Chamber Hypertrophy/Enlargement and Ventricular Conduction Defects

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UTHSCSA and STVAHCS

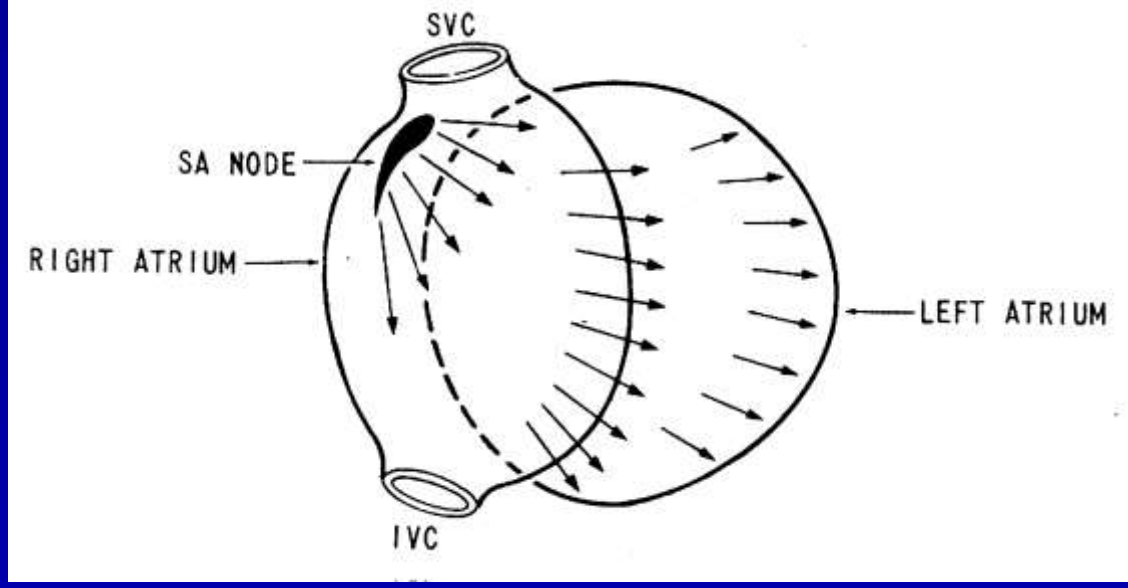
Surawicz B et al. Chou's Electrocardiography in Clinical Practice 5th ed. 2001; pp 28-74.

P wave

The normal atrial wall is only 1-2 mm thick

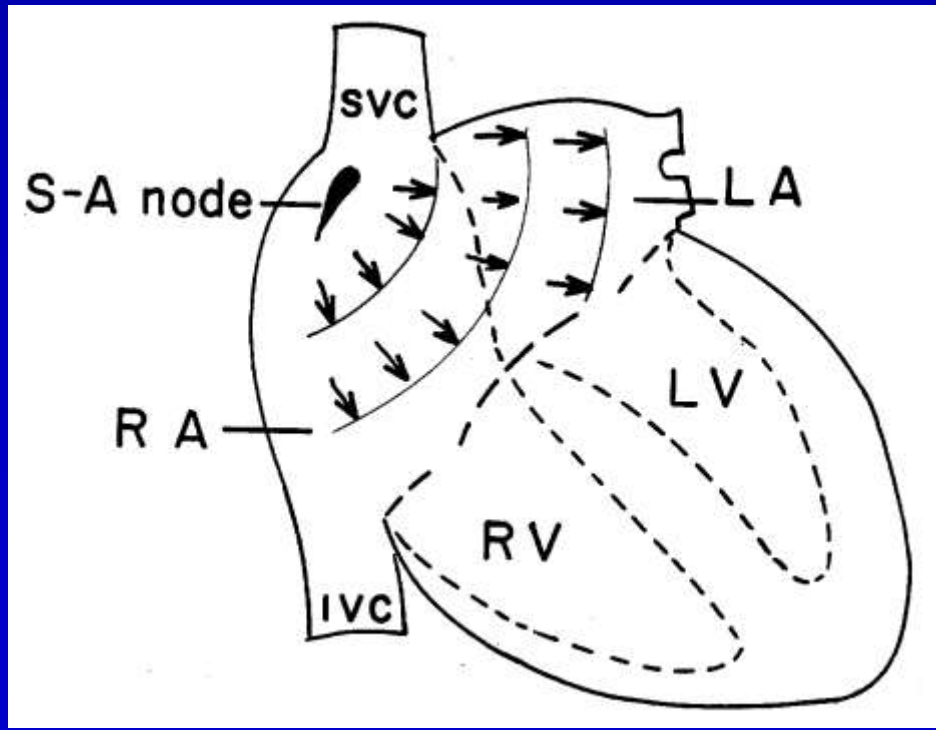
Each atrium weighs about 20 gm

Interatrial septum weighs about 10-20 gm

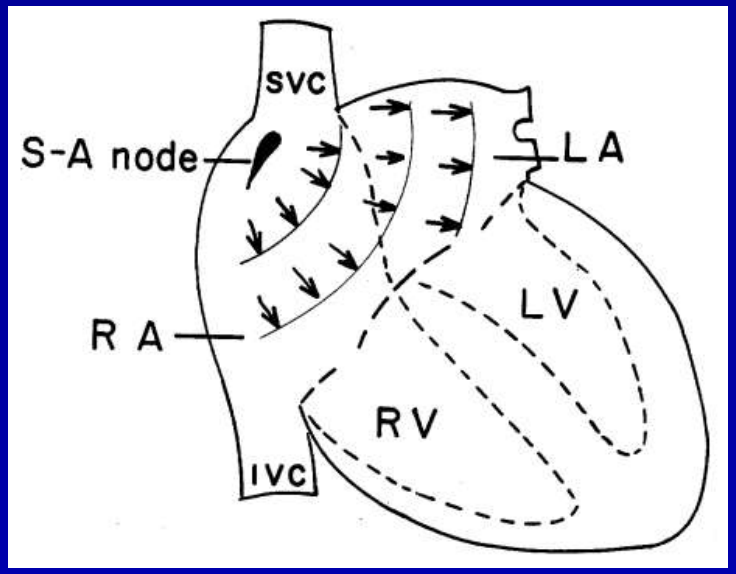


Atrial Abnormalities

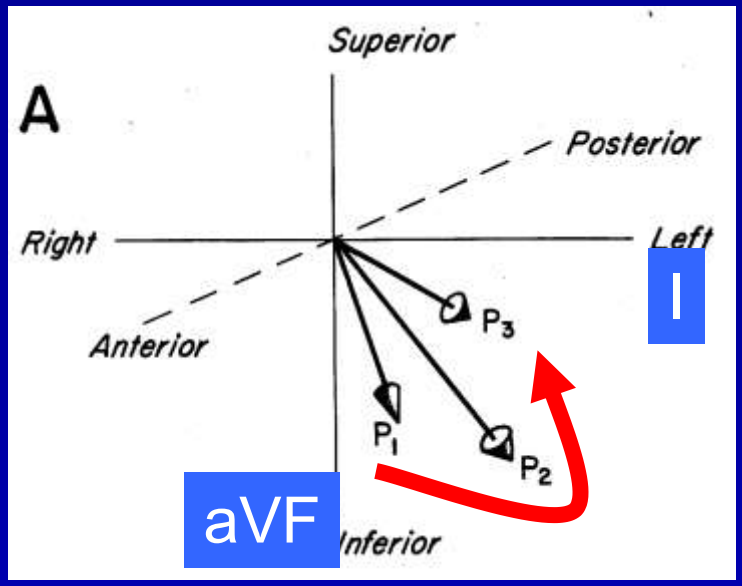
- Right atrial abnormality
- Left atrial abnormality
- Interatrial conduction disturbances

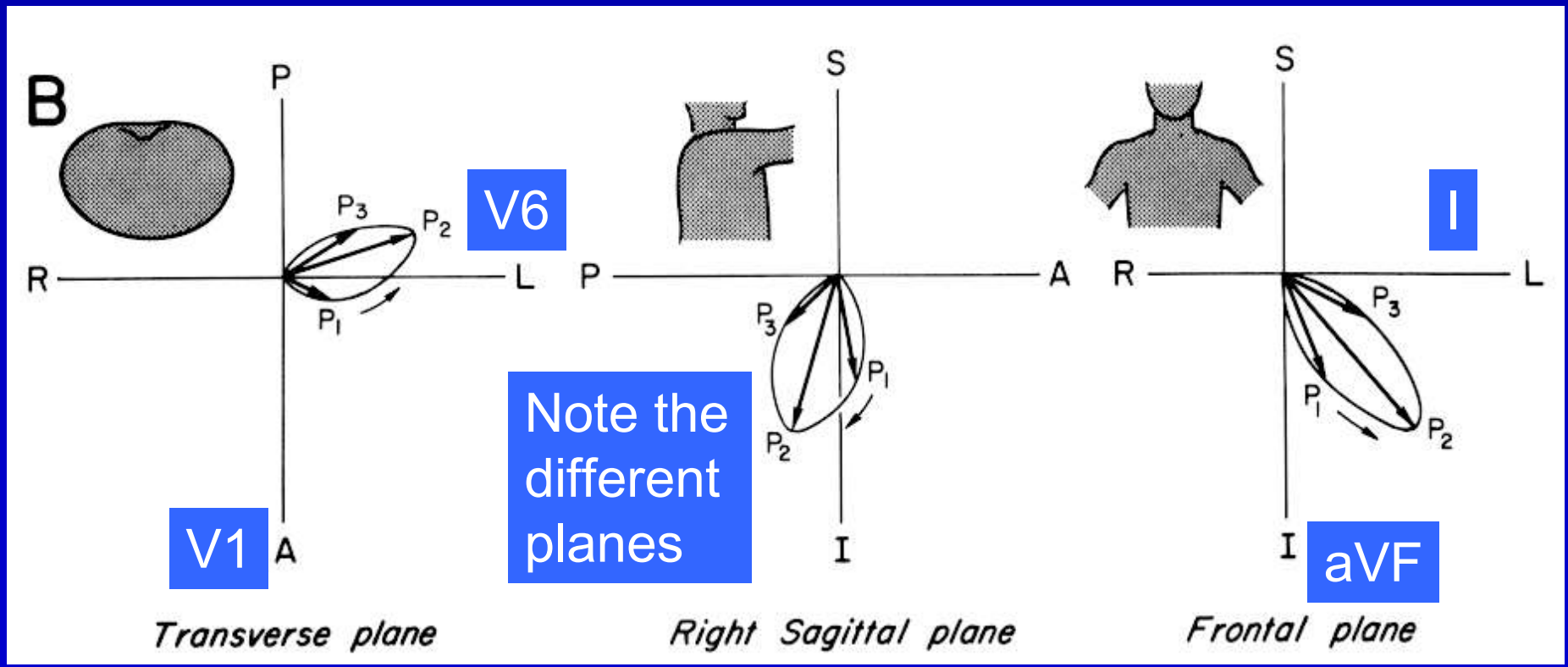


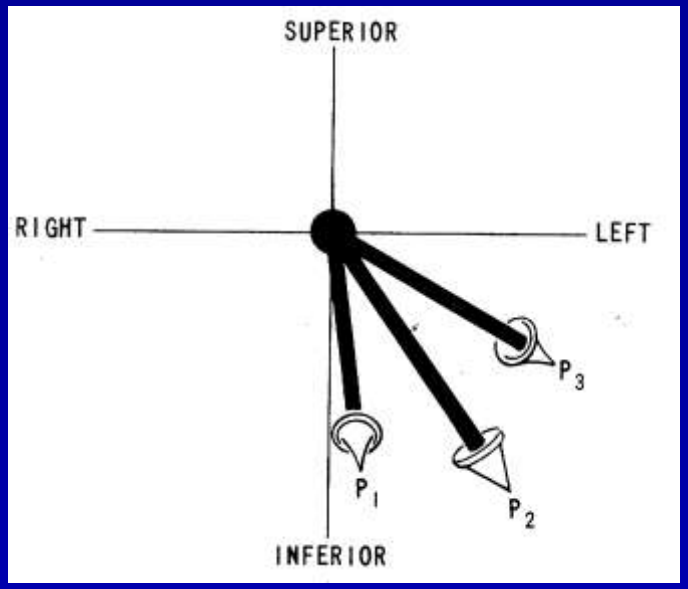
Friedman 1971; Chou 1974; Surawicz 2001



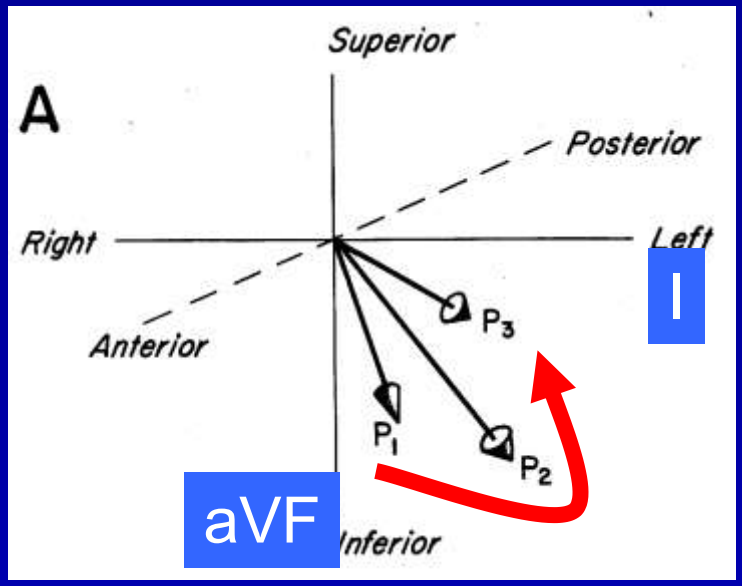
Normal P wave

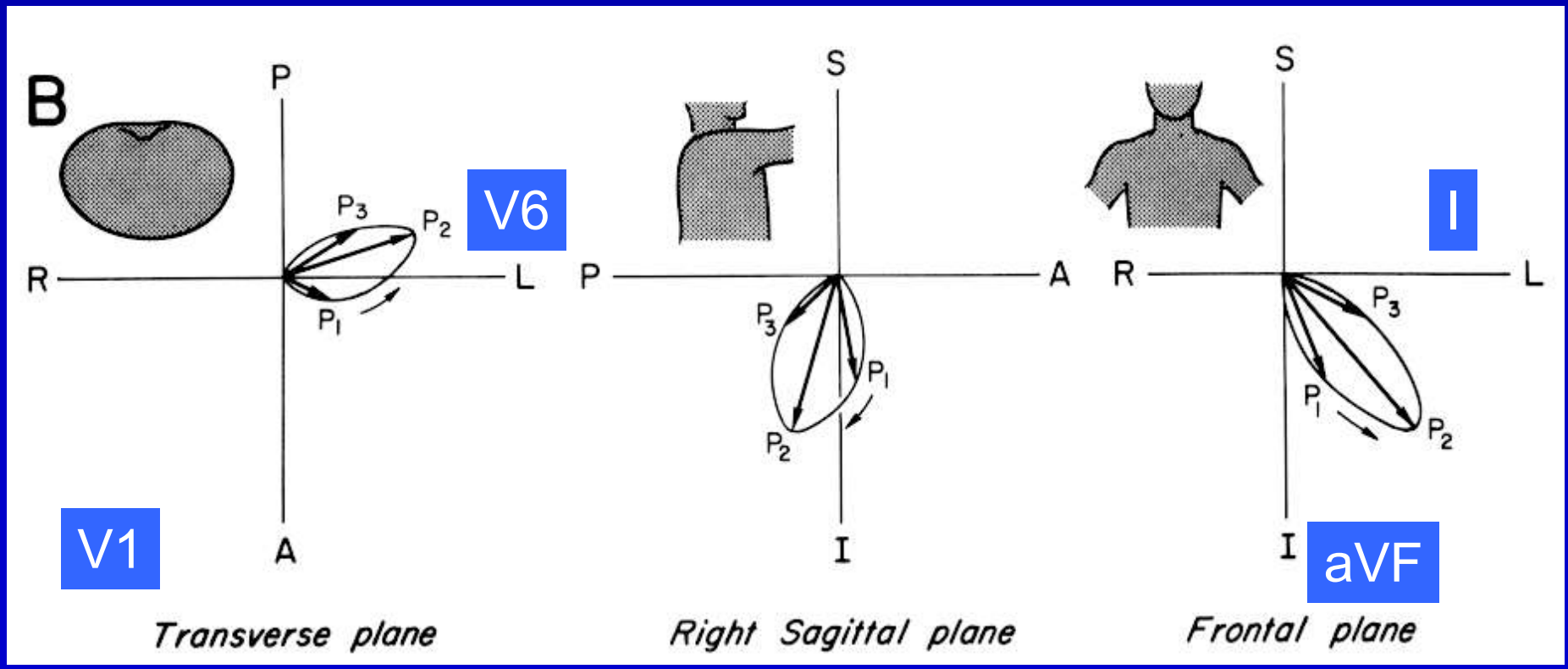


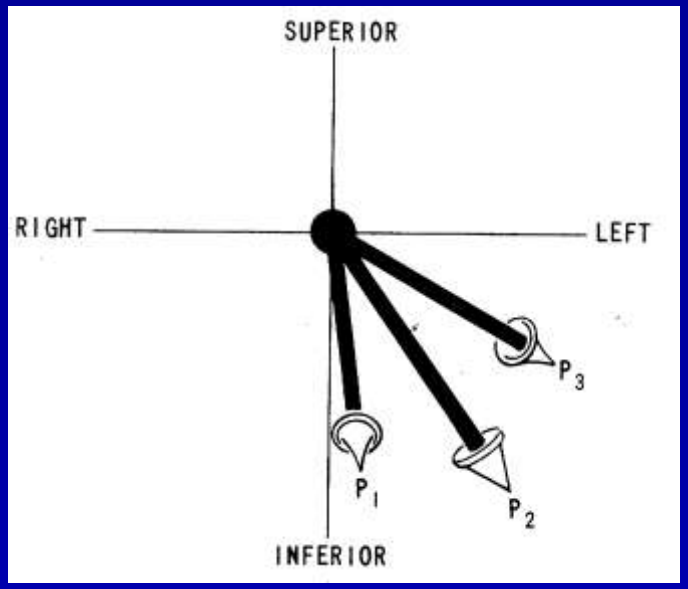


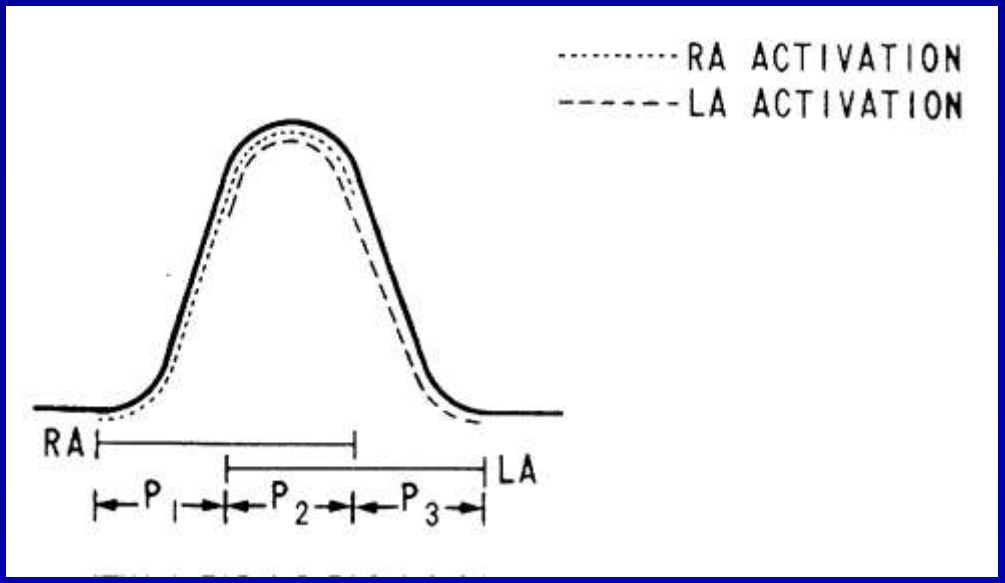


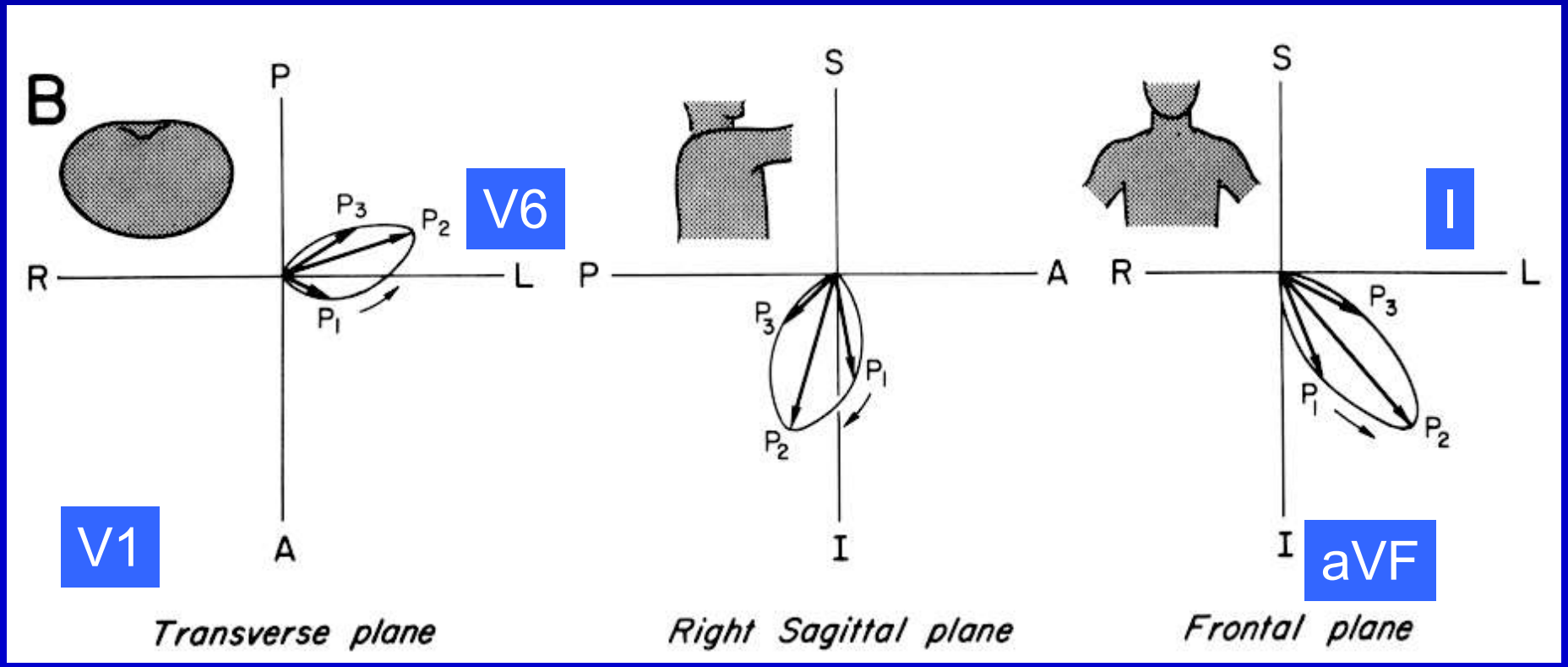
Normal P wave

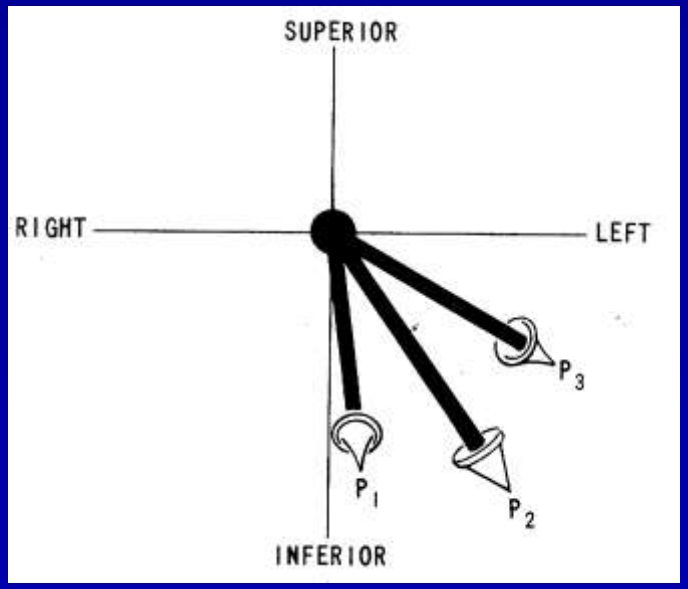


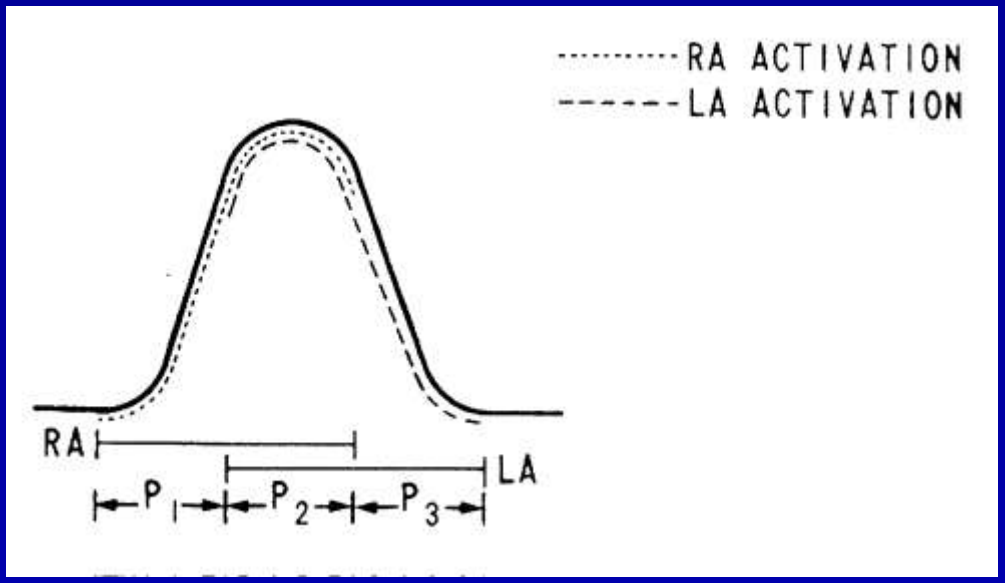


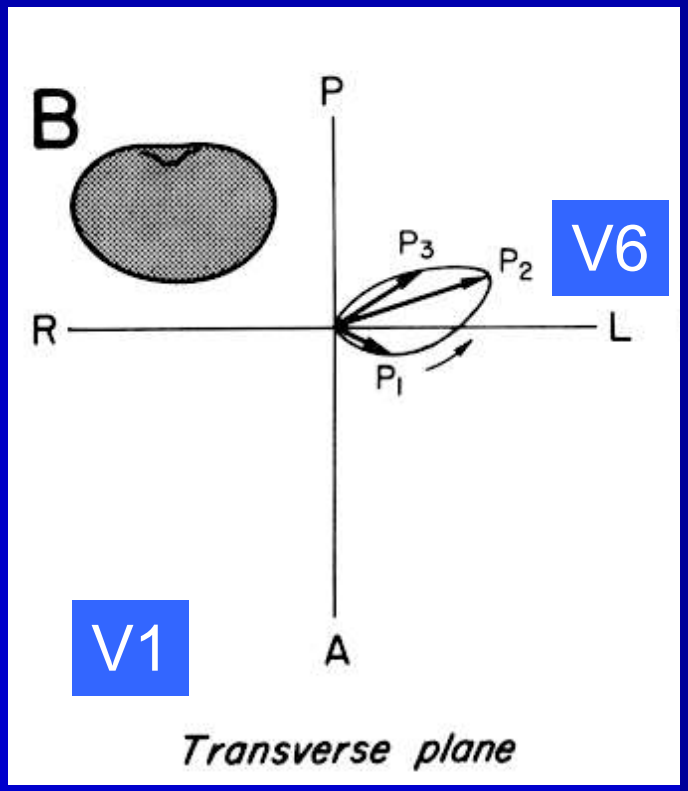

SUPERIOR
RIGHT
LEFT
INFERIOR
P1
P2
P3

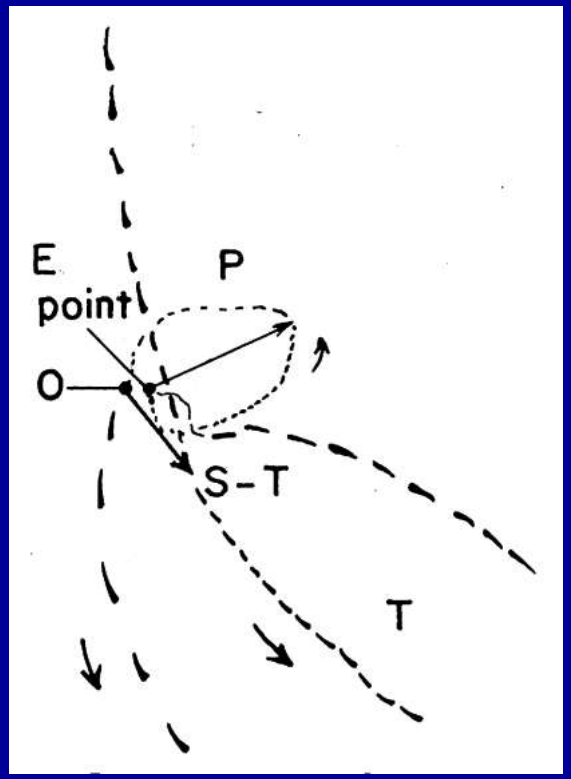

RA ACTIVATION
LA ACTIVATION
RA
LA
P1
P2
P3


B
P
R
L
A
P3
P2
P1
V6
V1
Transverse plane
S
P
A
I
P3
P1
P2
Right Sagittal plane
S
R
L
I
P3
P1
P2
I
aVF
Frontal plane

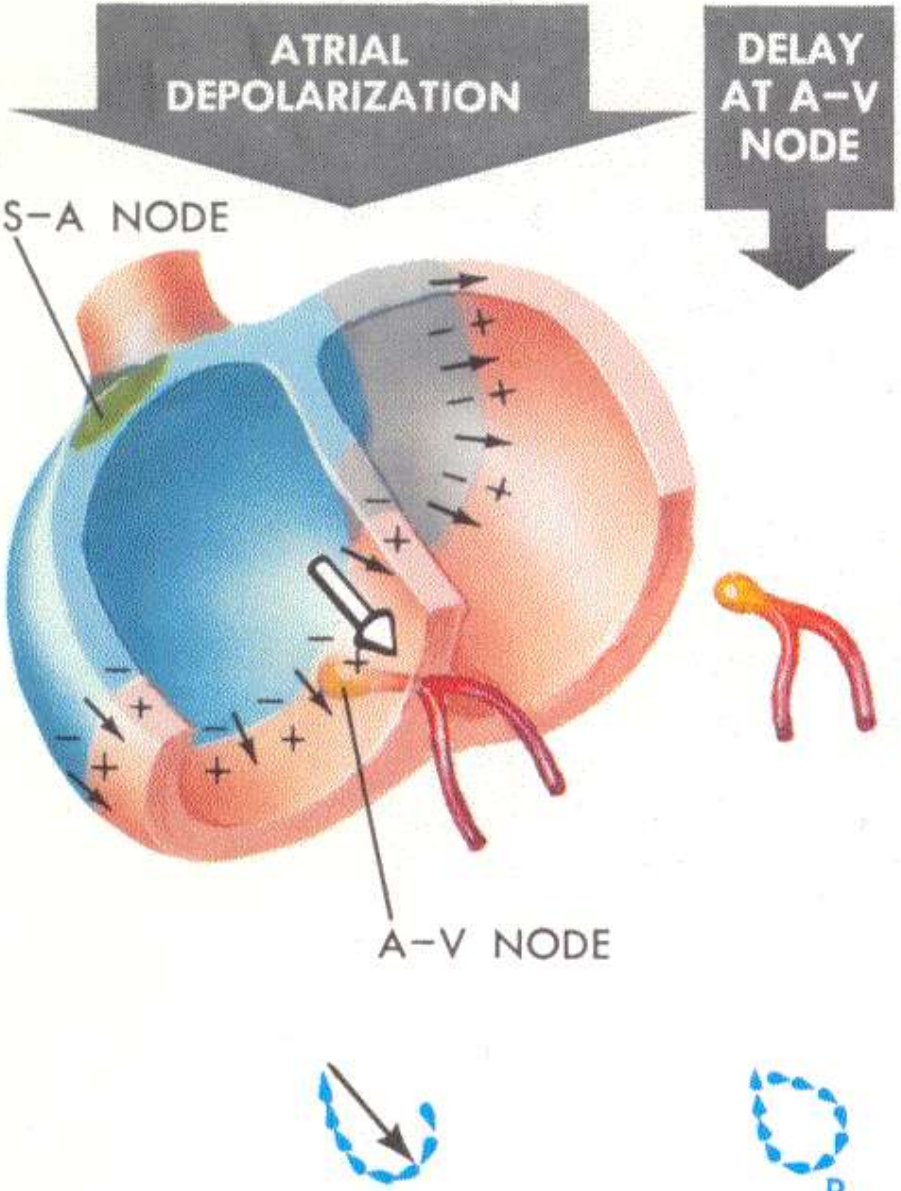

SUPERIOR
RIGHT
LEFT
INFERIOR
P1
P2
P3

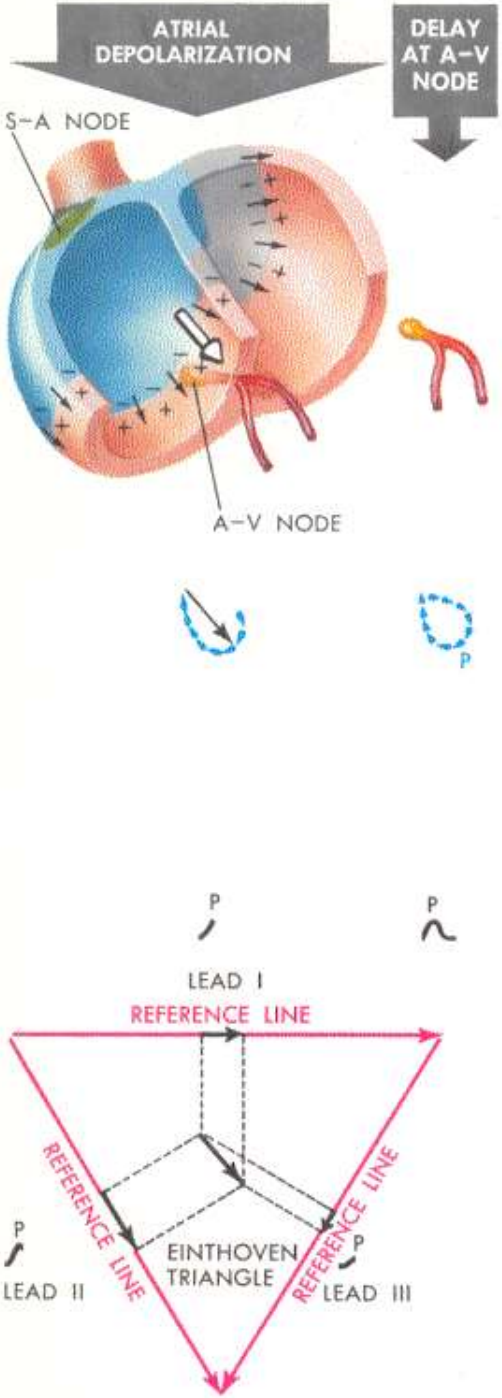

RA ACTIVATION
LA ACTIVATION
RA
LA
P1
P2
P3


B
P
P3
P2
R
L
P1
A
V1
V6
Transverse plane

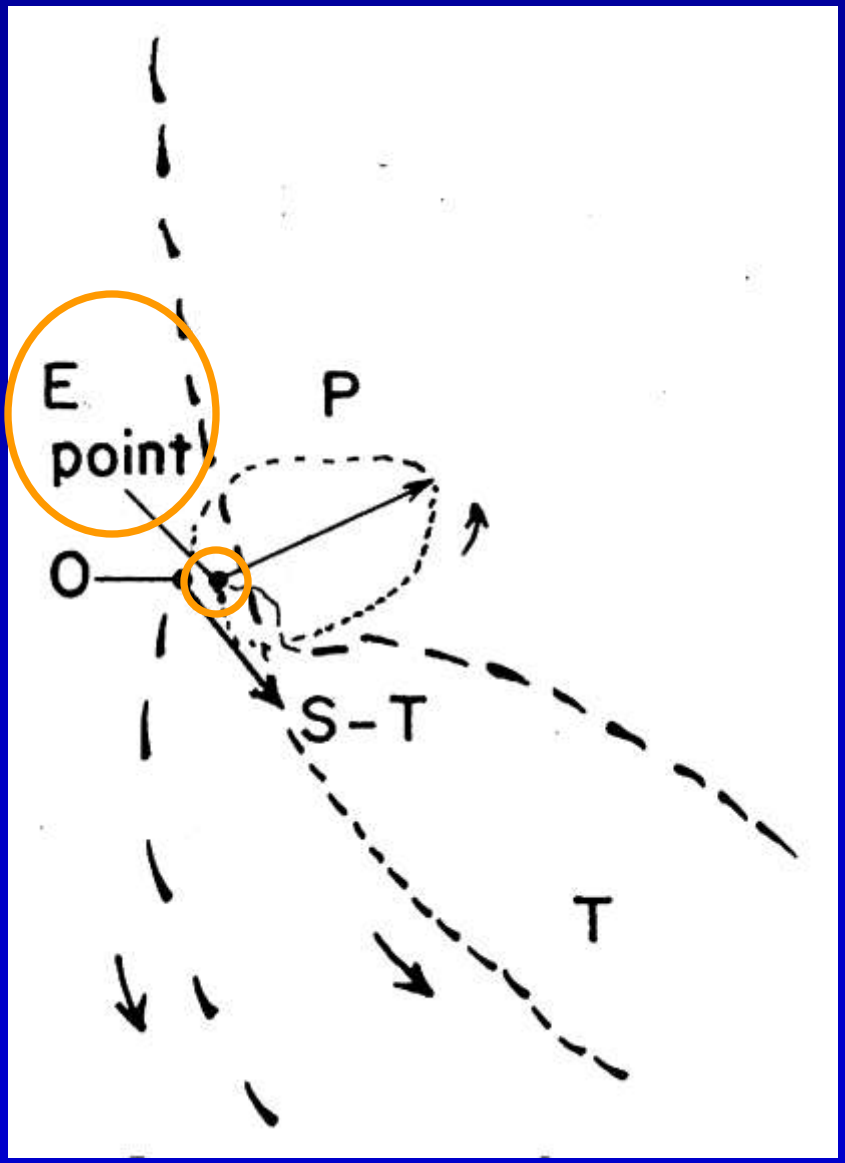

E
point
P
O
S-T
T

Atrial Activation



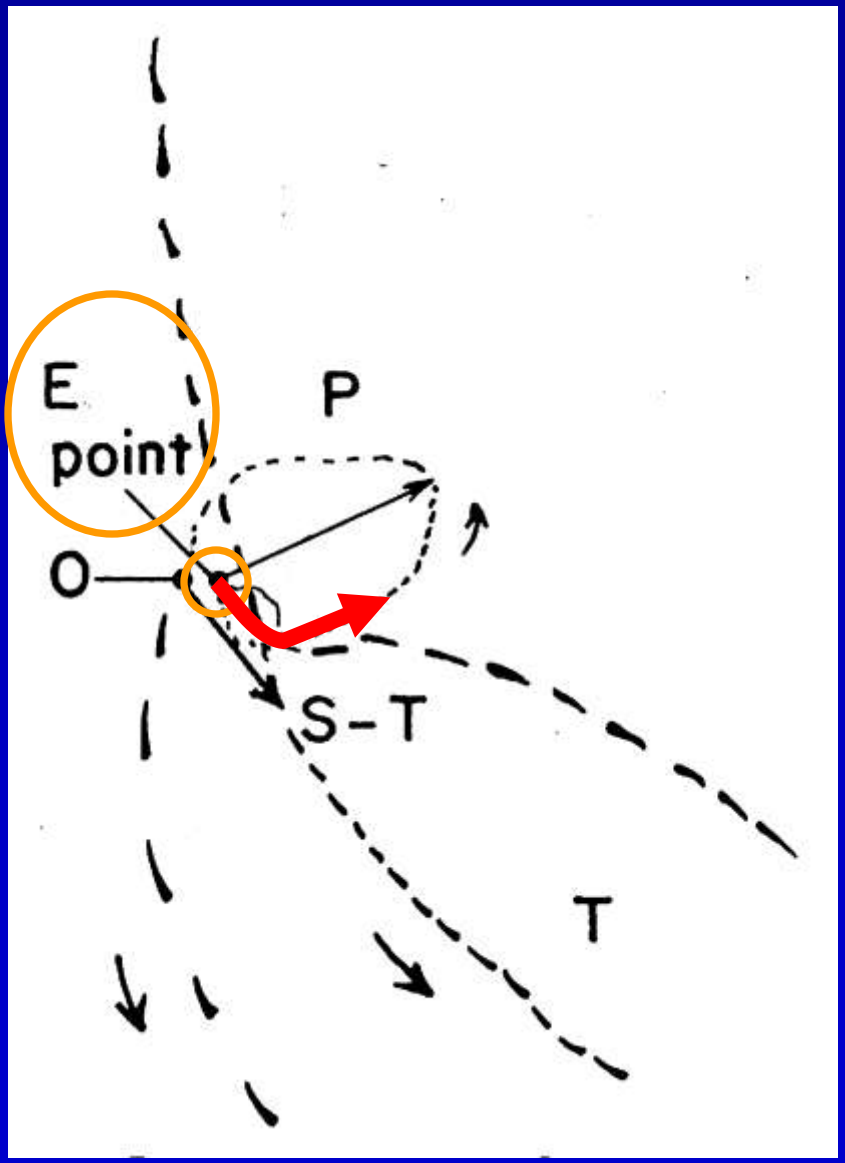


Transverse Plane



- E point is the beginning of the P wave, the end of the T-P segment
- O point is the end of the PR segment, the beginning of the QRS

Transverse Plane



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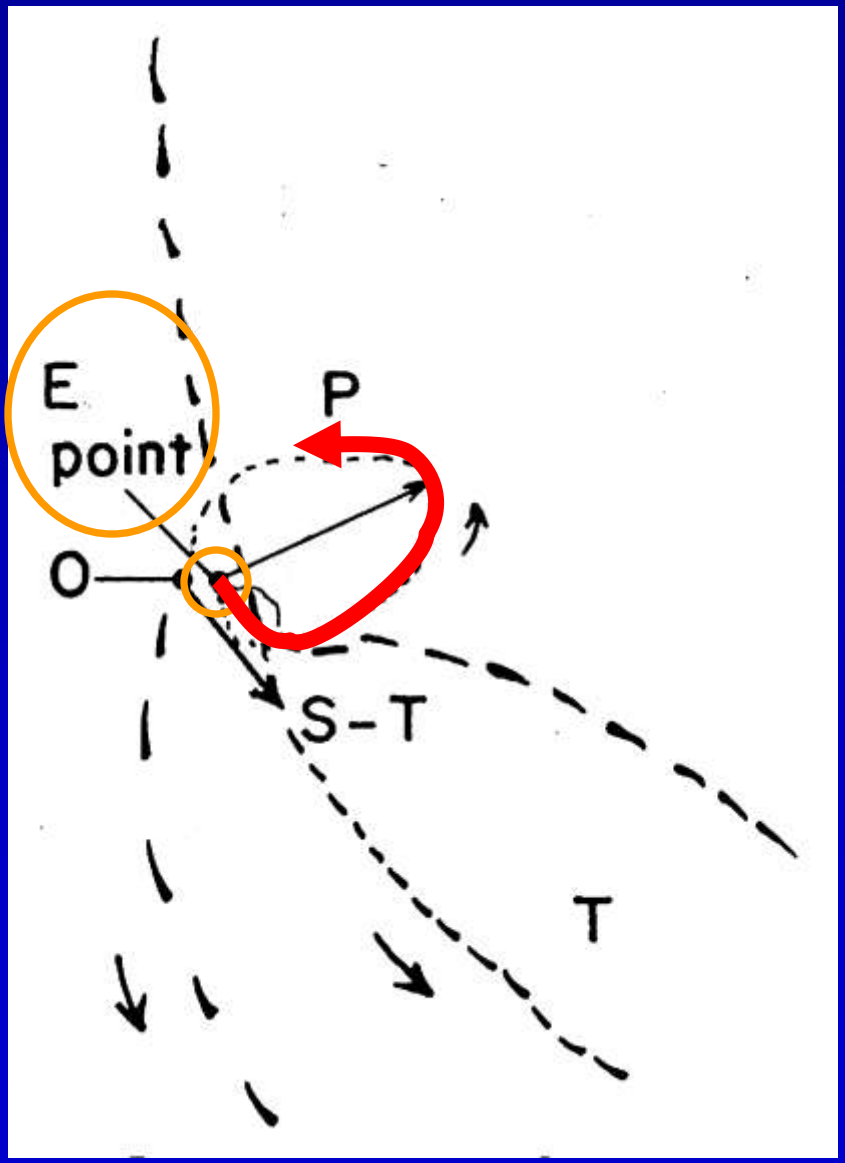
Transverse Plane



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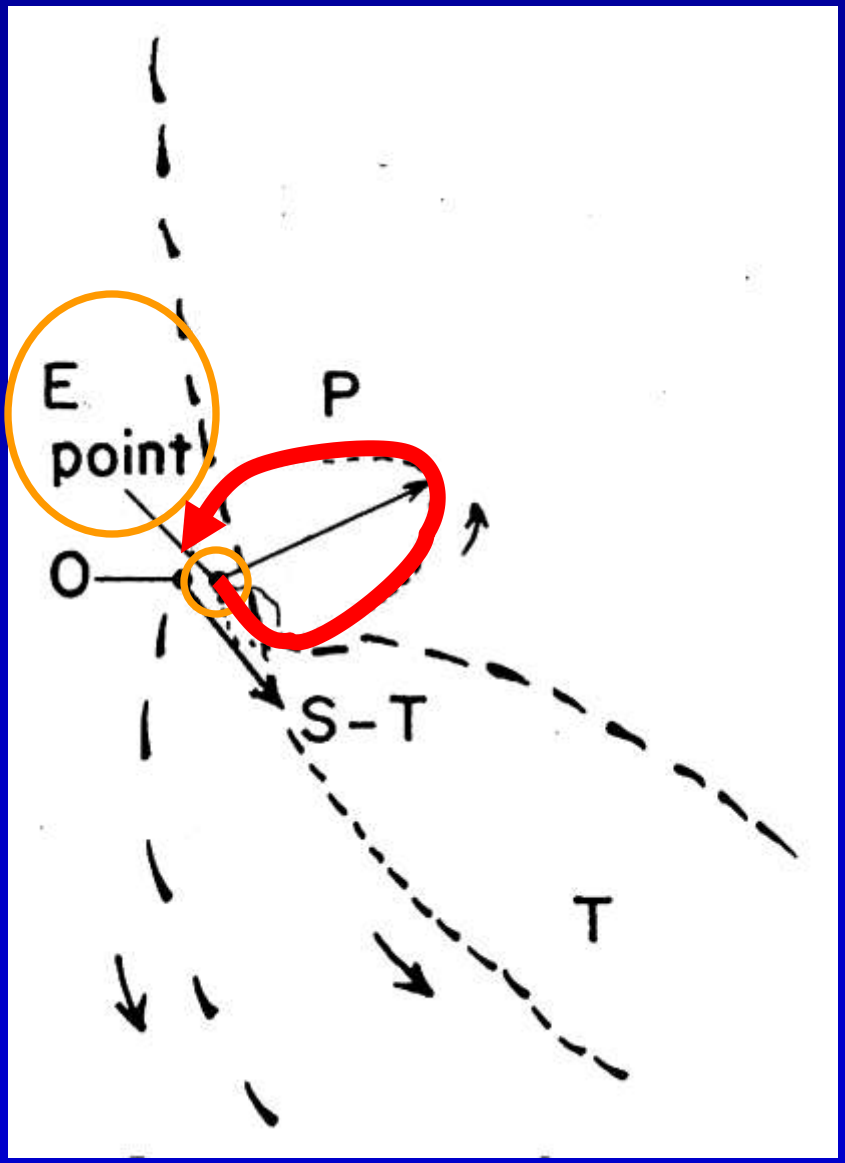
Transverse Plane



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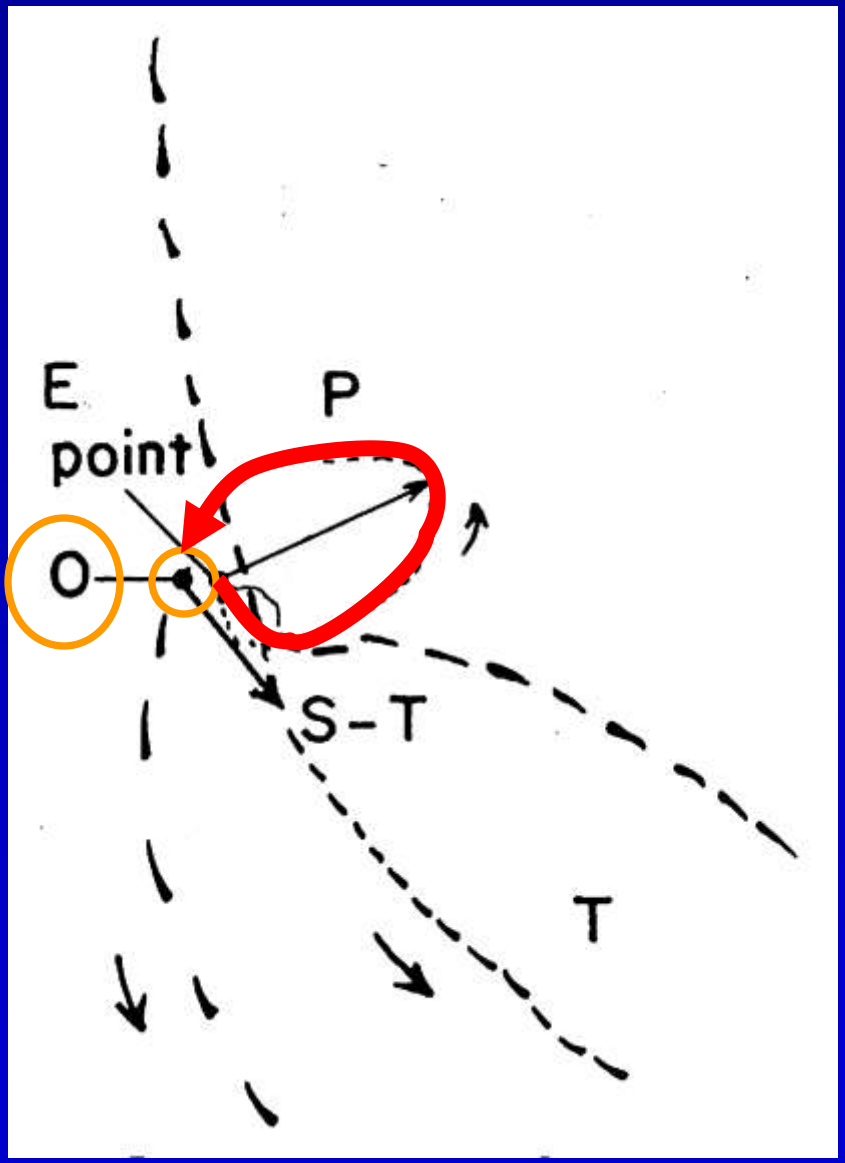
Transverse Plane



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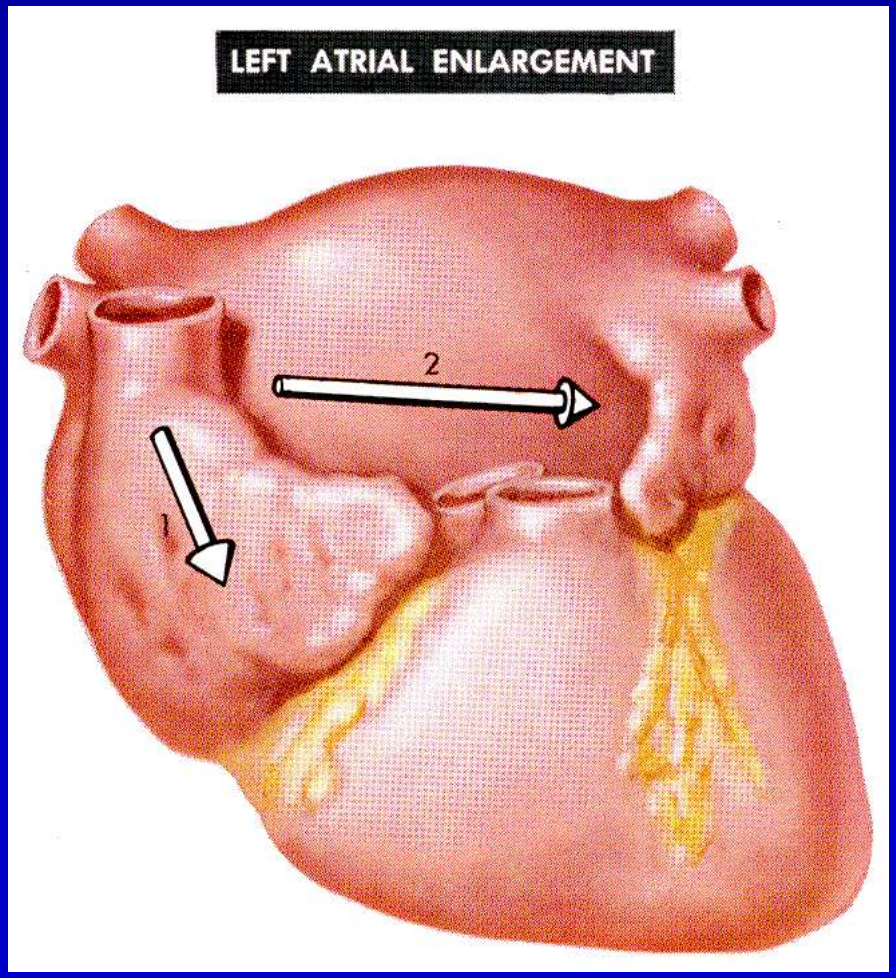
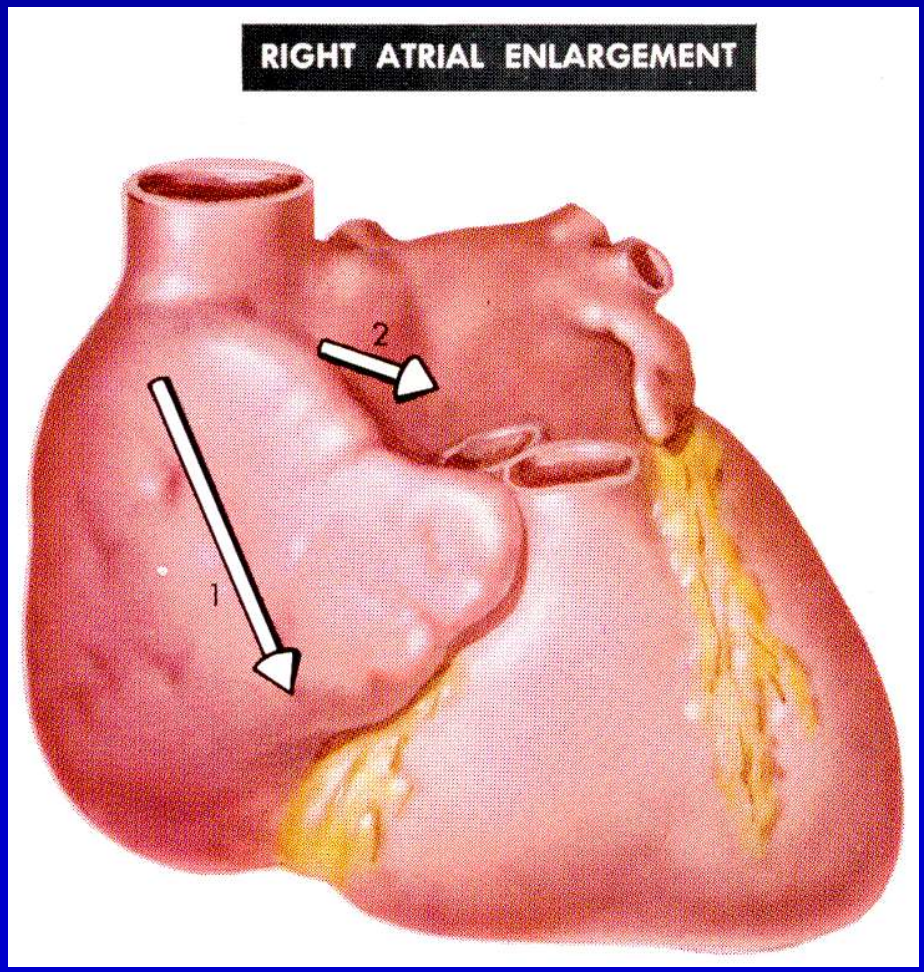
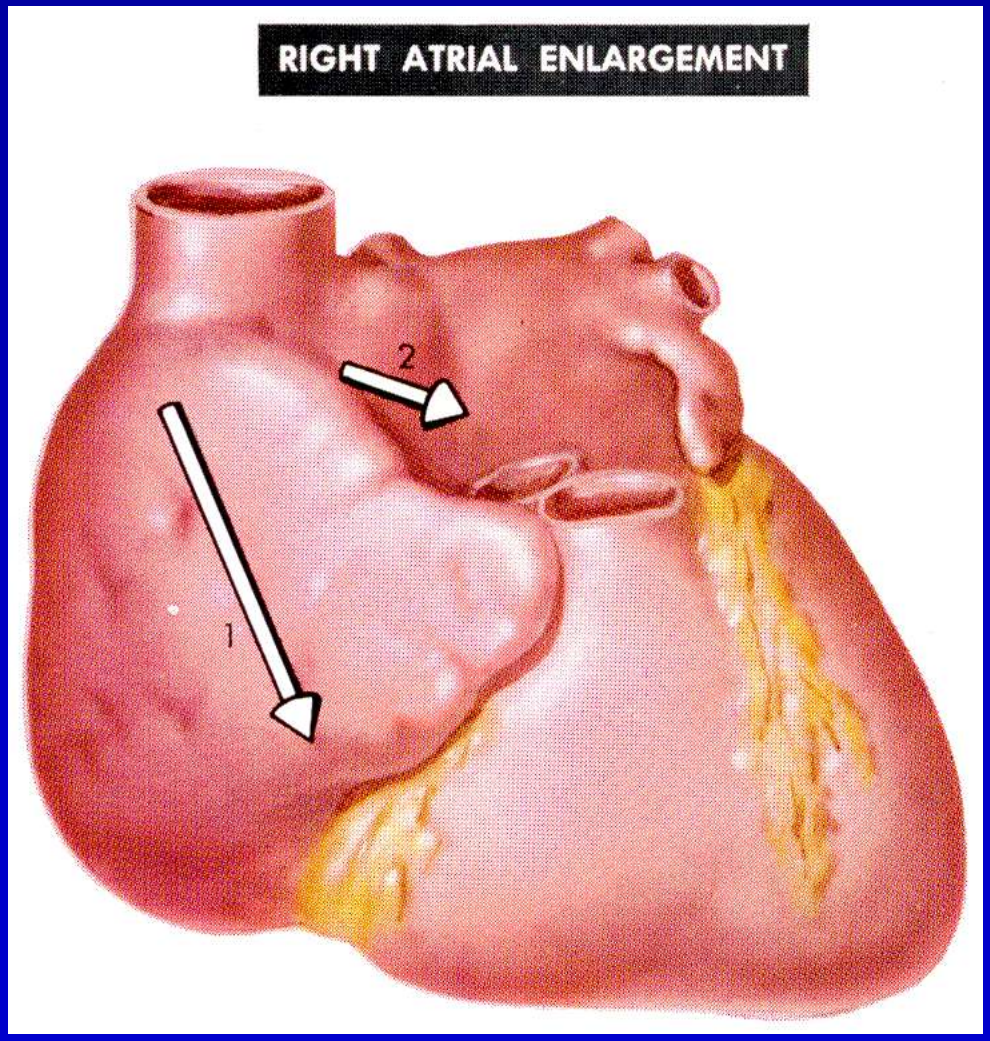
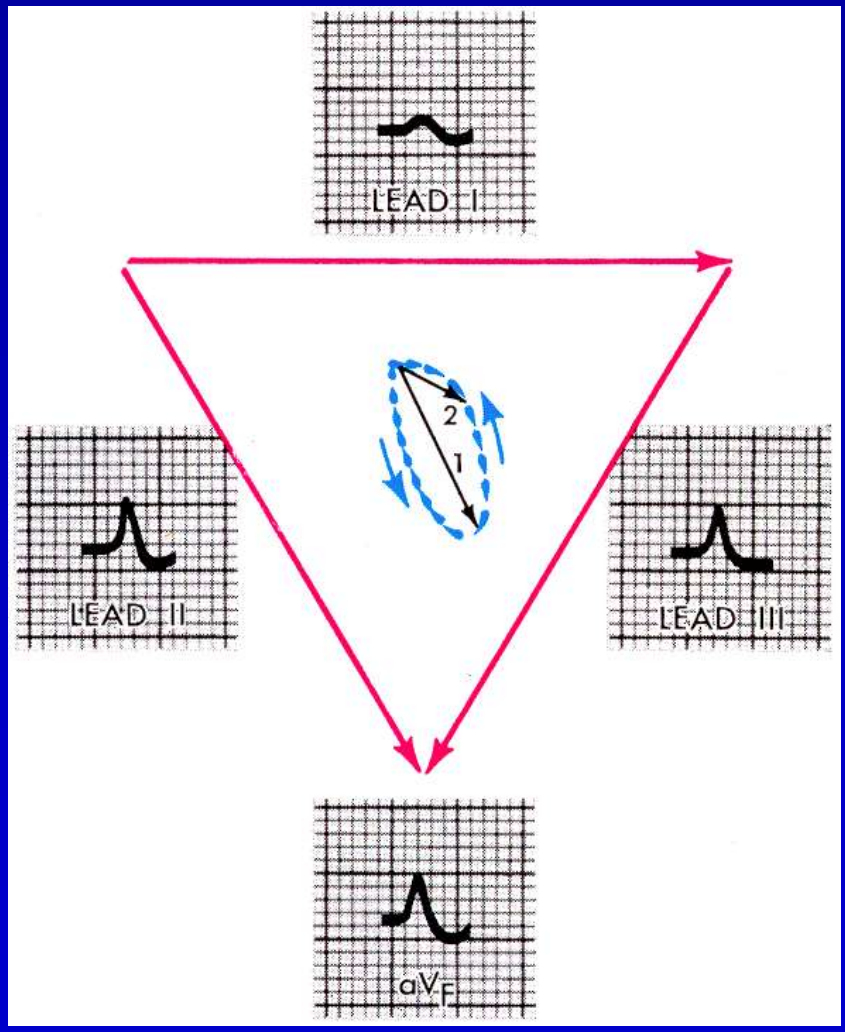
RAE vs LAE



