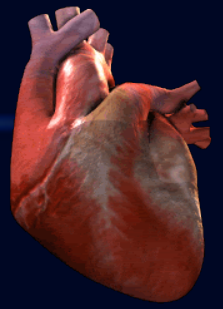
That Doesn't Look Quite Right: Refining Your EKG Reading Skills

Ben Taylor PhD, PA-C, DFAAPA



Disclosures

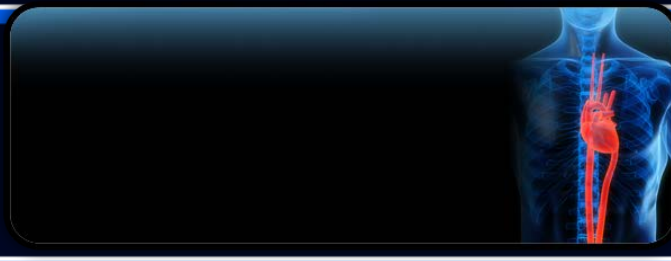
No relevant commercial relationships to disclose

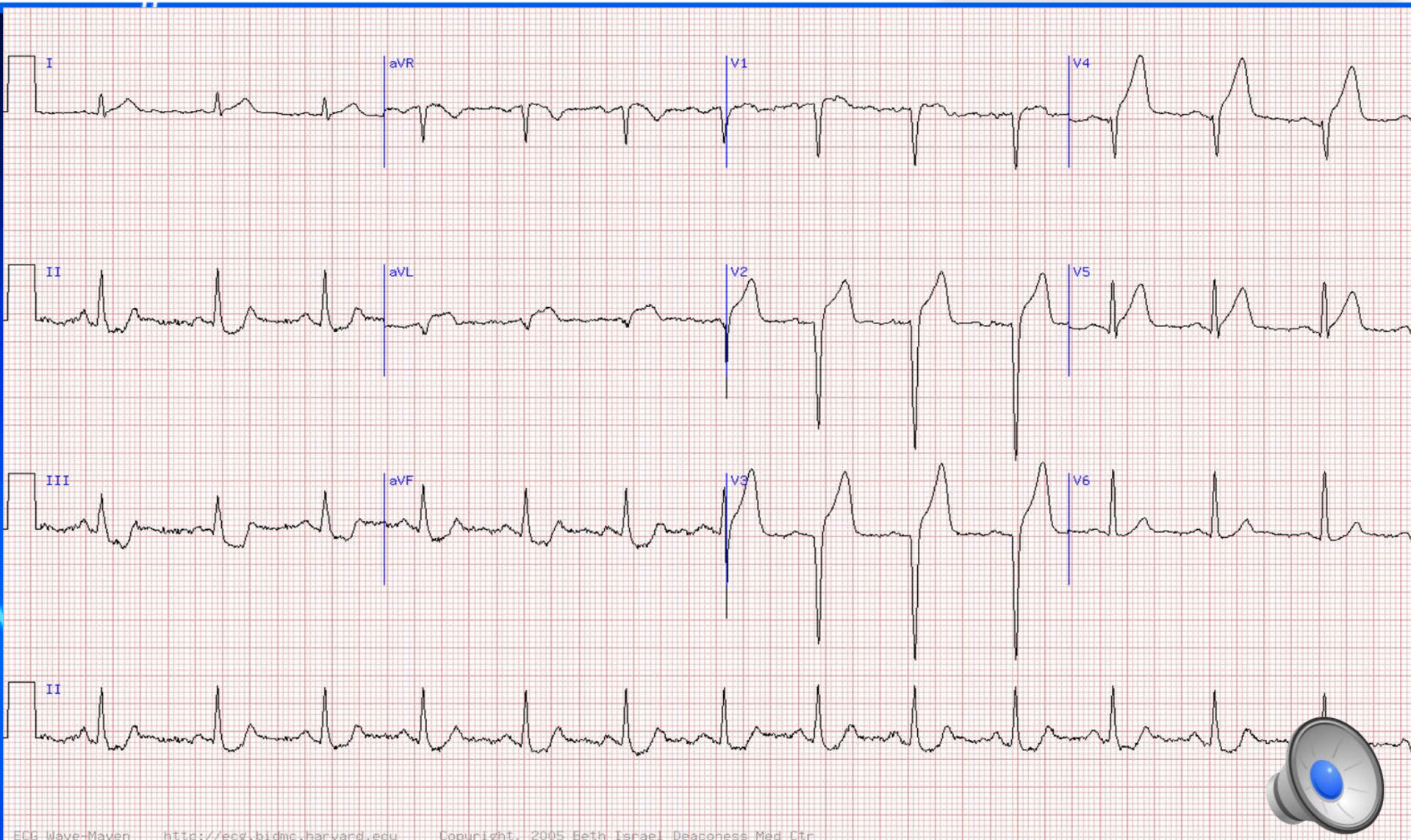
Learning Objectives

At the conclusion of this session, participants should be able to:

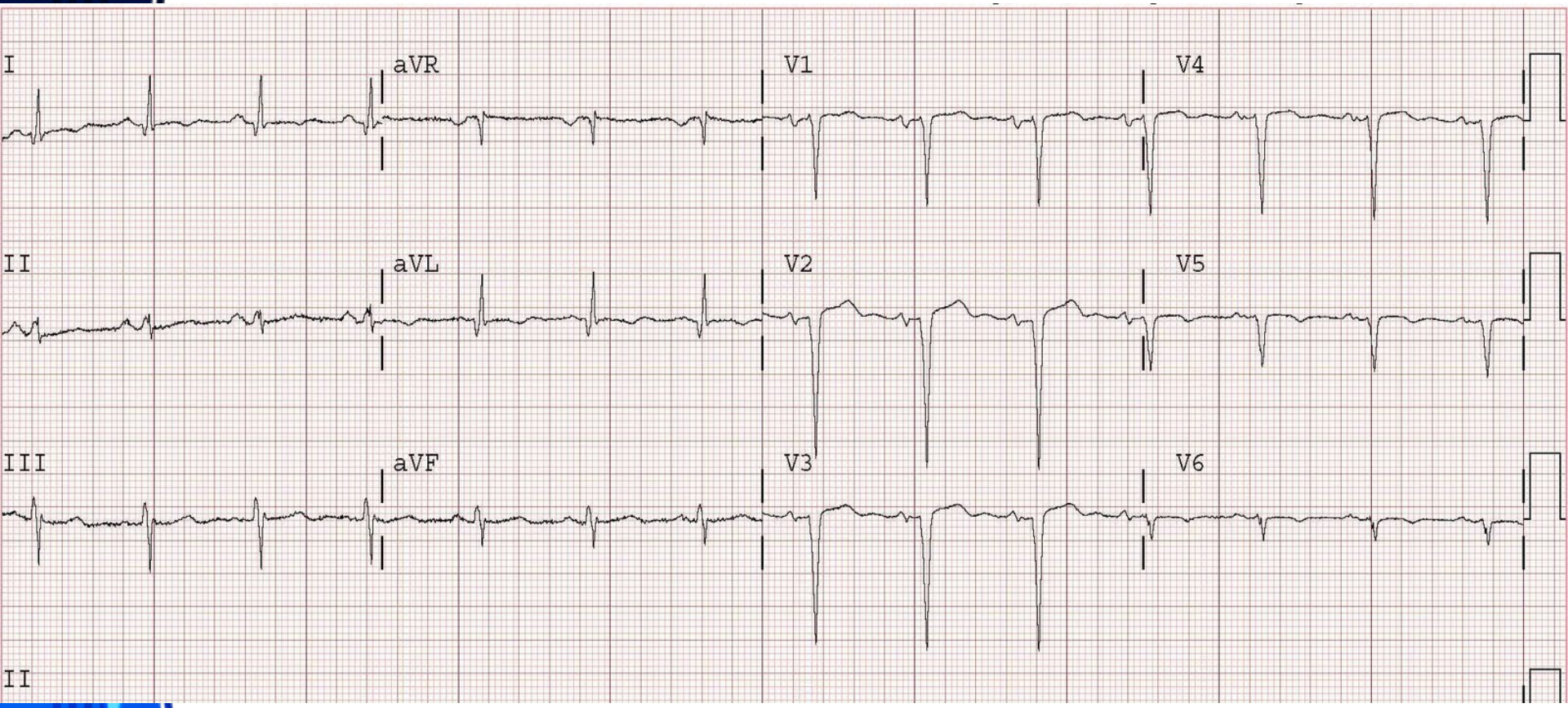
- Identify abnormalities on the EKG
- Discuss the EKG manifestations of Wellens' syndrome.
- Identify abnormal manifestations of a left main stem occlusion
- Recognize the ECG manifestations and clinical significance of ARVD
- Recognize the different types of WPW

We Wish!!




I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
ECG Wave-Maven http://ecg.bidmc.harvard.edu Copyright, 2005 Beth Israel Deaconess Med Ctr

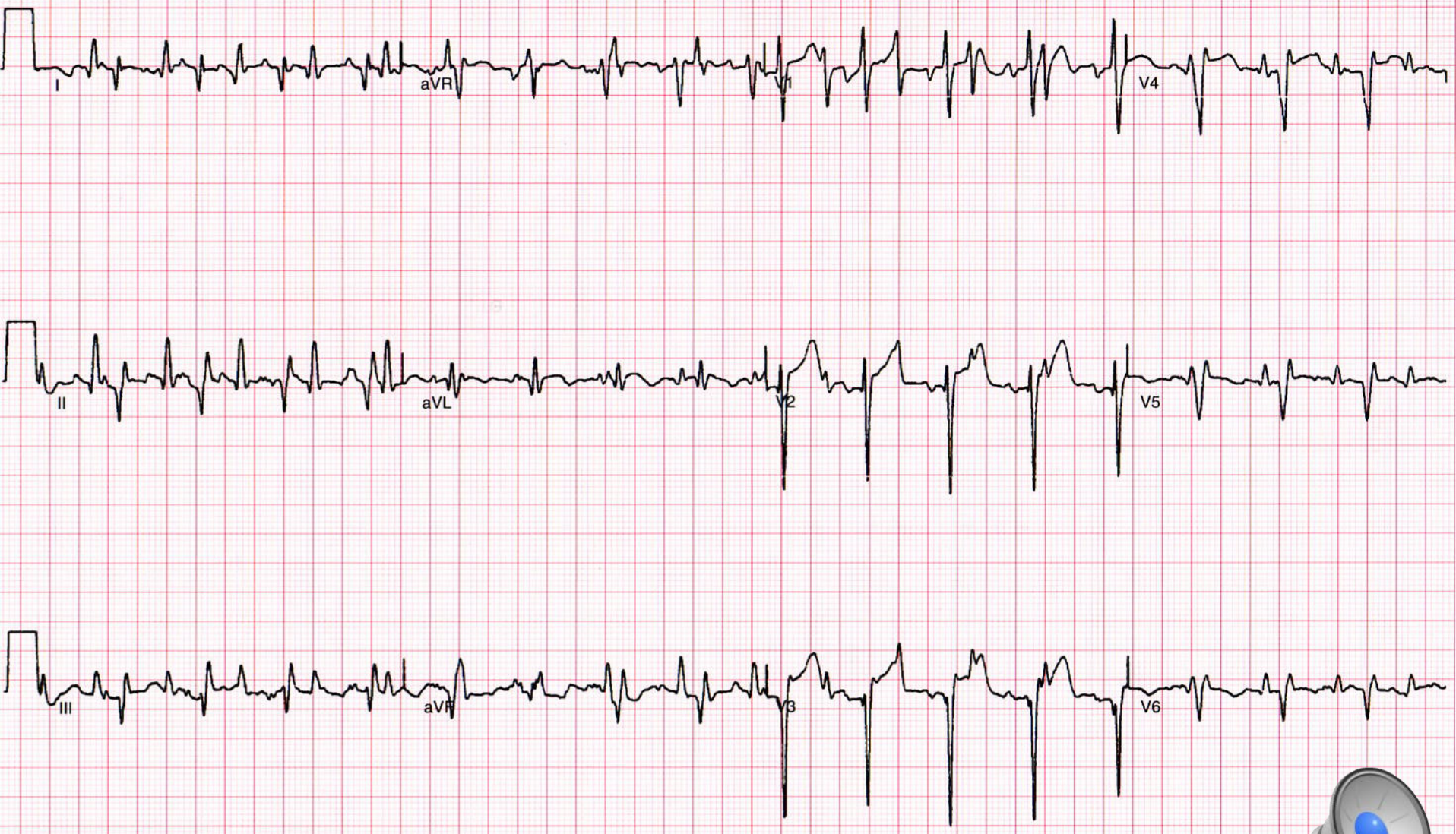
But Sometimes They Look Like This….



This MI was missed by the medical provider b/c of pts age (29)

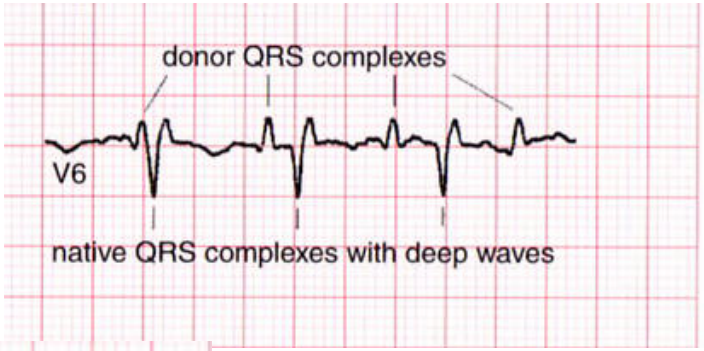
This is a good Starting point

- 42-year-old male presents to his PCP with a complaint of chest congestion and cough. He has a history of a heart transplant for ischemic cardiomyopathy 1 year ago. He has been afebrile, but his chest hurts from coughing.
- No tobacco, alcohol, or drugs. States he takes his medications as prescribed.
- He denies N/V/paresthesia's, upper back or neck pain.

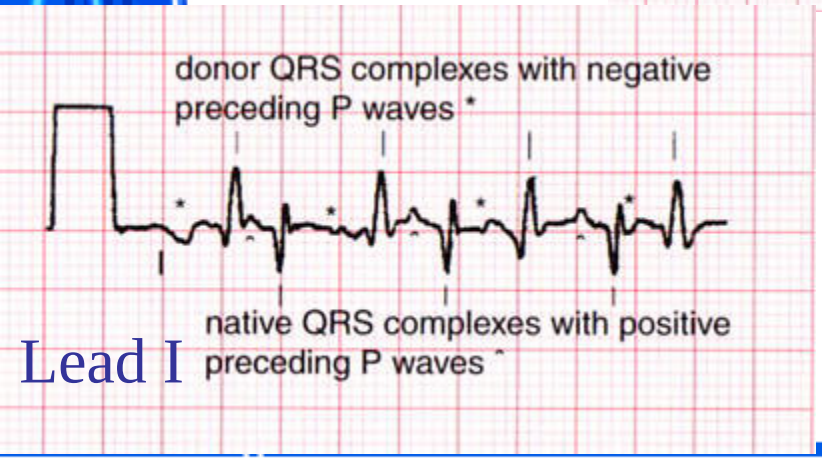

I
aVR
V4
II
aVL
V5
III
aVF
V6

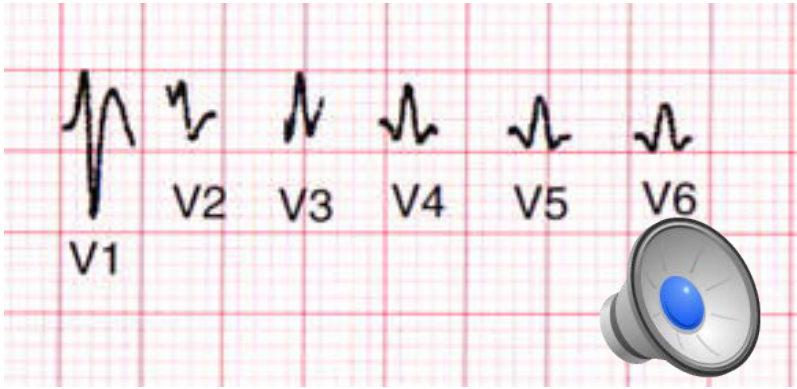
Auxiliary (Heterotopic or 'Piggyback') Heart Transplant

- Native heart
  - S. Tach, extreme RAD
  - Features of old anterolateral MI with deep Q-waves in leads I & V3-6

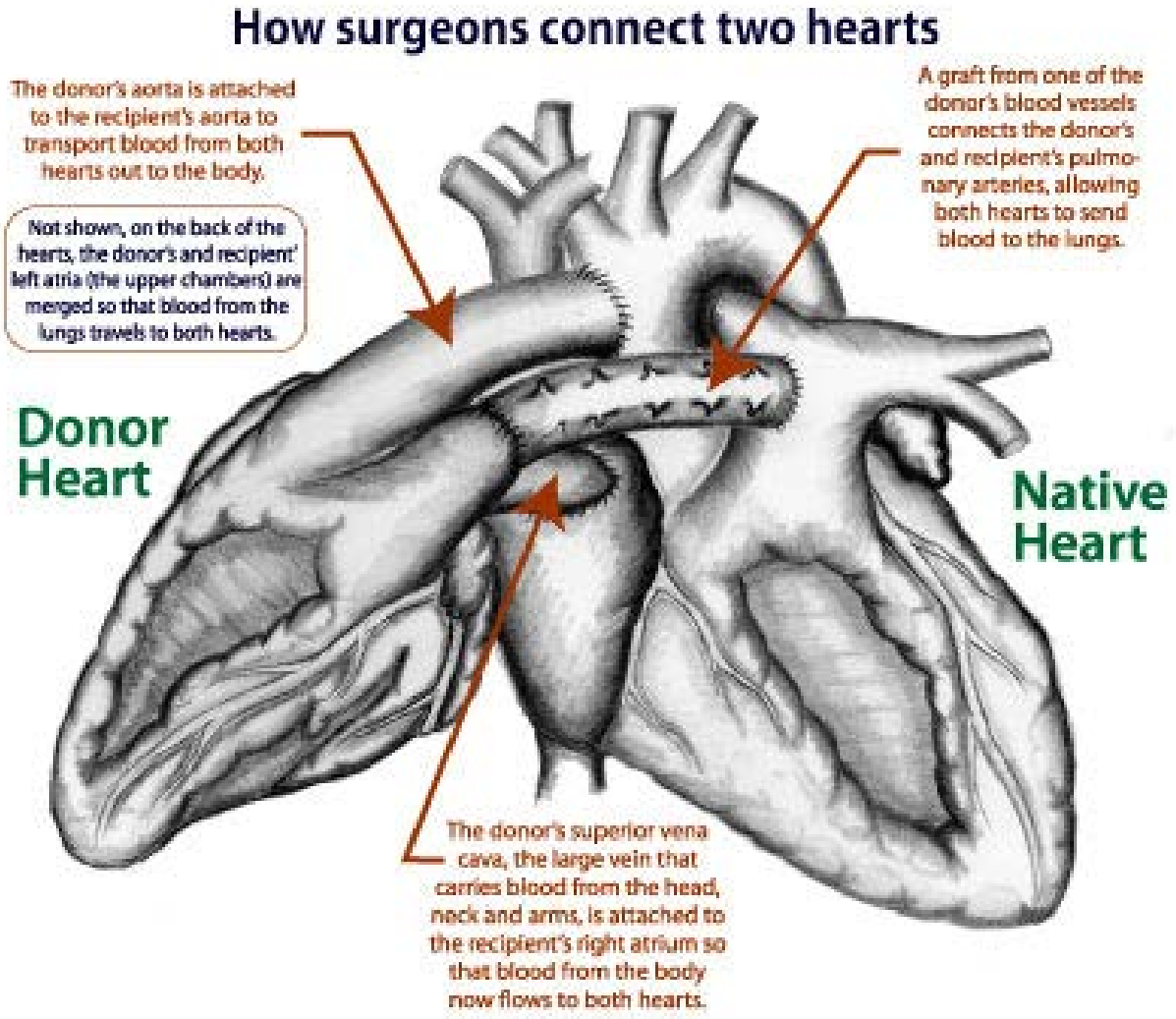


- **Donor heart**
  - **S. rhythm, normal axis**
  - **Features of Dextrocardia**
    - **Negative P in lead I**
    - **Small QRS complexes decreasing in size from V1 to V6**

