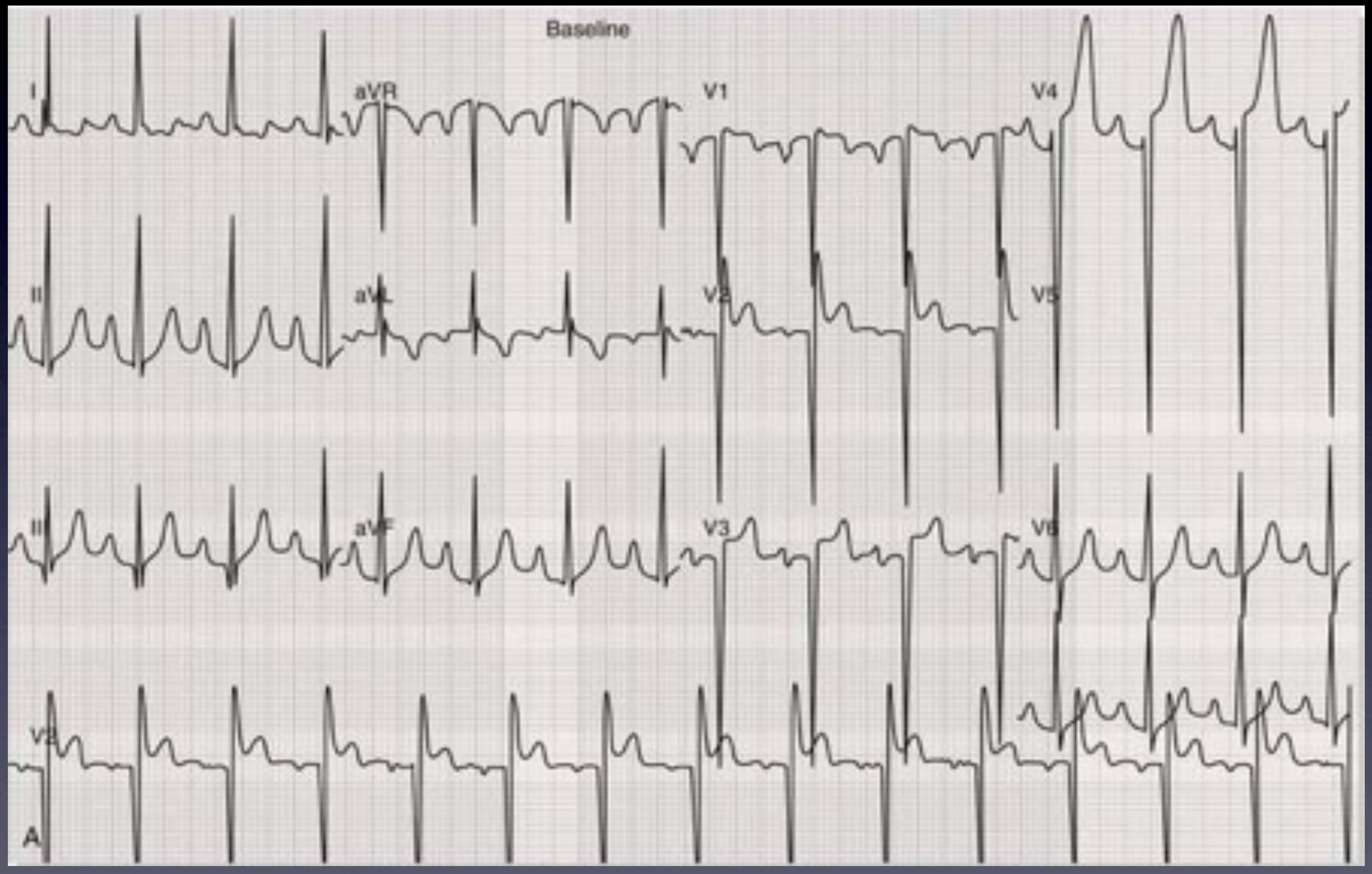
Bidirectional VT

- RBBB, alternating QRS polarity (- 60 to 90 to + 120 to130)
- VR is 140 to 200
- Digitalis excess
- CPVT

Brugada Syndrome

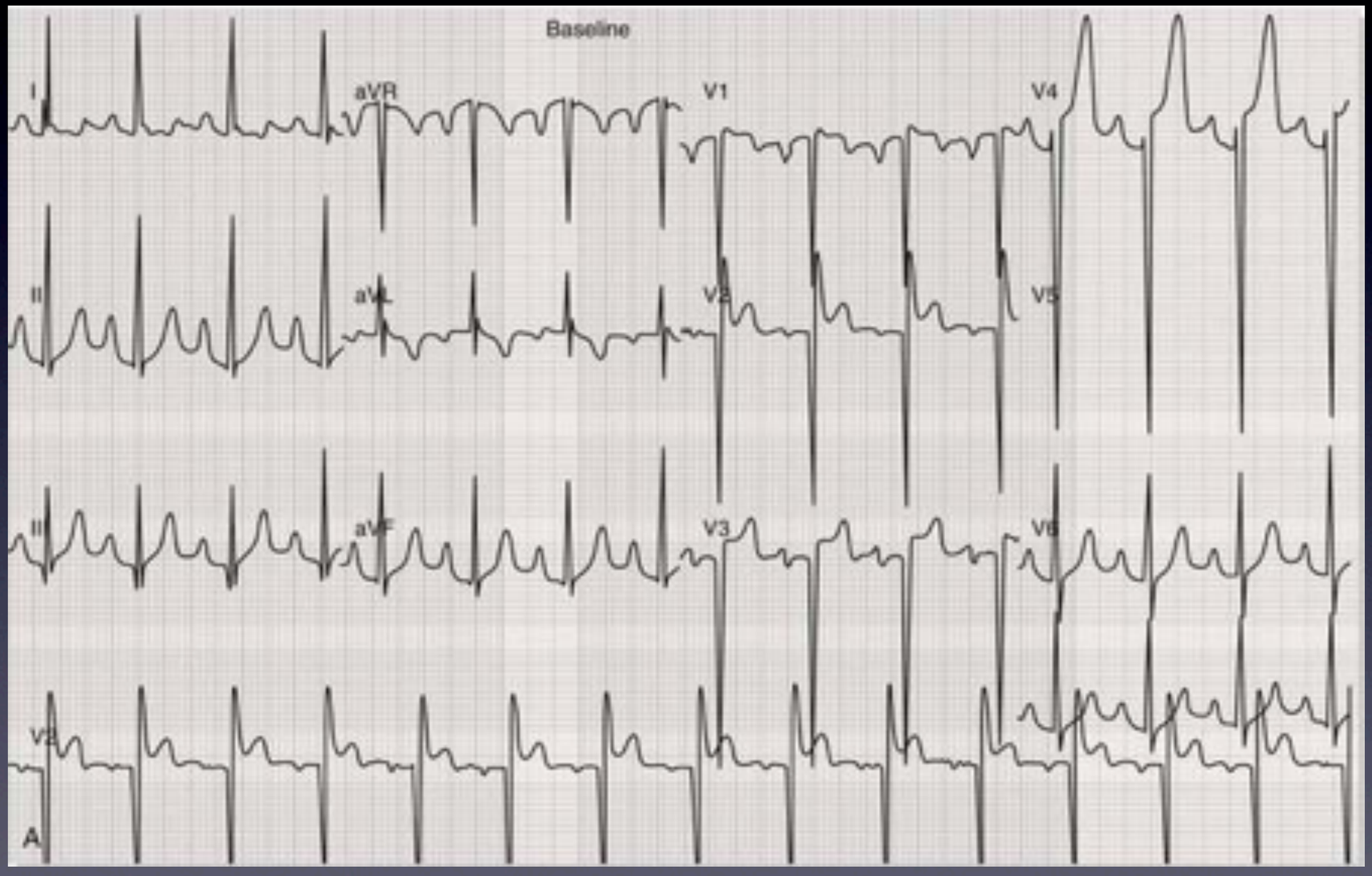
- idiopathic VF
- RBBB and ST elevation in anterior precordial leads
- No structural heart disease