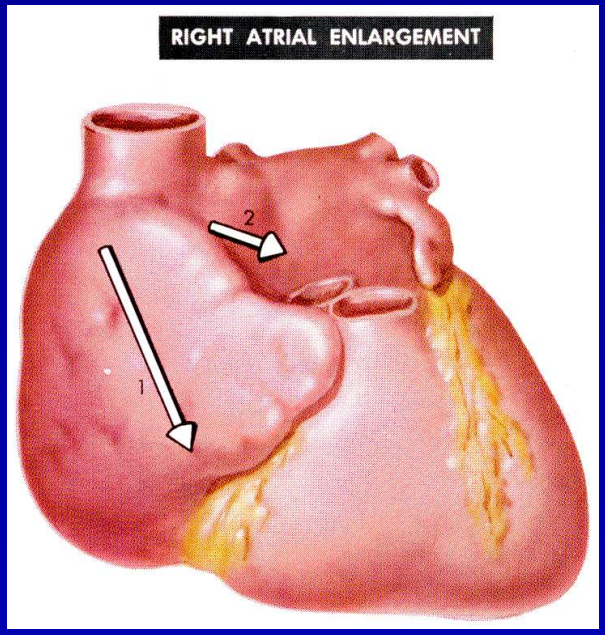


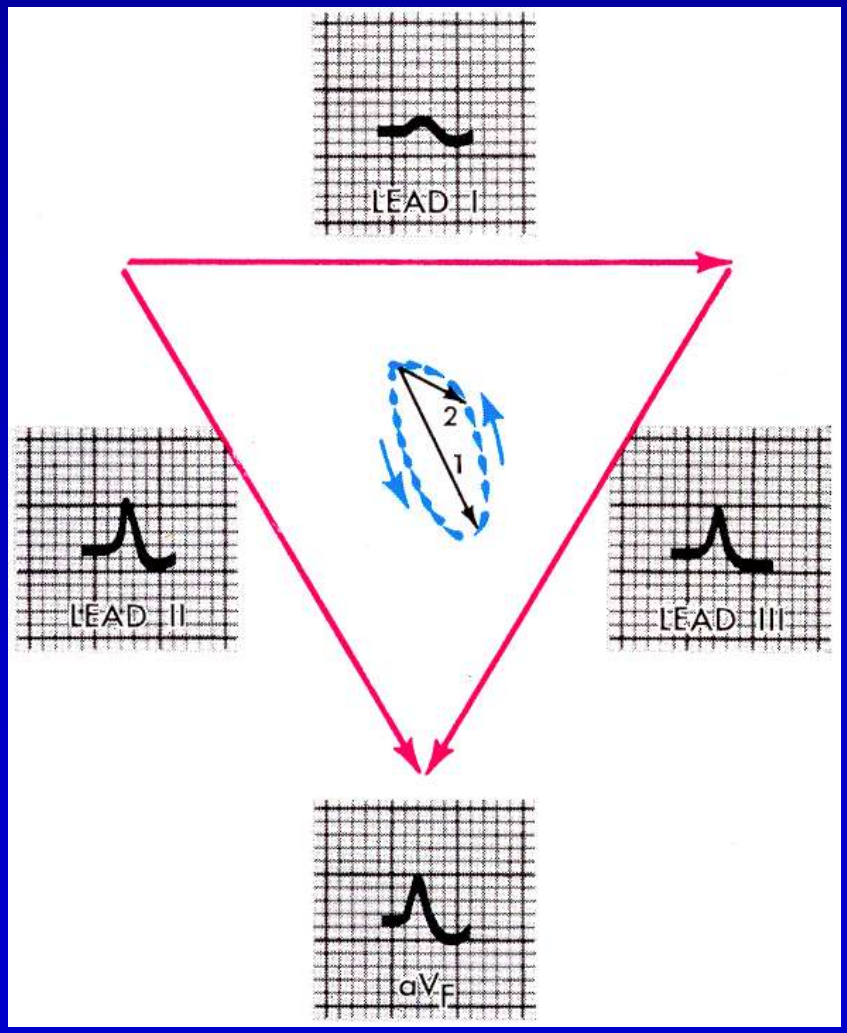
RAE

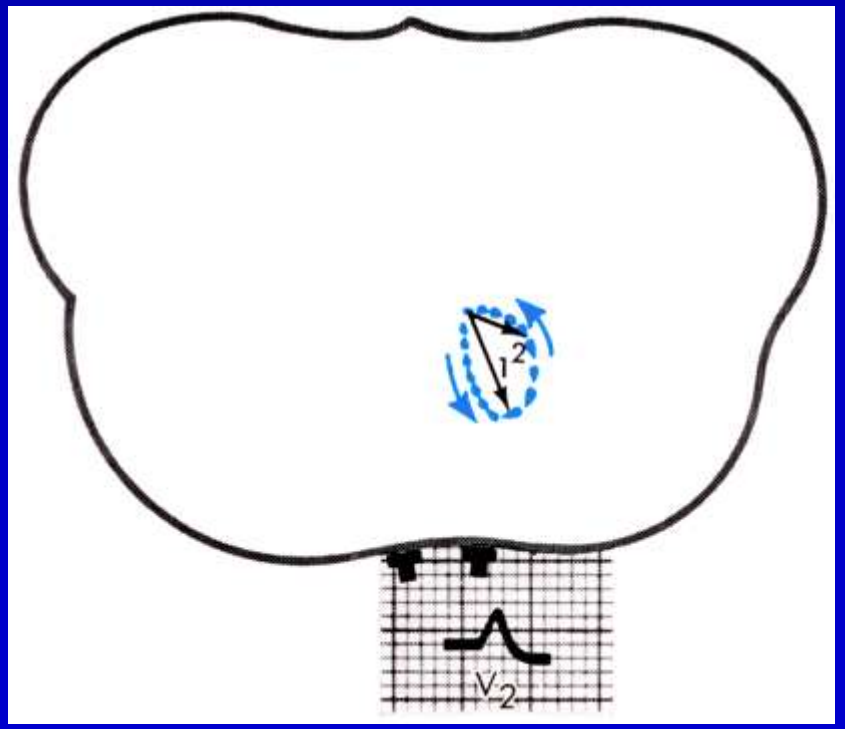




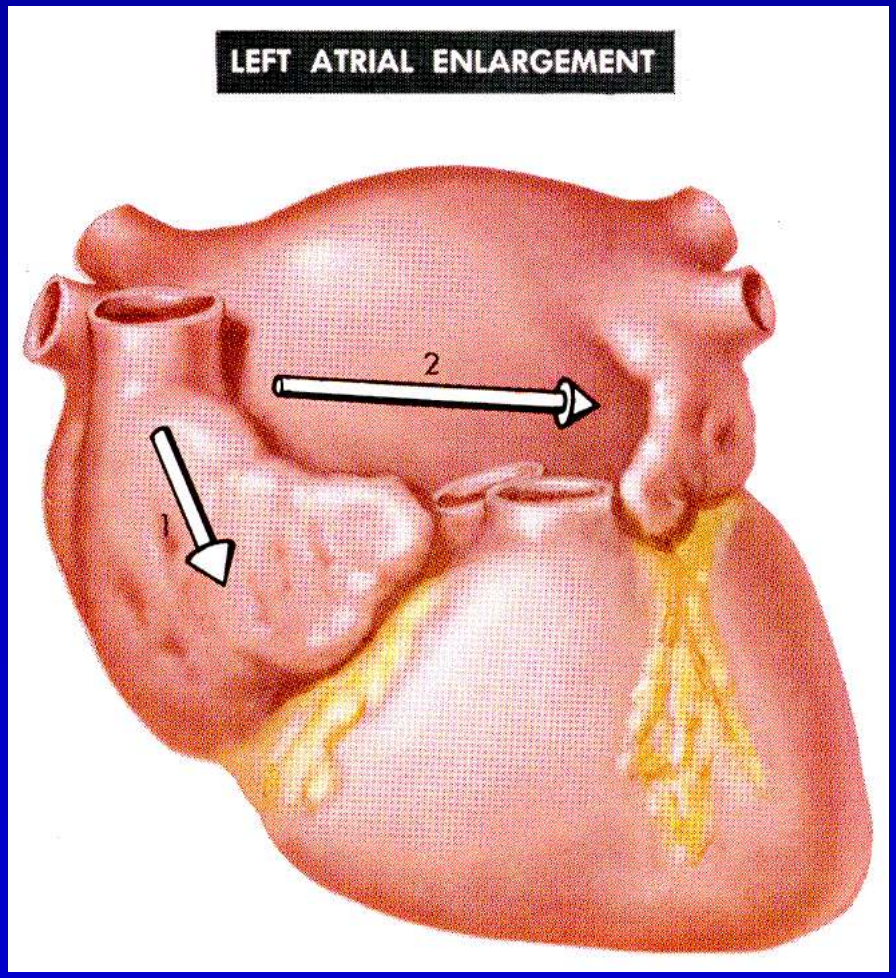
RAE

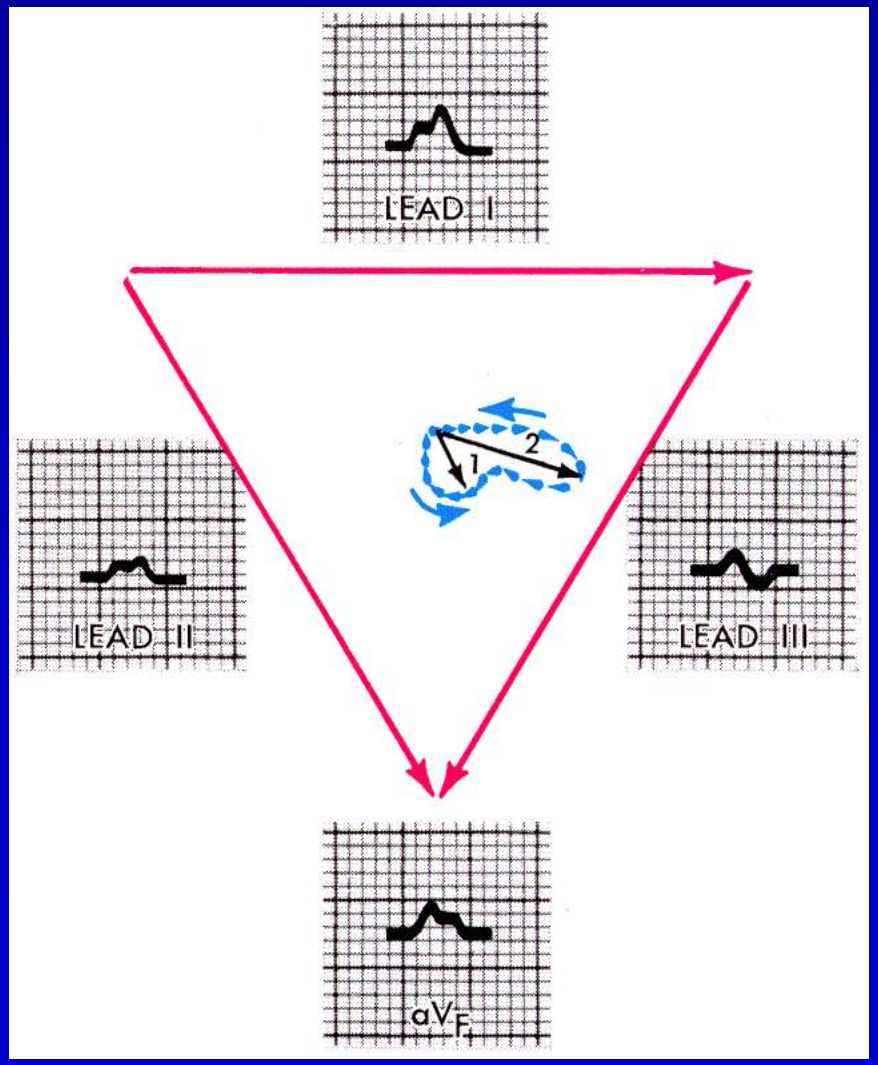

RIGHT ATRIAL ENLARGEMENT
2
1


LEAD I
LEAD II
LEAD III
aVF
2
1

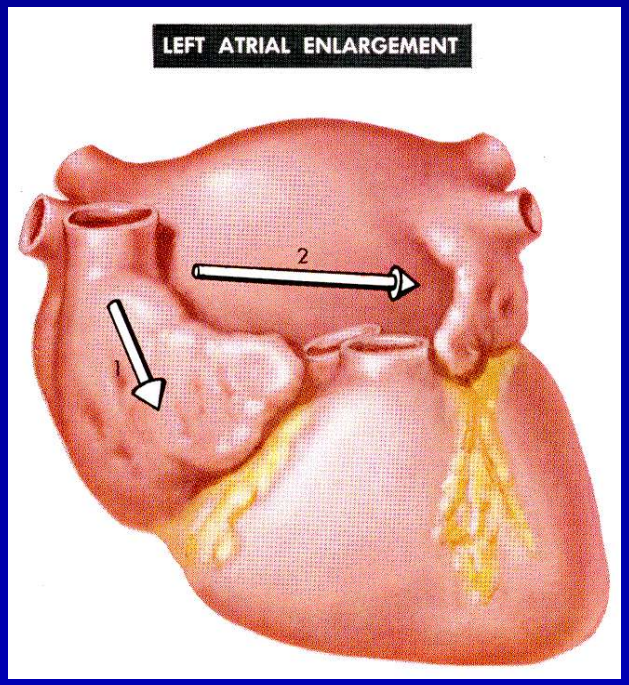

V2
1
2

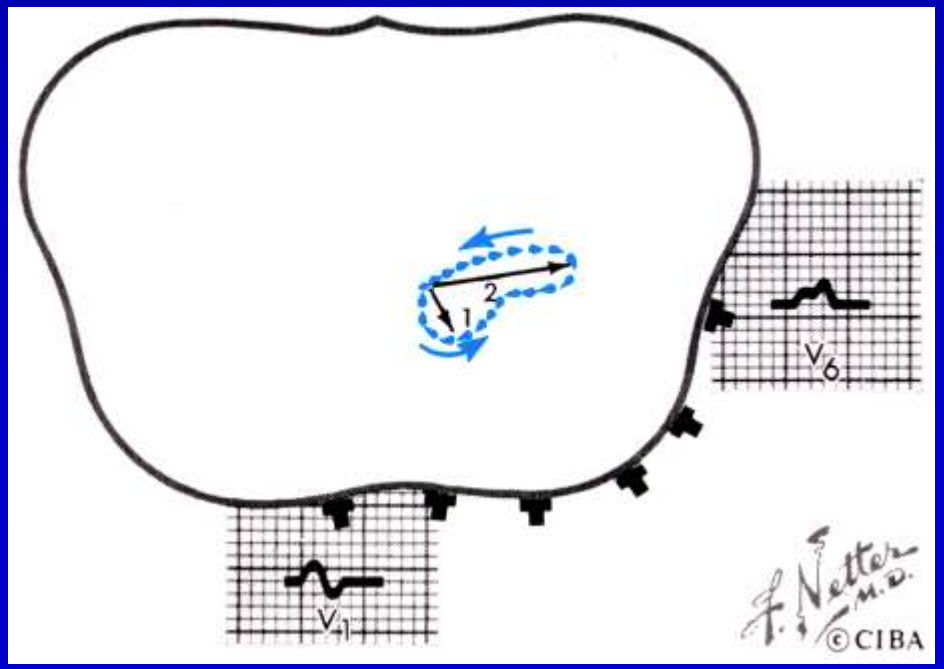
LAE

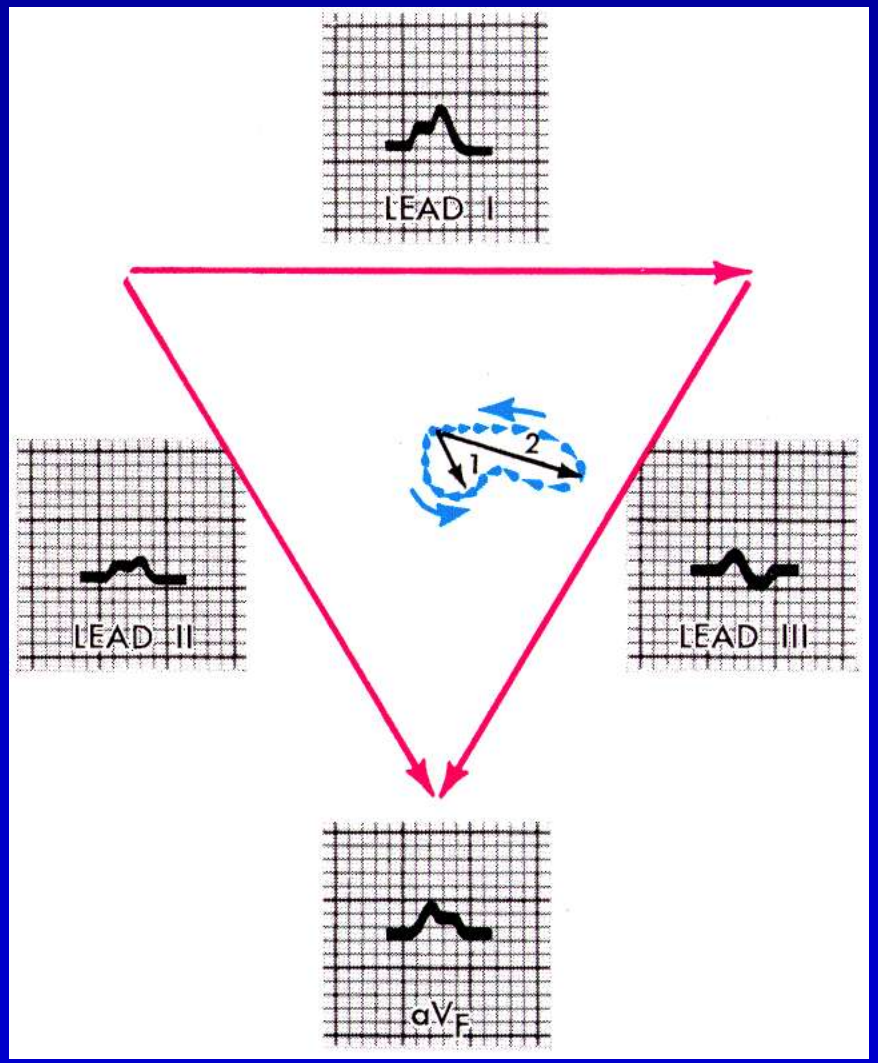




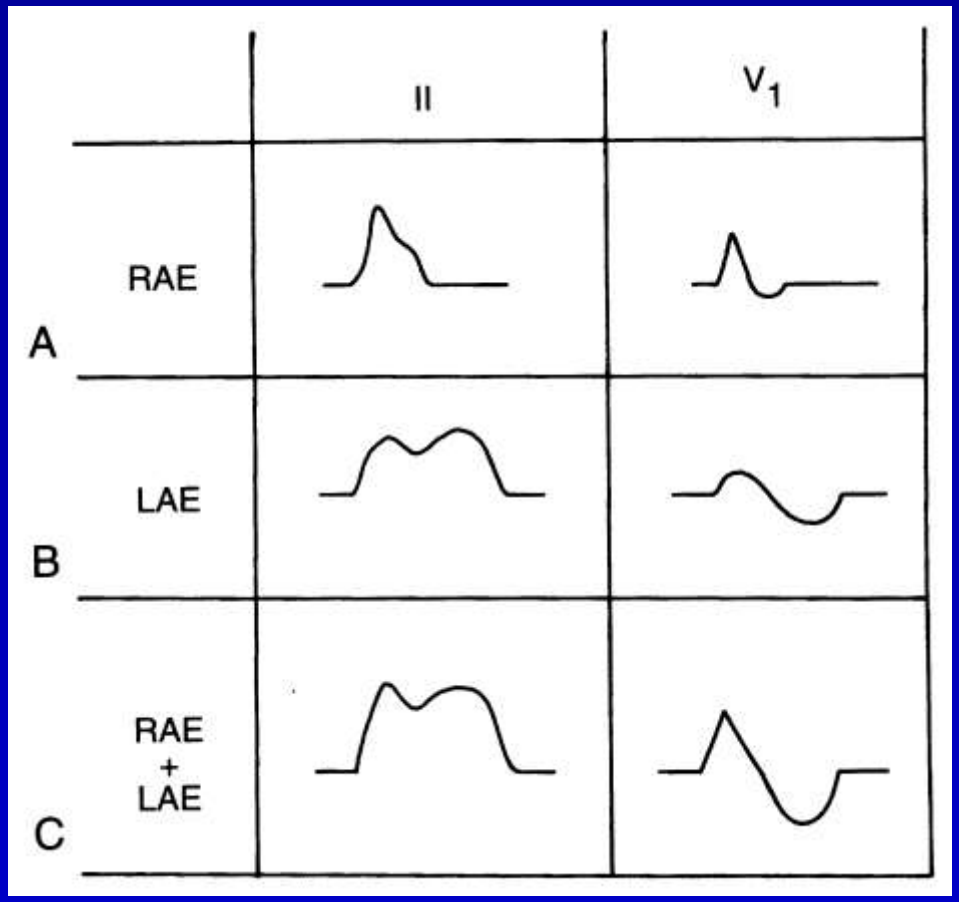
LAE

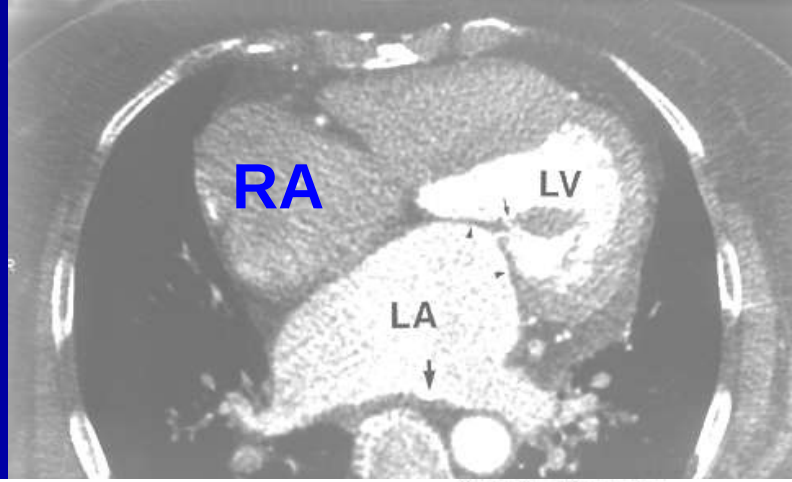

LEFT ATRIAL ENLARGEMENT
2
1

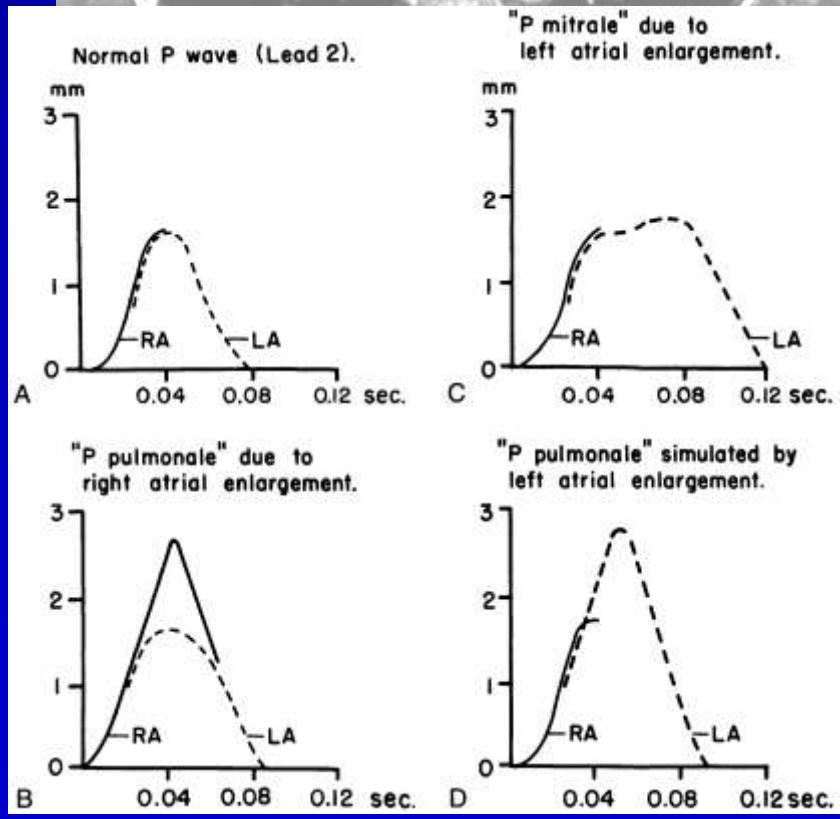

2
1
V6
V1
F. Netter M.D.
©CIBA


LEAD I
LEAD II
LEAD III
aVF
2
1

Atrial Enlargement



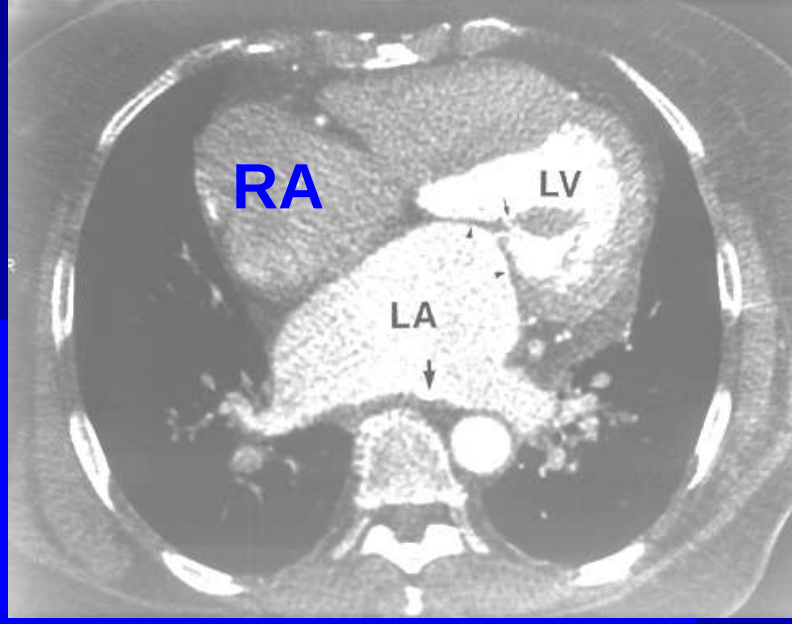




Surawicz B et al. Chou's ECG… 2001, p.35

Wagner GS. Marriott's Practical Electrocardiography 1994, p.58

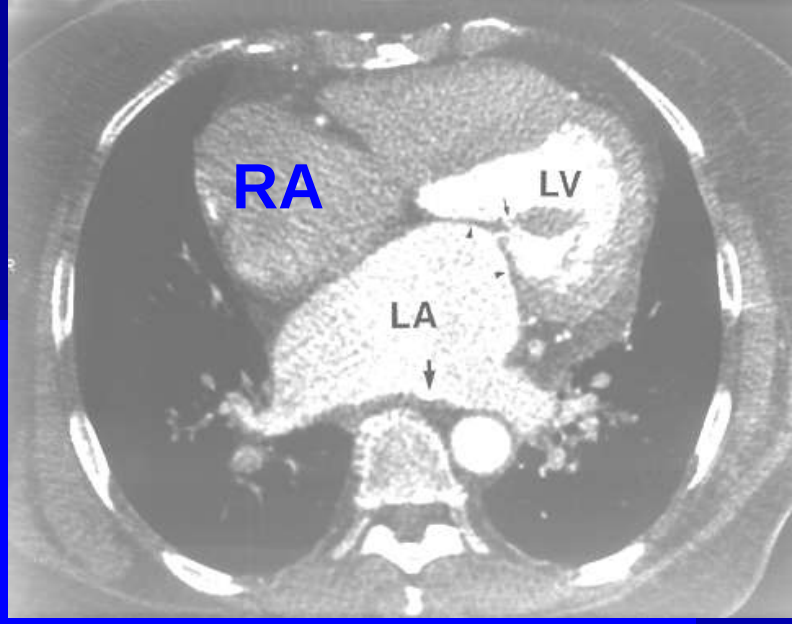
Atrial Enlargement or Hypertrophy



- Atrial abnormalities are usually with enlargement of one or both ventricles
- RAE (may wax and wane)
  - 1.5 mm tall P in V1 or V2 and rightward (>75) P axis are best
  - 2.5 mm tall P wave in II has false positives (sympathetic tone, standing position, low diaphragm position), can represent LAE too (“pseudo P pulmonale”)

Surawicz 2001 Ch. 2

Atrial Enlargement or Hypertrophy



- Atrial abnormalities are usually with enlargement of one or both ventricles
- LAE (may wax and wane)
  - Prolonged P duration >0.12 sec, and notch in P wave with 0.04 sec between peaks, also P axis <15 degrees
  - P terminal force more than 1 mm deep and wide in V1

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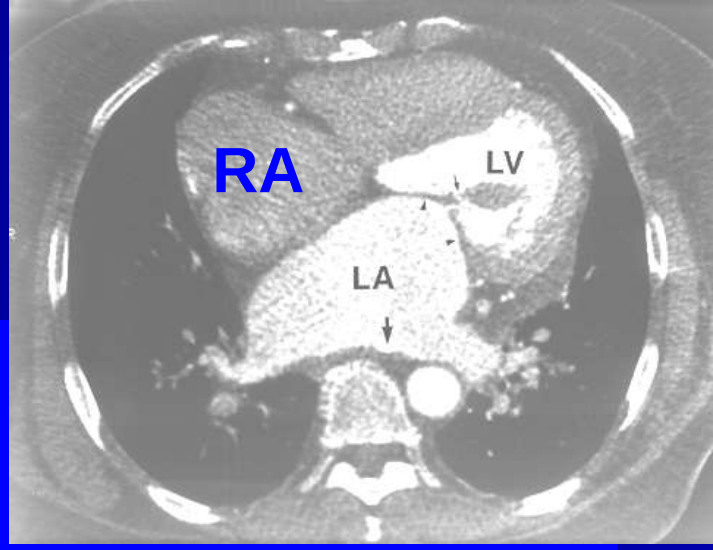
Atrial Enlargement or Hypertrophy



- False positives:
  - RA abnormality:
  - II tall in standing, in low diaphragm and in sympathetic tone
  - V1 tall in LA enlargement (pseudo P pulmonale)

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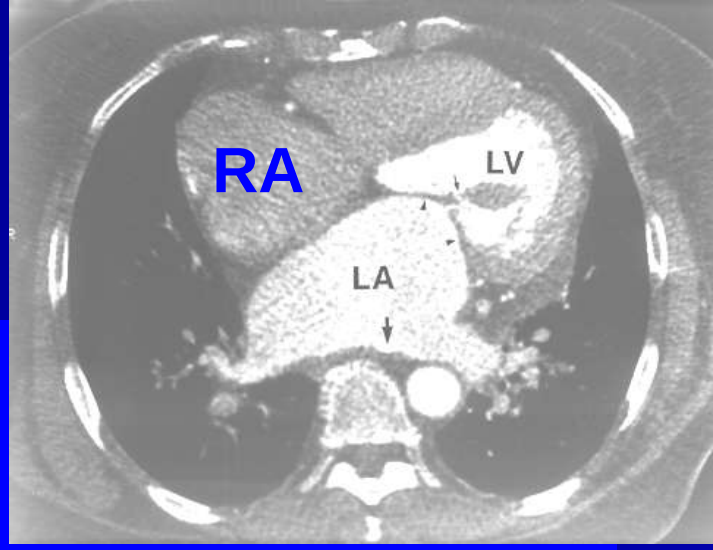
Atrial Enlargement or Hypertrophy



- False positives:
  - LA abnormality:
  - V1 P terminal force abnormal in COPD
  - V1 P terminal force abnormal in straight back syndrome and pectus excavatum
  - Occasionally in congenital disease with massive RA

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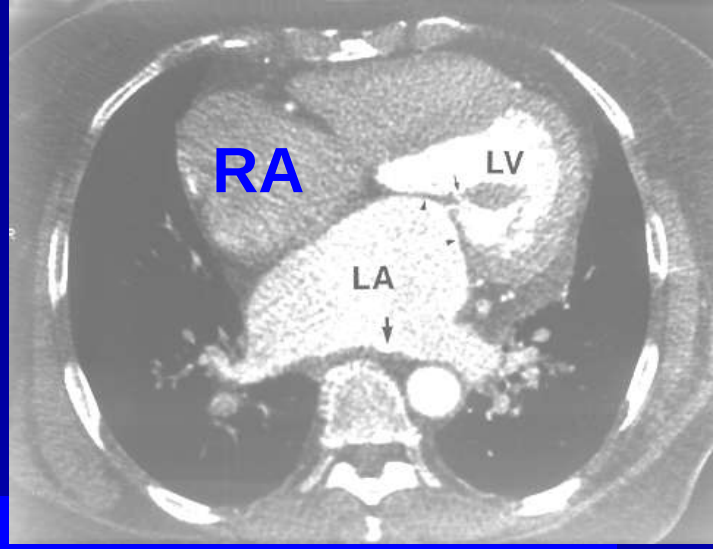
Atrial Enlargement or Hypertrophy



- Echo Correlation
  - RA abnormality:
  - Using V1, RAD of QRS and R/S>1 in V1, sens 48% and spec 100%
  - **LA abnormality:**
  - V1 Pterm>0.06 had PPV .58, NPV .83
  - V1 P term plus P dur >100ms had sens .82

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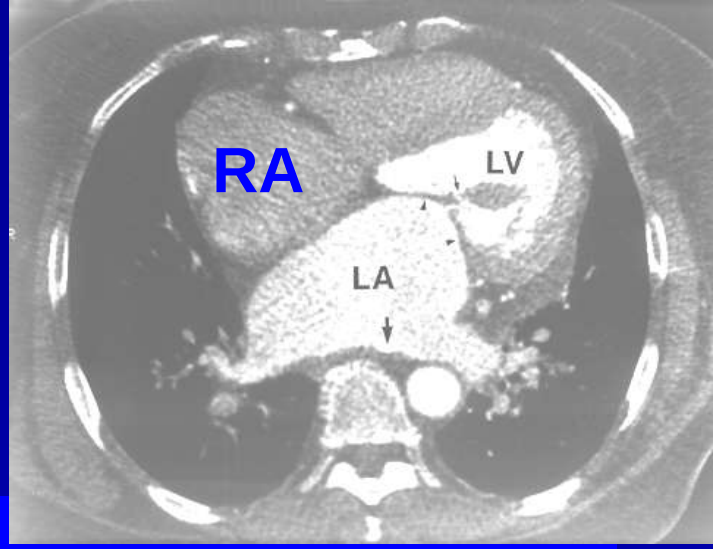
Atrial Enlargement or Hypertrophy



- Biatrial enlargement:
  - V1 has both RAE and LAE criteria
  - V1 has RAE criteria and inferior or lateral leads show wide notched P
  - Limb leads have both >2.5mm and >0.12 sec
- Atrial enlargement in atrial fibrillation (>1mm)
- Intra-atrial conduction disturbance
- Atrial repolarization may last 0.45 sec, about 1/3 height of P

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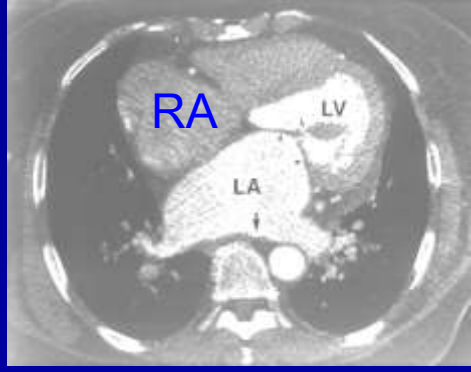
ECG - RAE

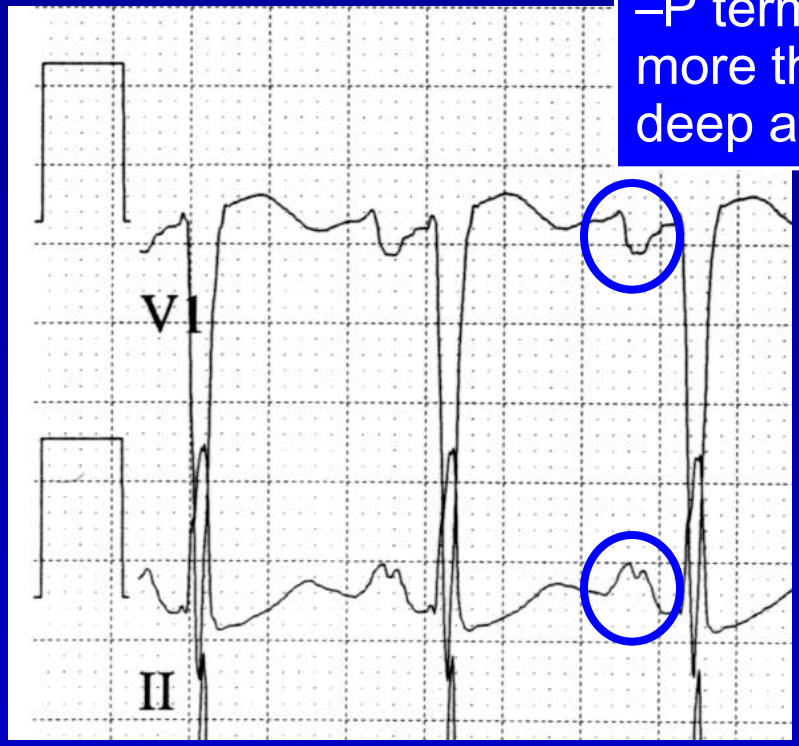


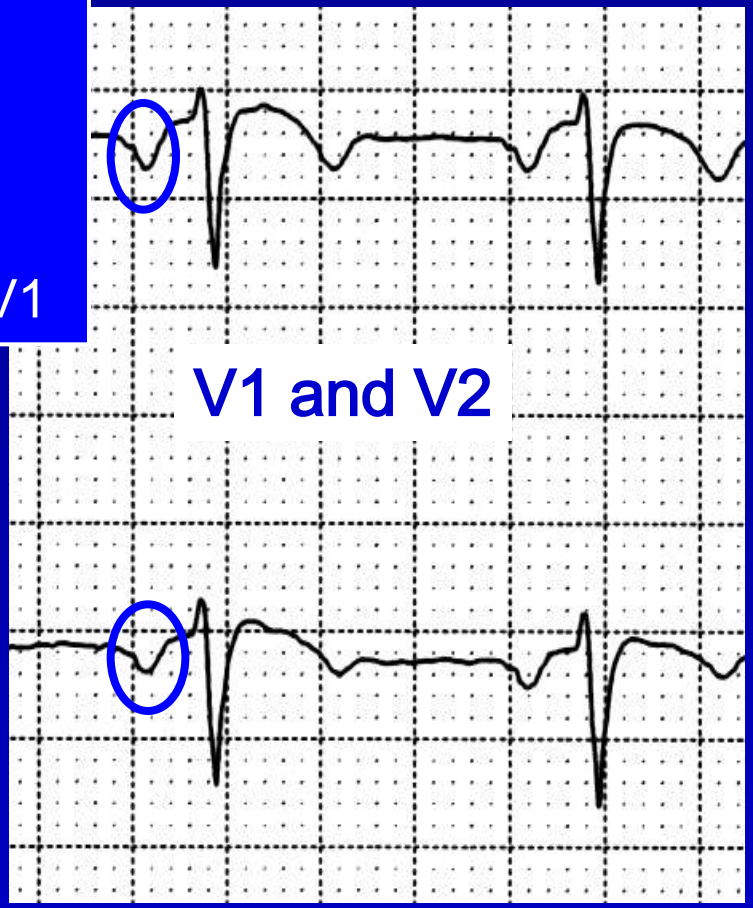
2.5 mm tall P wave in II, prominent initial force in V1

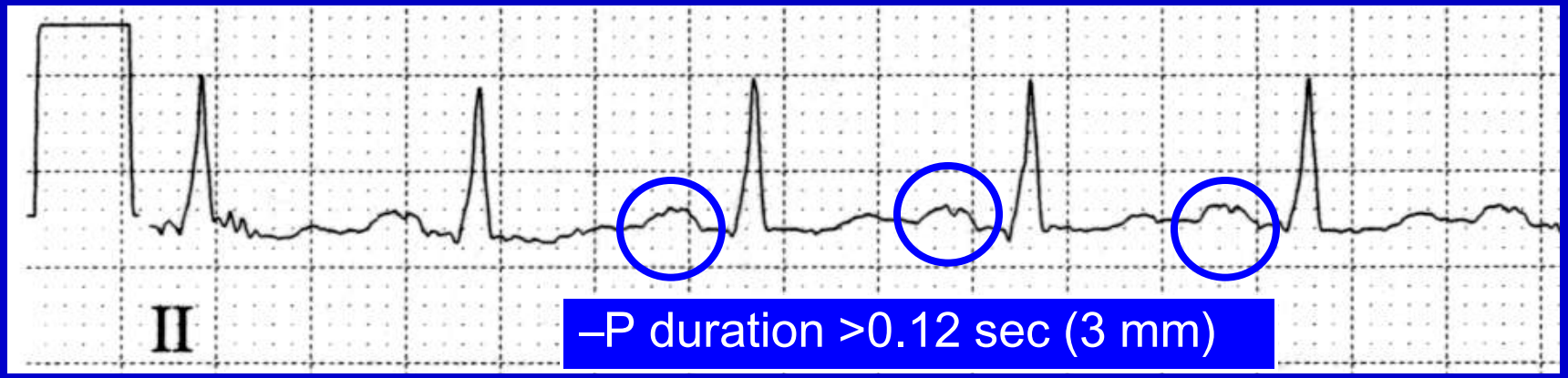
ECG - LAE

–Notch in P wave with 0.04 sec between peaks

–P terminal force more than 1 mm deep and wide in V1

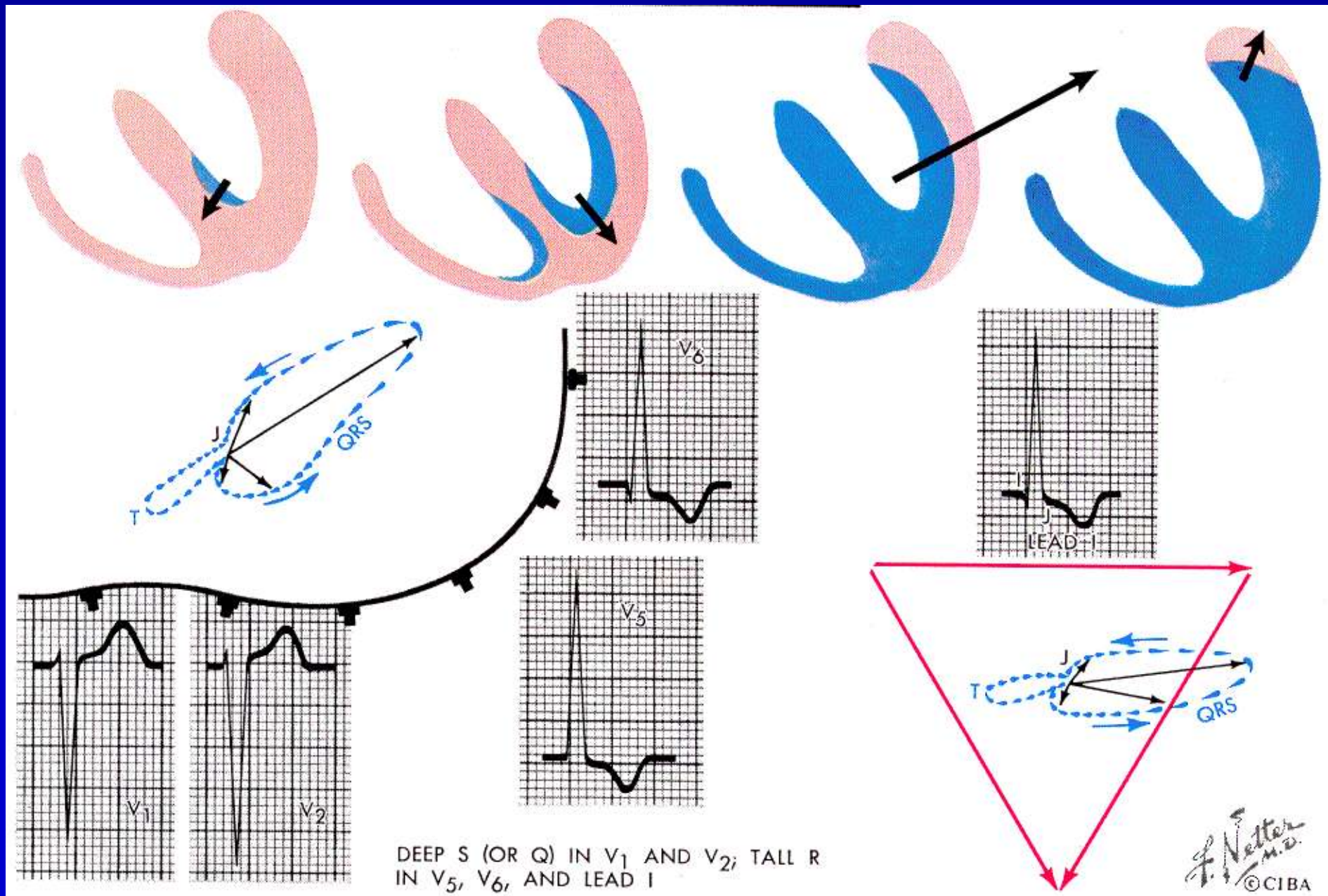




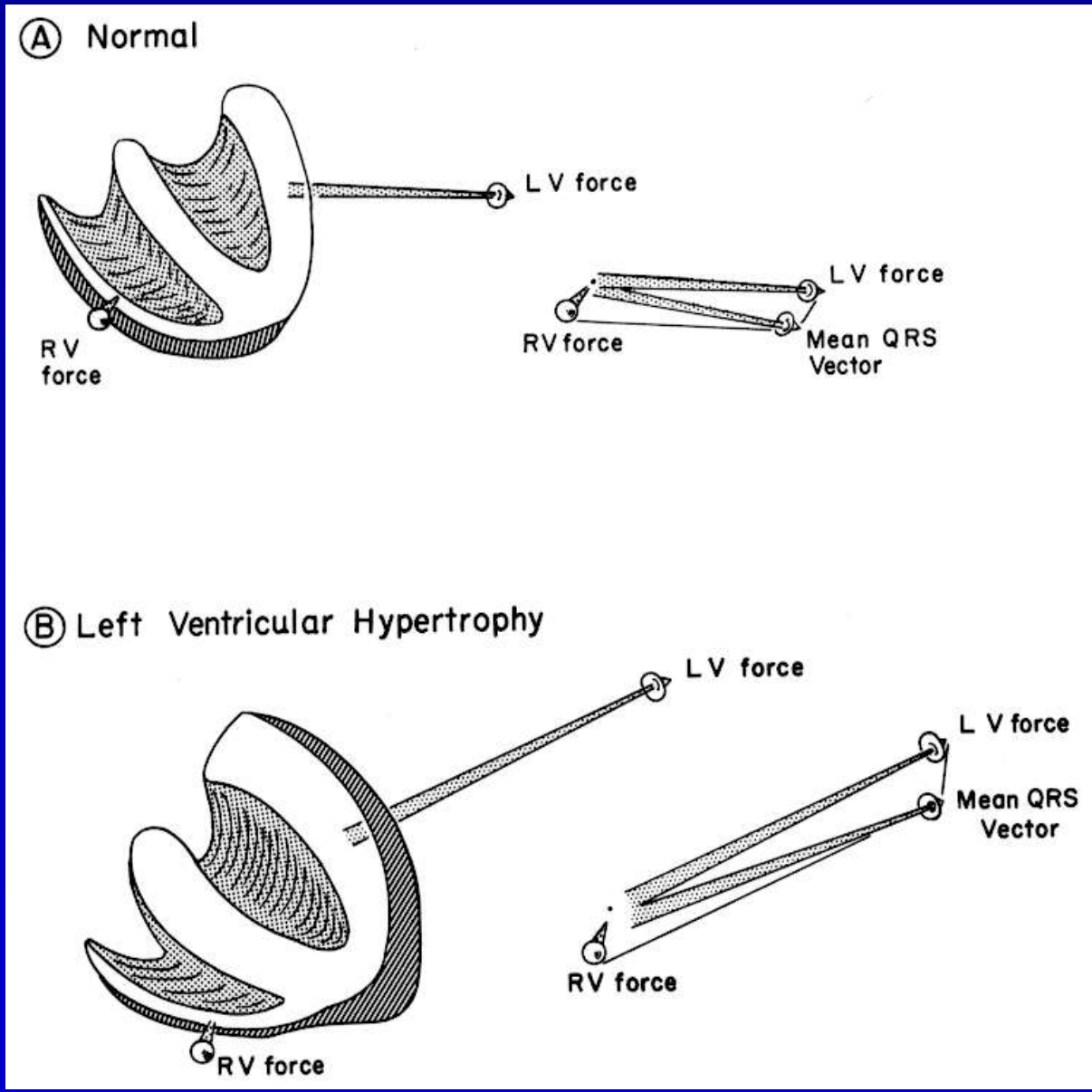


–P duration >0.12 sec (3 mm)

Left Ventricular Hypertrophy

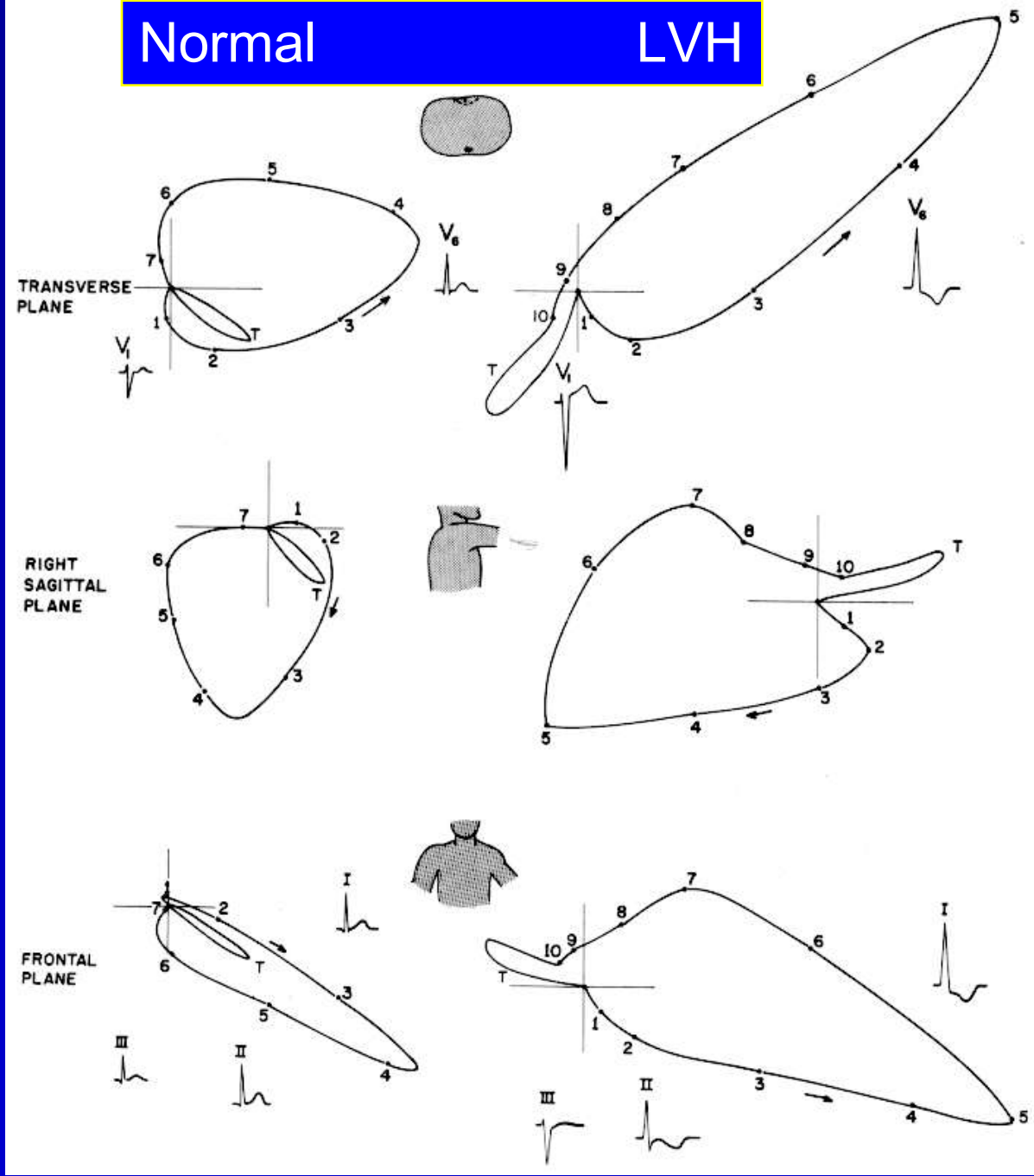


Left Ventricular Hypertrophy



Chou et al. Clinical Vectorcardiography 2nd ed 1974. p. 72.

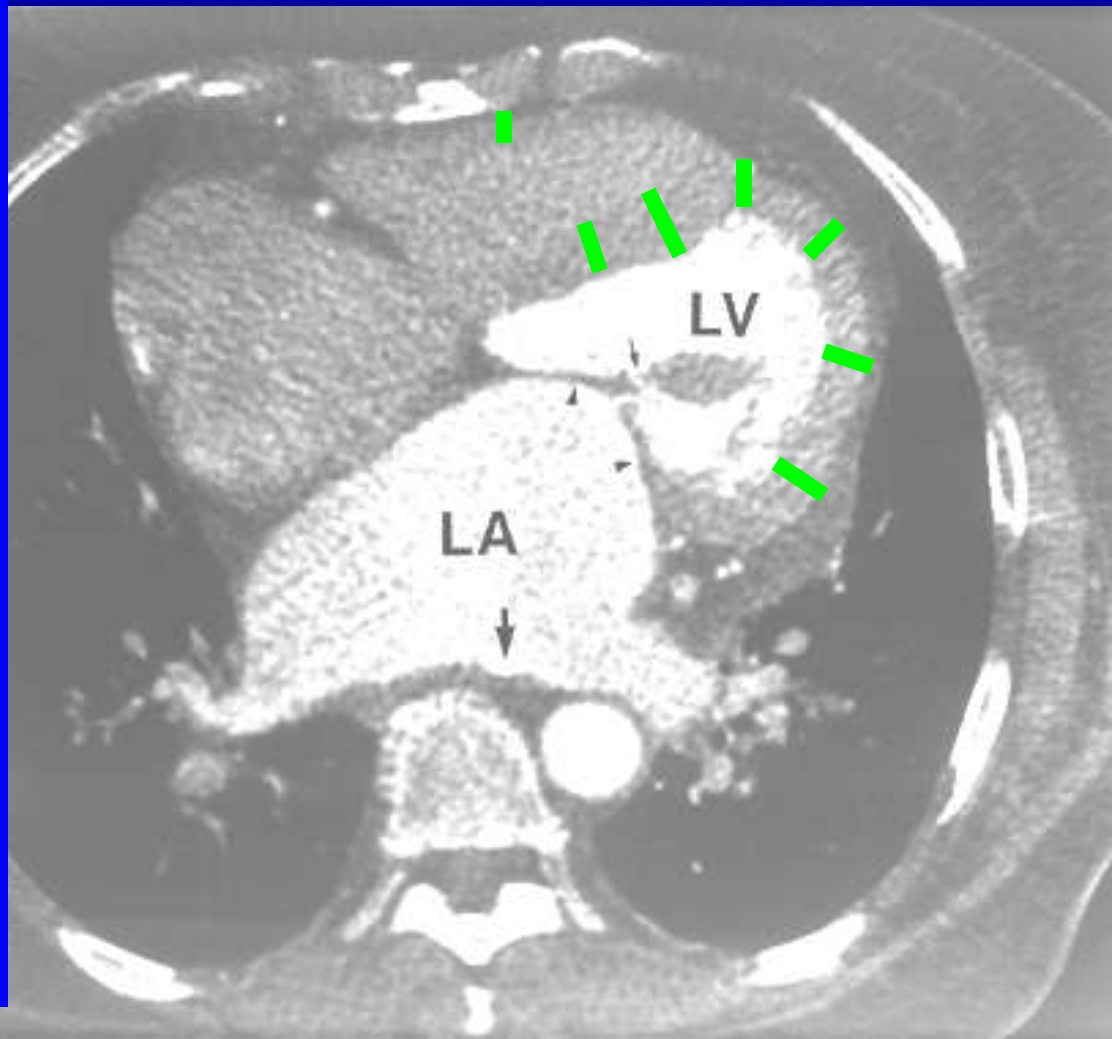
Left Ventricular Hypertrophy



Chou et al. Clinical Vectorcardiography 2nd ed 1974. p. 73.

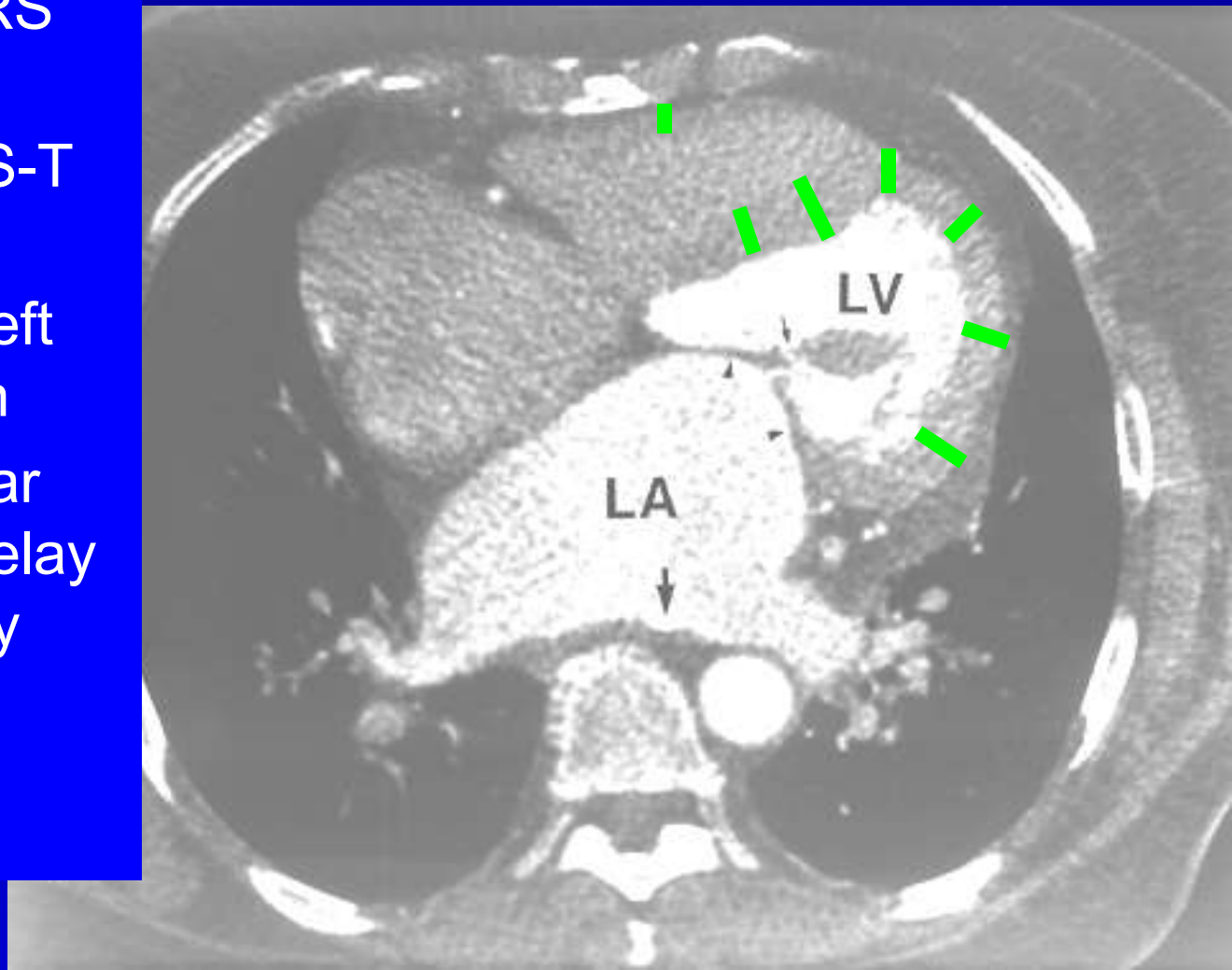
Left Ventricular Hypertrophy

- The LV normally dominates the QRS, since it is about 10 mm thick and the RV is only a few mm thick.
- When the LV mass increases, the ECG forces directed leftward and inferiorly and posteriorly increase.



Left Ventricular Hypertrophy

- Summary:
  - increased QRS amplitude,
  - widened QRS-T angle, and
  - tendency to left axis deviation
  - intraventricular conduction delay manifested by delayed intrinsicoid deflection



Left Ventricular Hypertrophy

Point Score System (Romhilt and Estes, 1968)

- 3 points: Voltage of R or S of 20 mm in limb lead, or 30 mm in chest lead
- 3 points: Left atrial abnormality in V1
- 3 points: Repolarization abnormal off dig.
- 1 point: Repolarization abnormal on dig.
- 1 point: Intrinsicoid deflection in V5-V6 >0.05 sec
- 1 point: QRS duration 0.09-0.10 sec
- 2 points: LAD <-30 degrees (not very helpful)

Sensitivity 54%

Specificity 97%

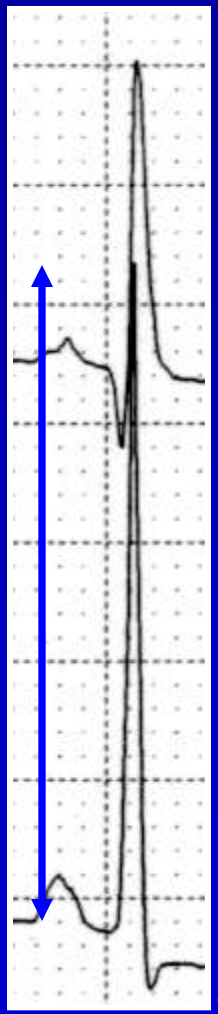
Surawicz 2001 Ch. 3.