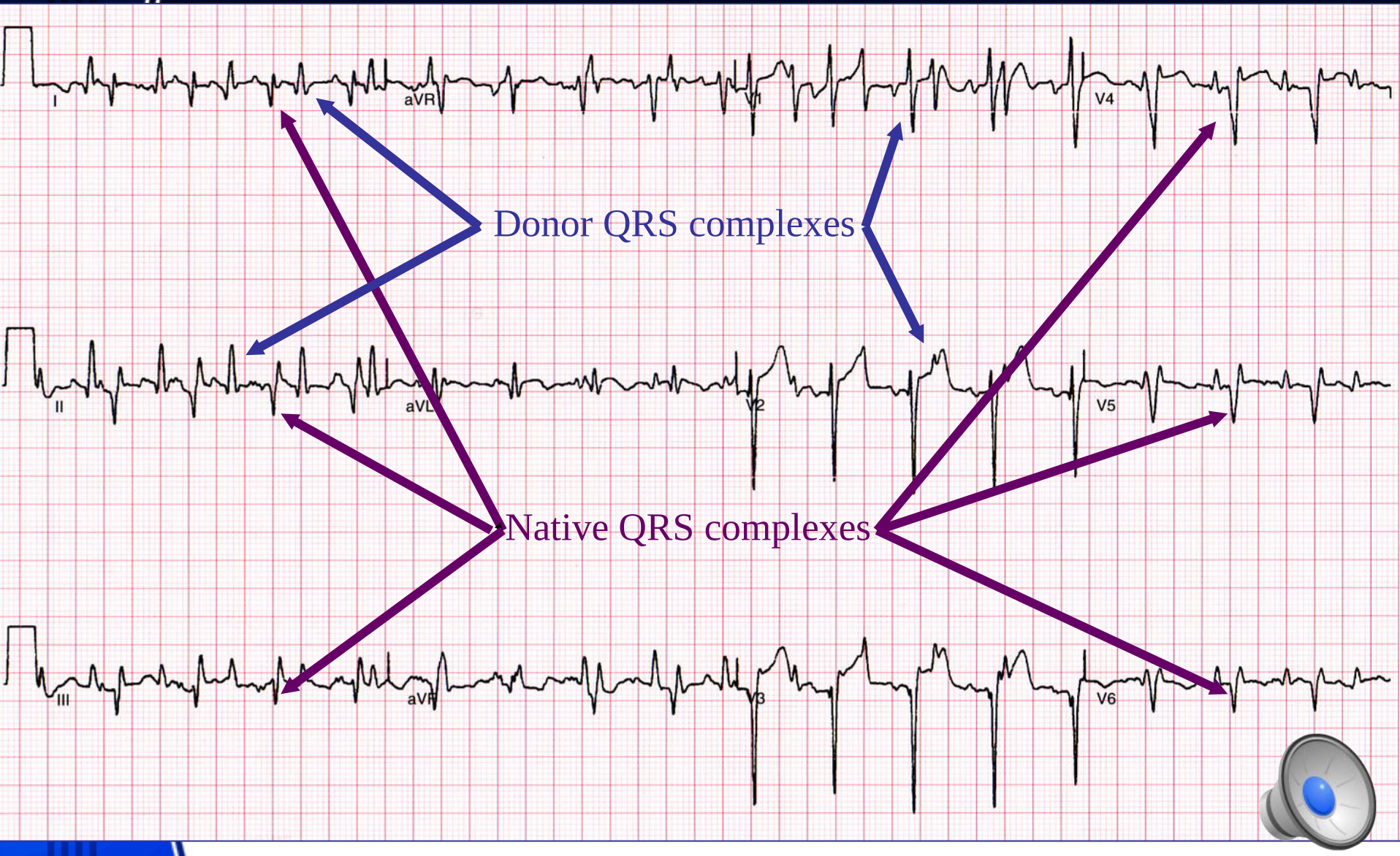




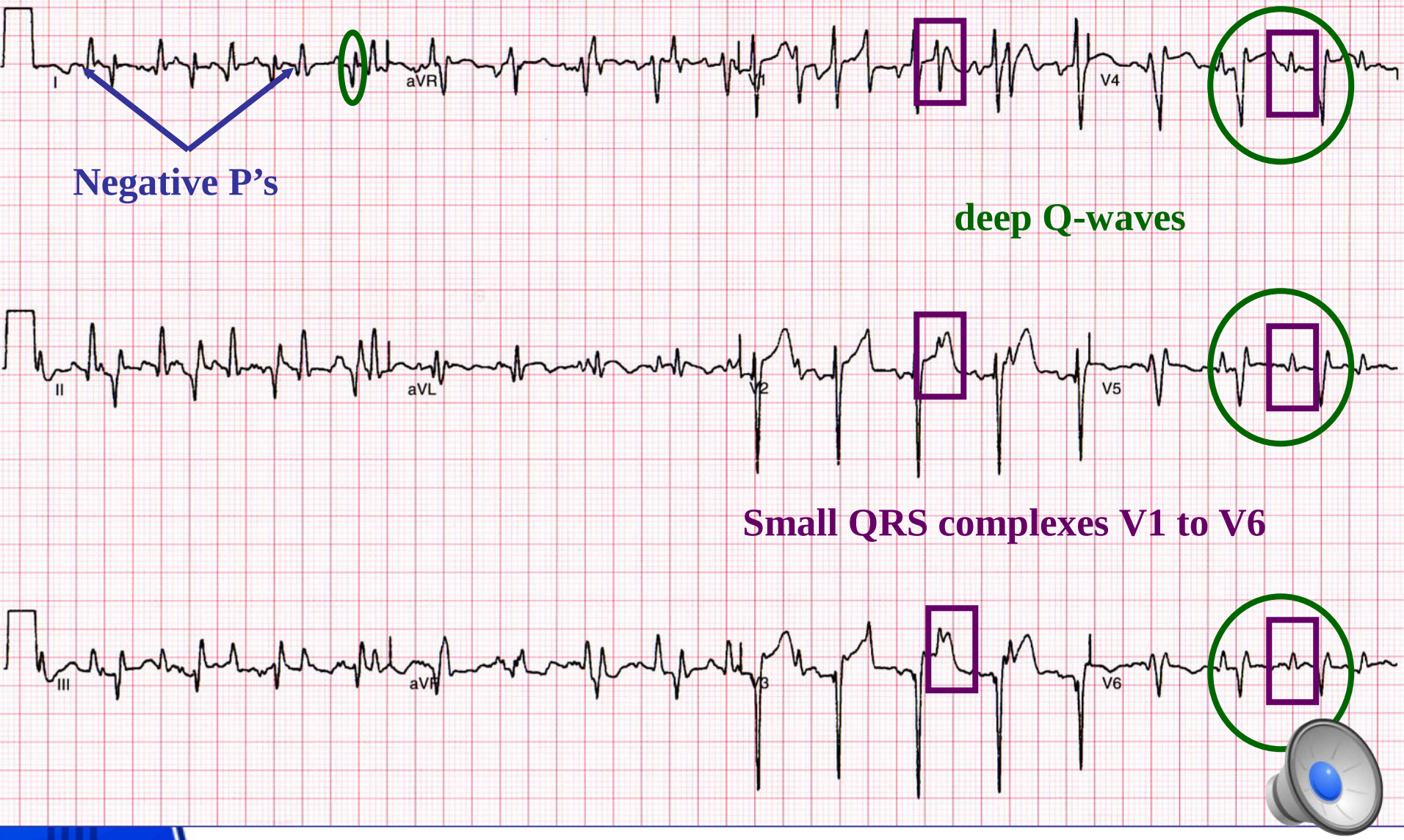
How It's Done


How surgeons connect two hearts
The donor's aorta is attached to the recipient's aorta to transport blood from both hearts out to the body.
Not shown, on the back of the hearts, the donor's and recipient' left atria (the upper chambers) are merged so that blood from the lungs travels to both hearts.
A graft from one of the donor's blood vessels connects the donor's and recipient's pulmonary arteries, allowing both hearts to send blood to the lungs.
Donor Heart
Native Heart
The donor's superior vena cava, the large vein that carries blood from the head, neck and arms, is attached to the recipient's right atrium so that blood from the body now flows to both hearts.

Auxiliary (Heterotopic or ‘Piggyback’) Heart Transplant



Auxiliary Heart Transplant



Our Next Patient…….

- **A 58-year-old male calls EMS complaining of chest discomfort that awoke him out of his sleep. Upon EMS arrival, the patient is found sitting on the edge of the bed. He is anxious but alert and oriented to person, place, time, and event.**
- **Onset: 30 minutes ago while sleeping**
- **Provoke: Nothing makes the pain feel better or worse**
- **Quality: Severe pressure or “ache”**
- **Radiate: The pain does not radiate**
- **Severity: 10/10**
- **Time: He has had chest pain before but “not this bad”**

- **Past medical history: HTN, dyslipidemia**

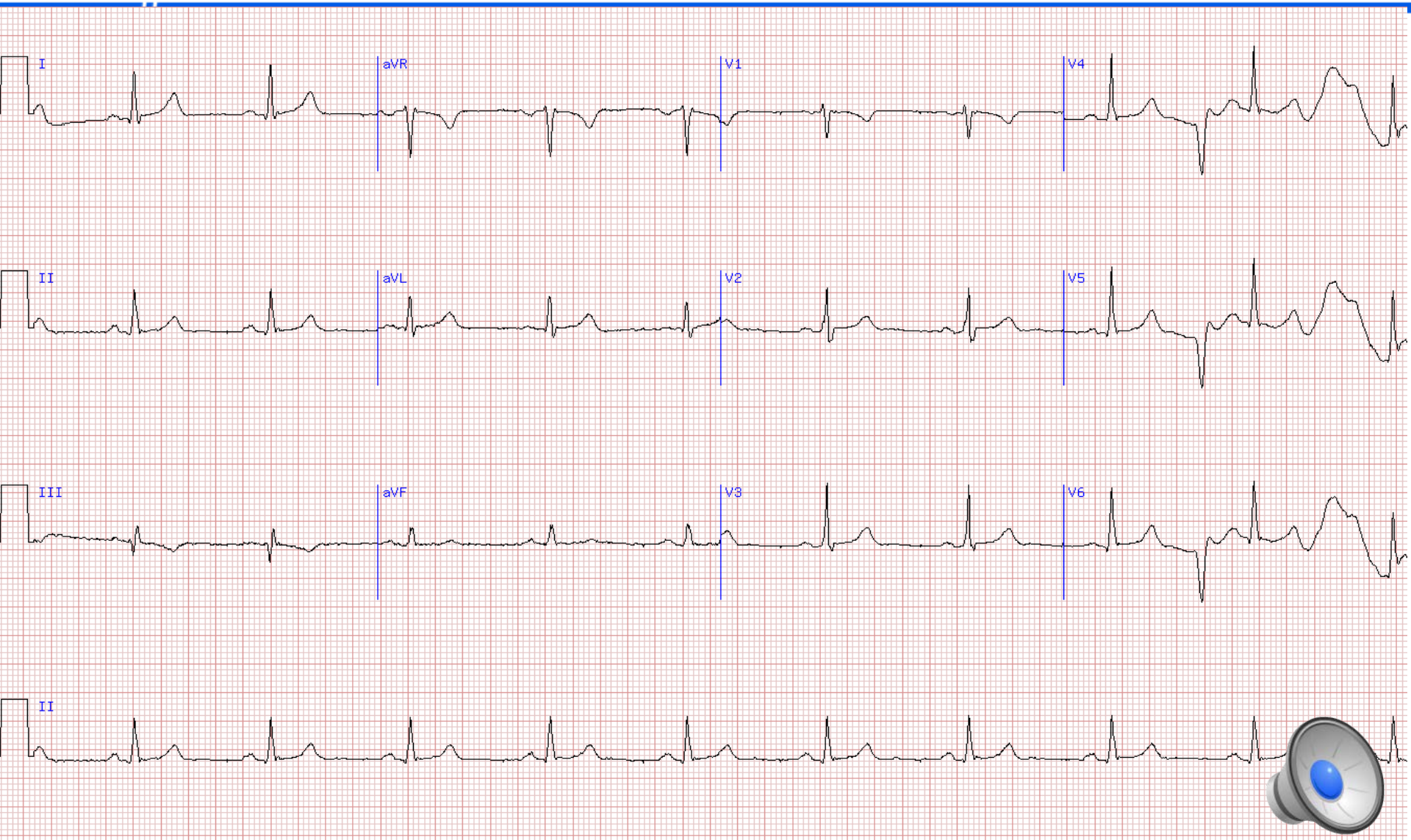
- **Medications: Lipitor, Norvasc, ASA**

- **Vital signs:**
- **RR: 24**
- **Pulse: 60**
- **BP: 160/98**
- **SpO2: 96 on RA**
- **Temp: 99.1**
- **BGL: 138**

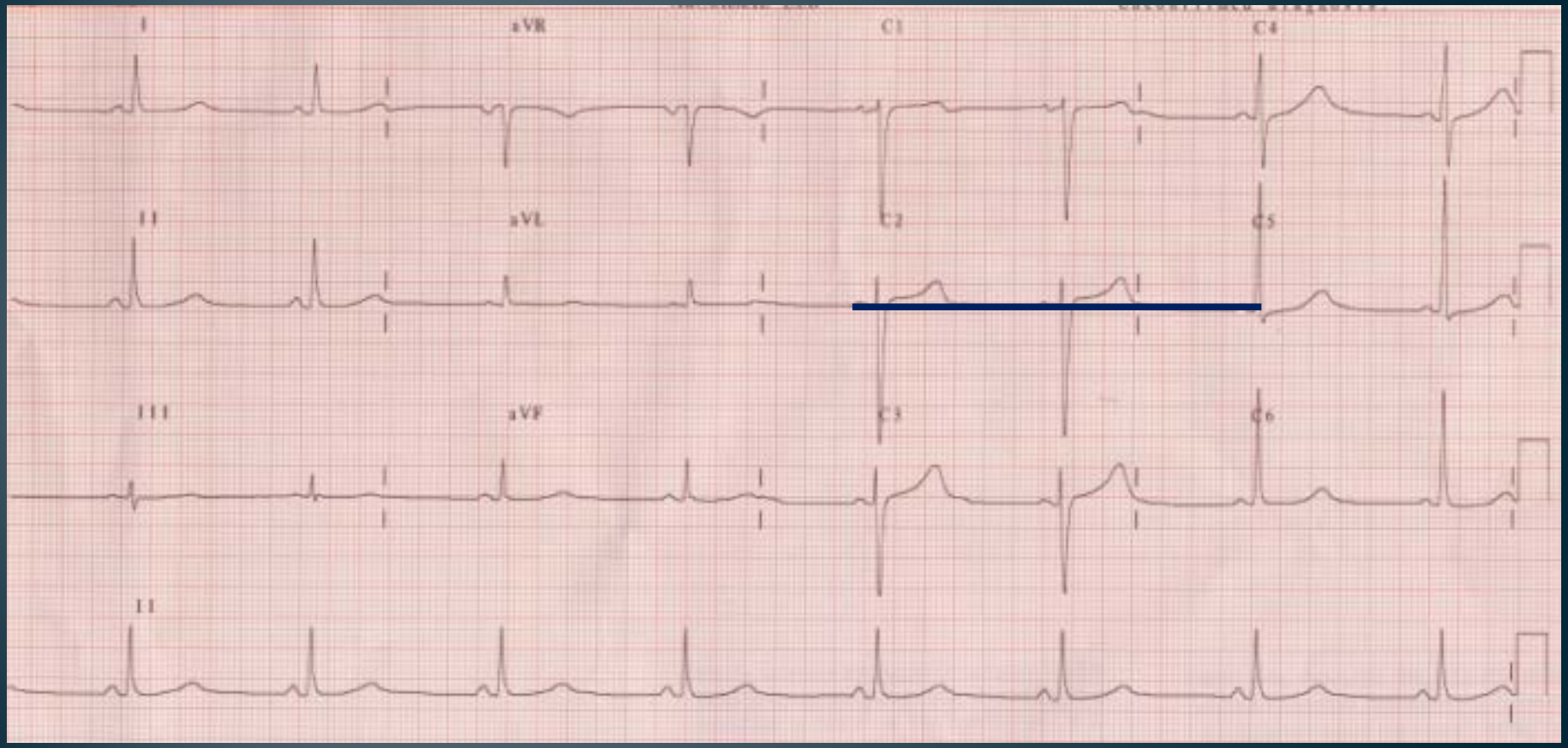
- **Breath sounds: basilar rales**

- **The patient admits to mild dyspnea. He states that he has “gained a little weight” recently and his docto getting ready to put him “on a water pill.”**

