EKG REVIEW

Asif Serajian, DO FACC

No disclosures

87 y/o female with palpitations

Patient has palpitations

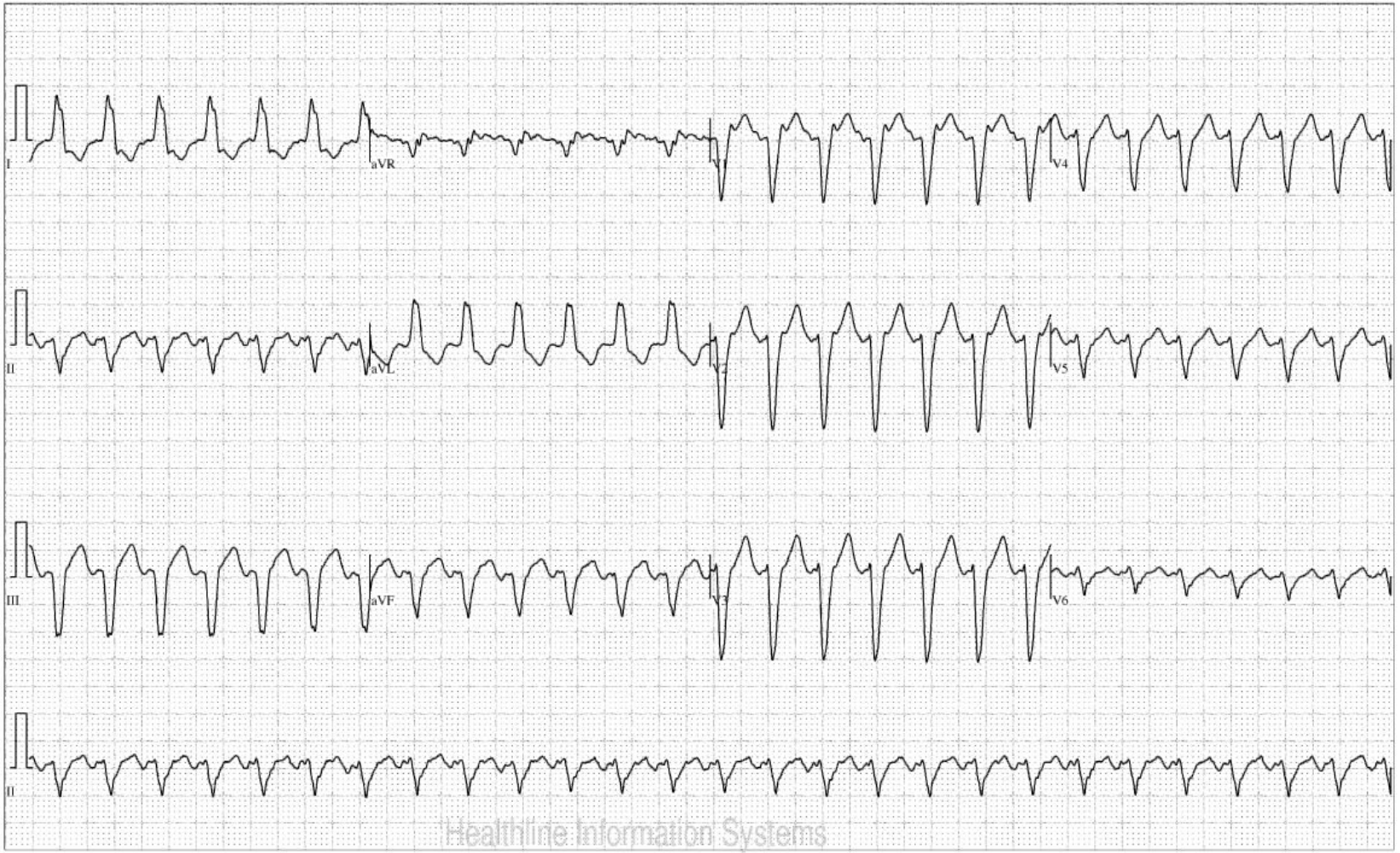
No chest pain

No syncope

Heart rate 160

BP is stable

Patient without distress



Speed:25 mm/sec Gain:10 mm/mv MYO:ON AC:ON DRIFT:ON Midmark Diagnostics Group Page 1 of 1 Version 8.6.0 ECG Analysis Ver. 8.6.0 Print Date 01/19/17 17:49:34

What is differential diagnosis?

How would you treat this patient?

Crit Care Med. 2009 Sep;37(9):2512-8. doi: 10.1097/CCM.0b013e3181a93661.

Adenosine for wide-complex tachycardia: efficacy and safety.

Marill KA[1], Wolfram S, Desouza IS, Nishijima DK, Kay D, Setnik GS, Stair TO, Ellinor PT.

Author information

Abstract

OBJECTIVES: : To determine whether adenosine is useful and safe as a diagnostic and therapeutic agent for patients with undifferentiated wide QRS complex tachycardia. The etiology of sustained monomorphic wide QRS complex tachycardia is often uncertain acutely.

DESIGN: : A retrospective observational study.

SETTING: : Treatment associated with emergency visits at nine urban hospitals.

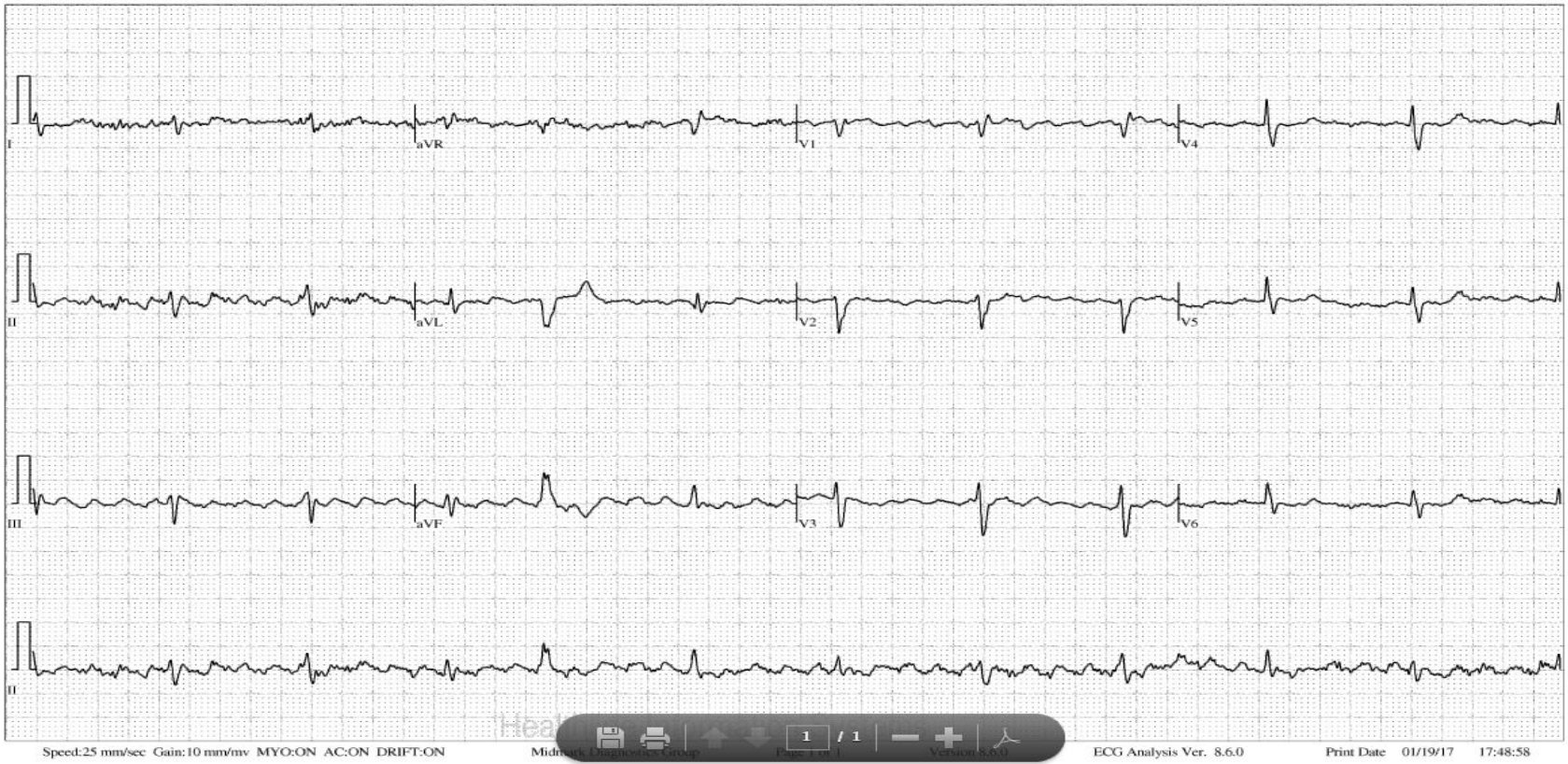
PATIENTS: : Consecutive patients treated with adenosine for regular wide QRS complex tachycardia between 1991 and 2006.

INTERVENTIONS: : Treatment with adenosine infusion.

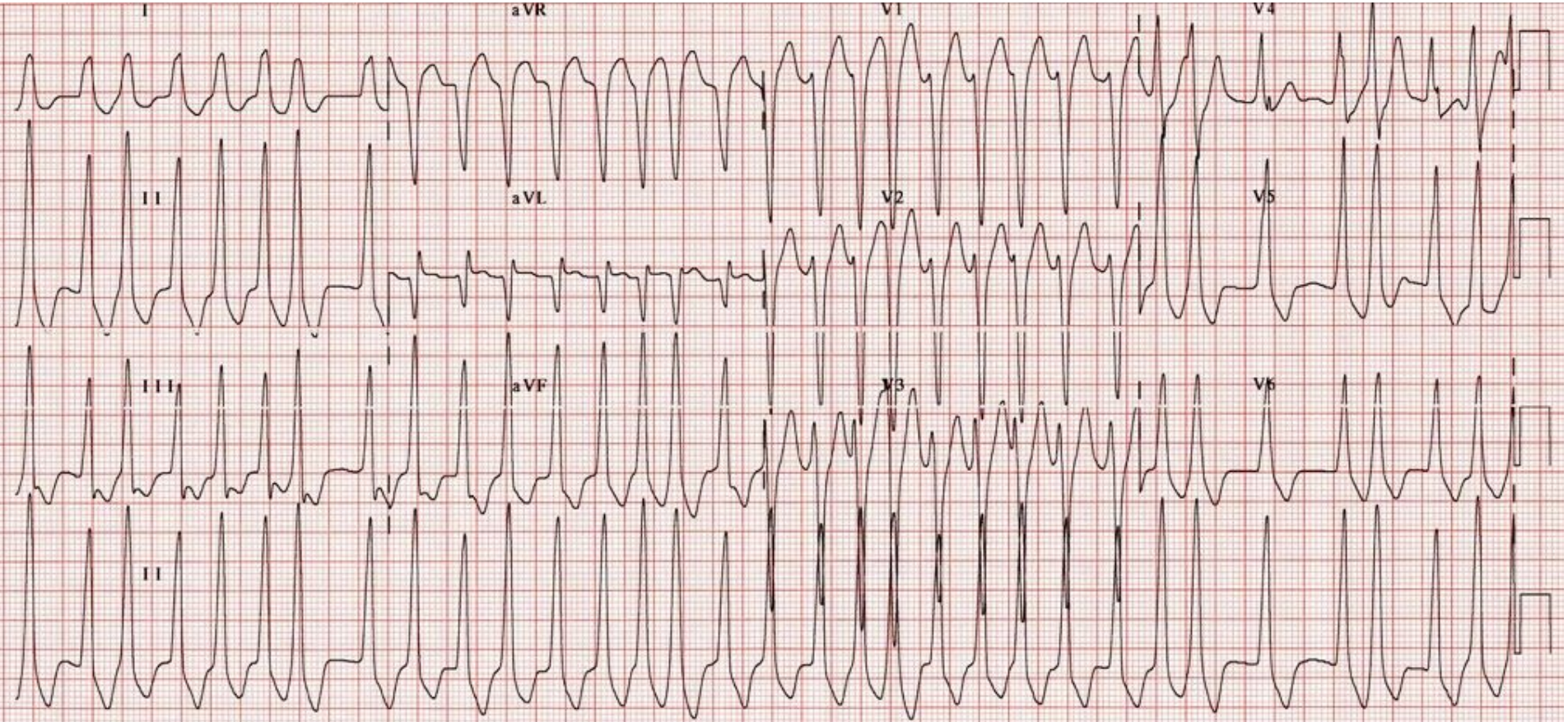
MEASUREMENTS AND MAIN RESULTS: : Measured outcomes included rhythm response to adenosine, if any, and all adverse effects. A positive response was defined as an observed change in rhythm including temporary atrioventricular conduction block or tachycardia termination. A primary adverse event was defined as emergent electrical or medical therapy instituted in response to an adverse adenosine effect. A rhythm diagnosis was made in each case. The characteristics of adenosine administration as a test for a supraventricular as opposed to ventricular tachycardia were determined, and the adverse event rates were calculated. A total of 197 patients were included: 104 (90%) of 116 (95% confidence interval, 83%-95%) and two (2%) of 81 (95% confidence interval, 0.3%-9%) supraventricular tachycardia and ventricular tachycardia patients demonstrated a response to adenosine, respectively. The odds of supraventricular tachycardia increased by a factor of 36 (95% confidence interval, 9-143) after a positive response to adenosine. The odds of ventricular tachycardia increased by a factor of 9 (95% confidence interval, 6-16) when there was no response to adenosine. The rate of primary adverse events for patients with supraventricular tachycardia and ventricular tachycardia was 0 (0%) of 116 (95% confidence interval, 0%-3%) and 0 (0%) of 81 (95% confidence interval, 0%-4%), respectively.