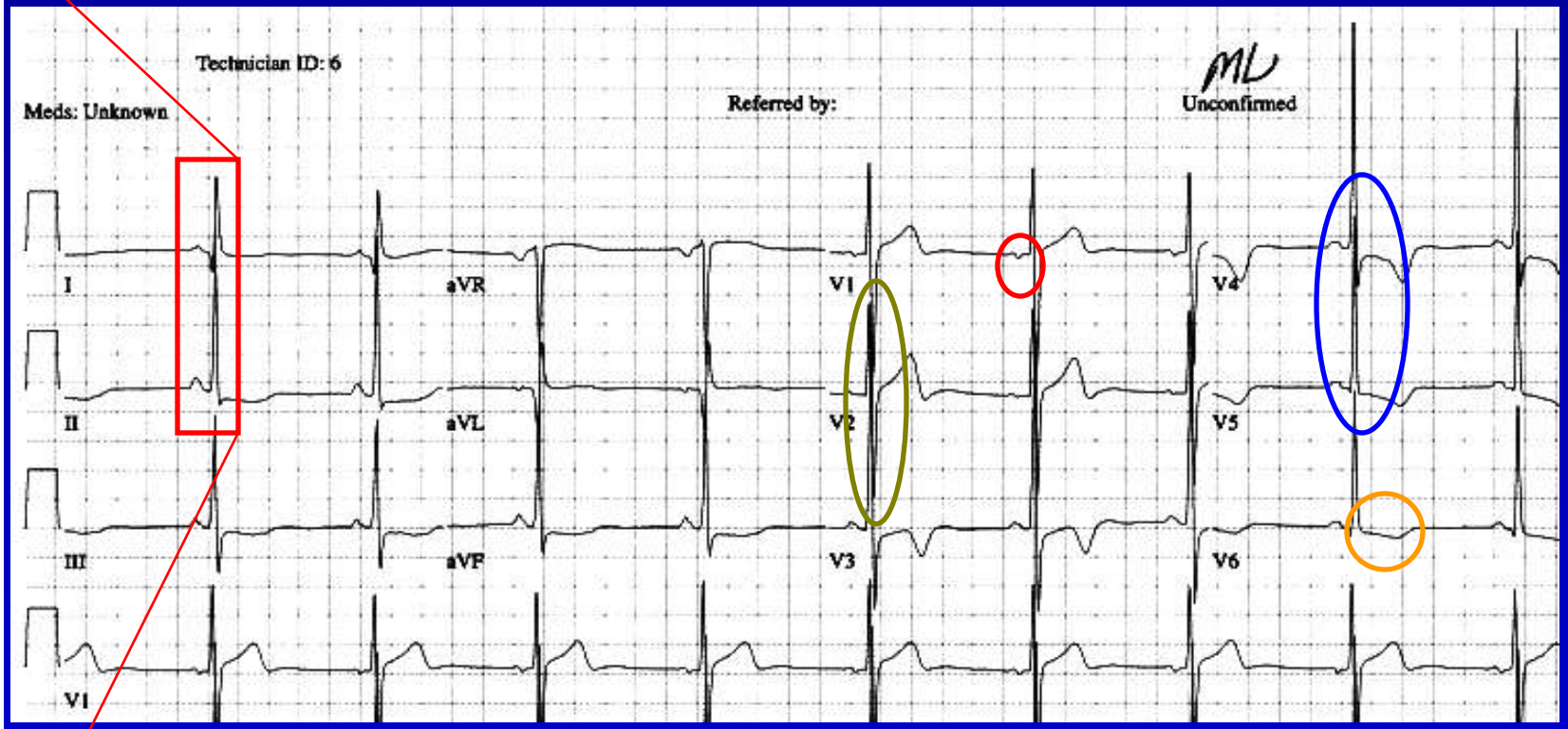
Left Ventricular Hypertrophy

- Cornell Voltage Criteria
- R wave in aVL + S wave in lead V3!
- Women: >2.0 mV (20 mm)
- Men: >2.8 mV (28 mm)

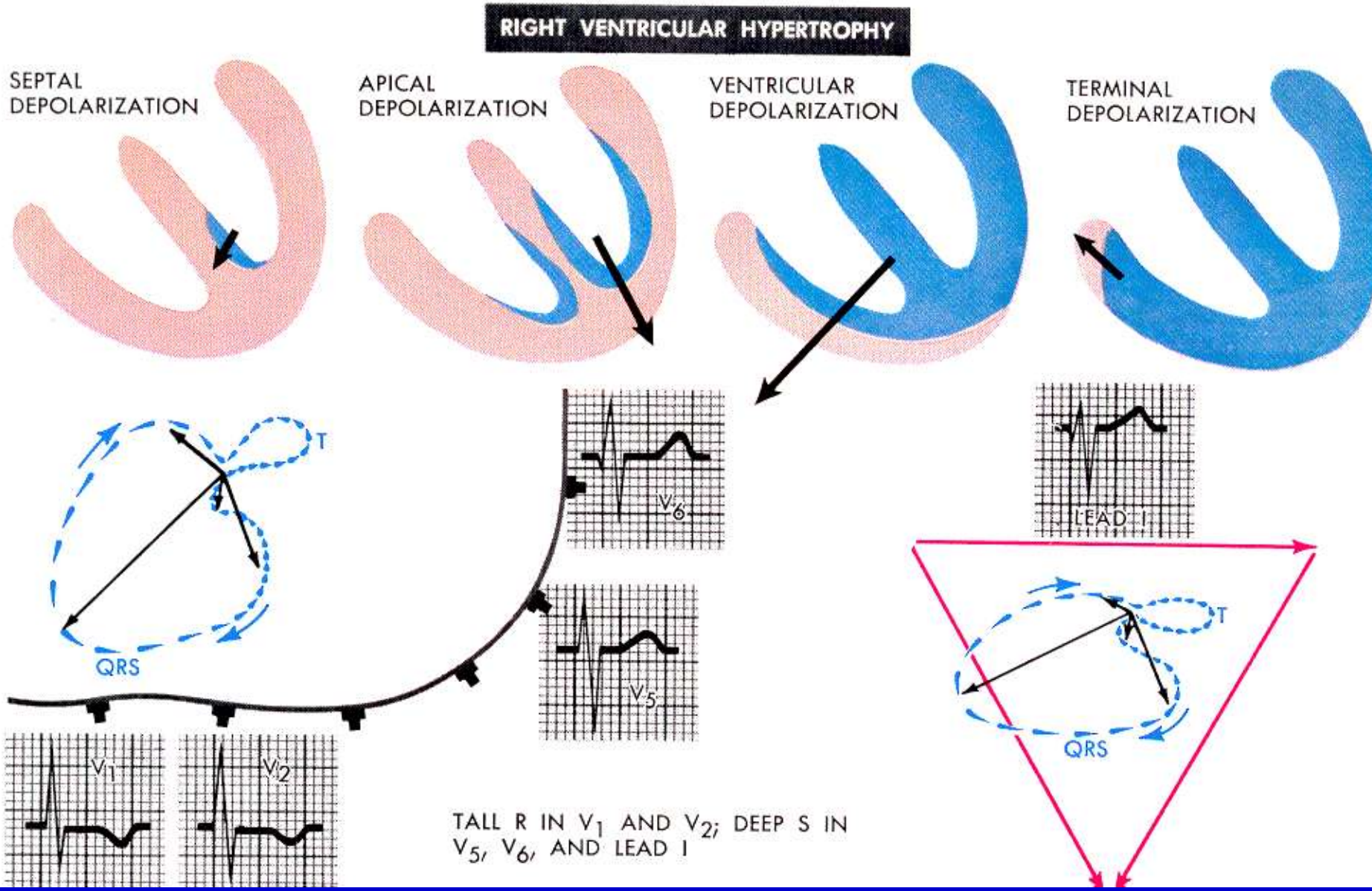
Surawicz 2001 Ch. 3. p. 46.

ECG LVH

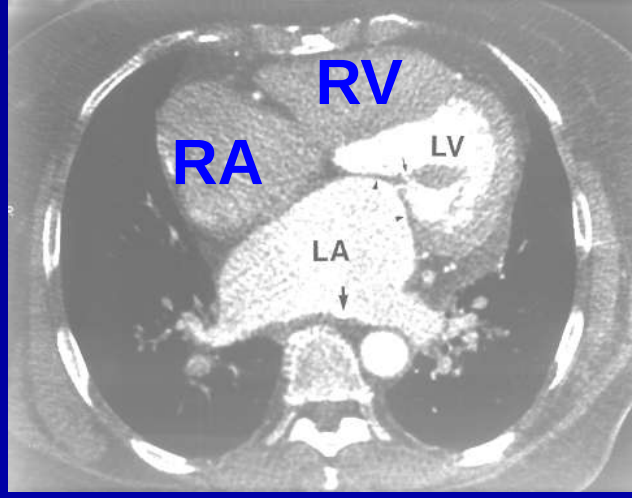



Technician ID: 6
Meds: Unknown
Referred by:
Unconfirmed
I
II
III
VR
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6

Right Ventricular Hypertrophy

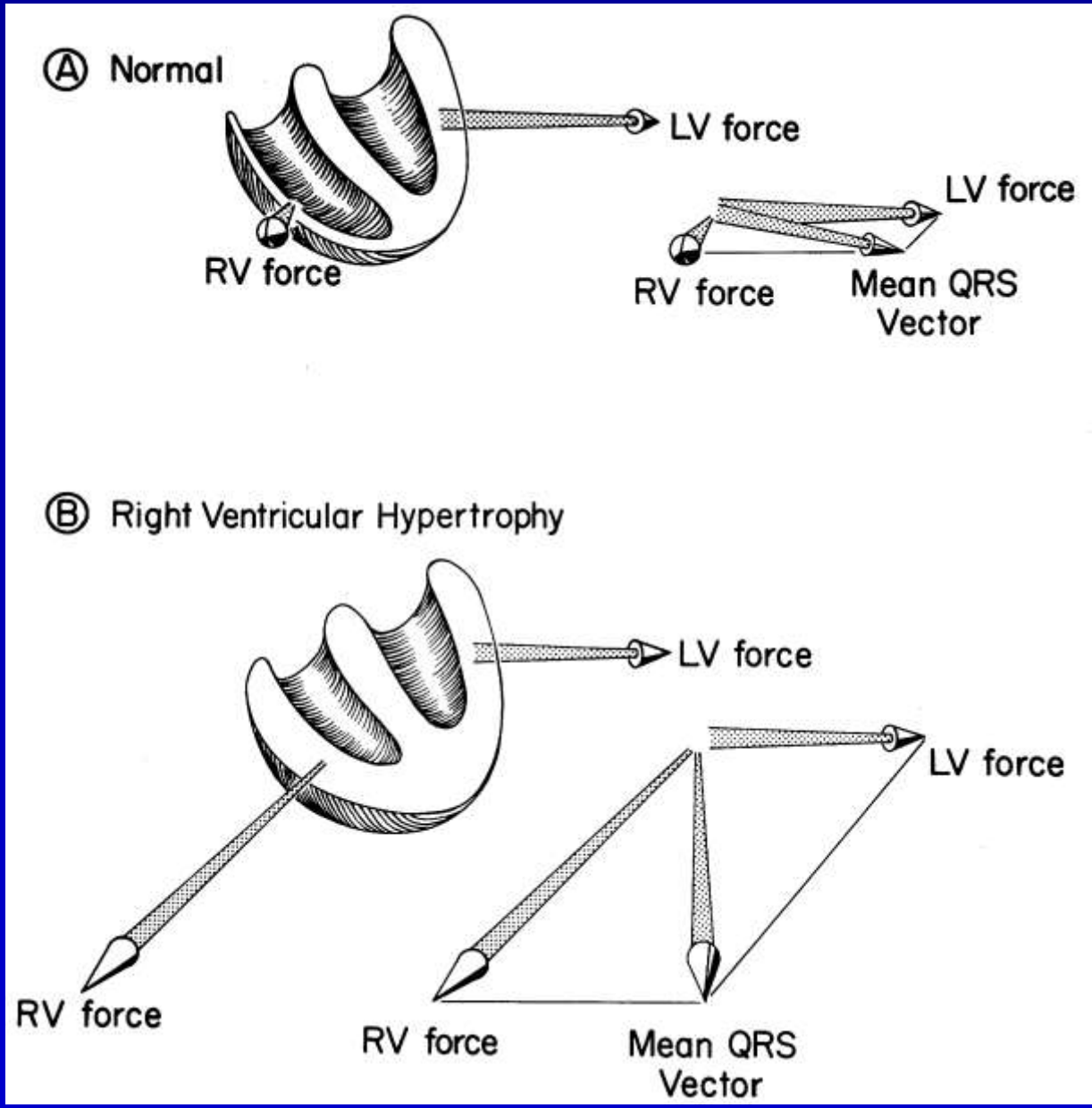


Right Ventricular Hypertrophy



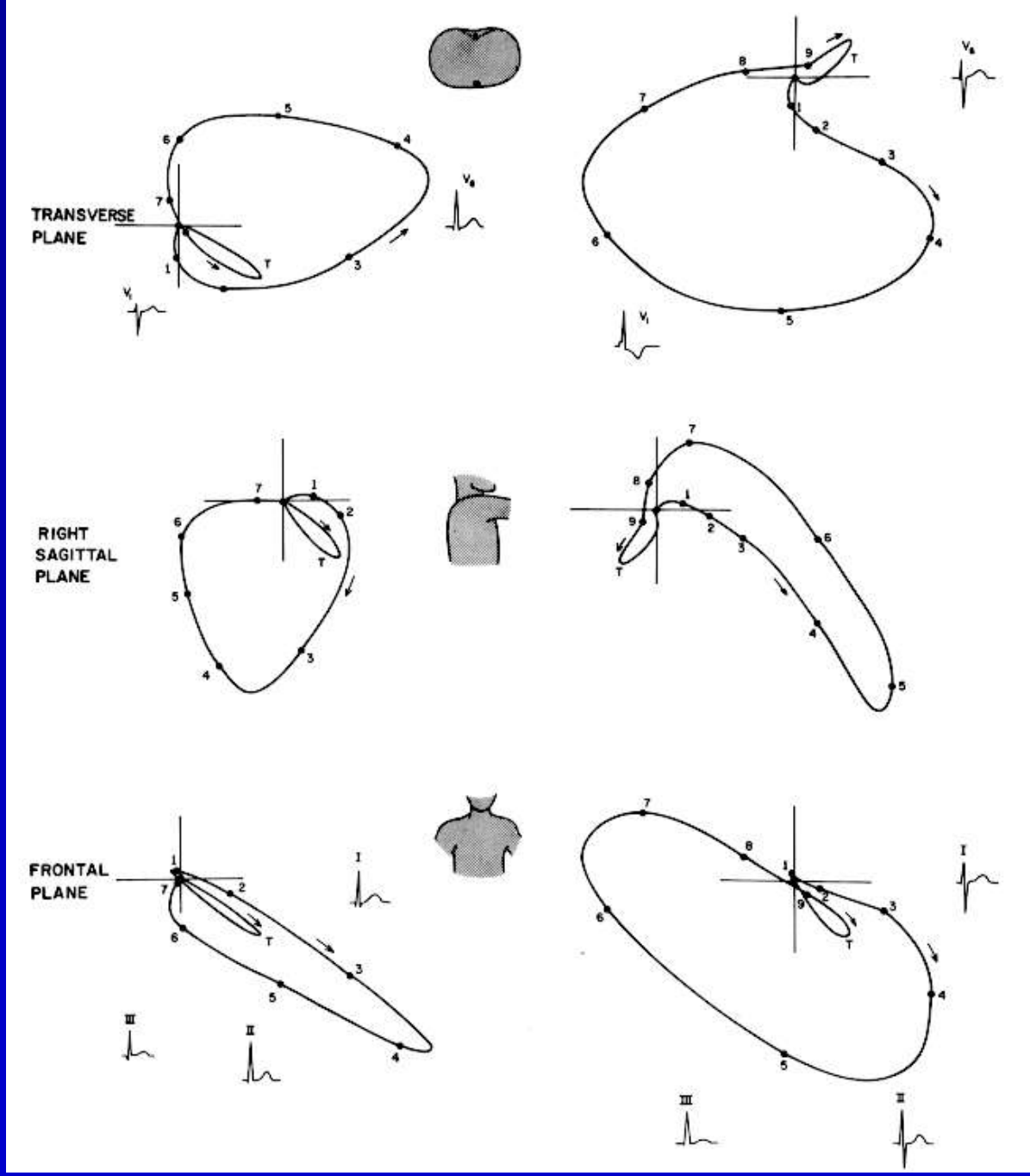
- Tougher than LVH, because RV forces must increase more to overcome the normally dominant LV forces
- RV forces are rightward and anterior and inferior
- Criteria
  - Right axis >110 degrees
  - R/S in V1 or V3R >1
  - R in V1 >7mm
  - S in V1 <2mm
  - qR pattern in V1 or V3R
  - rSRprime in V1 with Rprime >10mm
  - also, ST depression and T inversion in V1-V2

Right Ventricular Hypertrophy



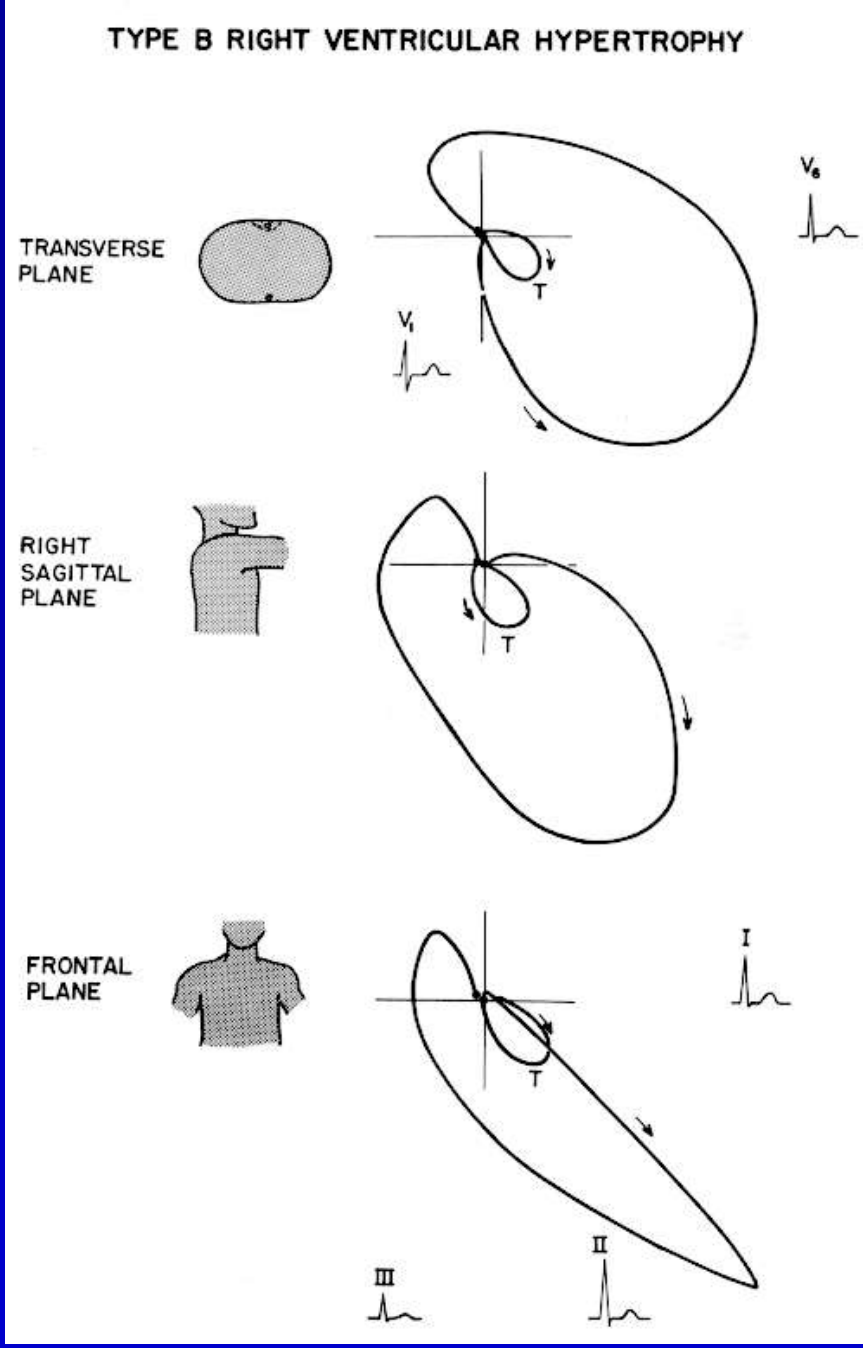
Chou et al. Clinical Vectorcardiography 2nd ed 1974. p. 86.

Right Ventricular Hypertrophy Type A



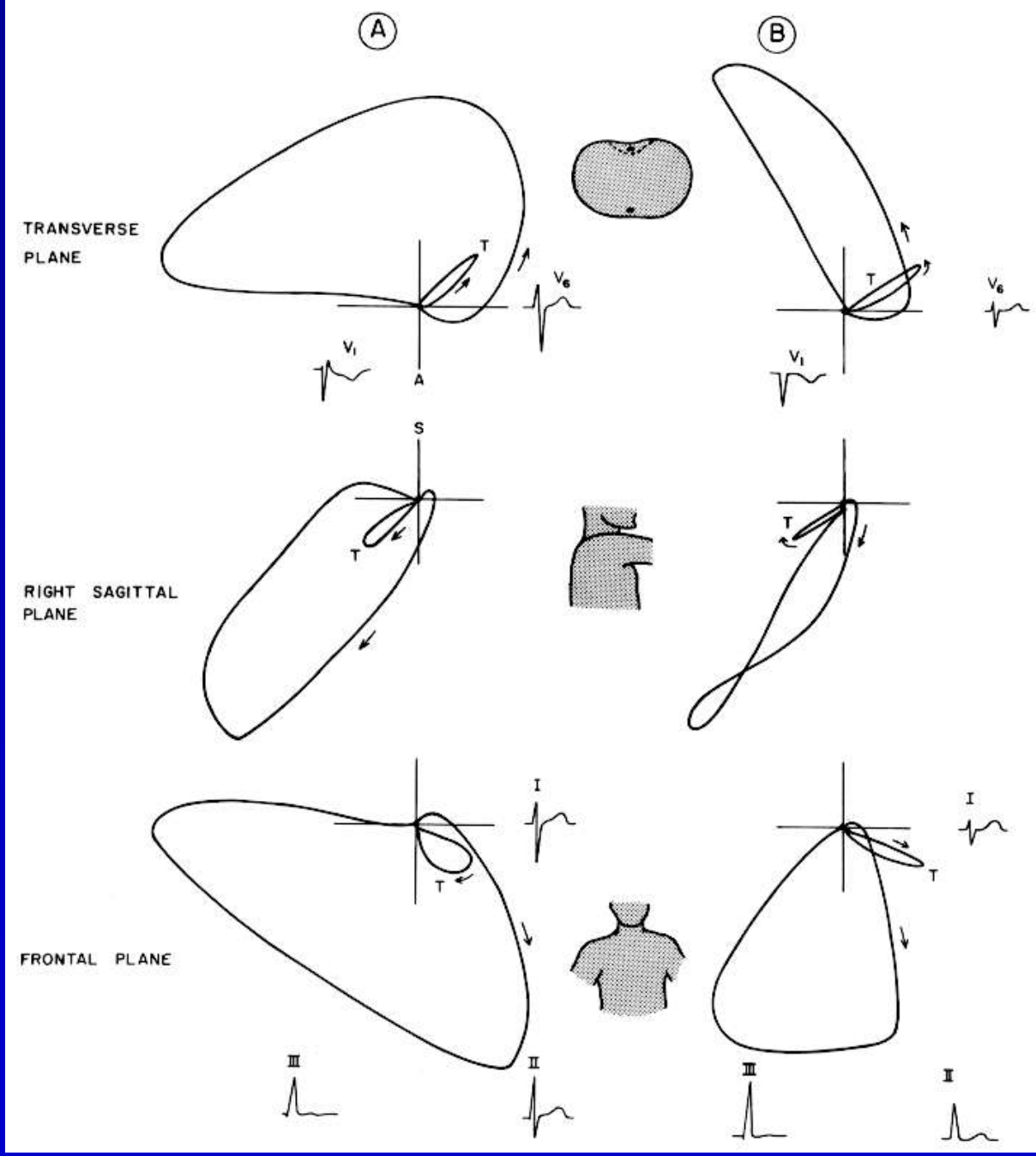
Chou et al. Clinical Vectorcardiography 2nd ed 1974. p. 87.

Right Ventricular Hypertrophy Type B



Chou et al. Clinical Vectorcardiography 2nd ed 1974. p. 99.

Right Ventricular Hypertrophy Type C

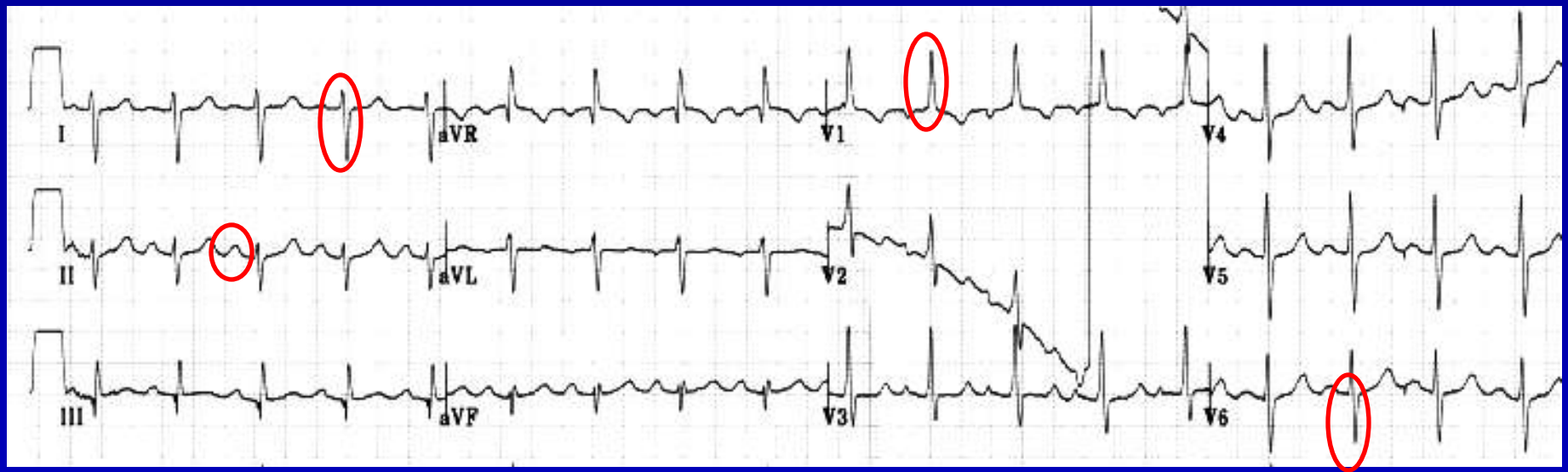


Chou et al. Clinical Vectorcardiography 2nd ed 1974. p. 102.

Types of RVH by ECG

	V1	V6
Normal	-	+
RVH type A	+	-
RVH type B	+	+
RVH type C	-	-

ECG - RVH



–Right axis >110 degrees

–R/S in V1 or V3R >1

–R in V1 >7mm

–S in V1 <2mm

–qR pattern in V1 or V3R

–rSRprime in V1 with Rprime >10mm

–also, ST depression and T inversion in V1-V2

ECG - RVH

–Right axis >110 degrees

–R/S in V1 or V3R >1

–R in V1 >7mm

–S in V1 <2mm

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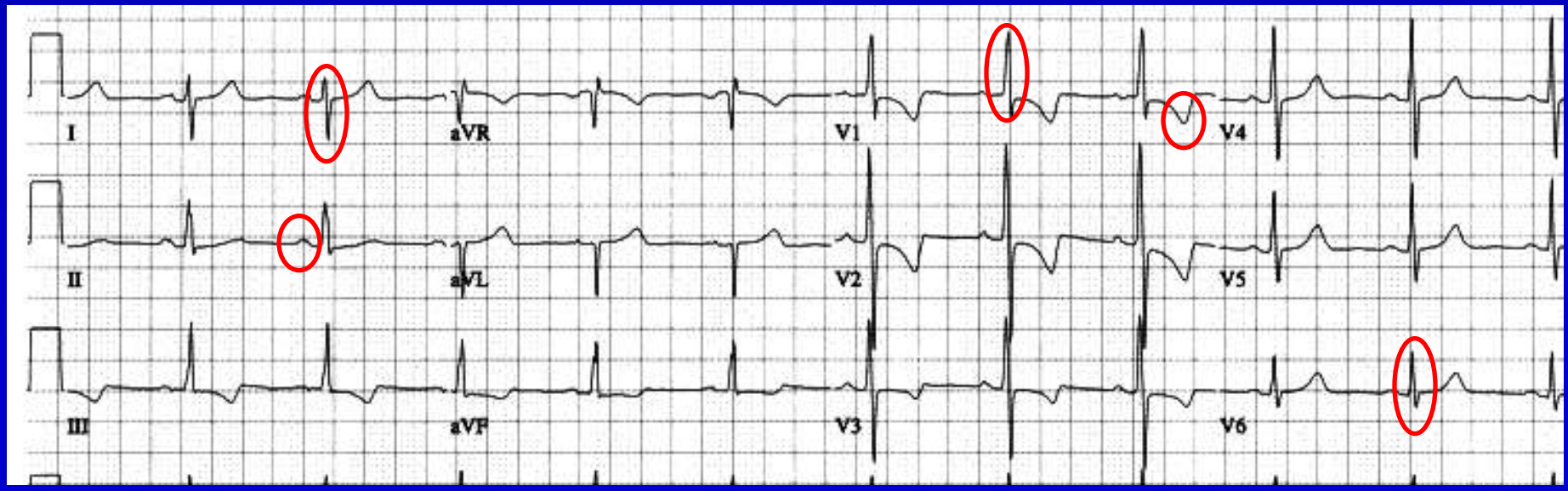
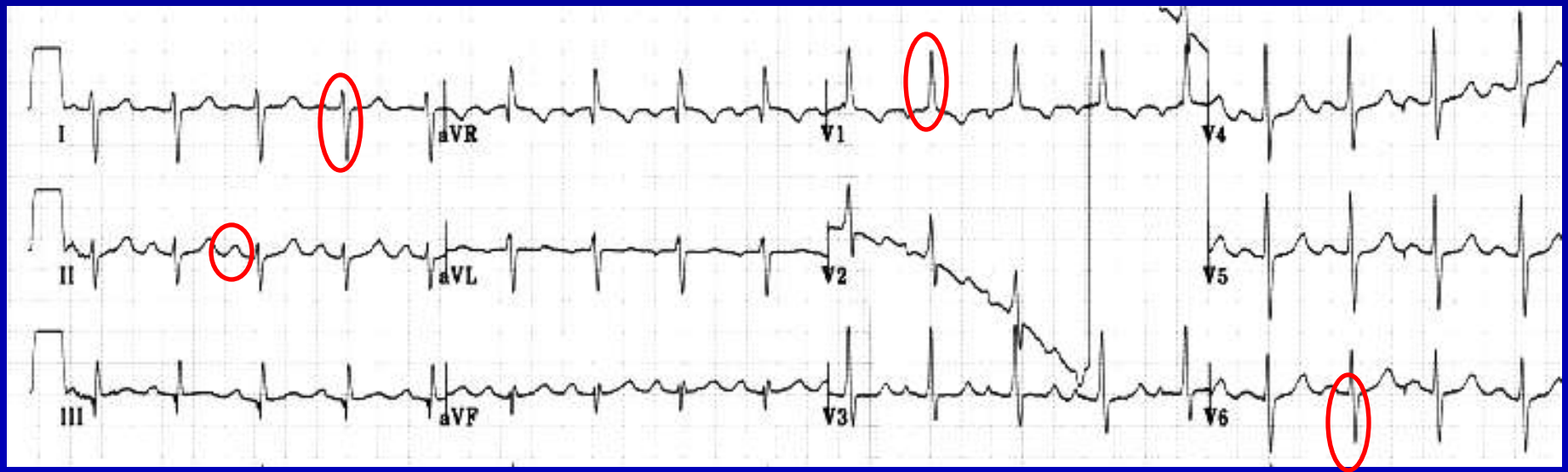
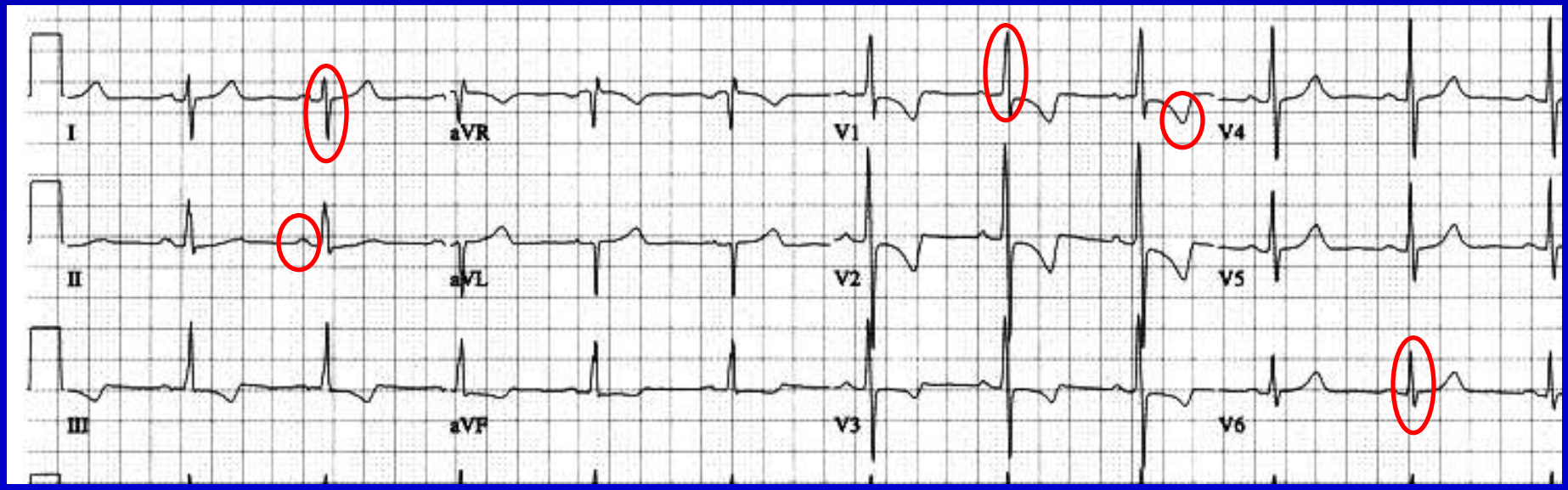
–also, ST depression and T inversion in V1-V2



ECG - RVH


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


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

Biventricular Hypertrophy

- The same ECG meets one or more criteria for both isolated LVH and RVH
- Precordial leads look like LVH but QRS axis in limb leads is > 90 degrees
- Signs of LVH and R>Q in aVR and S>R in V5 and T inversion in V1

Surawicz 2001 Ch. 3. p. 68.

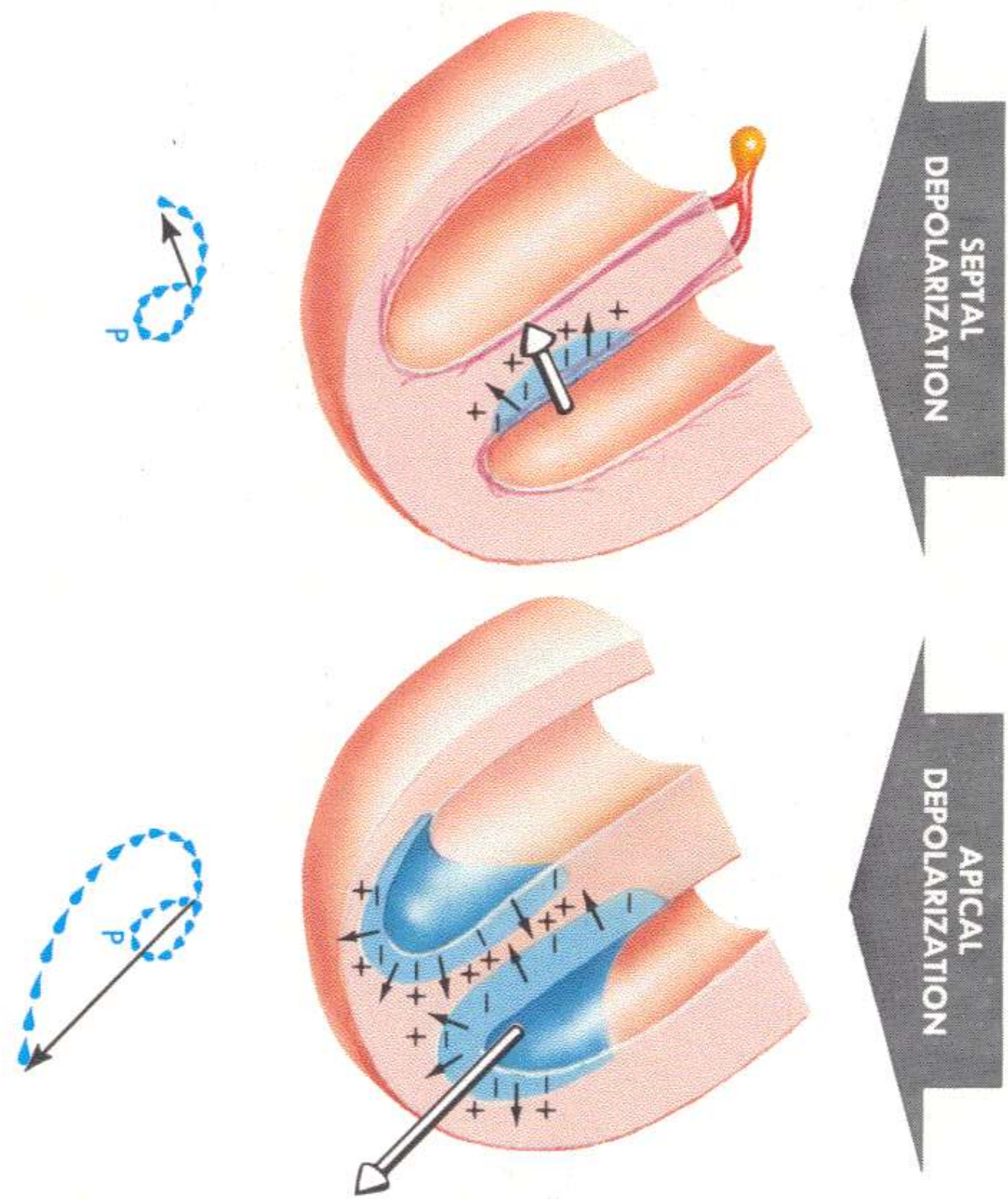
Conduction Abnormalities

- Normal ventricular activation
- LBBB
- LAFB, LPFB
- RBBB
- IVCD

Causes of wide QRS:

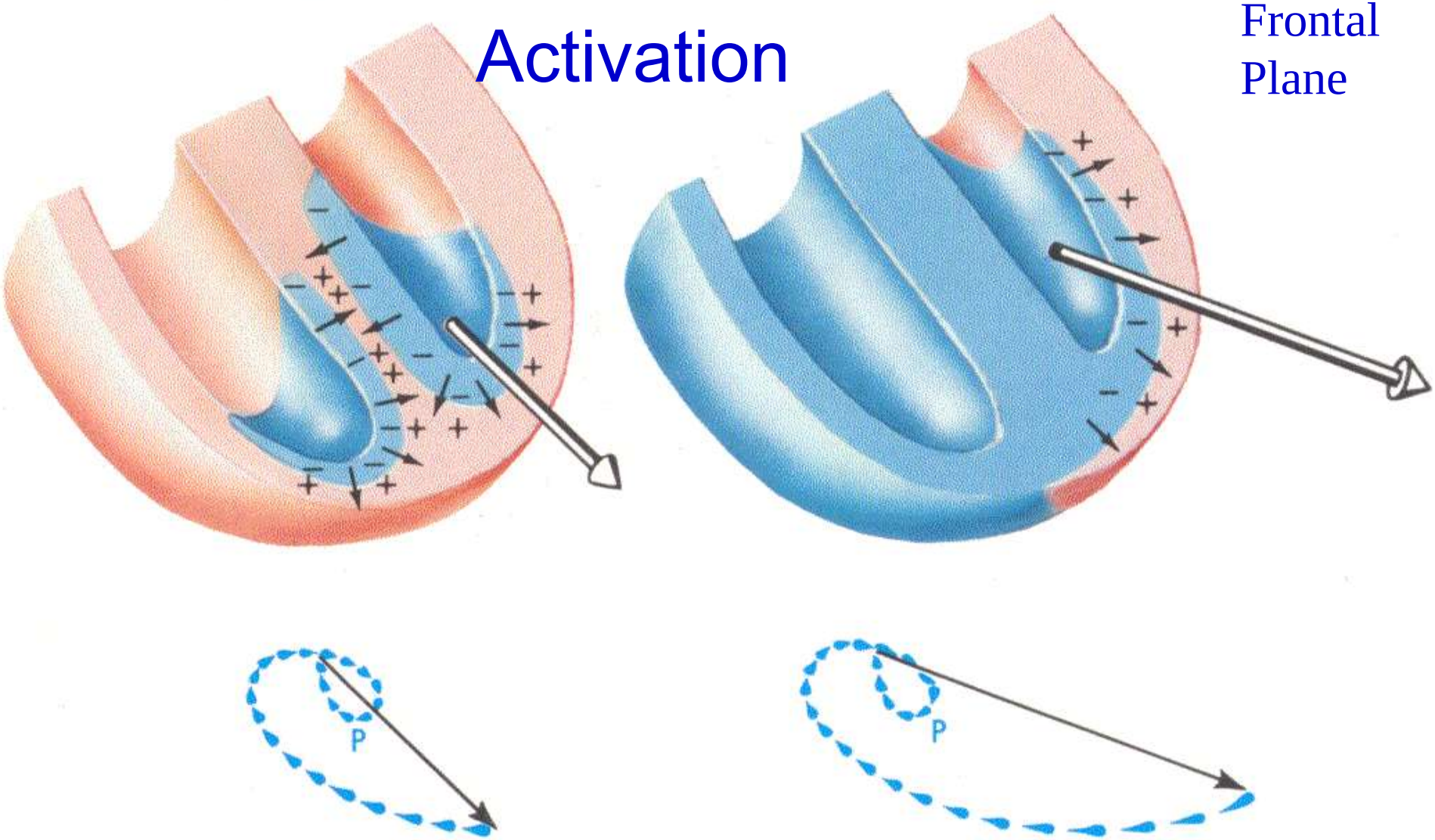
RBBB and bifascicular
LBBB
IVCD
WPW
Non-His origin beat
Paced QRS

Ventricular Activation



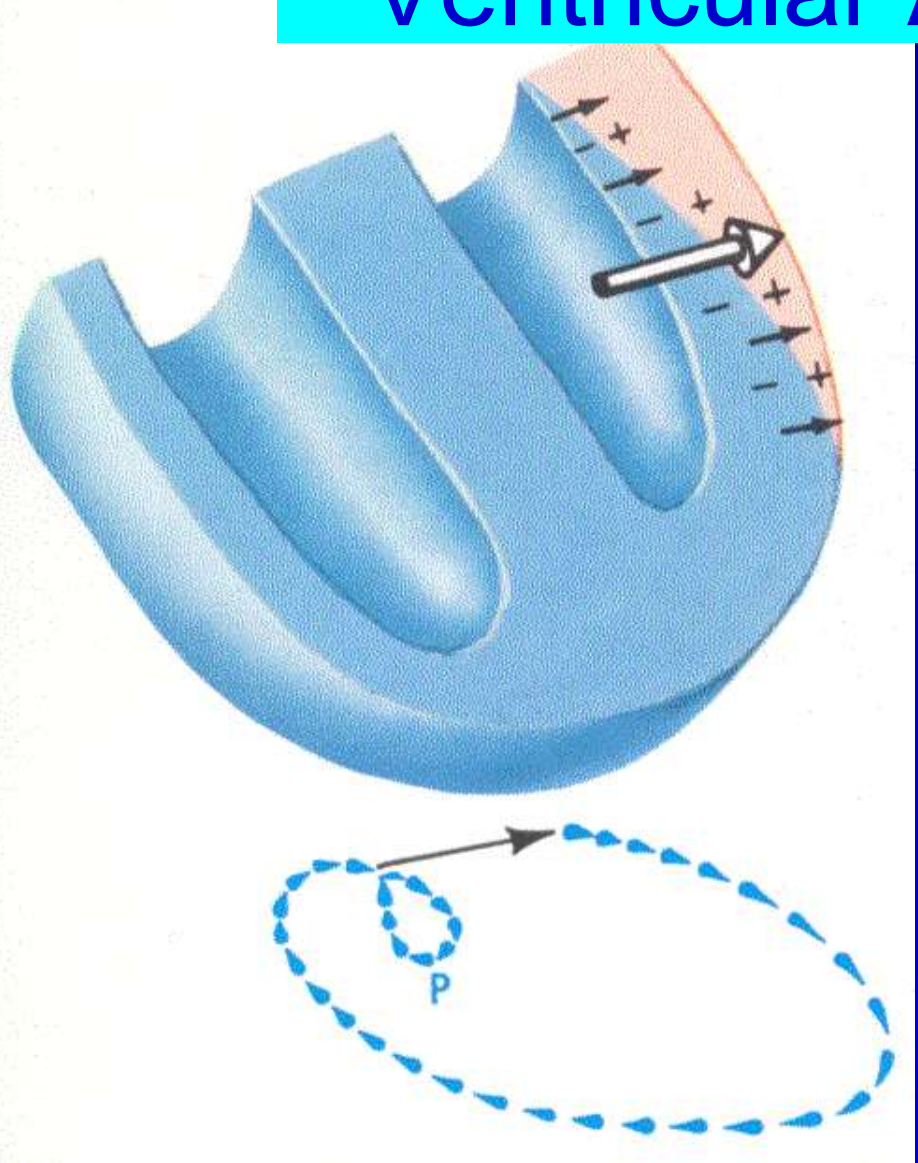
Ventricular Activation

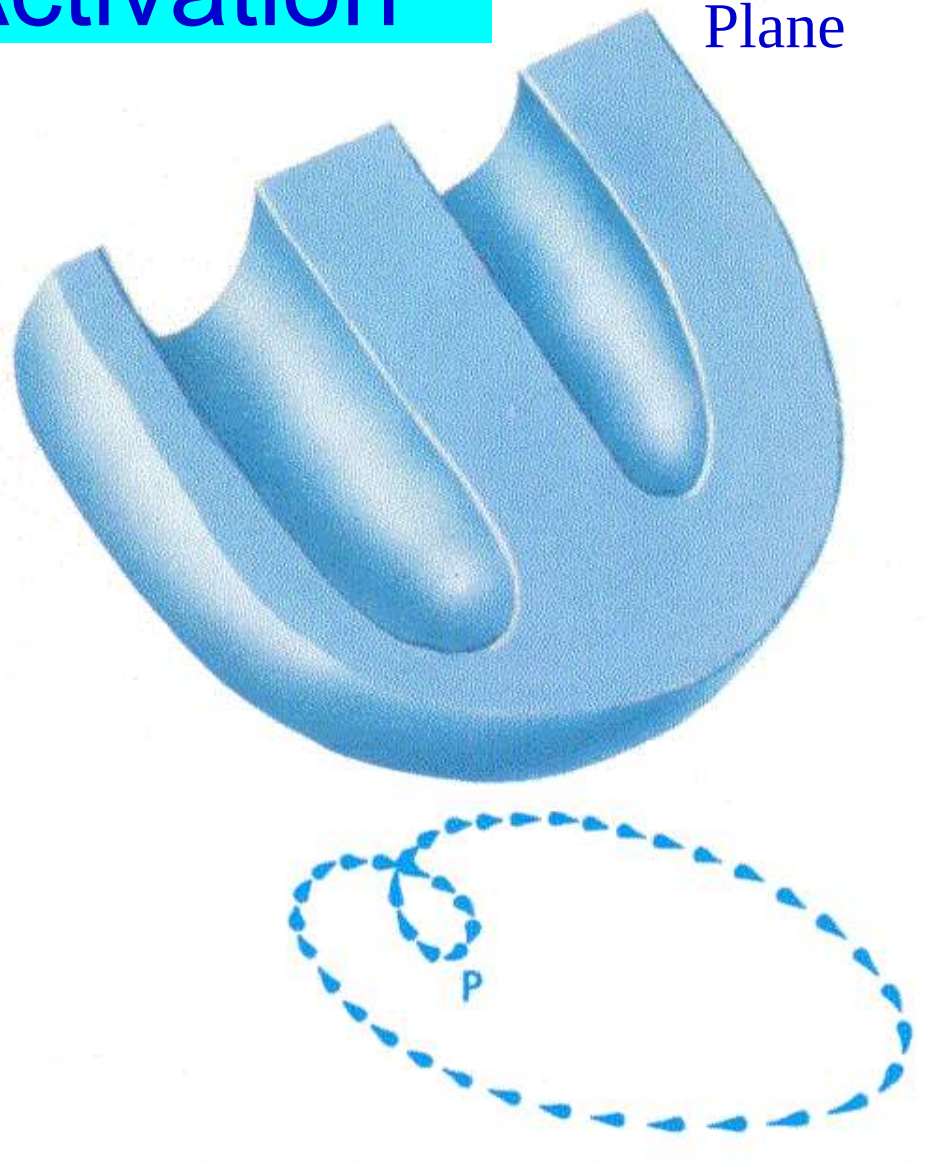
Frontal Plane



Ventricular Activation

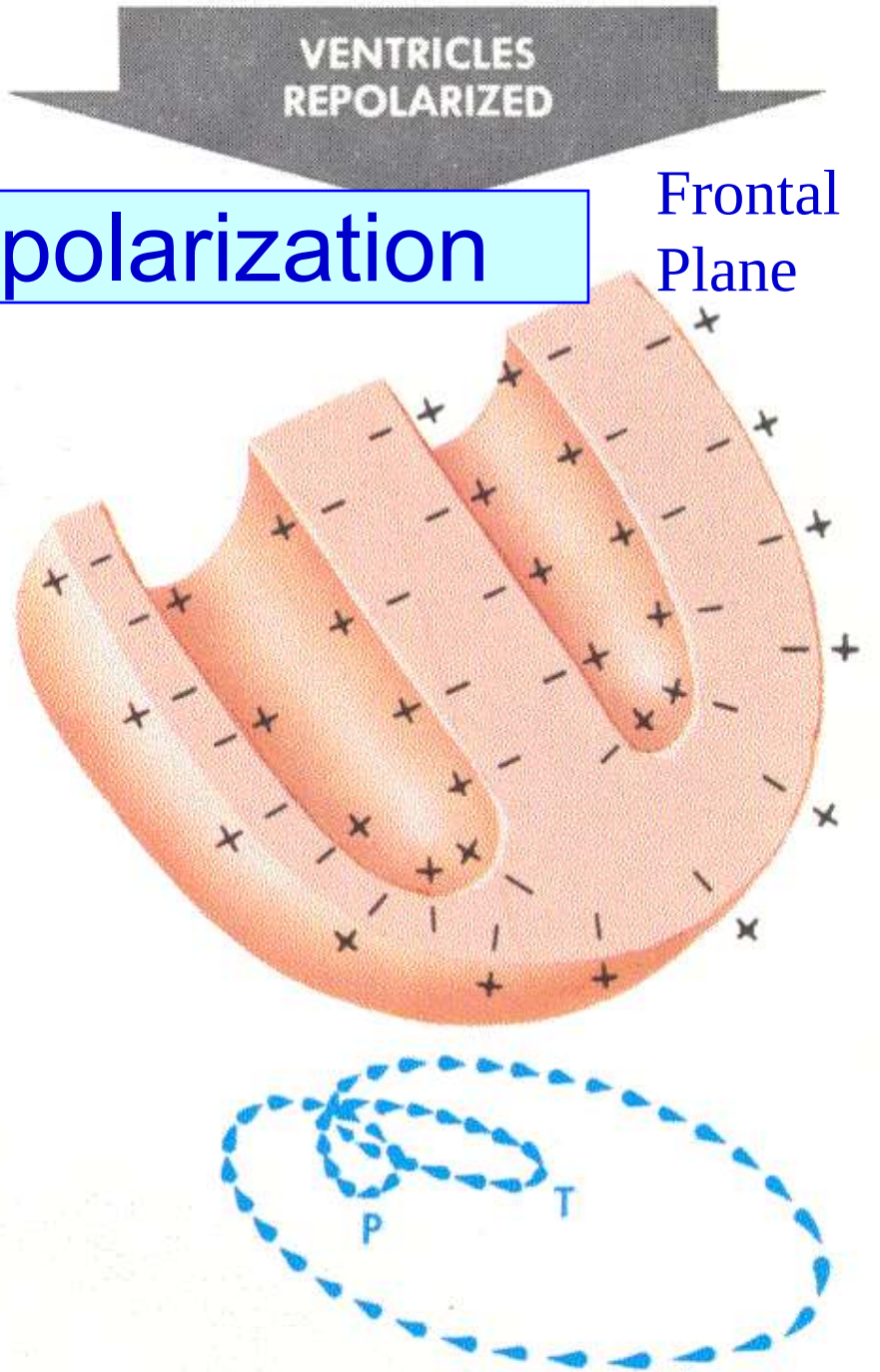
Frontal Plane

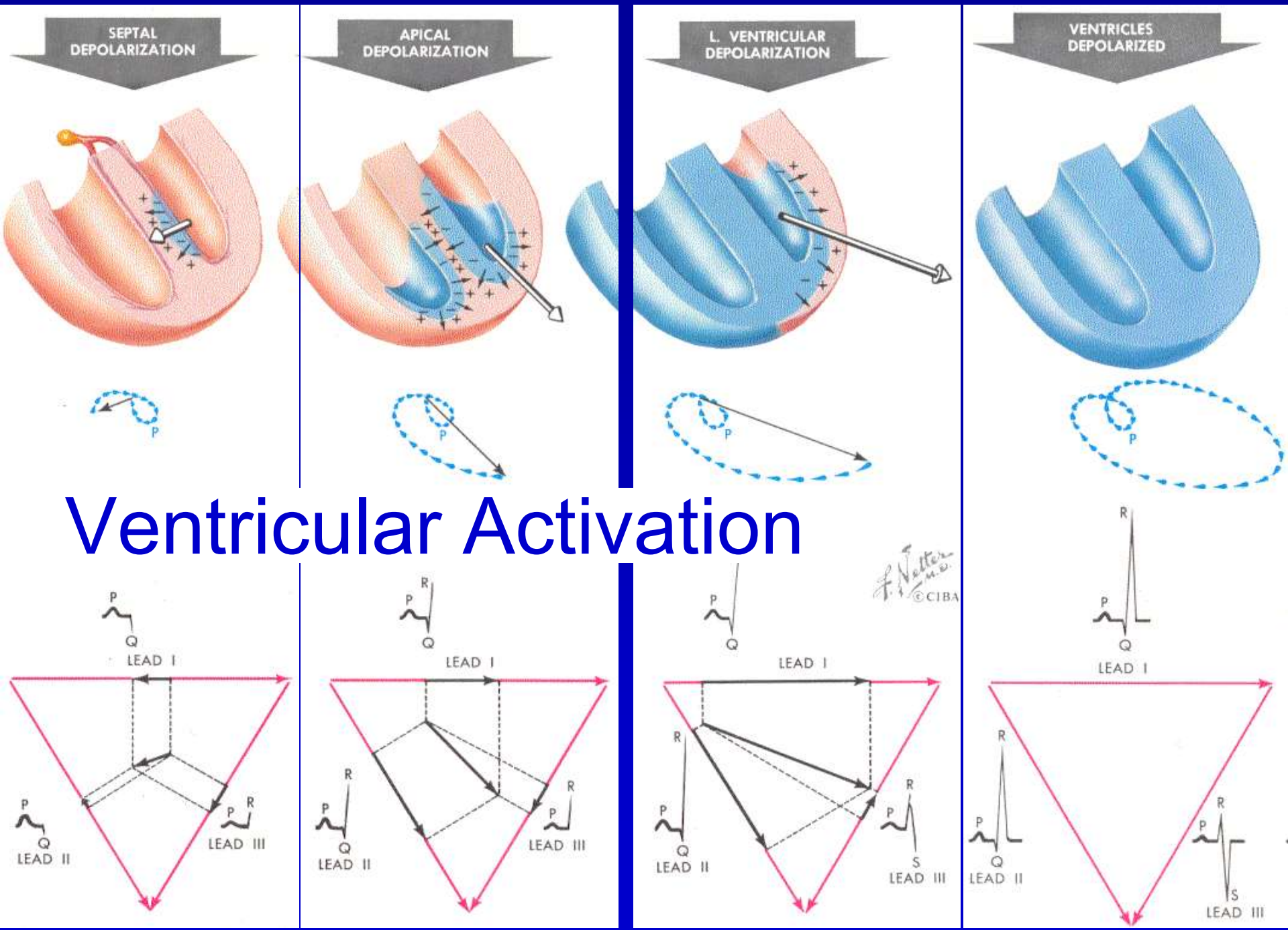




Ventricular Repolarization

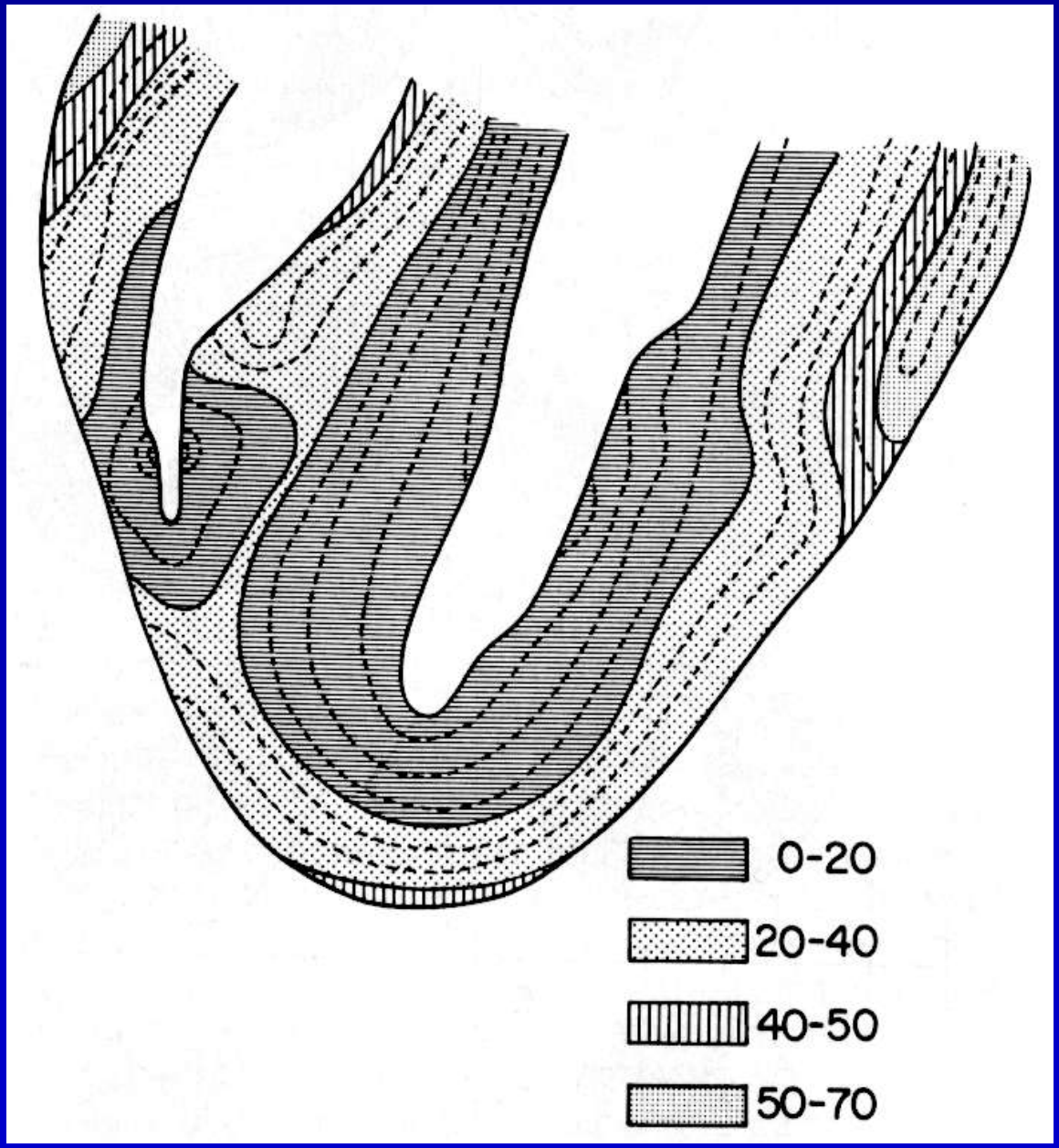
Frontal Plane



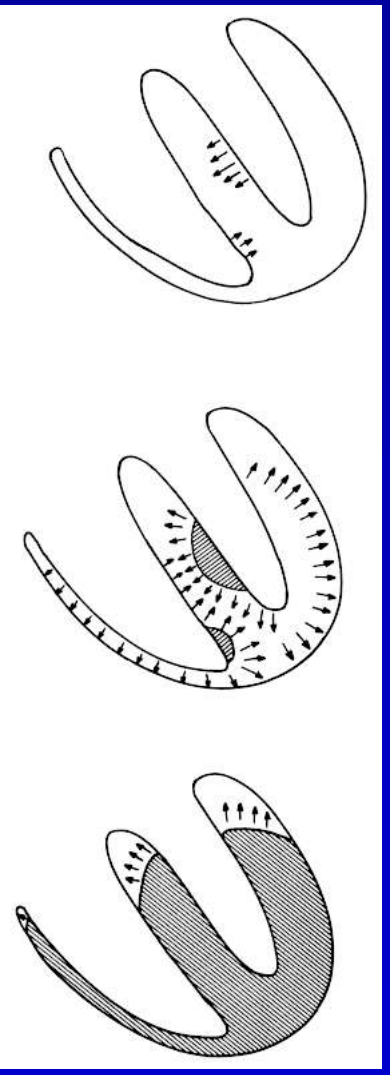


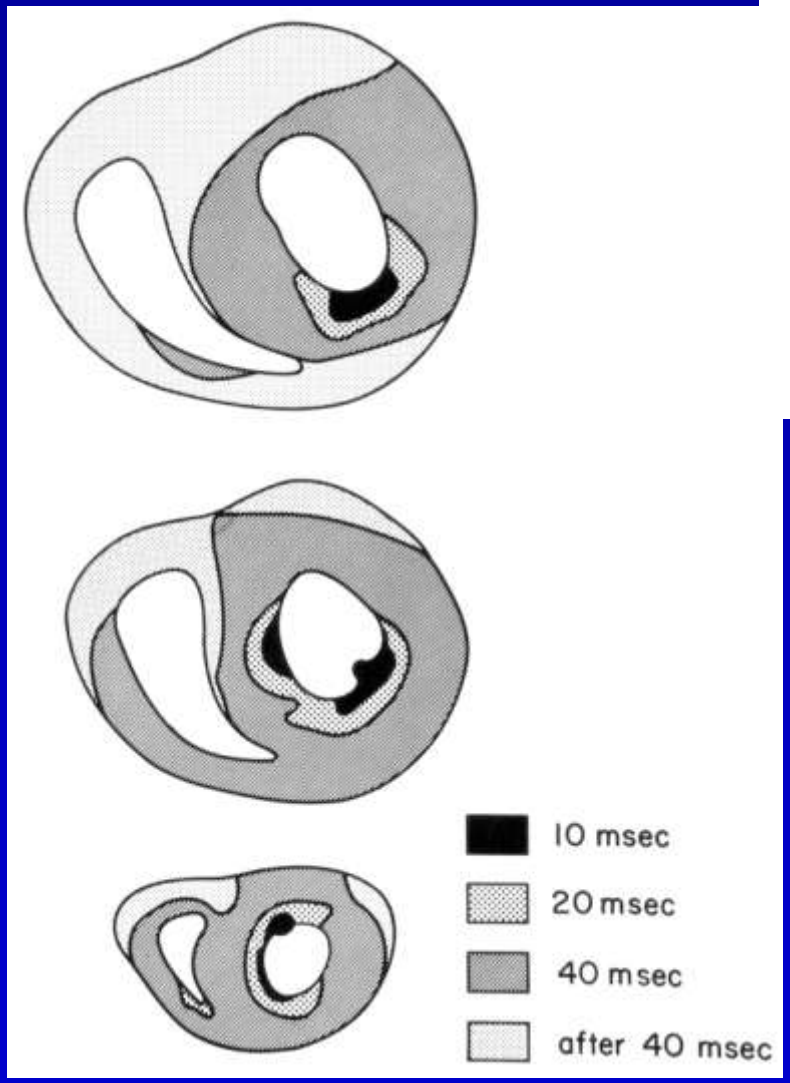
Ventricular Activation

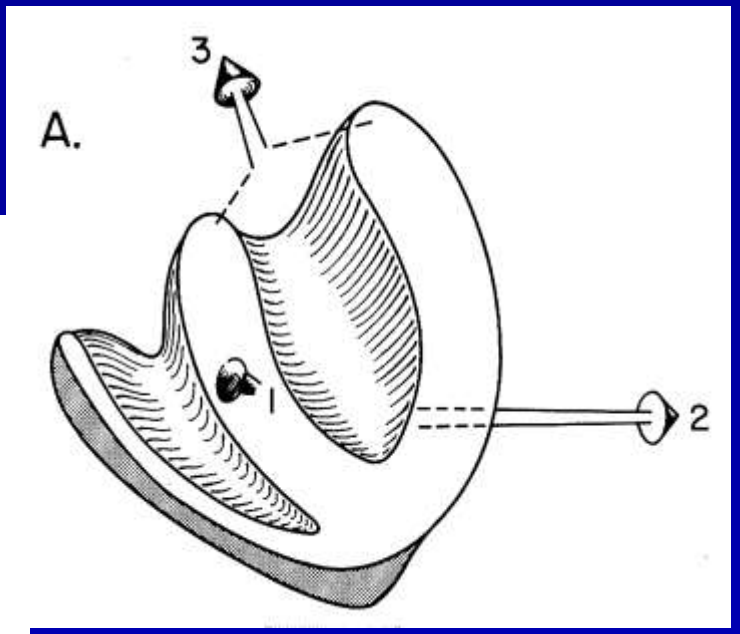
Sequence of Depolarization

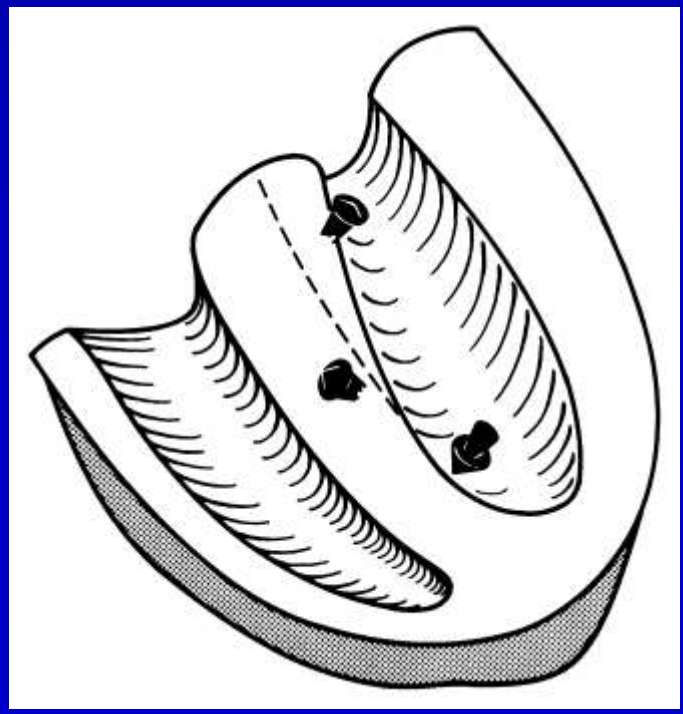


Sequence of Depolarization



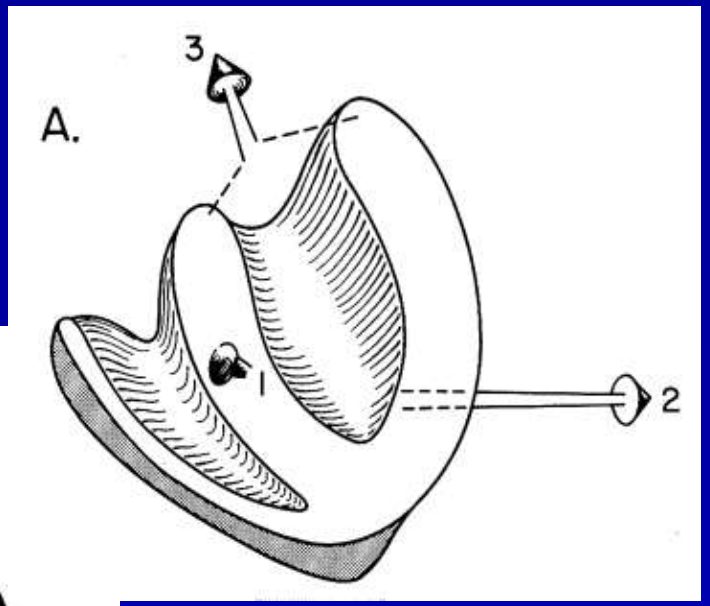


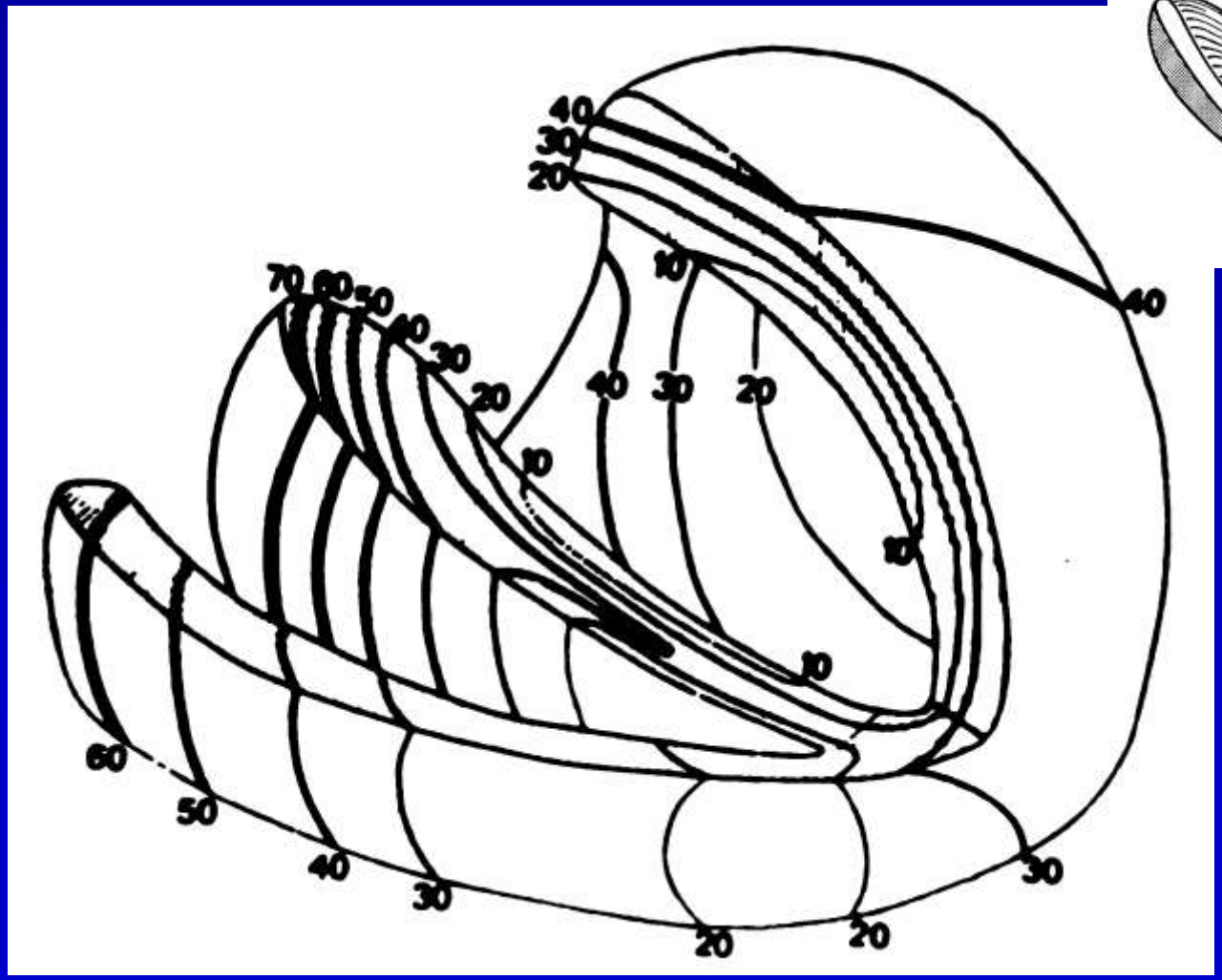




Chou, Vectorcardiography, 2nd ed. 1974 p. 36-9.