- SCN5A sodium channel gene mutation
- young southeast asians
- 40-60% of all cases of idiopathic VF


Baseline
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
A

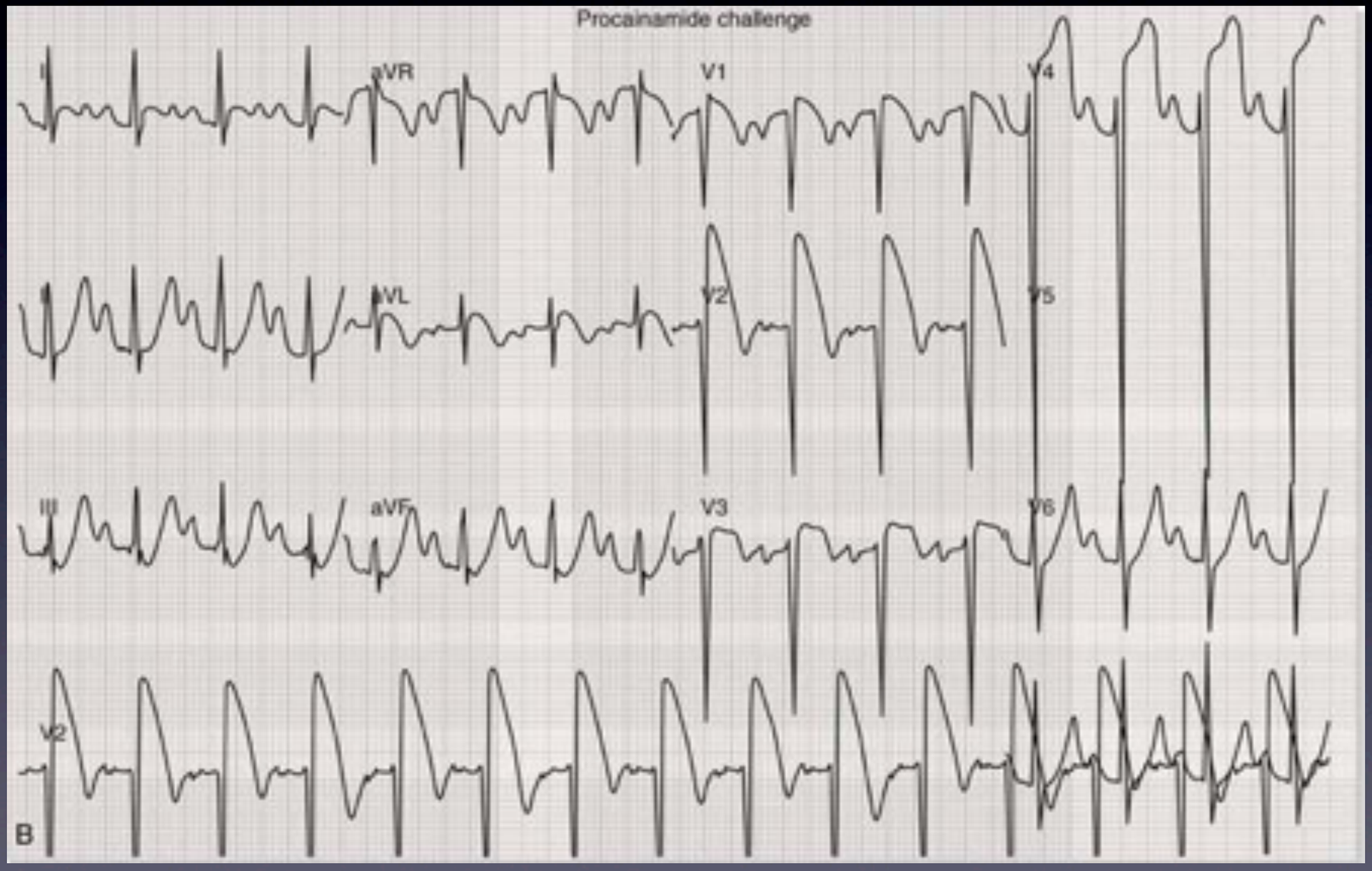
Type 2 brugada

RBBB



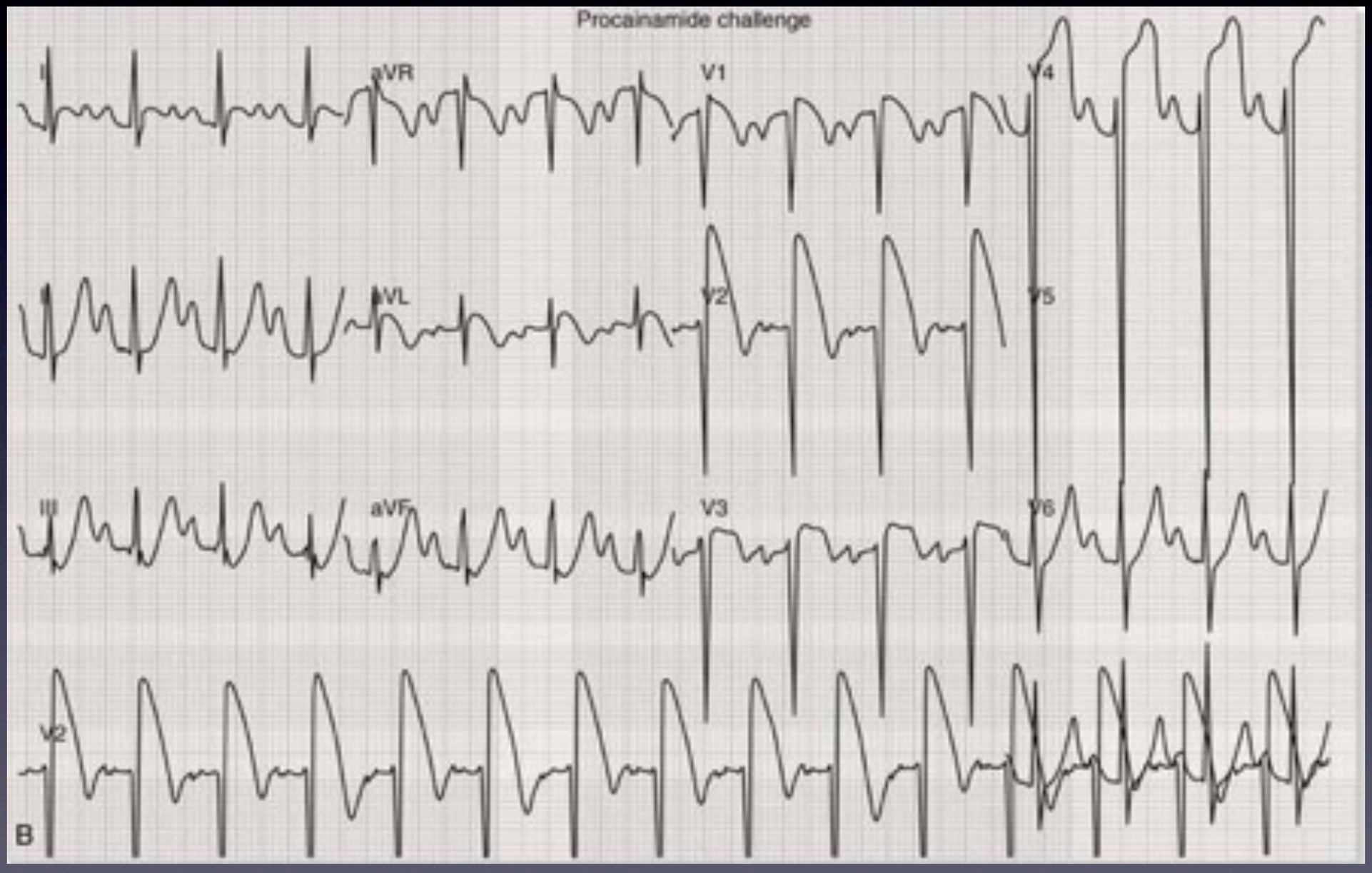
ST elevation in V1-V3

Saddleback ST elevation and bipasic T wave in V1


Procainamide challenge
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V2
B

Type I brugada pattern