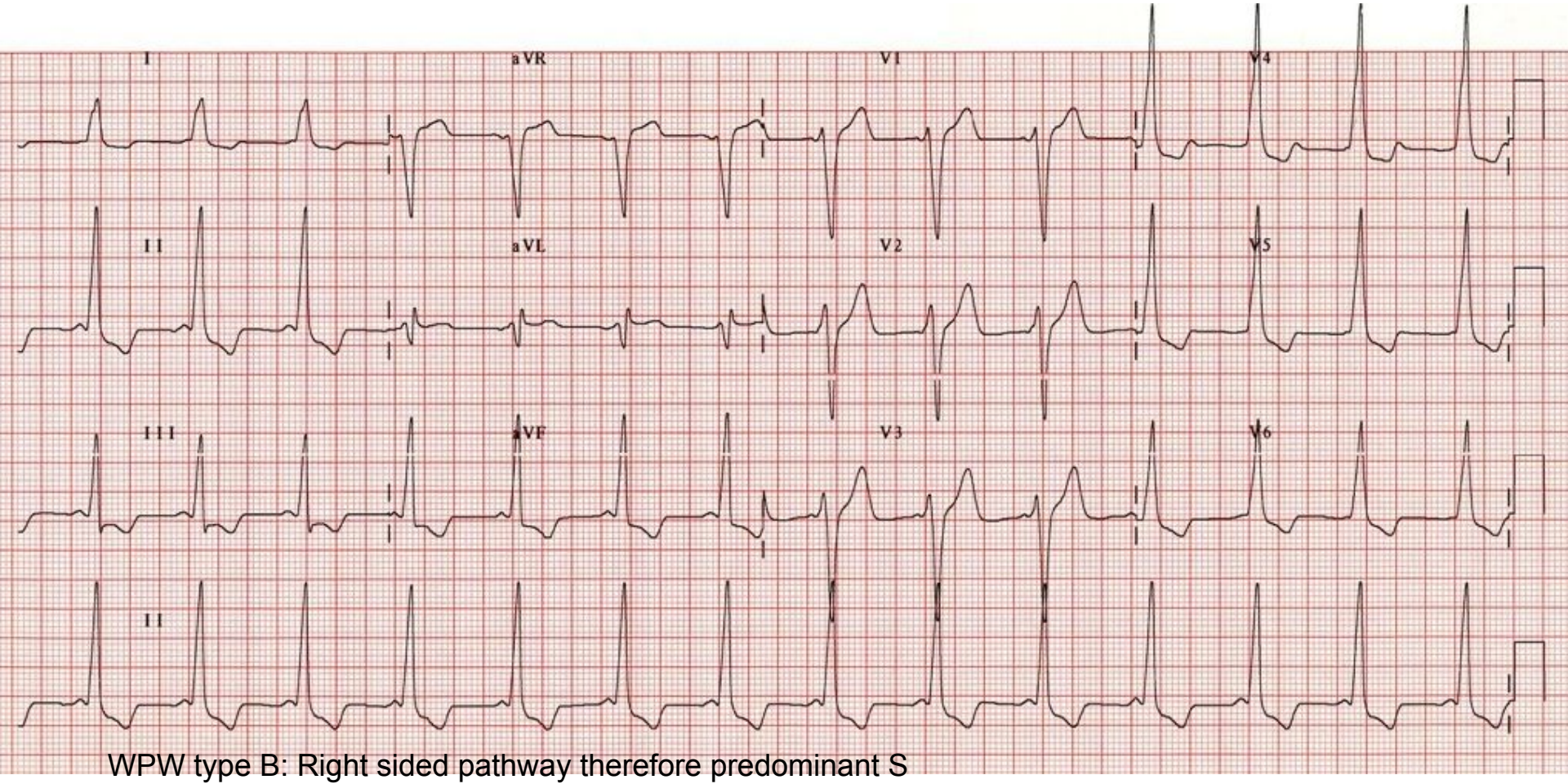
CONCLUSIONS: : Adenosine is useful and safe as a diagnostic and therapeutic agent for patients with regular wide QRS complex tachycardia.

Subsequent ECG

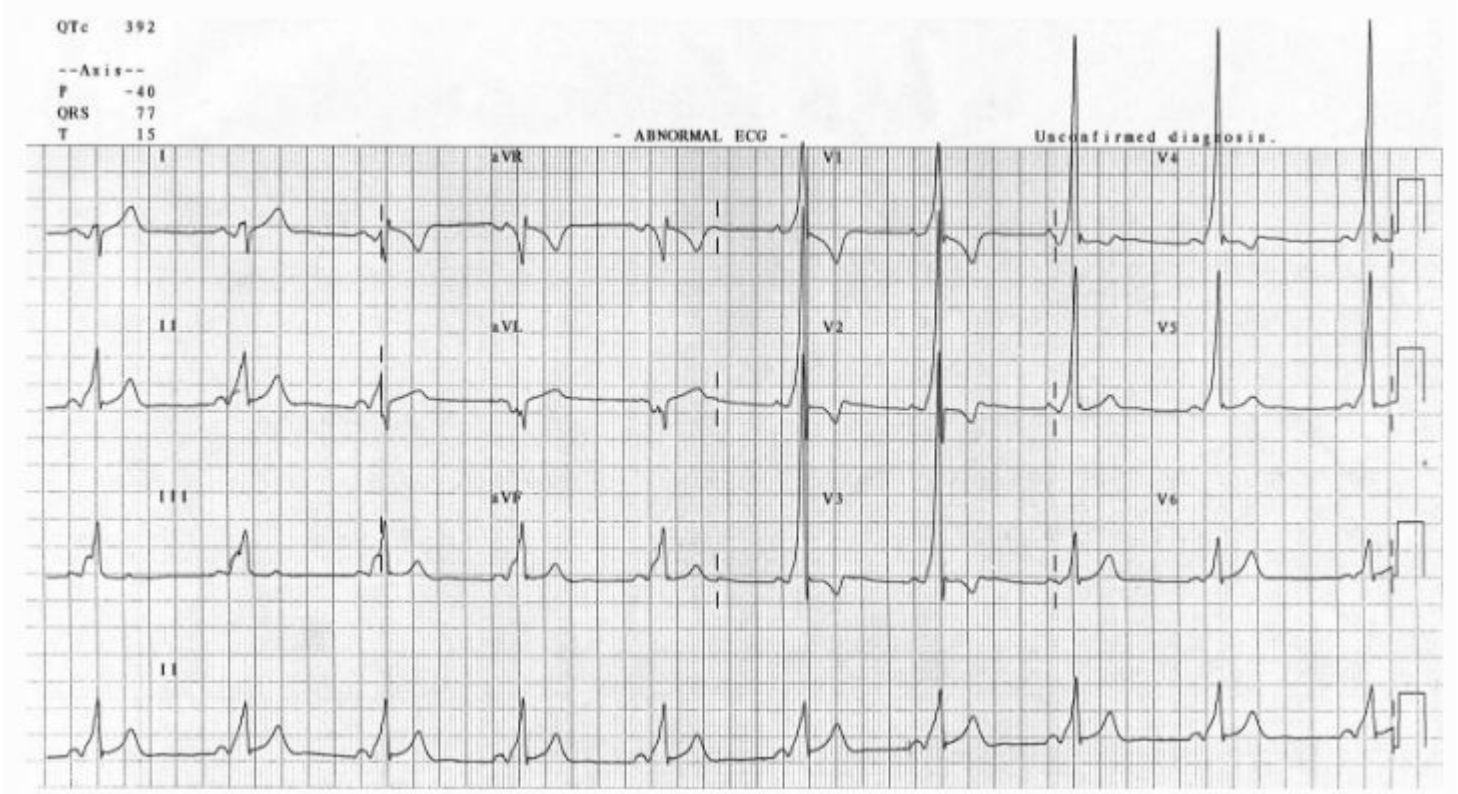


What about adenosine for this patient

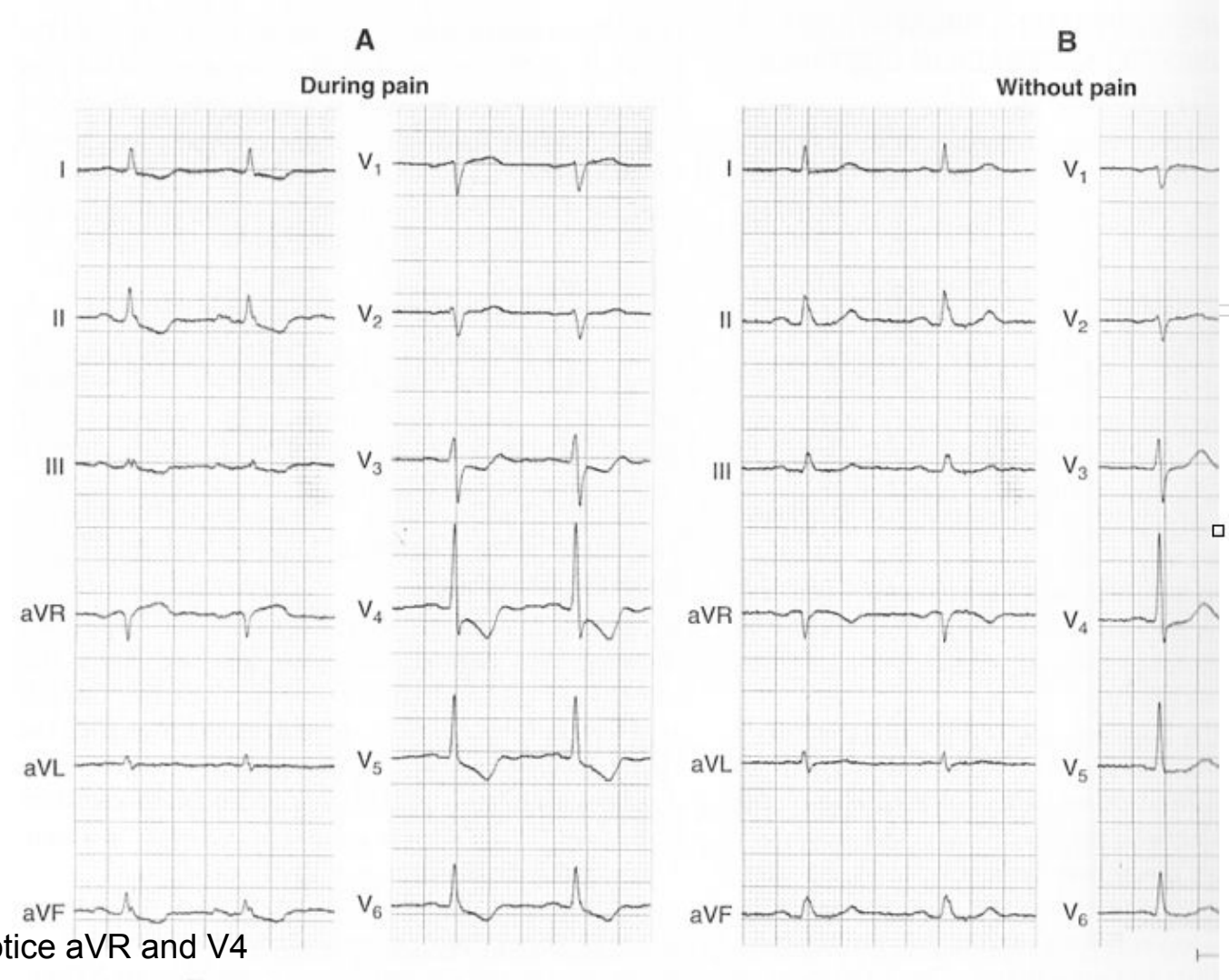




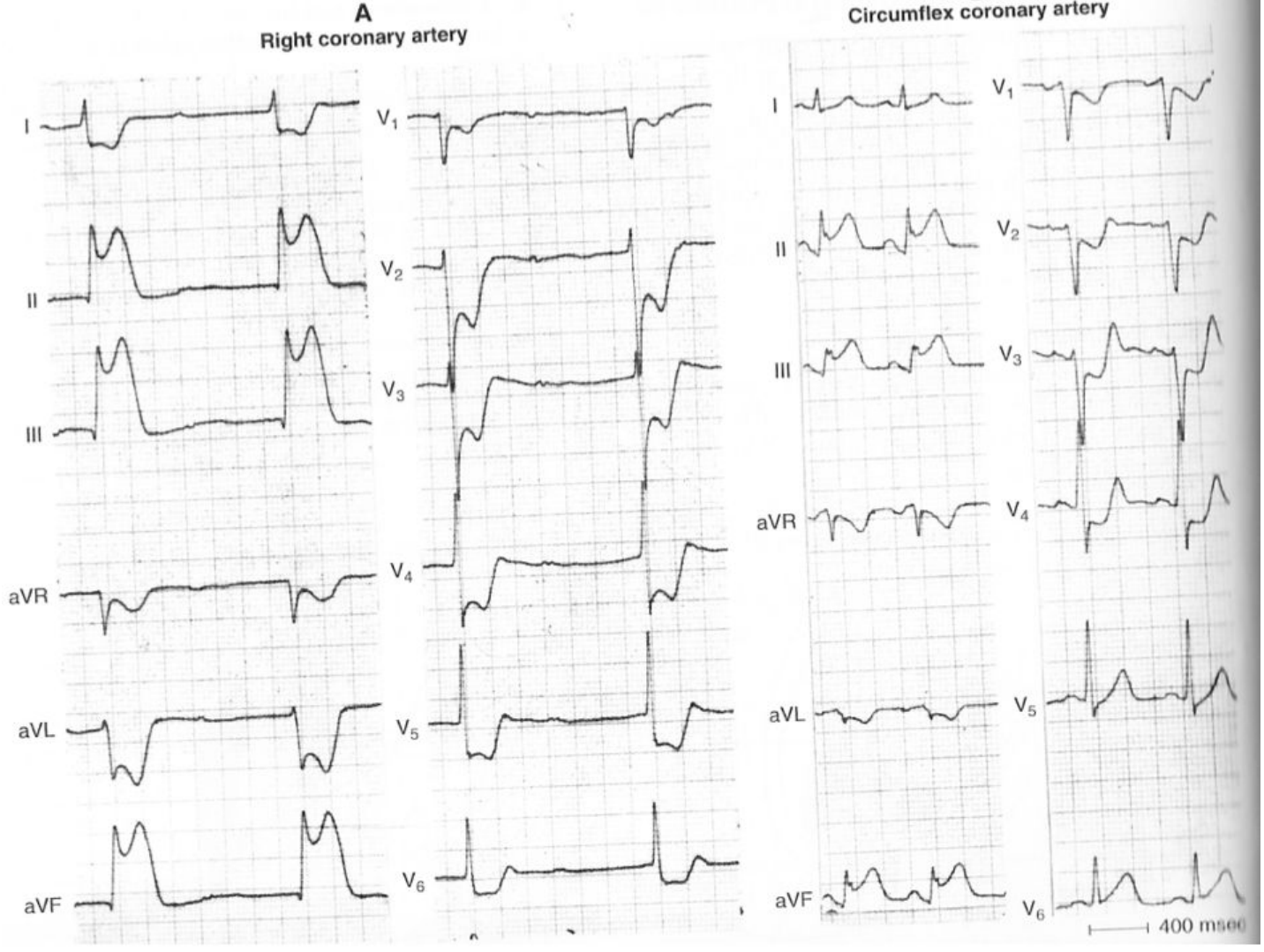
WPW type B: Right sided pathway therefore predominant S wave in V1, short PR (< 120 ms) [B right!]