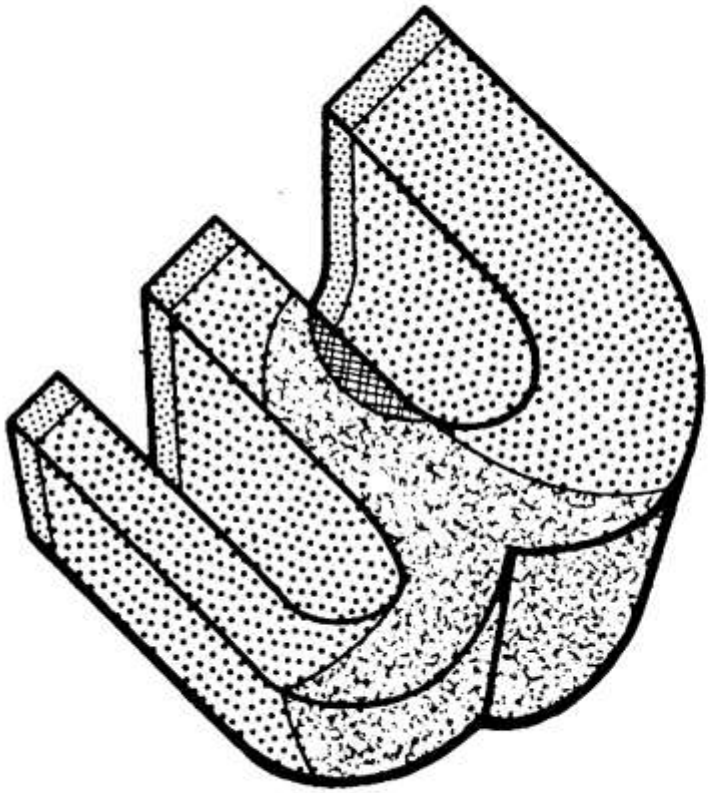
Sequence of Depolarization





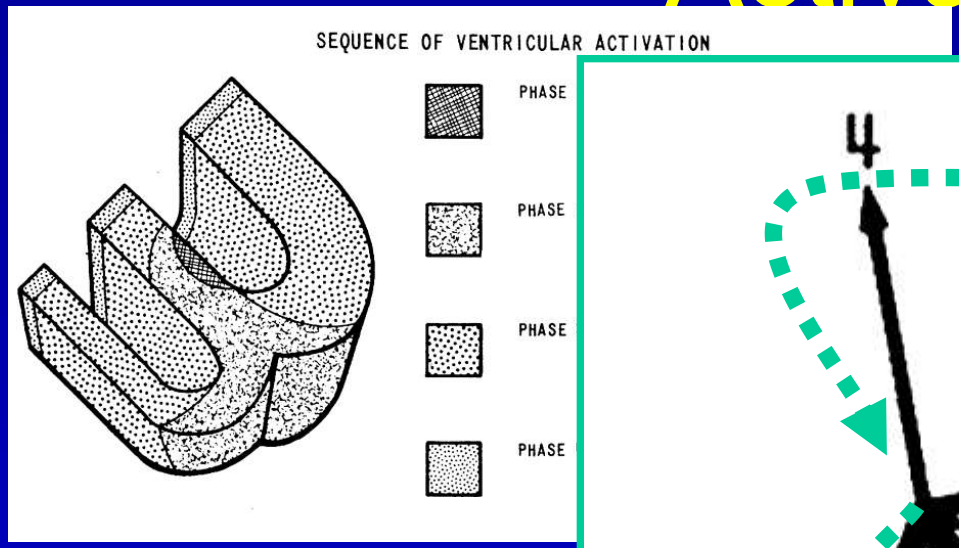
Chou, 2001, p.12-13
From Durrer 1968
Human heart

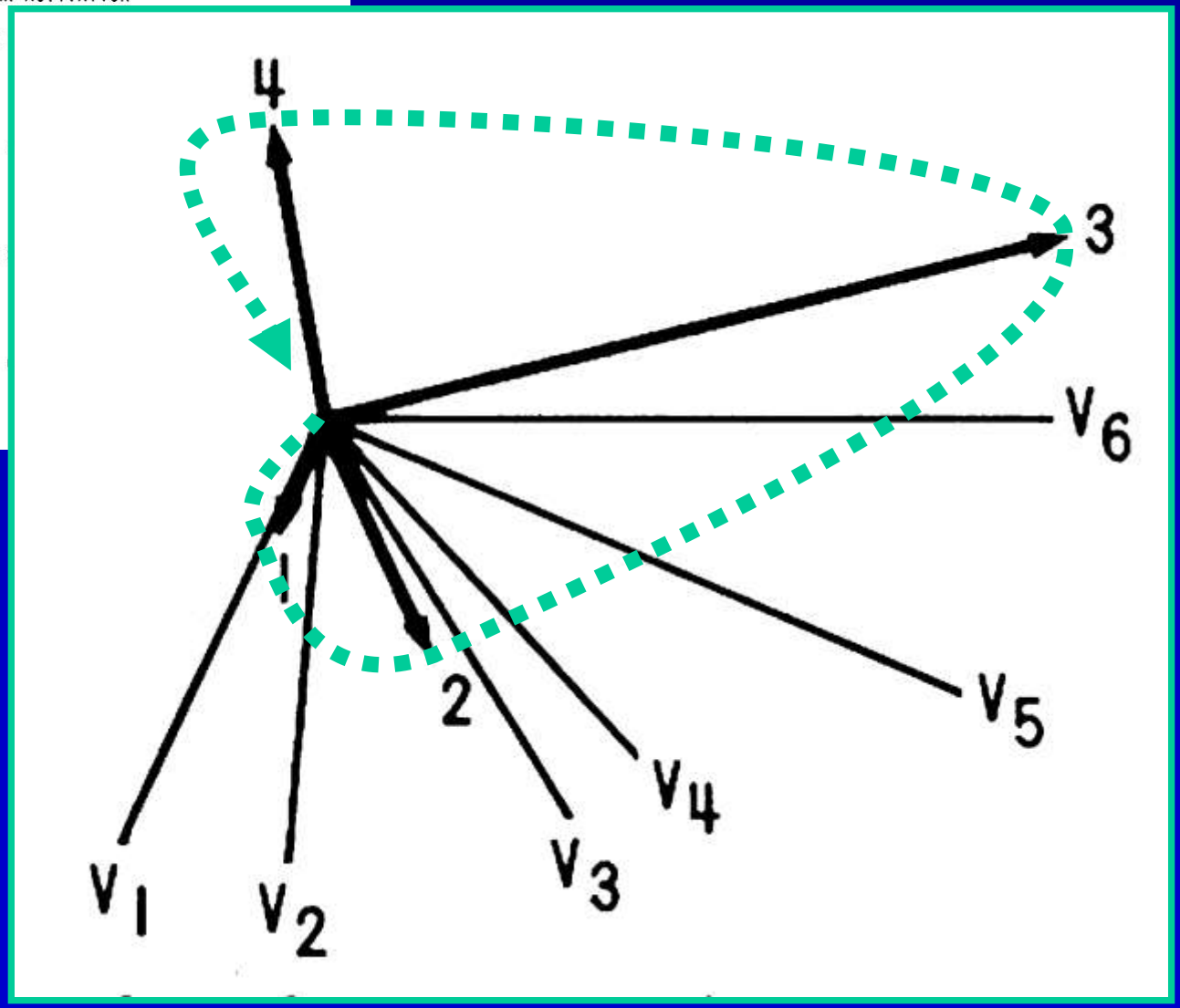
Sequence of Ventricular Activation



Friedman HH, 1971, p. 51.

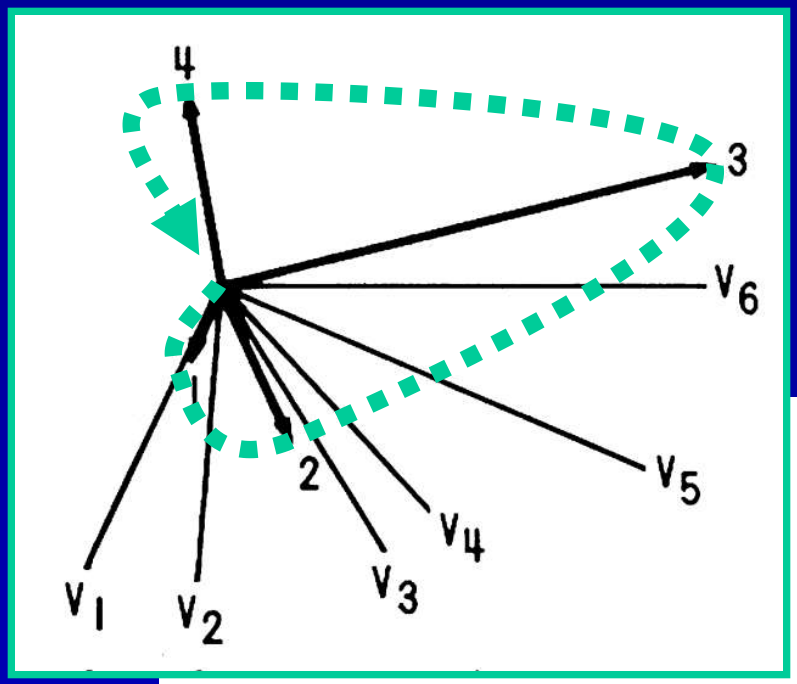
Sequence of Ventricular Activation

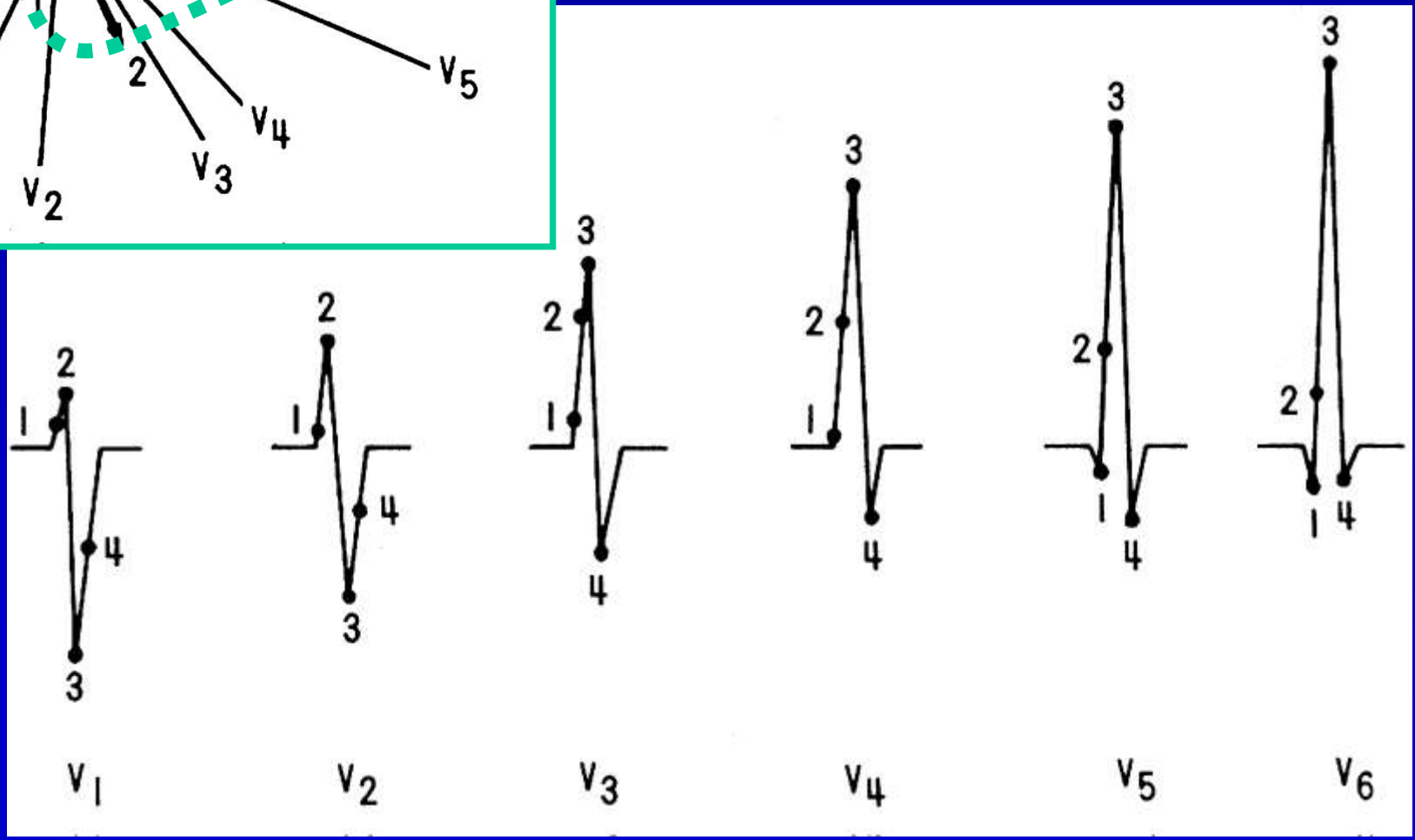




Friedman HH, 1971

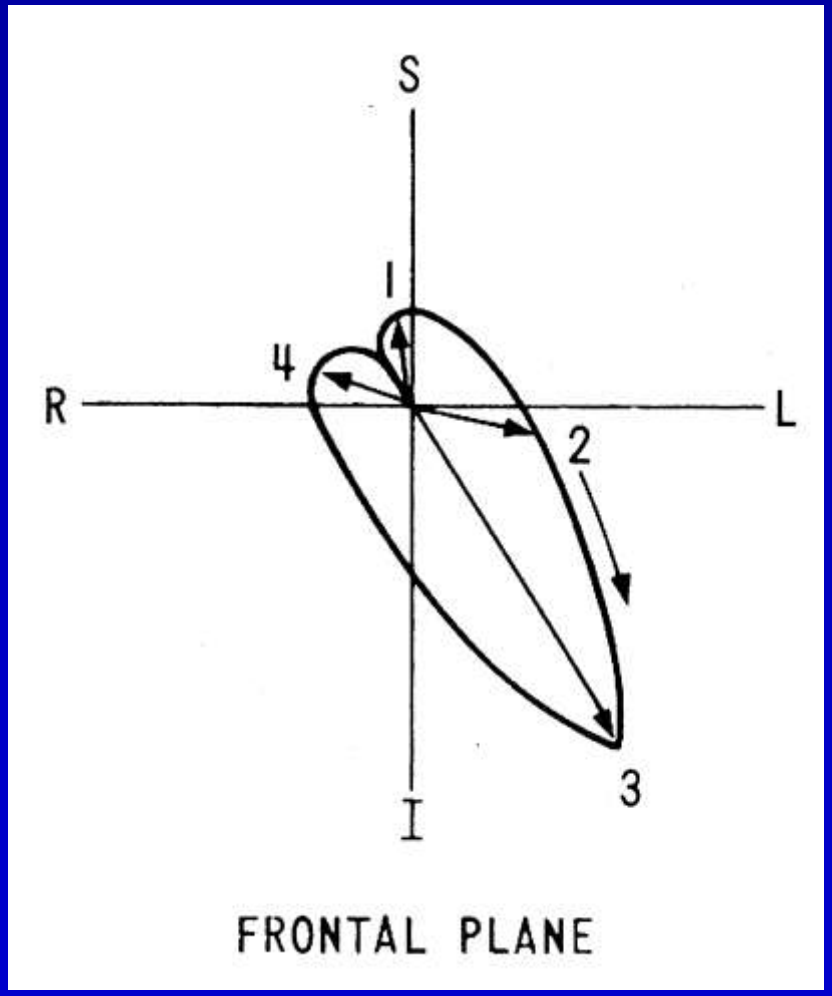
Sequence of Ventricular Activation

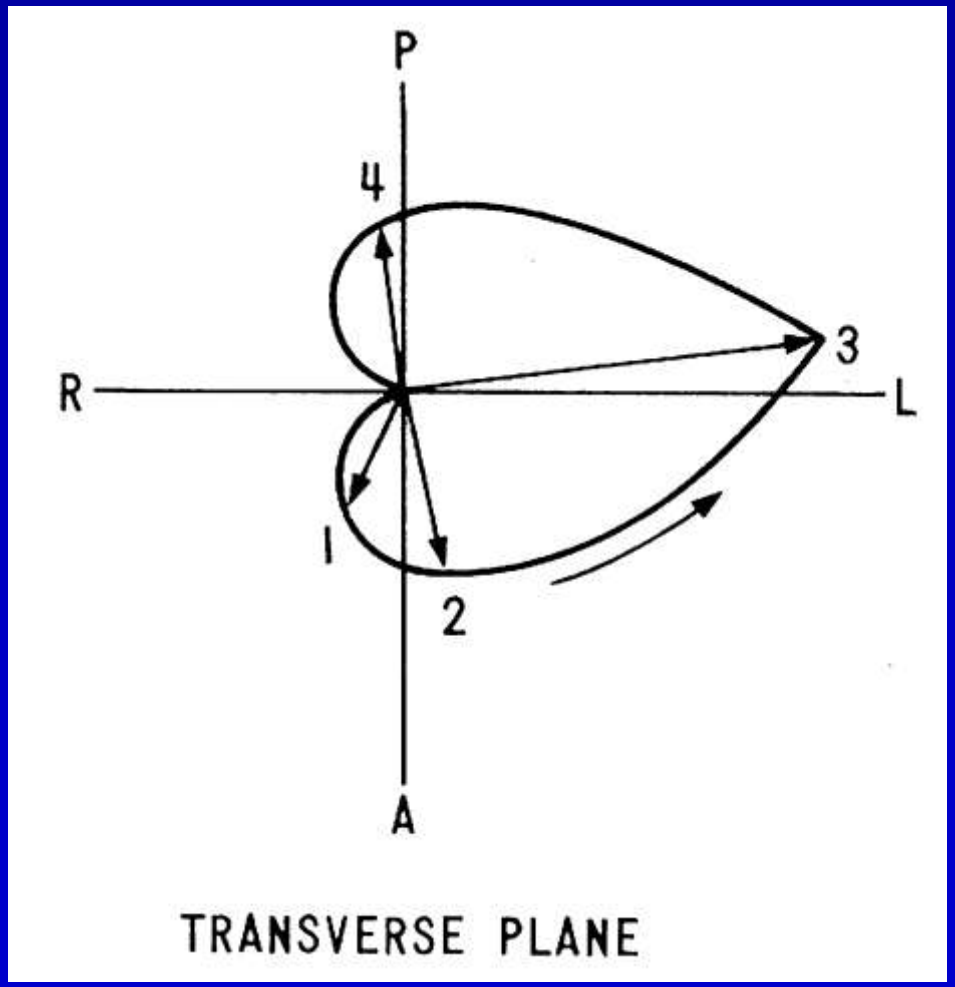




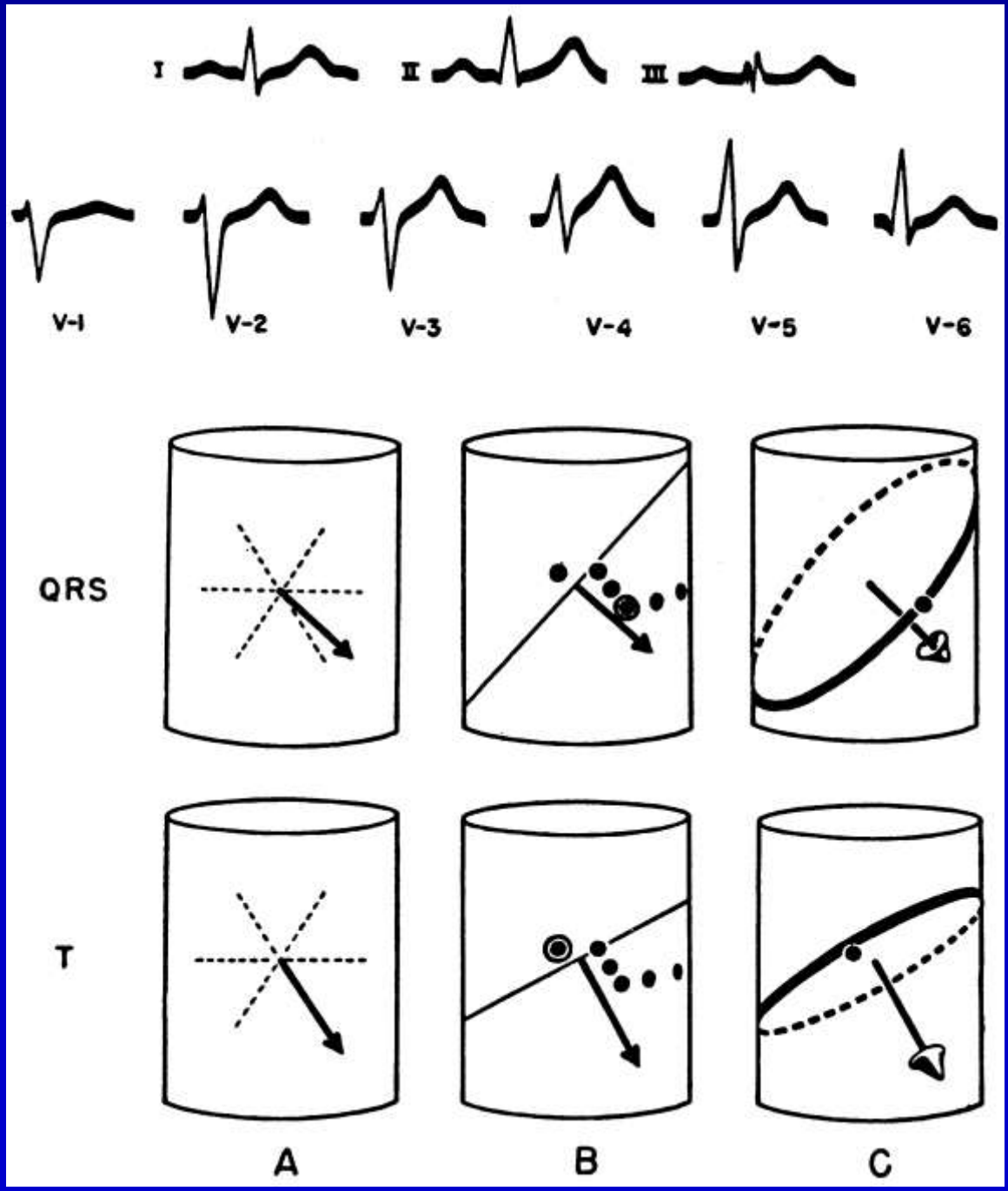
Friedman HH, 1971

Sequence of Activation



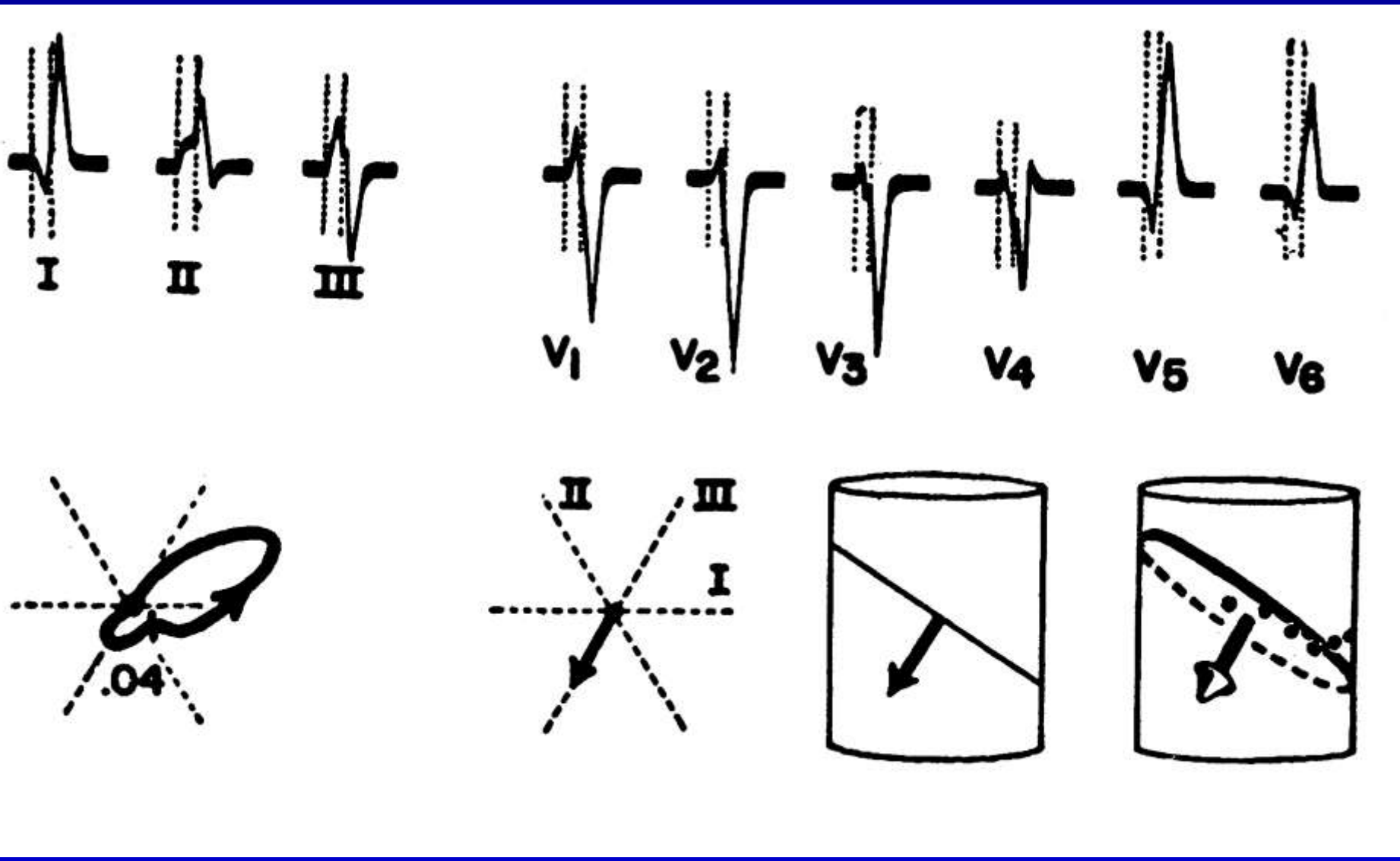


Deriving the Vector from the Scalar



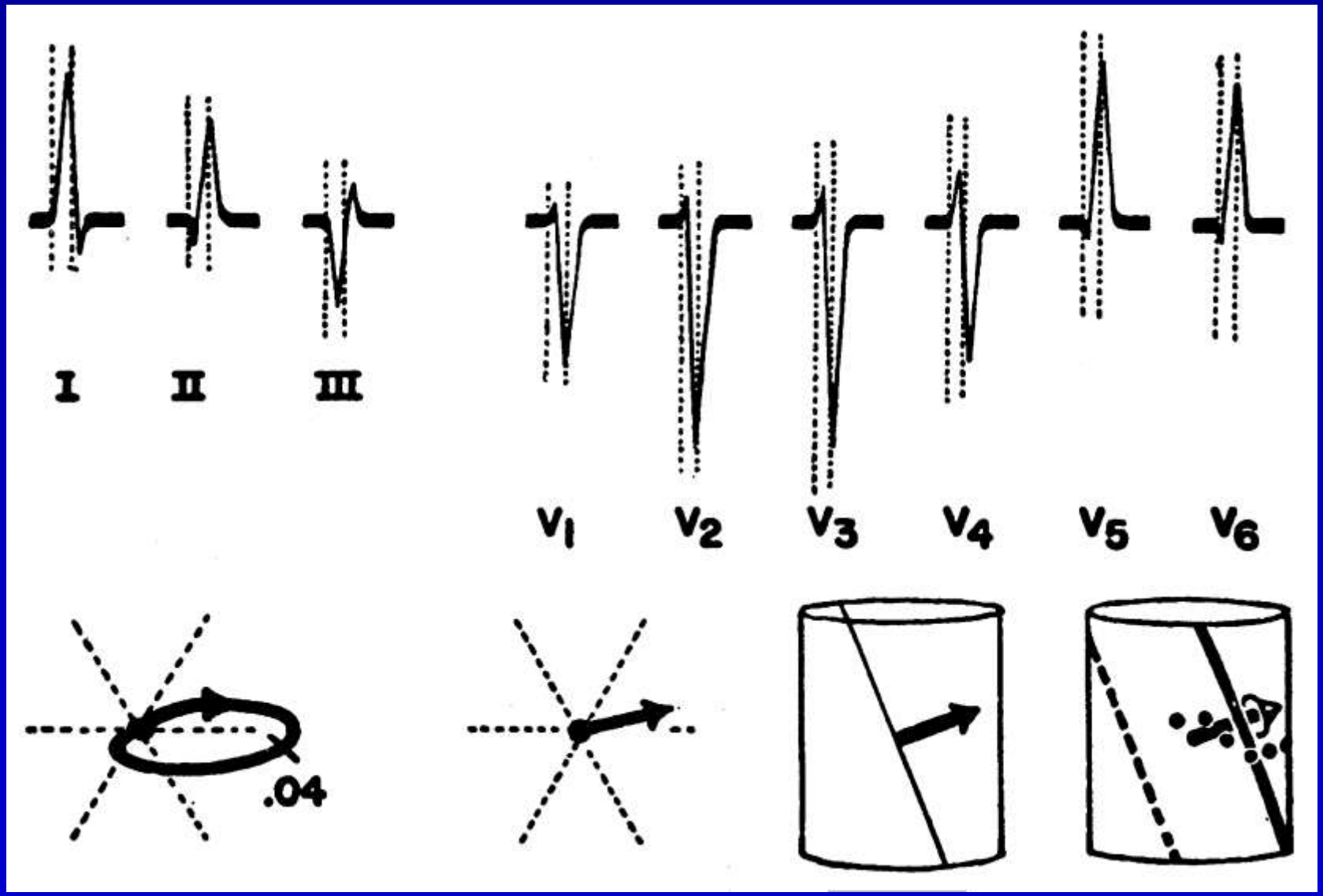
From Grant RP. Clinical Electrocardiography. McGraw-Hill. 1957

Deriving the Vector from the Scalar



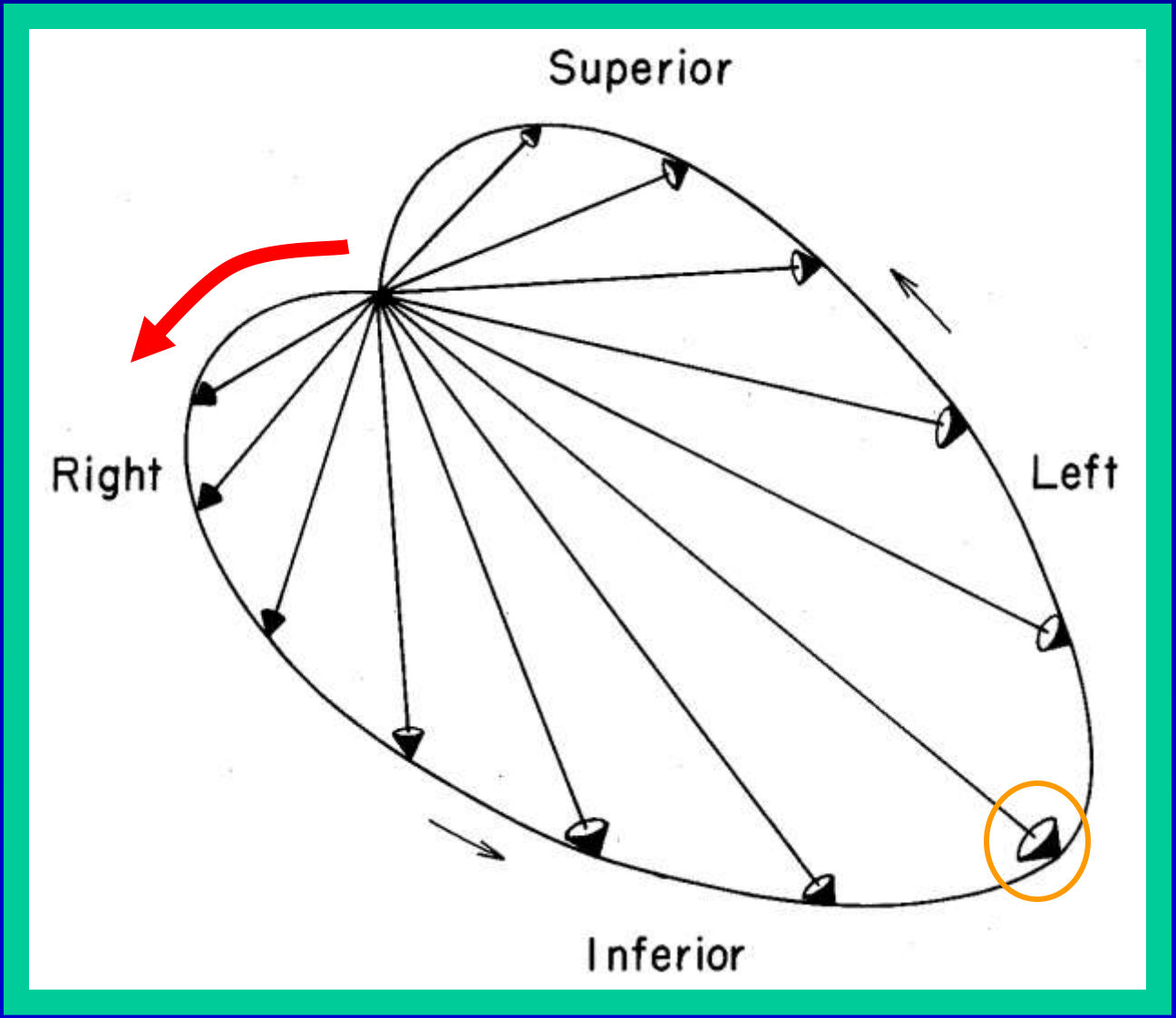
From Grant RP. Clinical Electrocardiography. McGraw-Hill. 1957

Deriving the Vector from the Scalar



From Grant RP. Clinical Electrocardiography. McGraw-Hill. 1957

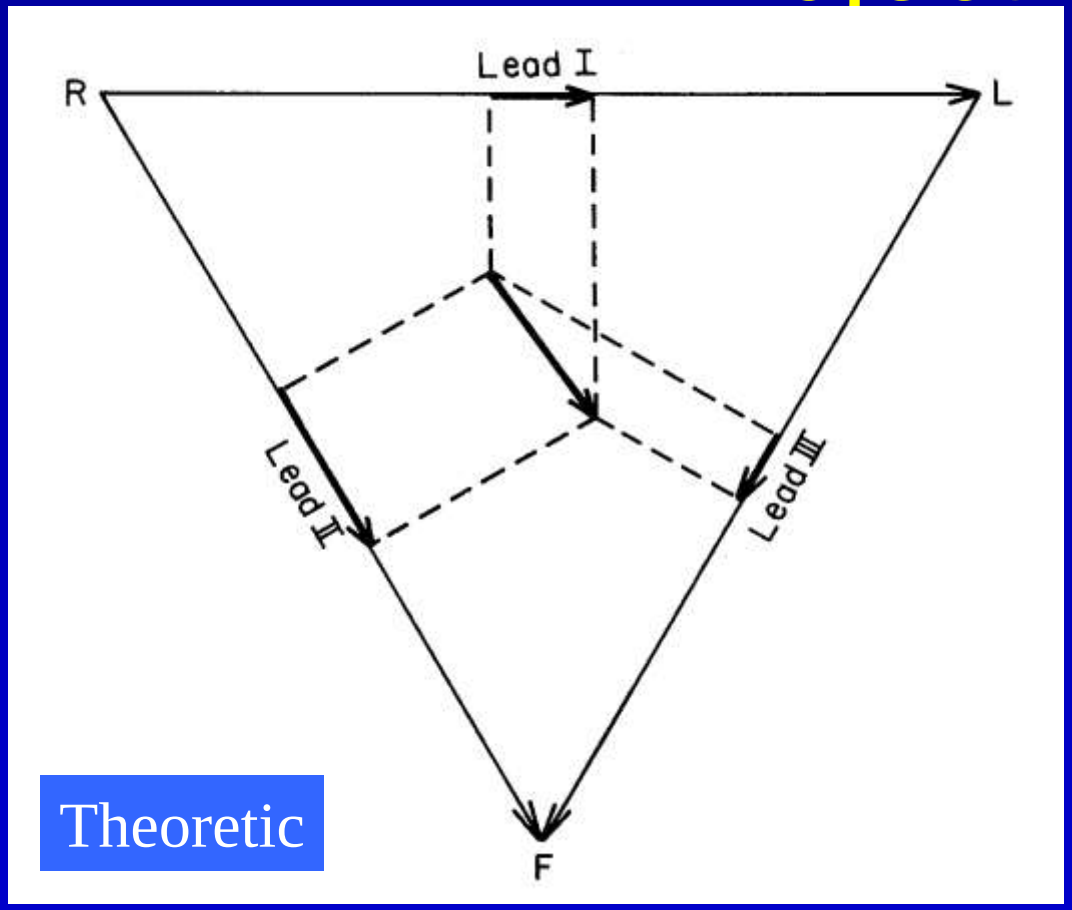
Normal Frontal Plane QRS Loop



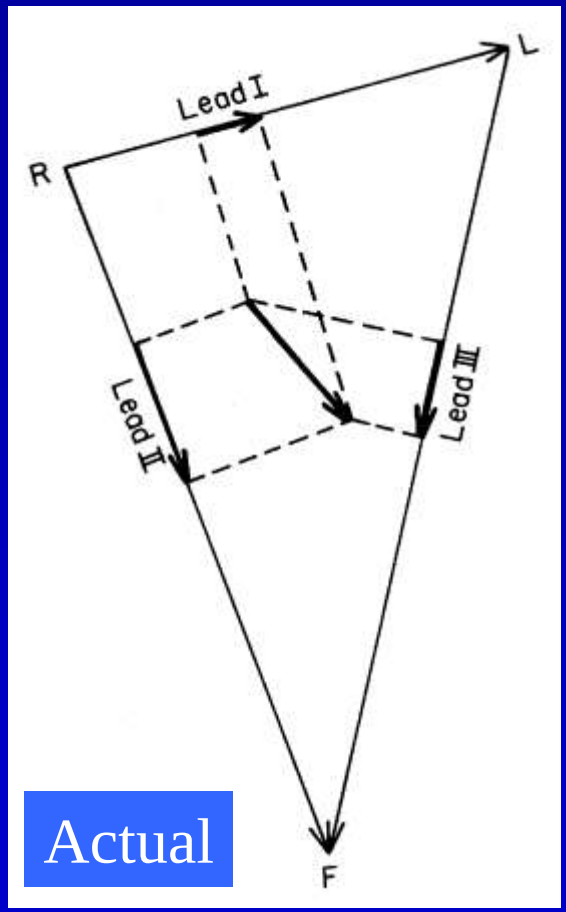
Mean maximal vector

Chou TC et al. Clinical Vectorcardiography, 2nd ed, 1974, p. 2.

Standard Limb Lead Projection



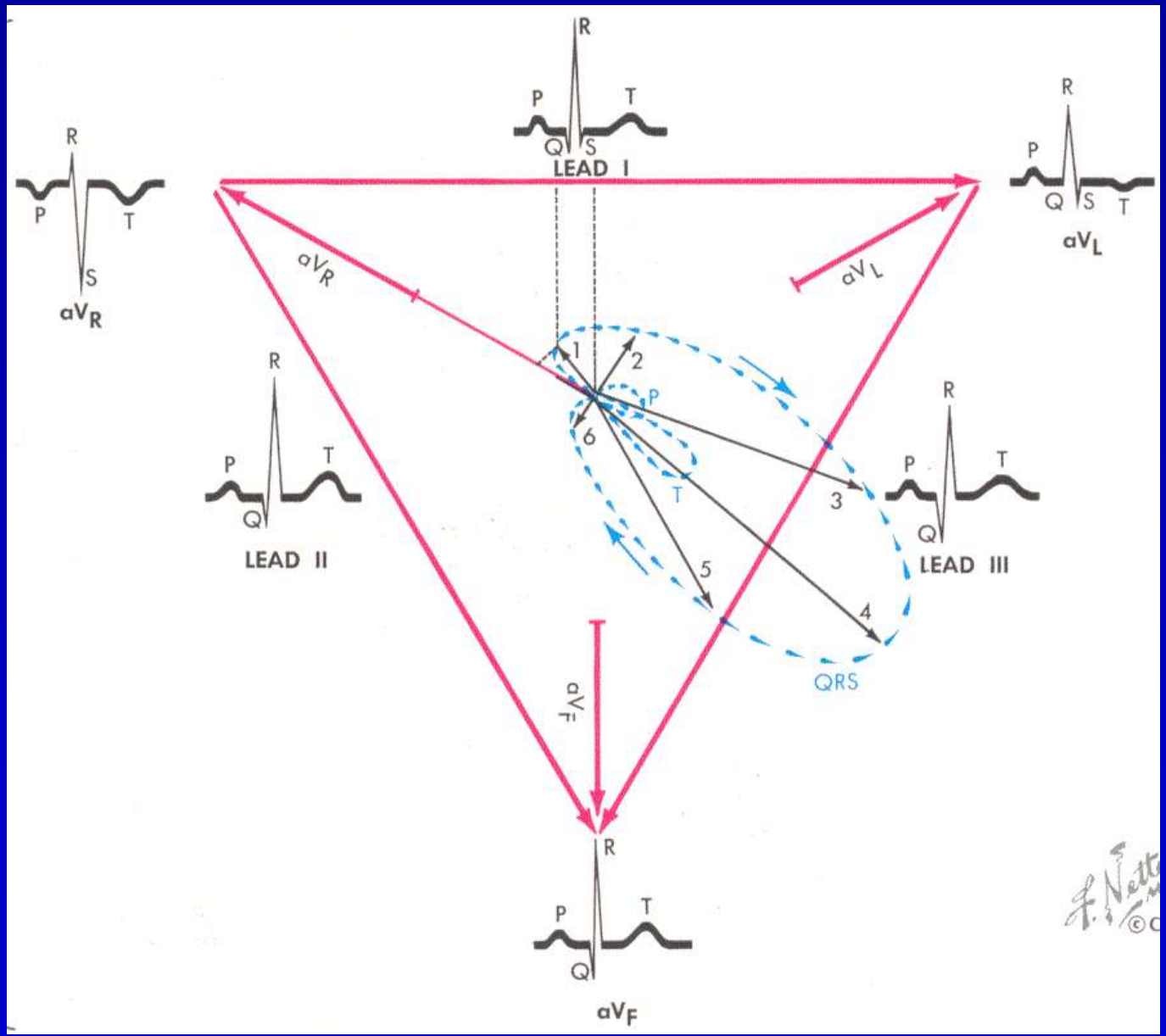
Theoretic



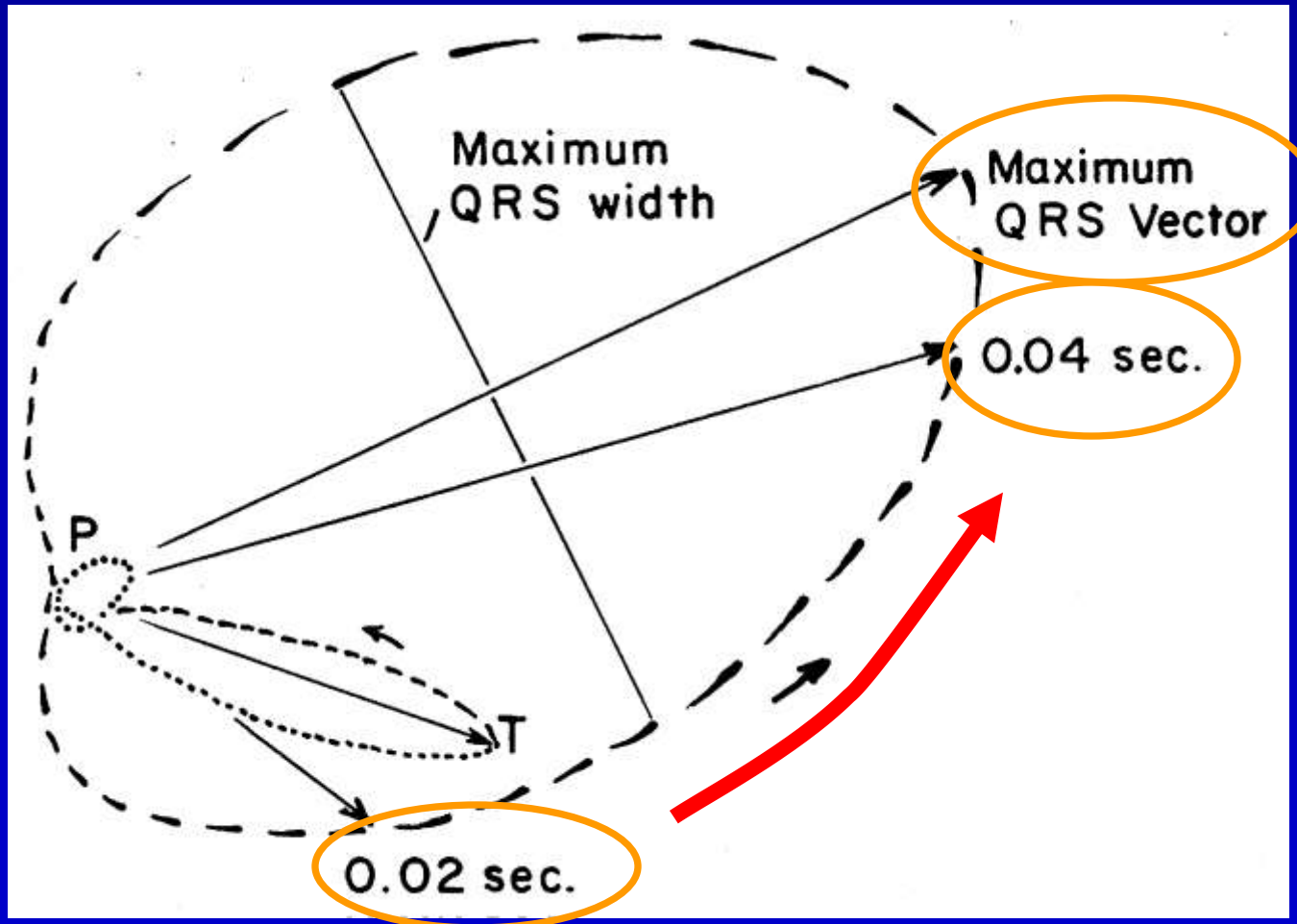
Actual

Chou TC et al. Clinical Vectorcardiography, 2nd ed, 1974, p. 3, 4.

Limb Lead ECG and VCG



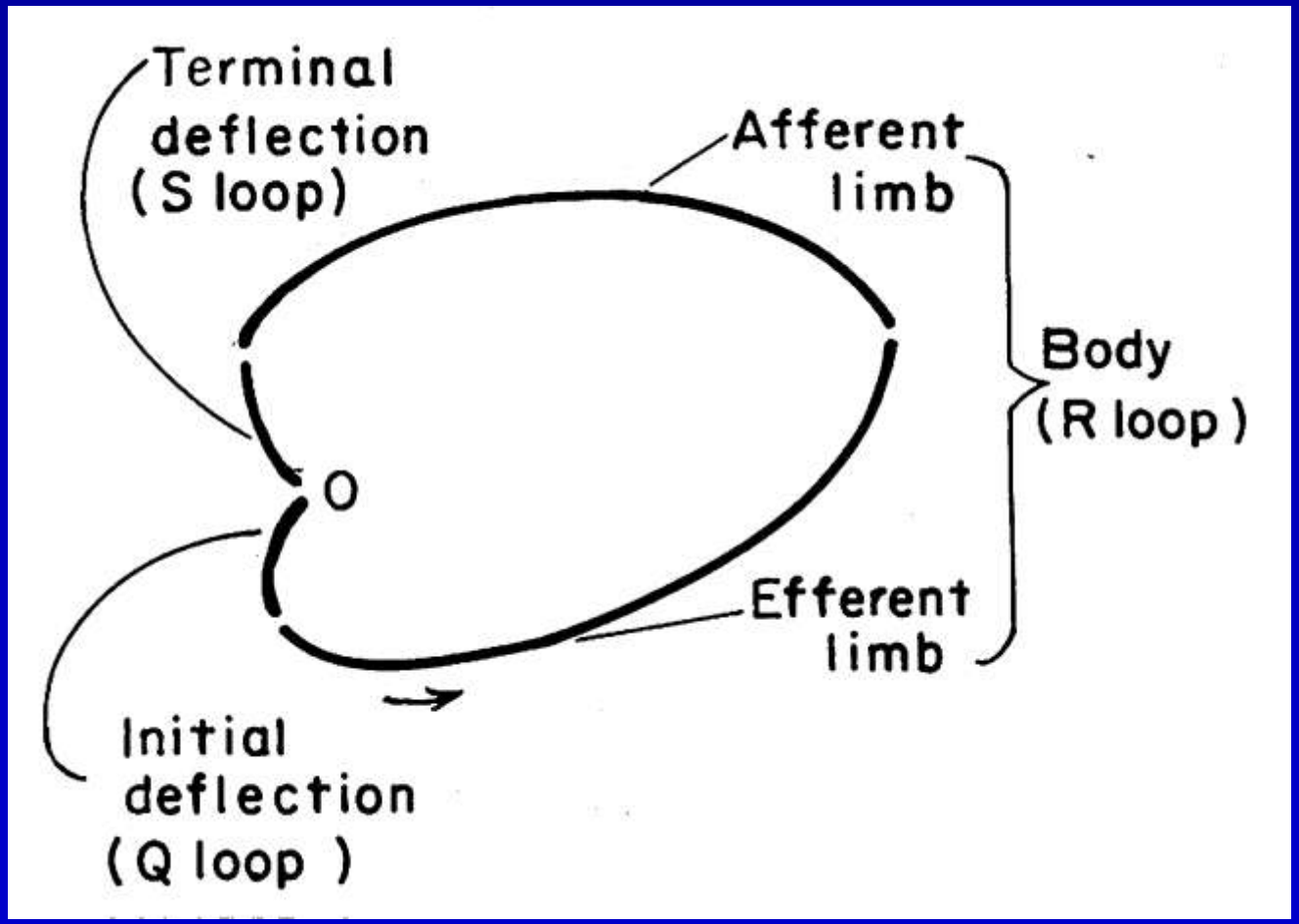
Vectorcardiographic Measurements



Transverse plane

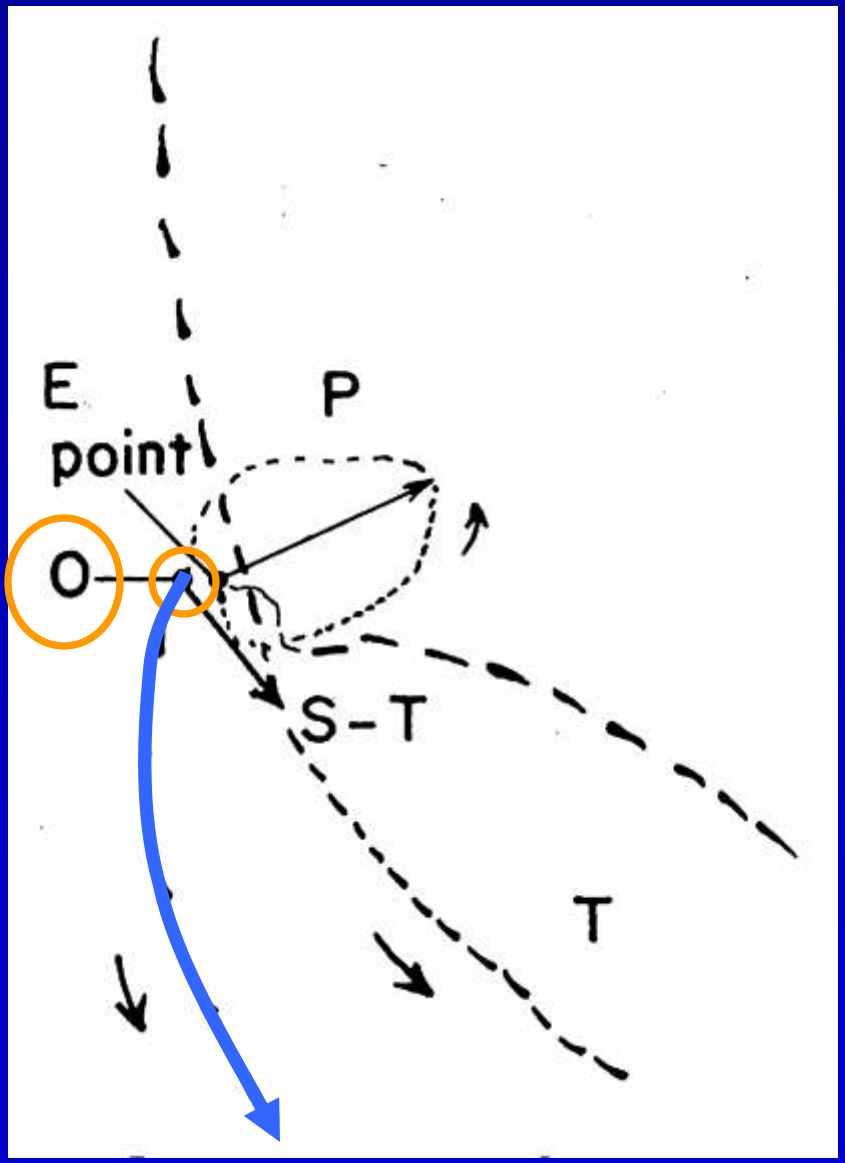
Chou TC et al. Clinical Vectorcardiography, 2nd ed, 1974, p. 42.