coved ST elevation



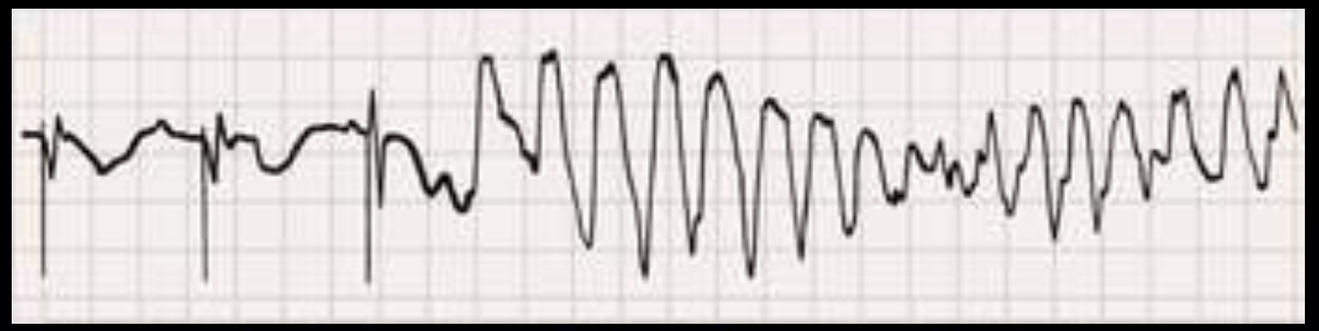
Negative t waves V1-V3

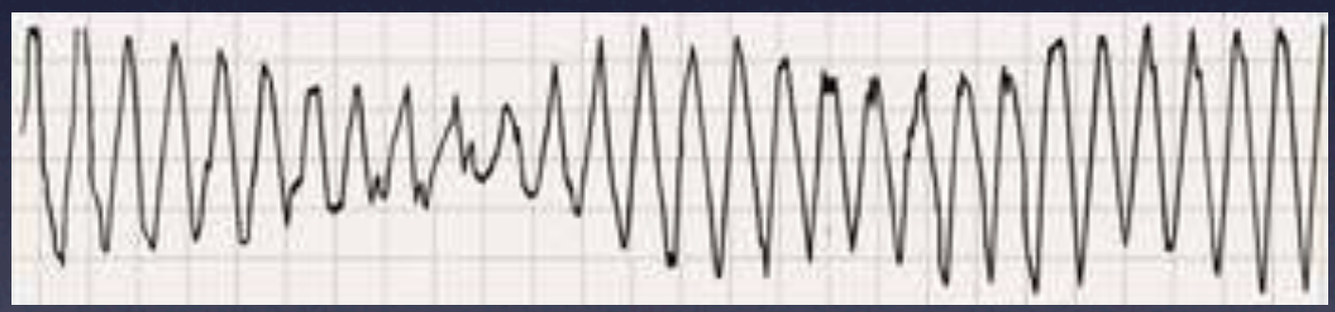
After procainamide challenge

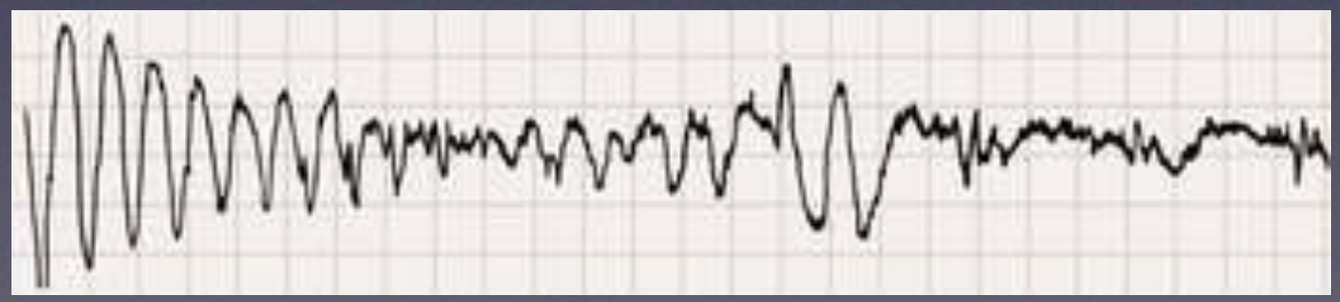
Torsades de Pointes

- QRS complexes of changing amplitude
- appears to twist around the isoelectric line
- rates of 200 to 250/min
- prolonged ventricular repolarization
- QT interval > 500 ms
- prominent u wave, may merge with the t wave