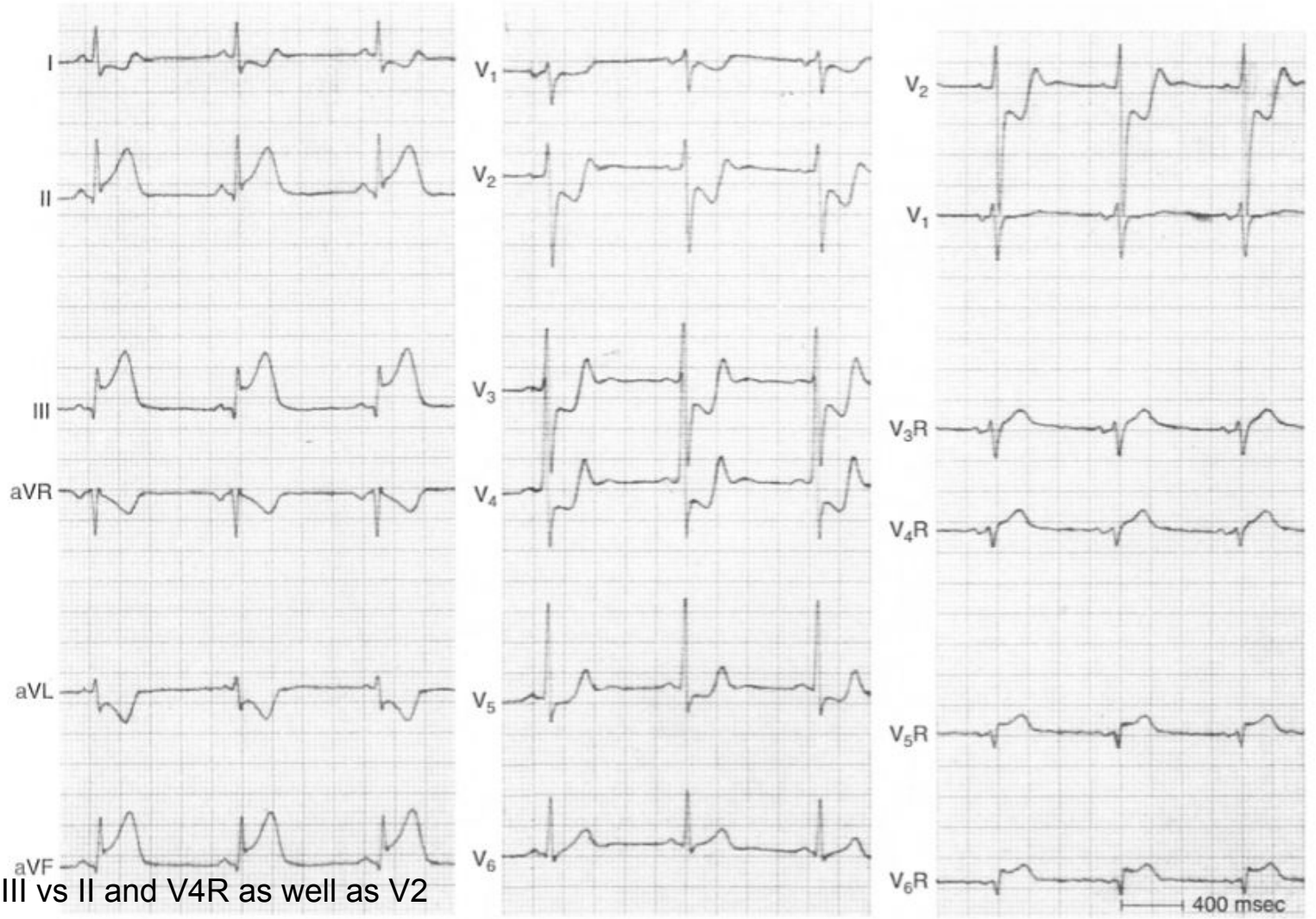


WPW type A: Prominent R wave in V1 so it is left sided pathway



Notice aVR and V4


A
Right coronary artery
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
Circumflex coronary artery
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
400 msec



Notice III vs II and V4R as well as V2

ST elevation MI

2 contiguous leads

> 2 mm ST elevation in V1 V2 V3

> 1 mm ST elevation in other leads

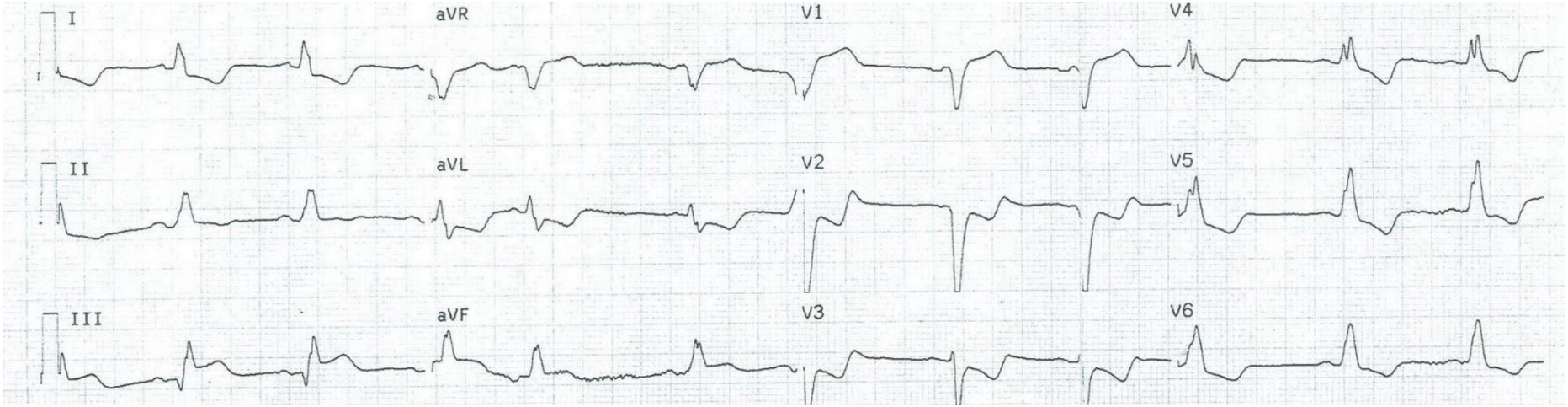
Other causes of ST elevation: ventricular aneurysm (note Q waves), LVH, BBB, hyperkalemia*, pericarditis, myocarditis, early repolarization

STEMI with pre-existing LBBB

LBBB alters myocardial depolarization and repolarization and therefore it creates secondary ST-T changes that are discordant to the QRS complex

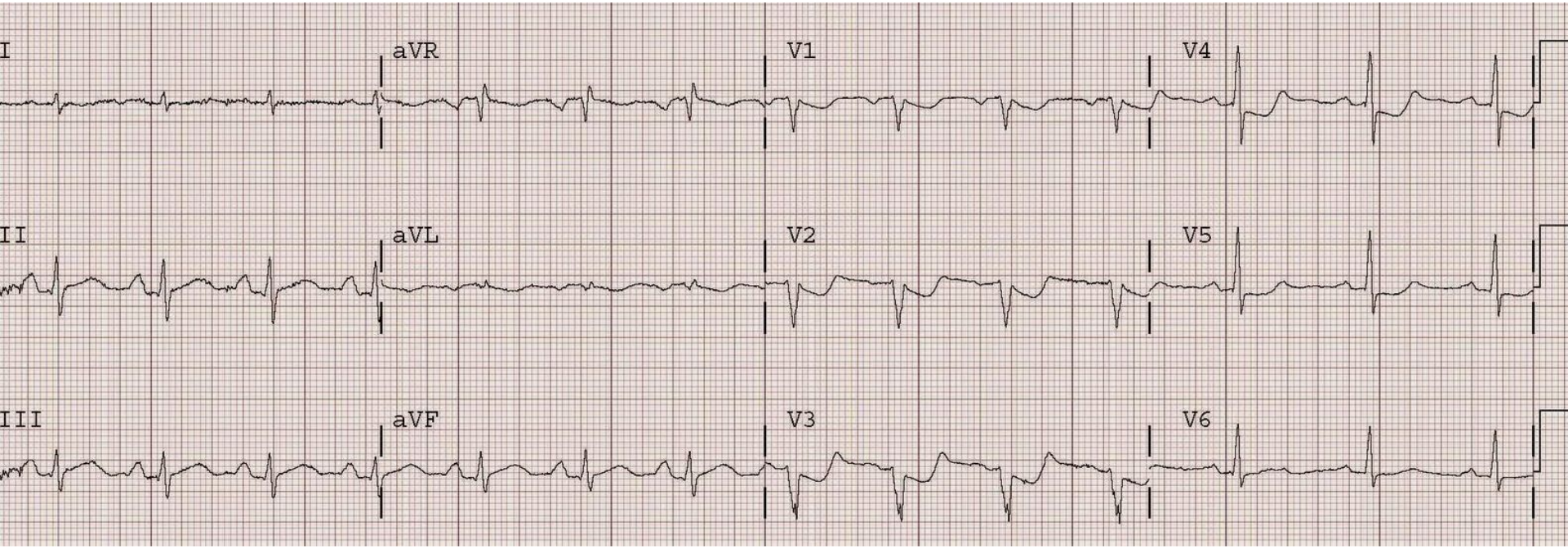
RBBB only alters V1 and V2 repolarization

ST elevation myocardial infarction can be diagnosed with RBBB however with LBBB any concordant ST elevation or 5 mm discordant ST elevation is relevant

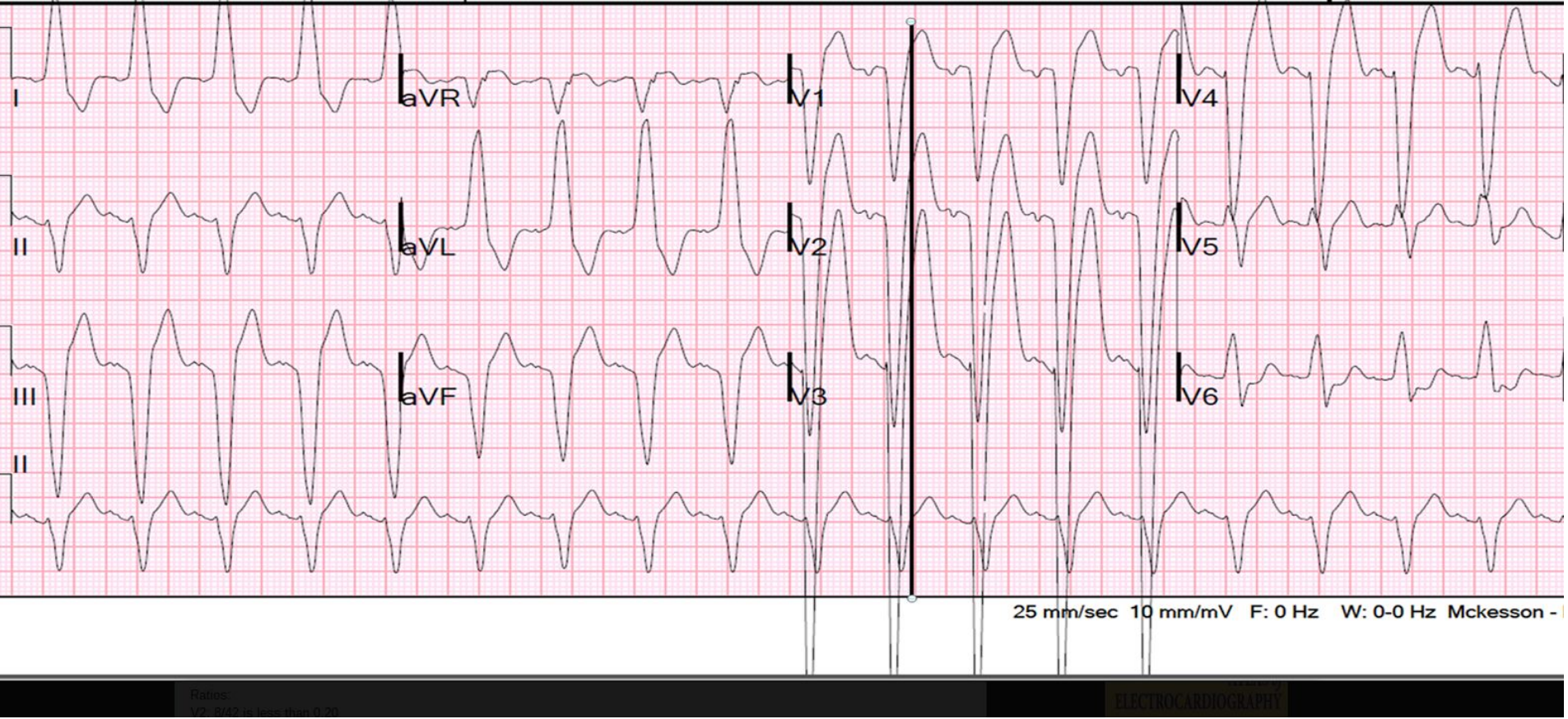


Concordant ST elevation in the inferior leads

True Posterior MI

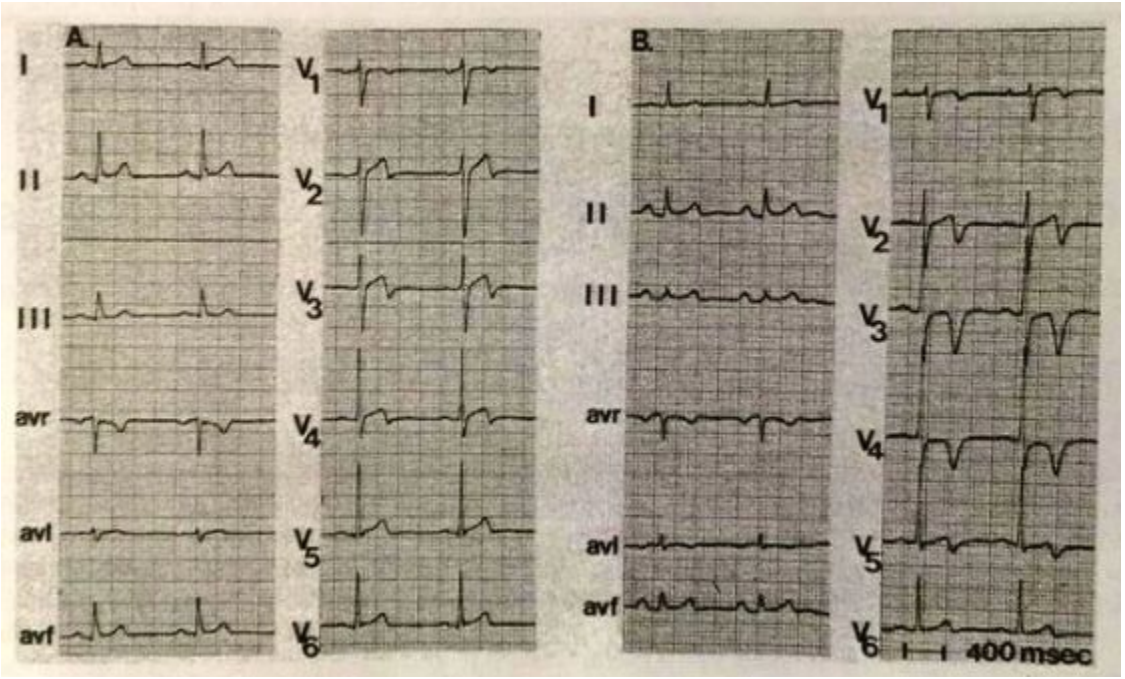


May see a dominant R wave (mirror image of Q wave)
STdepression (mirror image of ST elevation)