Vector Terminology



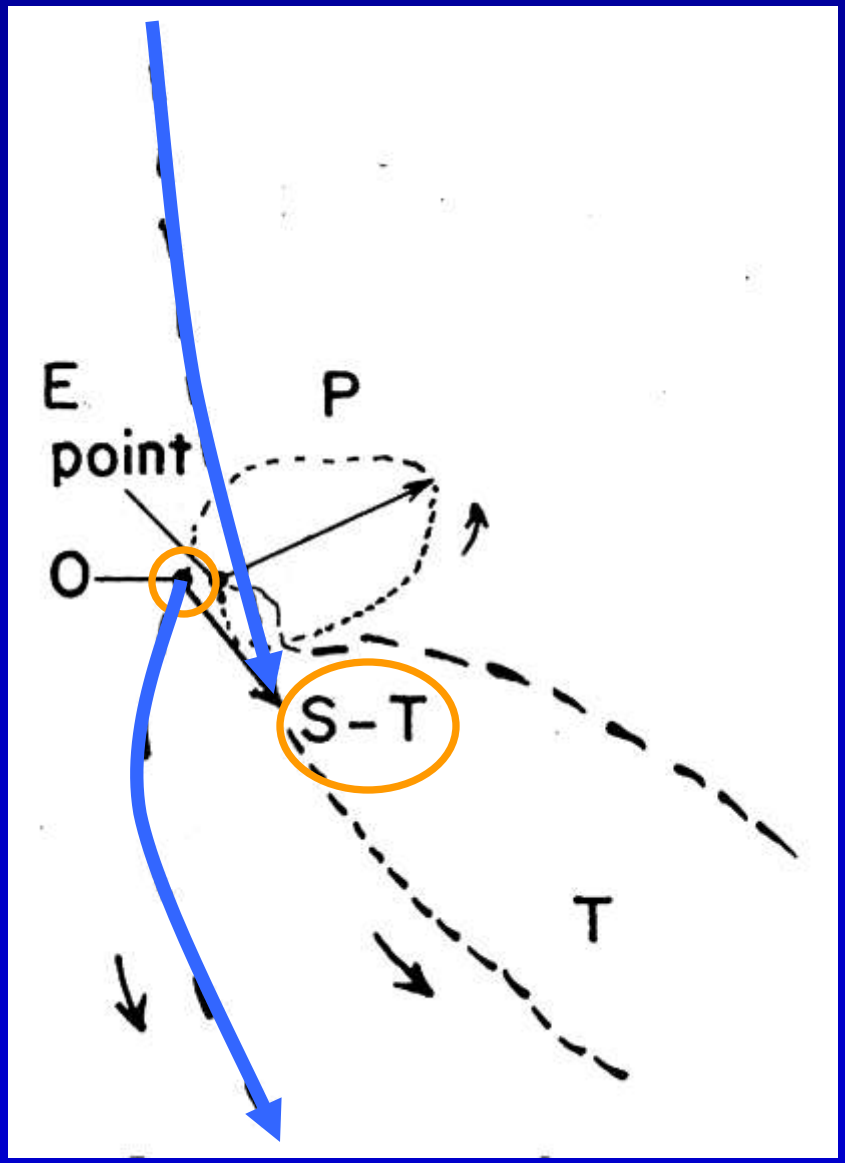
Chou TC et al. Clinical Vectorcardiography, 2nd ed, 1974, p. 42.

Transverse Plane



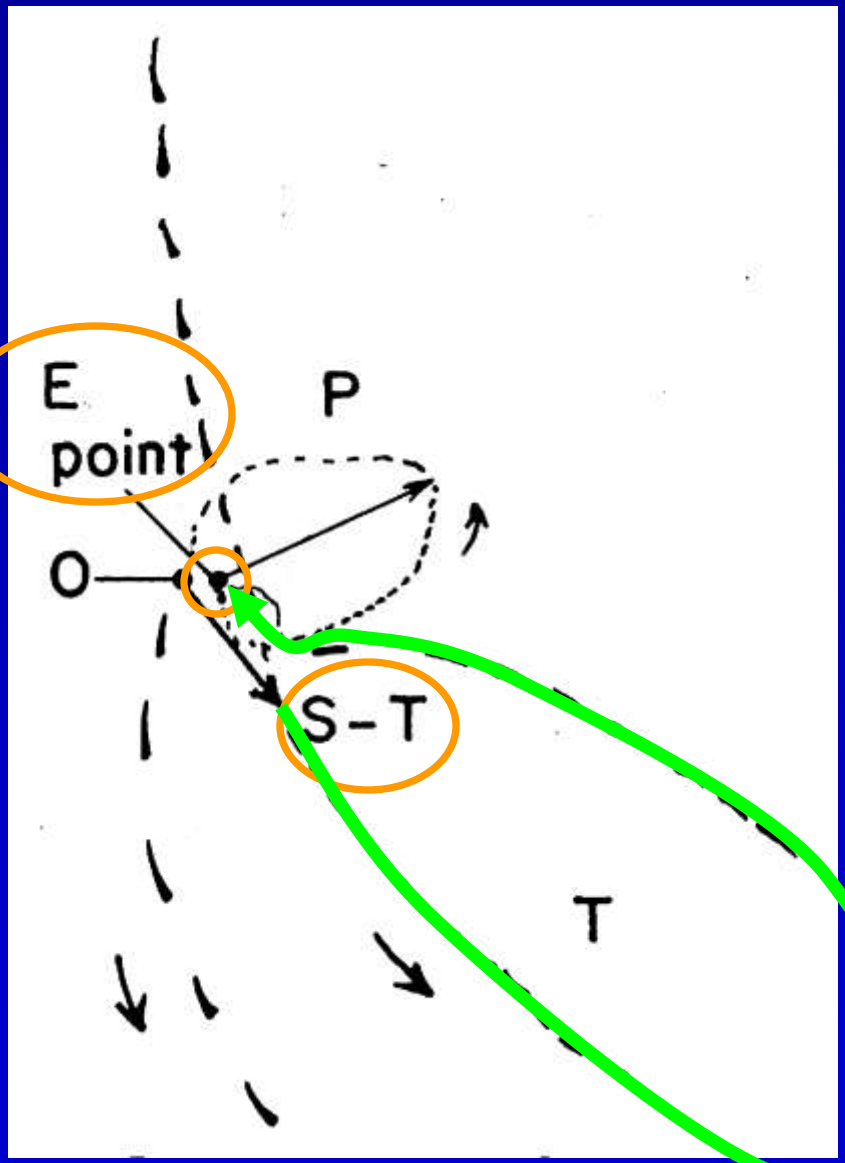
Chou TC et al. Clinical Vectorcardiography, 2nd ed, 1974, p. 42.

Transverse Plane

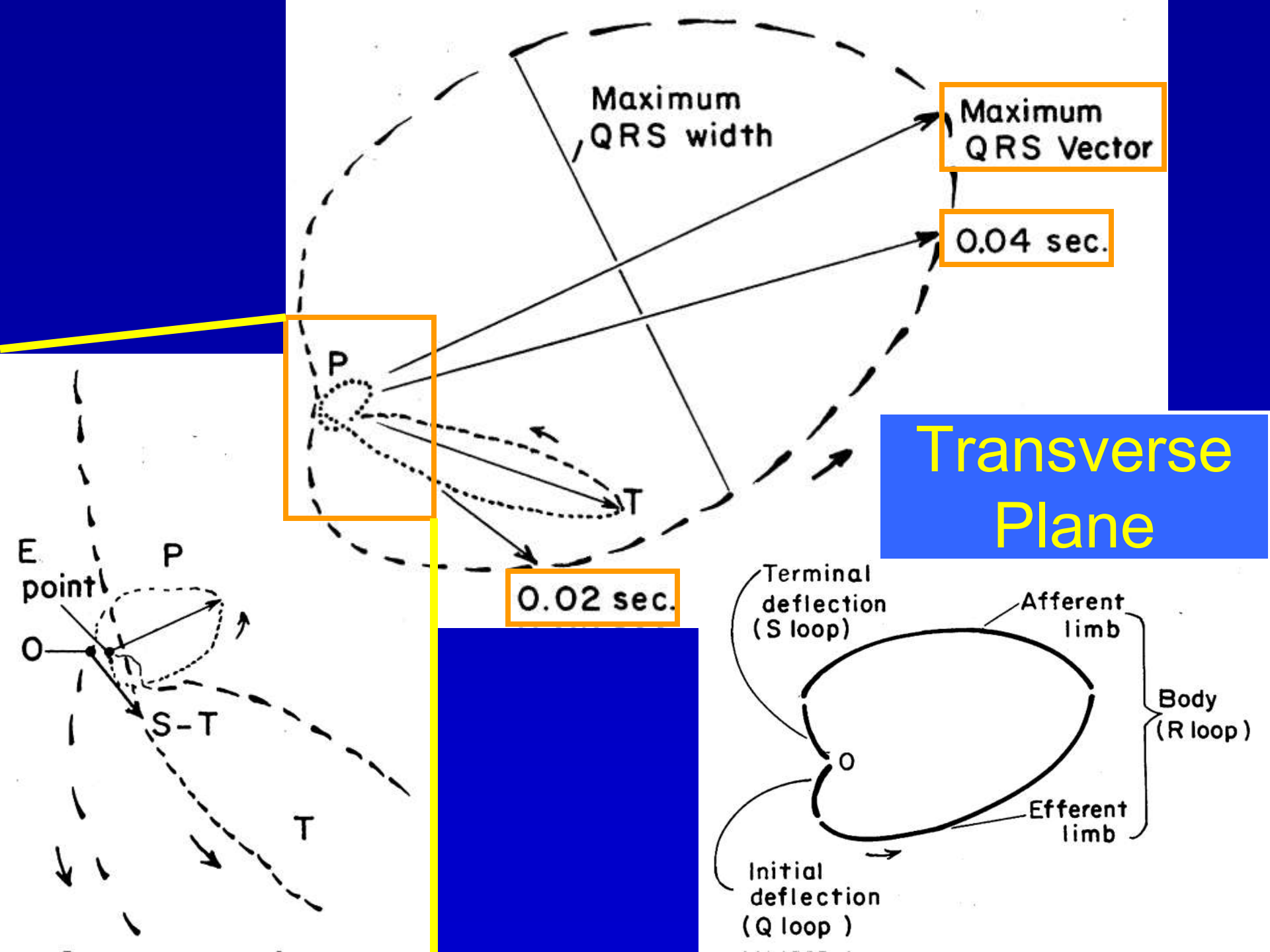


- The end of the QRS is not precisely at the same point as the beginning, so there is a normal ST segment, especially in the transverse plane.

Transverse Plane



- The end of the T loop is back to the E point

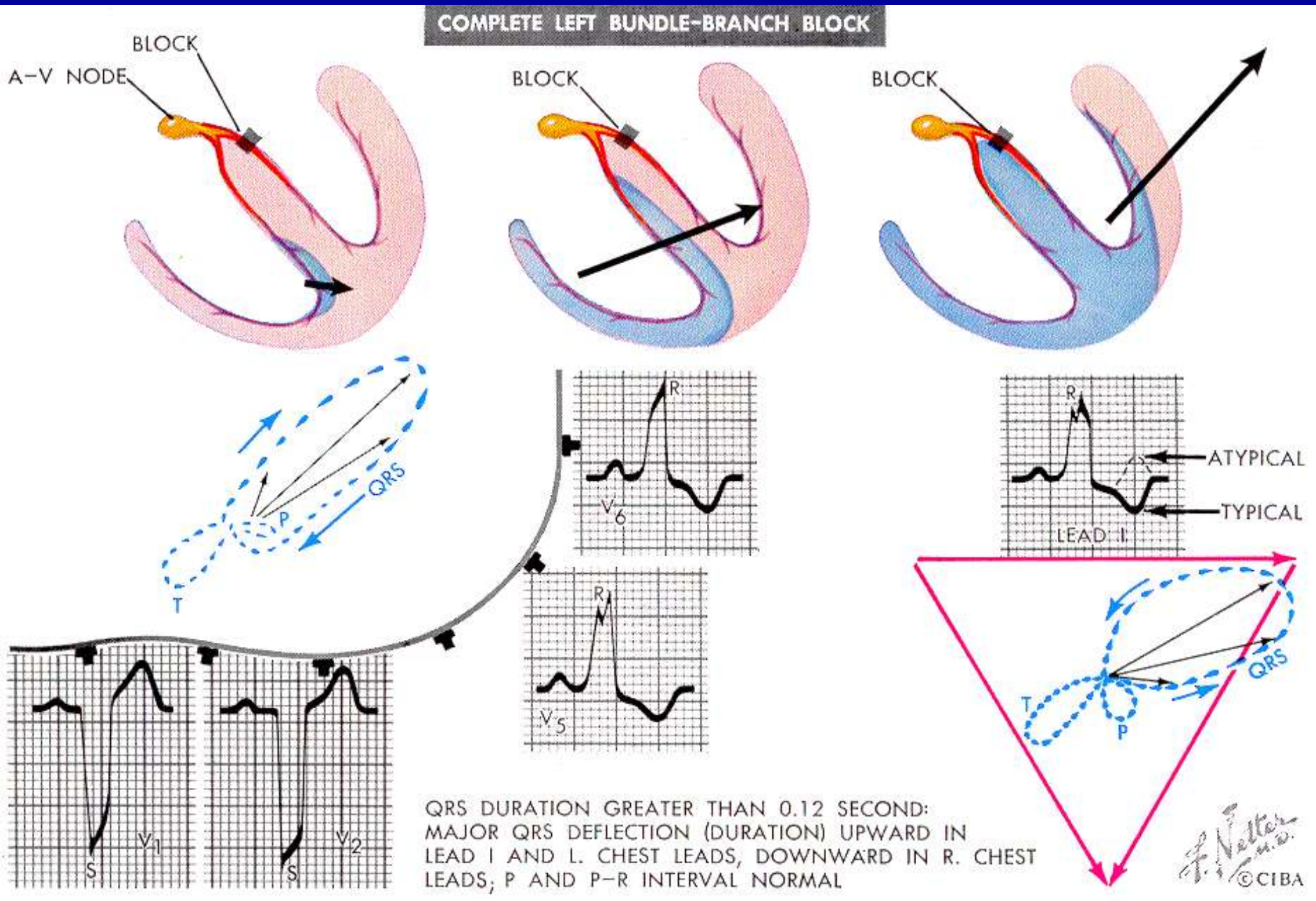


Transverse Plane

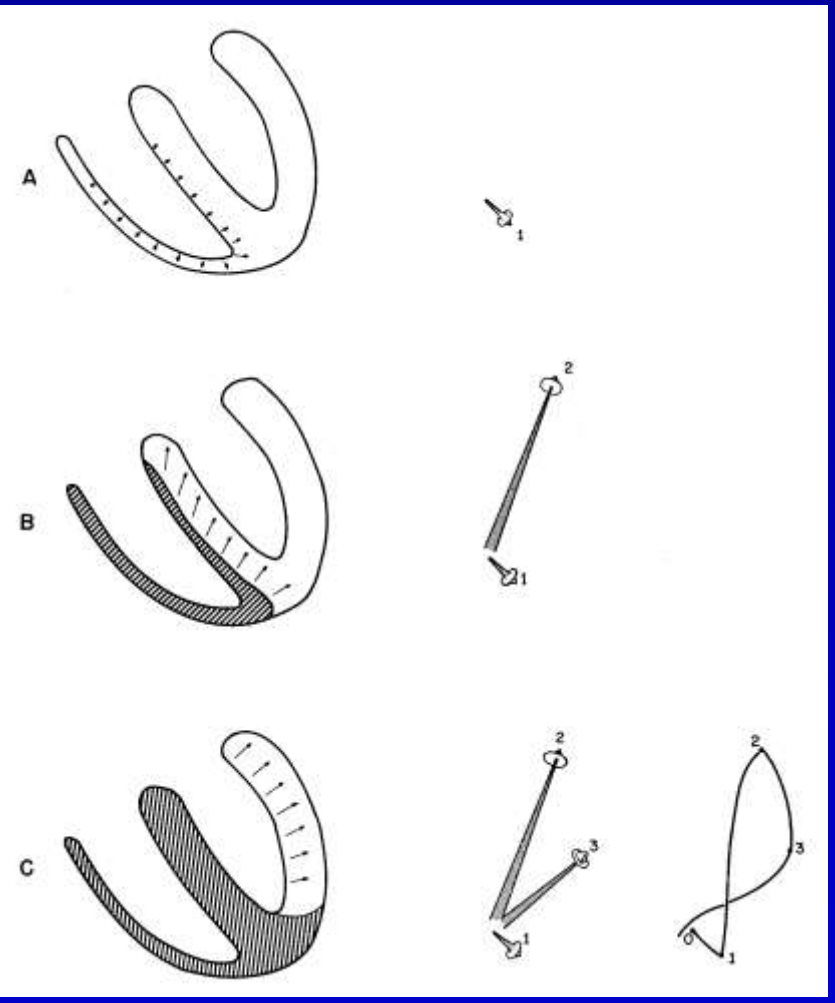
Left Bundle Branch Block

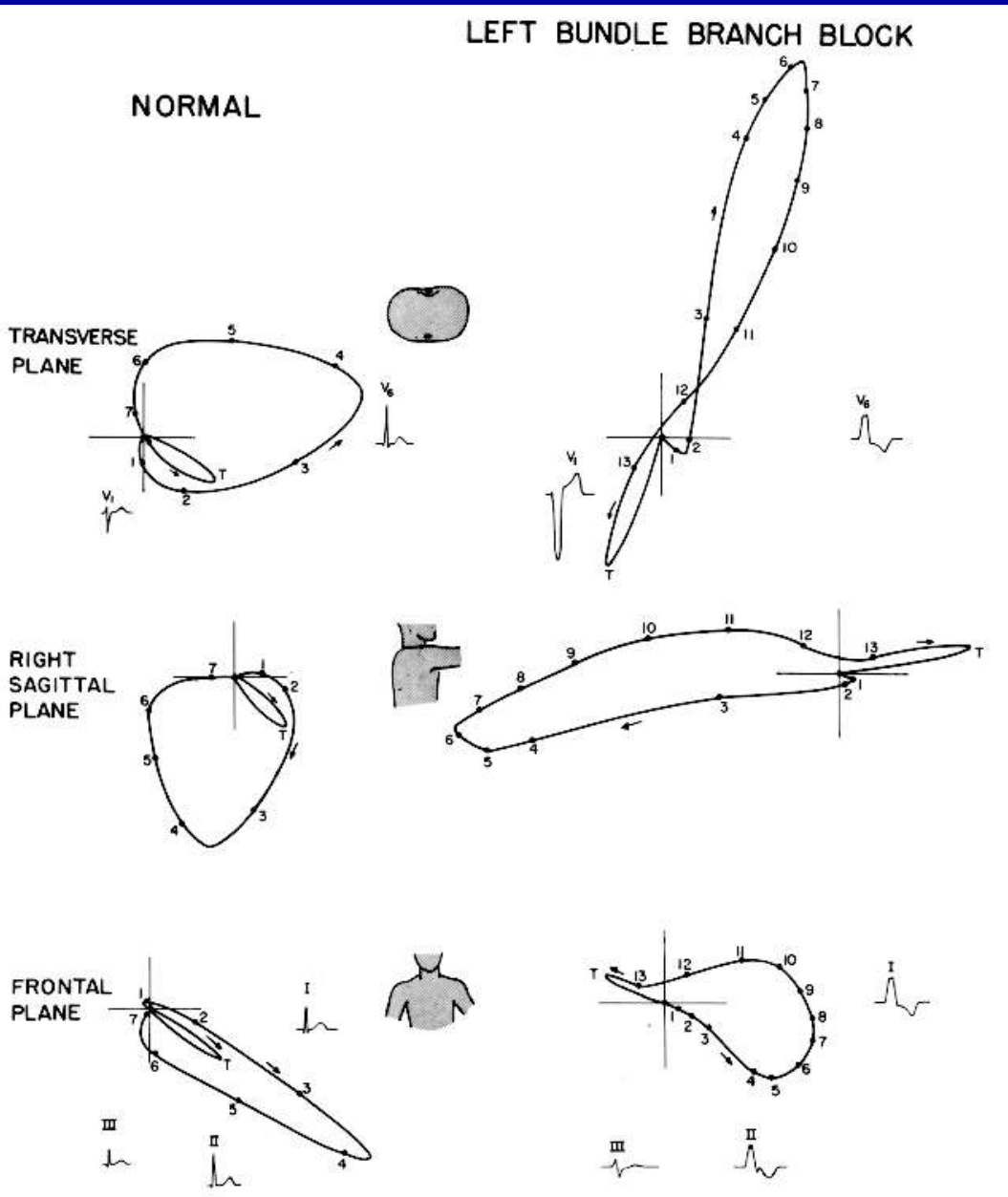
- Criteria
- Axis
- Atypical features
- Incomplete
- Corrected transposition
- LVH
- RVH
- Infarction/ primary T wave

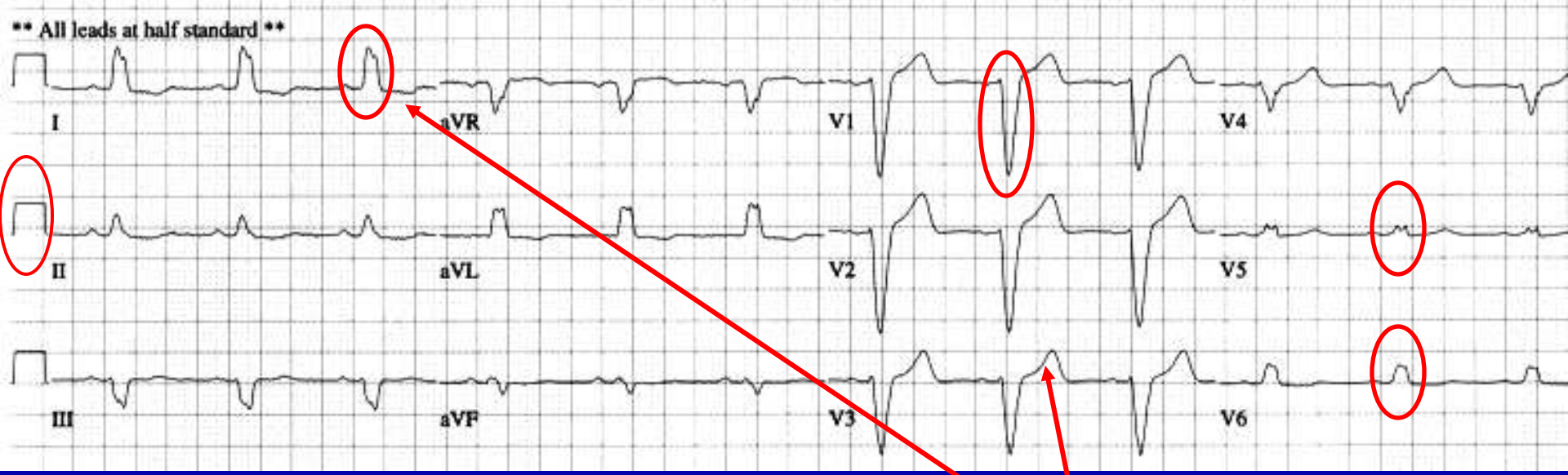
Left Bundle Branch Block



Left Bundle Branch Block







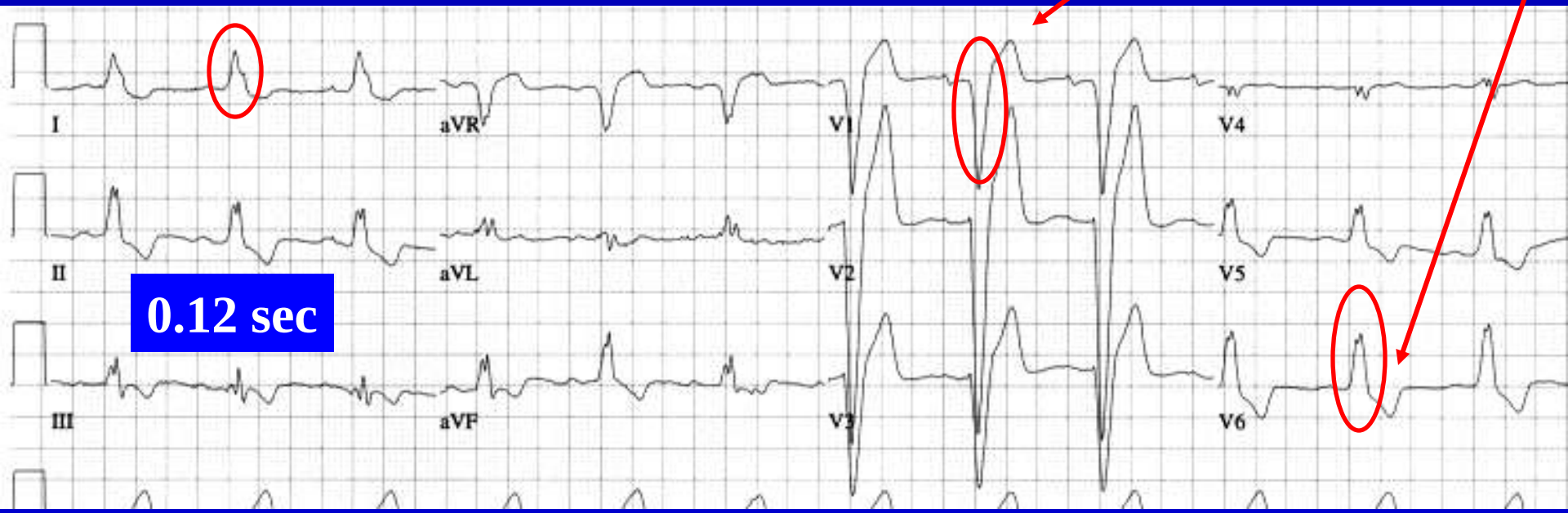
Discordant T wave

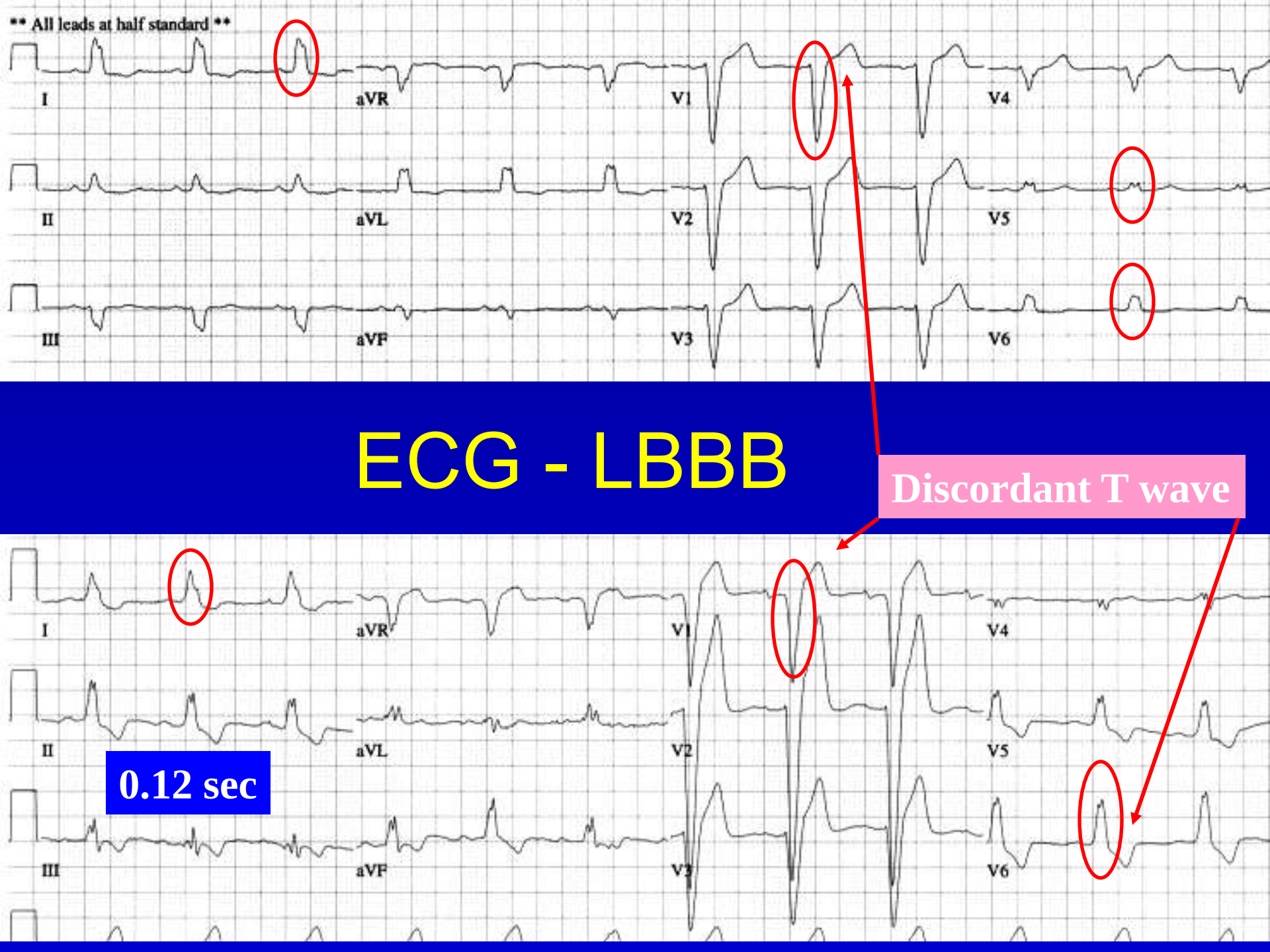
ECG - LBBB

- QRS duration >= 0.12 sec
- Broad (usually notched or slurred) R in aVL, V5, and V6
- Absent Q waves in I, V5, and V6
- Delay in R peak time in V5 and V6 >0.06 sec
- Broad Deep S wave or QS complex in V1
- Repolarization: ST segment and T wave directed opposite to mean QRS

ECG - LBBB

- QRS duration >= 0.12 sec
- Broad (usually notched) R in aVL, V5, and V6
- Absent Q waves in I, V5, and V6
- Delay in R peak time in V5 and V6
- Broad Deep S wave or QS complex in V1
- Repolarization: ST segment and T wave directed opposite to mean QRS




** All leads at half standard **
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
ECG - LBBB
Discordant T wave
0.12 sec

Left Bundle Branch Block

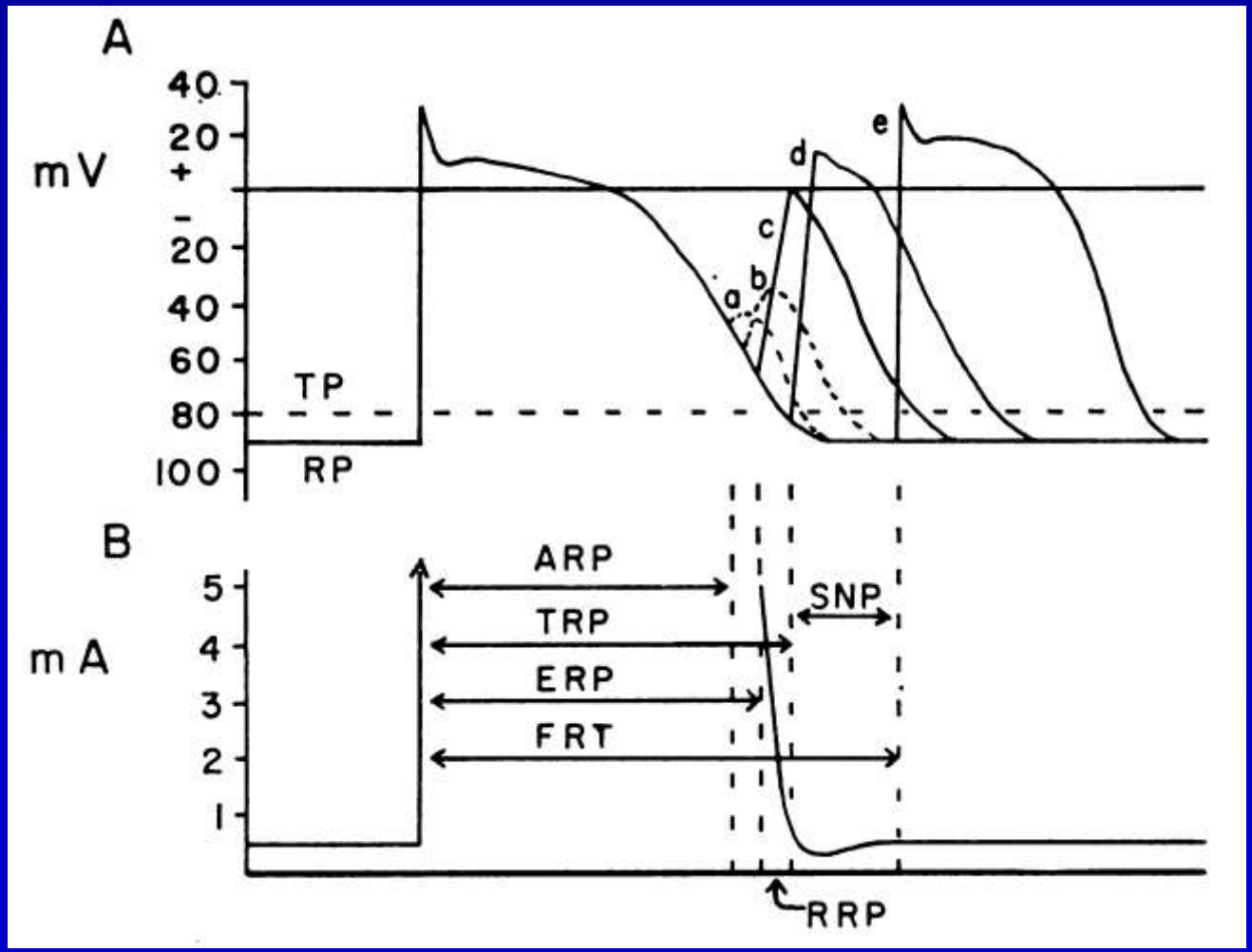
- Axis may shift leftward or not
- Atypical features, RS in V5-6, Q in V1-2
- Incomplete, QRS 0.11 and R peak time 0.06 and no Q in V5 and V6 and I (excessive >0.15 or 0.18)
- Corrected transposition – absent rightward initial force, otherwise normal (due to ventricular inversion)
- LVH: S in V2 plus R in V5>45mm; QRS 0.16; probably anyway
- RVH: probably not
- Infarction/ primary T wave
  - Qs in I and aVL, V5 or V6; notch in S in V3-5; inf Q >30ms and T inversion – all these probably aren't really any good, also for paced beats
  - Concordant ST elevation is diagnostic, especially lateral

Left Bundle Branch Block

- Intermittent:
  - Acceleration dependent – phase 3 block, voltage or time-dependent block, usual refractoriness
  - Deceleration dependent – phase 4 block, more controversial, spontaneous depolarization
  - Non rate-related
  - Ischemic, autonomic tone variations
- Complete may be relative, and can occasionally be made worse by pacing or premature beats

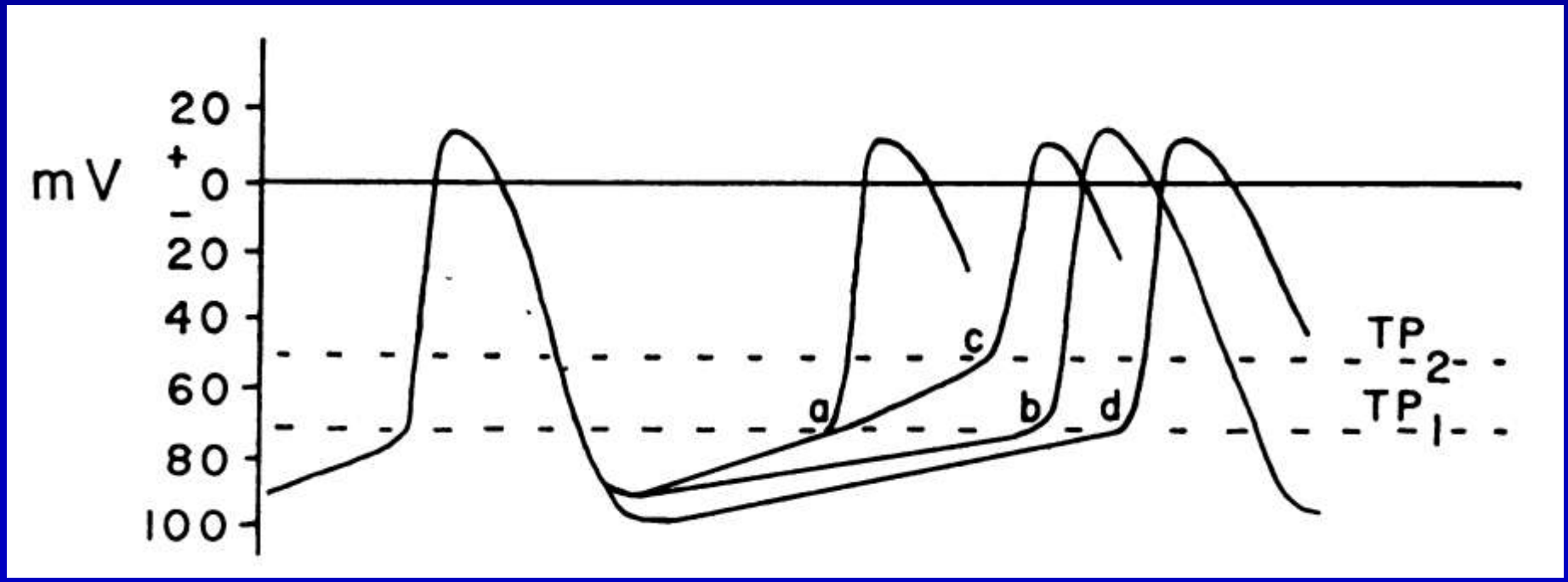
Left Bundle Branch Block

- Intermittent
  - Phase 3
  - Phase 4



Bellet S. 3rd ed. 1971, p. 45.

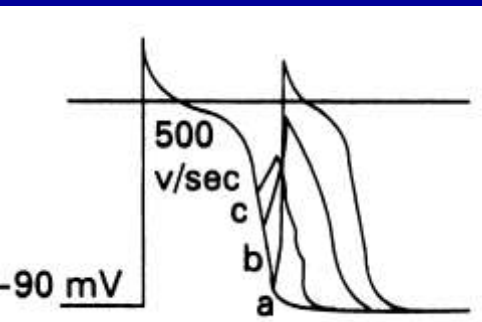
Left Bundle Branch Block

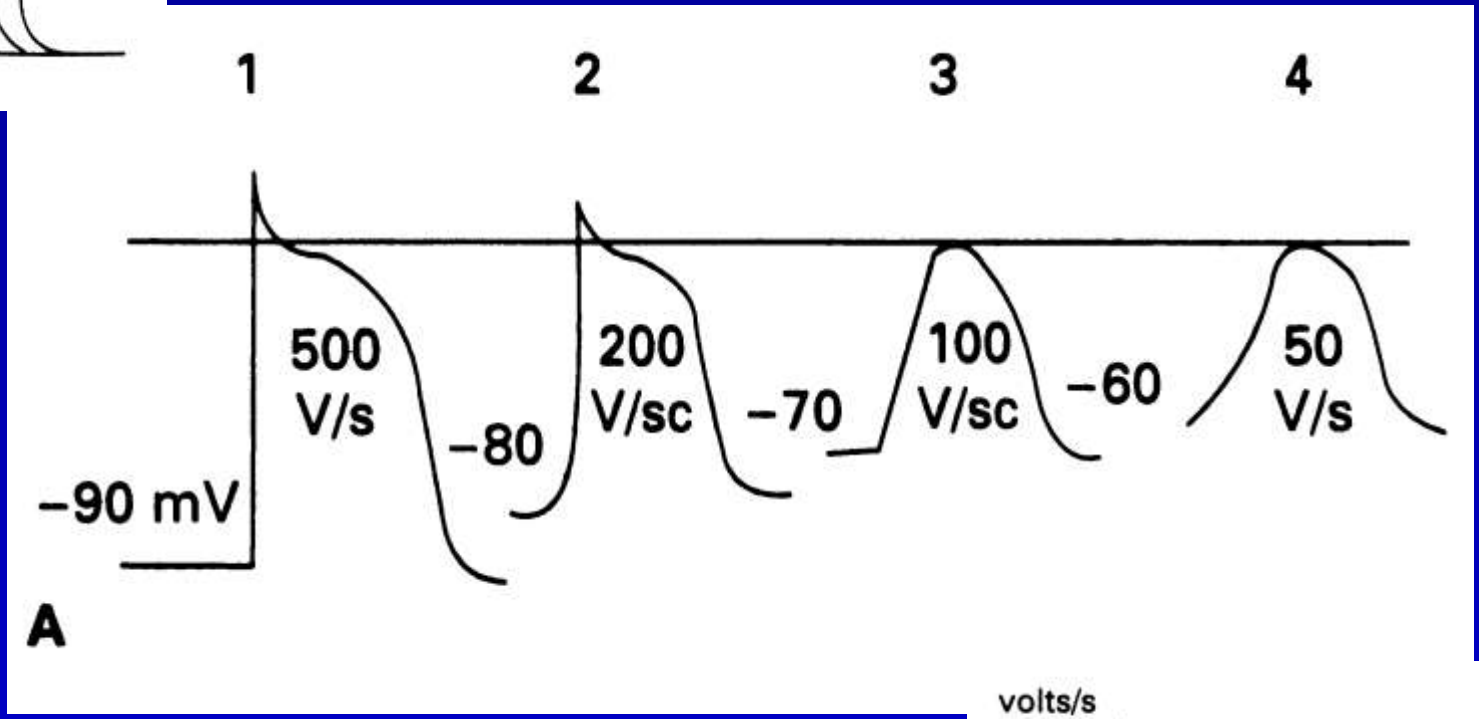


- Automaticity depends on threshold, on maximal polarized potential and on phase 4 slope
- A partially depolarized cell may not propagate an action potential normally

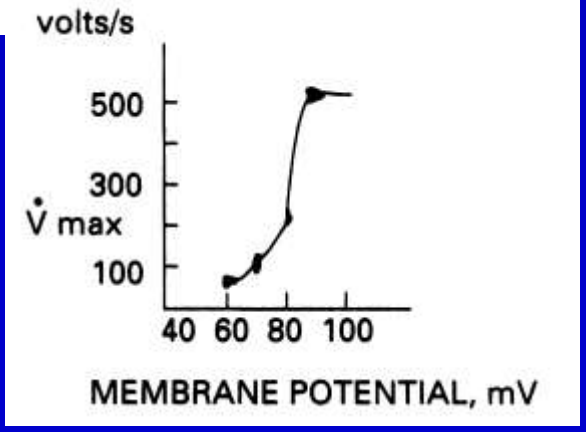
Bellet S. 3rd ed. 1971, p. 45.

Left Bundle Branch Block



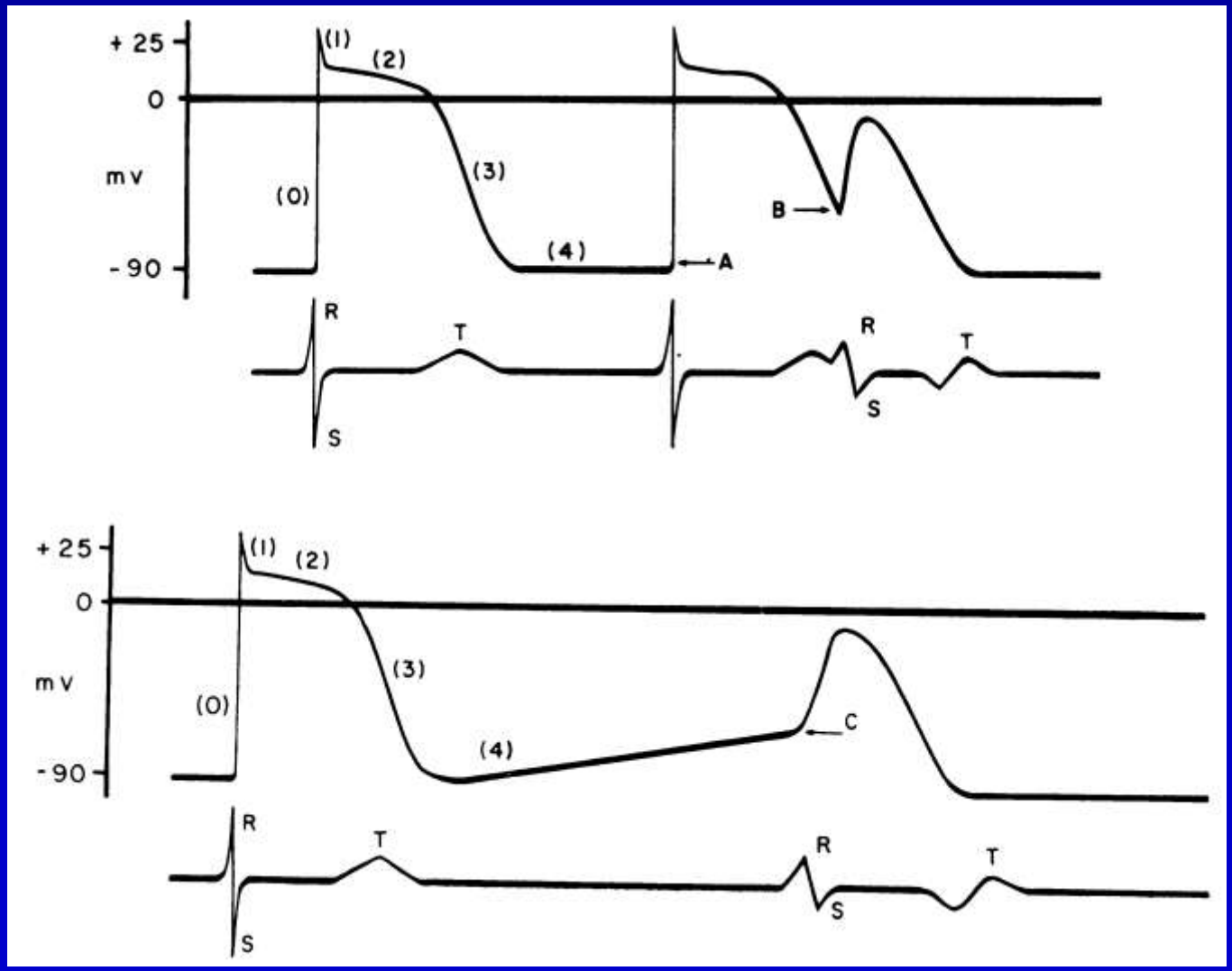


- Automaticity depends on threshold, on maximal polarized potential and on phase 4 slope
- A partially depolarized cell may not propagate an action potential normally



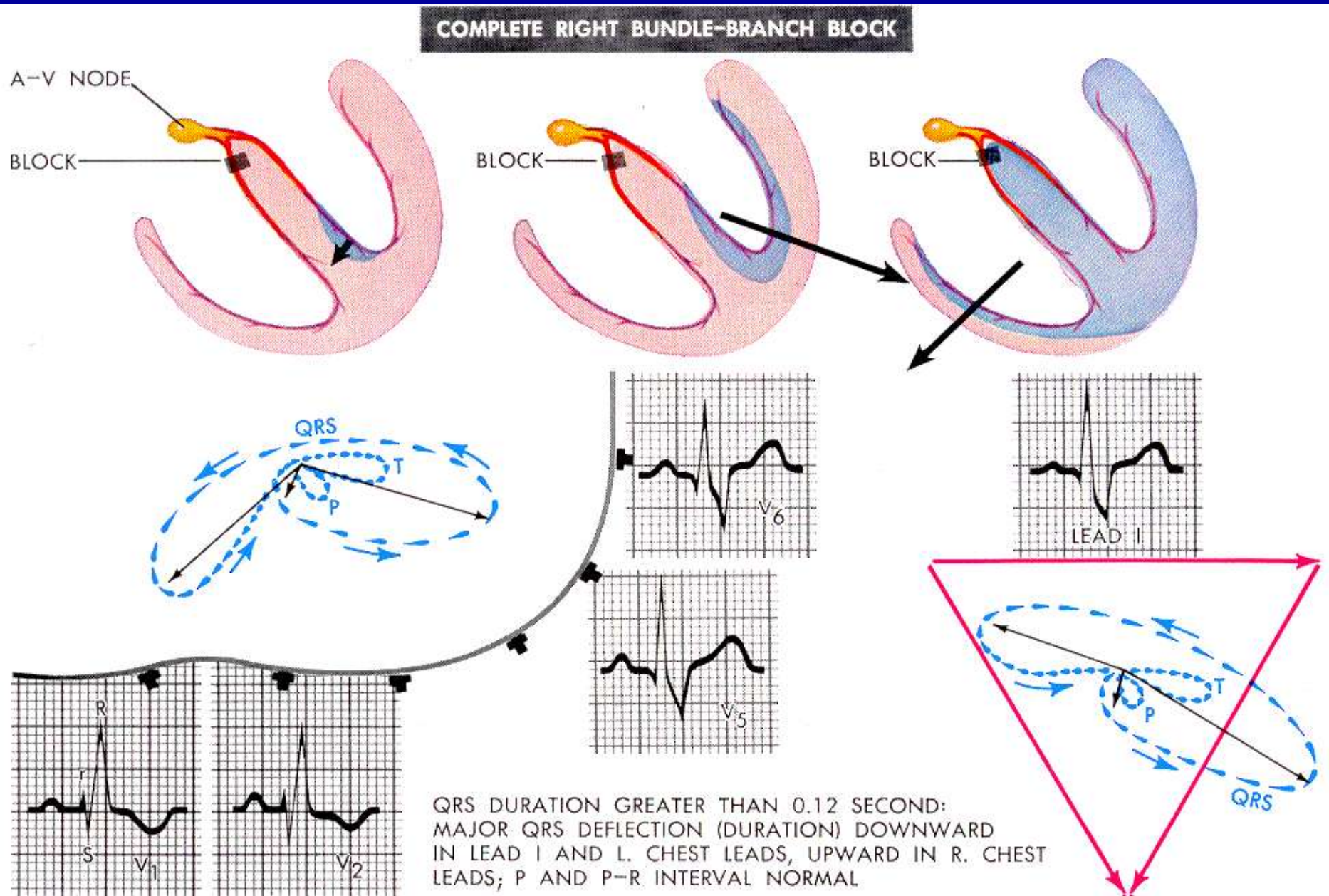
Left Bundle Branch Block

- Intermittent
  - Phase 3
  - Phase 4

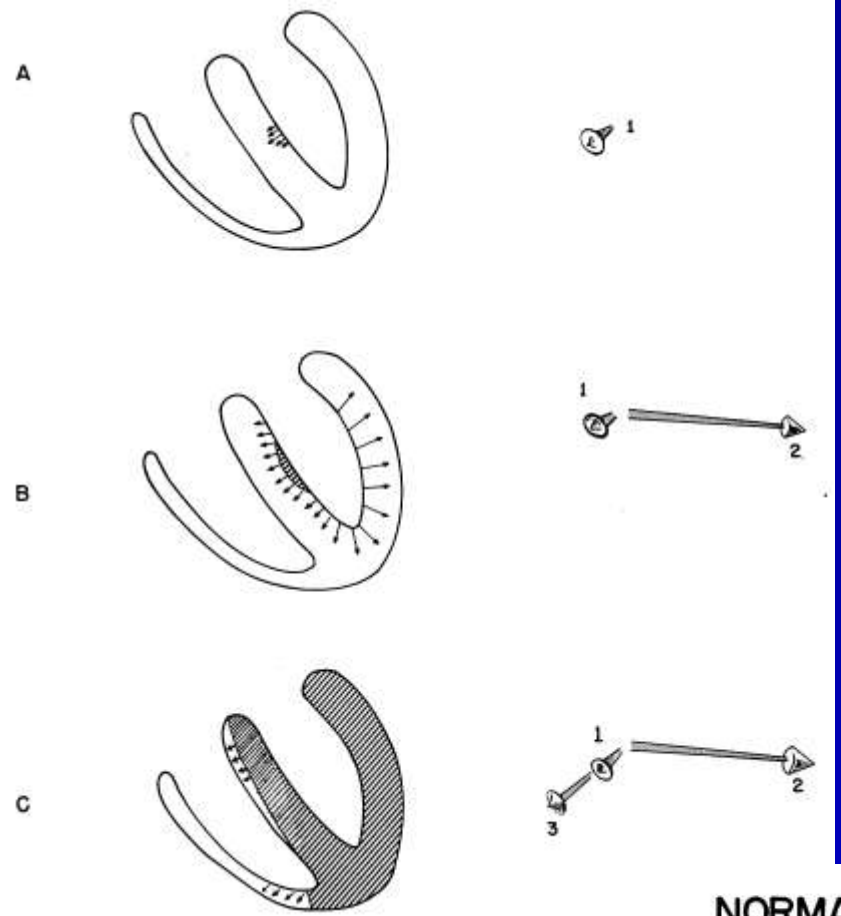


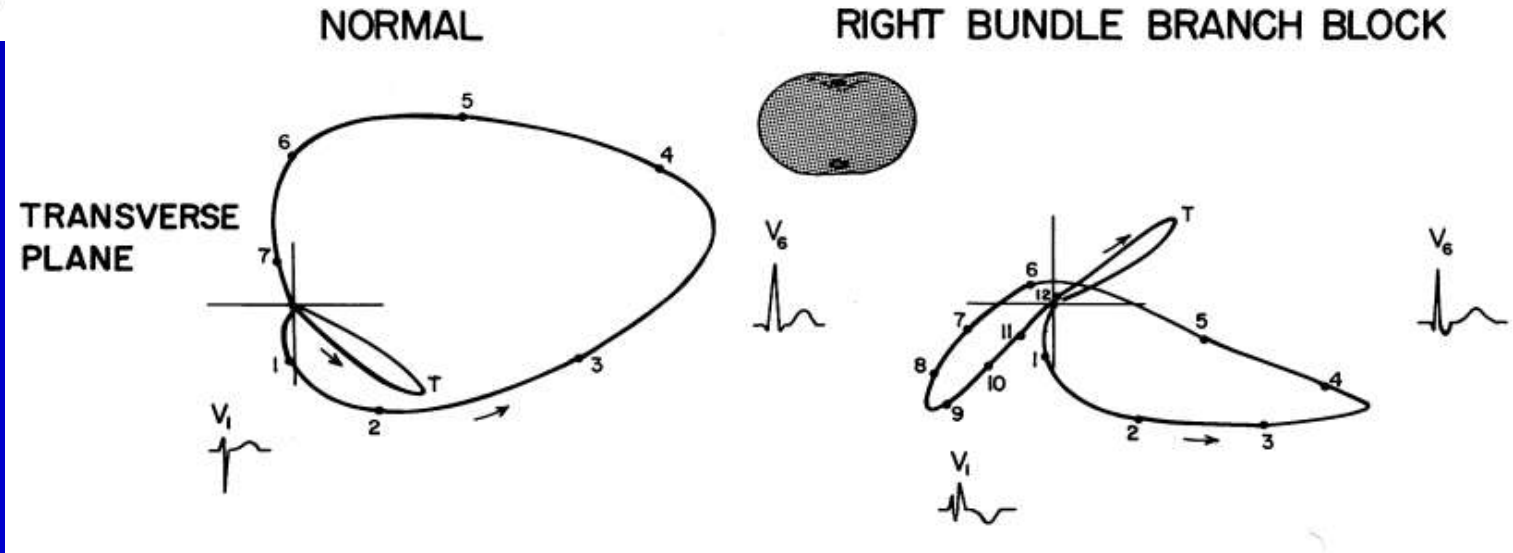
Bellet S. 3rd ed. 1971, p. 62.

Right Bundle Branch Block

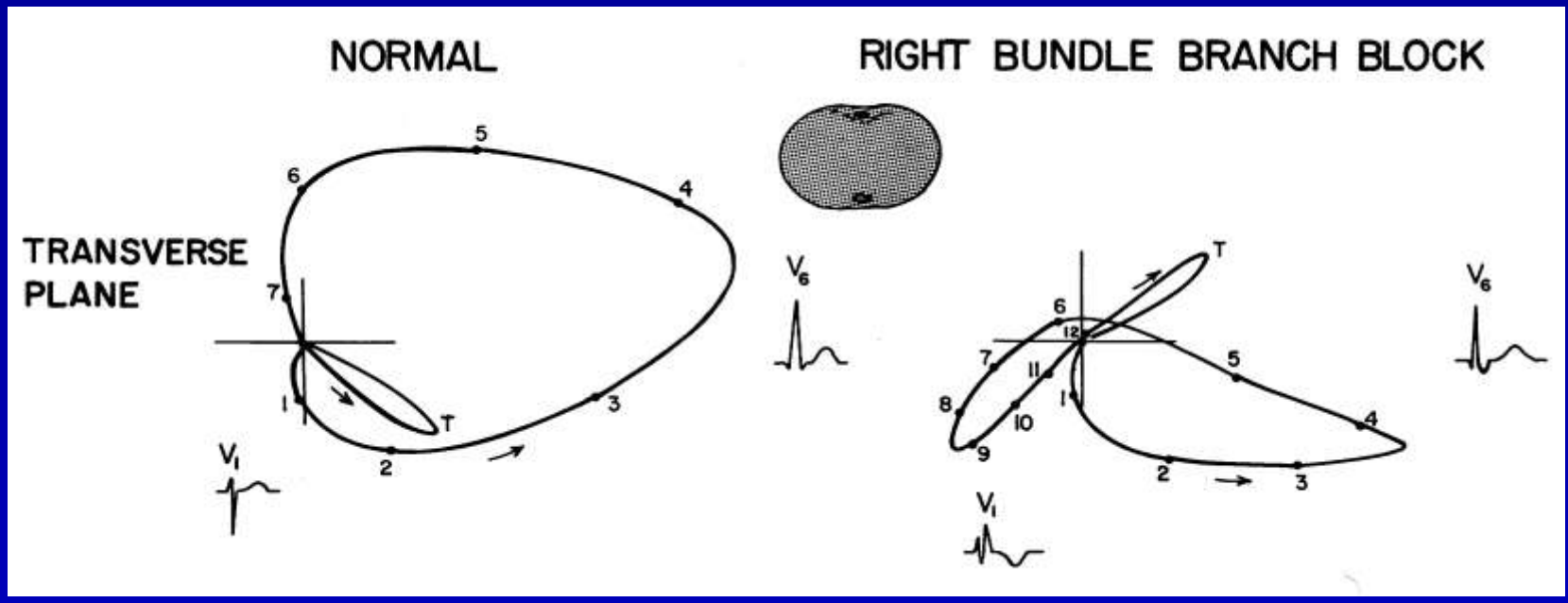


Right Bundle Branch Block





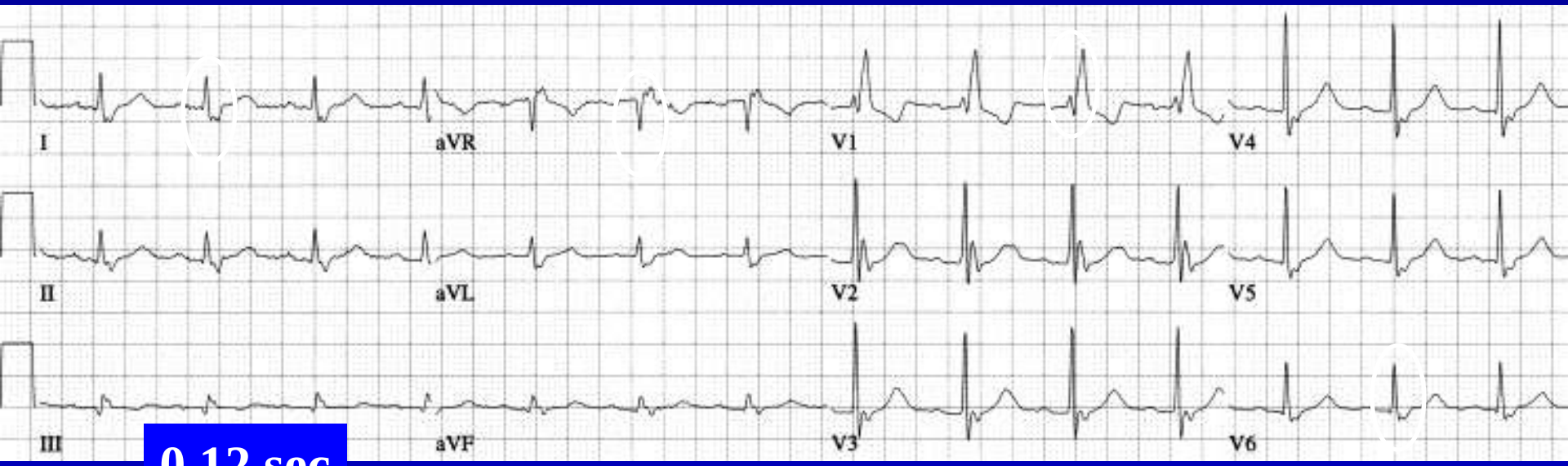
Right Bundle Branch Block



Right Bundle Branch Block

- QRS duration >= 0.12 sec
- R prime in V1 or V2 larger than R wave
- R prime in V1 usually 0.06 sec wide
- Wide (slurred) S wave in I, V5 and V6, often distinct onset
- Preserved initial forces
- Repolarization: T wave opposite the terminal delay

ECG - RBBB



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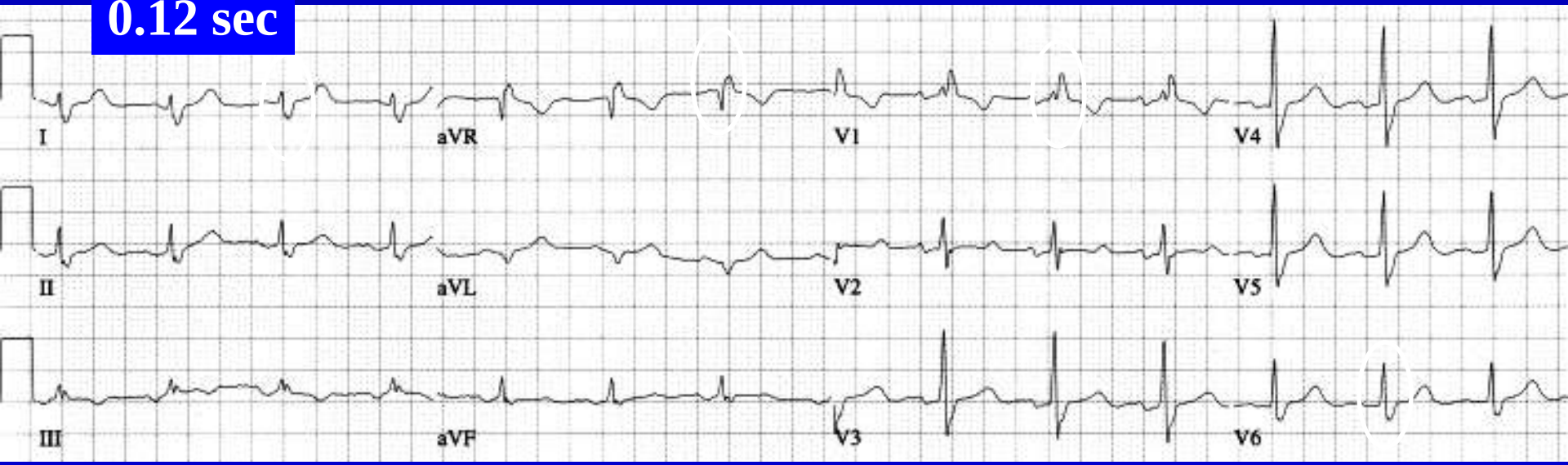
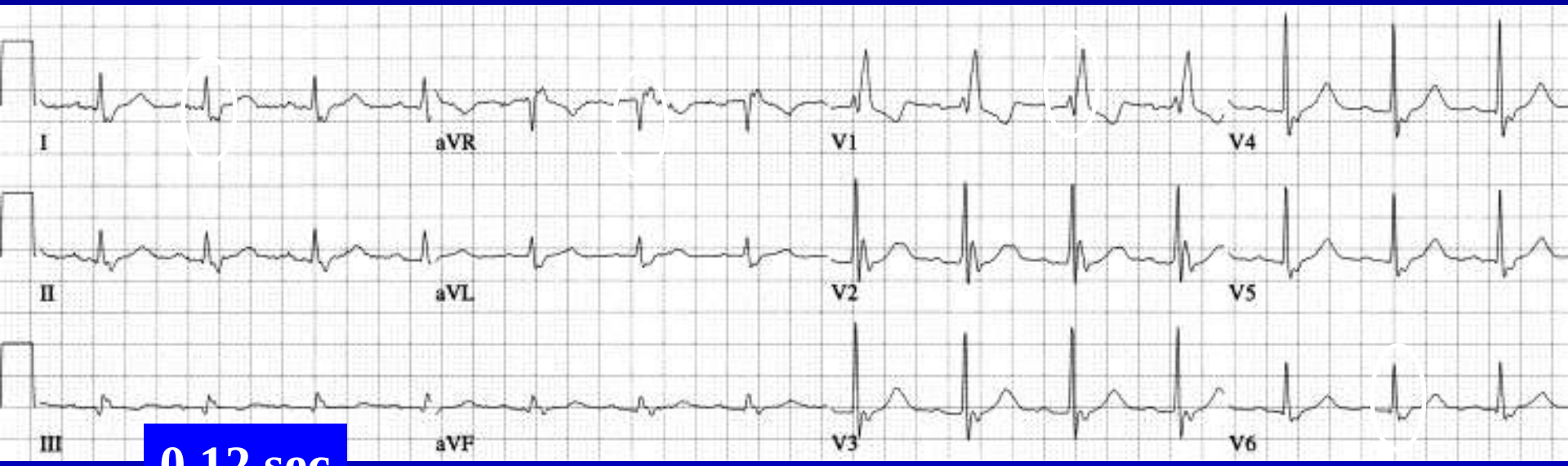
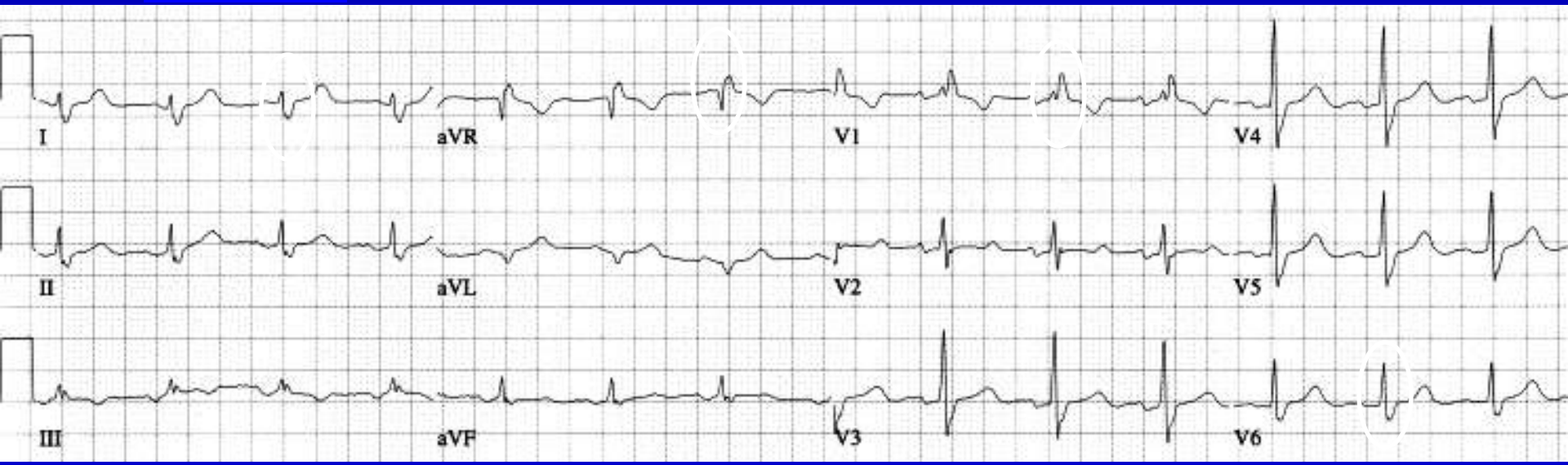


ECG - RBBB



0.12 sec



Right Bundle Branch Block

- Rightward anterior terminal conduction delay
  - QRS 0.12, rsrprime in V1 or 2, S>40 ms or S longer than R in V6 and I, normal R peak time in V5-6 but >0.05 sec in V1
- Initial 0.06 sec unaltered, terminal delay at least 0.04 sec, transition often is obvious
- T wave directed opposite the terminal conduction delay
- ST segment unaltered except possibly depressed inV1-2
- Diagnosis of infarction Q waves and lateral ST depression is accurate

Right Bundle Branch Block

- May occur in normal persons, 1.8/1000
- Acute RBBB in anterior MI is worse prognosis
- Associated with hypertension, CAD, RHD, cor pulmonale, myocarditis incl Chagas' disease, DCM, sclerosis of the cardiac skeleton and degenerative conduction system disease, chest trauma, transplant
- LVH more difficult to diagnose if RBBB
- RVH maybe if R prime in V1 >15 mm, not good sensitivity or specificity
- Incomplete RBBB: usually the RBB is pathologically normal

Left Anterior Fascicular Block

- "…the left bundle branch "system" is composed of a main stem and two main divisions"

Rosenbaum MB. J Electrocard. 1969;2:197

Left Anterior Fascicular Block

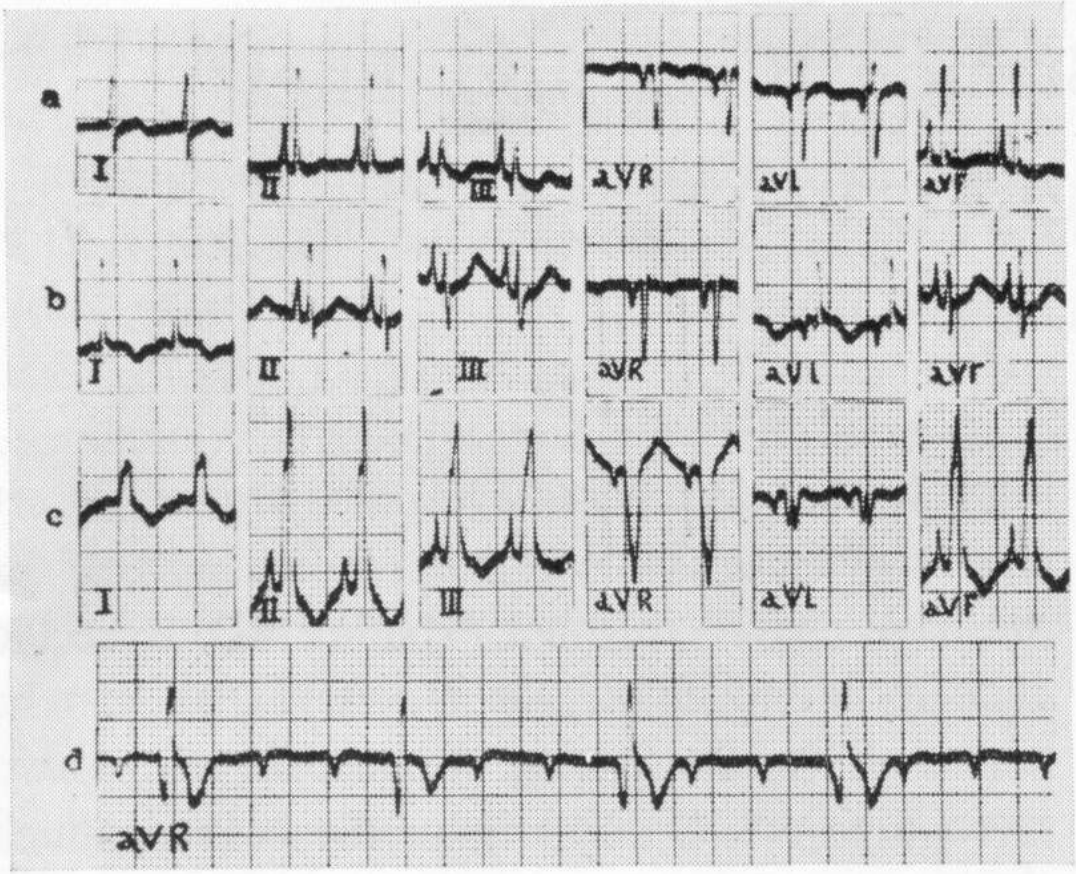


Fig. 1. "Divisional" LBBB and "trifascicular" heart block produced experimentally in the dog. a) Control tracing. b) Pure LAH, after cutting the anterior division of the left bundle branch. c) "Divisional" LBBB after subsequently cutting the posterior division. d) The right bundle branch is also cut, and "trifascicular" complete heart block is produced.