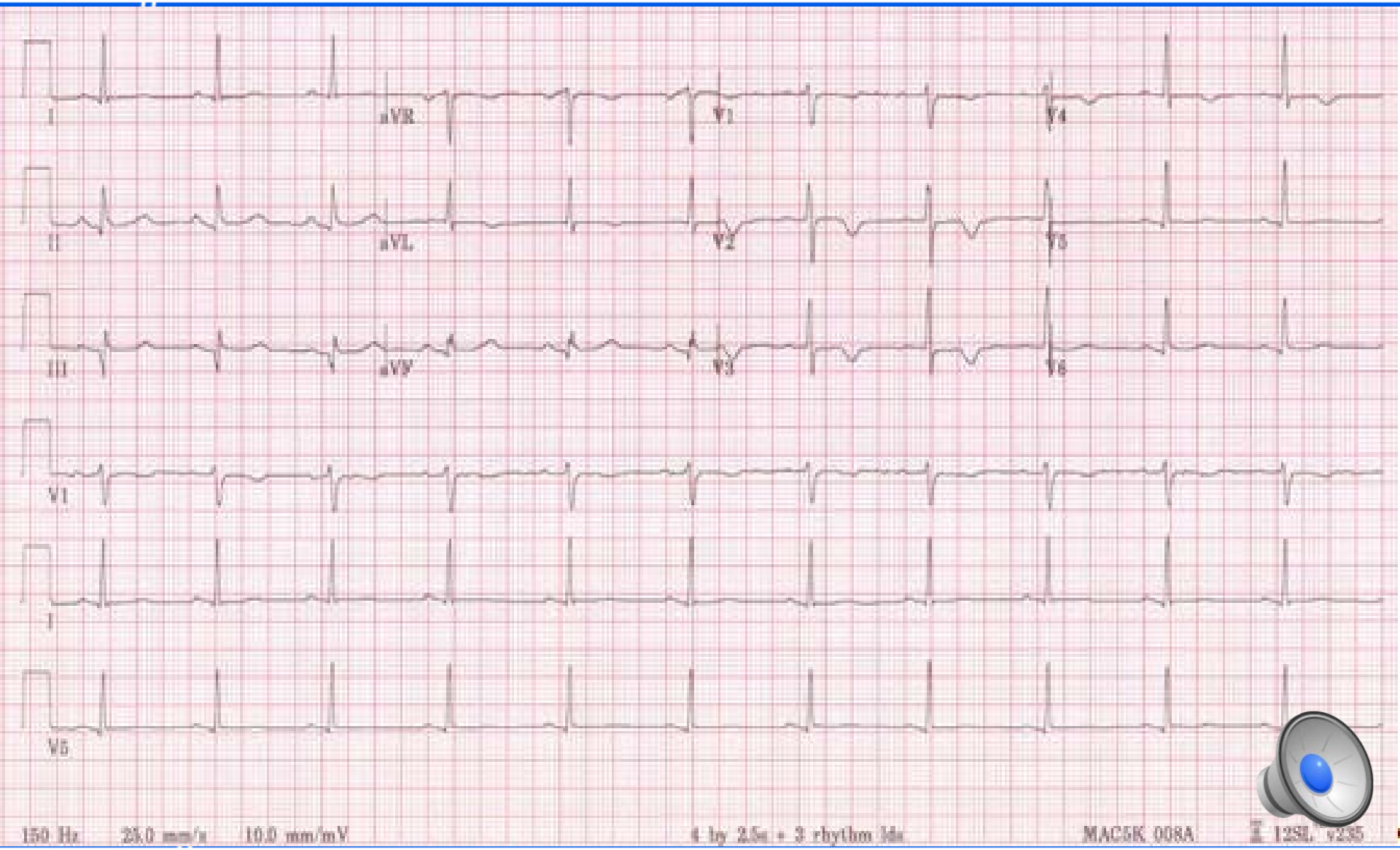
Can I Send Him Home?



One Hour Later



His EKG 4 days Later
What Do You See?



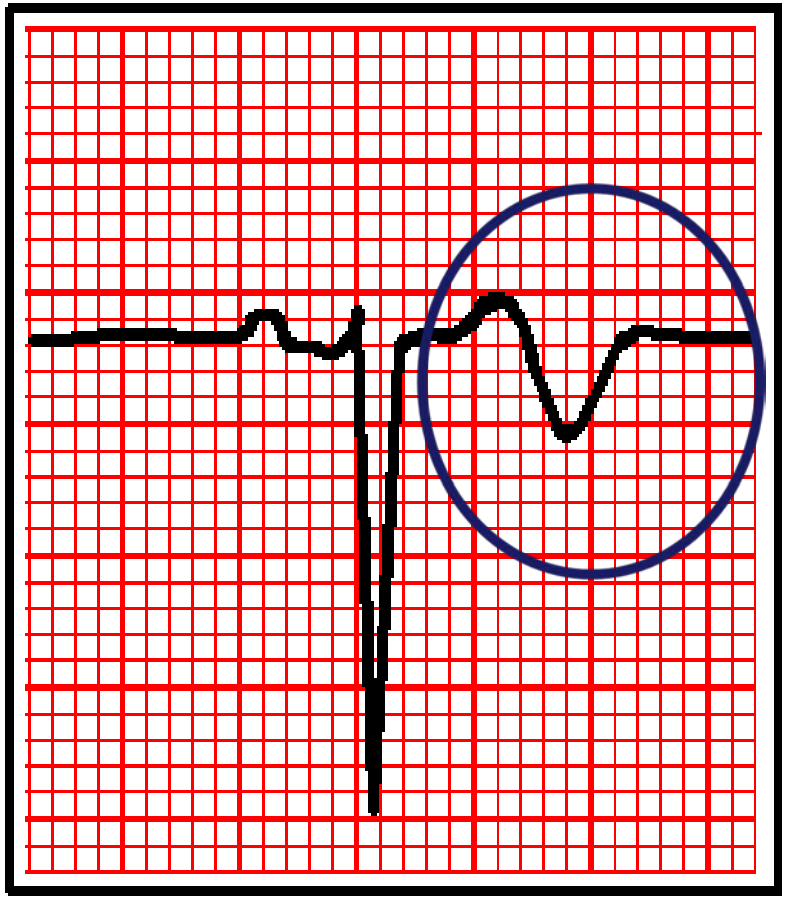
Wellens Syndrome

Wellens Syndrome is an easy to identify cardiac syndrome which indicates a critical high-grade occlusion of the proximal LAD. If not identified and properly treated the mean time from onset of symptoms to extensive anterior wall MI is 8.5 days

Criteria of Wellens Syndrome

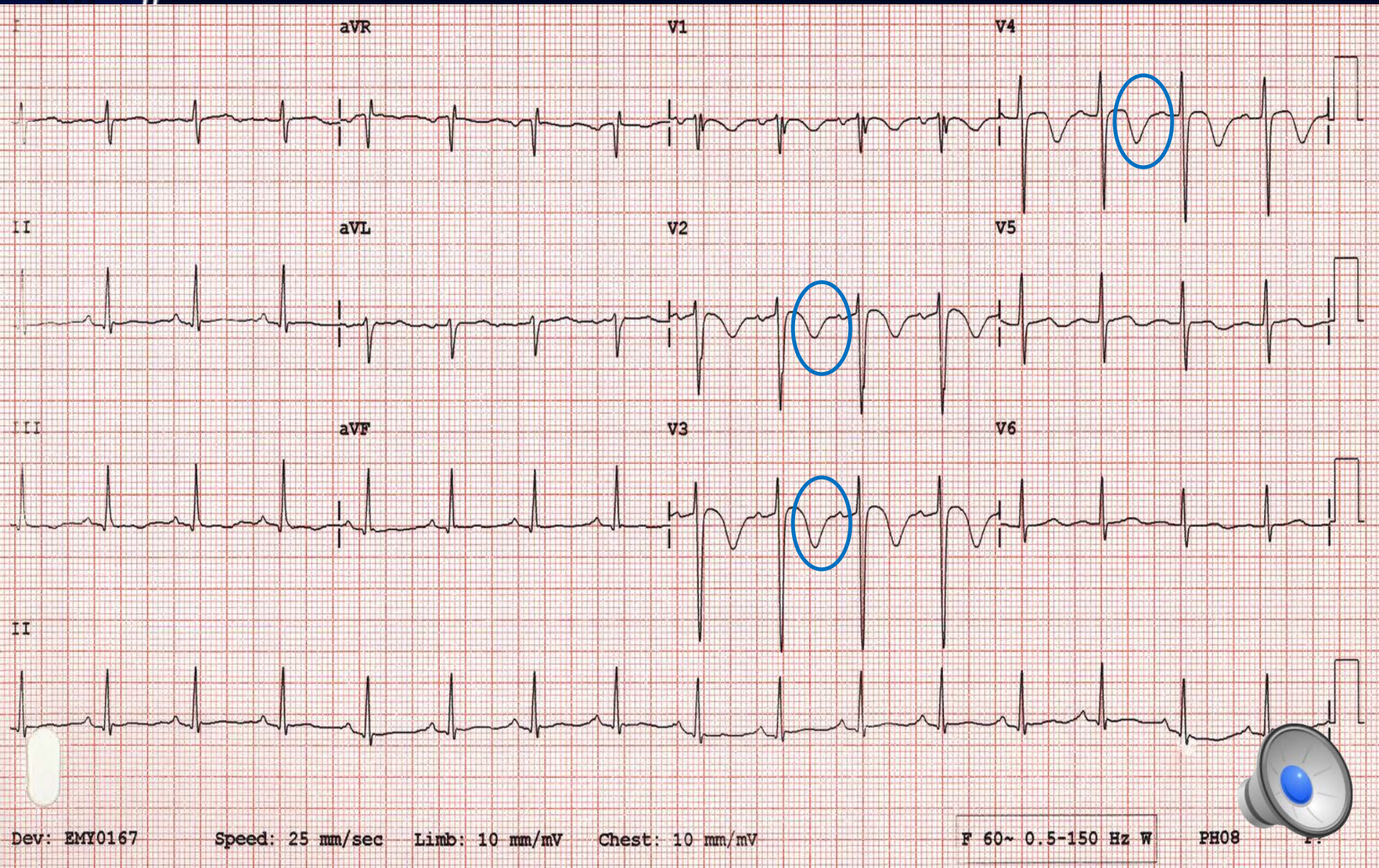
- Prior history of chest pain/angina
- Chest pain with normal EKG
- Normal or minimally elevated cardiac enzymes
- No pathologic precordial Q waves or loss of R waves
- ST segment in V2 and V3 that is isoelectric or minimally elevated (1mm), concave or straight
- Symmetric and deep T-wave inversion or biphasic T-waves in V2-V5 or V6 in pain free periods
- Proximal LAD stenosis

Wellens Syndrome

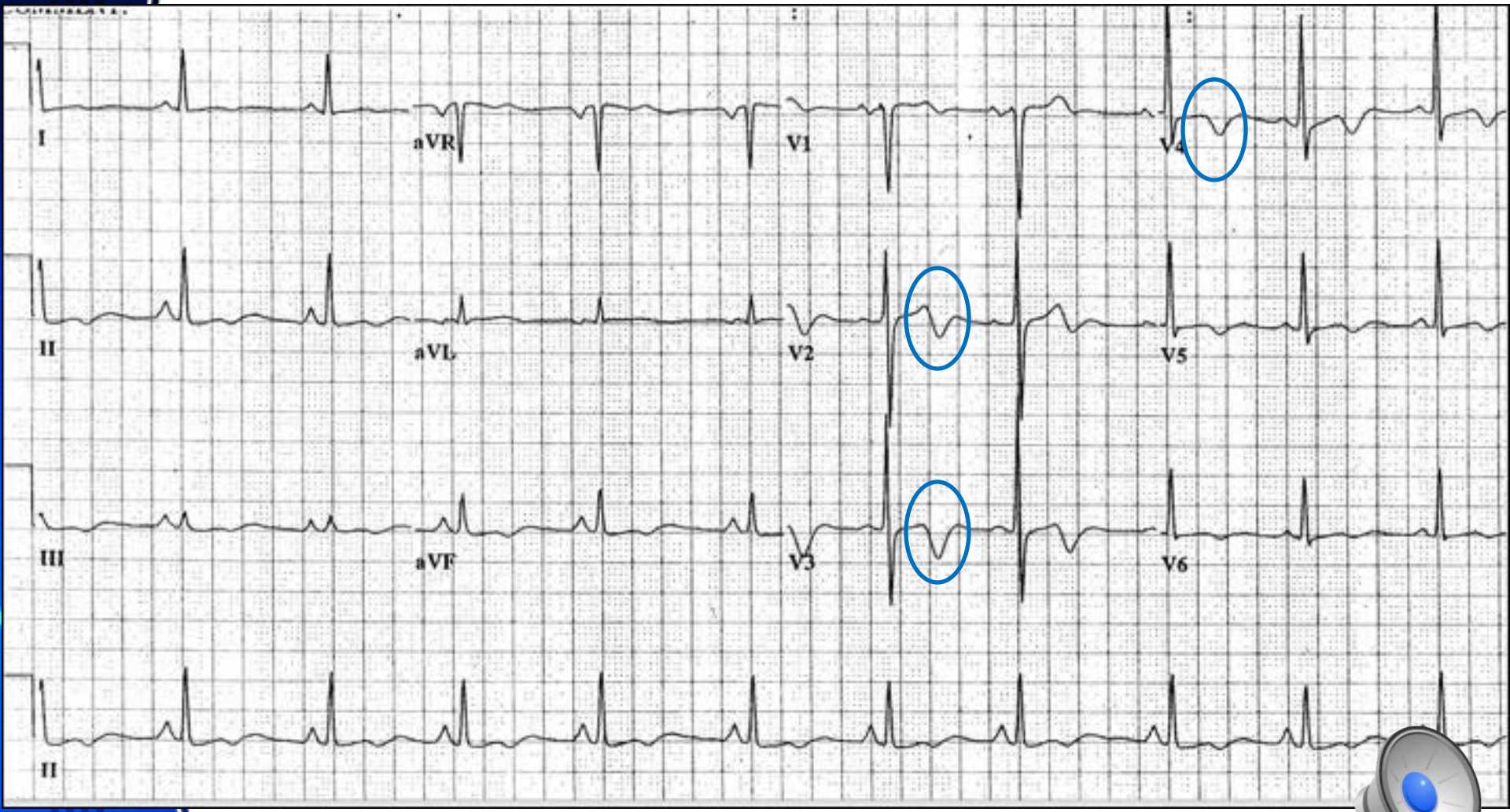


Biphasic T-wave

Wellens Syndrome Type I



Wellens Syndrome (Type II)