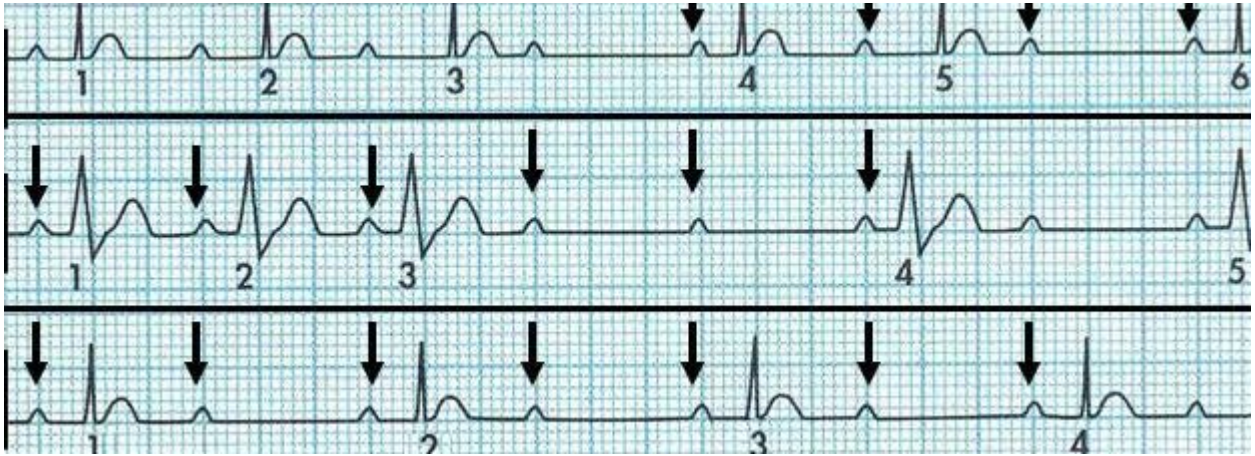


Criteria for STEMI is > 5 mm ST elevation if discordant to QRS
> 1 mm ST elevation if concordant

2 types of Wellen's pattern



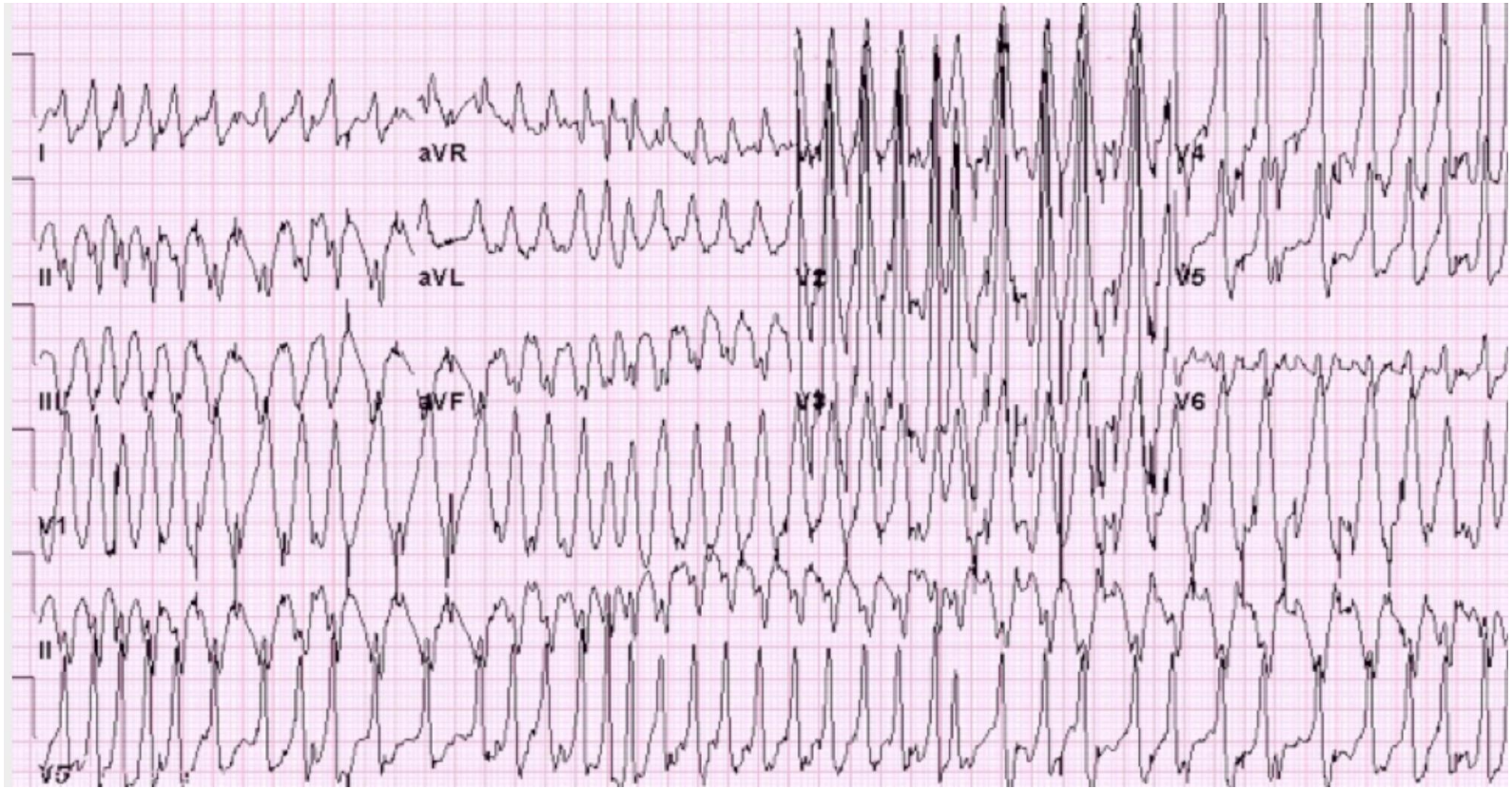
Type 2 Blocks categorized into 3 types



1st strip: Mobitz 1. PR lengthens with dropped QRS. Block higher at the AV node, has narrow complex. Associated with inferior MI. Look for group beating due to shortening of RR interval. Non-conducted P wave is < 2 RR.
2nd strip: Mobitz 2. PR unchanged with dropped QRS. Block is in His and may have wide complex. Associated with anterior MI. ~~atropine~~
3rd strip: 2:1. Could be Mobitz 1 or 2. Need to find other strips with lengthening PR to determine

Classic Wenckebach pattern may not be seen if there is sinus arrhythmia also present related to changes in autonomic tone

However, look for prolongation of the PR interval



WPW with atrial fibrillation. Do not treat with adenosine! Use procainamide

WPW pattern

PR < 0.12 seconds

Slurred PR segment

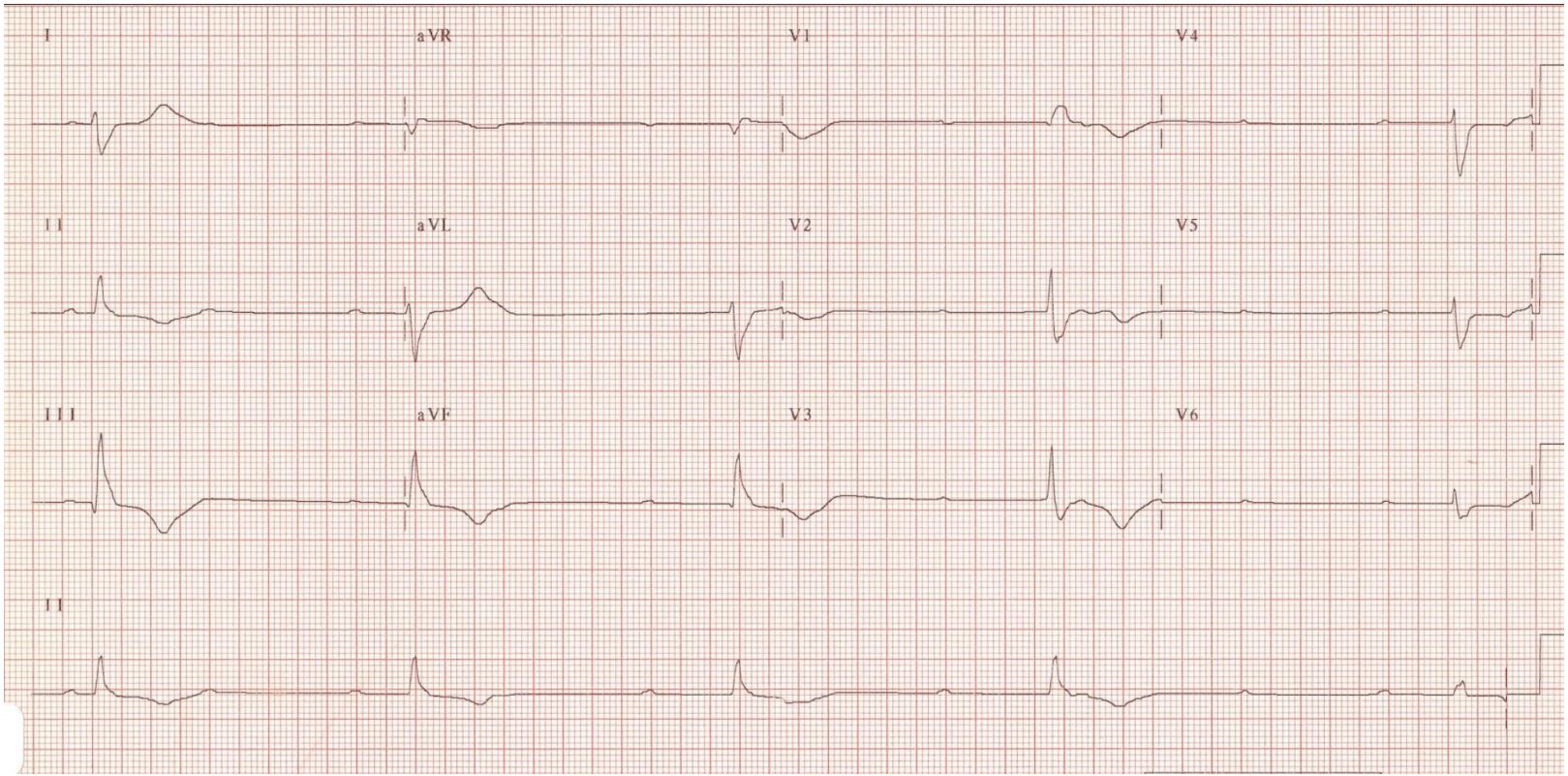
Secondary ST-T changes

QRS duration is > 0.10 seconds

Complete heart block

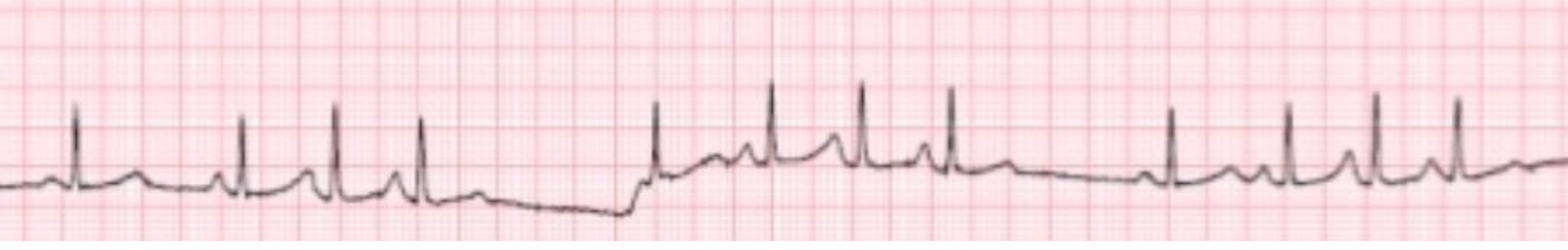
Sinus rate higher than escape rate

If block at the AV junctional escape rhythm is narrow complex junctional rhythm at 40-60