Rosenbaum MB. J Electrocard. 1969;2:197

Left Anterior Fascicular Block

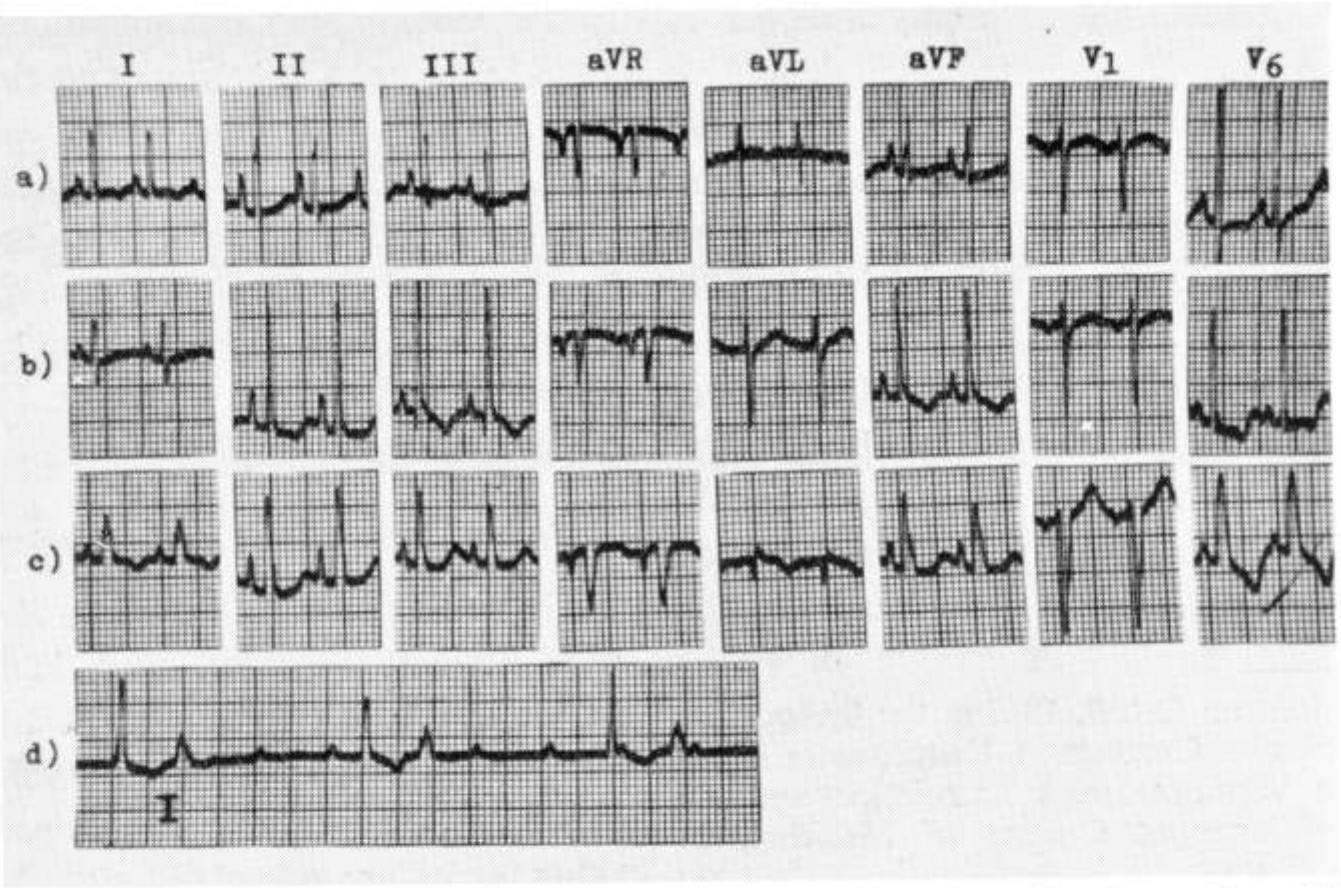


Fig. 2. Pure LPH, "divisional" LBBB and "trifascicular" heart block produced experimentally in the dog. a) Control tracing. b) Pure LPH, after cutting the posterior division of the left bundle branch. c) "Divisional" LBBB, after subsequently cutting the anterior division. d) The right bundle branch is also cut, and "trifascicular" heart block develops.

Rosenbaum MB. J Electrocard. 1969;2:197

Left Anterior Fascicular Block

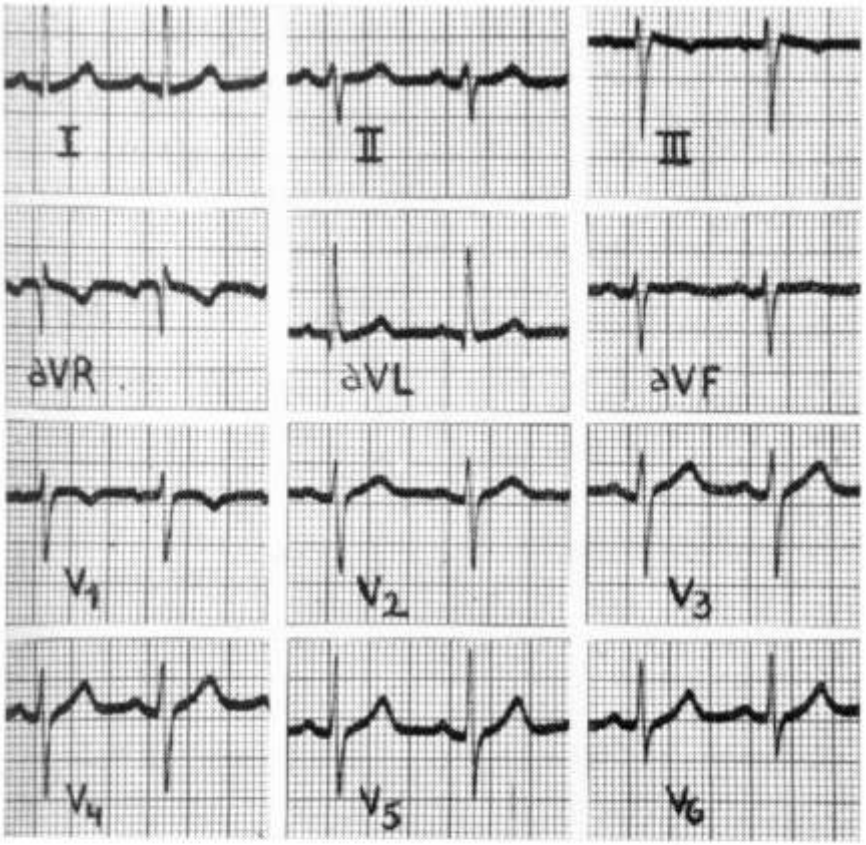


Fig. 3. Typical, uncomplicated LAH. ÂQRS close to −60° Q_I S_{III} and QRS interval of 0.08 sec. Notice the deep S wave up to V_6 and the lack of Q wave in $V_4 - V_6$. Tracings of this kind are considered by some authors as indicating counterclockwise rotation of the heart on its longitudinal axis because of the presence of Q_I S_{III} and by others, as indicating clockwise rotation because of the leftward shift of the transitional zone. Actually, neither one is present for both are due to LAH.

Rosenbaum MB. J Electrocard. 1969;2:197

Left Anterior Fascicular Block

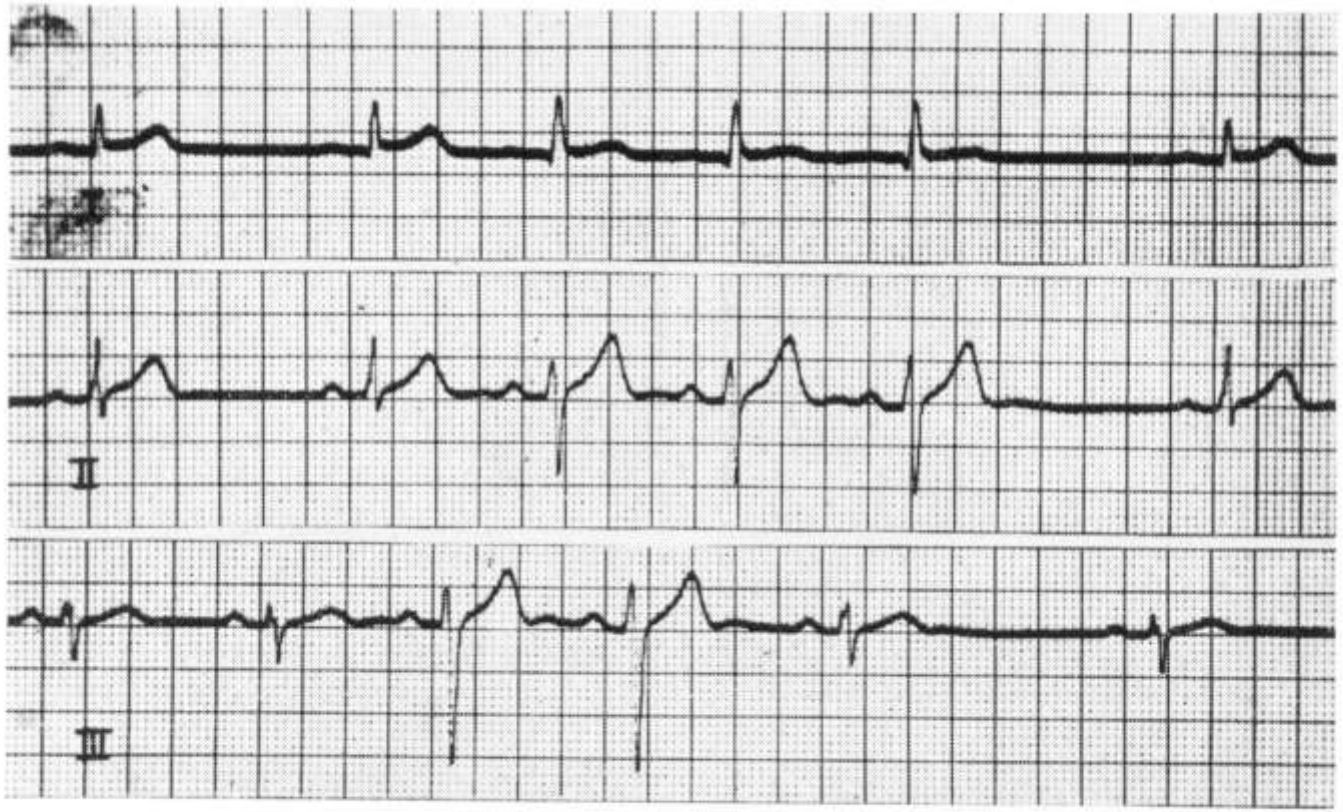


Fig. 4A. A case of intermittent LAH. LAH (beats 3, 4 and 5 in lead I-II; 3 and 4 in lead III) appears when the heart cycle shortens, and disappears when it lengthens.

Rosenbaum MB. J Electrocard. 1969;2:197

Left Anterior Fascicular Block

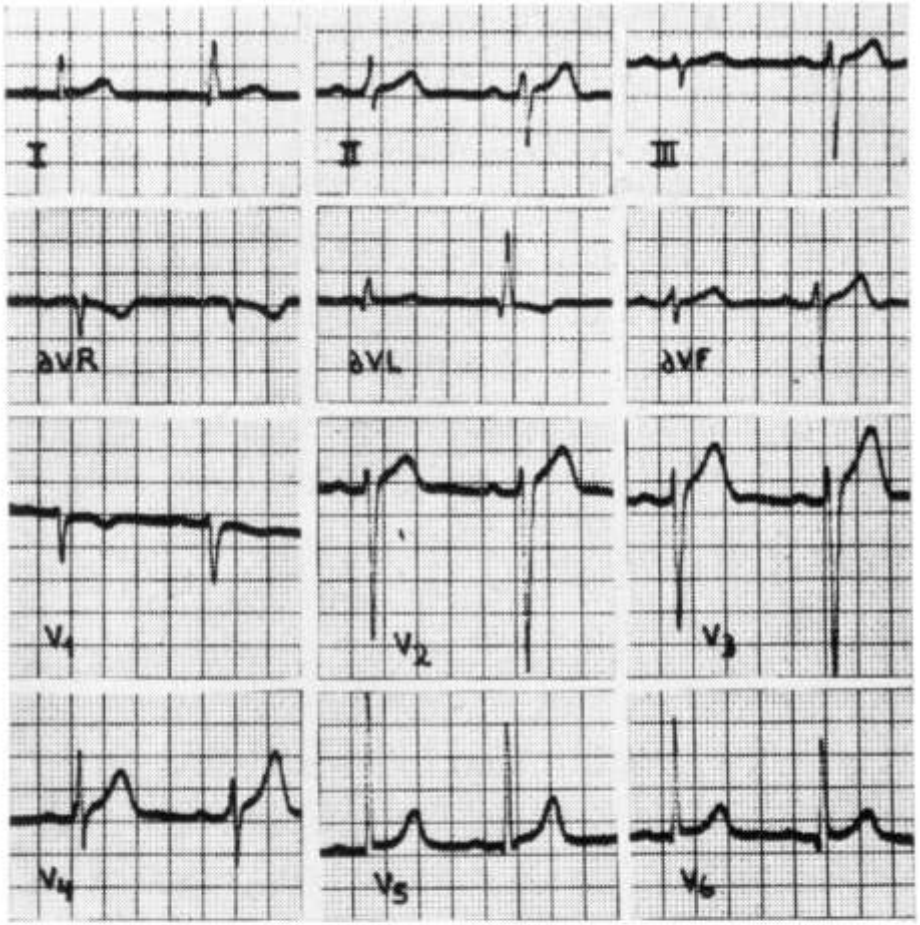


Fig. 4B. Same case as in 4A. In each lead the first beat is without and the second with LAH. LAH shifts ÂQRS from 0 to −60°; widens the QRS interval from 0.08–0.09 to 0.10–0.11 sec; slightly increases the Q wave in leads aVL and III; diminishes the R wave in V_5 and V_6; and simulates left ventricular hypertrophy in aVL.

Rosenbaum MB. J Electrocard. 1969;2:197

Left Anterior Fascicular Block

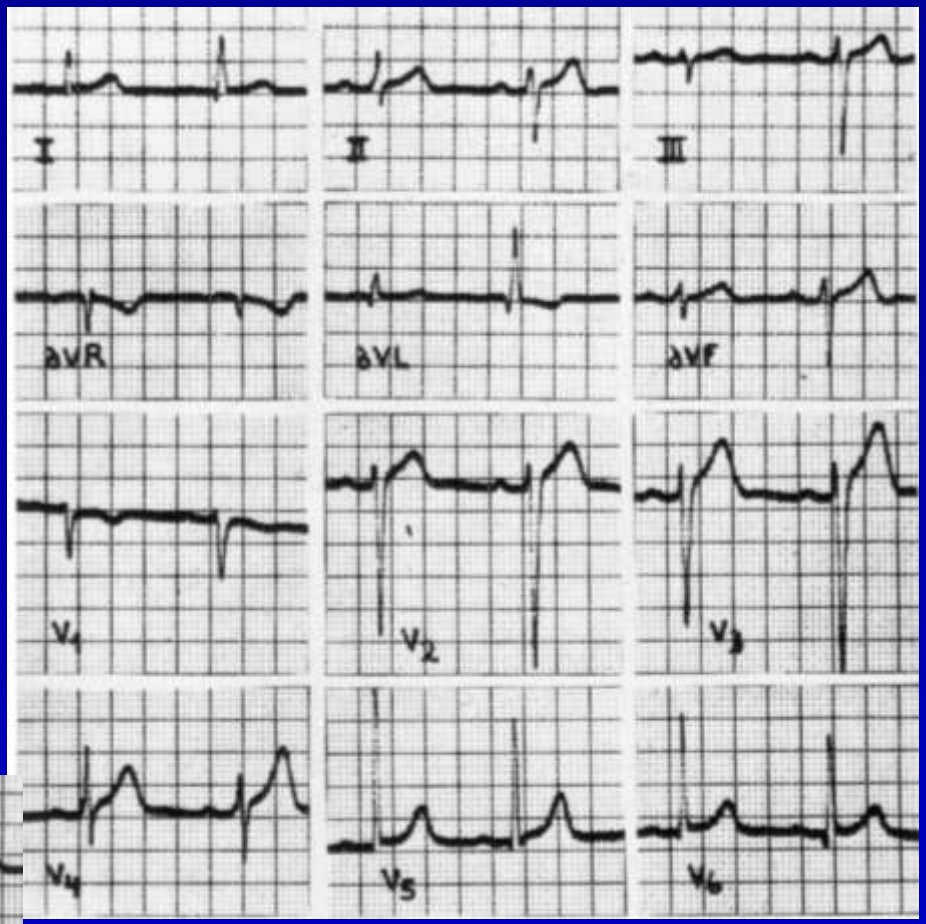


Fig. 4B. Same case as in 4A. In each lead the first beat is without and the second with LAH. LAH shifts ÂQRS from 0 to −60°; widens the QRS interval from 0.08–0.09 to 0.10–0.11 sec; slightly increases the Q wave in leads aVL and III; diminishes the R wave in V_5 and V_6; and simulates left ventricular hypertrophy in aVL.

Rosenbaum MB. J Electrocard. 1969;2:197

Left Anterior Fascicular Block

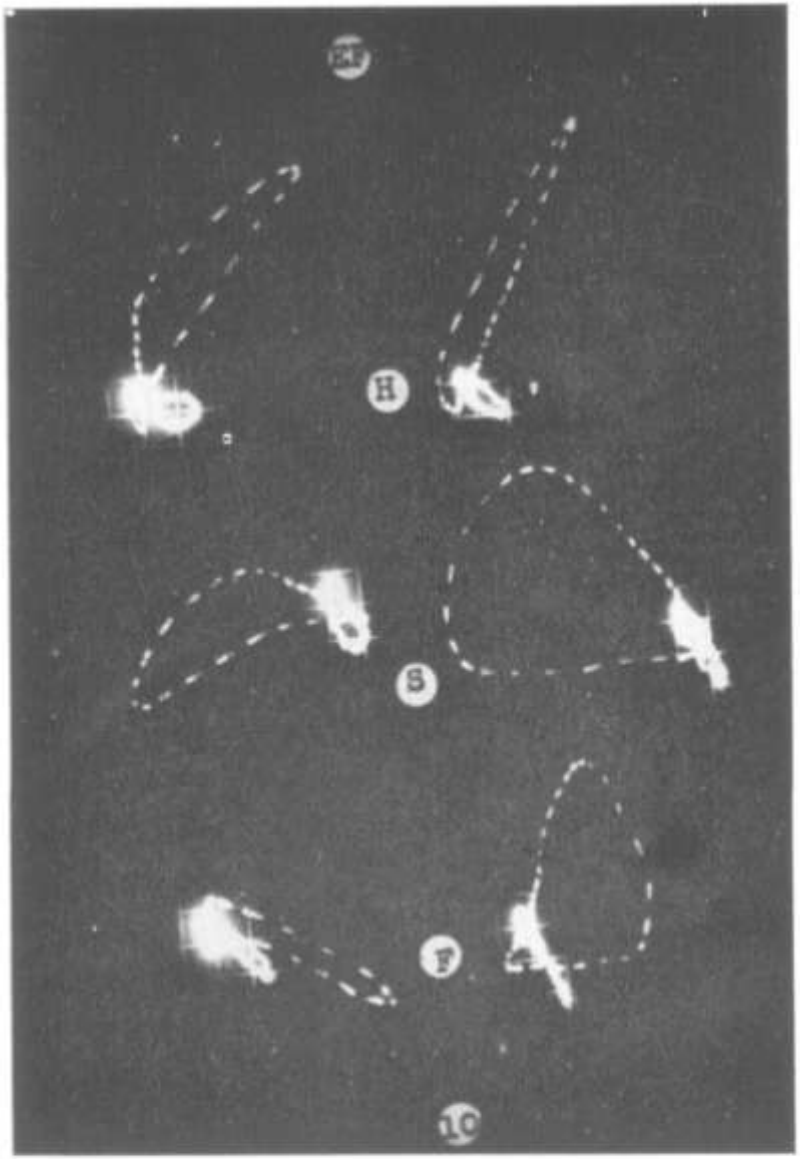


Fig. 4C. VCG from the same case as in 4A-B, with and without LAH. Notice how LAH (right side of picture) produces an initial force pointing inferiorly and to the right, and main QRS forces superiorly and to the left.

Rosenbaum MB. J Electrocard. 1969;2:197

Left Anterior Fascicular Block

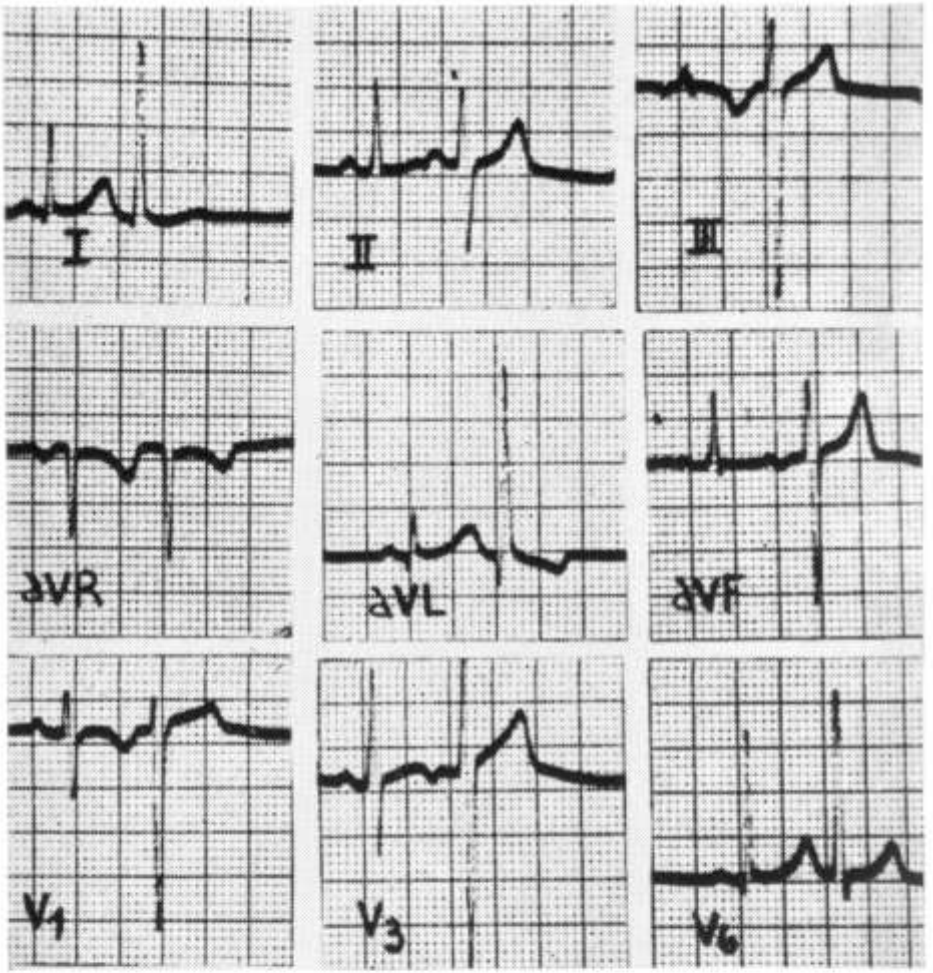


Fig. 5A. "Physiological" LAH caused by aberrant ventricular conduction of atrial premature beats (the second beat in each lead), in a healthy 12 year-old boy. LAH shifts ÂQRS from +35 to −30°, barely widens the QRS interval from 0.07 to 0.08 sec. and obliterates the Q wave from V_6. Besides, it simulates left ventricular hypertrophy in the extremity leads and to a lesser degree in the precordial leads.

Rosenbaum MB. J Electrocard. 1969;2:197

Left Anterior Fascicular Block

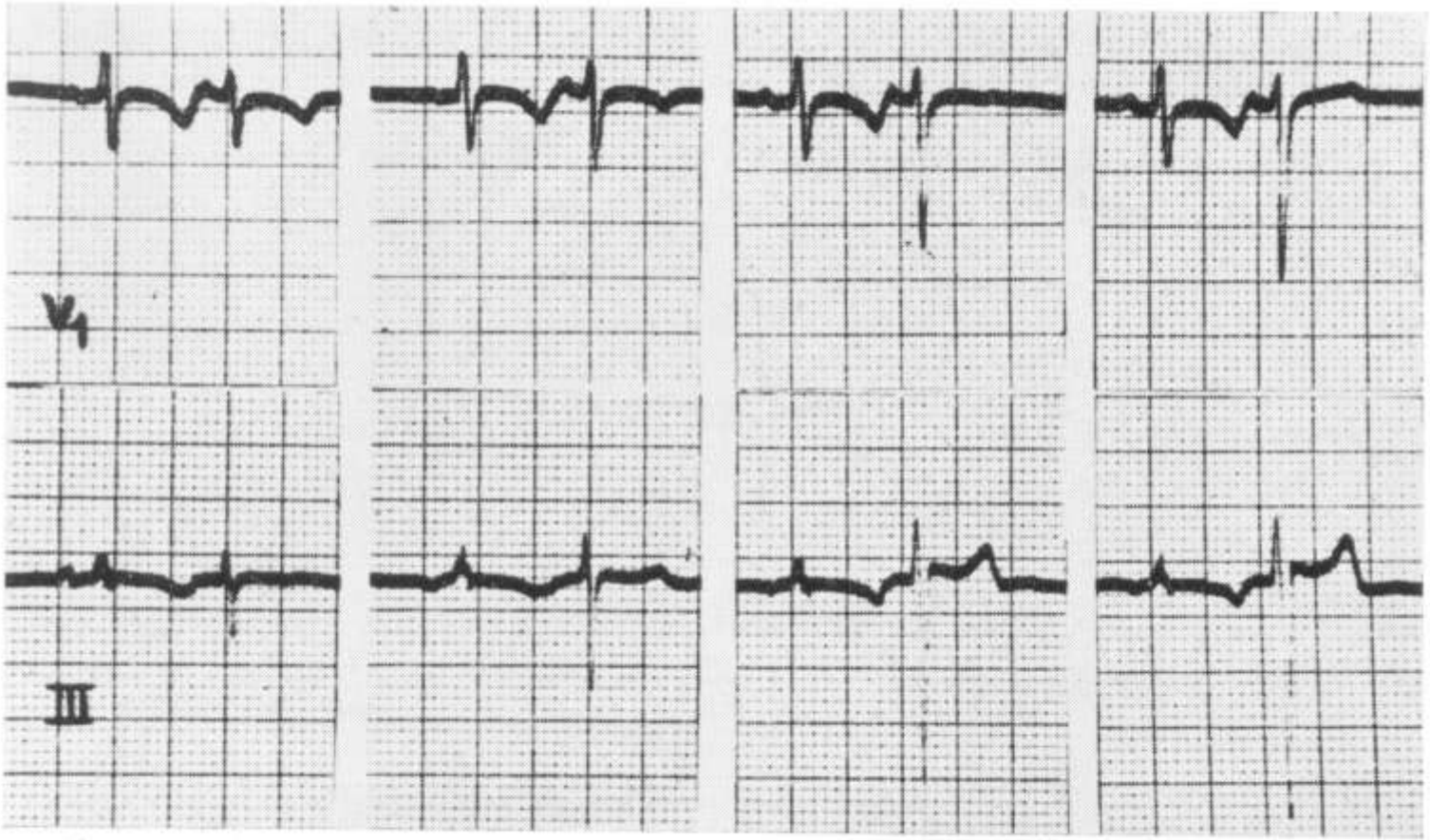


Fig. 5B. Same case as in 5A. Four different and progressive degrees of LAH.

Rosenbaum MB. J Electrocard. 1969;2:197

Left Anterior Fascicular Block

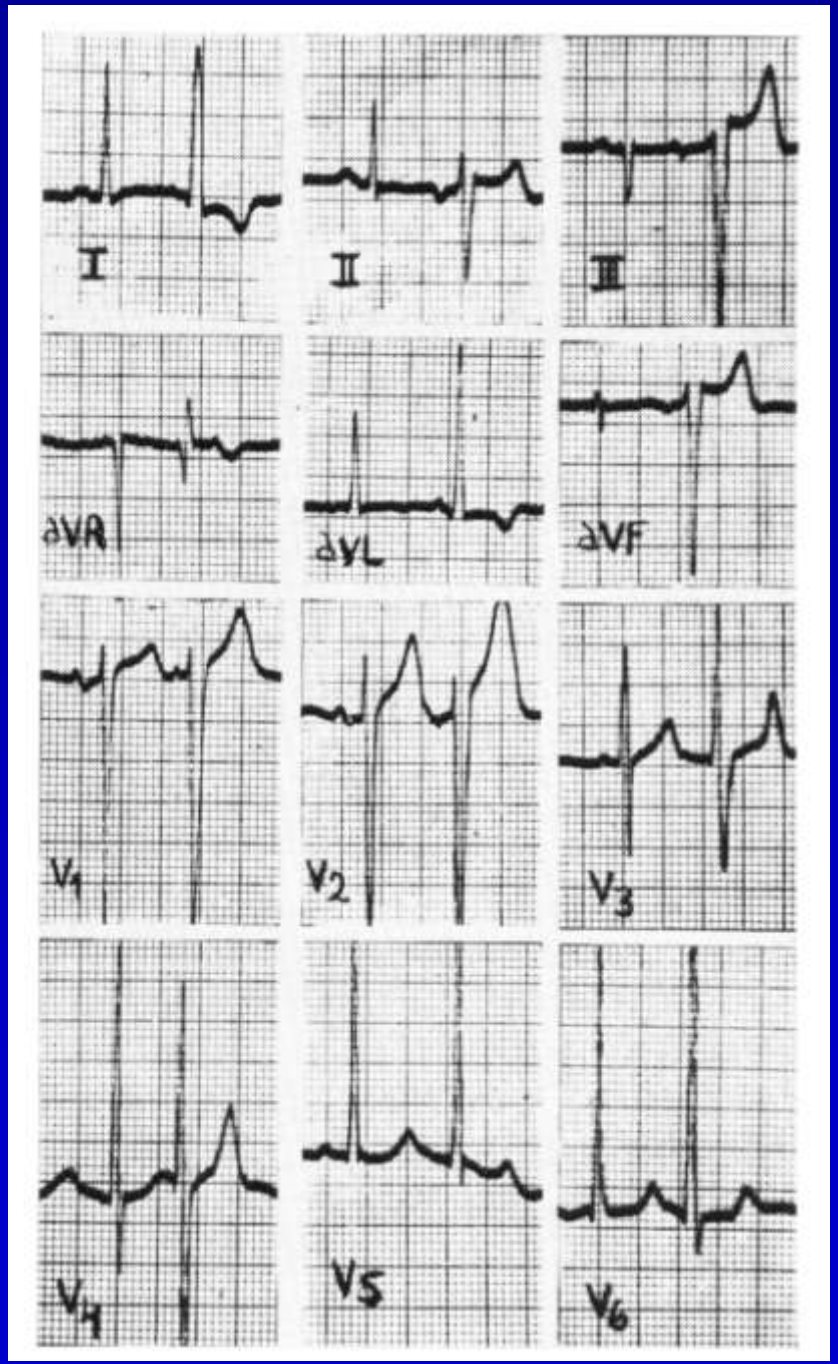


Fig. 6A. "Physiological" LAH caused by aberrant ventricular conduction of atrial extrasystoles (the second beat in each lead), in a 65-year-old man with arterial hypertension and left ventricular hypertrophy. In addition to the superior shift of the main QRS forces, LAH produces signs of "left ventricular strain" in the standard leads.

Rosenbaum MB. J Electrocard. 1969;2:197

Left Anterior Fascicular Block

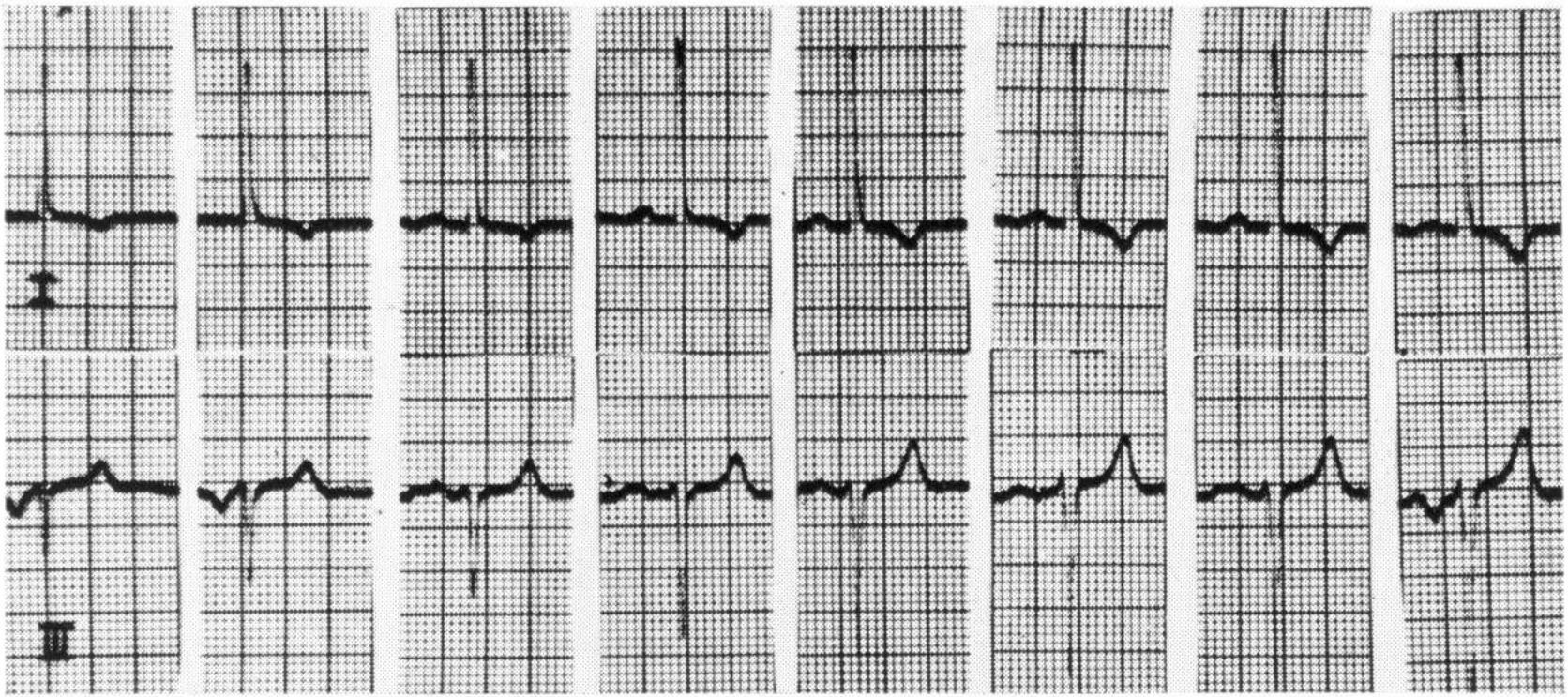


Fig. 6B. Same case as in 6A. Eight different and progressive degrees of LAH.

Rosenbaum MB. J Electrocard. 1969;2:197

Left Anterior Fascicular Block

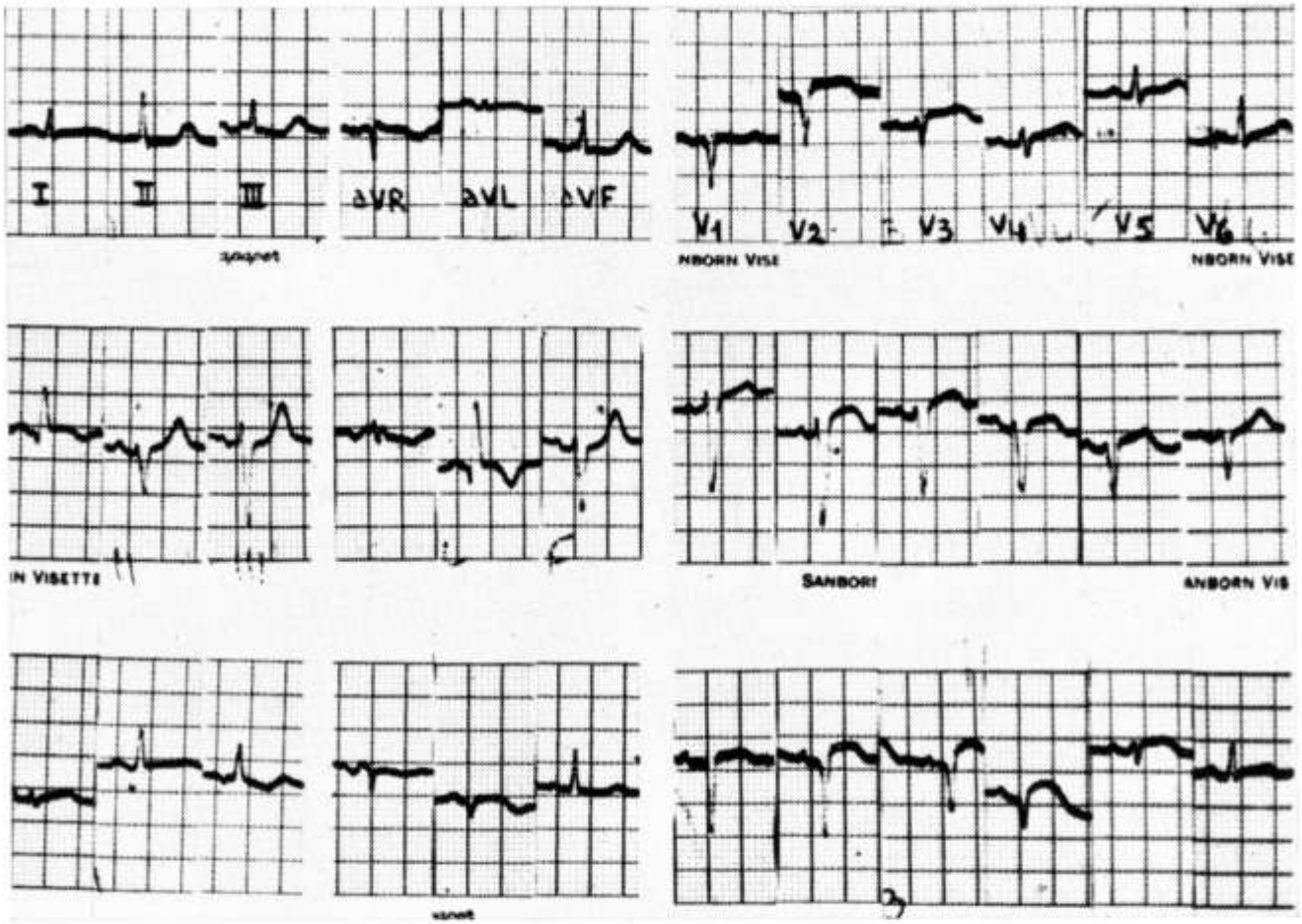


Fig. 7. Transient LAH due to an acute anteroseptal infarction. *Top:* Acute anteroseptal myocardia infarction. ÂQRS: +60°; QRS Interval: 0.08 sec. *Middle:* Two days later LAH has developed. ÂQRS: −60°; QRS interval: 0.10 sec. LAH conceals some of the signs of the anteroseptal infarction. *Bottom:* One day later, LAH has vanished.

Rosenbaum MB. J Electrocard. 1969;2:197

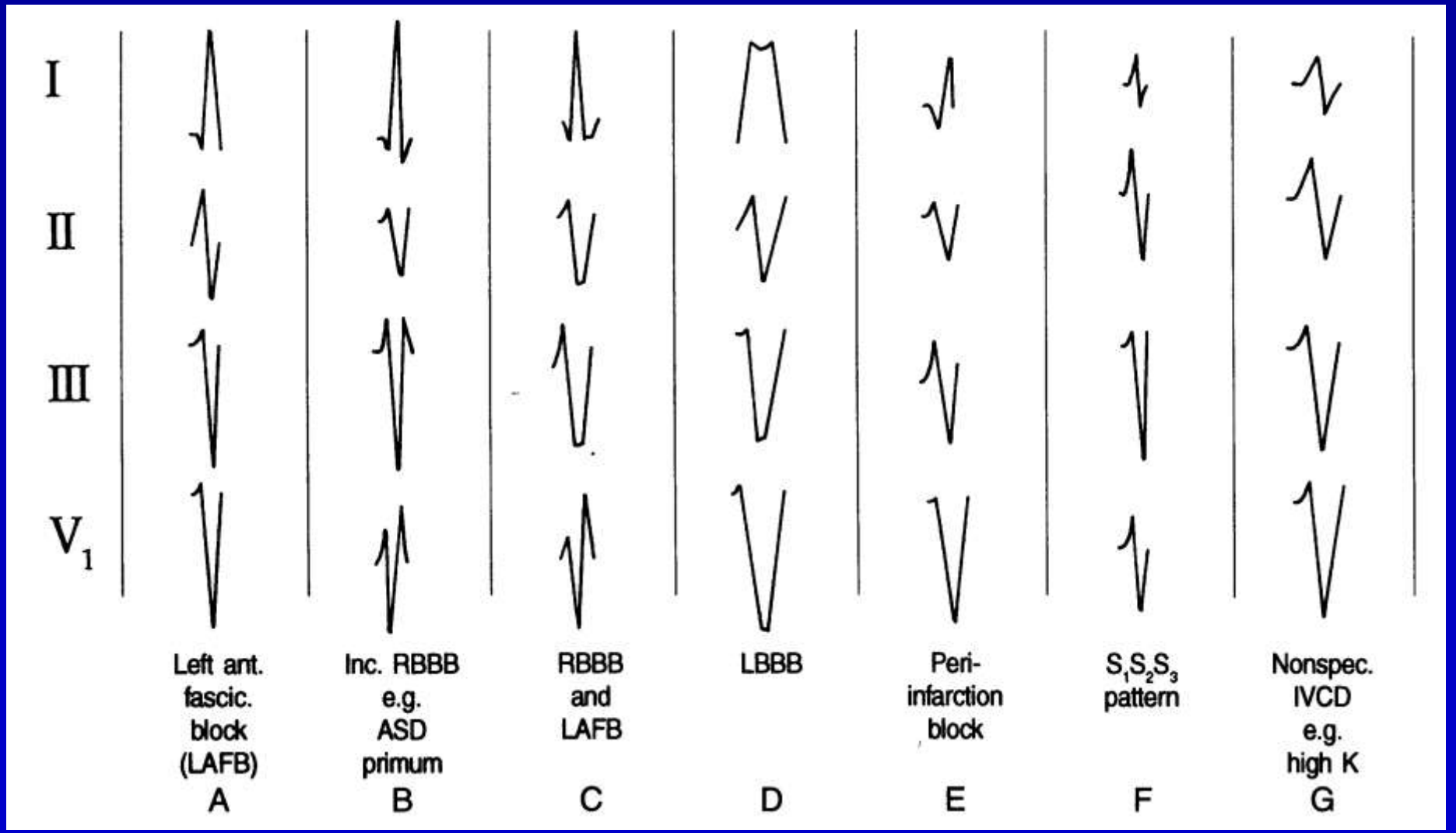
Other IVCD

- LAFB: qR in left leads, rS in inferior leads, often PPRWP, persistent S in V5-6, often T wave more upright in inferior leads (discordant), may diagnose concomitant inferior MI if peak of R in aVR is later than aVL (counterclockwise loop), but R in aVR and aVL must be terminal
- AFB can cause false positive voltage for LVH in I and III >25 mm
- LAD present (<-30) in 1.9% of normals

Other IVCD

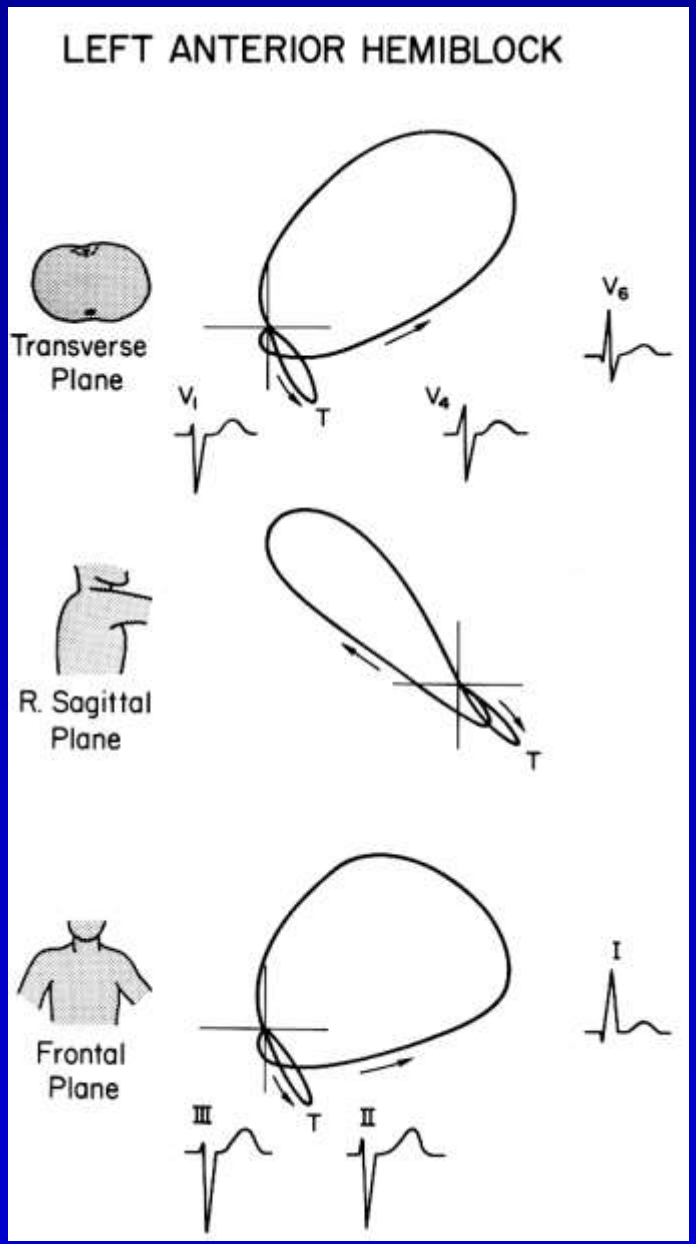
- LPFB: rS in I and aVL, and qR in III and aVF, QRS axis >90 and <180 and QRS <0.12 sec, left precordium may give RS complex – seldom found in absence of RBBB
- Mid-septal fascicles: uncertain
- Bilateral BBB, alternating BBB
- Bifascicular is RBBB plus either AFB or PFB
- Trifascicular block, add FAV to bifascicular
- Periinfarction block: terminal QRS delay directed toward the infarcted area
- Nonspecific IVCD: wide QRS without RBBB or LBBB

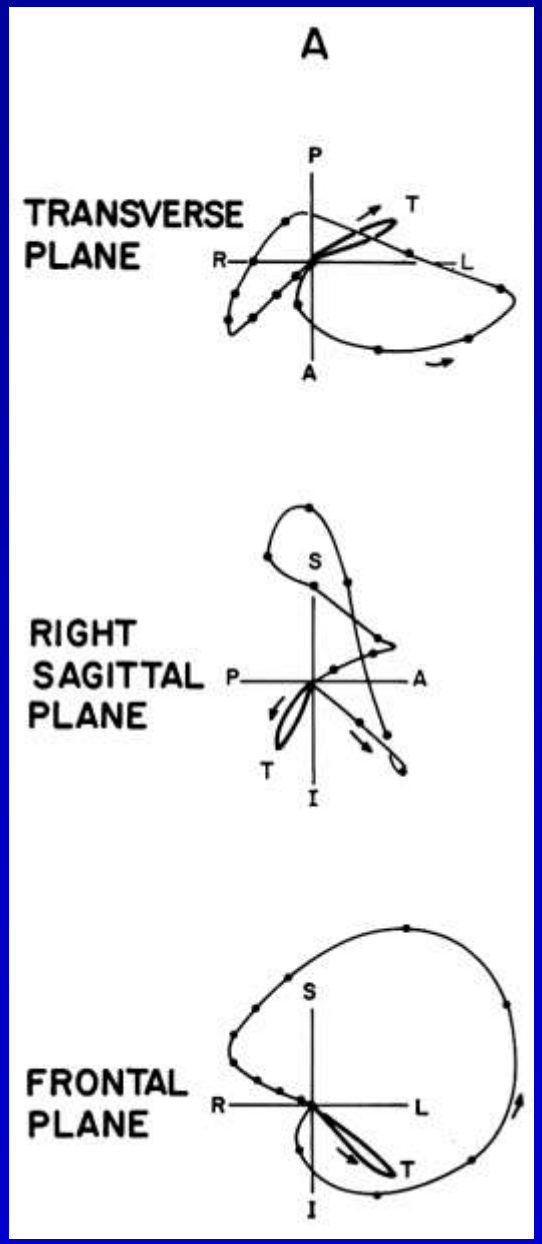
Causes of Left Axis Deviation



Surawicz B et al. P.109, 2001

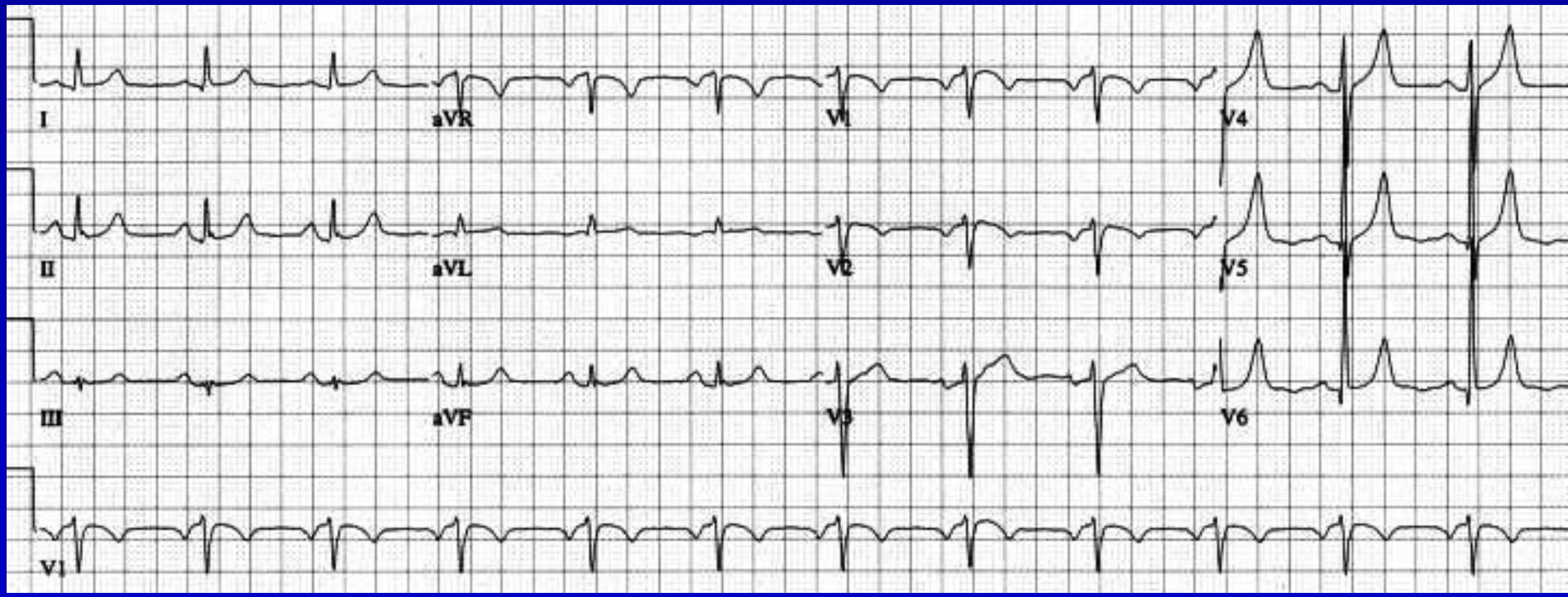
Left Anterior Fascicular Block



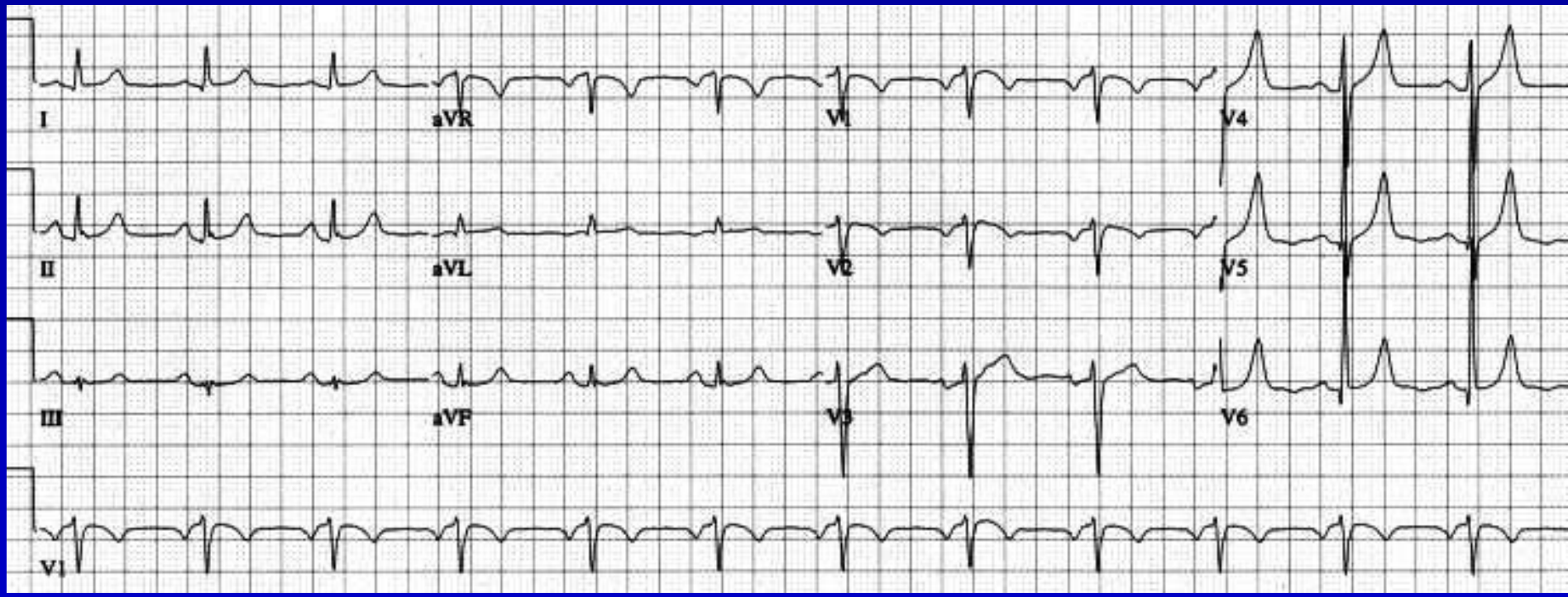


RBBB plus LAFB

Unknown 1

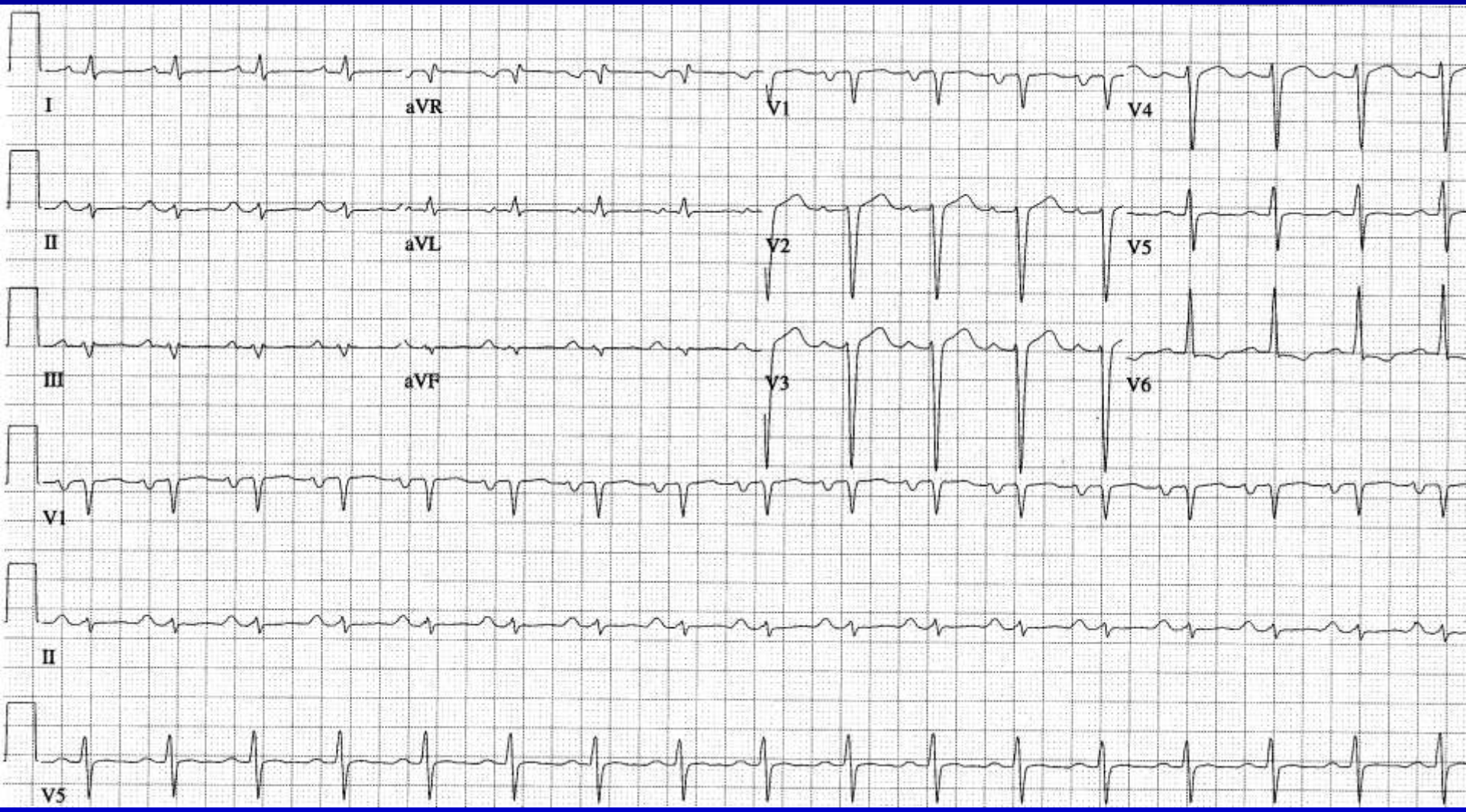


Unknown 1

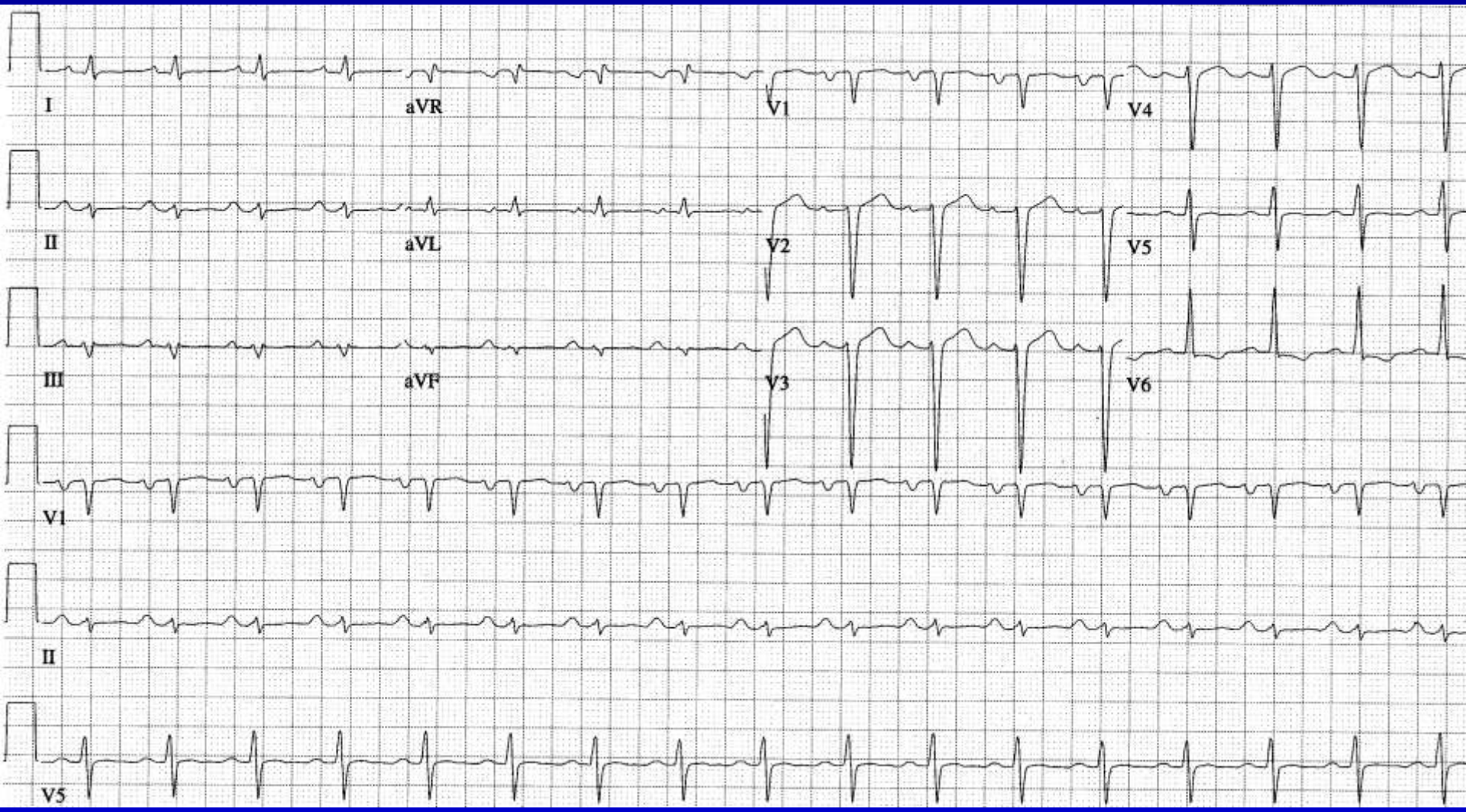


LAE with deep P in V1 and V2 and biphasic in V3
Atrial repolarization in V1 and II
Large voltage
Prominent upright T waves, but inverted U waves in V5 and V6