- the beat before the onset of torsades will typically have a long QT (e.g., after a PVC)
- long-short R-R cycle sequences commonly precedes the onset of torsades







congenital long QT

TU wave alternans

late premature complex

- short couple variant: malignant dz, high mortality rate, initiated with a close coupled PVC, usually does not involve preceding pause or bradycardia
- important to distinguish from polymorphic VT with a normal QT

Mechanism of Torsades

- early afterdeplolarization causes both long QT and torsades
- Perpetuation most likely due to transmural reentry
- Putative M cells in the midmyocardium, have prolonged repolarization and may play a role.

Who is prone?

- congenital long QT
- severe bradycardia
- K,Mg depletion (e.g, diarrhea)
- Class 1A or III anti-arrhythmics
- Drugs
- F > M due to longer QT interval

Rx

- IV magnesium followed by temporary atrial or ventricular pacing, isoproterenol can be used to increase the rate till pacing instituted
- Lidocaine
- Phenytoin
- Mexiletine
- Avoid Class 1A, IC and III agents

Long-QT Syndrome

- QTc ~ upper limit of normal is 440 msec
- Men ~ 460 ms +/- 15%
- Female ~ 470 ms +/- 15%
- probability of arrhtythmia is directly propotional to the length of the QTc
- t wave “humps” may suggest LQTS (caused by early afterdepolarizations)

Congenital LQTS

- Jervell and Lange-Nielsen syndrome: AD, associated sensorineural deafness
- Romano-Ward syndrome: AD, normal hearing

Acquired LQTS

- Drugs: quinidine, procainamide, sotalol, amiodarone, disopyramide, phenothiazines, tricyclics, erythromycin, pentamidine, some antimalarials, cisapride and probucol
- Hypokalemia and hypomagnesemia
- Liquid protein diet and starvation
- CNS lesions
- significant bradyarrythmias
- Cardiac ganglionitis
- mitral valve prolapse

- syncope (at times misdiagnosed as epilepsy)
- sudden death
- LQT1 ~ swimming, emotional stress
- LQT3 ~ sleep
- LQT2 ~ emotional stress or sudden loud noise (e.g, alarm clock)

Workup for suspected LQTS

- Stress Testing: can prolong the QT and produce t wave alternans
- ECG's of family members
- Prolonged ECG recording with various stresses (auditory, cold, exercise, psychological) to evoke VT
- Valsalva maneuver
- Catecholamine infusion
- PVC stimulation and EPS ~ not helpful
- often preceded by sinus tachycardia as compared to the aquired form (bradycardia or short-long RR)

Management of LQTS

- Beta-blockers at max tolerated dose
  - symptomatic
  - family hisory of early SCD
  - QTc >500 ms
- ICD
  - syncope or aborted SCD
  - BB + overdrive atrial pacing (prevent ICD discharge)
  - algorithms to prevent post-PVC pauses
- Left-sided cervicothoracic sympathetic ganglionectomy
  - syncope despite max drug therapy
- No competitive sports

Torsades in congenital long QT

- beta-blockers
- surgical sympathectomy
- Pacing
- ICD
- may obtain ECG from close relatives in borderline cases