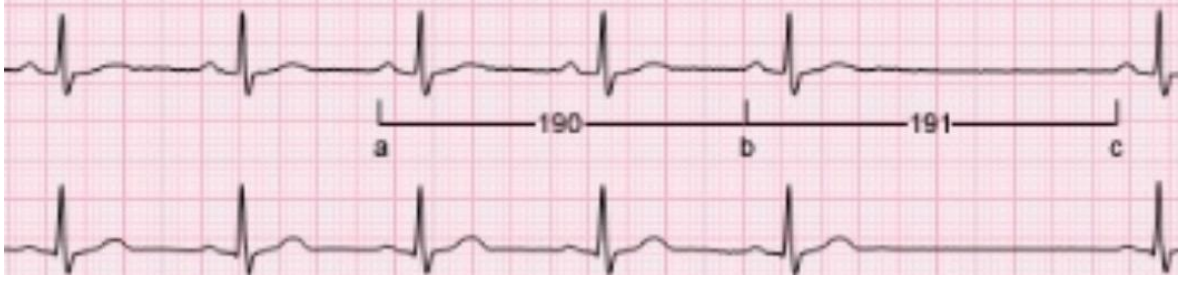

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

Sinoatrial exit block types

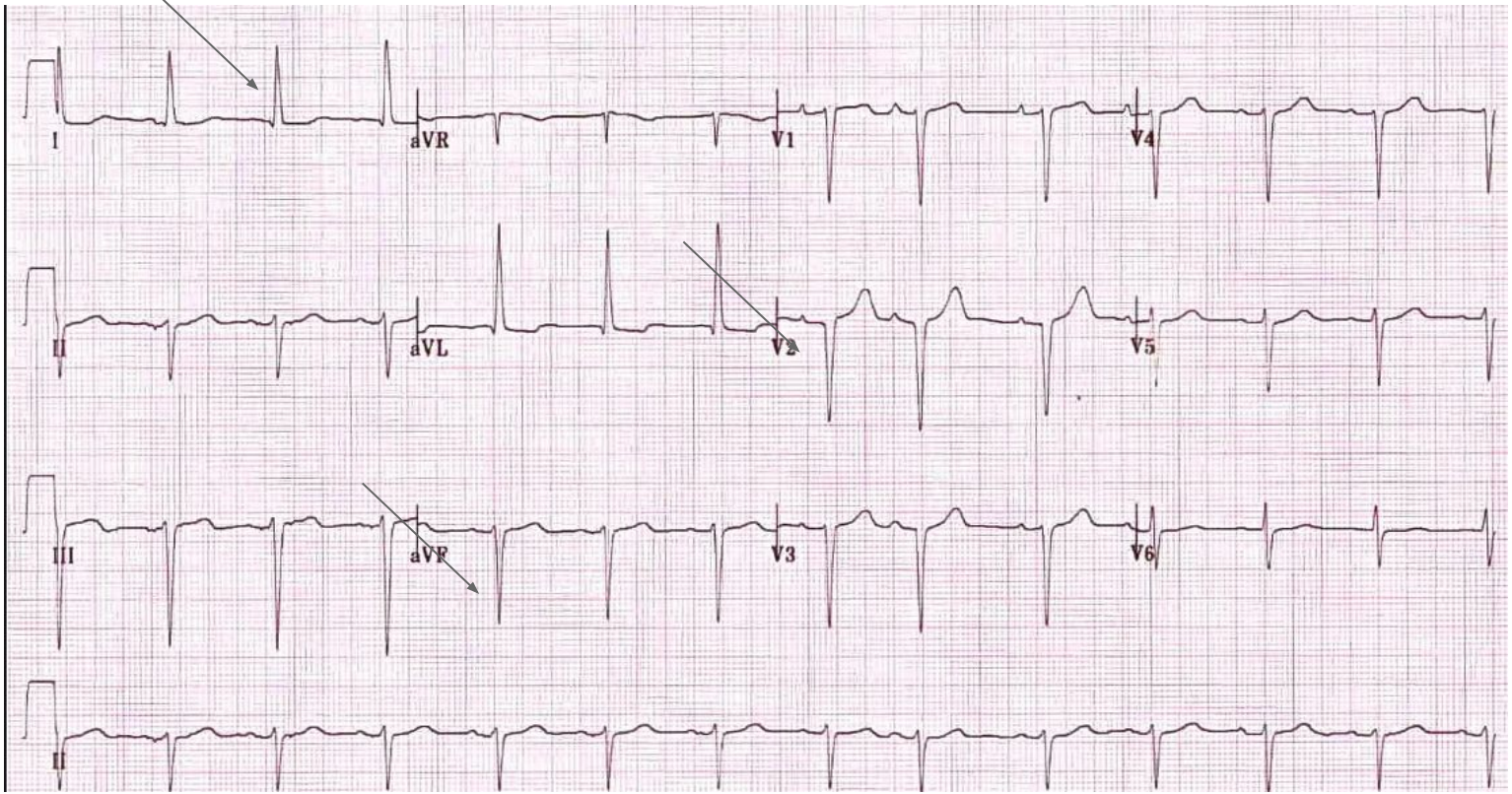
1: Shortening of P-P interval until no P. Grouped beating.



2: No shortening of P-P interval, pause is 2x P-P.



Left anterior fascicular block



LAFB

Left axis deviation over 45 degrees

May mimic anterior and inferior infarction

rS pattern in aVF

qR pattern in I

Slight prolongation of QRS interval

May mask an inferior myocardial infarction

LPFB

RAD +100 to +180

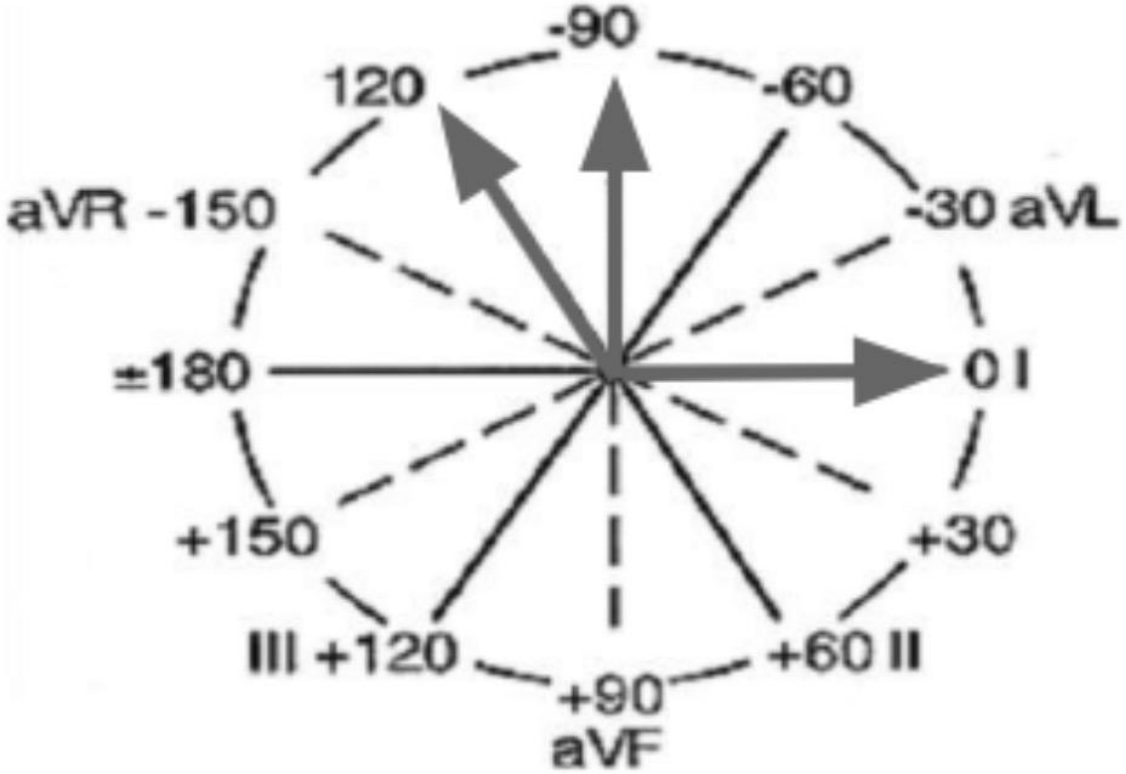
Slight prolongation of the QRS

Diagnosis of exclusion if no other cause of RAD such as RVH, pulmonary embolism, emphysema

Most common cause of this is coronary artery disease

May mask a lateral infarction

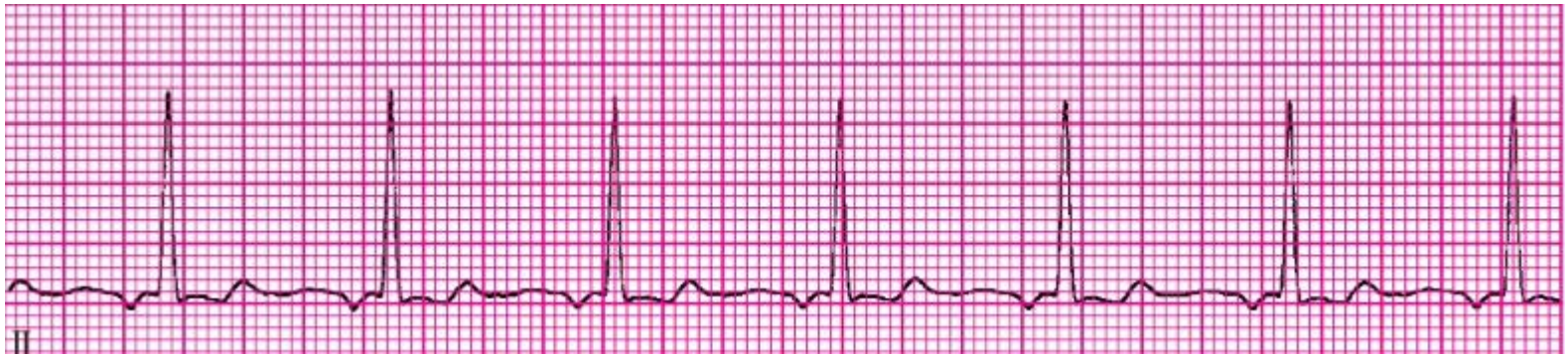
Calculating axis



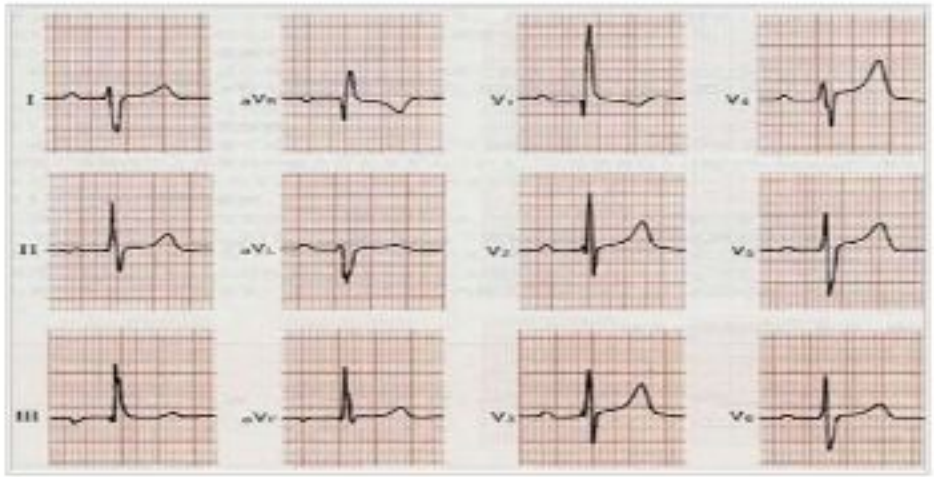
Distinguishing sinus rhythm

Upright P wave in II, III, aVF

Negative P wave could be low sinus rhythm however if PR is < 120 ms (3 boxes) it is retrograde P wave

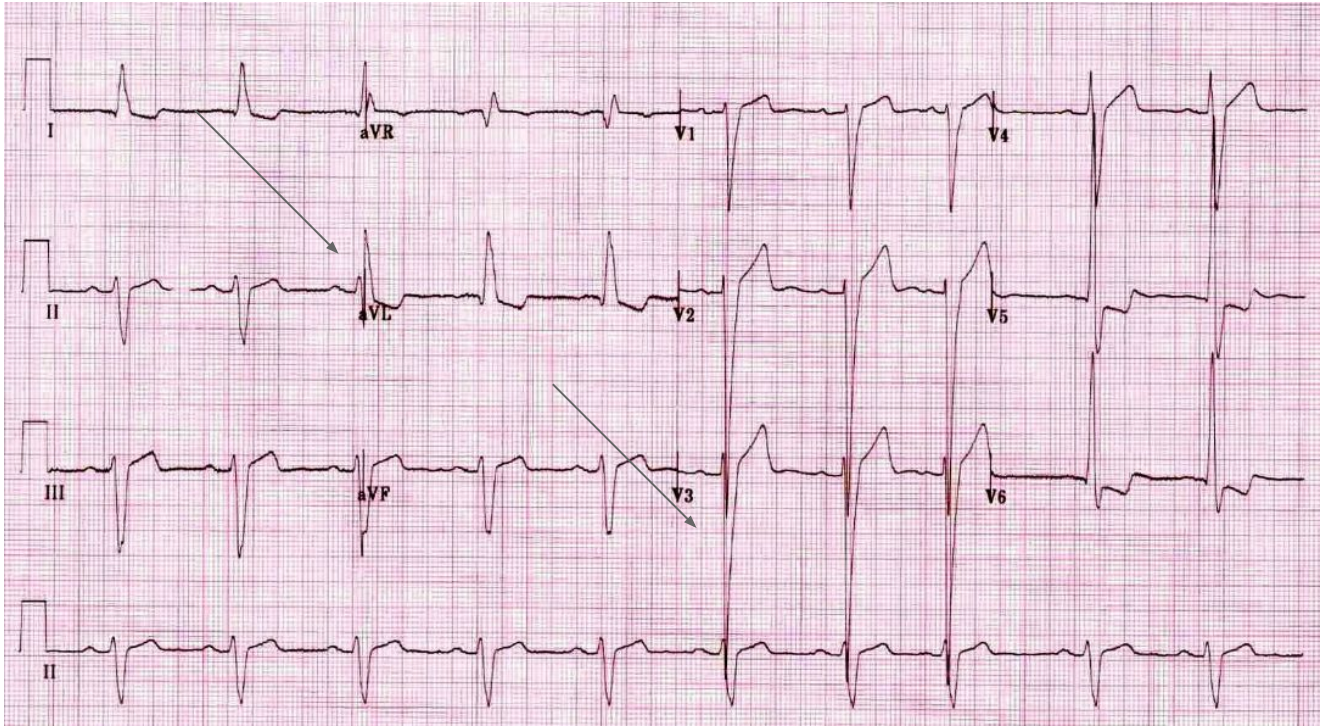


Right Ventricular Hypertrophy



Tall R wave > 7 mm in V1 or rSR' greater than 10 mm
Right axis deviation > 100
R in V1 and S in V5 or V6 > 10.5 mm

Left Ventricular Hypertrophy



S in V3 + R in aVL > 28

Secondary repolarization changes

LVH criteria

Several criteria, too hard to remember all of them

Cornell (most accurate): S in V3 + R in aVL > 28 in males and 20 in females

Look for repolarization ST-T changes, left axis deviation, IVCD and la enlargement as other clues

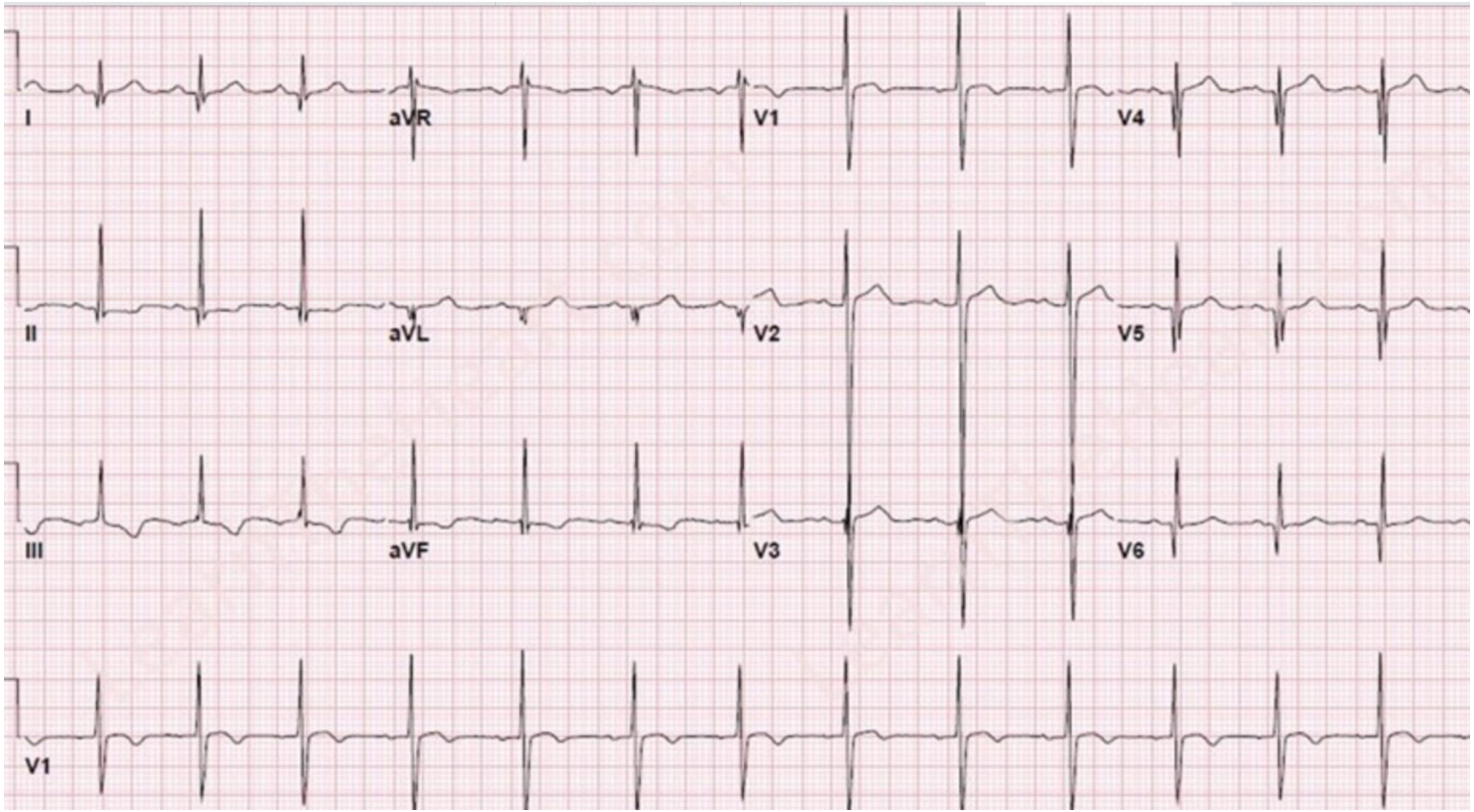
R in aVL > 12 mm (except for LAFB may cause this also)