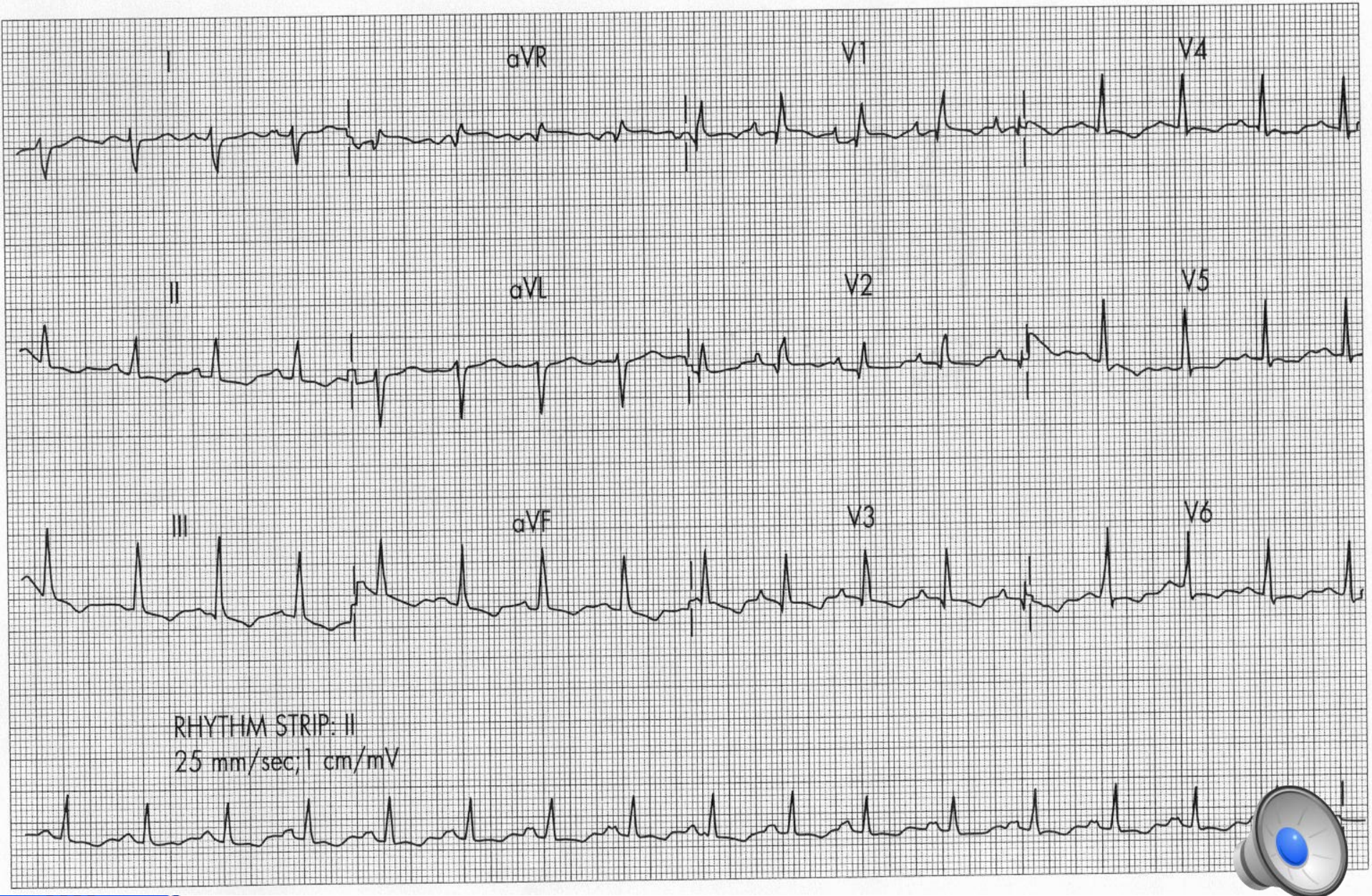
Wellens Syndrome Management

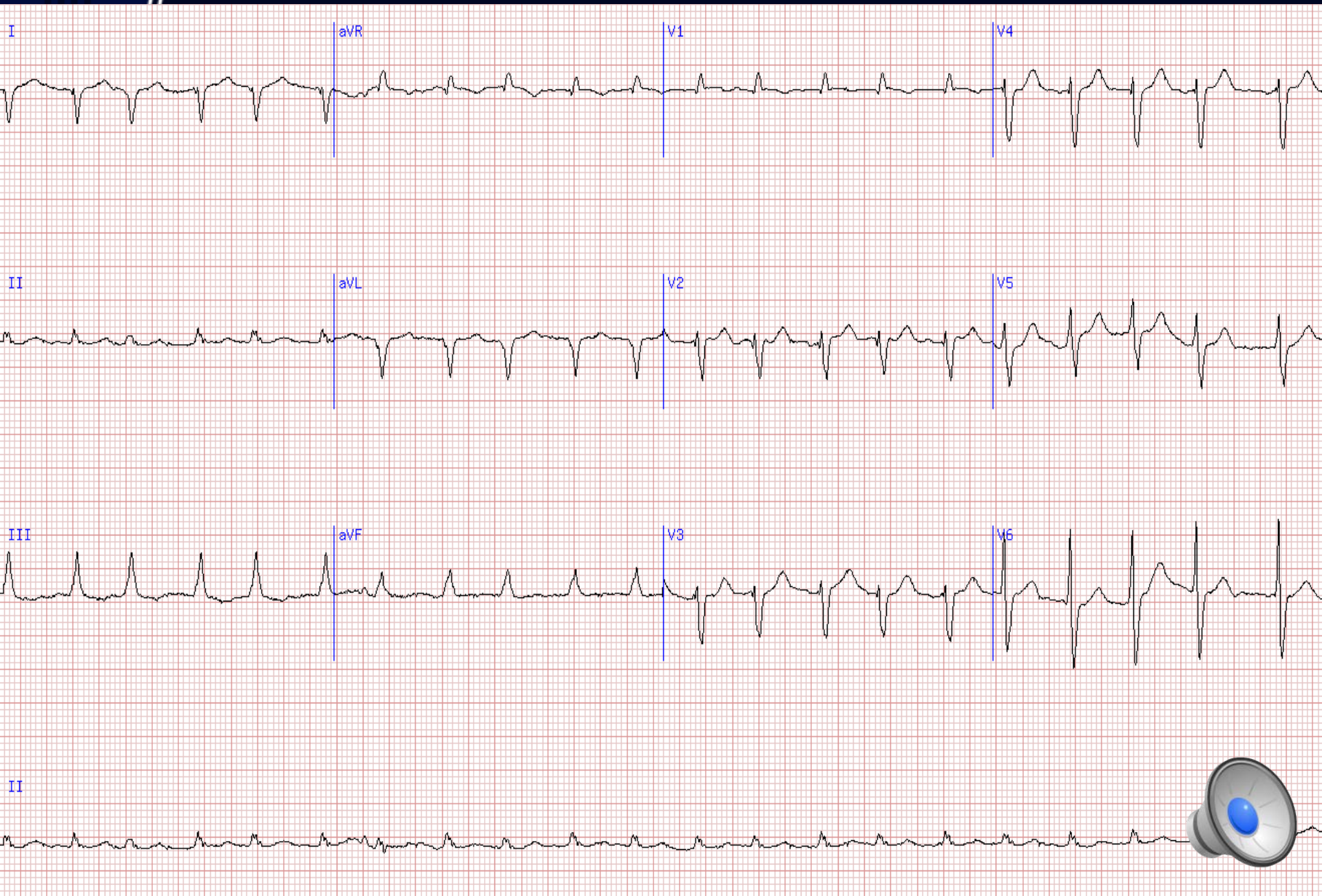
Transport to a facility capable of:

- ✓ Prompt percutaneous transluminal angioplasty
- ✓ Cardiac catheterization or
- ✓ CABG surgery

Right Ventricular Hypertrophy

- RVH increases the height of the R wave in V1. An R wave in V1 that is greater than 7 boxes in height, or larger than the S wave, is suspicious for RVH. Other findings are necessary to confirm the ECG diagnosis.
  - Other findings include right axis deviation, taller R waves in the right precordial leads (V1-V3), and deeper S waves in the left precordials (V4-V6). The T wave is inverted in V1 (and often in V2).
- True posterior infarction may also cause a tall R wave in V1, but the T wave is usually upright, and there is usually some evidence of inferior infarction (ST-T changes or Qs in II, III, and AvF).


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
RHYTHM STRIP: II
25 mm/sec;1 cm/mV


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

Left Ventricular Hypertrophy

- Caused by increased loads on the left ventricle.
- Etiologies include:
  - hypertension, aortic stenosis or regurgitation, mitral regurgitation
- Left ventricular hypertrophy (LVH) may be difficult to diagnose with certainty from the ECG.
  - Different scoring criteria have been recommended.
- One of the simplest uses five criteria, with the certainty of diagnosis based on the number of criteria present. If one is present, diagnose “possible LVH”; if two, “probable LVH”; if three are found, “definite LVH.”

Multiple LVH Criteria

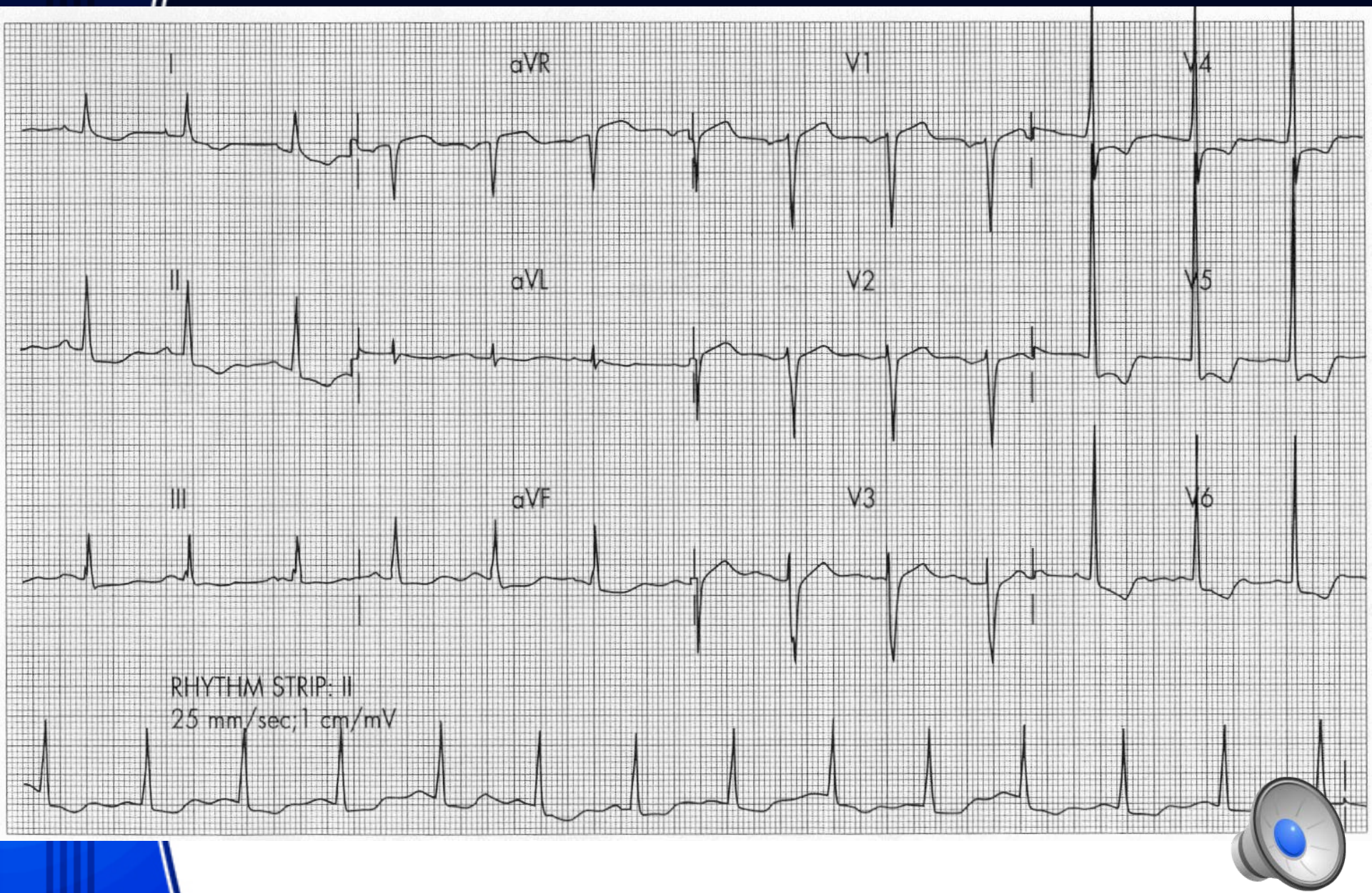
LVH Criteria #1:
Increased limb lead QRS voltage: R in lead I plus S in lead III greater than 25 mm.

LVH Criteria #2:
Increased precordial QRS voltage: S in lead V1 plus R in either V5 or V6 greater than 35 mm.

LVH Criteria #3:
Typical ST and T abnormalities: ST depression or T wave inversion (or both) in the “lateral” leads (I, aVL, V4-V6)

LVH Criteria #4: (voltage criteria)
Large leftward voltage: R wave in lead aVL greater than 11 mm, R wave in aVF > 20 mm, or S wave in aVR > 14 mm.

LVH Criteria #5:
Left atrial enlargement: Wide (greater than 0.11 msec) P wave or LAD. These criterion are used IN SUPPORT of the diagnosis, r alone.


I
aVR
V1
V4
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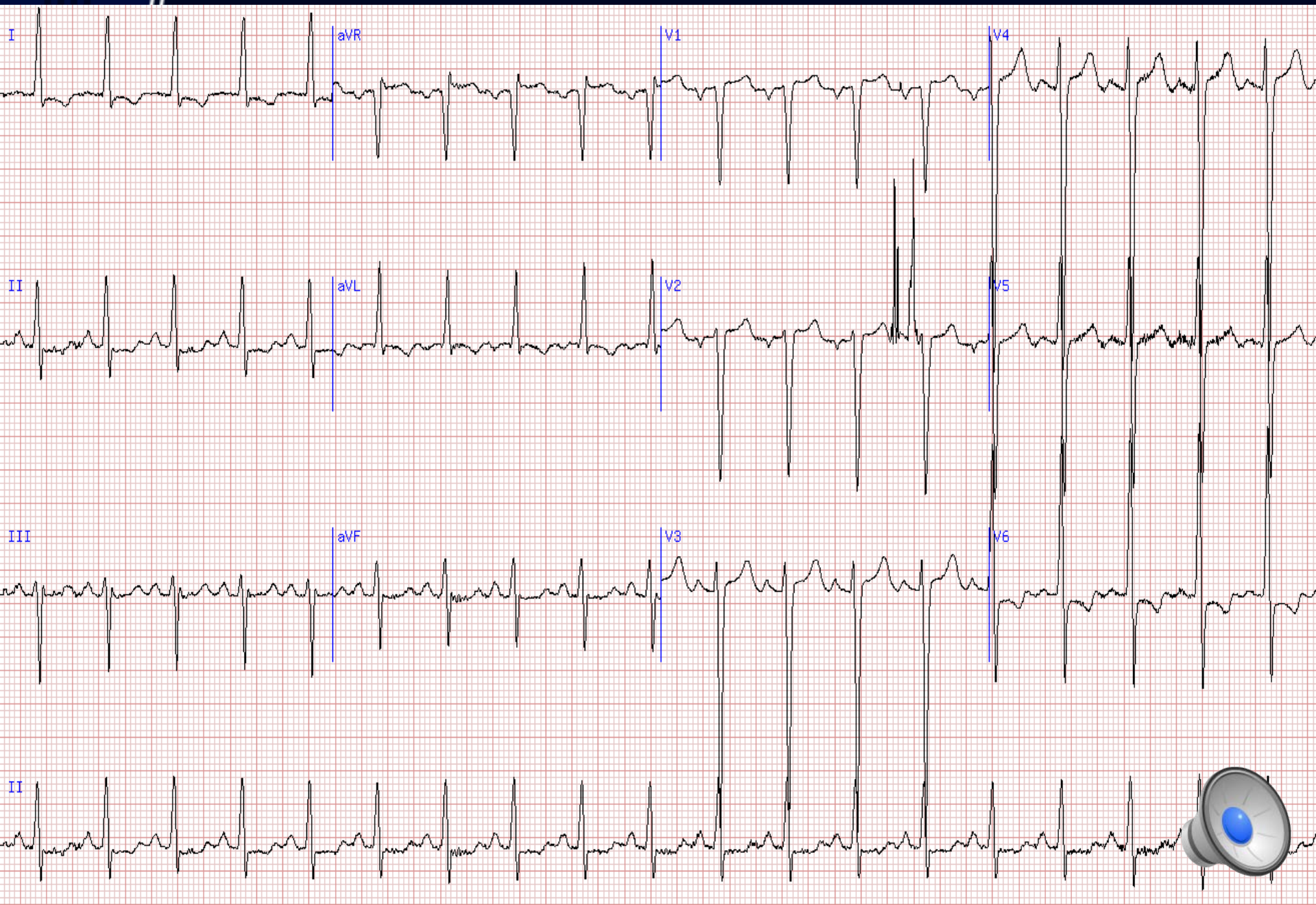
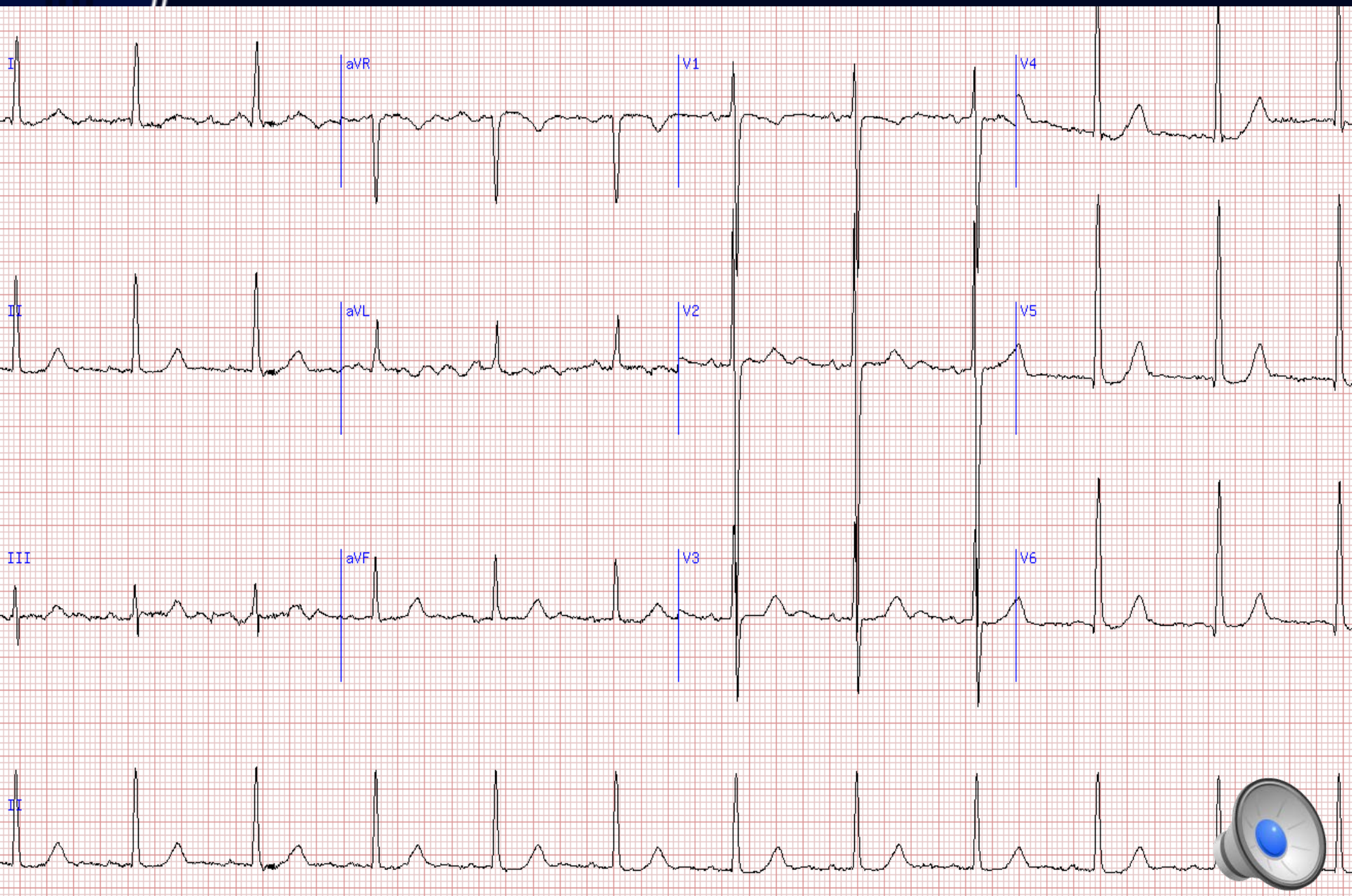
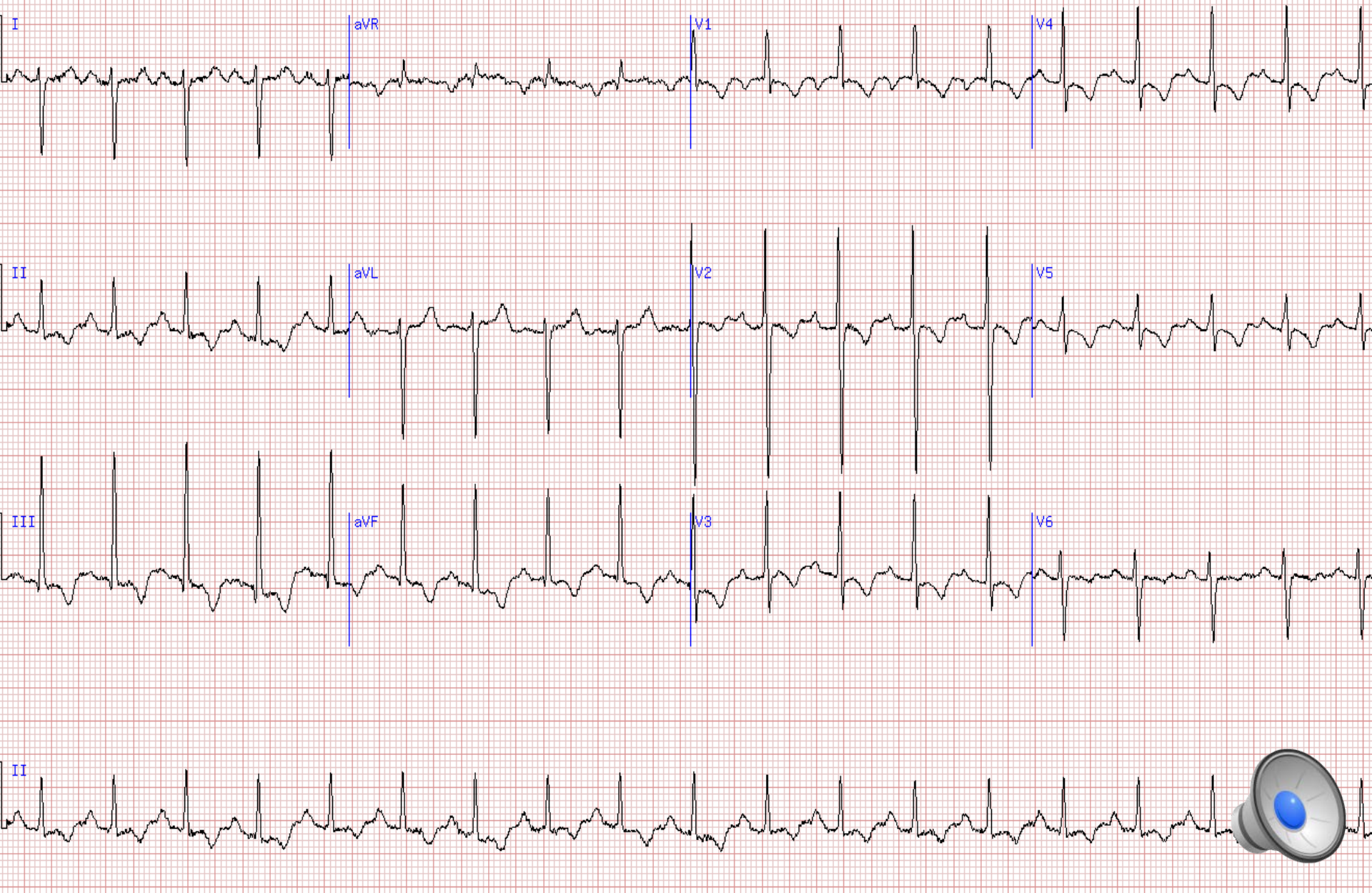
V3
V6
RHYTHM STRIP: II
25 mm/sec;1 cm/mV


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

Right or Left?