Unknown 2

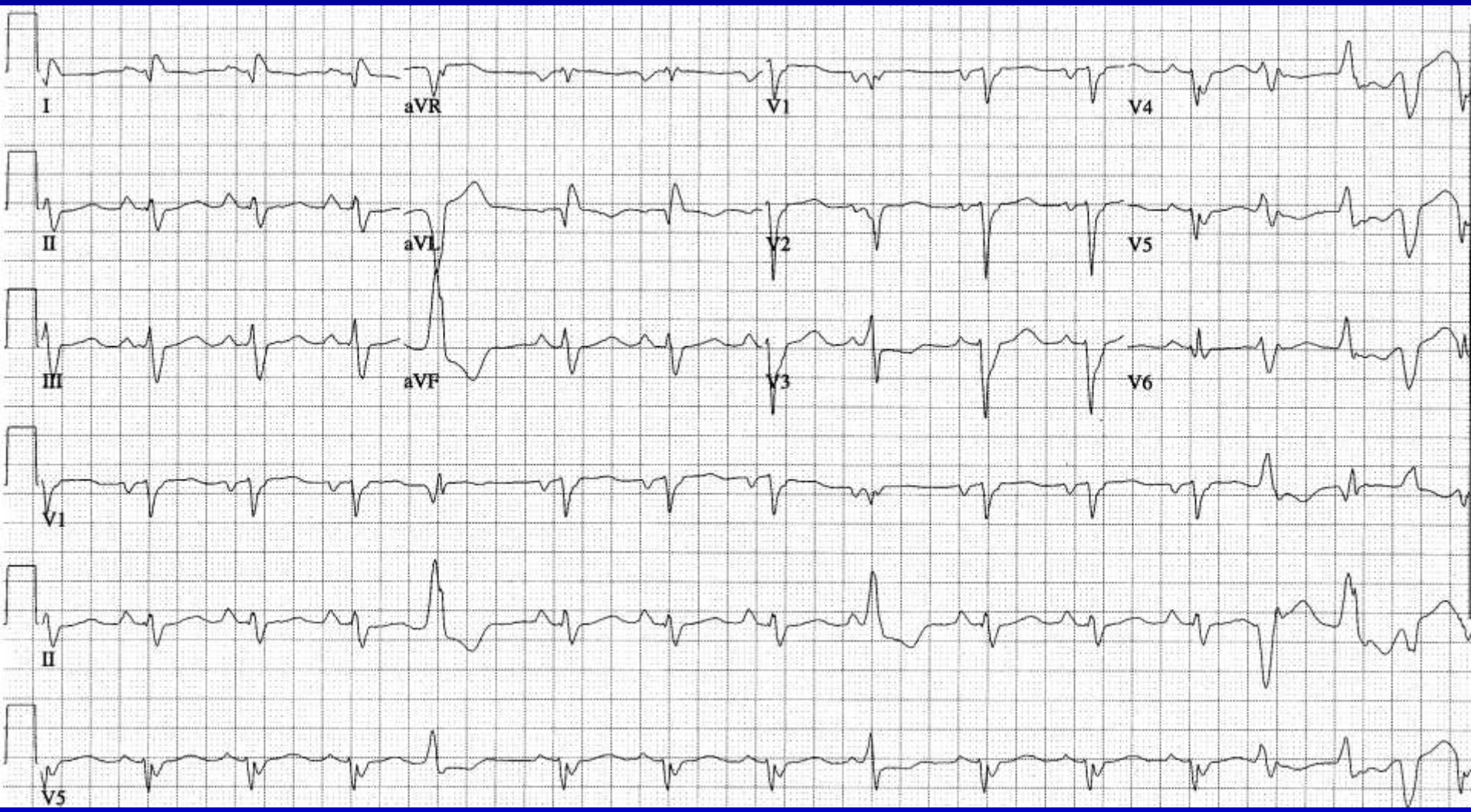


Unknown 2

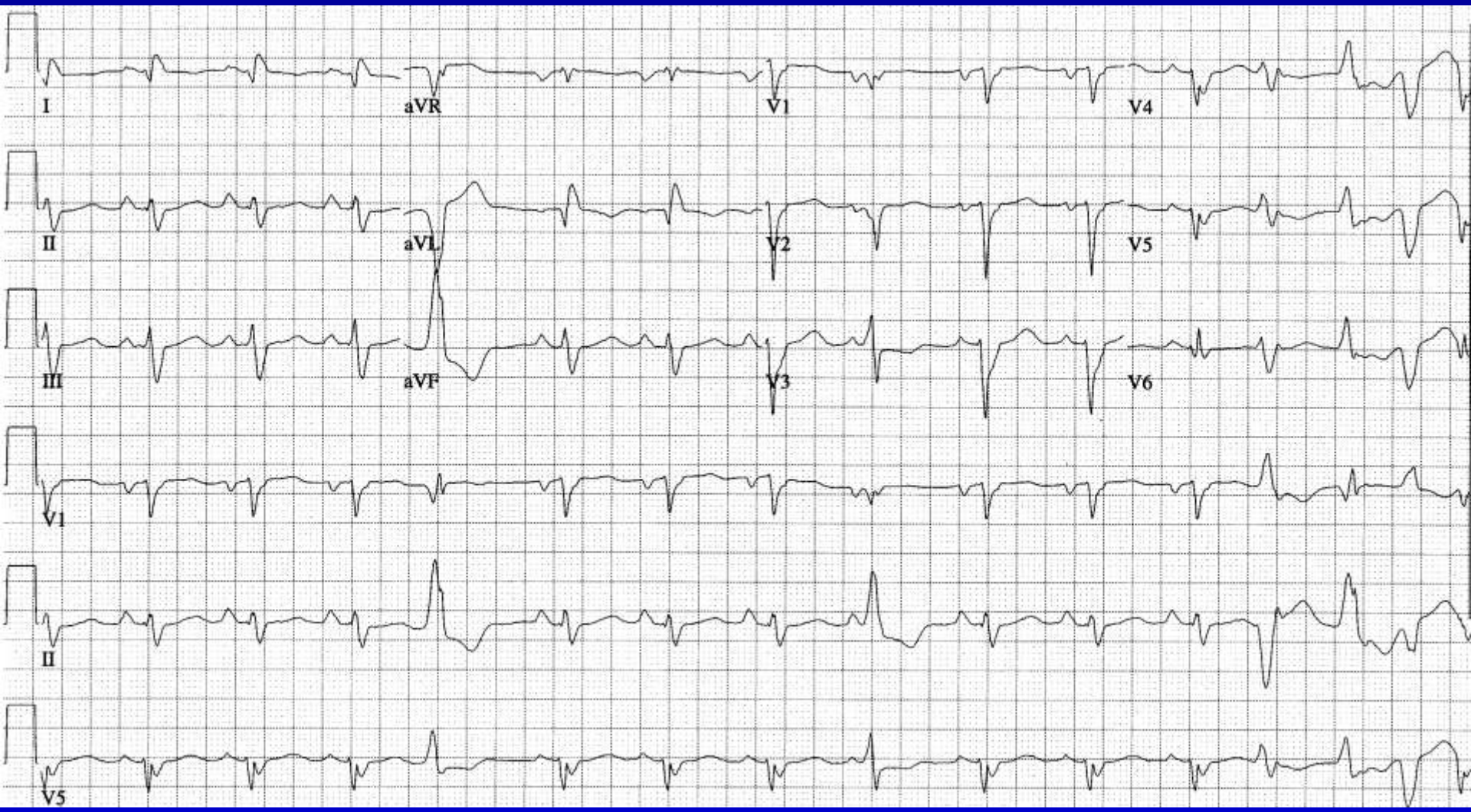


27 yo man short of breath, with sinus tachycardia, QRS duration 0.10, axis –30, LAE, poor R progression, diffuse T abnormality

Unknown 3

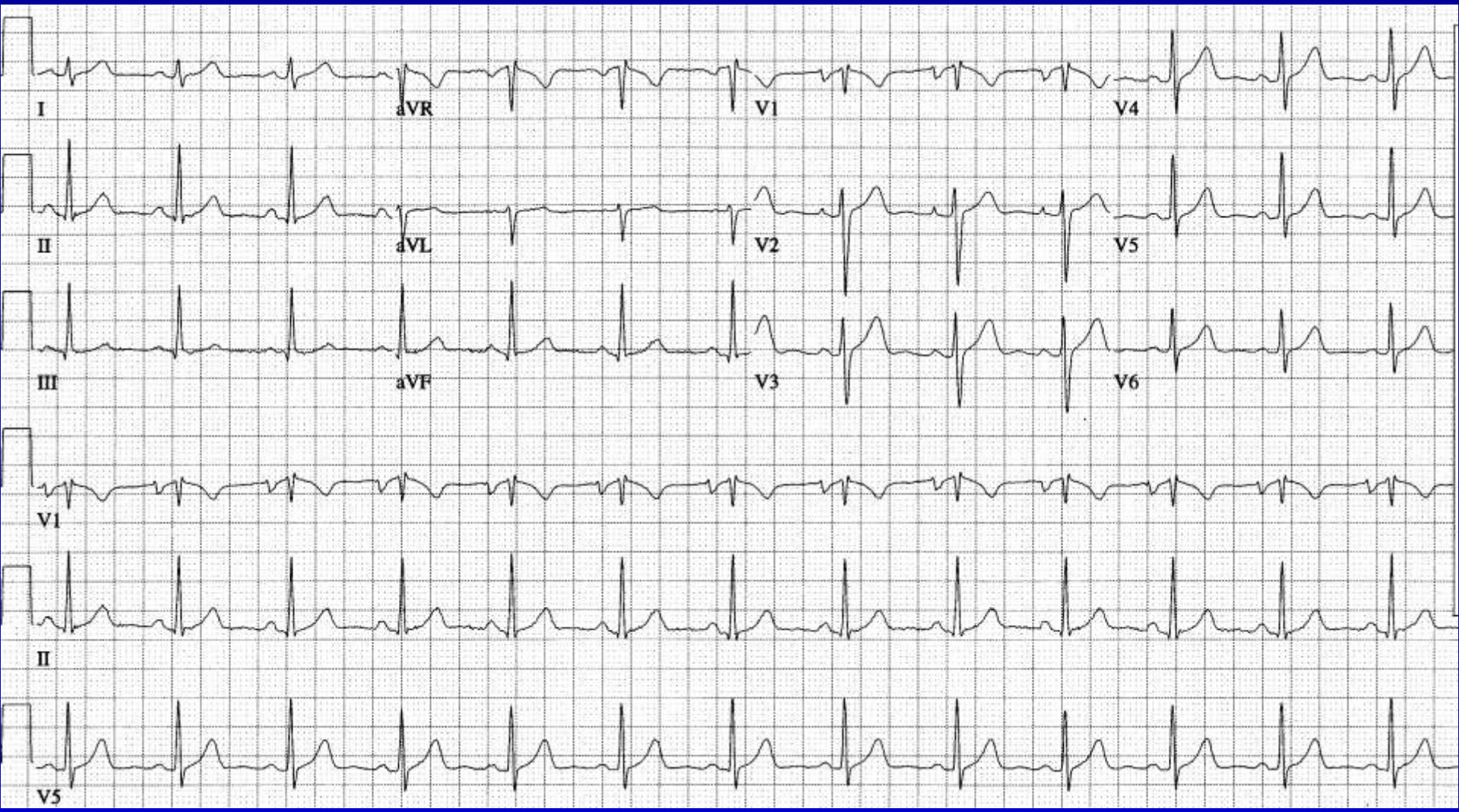


Unknown 3

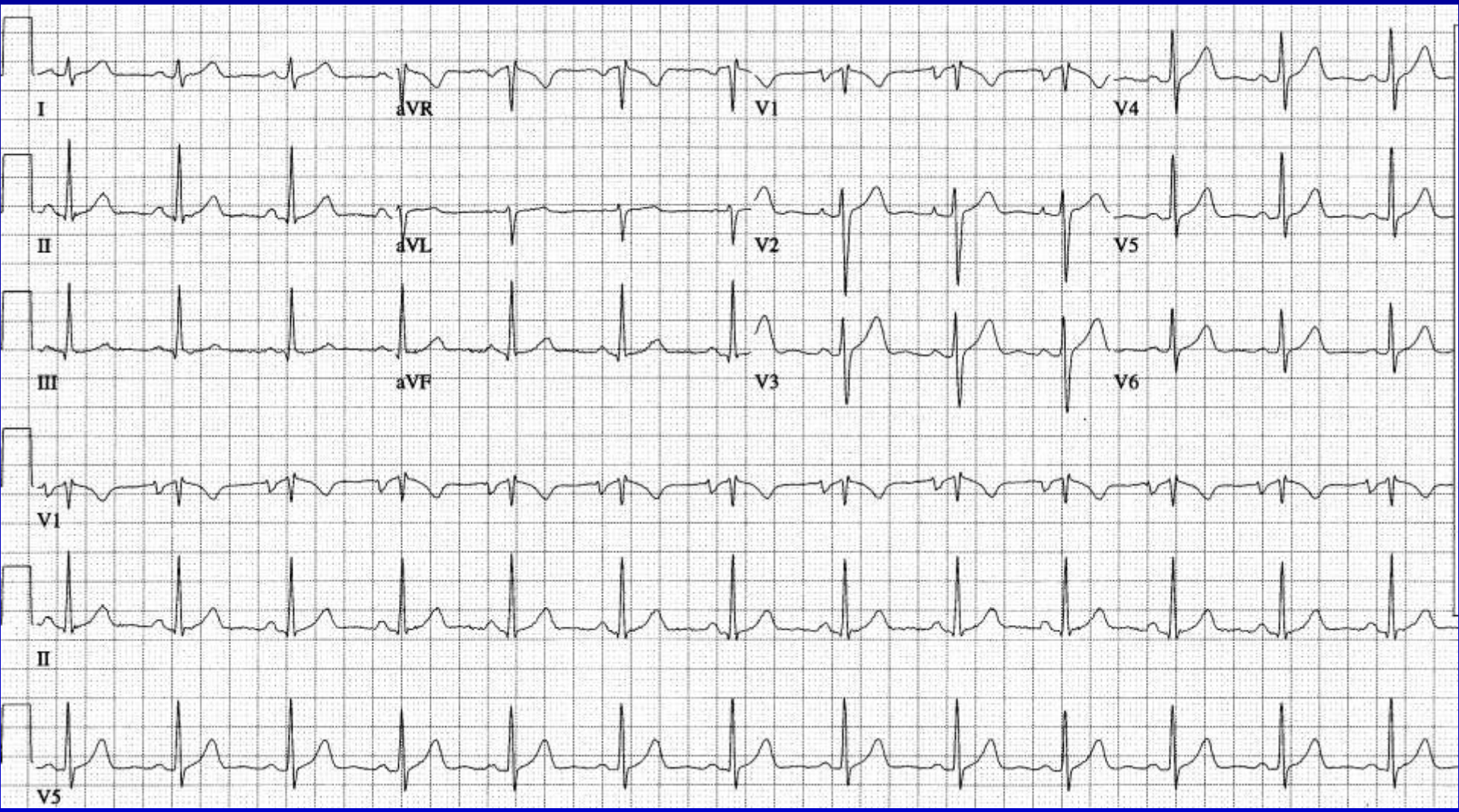


45 yo woman wt 250, sinus rhythm rate 90, LAE, QRS 0.15 sec, ?lateral wall MI, PVC's with triplet or quadruplet

Unknown 4

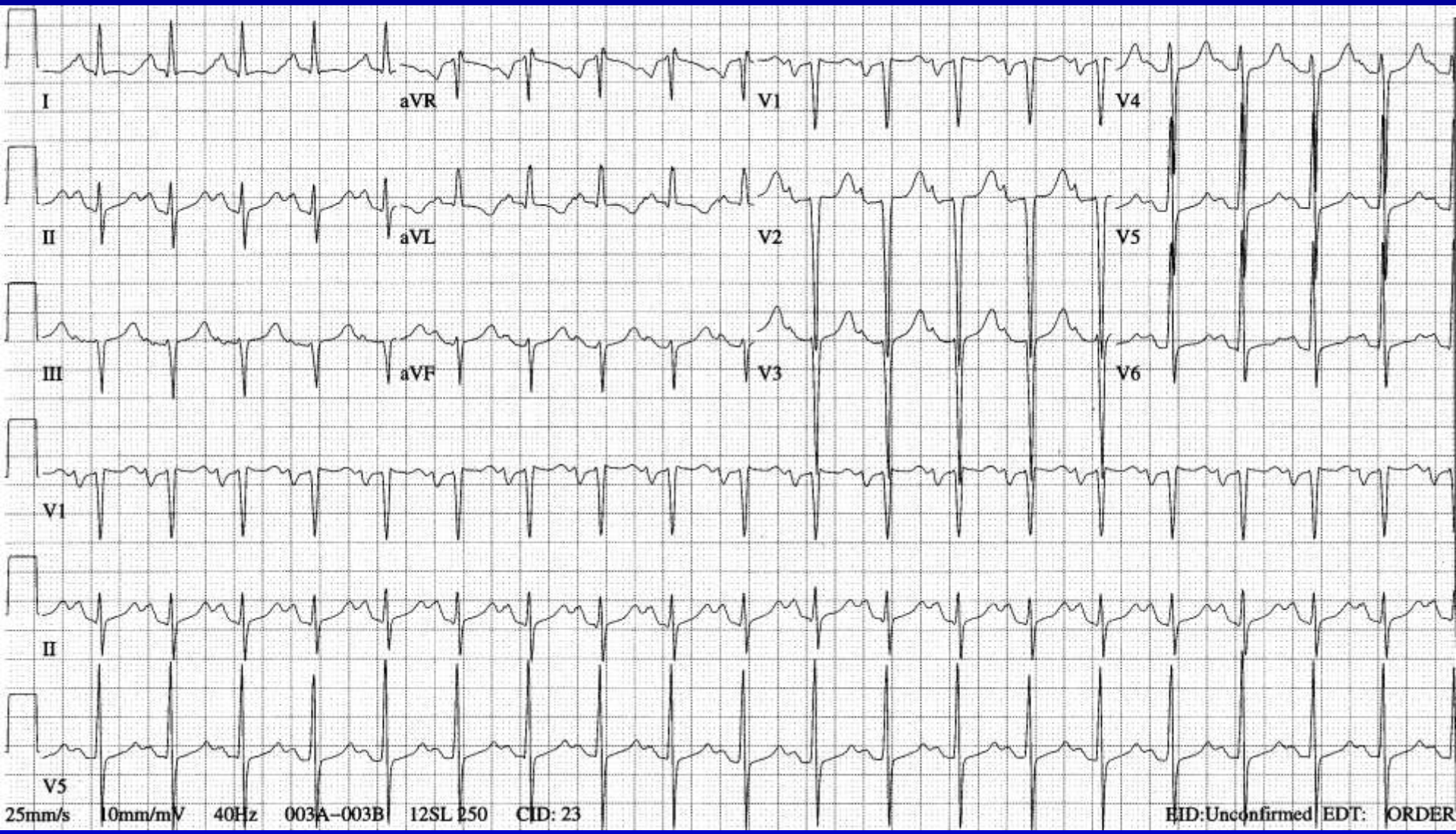


Unknown 4



28 yo man, PR 0.15, QRS 0.08, axis 85, LAE but no other problem.
Possible MS, possible acute MR, less likely myocarditis or DCM

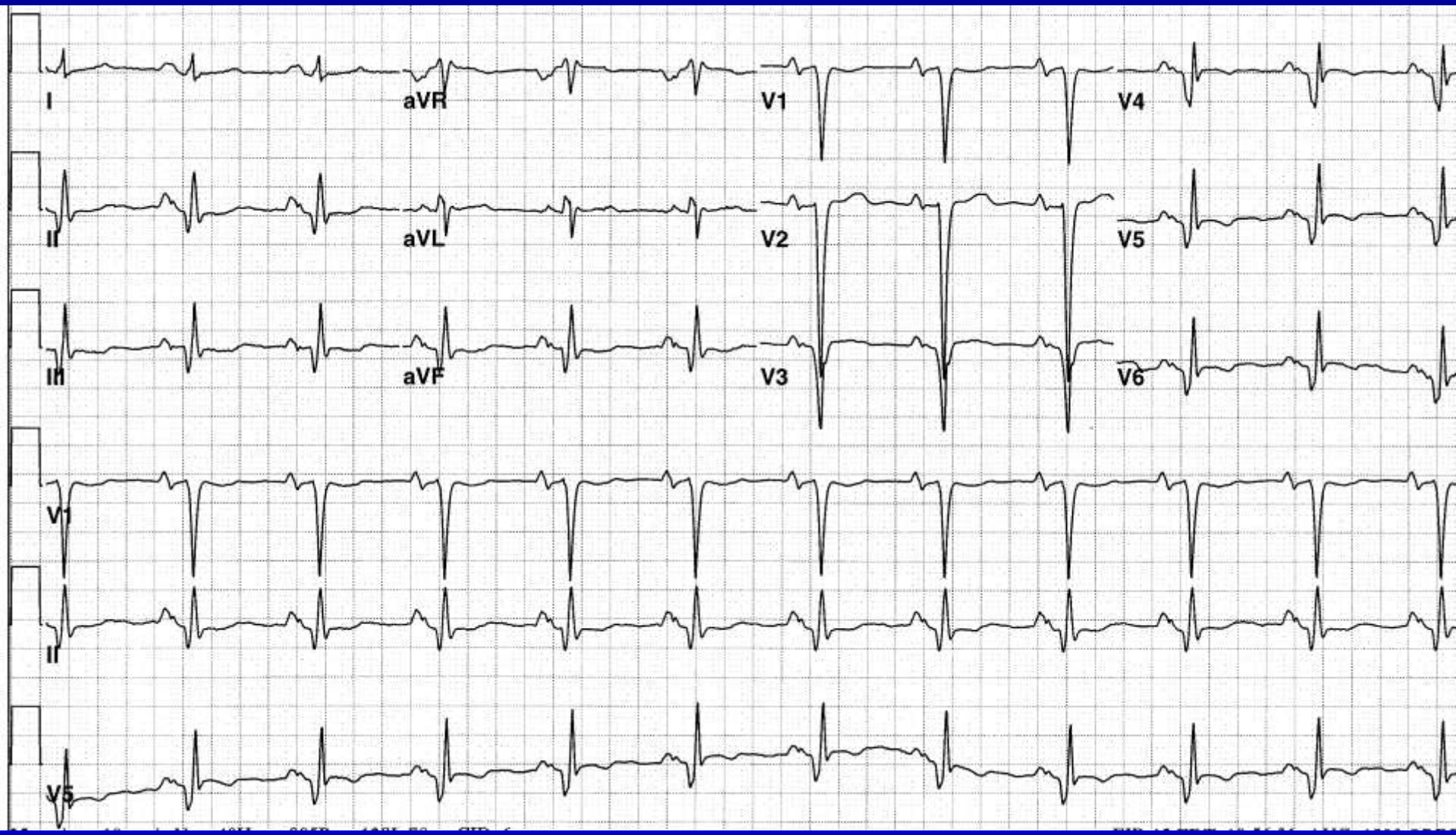
Unknown 5


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
25mm/s
10mm/mV
40Hz
003A-003B
12SL 250
CID: 23
EID:Unconfirmed
EDT:
ORDER

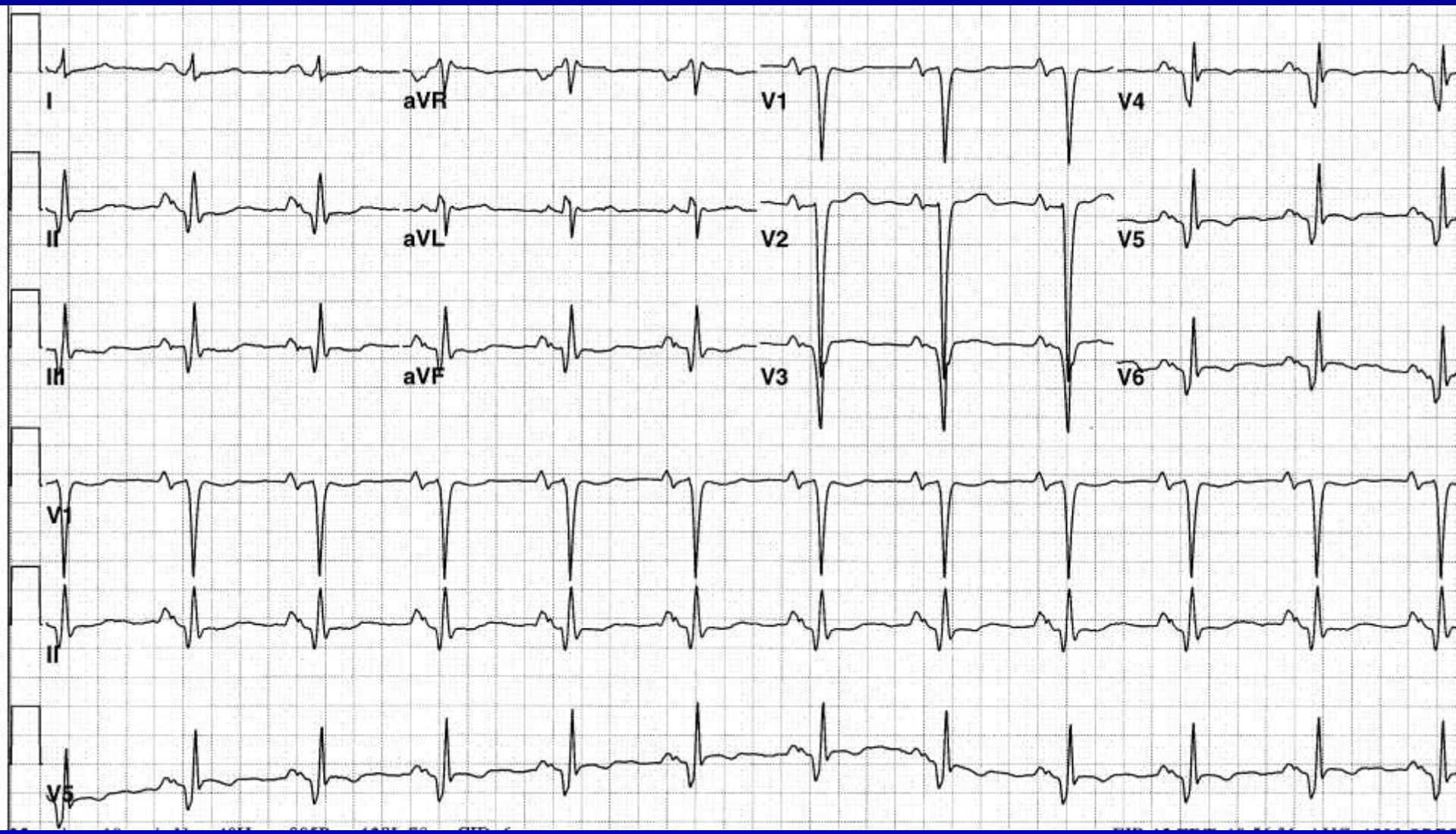
Unknown 5

32 year old woman from renal clinic. Notice leftward axis.
LAE, LVH secondary repolarization changes not fully developed

Unknown 6

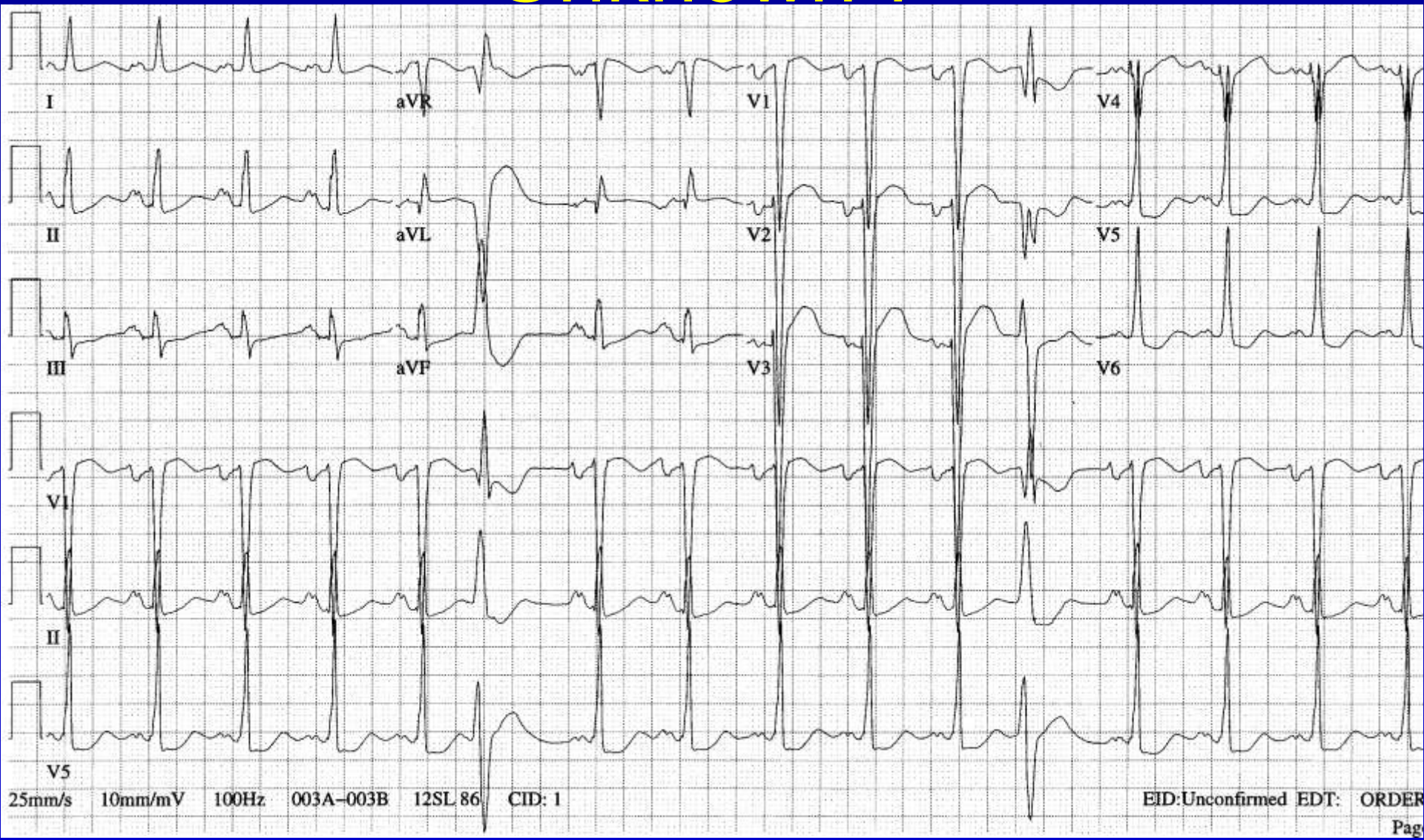


Unknown 6

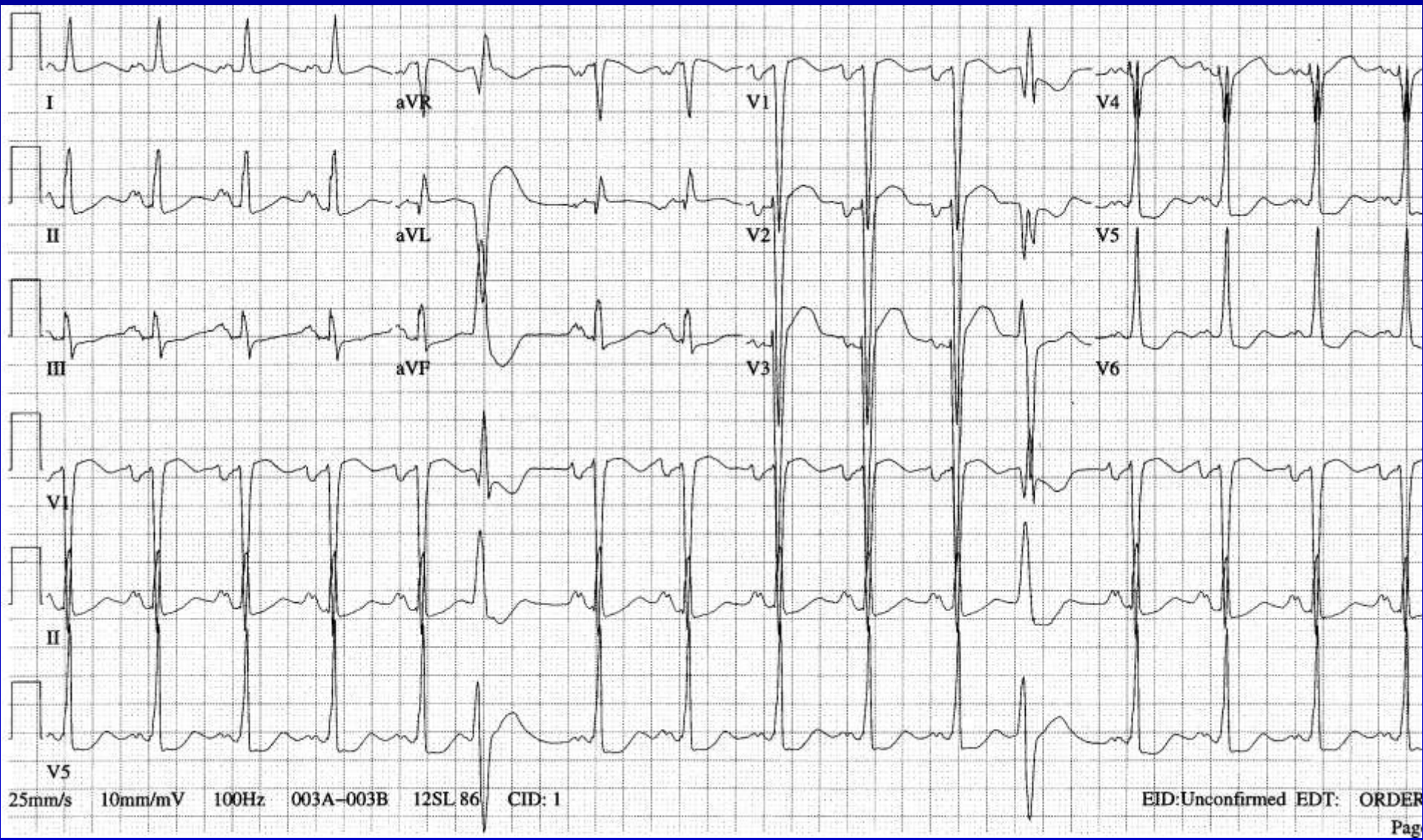


52 year old man with criteria for RAE and inferior MI and anterolateral MI and LVH. Clinically probably LA enlargement

Unknown 7

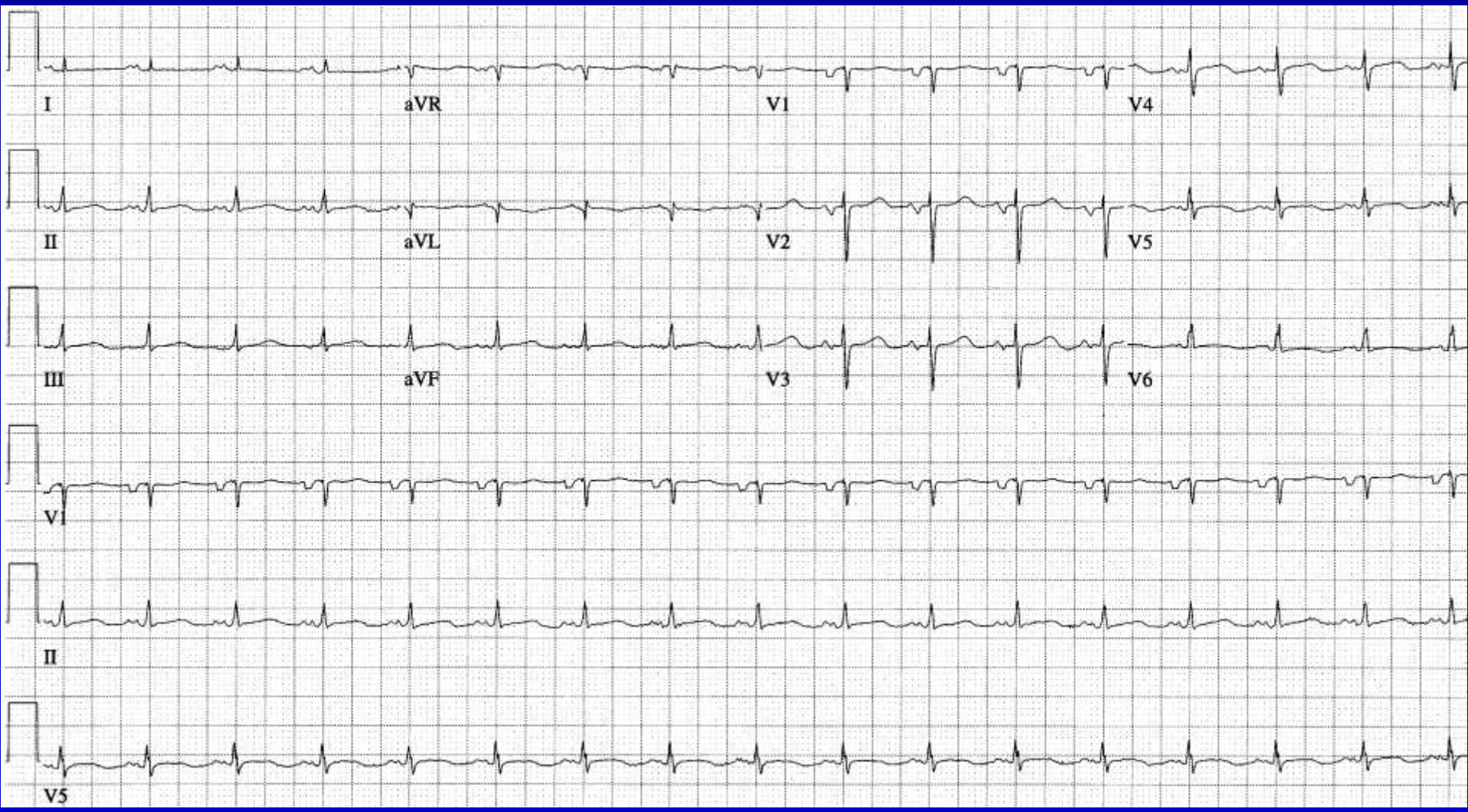

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
25mm/s 10mm/mV 100Hz 003A-003B 12SL 86 CID: 1
EID:Unconfirmed EDT: ORDER

Unknown 7

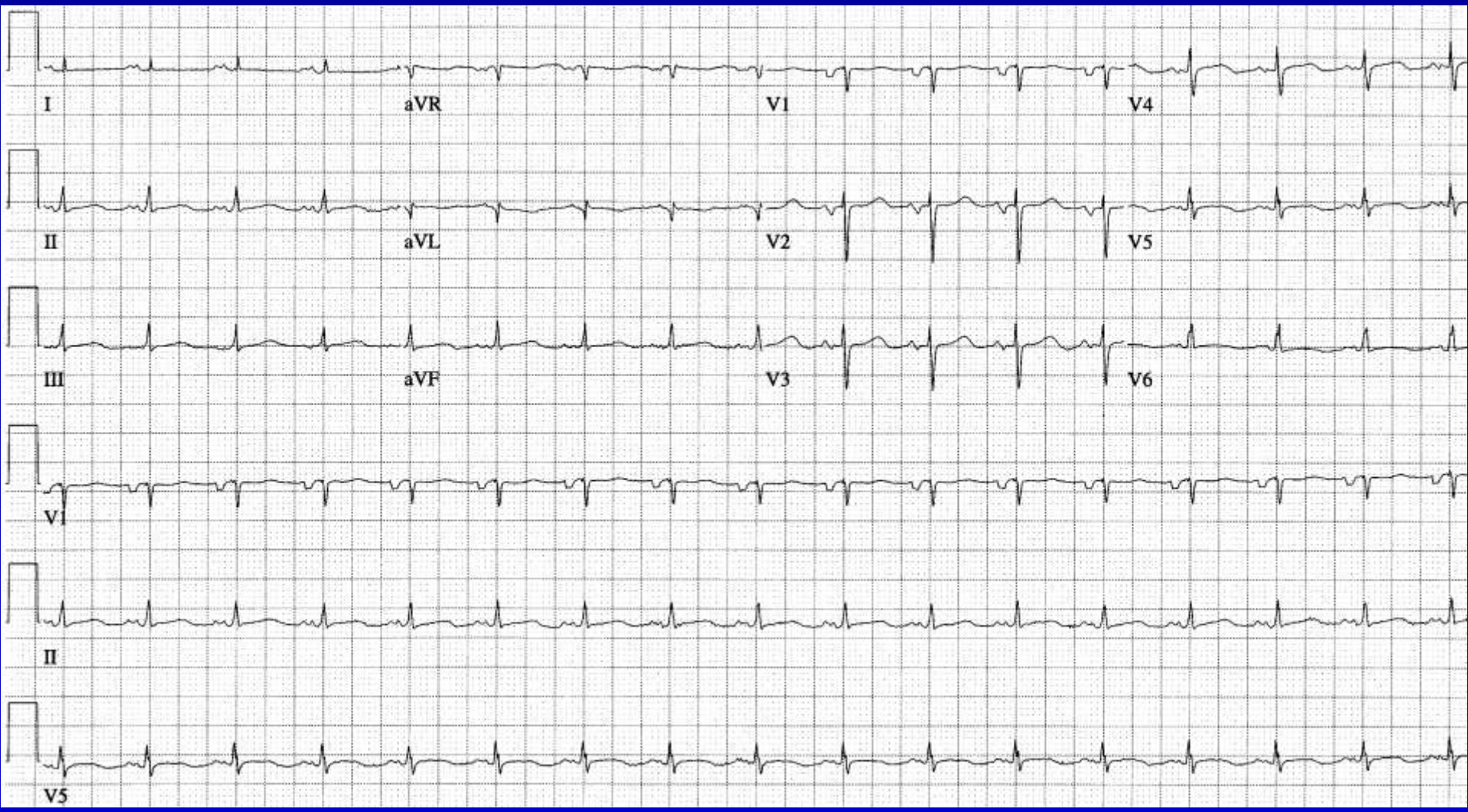


44 yo man in MICU. LAE unequivocal. QRS 0.11, probable significant LVH. Normal standard. Notch in P in II, PVC's ST-T abnormal

Unknown 8

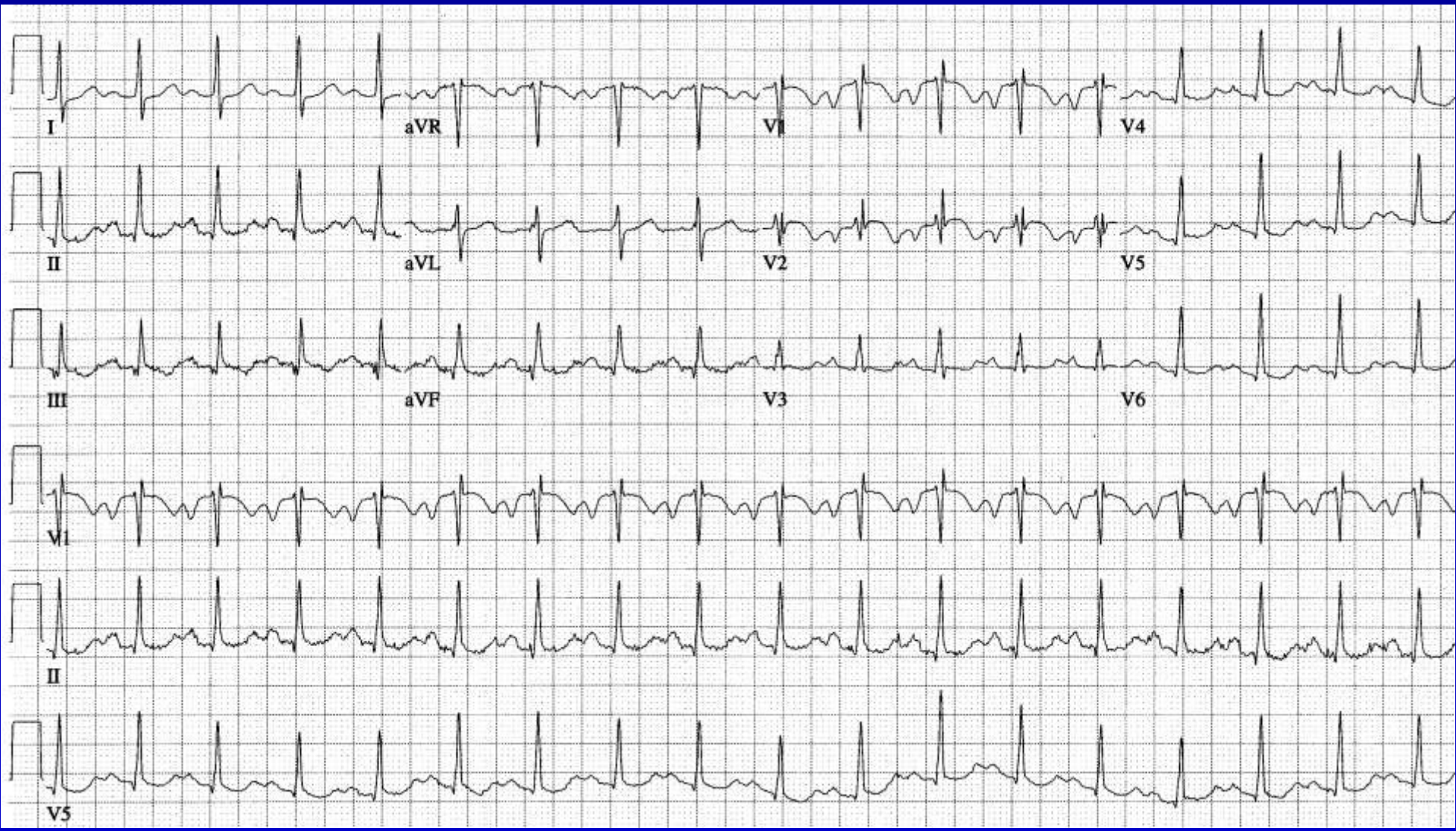


Unknown 8

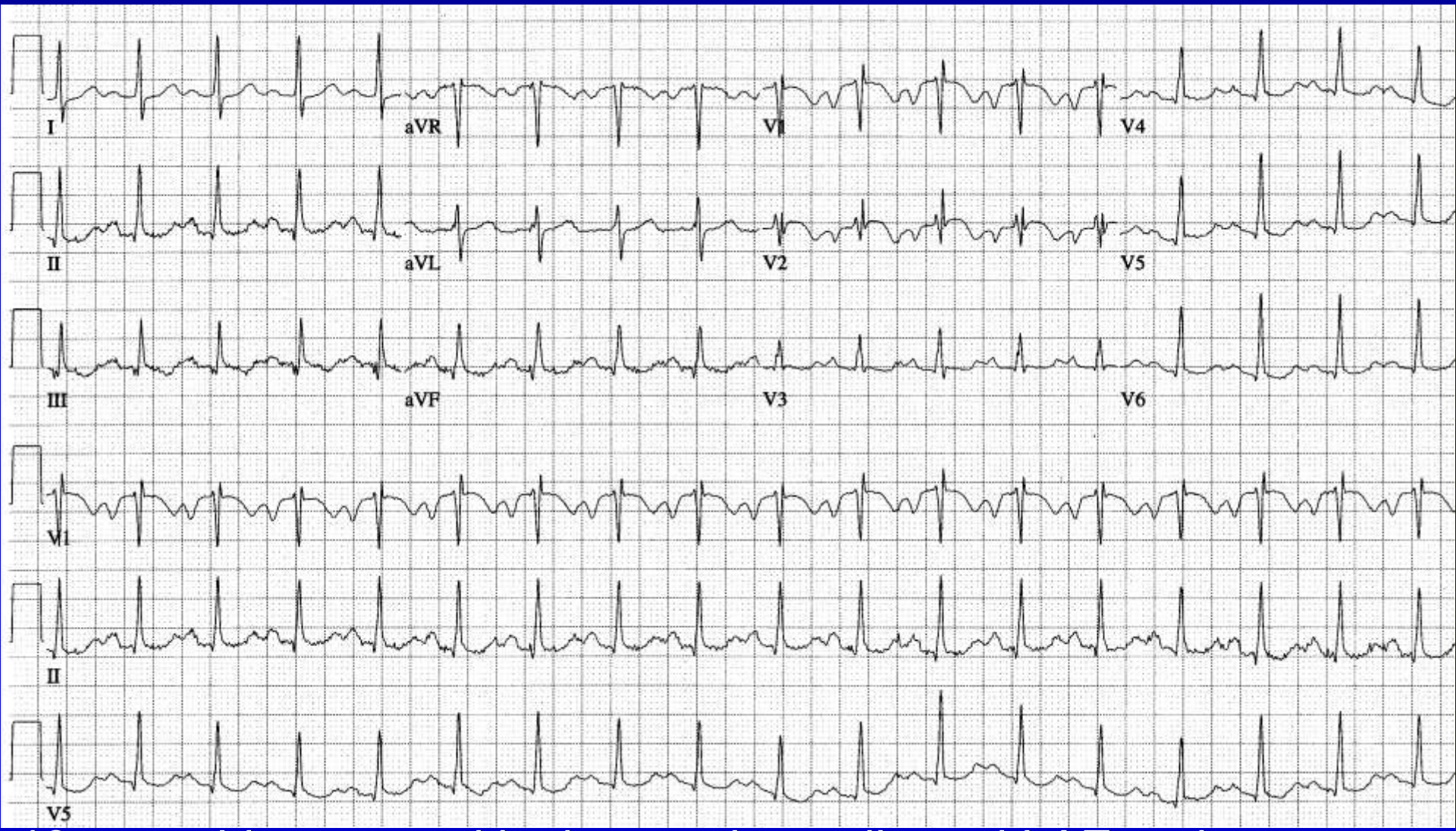


54 yo woman. Clear LAE. No LVH and no clear RVH, but QT is long

Unknown 9

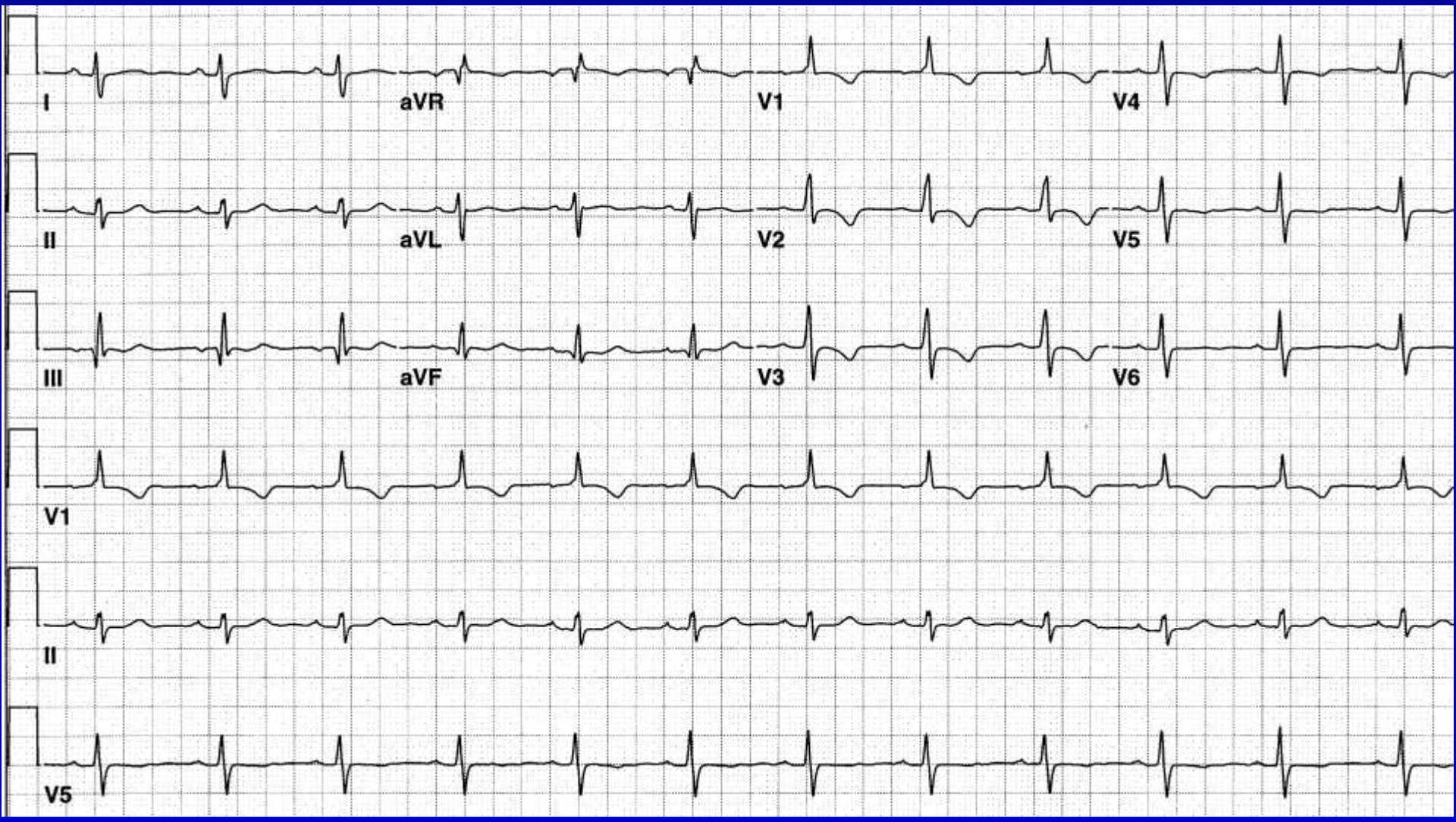


Unknown 9

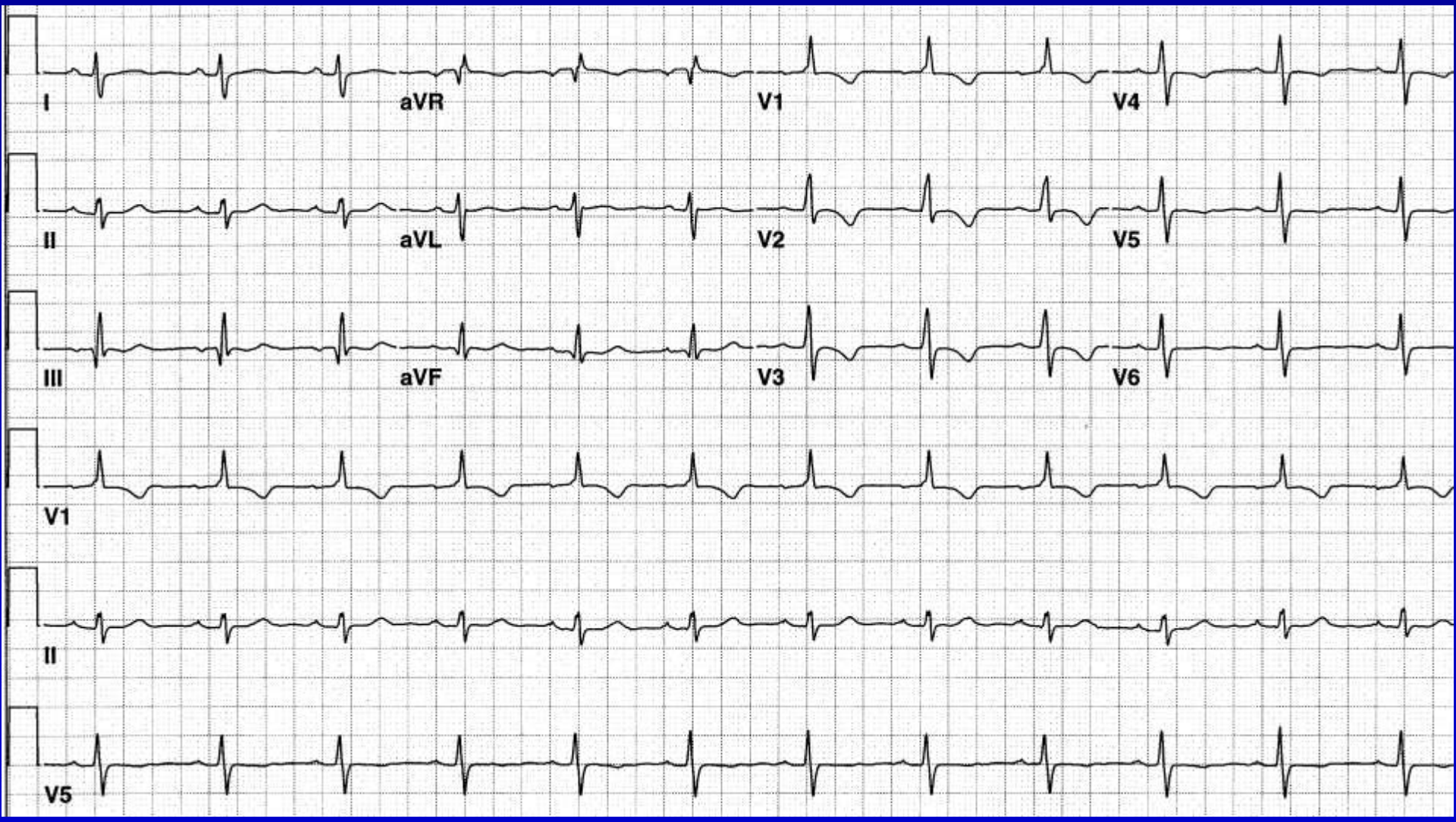


46 year old woman with sinus tachycardia and LAE and nonspecific ST and T wave abnormality and QRS duration of 0.09