R in I > 14 mm

HOCM

Abnormal septal thickening

Presents as large septal depolarization q waves (pseudoinfarction) noted in V4 to V6 I and aVL

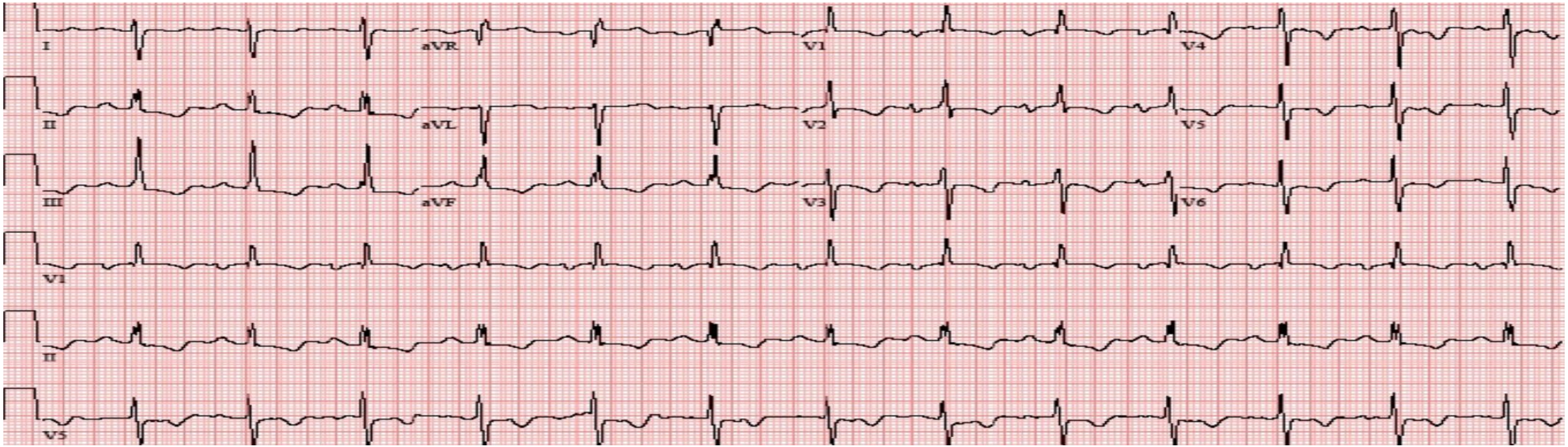


Notice septal Q waves, notice they are deep but not wide

Left atrial dilation

P wave amplitude is 1 small box wide and down in V1

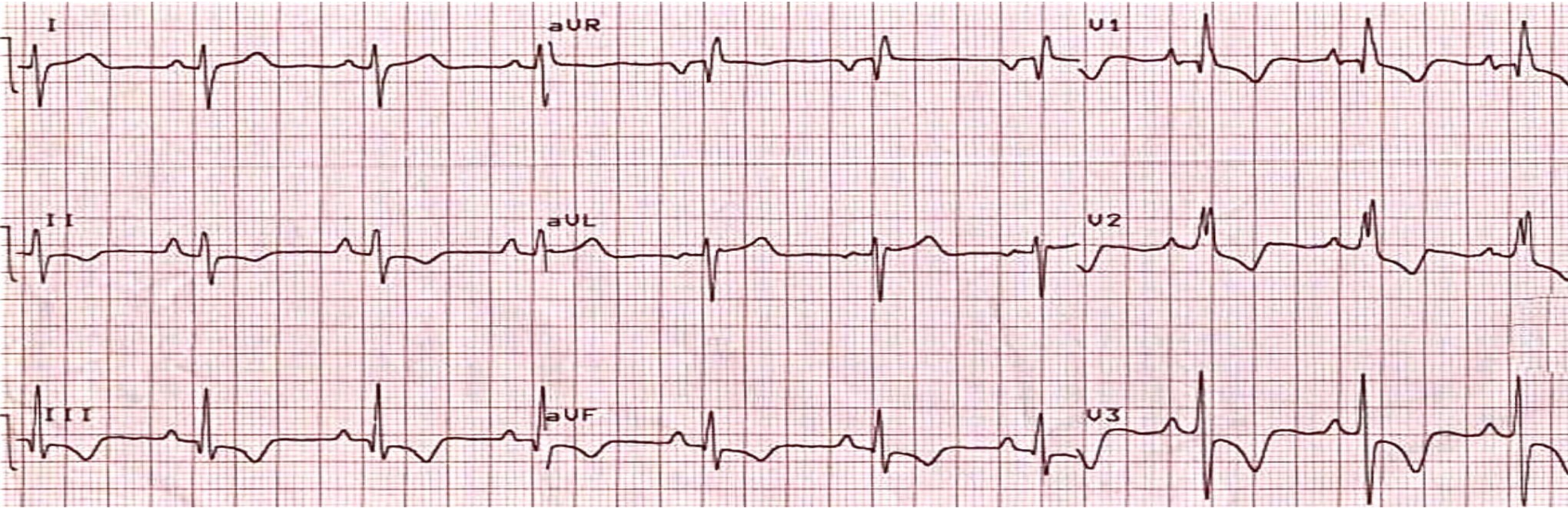
Notched P wave in inferior leads > 0.12 seconds (3 small boxes)



Right atrial dilation

P wave > 2.5 mm in II, III, aVF

P wave > 1.5 mm in V1 V2



Sick sinus syndrome

Bradycardia alternating with tachycardia

Marked sinus bradycardia

Atrial fibrillation with slow ventricular response or pause after conversion to sinus

Acute CNS Event

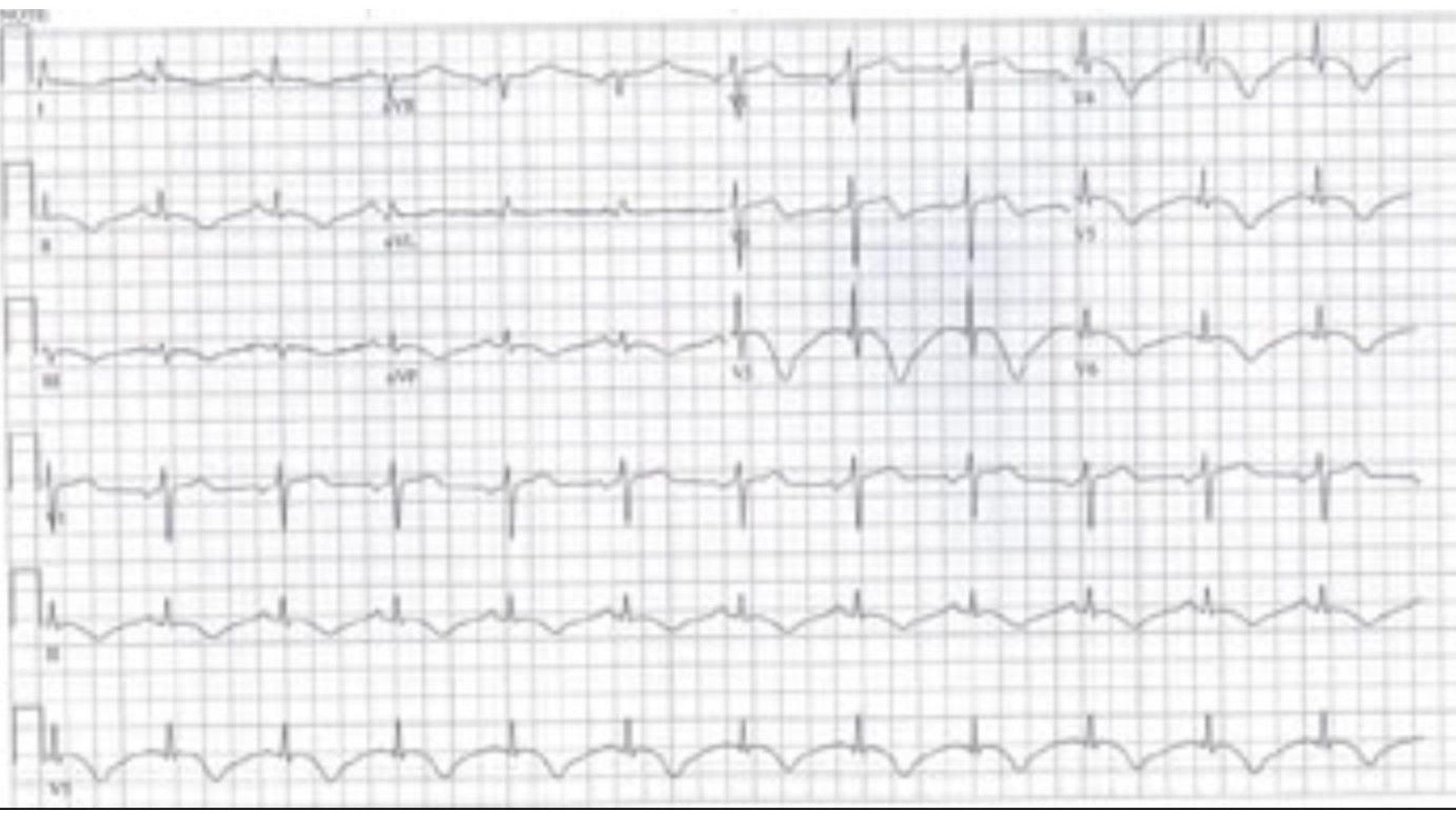
ST elevation (Takotsubo can present as ST elevation myocardial infarction)

Deep T wave inversion

Marked QT prolongation

Large U waves

CNS event can be intracranial hemorrhage, SAH



QT interval

Normal < 440 mS

Should be < 50% of RR interval

Shortens with faster heart rate

Prolongation causes: Hypomagnesemia, hypocalcemia and hypokalemia, hypothyroidism

If prolonged: Should replace K, Mag, Ca

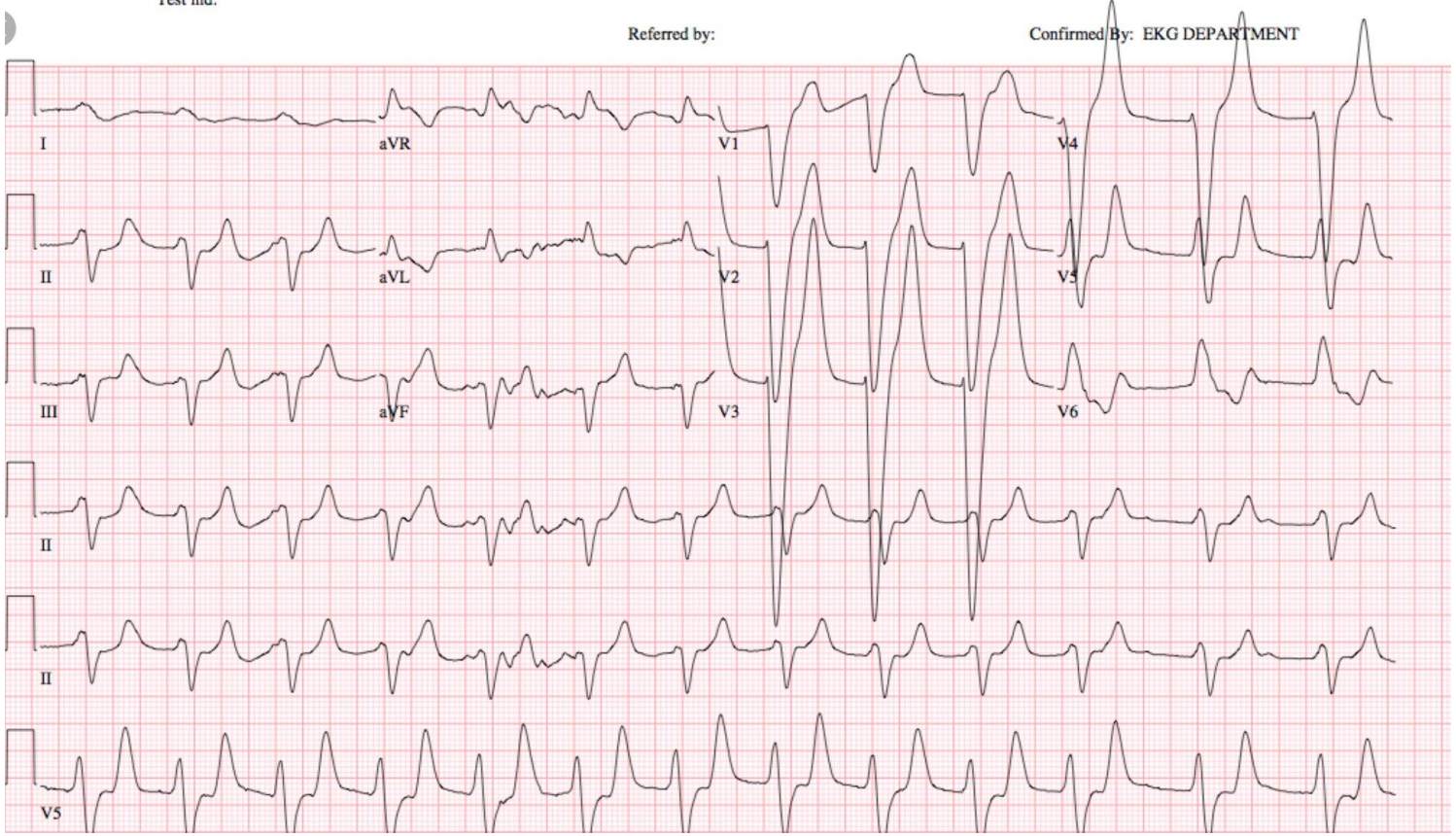
Hyperkalemia

Peaked T waves (defined at T wave > 6 mm in limb leads or > 10 mm in precordial leads