Right or Left?

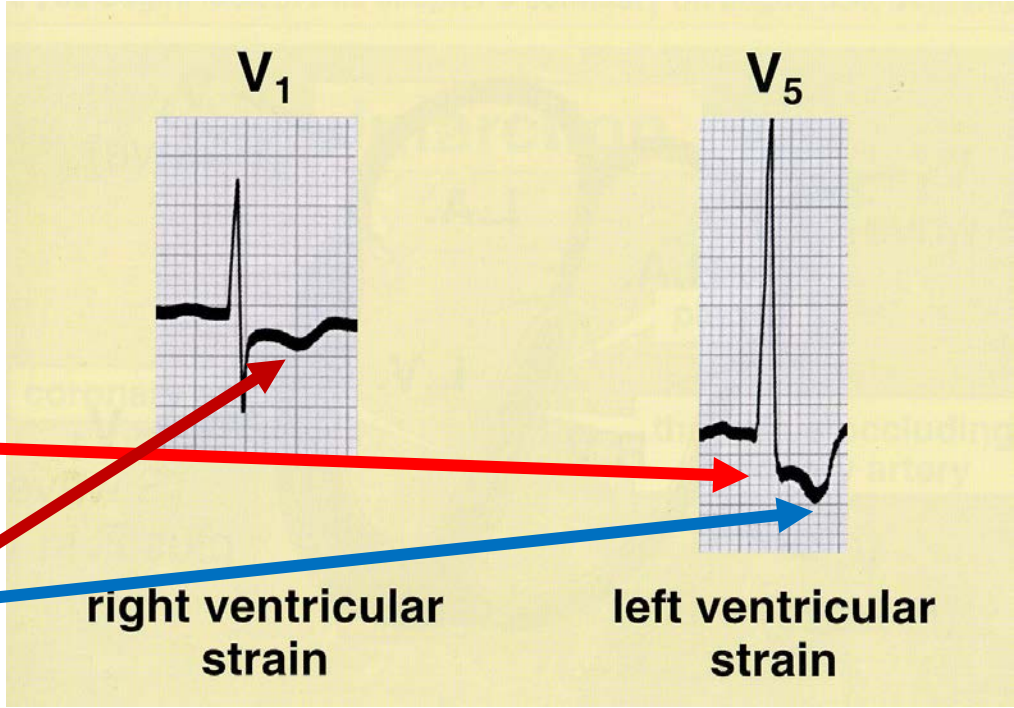


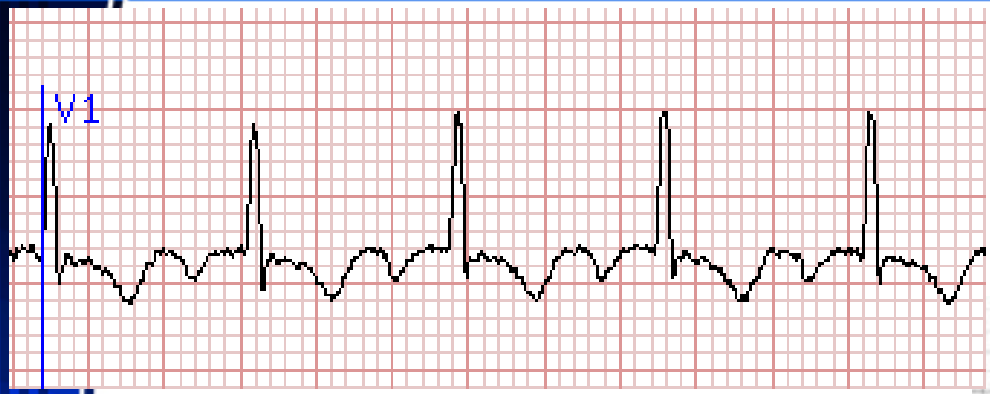
Strain in Hypertrophy

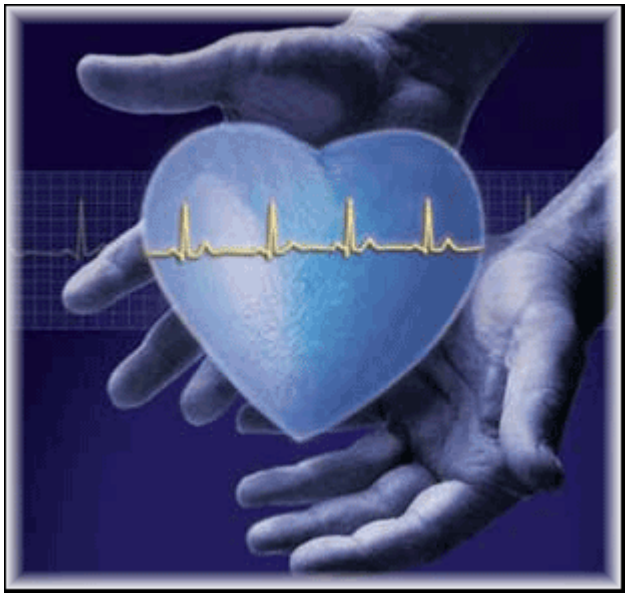
- Strain is usually associated with ventricular hypertrophy since a ventricle that is straining against some kind of resistance will become hypertrophied in its attempt to compensate.
- Ventricular strain depresses the ST segment, which generally humps upward in the middle of the segment.

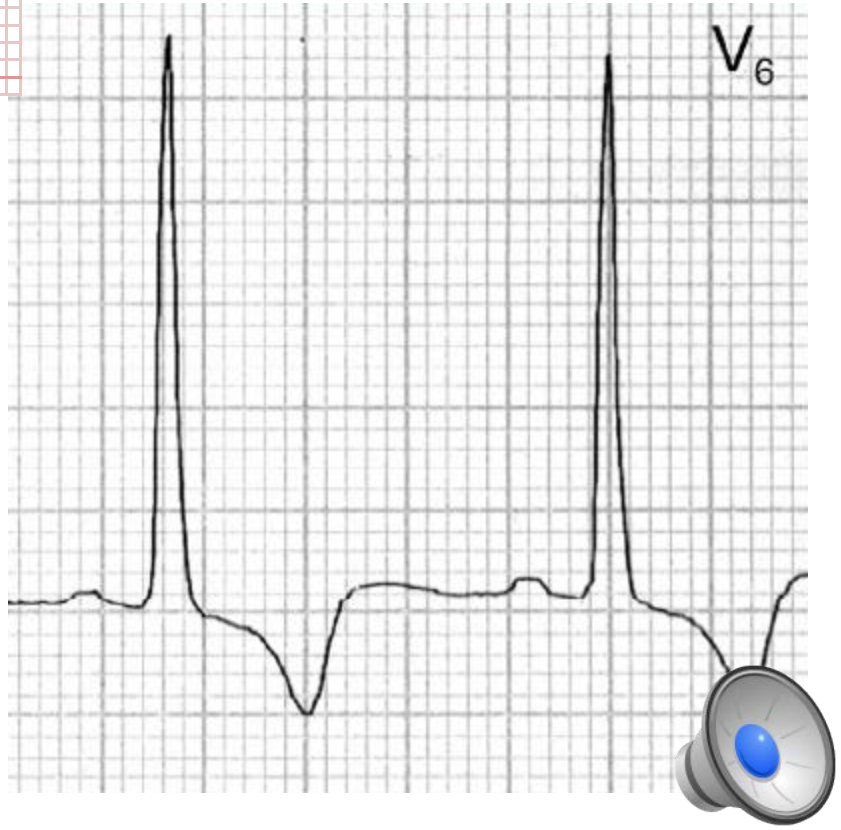
Strain

- Look for these changes when hypertrophy is present to determine if there is strain present:
  - Downsloping ST segment & ST depression
  - T-wave inversion

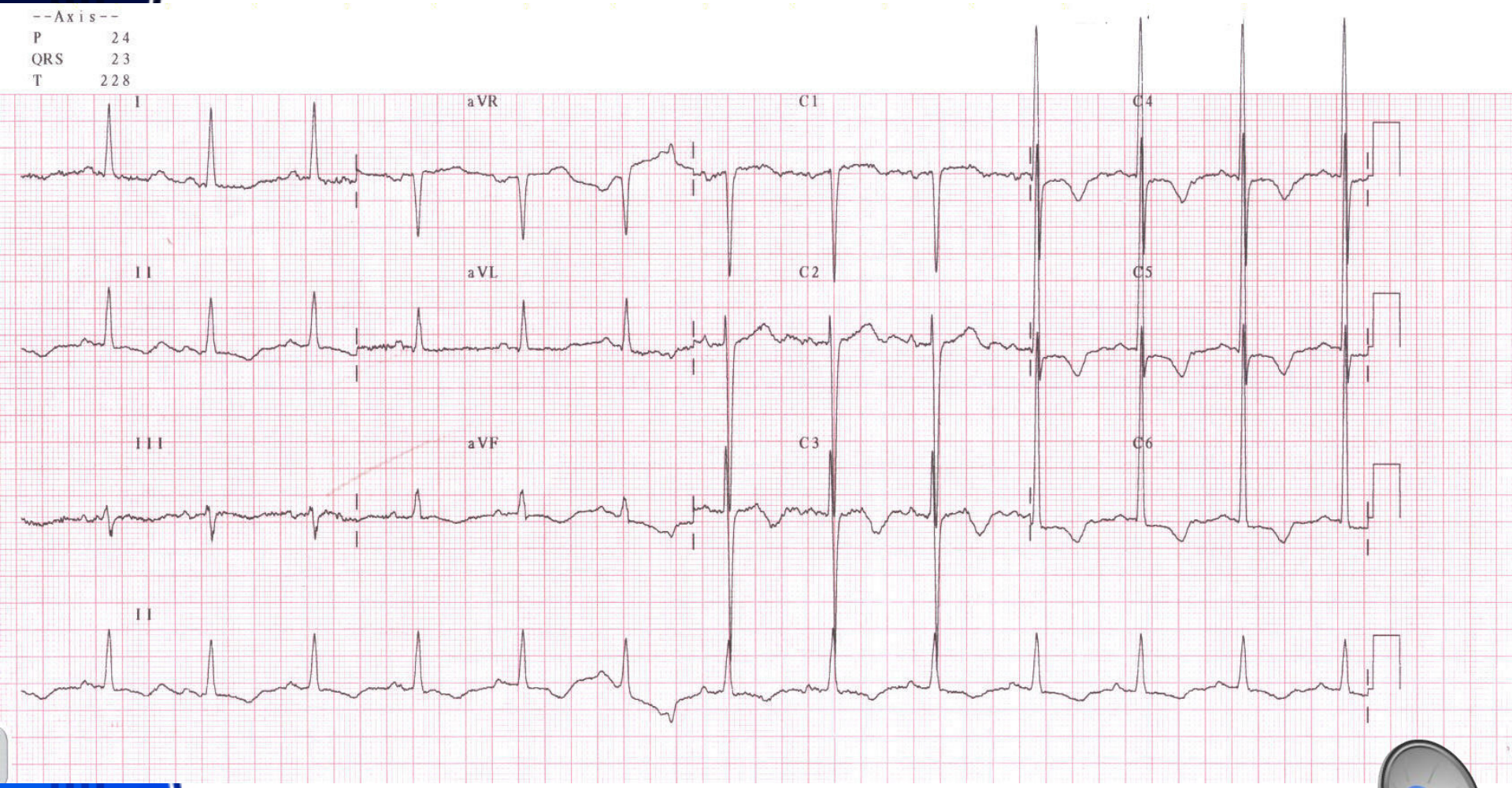



V1




V6

LVH with Strain

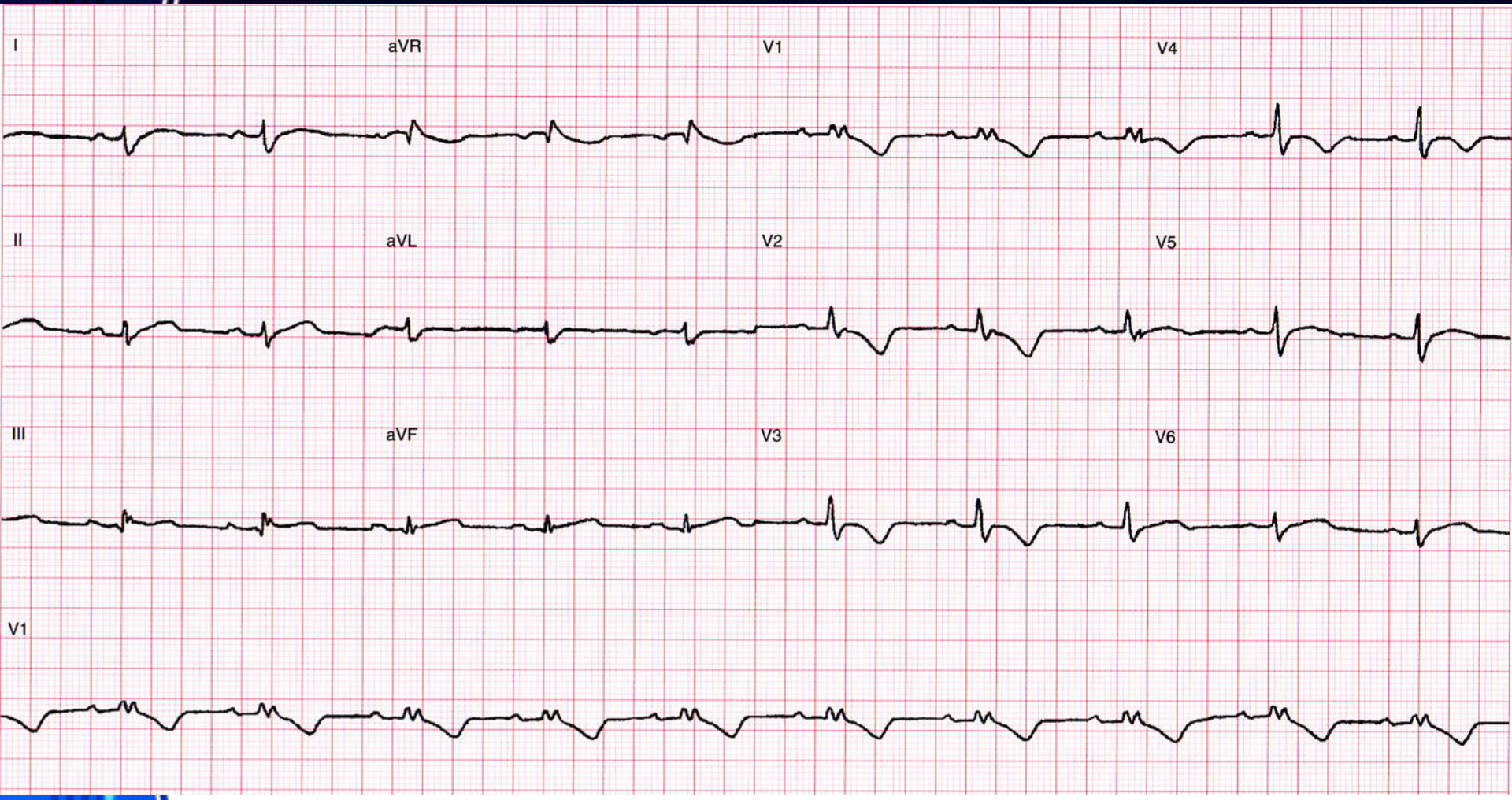


Summary

- **RVH Criteria**
  R in V1 > 7 mm or > S wave
  T in V1 inverted
  Right axis deviation
  S waves in V5-V6

- **LVH Criteria**
  1) R-I + S-III >25 mm
  2) S-V1-2 + R-V5-6 >35 mm
  3) ST-Ts in left leads
  4) R-L >11 mm
  5) LAE + other criteria
- Positive Criteria:
  1=possible 2=probable
  3=definite

So what's wrong with this patient?