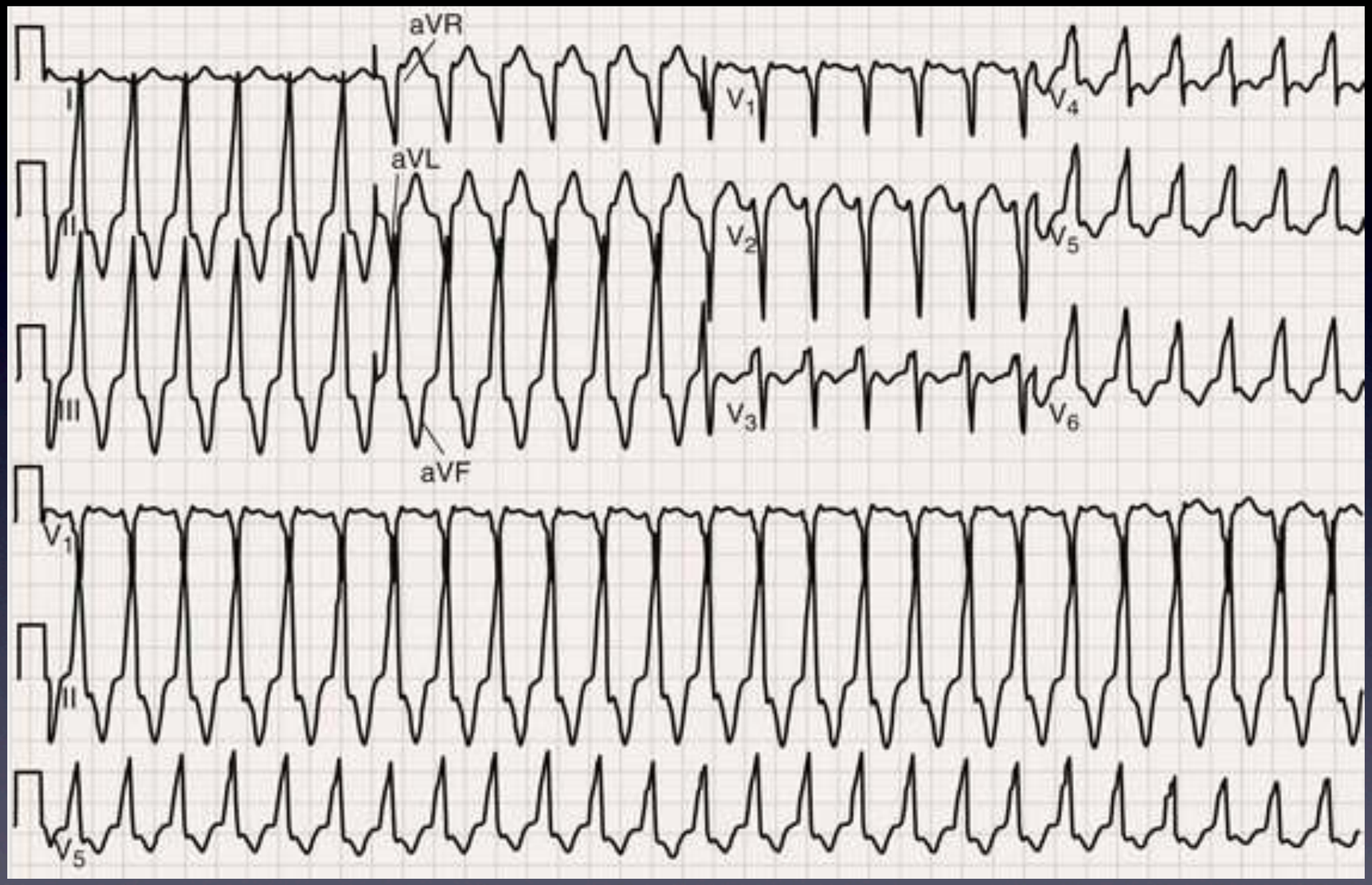
Short-QT Syndrome

- “idiopathic VF”
- prone to Afib
- gain-of-function mutations
- QT < 350 ms at HR < 100
- short QT persists, short/absent ST, tall and narrow T waves
- r/o Hyeprkalemia, hypercalcemia, hyperthermia acidosis and digitalis
- Rx ~ ICD
- Quinidine effective in HERG (KCNH2) gain of function mutation

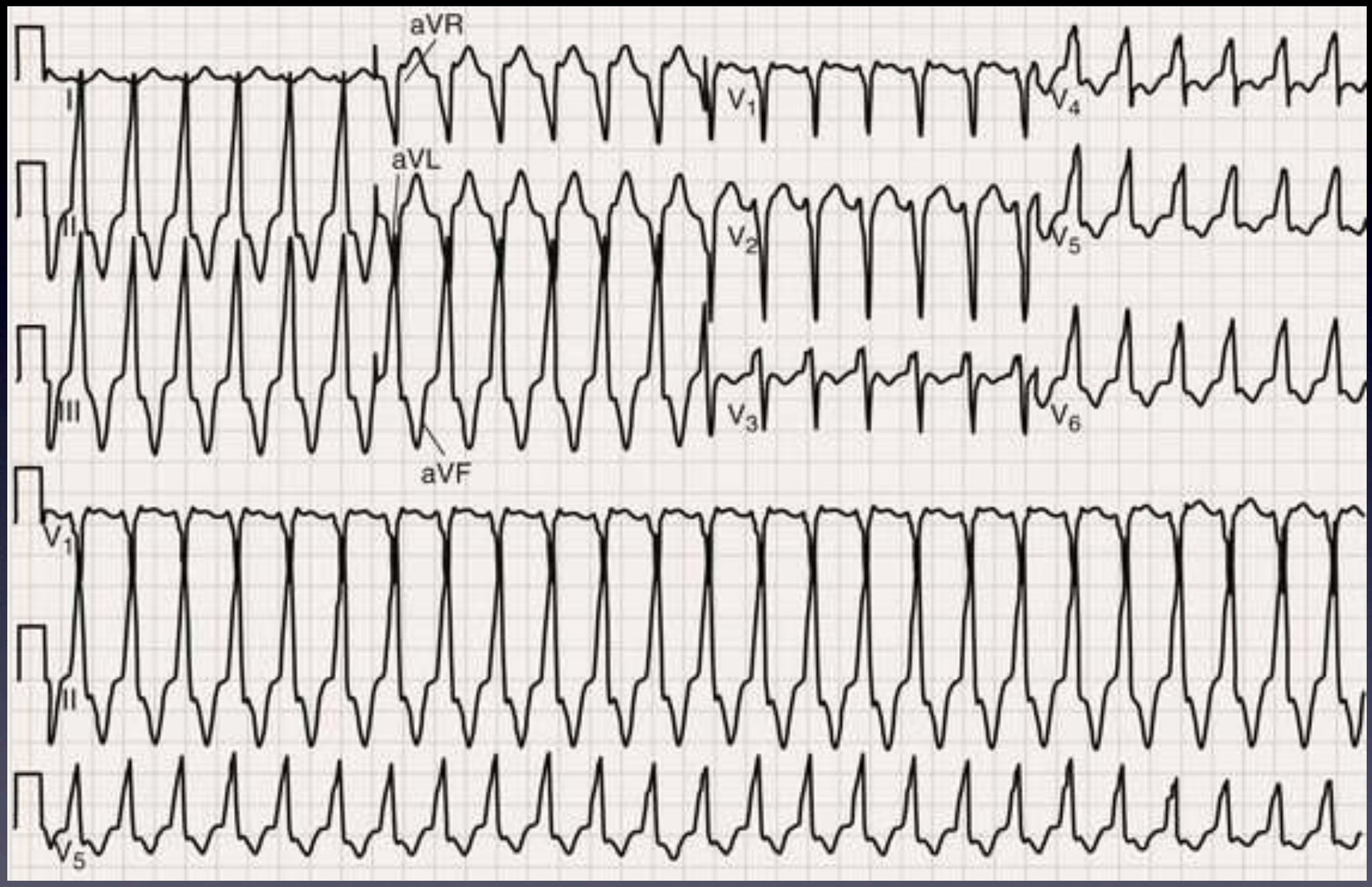
Idiopathic VT

- monomorphic VT in the absence of structural heart disease or CAD
  - Outflow tract
  - Annular
  - Fascicular