K 5.5 - 6.5: QT shortens, peaked T waves, LAFB or LPFB

K 6.5 - 7.5: Flat P wave, first degree AV block, ST depression, QRS widening

K > 7.5 sine wave, ventricular tachycardia and VF

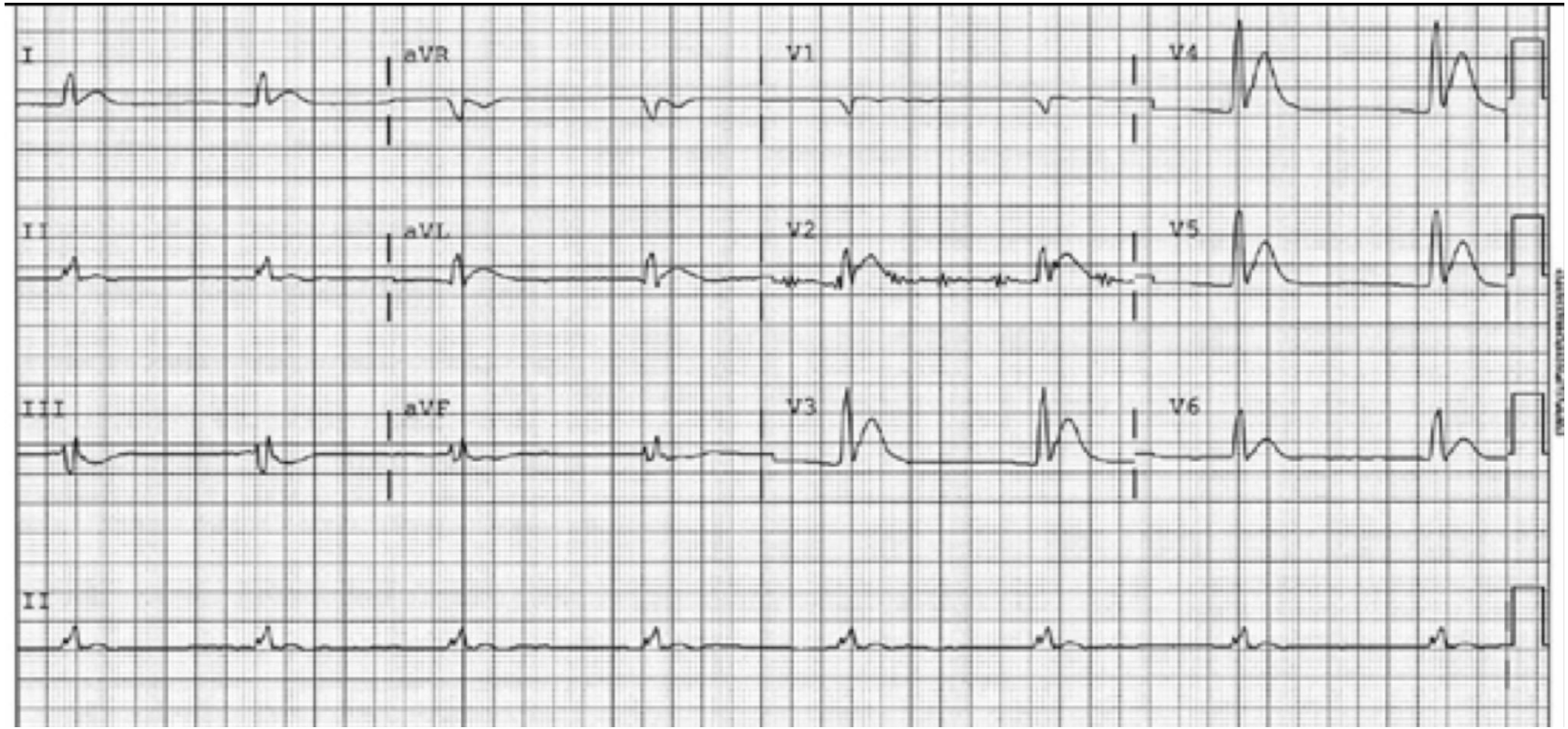


Peaked T wave, flat P wave, First degree AV block

Hypercalcemia

QT shortening

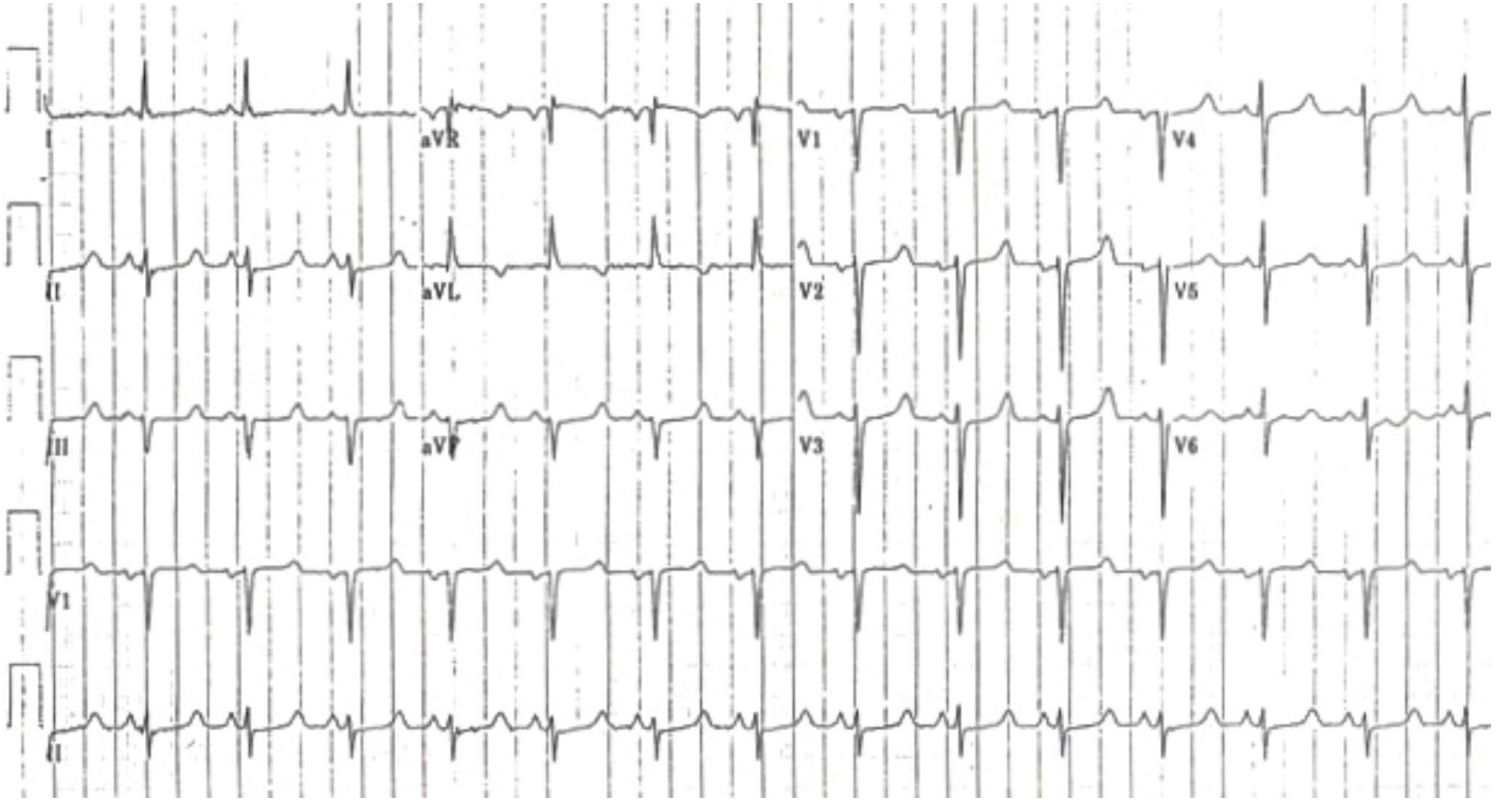
PR prolongation


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

Hypocalcemia

QT prolongation

Specifically ST segment is prolonged


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

Hypokalemia

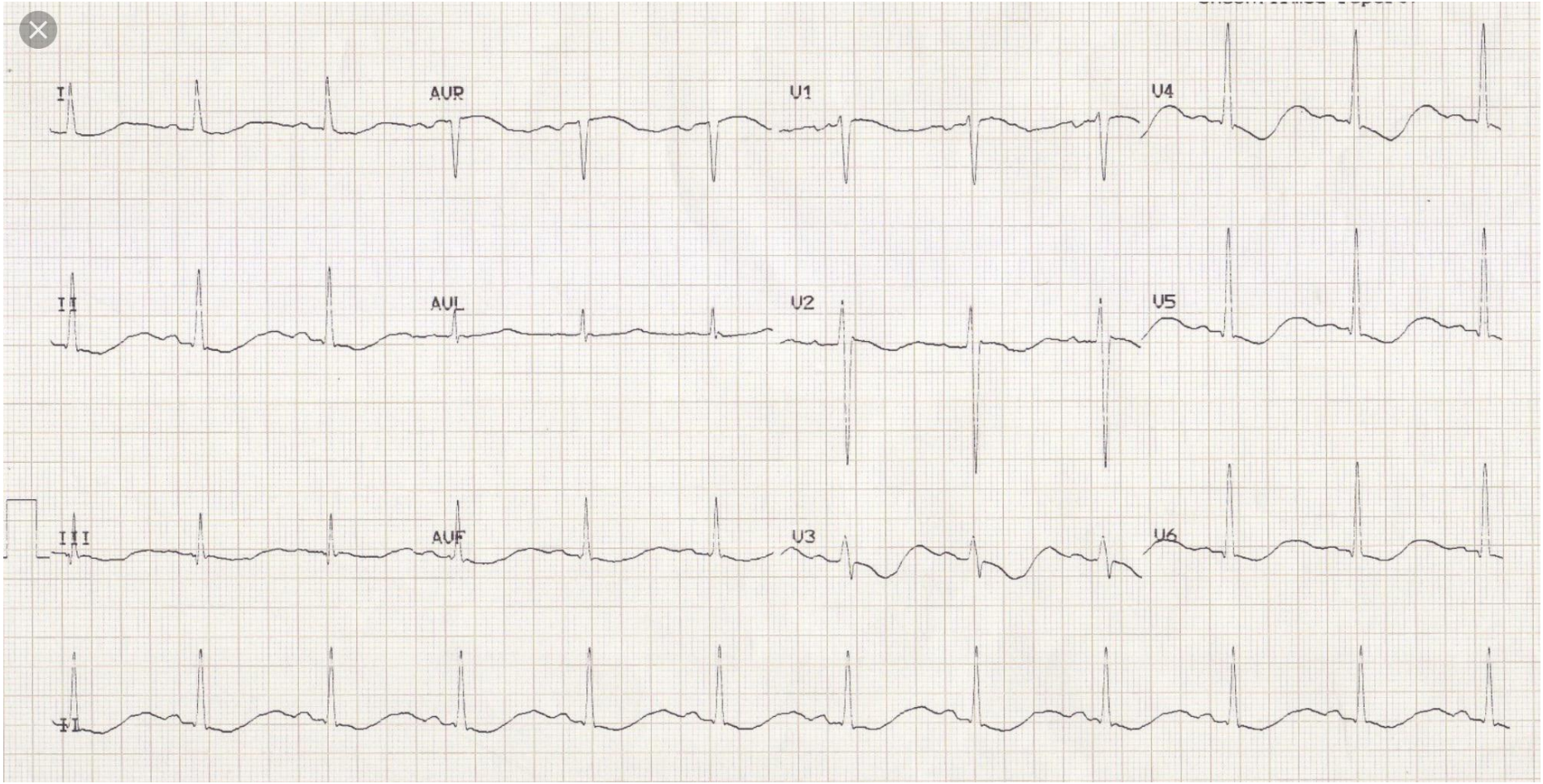
Prominant U wave

Flat T wave

ST depression

Prolonged QT

May cause PVCs, ventricular tachycardia, VF


I
AUR
V1
V4
II
AUL
V2
V5
III
AUF
V3
V6
II

Supraventricular tachycardia

Narrow complex tachycardia from any cause that is supraventricular such as atrial flutter, atrial fibrillation, AV nodal reentrant tachycardia, AV reciprocating tachycardia

However, in lay terms it is usually used to describe most probably AV nodal reentrant tachycardia

AVNRT

70% of all supraventricular tachycardia

retrograde P wave buried within the QRS, may be seen as a RSR’ in V1 where the R’ is not seen when the patient is in sinus rhythm

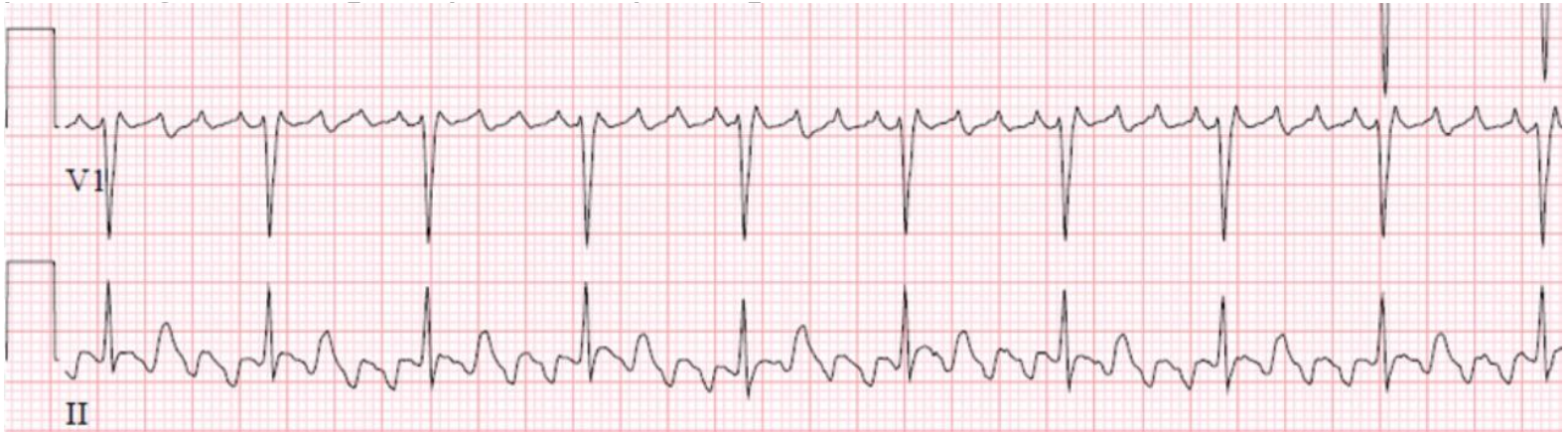
Usually initiated with a PAC and terminates spontaneously, by vagal maneuvers or adenosine