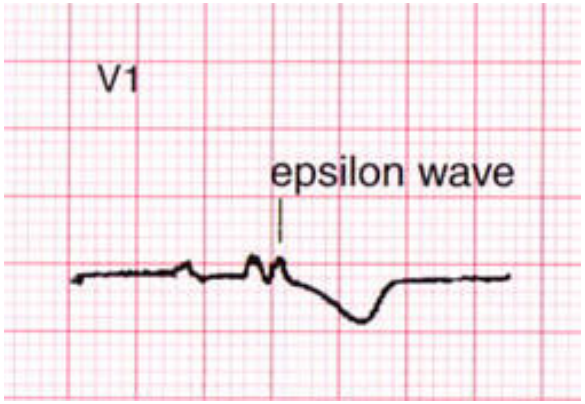


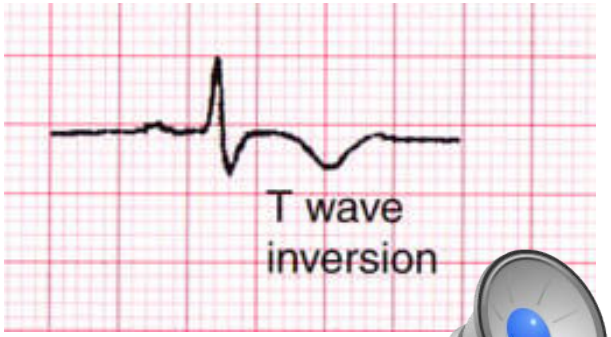
A 35 year old male presents following a syncopal episode while running.

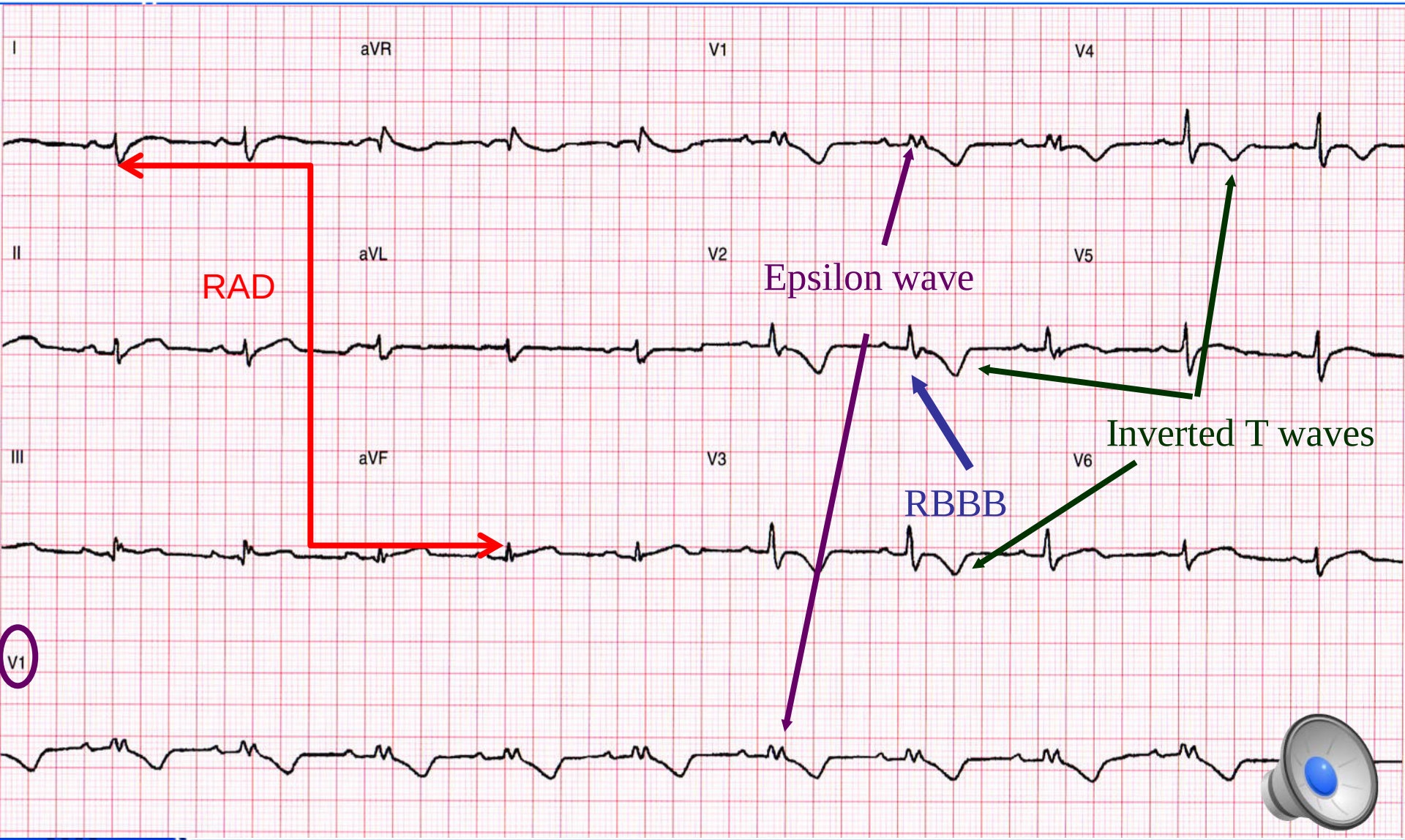
Arrhythmogenic Right Ventricular Dysplasia

Features of this EKG

- SR w/1^0 AVB, RAD, ischemic changes anteriorly
- Features of ARVD:
  - Incomplete BBB (V3)
  - Epsilon wave in V1-2
  - T-wave inversion in V1-4






I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
RAD
Epsilon wave
RBBB
Inverted T waves

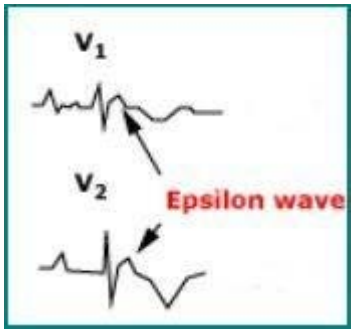
Arrhythmogenic Right Ventricular Dysplasia

- Incidence 1:5000
- 30-40% inherited: autosomal dominant with incomplete penetrance
- No specific genetic test
- Usually presents in young adult male > female
- After HCM, commonest cause of death during exertion in young adults

ARVD

- Progressive disorder anatomically
- Fatty/fibrous infiltration of right ventricle
- Patchy initially, more diffuse later
- Septal and LV involvement only at advanced stage

- RV dysfunction (late)
- Arrhythmia (potentially fatal)

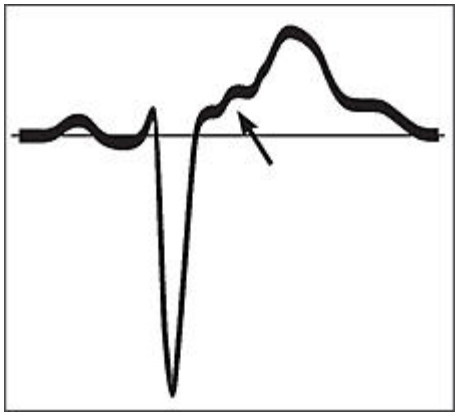


ARVD
Clinical Features

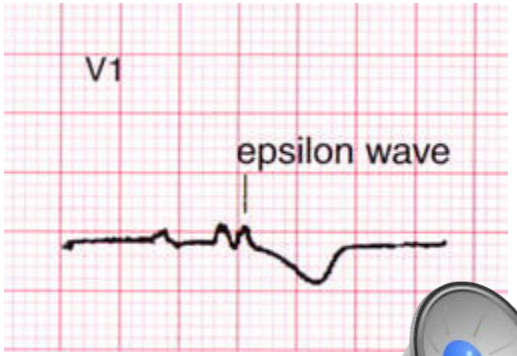
- Presentation
  - Dyspnea, palpitation, dizziness, syncope
  - Abnormal ECG
  - Frequent VEs
  - Abnormal echo
  - Sudden death
- Characteristic VT (or VEs)
  - LBBB pattern with inferior axis

EKG Features of ARVD

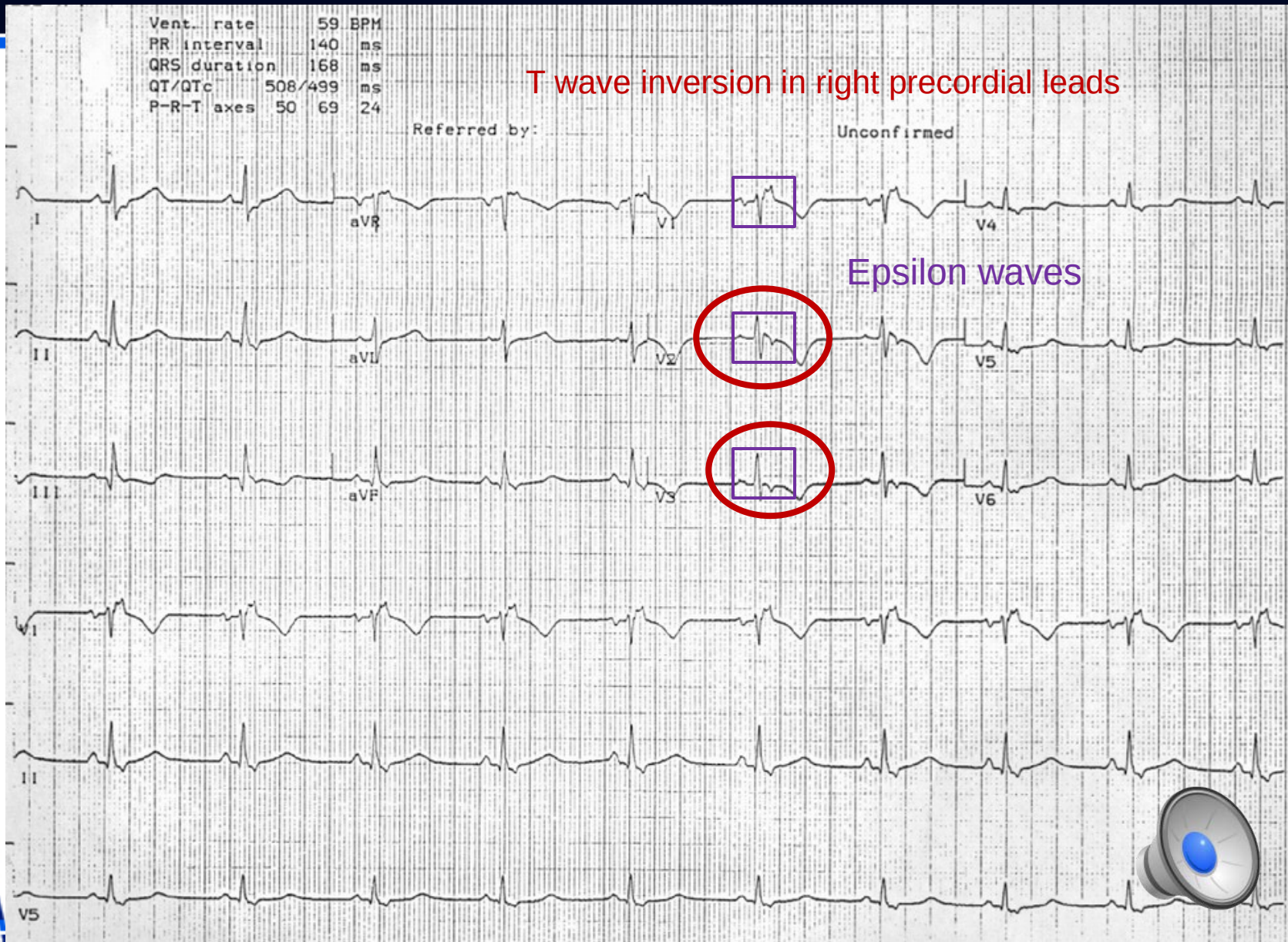
- Complete or incomplete BBB (may have either L or RBBB)
- Epsilon wave
  - Epsilon waves are small deflections just beyond the QRS complex; they are best visualized in leads V1 through V3.
  - Any potential in leads V1 through V3 that exceeds the QRS duration in lead V6 by more than 25 milliseconds should be considered an epsilon wave.
- T wave inversion in the right precordial leads.



Epsilon wave – a late positive deflection in the terminal QRS complex



ARVD


Vent. rate 59 BPM
PR interval 140 ms
QRS duration 168 ms
QT/QTc 508/499 ms
P-R-T axes 50 69 24
Referred by:
Unconfirmed
T wave inversion in right precordial leads
Epsilon waves
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5

ARVD
High Risk Features

- Extent of myocardial involvement (progressive)
  - Extensive RV involvement
  - LV involvement
- History of syncope/cardiac arrest
- Family history of sudden death
- Early onset of symptoms
- VT on Holter
- Sudden death may occur in absence of these features

ARVD Treatment

- Reduce symptoms due to VT
  - B blocker (sotalol)
  - Amiodarone
- Reduce risk of sudden death
  - Lifelong screening (echo/MRI, Holter monitor)
  - ? Drugs (stimulants/nicotine)
  - Avoid competitive sport
  - ICD in selected cases
  - Surgical RV isolation

Screening in ARVD

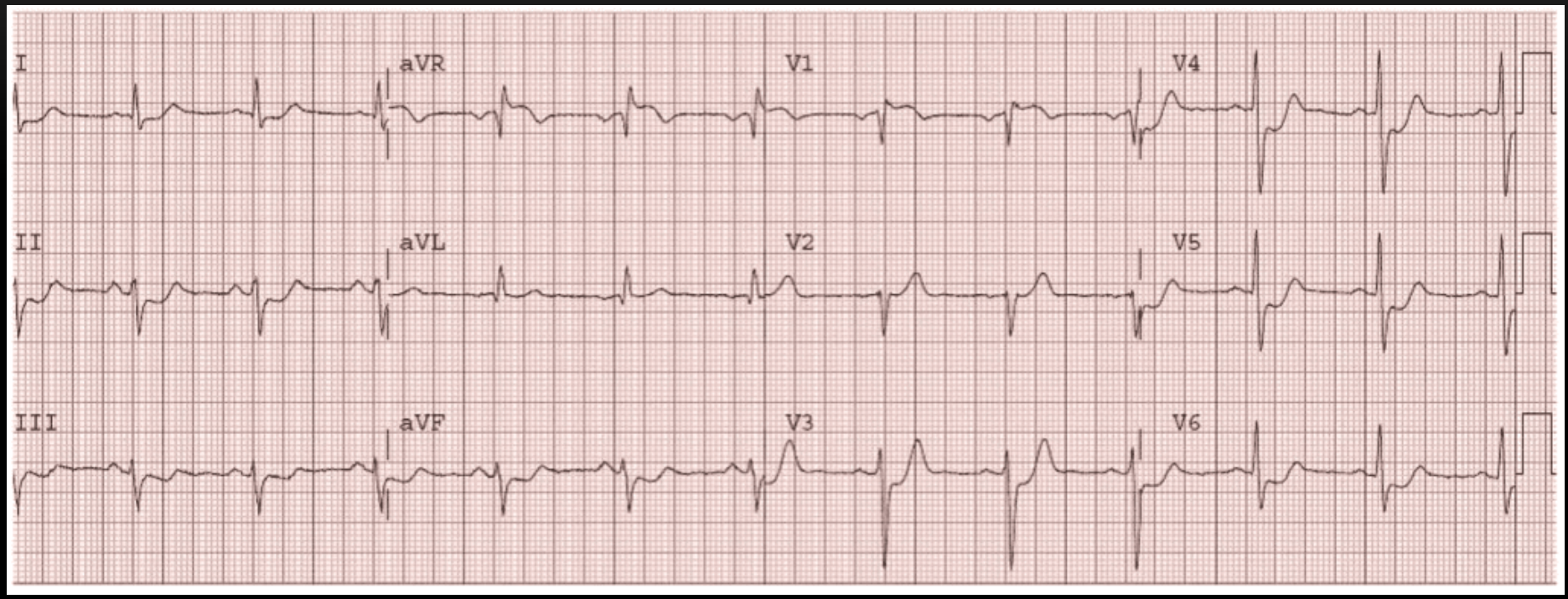
- 50% risk in 1st degree relatives
- Diagnostic criteria less clear
- Prognostic criteria less clear

- From 20 onwards, ECG/echo every 5 years.

Would Have Never Guessed

- A 59-year-old man with a history of hypertension, smoking 1 ppd for 40 years, and body mass index (BMI) of 43 developed severe persistent central chest pain at rest that started this AM. His temperature is 100.0, blood pressure is 140/90 mm Hg, respirations are 22 per minute, heart rate is 70 beats per minute and O2 Sat is 92% on room air. His EKG from EMS while in route is presented to you upon arrival. What do you do?

His EKG - What Do You See?



Left Main STEMI

- Lead aVR has often been called the “forgotten lead”, but it is worth paying attention to because ST-segment elevation in aVR portents a worse prognosis in ACS.
- ST elevation in aVR ≥ 1mm is the strongest independent predictor of either severe LMCA or triple-vessel disease requiring CABG in patients with NSTEMI.
- Elevation in aVR of ≥ 0.5 mm is an independent predictor of mortality in patients with STEMI.