Outflow tract VT


aVR
I
V1
V4
aVL
II
V2
V5
III
V3
V6
aVF
V1
II
V5

LBBB pattern in V1

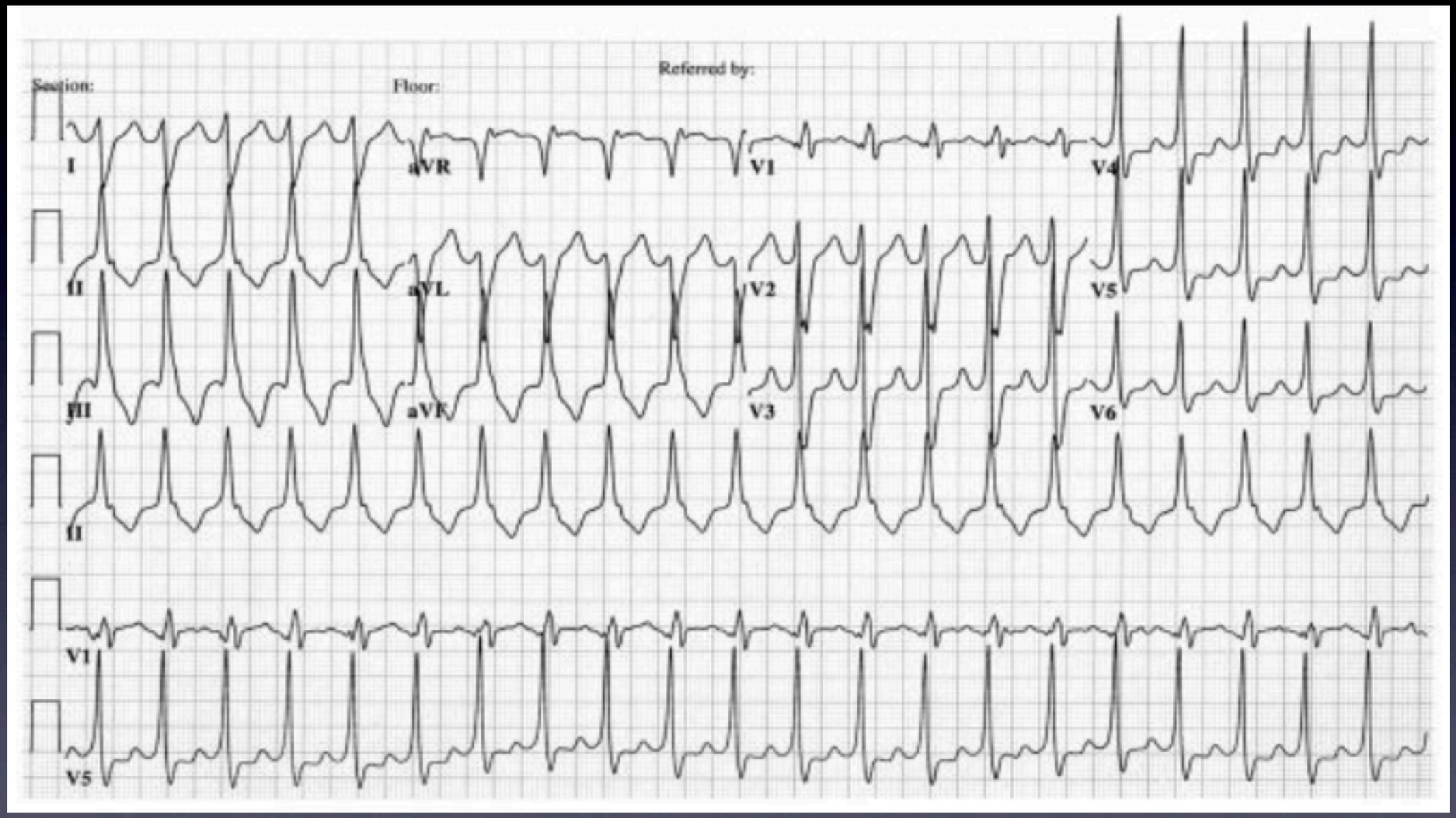


Inferior axis

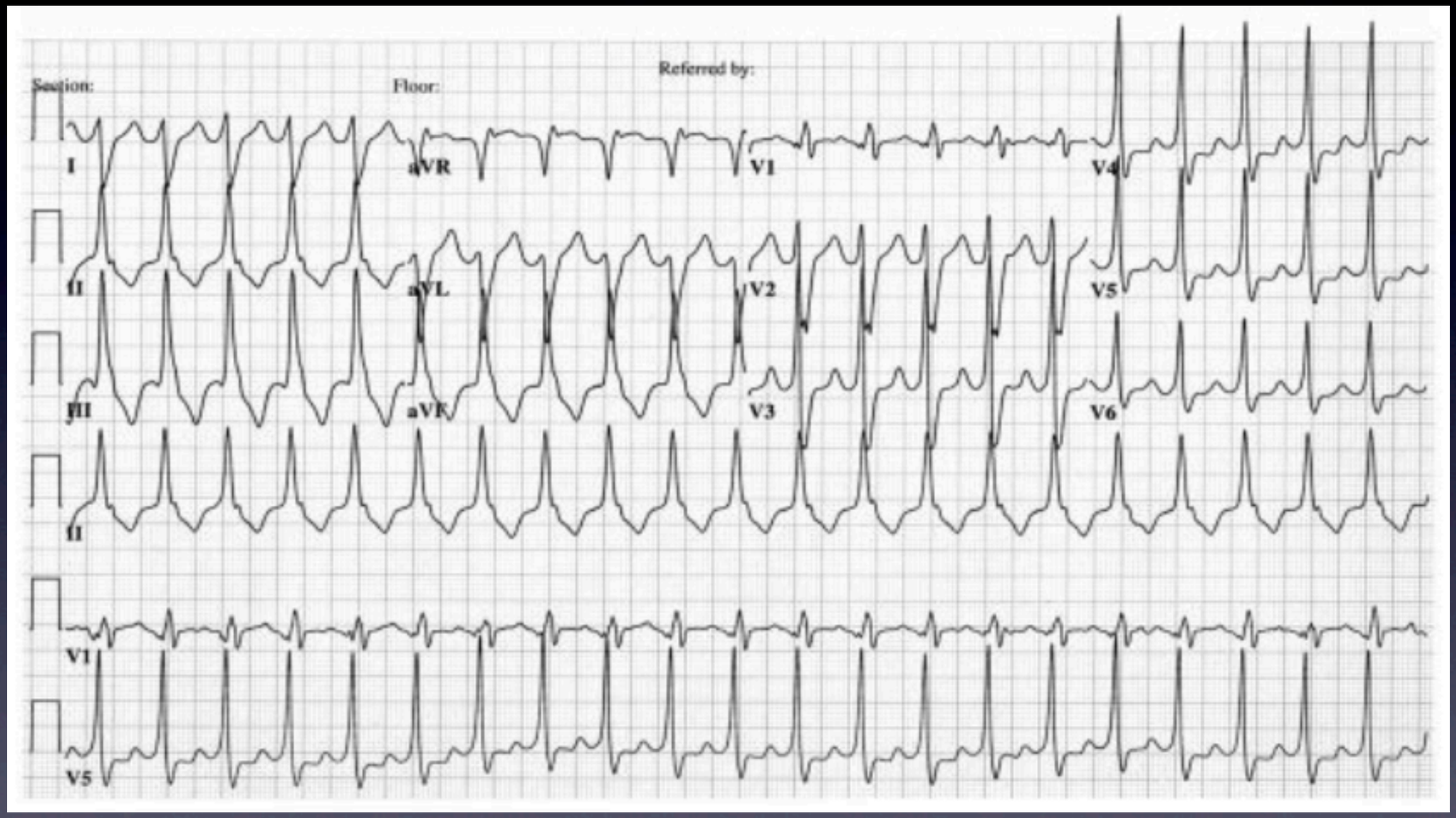
RVOT VT

RVOT VT

- LBBB in V1 + Inferior Axis
- vagal maneuvers, adenosine ~ terminate VT
- exercise, stress, isoproterenol, rapid or premature stimulation ~ initiate VT
- BB and verapamil can suppress the VT
- early or delayed after depolarizations ~ cAMP triggered activity
- Paroxysmal form ~ exercise or stress induced
- Repetitive monomorphic type ~ occurs at rest, ppt by transient increase in sympathetic activity unrelated to exertion
- Occasionally arises from RV inflow or RV apex