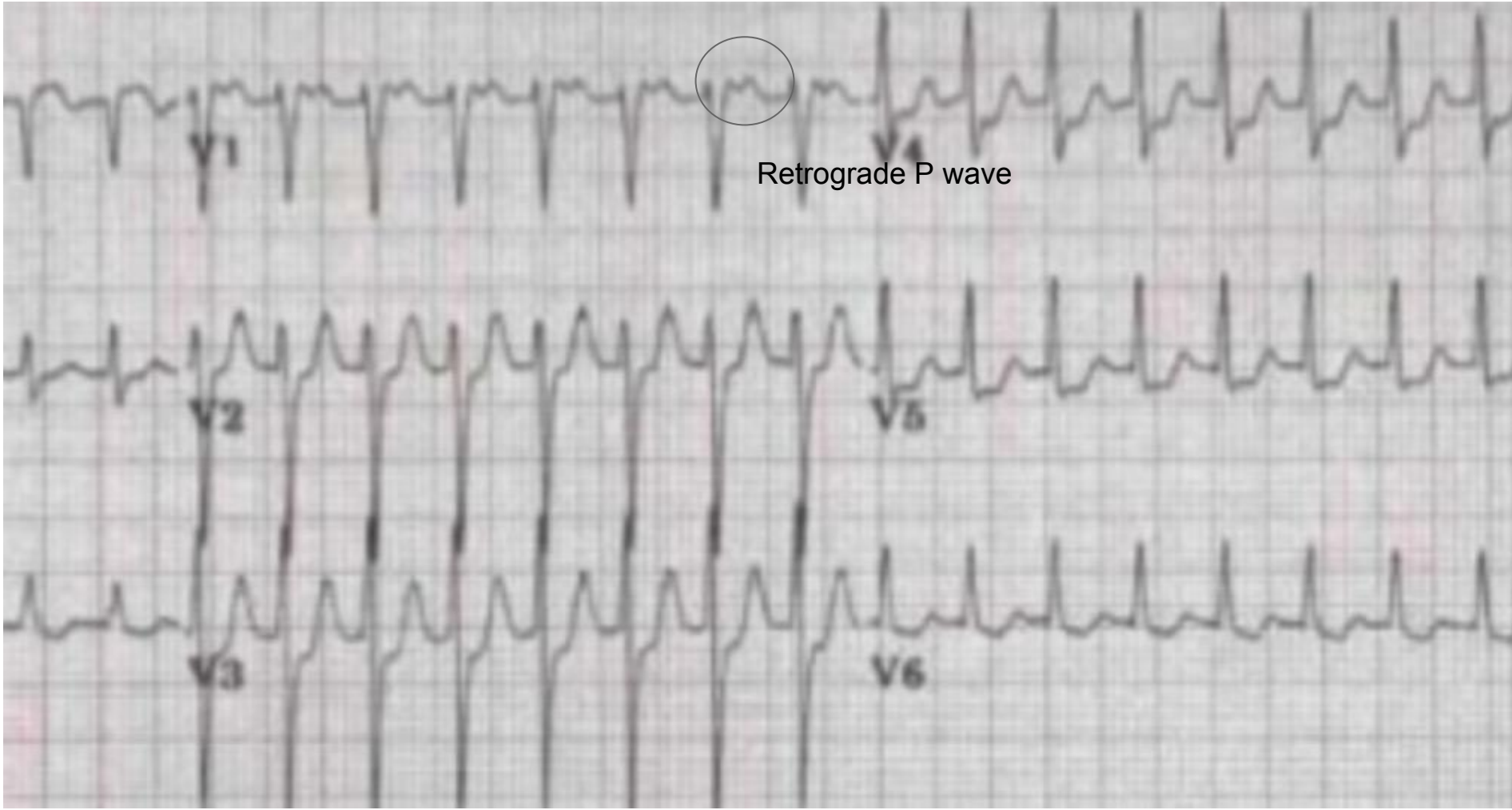
Reentry usually occurs down the slow pathway and up the fast pathway in the AV node

Atrial flutter versus tachycardia

Atrial tachycardia, rate is 100 to 240, iso-electric baseline noted

Atrial flutter, sawtooth negative appearance if typical in t



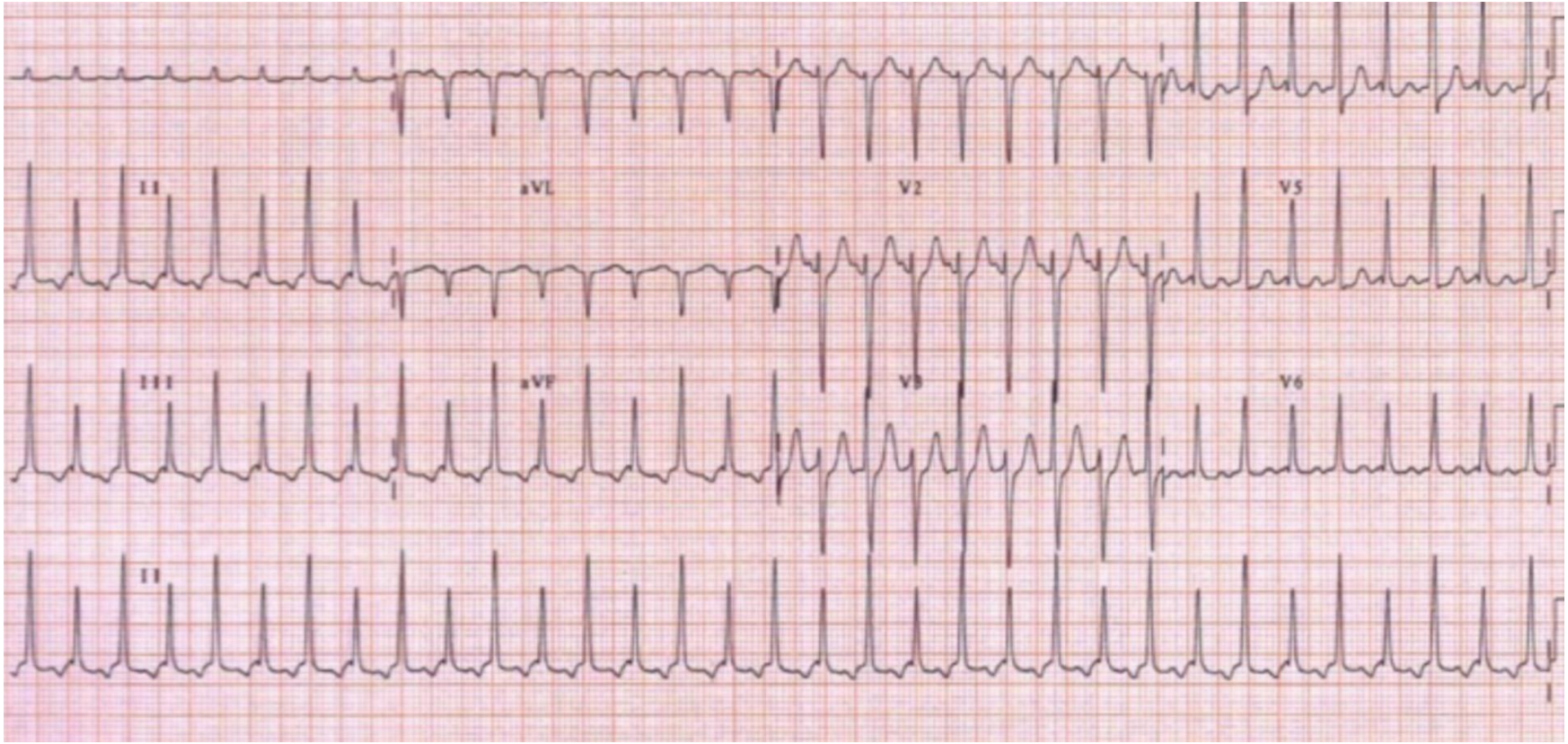

V1
V4
Retrograde P wave
V2
V5
V3
V6

Low Voltage

R wave < 5 mm in limb leads and < 10 mm in precordial leads

Can be caused by several abnormalities including body habitus, pericardial effusion, emphysema, cardiomyopathy

If seen along with electrical alternans consider pericardial effusion



Electrical alternans

Ventricular tachycardia

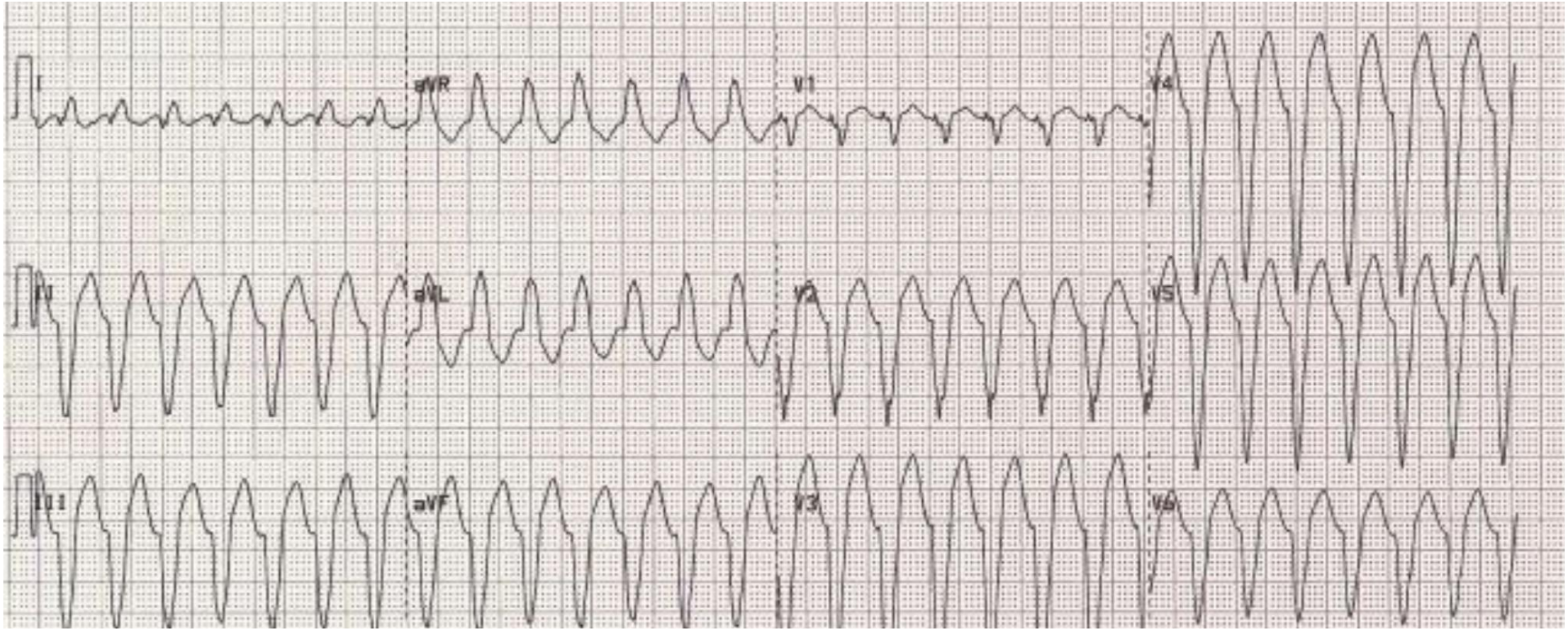
Wide complex > 140 ms for RBBB morphology or > 160 ms for LBBB morphology

AV dissociation

Capture beat - sinus beat conducts into the ventricle

Fusion beat - normal R wave occurs simultaneous to VT

Precordial concordance (all positive or all negative)



Concordance, extreme axis, marked prolonged qrs

Digitalis Toxicity

May manifest itself as atrial fibrillation with regular ventricular response due to a complete heart block and AV junctional escape rhythm but can cause any type of heart block with or without atrial tachycardia

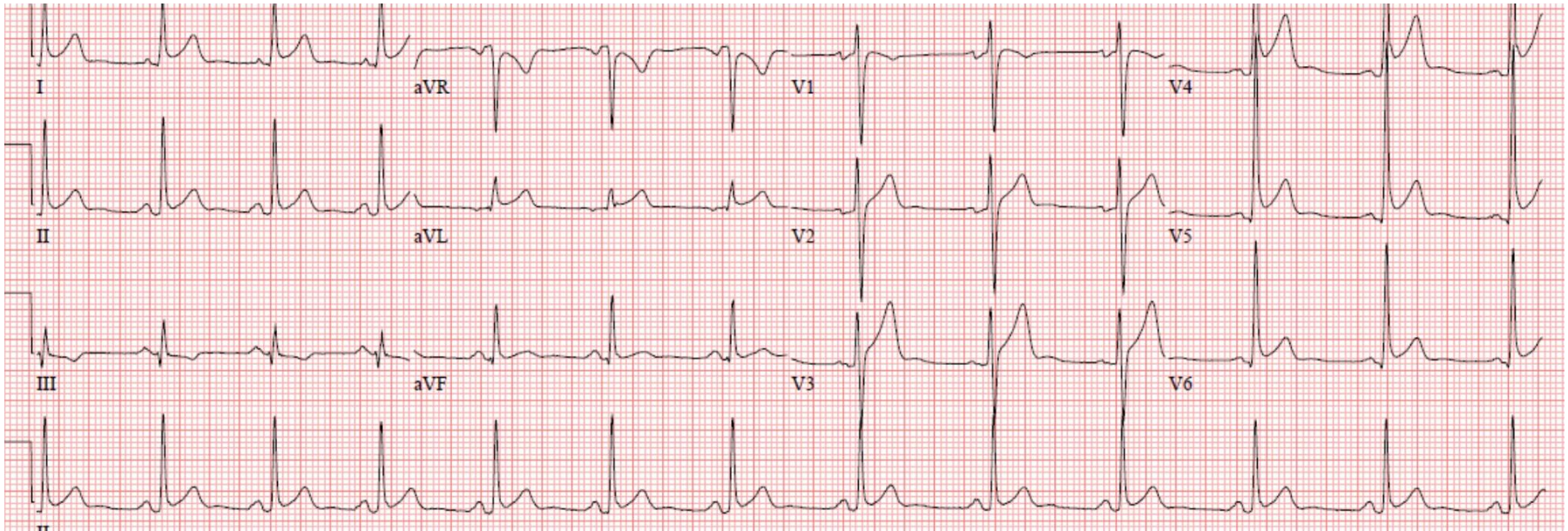
Does **not** cause bundle branch block

If there is digitalis toxicity, do not cardiovert as it may lead to ventricular fibrillation

Acute pericarditis

Diffuse ST elevation in all leads except aVR

PR depression in all leads except aVR



Thank You