Left Main Coronary Artery Disease

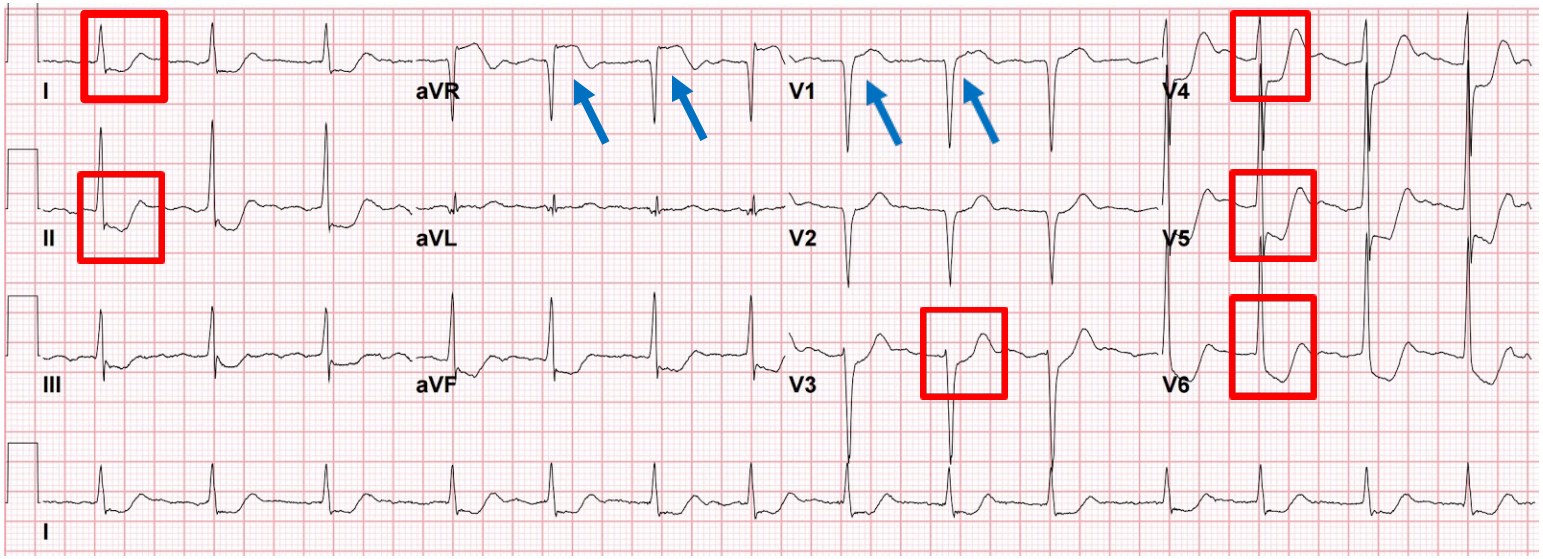
- Early identification of LMCA disease is critical because acute occlusion can cause rapid hemodynamic and electrical deterioration.
- LMCA insufficiency due to critical stenosis of the left main artery is important to recognize because these patients can progress to complete occlusion and are likely to require surgical intervention (such as CABG).

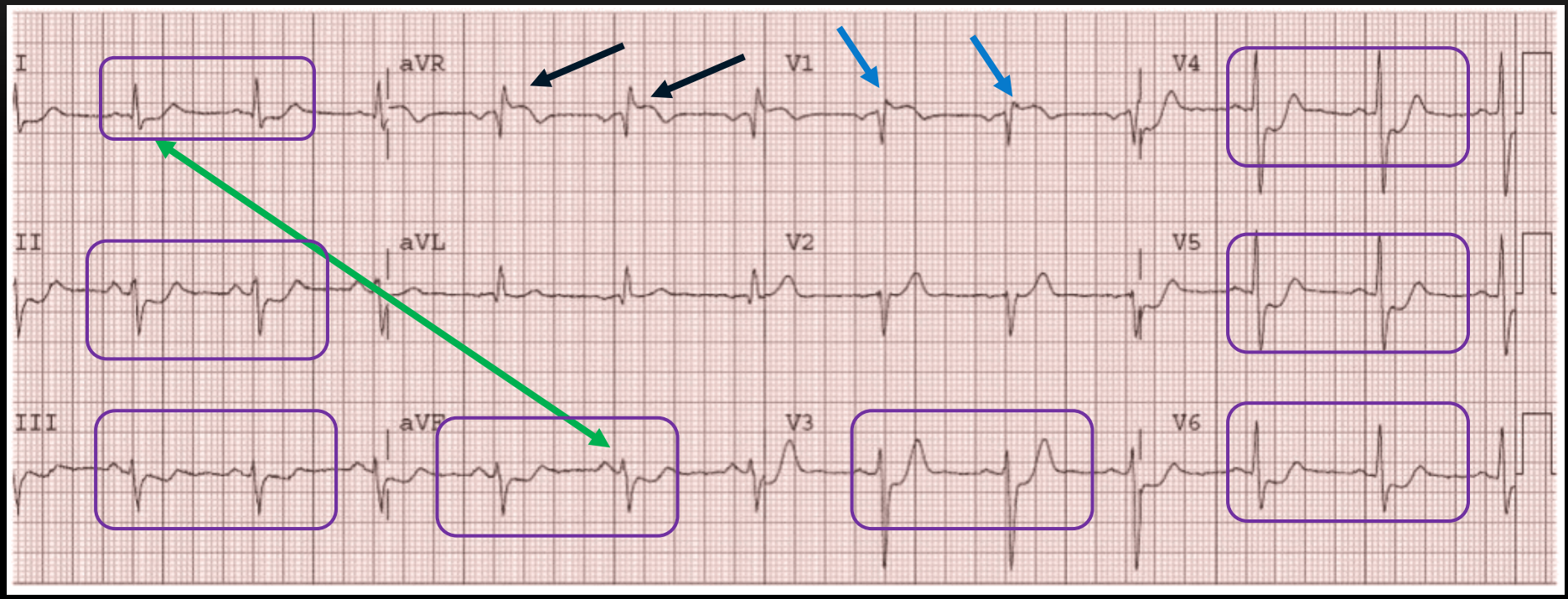
How it Occurs

- ST-elevation in aVR occurs by the following mechanisms:
  - Critical narrowing of the LMCA causing subendocardial ischemia due to insufficient blood flow.
  - Transmural infarction of the basal septum due to a very proximal LAD occlusion or complete LMCA occlusion.
  - Severe multi-vessel coronary artery disease. **
  - Diffuse subendocardial ischemia from oxygen supply/demand mismatch.

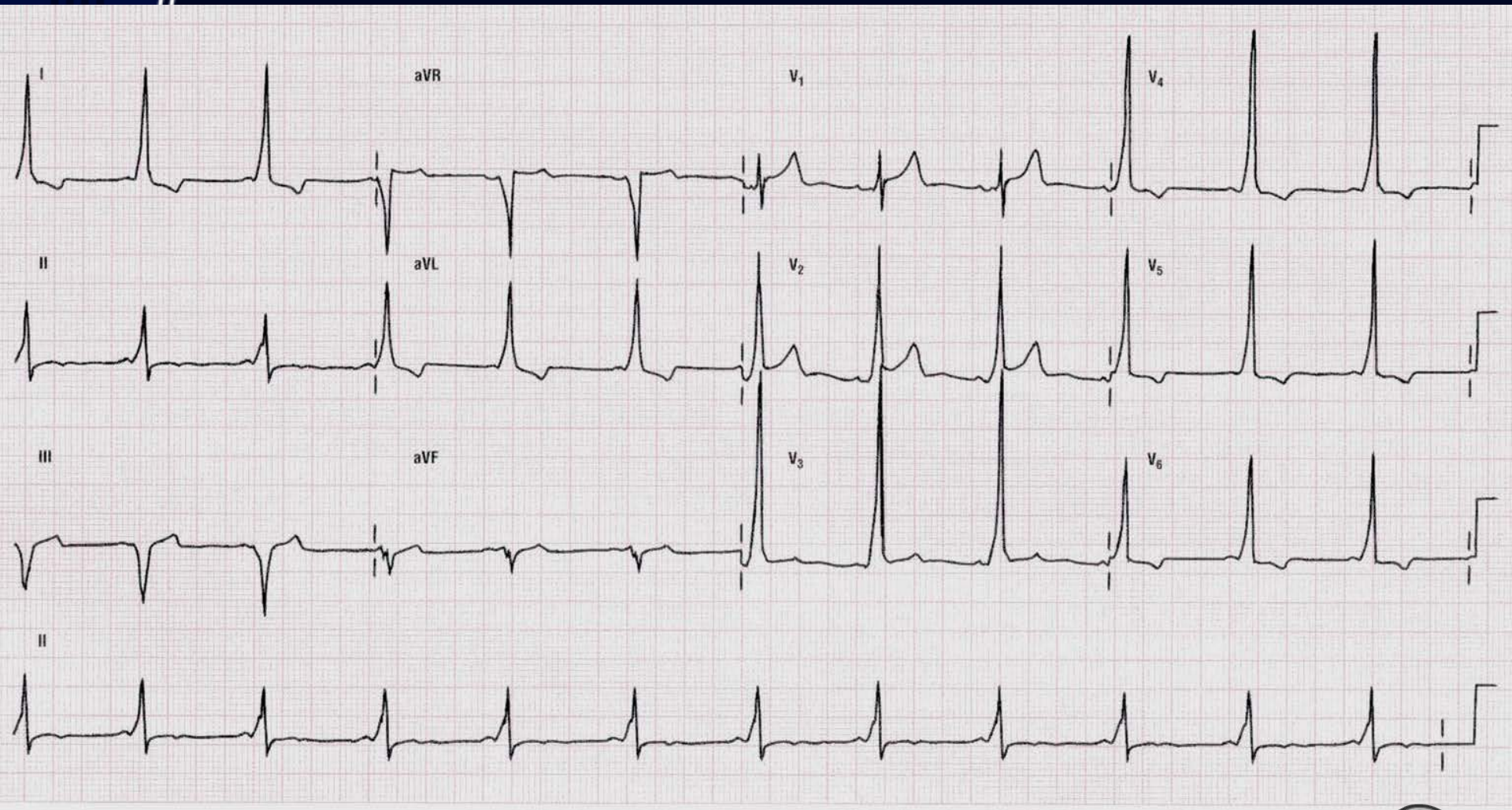
Classic Findings on ECG

- ST depression in leads I, II, aVL and V4-6
- ST elevation in aVR ≥ 1mm
- ST elevation in aVR ≥ V1




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

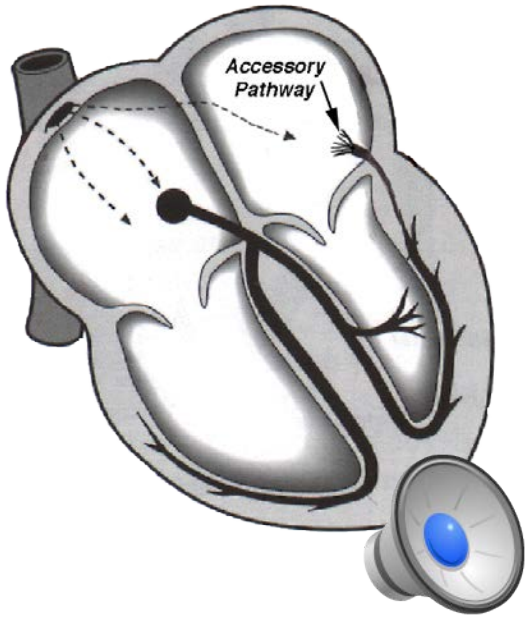
What Do You See Here?

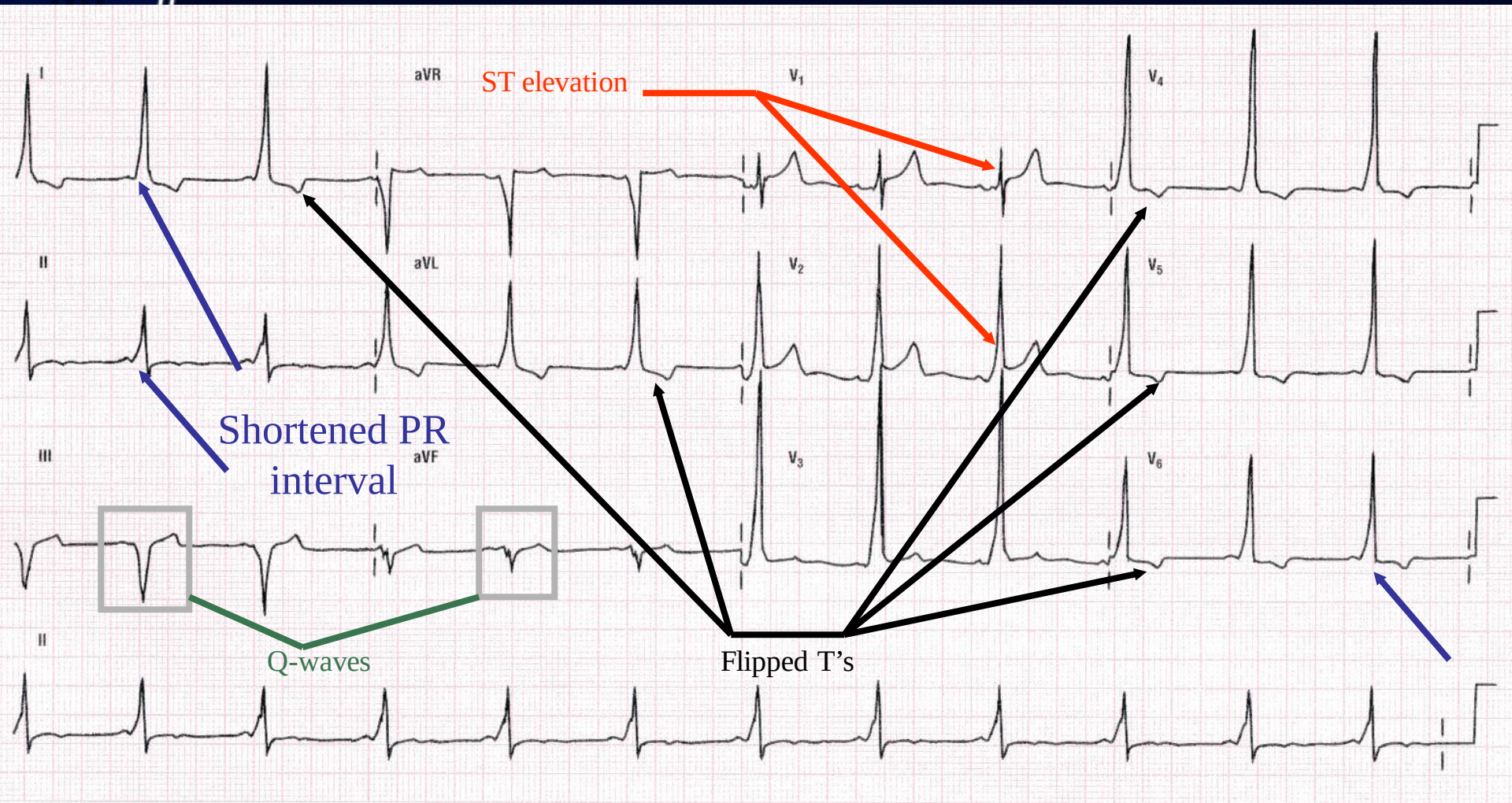


Interpretation

- Wolf-Parkinson-White Syndrome (WPW)
- The accessory conduction pathways act as short circuits, allowing the atrial wave of depolarization to bypass the AV node and activate the ventricles prematurely.

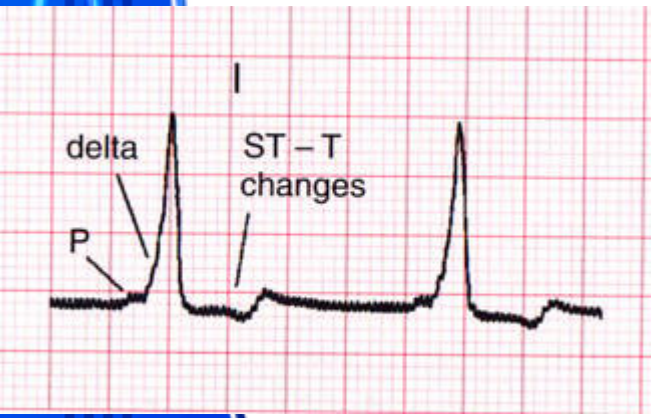
In this EKG: LAD, Q-waves, T-wave inversion in I, AVL, V3-6 representing ischemic changes.

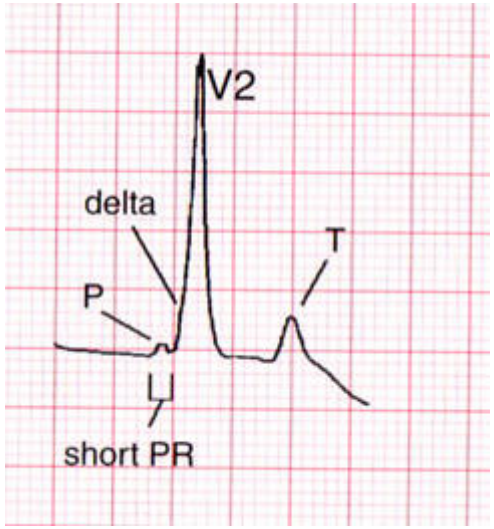


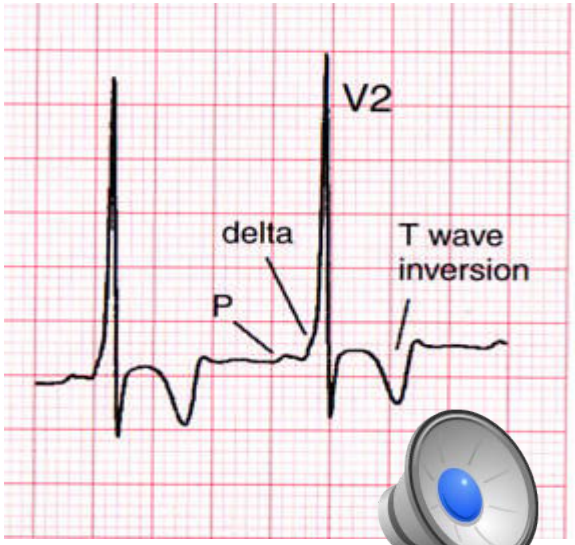

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
ST elevation
Shortened PR interval
Q-waves
Flipped T's

WPW Criteria

- Short PR interval, less than 3 small squares (< 0.12 sec) with a normal p wave
- Slurred upstroke to the QRS indicating pre-excitation (delta wave)
- Wide QRS complex (≥ 0.11 sec)
- Secondary ST and T wave changes
- Associated with paroxysmal tachycardia's