Section:
Floor:
Referred by:
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
II
V1
V5

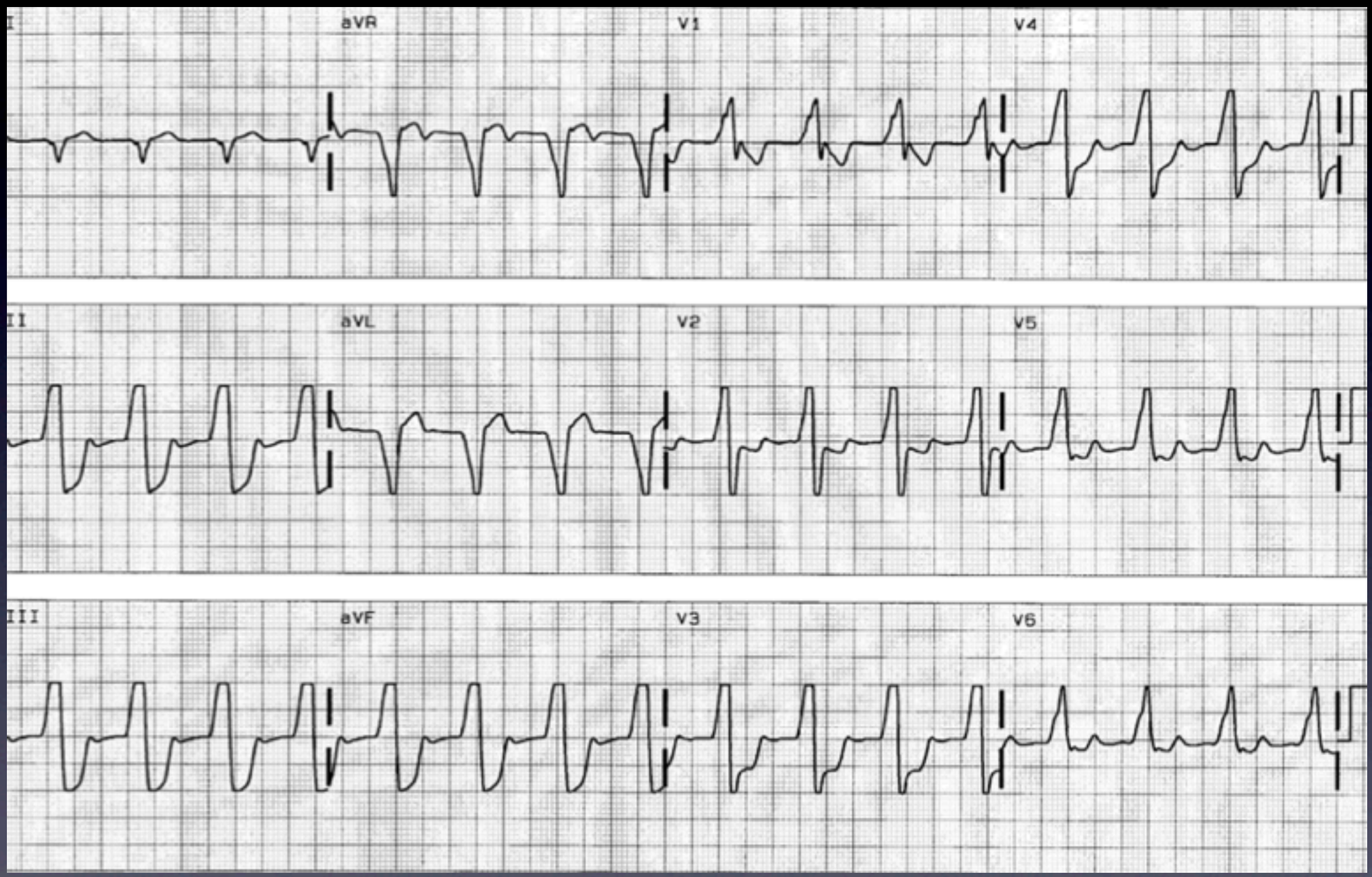
s wave in V1 and early R wave transition