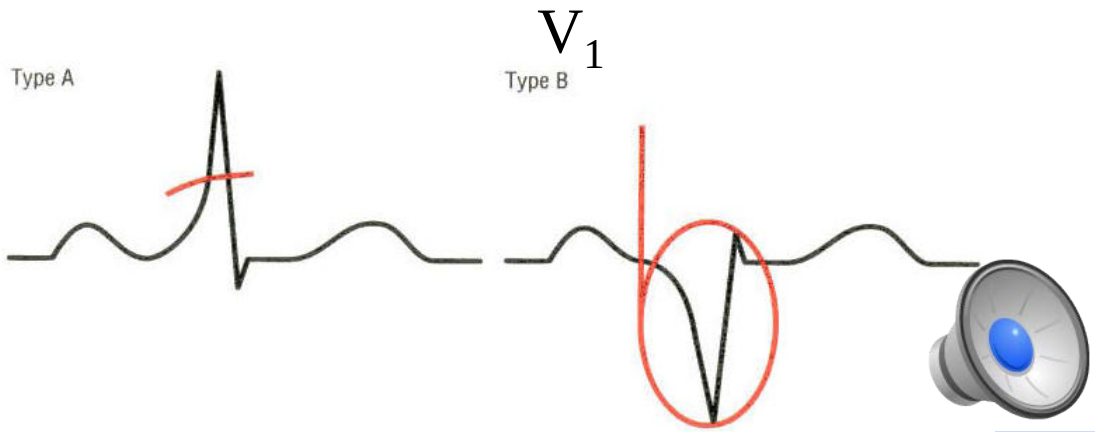




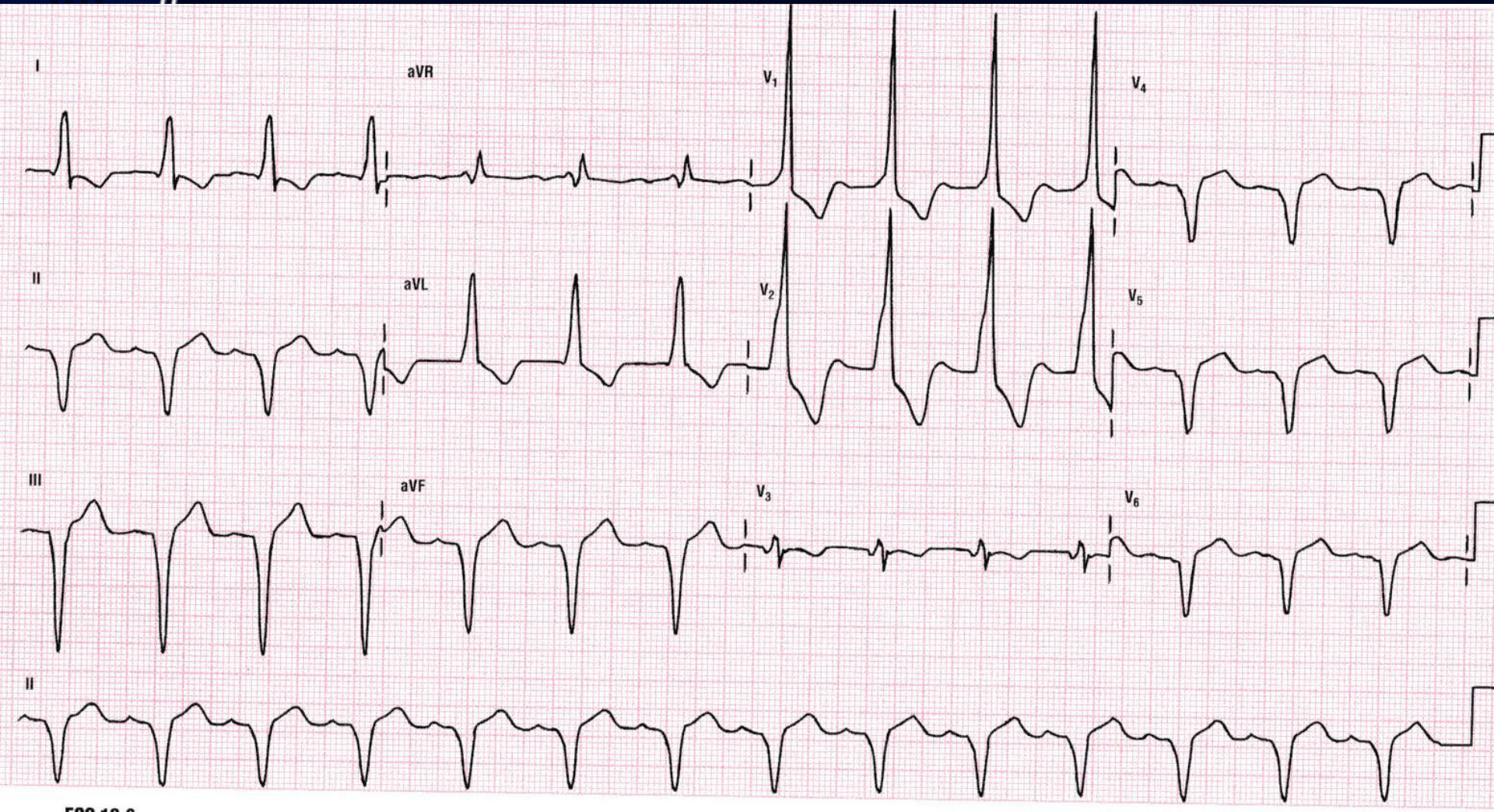


WPW Types

- Type A:
  - –QRS complexes are primarily upright in precordial leads.
  - –Associated with accessory pathways in the left side of the heart
- Type B:
  - –QRS complexes are negative in V1 or V2, and upright in the left sided precordial leads (V5-V6).
  - –Associated with accessory pathways in the right side of the heart
- Type C:
  - – QRS complexes are positive in V1-V4, and negative in V5 & V6.
  - –This type is rare

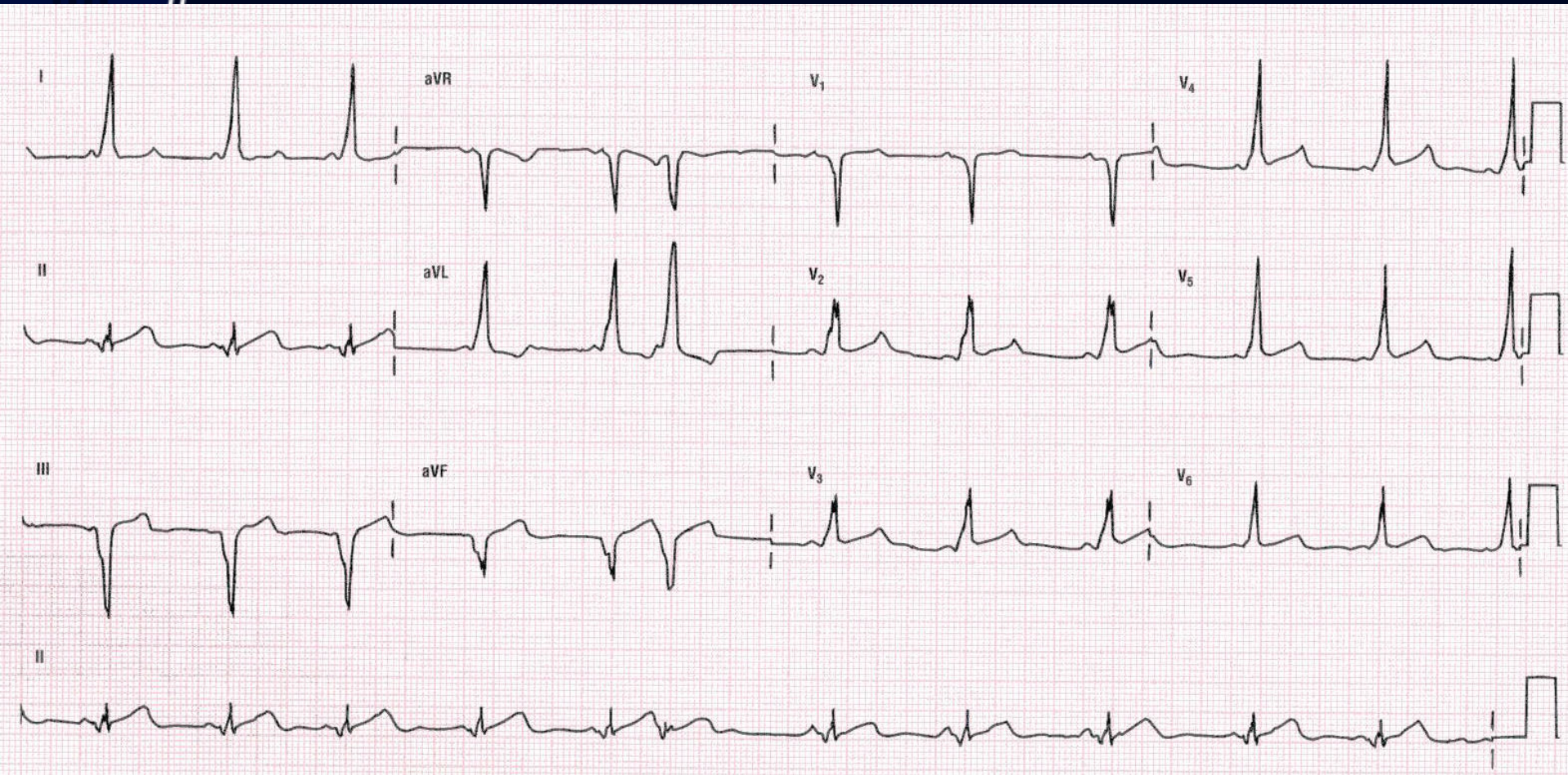


Which Type?



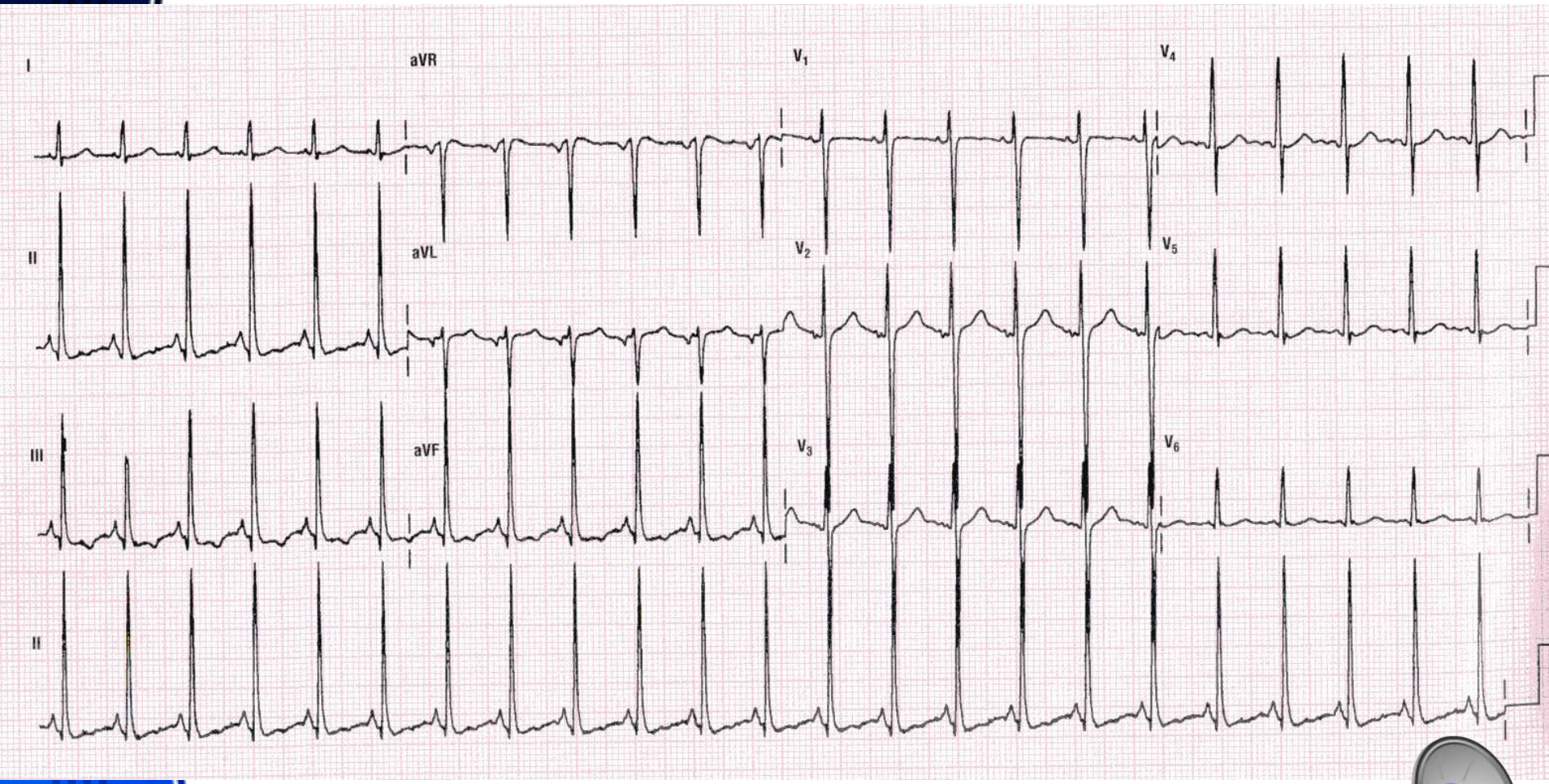
Type C. Starts off as type A but has negative delta waves in left lateral precordial leads.

Which Type?



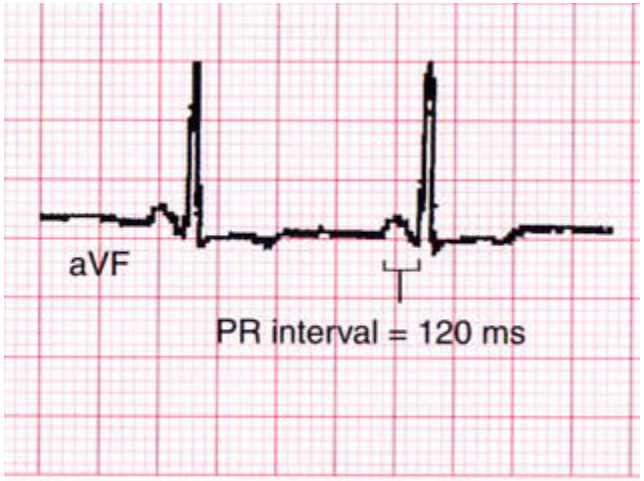
Type B with pseudoinfarct pattern in the inferior leads. Also noted RBBB & LAD (bifasicular block?). The 6th complex is an PAC. Underlying rhy
wandering atrial pacemaker

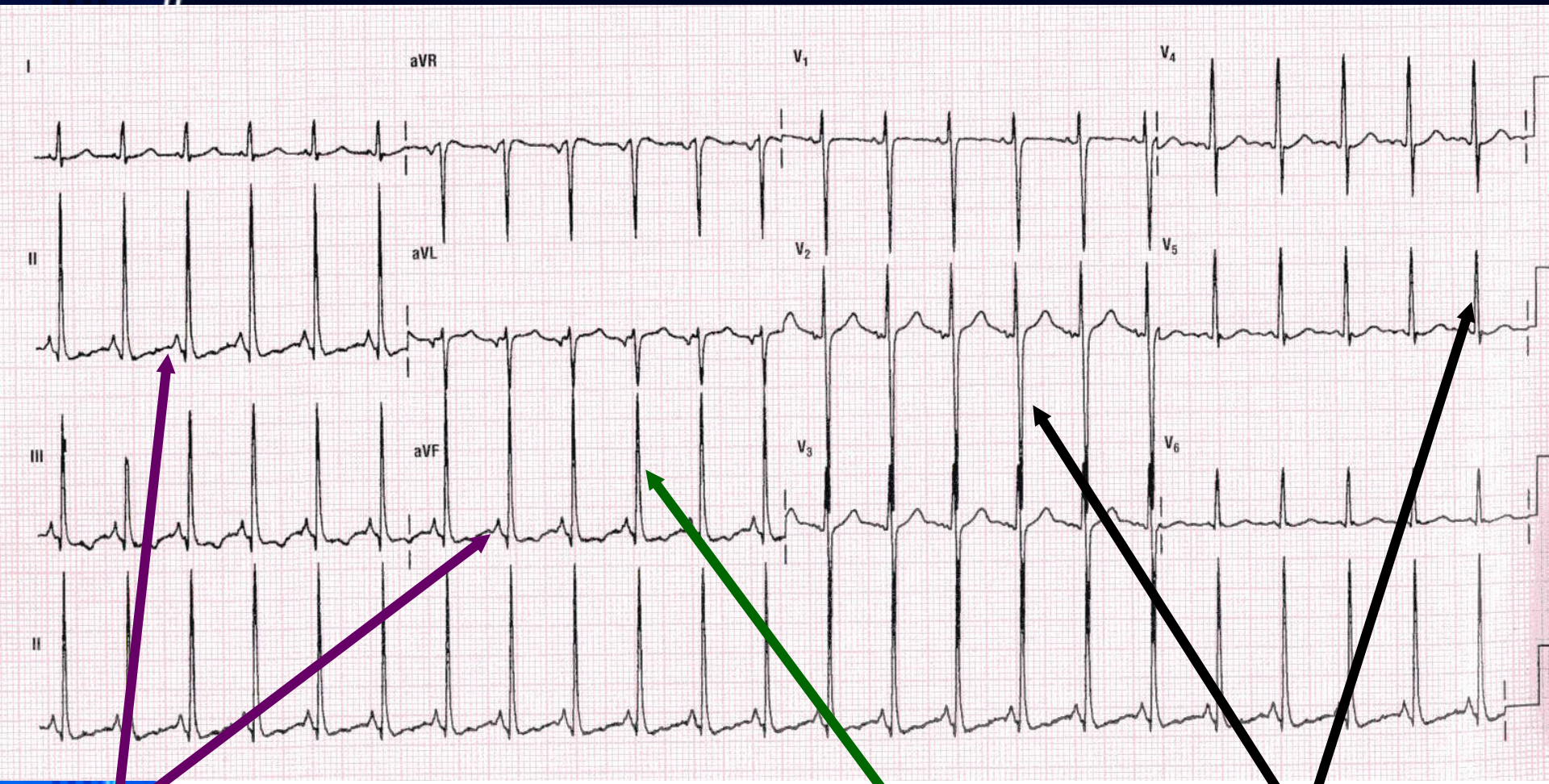
And this?



Interpretation

- Lown Ganong Levine Syndrome
- Criteria for LGL includes:
  - PR interval less than or equal to 0.12 seconds
  - Normal QRS complex duration
  - Rare occurrence of supraventricular tachycardia but not atrial fibrillation or atrial flutter.
  - No delta wave





Short PR interval, LVH by two criteria: R in aVF exceeds 20 mm & S+R is > 35 mm)

EKG Interpretation Tips

- Don’t be scared to look at all EKGs you see!
- Use a systemic approach
- Know the basic rhythms
- Learn benign ST changes
- Identify lethal rhythms first
- When considering LVH, don’t just look at the chest leads
- Be suspicious if you suspect sinus arrhythmia in the elderly
- Don’t rely on the computer interpretation!!!!!!!

Questions?

References:
Interpreting ECGs: A Practical Approach by Bruce Shade