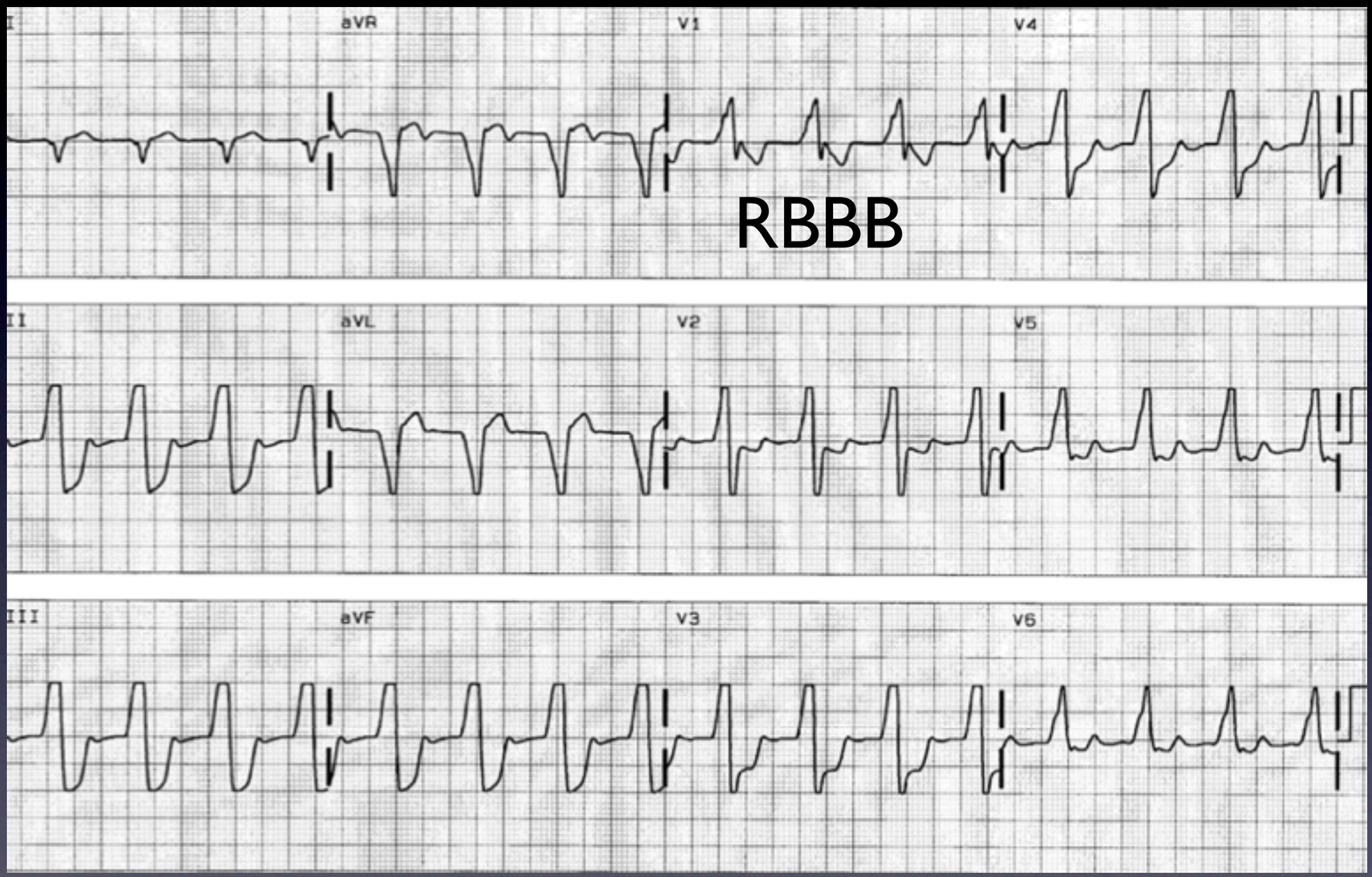
LVOT VT

LVOT VT

- s wave in V I
- early precordial R wave transition (V1-V2)
- Good prognosis
- Rx: ablation or AAD


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

monophasic R or Rs in V2 to V6



RBBB

mitral annular VT

S in V6

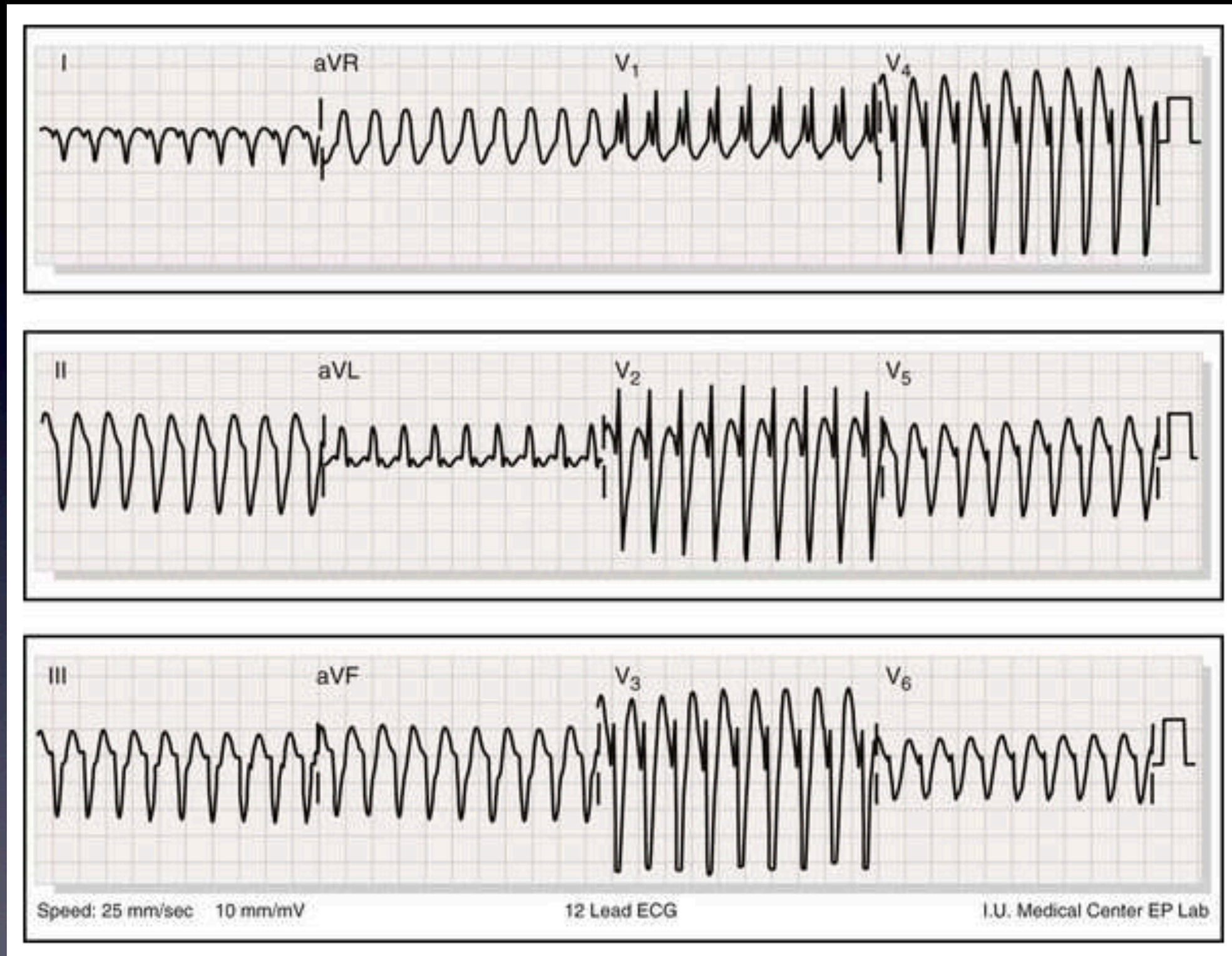
Annular VT

- Mitral or tricuspid
- ~ 4 to 7% of idiopathic VT
- tricuspsid VT: LBBB (Qs in V1), early precordial transition (V3) and narrower QRS
- similar to ouflow tract VT in prognosis and drug response
- amenable to ablation

Fascicular VT

Rightward axis

RBBB



Left Septal VT

- a.k.a left septal VT or fascicular tachycardia
- arises in the left posterior septum
- re-entry ~ entrainment demonstrated in some
- verapamil (iv better) or diltiazem sensitive
- adenosine resistant
- started by rapid pacing, exercise or isoproterenol
- good prognosis
- ablation is effective

Bundle branch

reentrant VT

- correctly timed antegrade right bundle and retrograde left bundle reentry
- started after a critical S2-H2 or S3-H3 delay
- the H-V interval of the BB reentrant complex equals or exceeds that of a normally conducted complex
- seen in dilated CMP or structural heart disease
- Pace termination effective
- Catheter ablation is effective

Thank You