Unknown 10

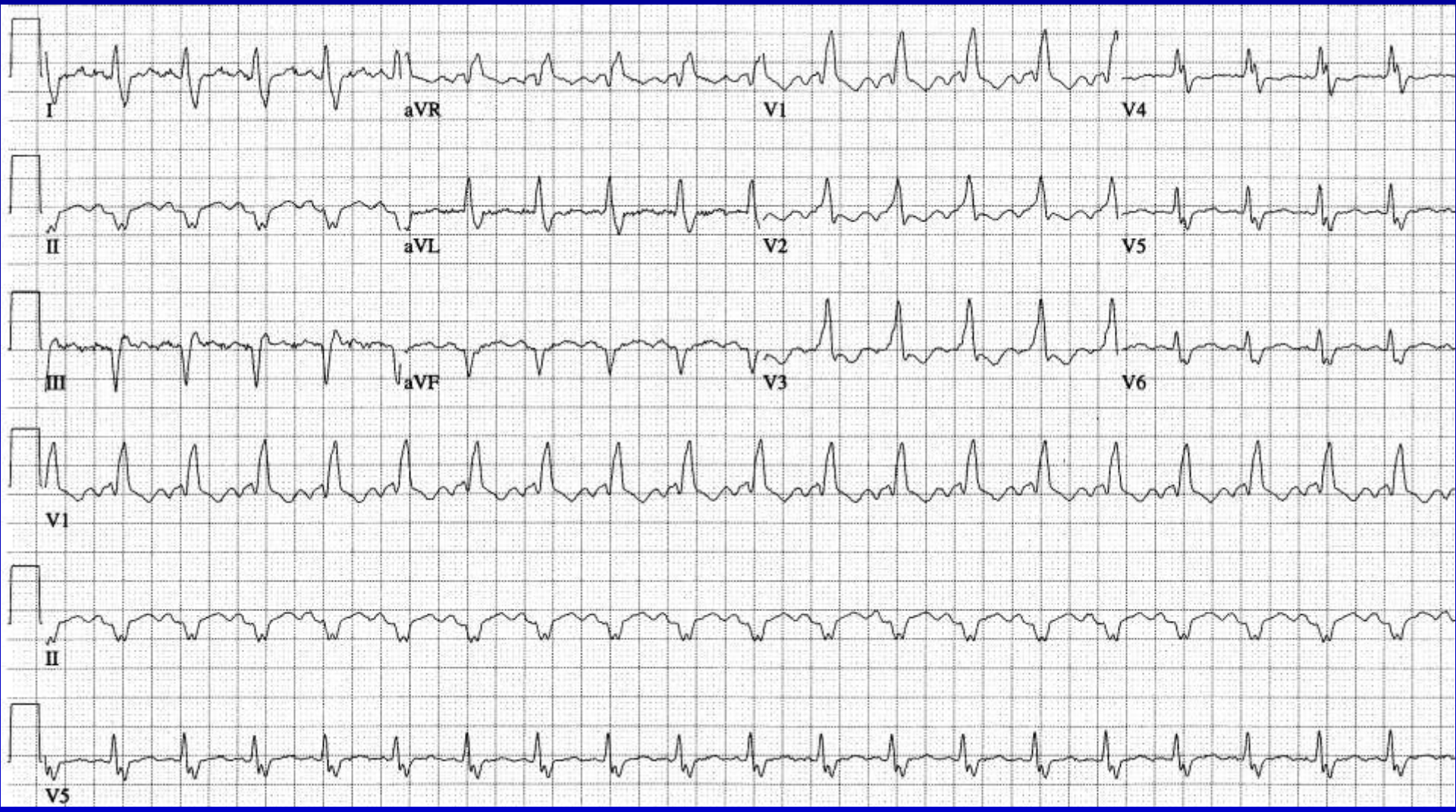


Unknown 10

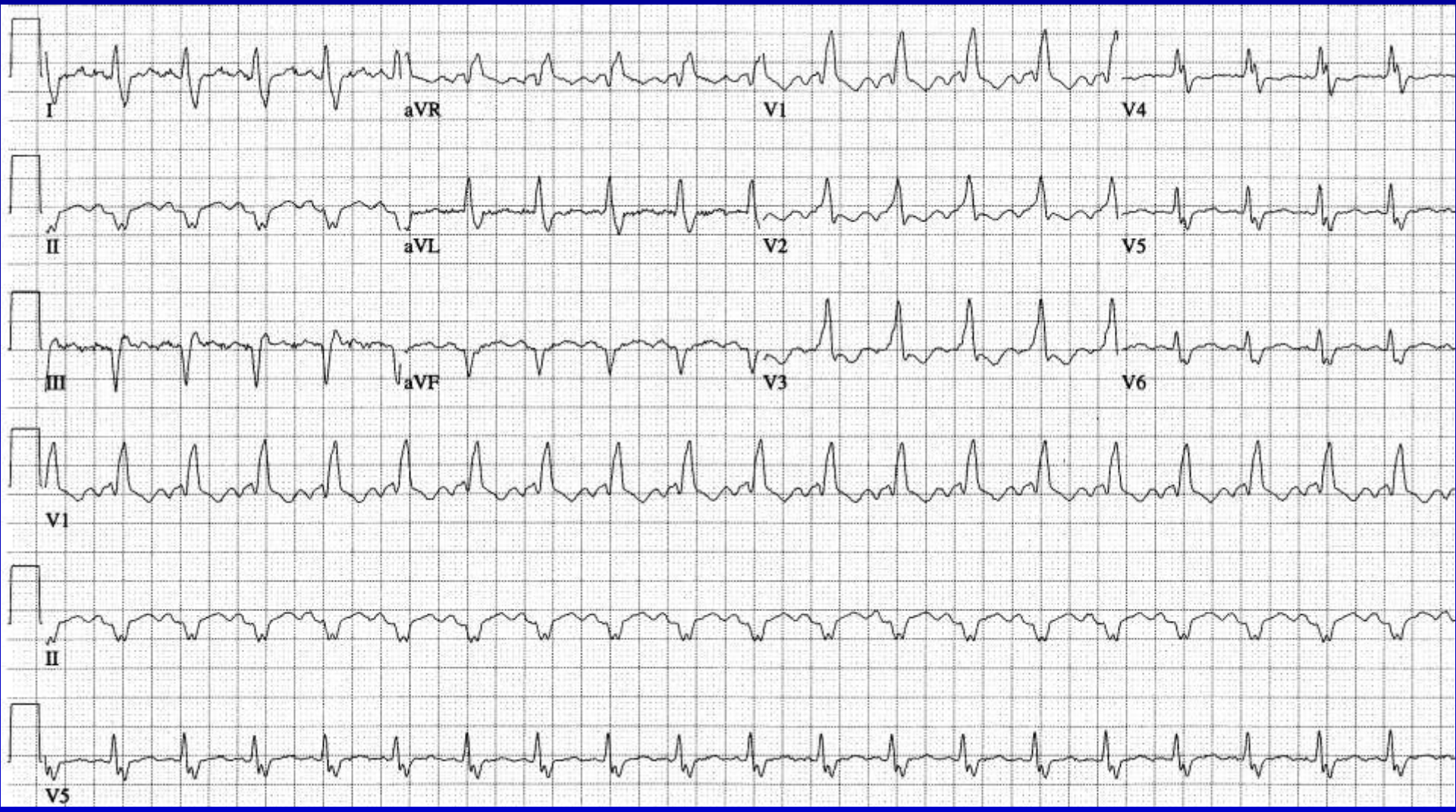


38 year old woman, 62 inches, 200 lb, read as inferoposterior MI
Actually RVH without RAE or LAE. This would be type A-B

Unknown 11

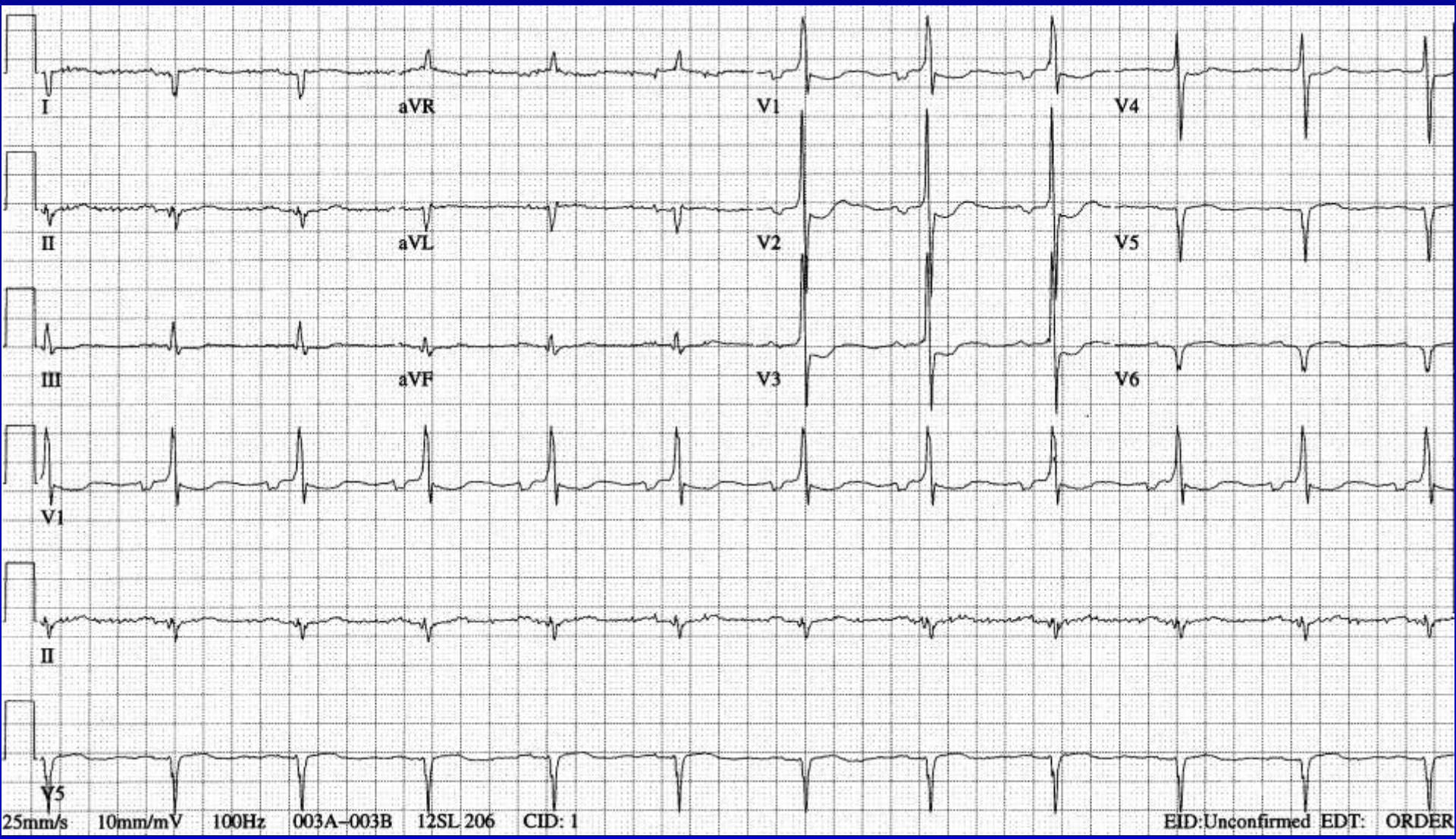


Unknown 11

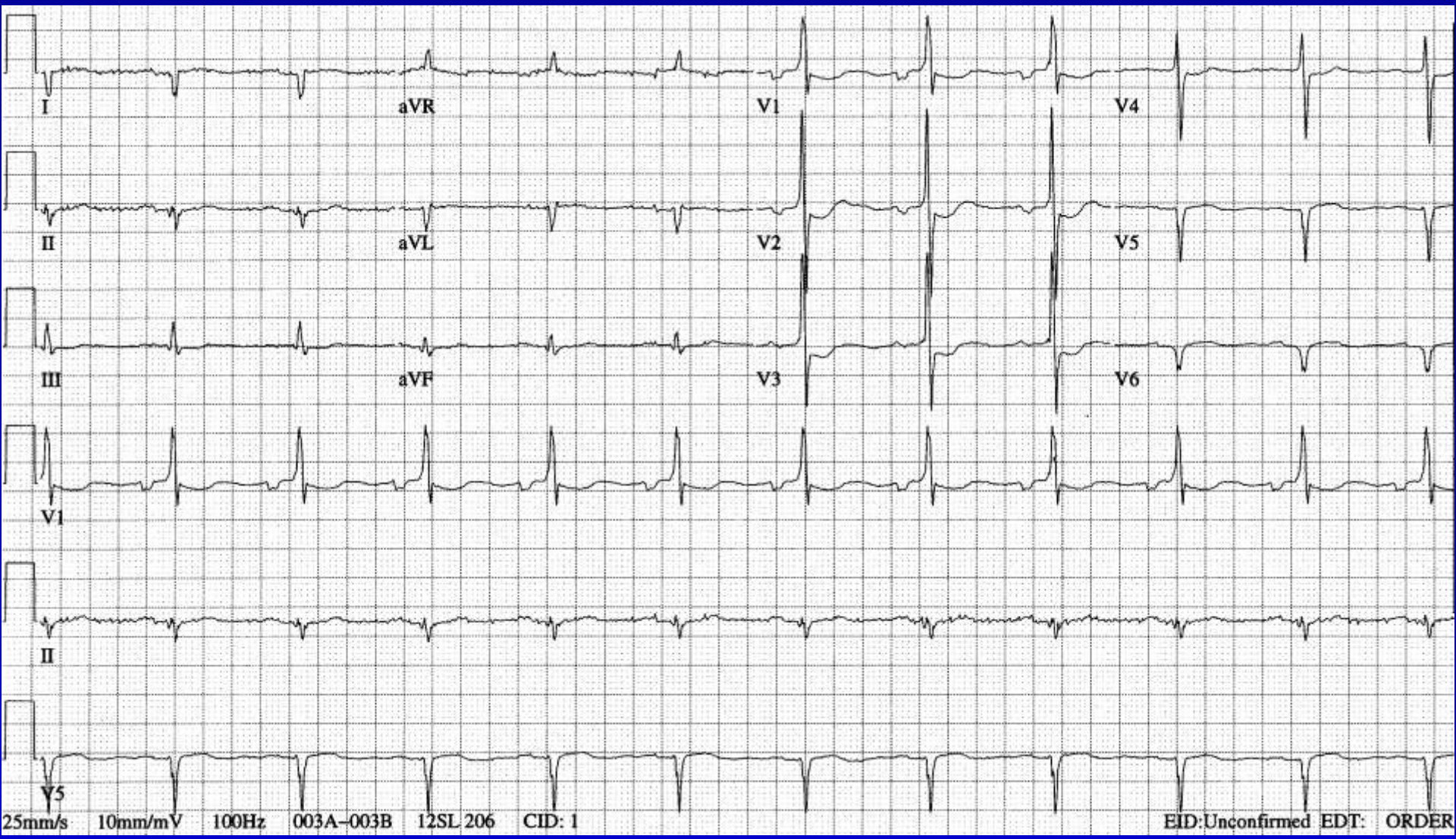


49 year old man with chest pain in the emergency department. Sinus tachycardia (not flutter), LAE, RBBB, ?inferior MI, ?posterior MI

Unknown 12

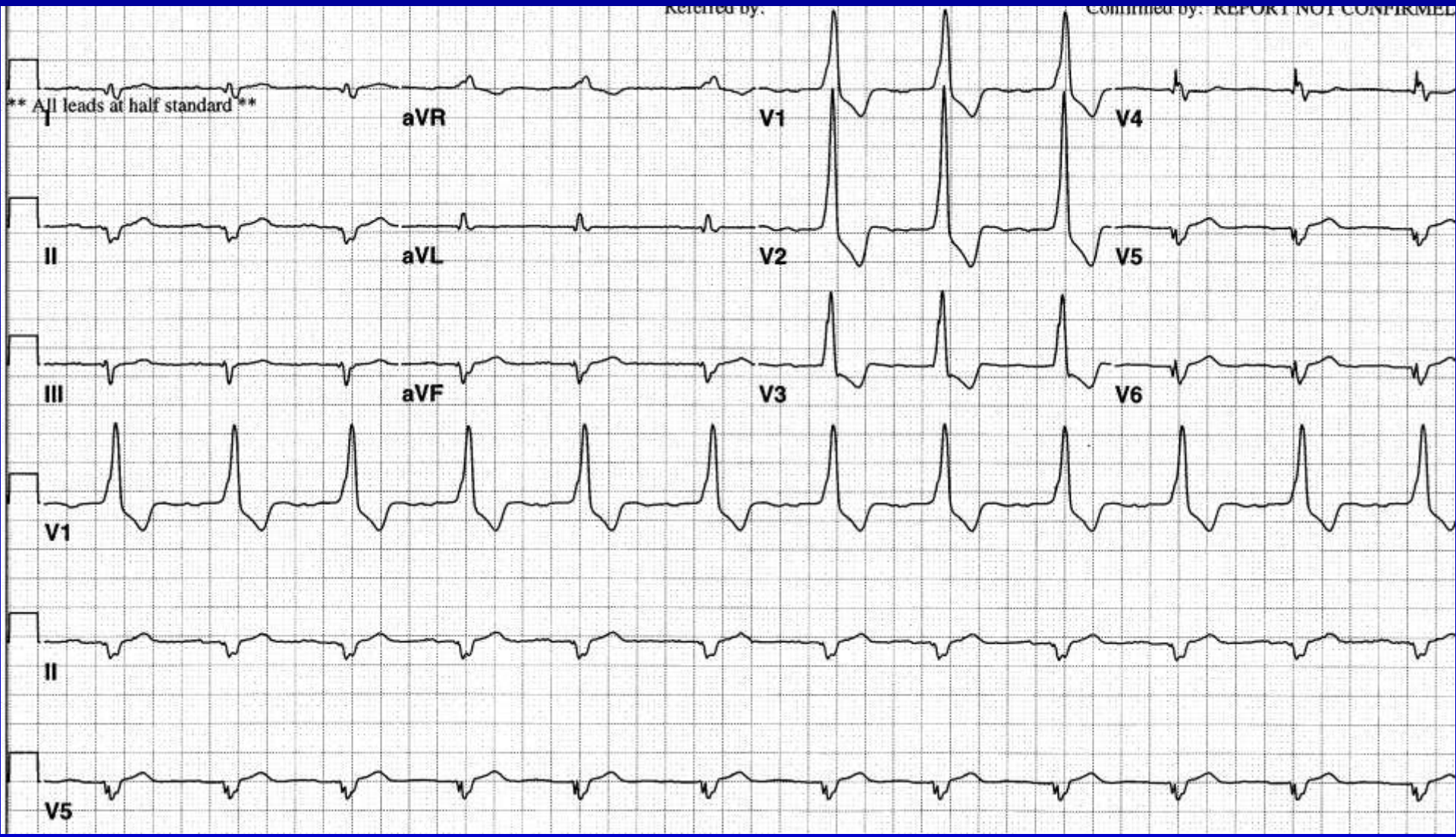

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
25mm/s 10mm/mV 100Hz 003A-003B 12SL 206 CID: 1
EID:Unconfirmed EDT: ORDER

Unknown 12



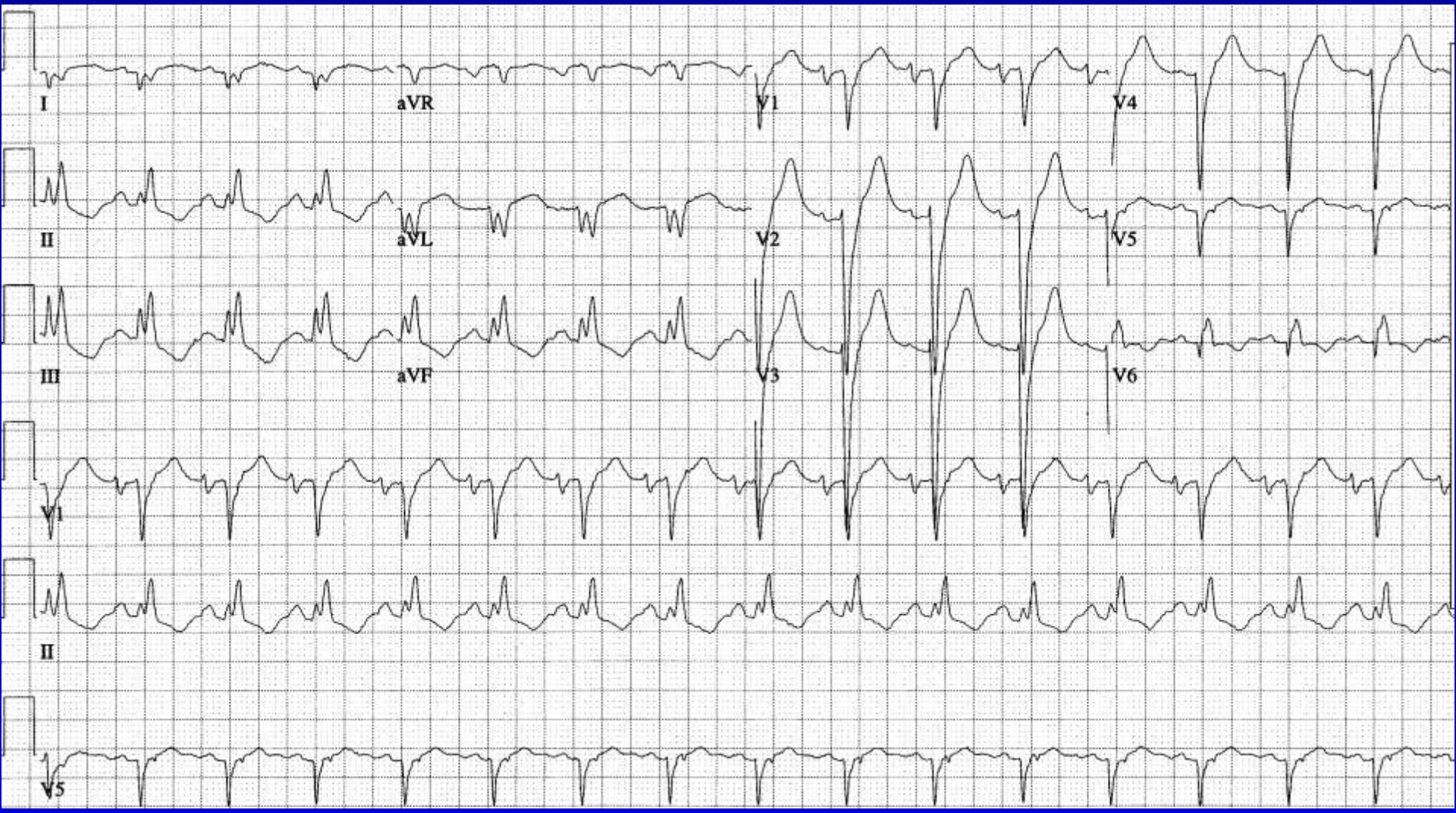
58 year old man with LAE and posterolateral MI, not RVH, because in I is a QS not an RS. Later developed RBBB too!

Unknown 12

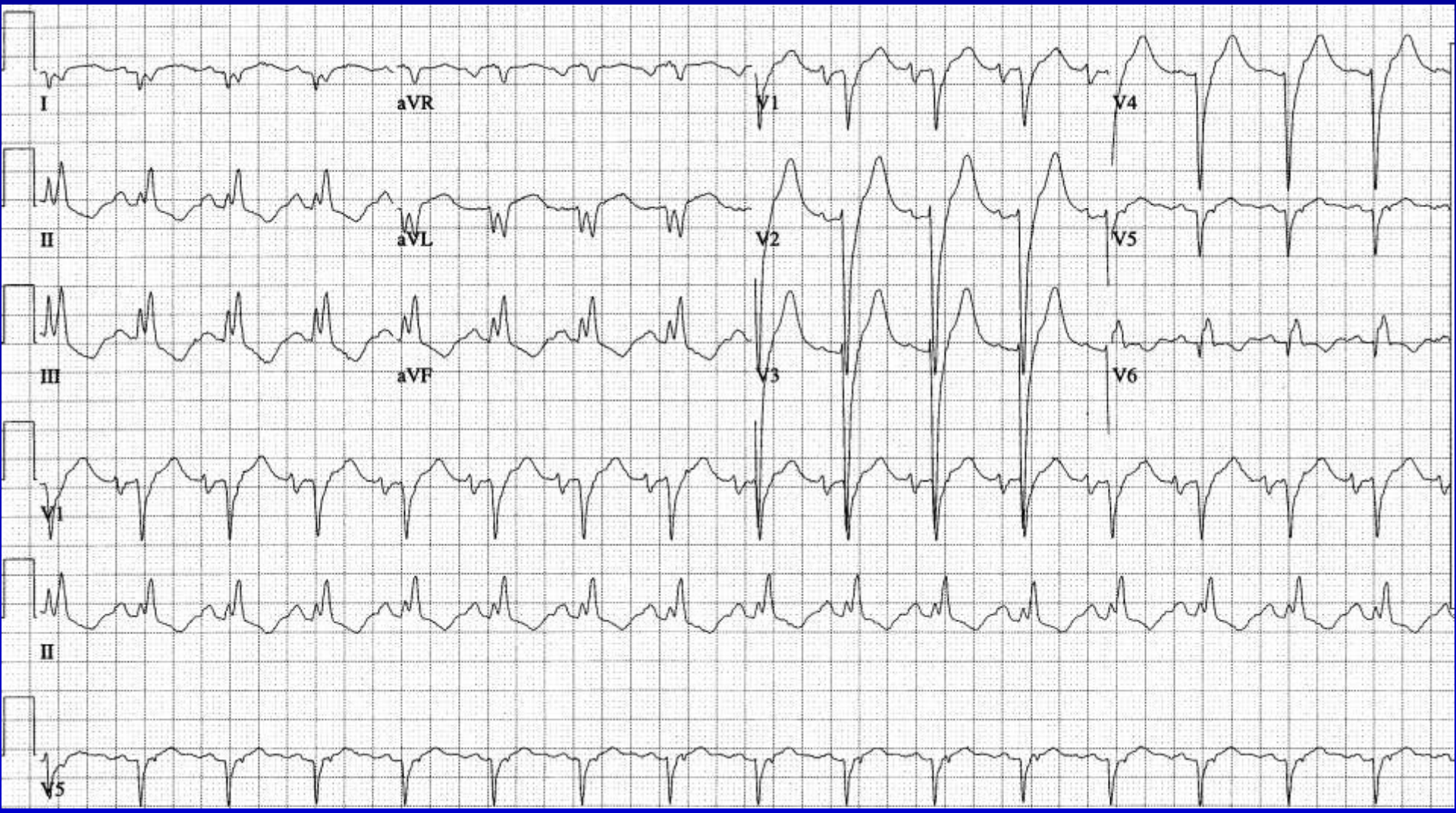


58 year old man with LAE and posterolateral MI, not RVH, because in I is a QS not an RS. Later developed RBBB too!, now all leads at half std

Unknown 13

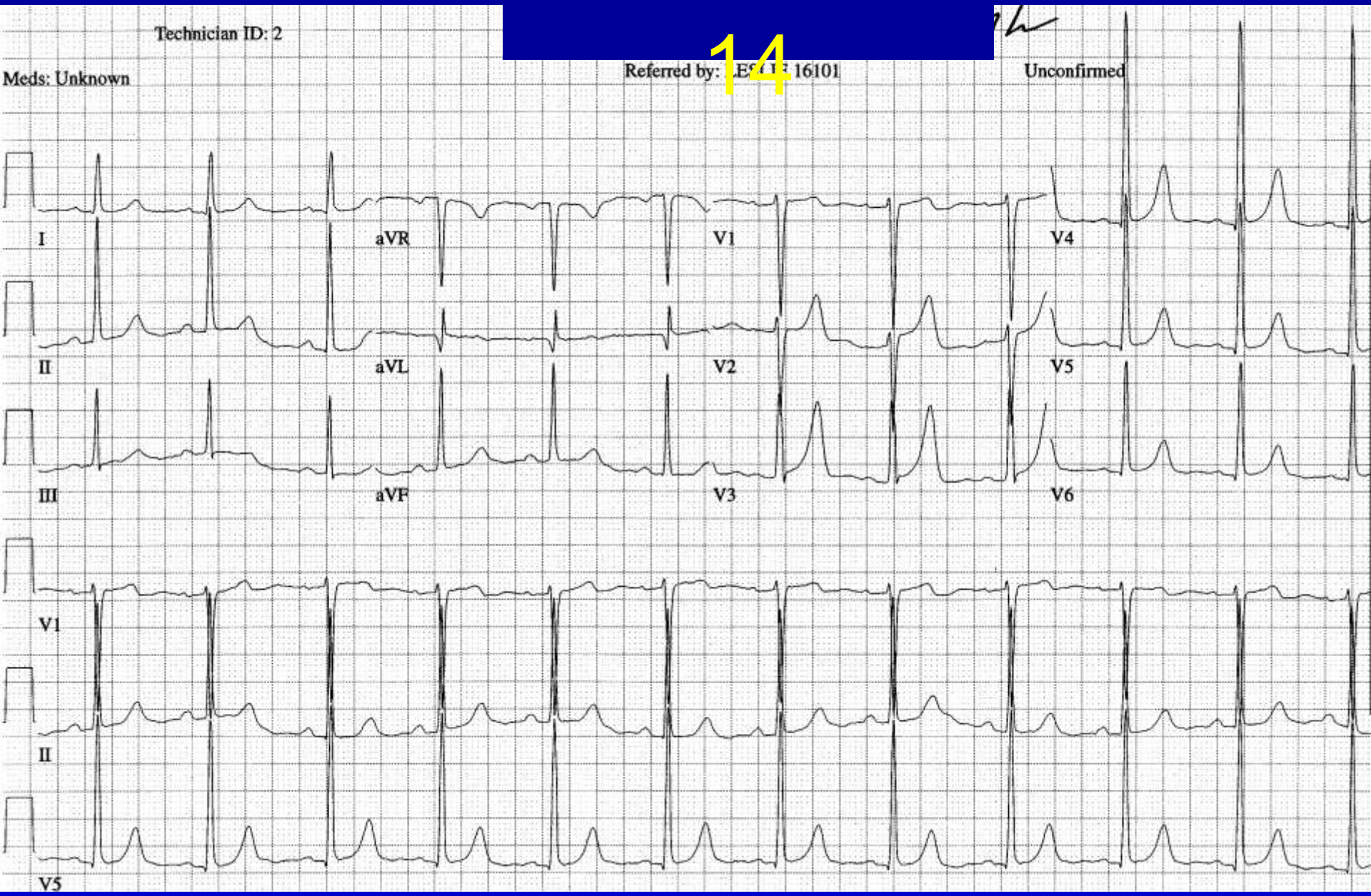


Unknown 13

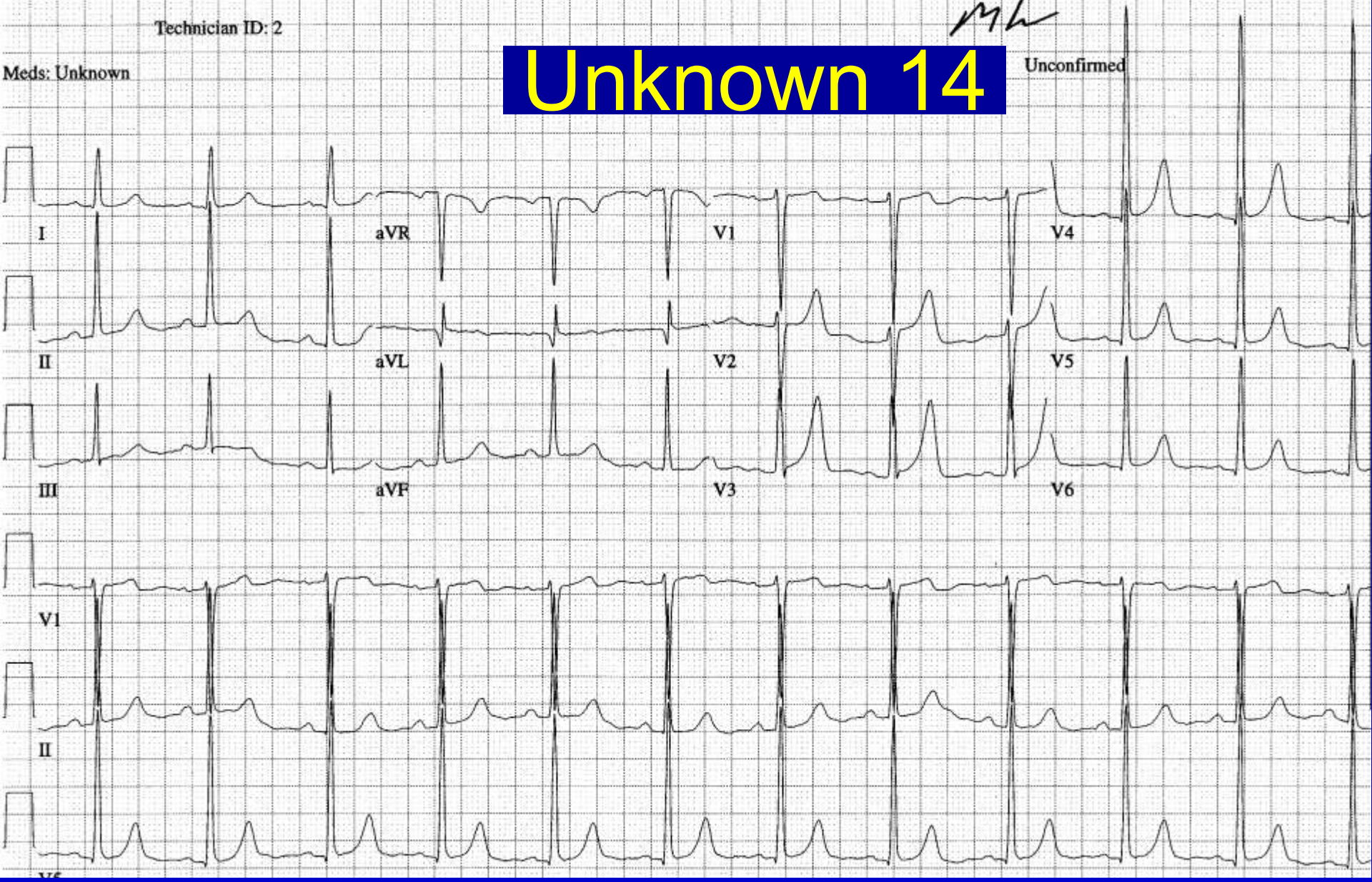


LAE and IVCD. IVCD looks like RBBB in I, but LBBB in V1, large voltage but no LVH because of IVCD. Anterolateral MI? CHF?

Unknown 14

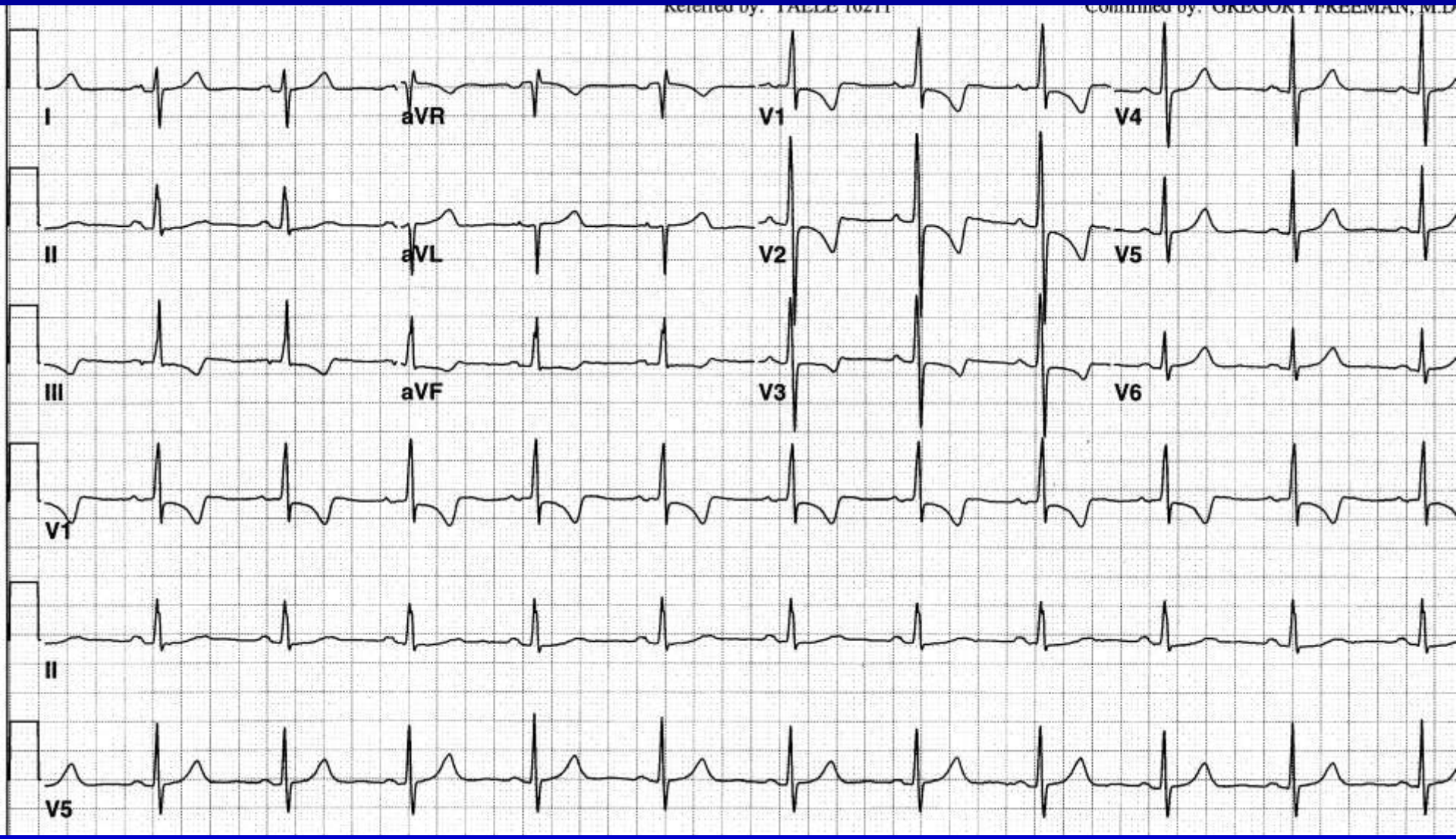

Technician ID: 2
Meds: Unknown
Referred by:
16101
Unconfirmed
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5

Unknown 14

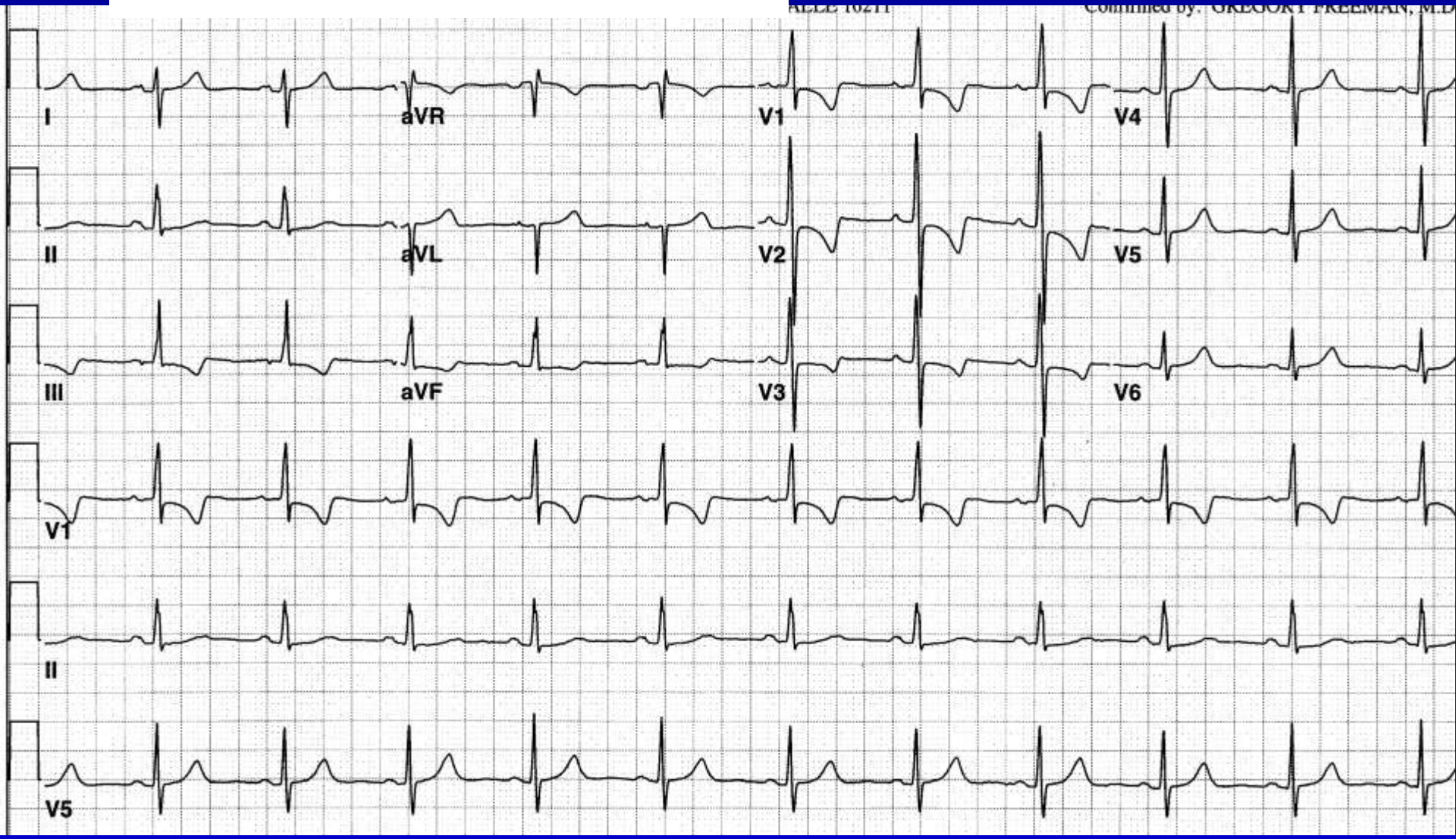


37 year old man, QRS duration 0.10. R peak time in V6 is 0.05 and in V5 is 0.05. LVH? – I think so, but not by strict application because he is less than 40 years old.

Unknown 15

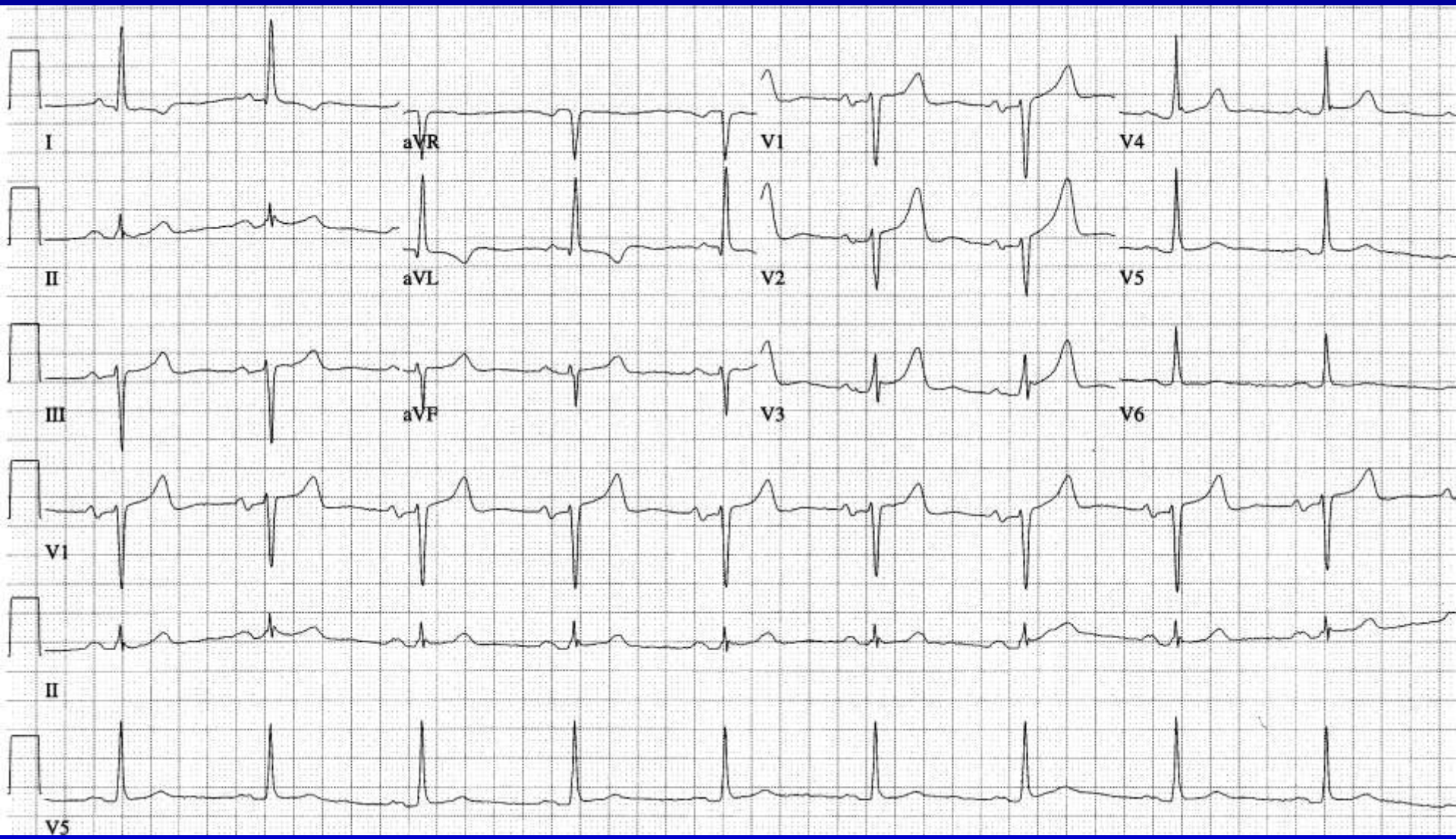


Unknown 15

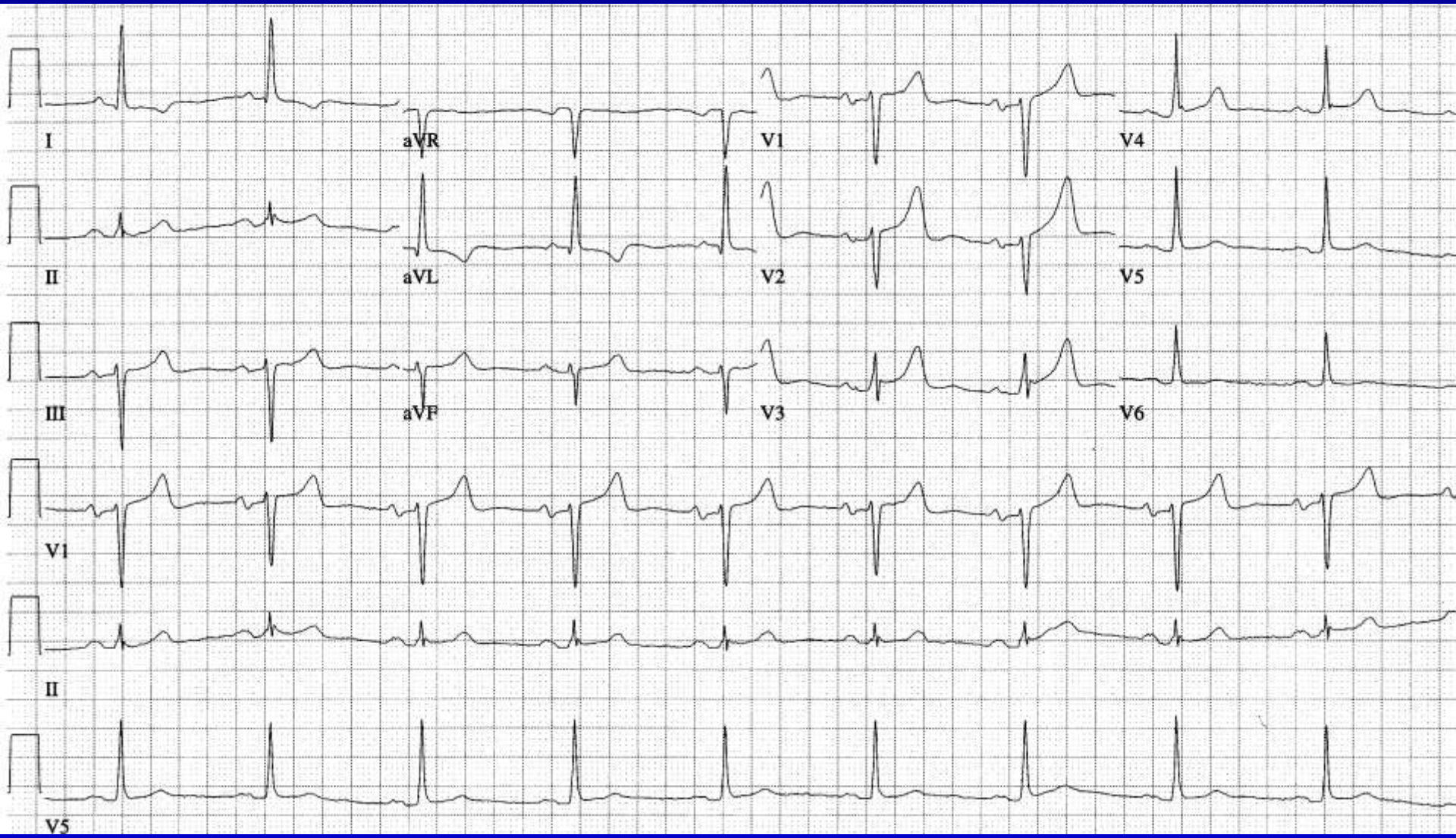


45 year old woman with no atrial abnormality, but right axis deviation and classic abnormality in V1 with T inversion – RVH. 4 months earlier the RVH was type A and there was also RAE ?embolism

Unknown 16

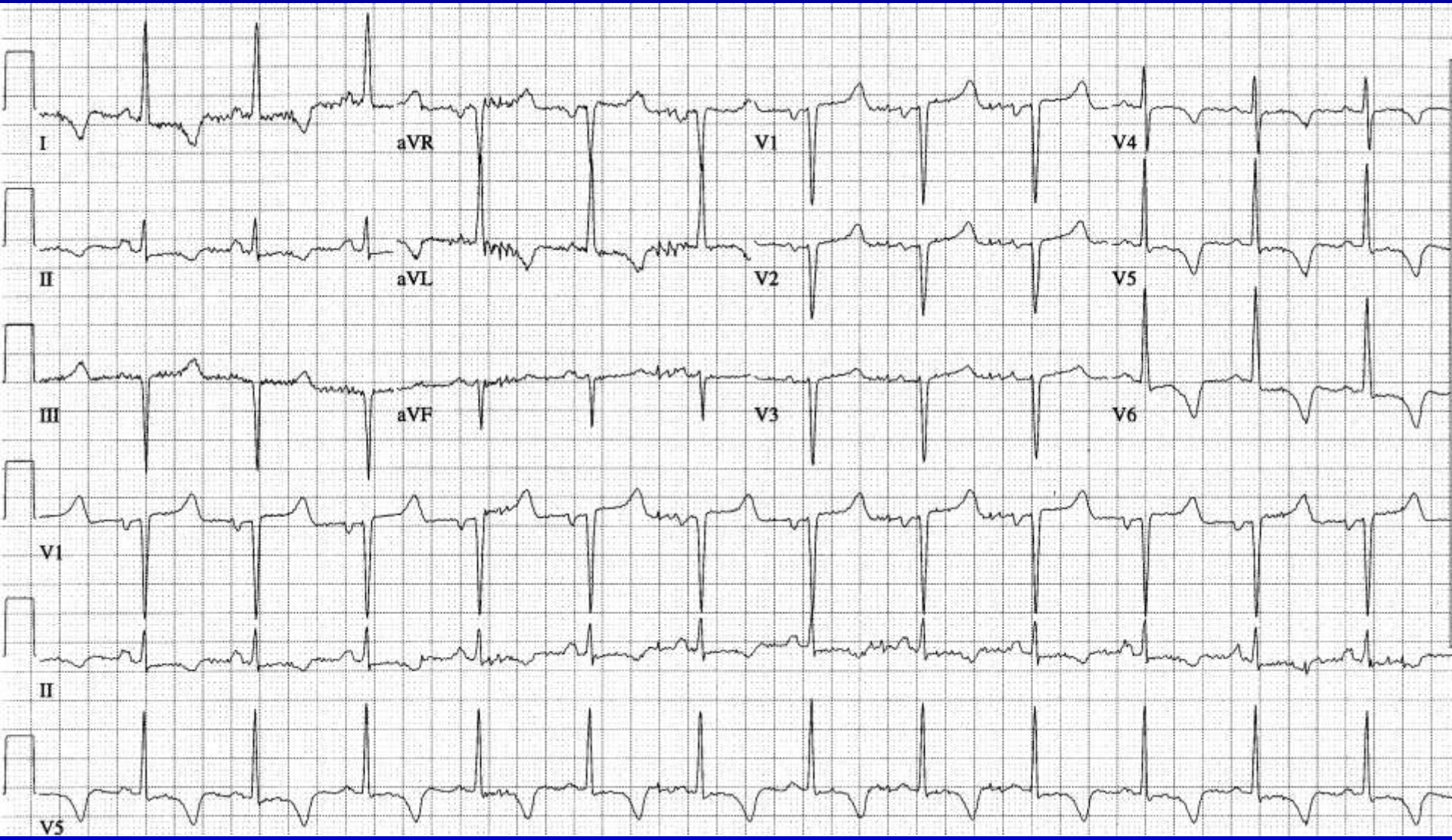


Unknown 16

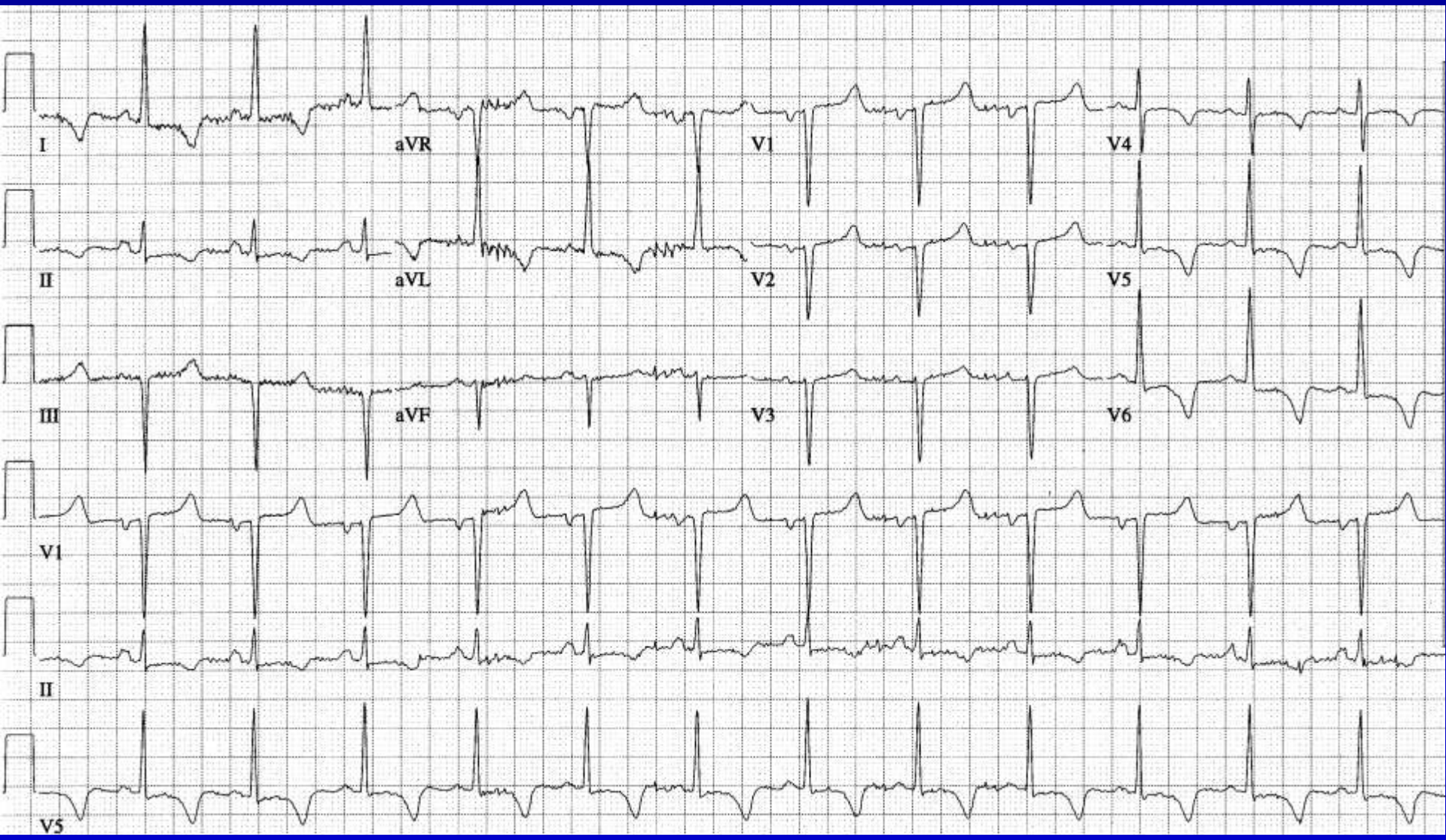


56 year old man in preparation for outpatient ETT! Sokolow-Lyon criterion of 25 mm I and III and repolarization abnormality and QRS duration of 0.10 and LAE and normal R peak time

Unknown 17

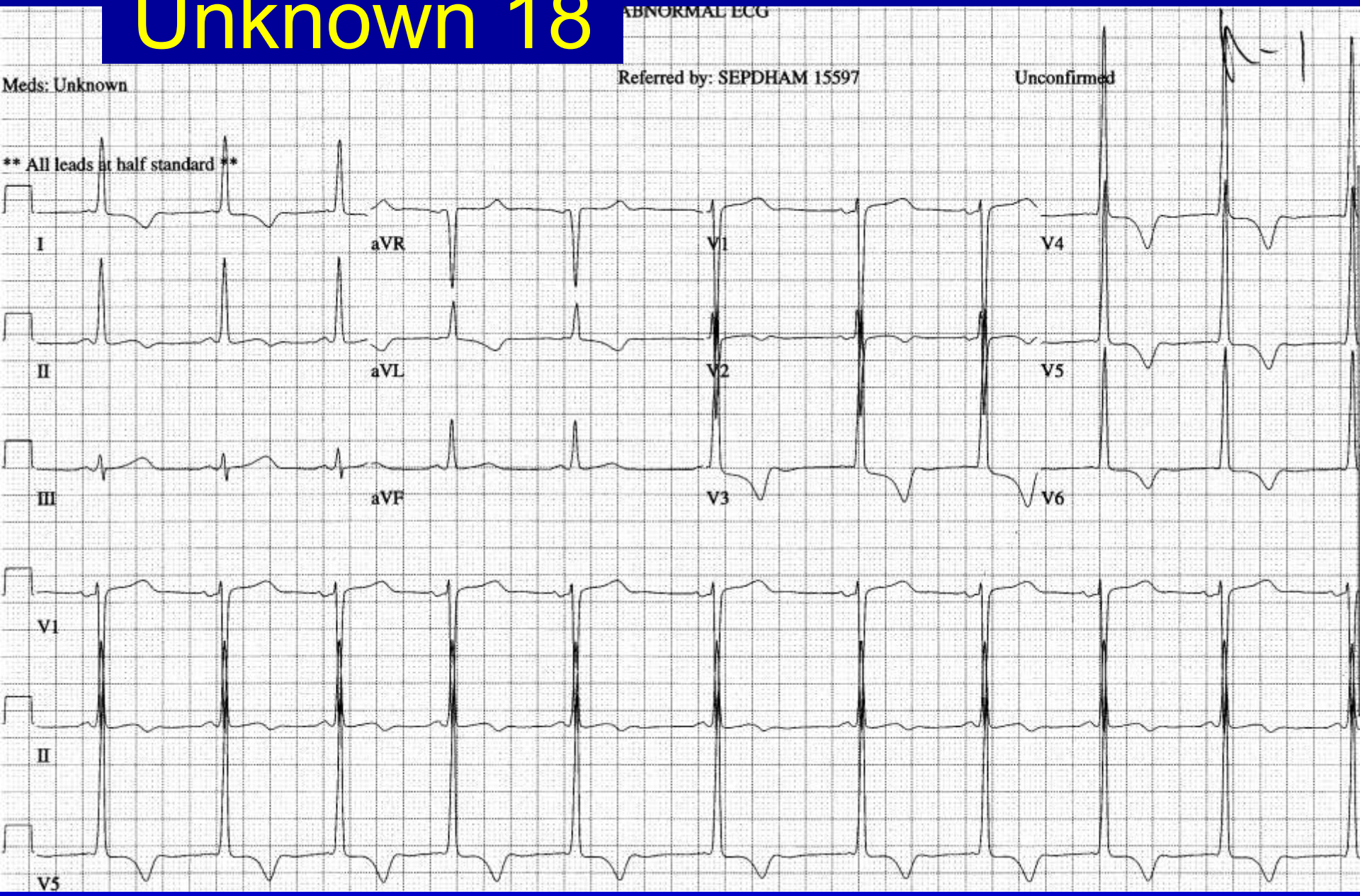


Unknown 17

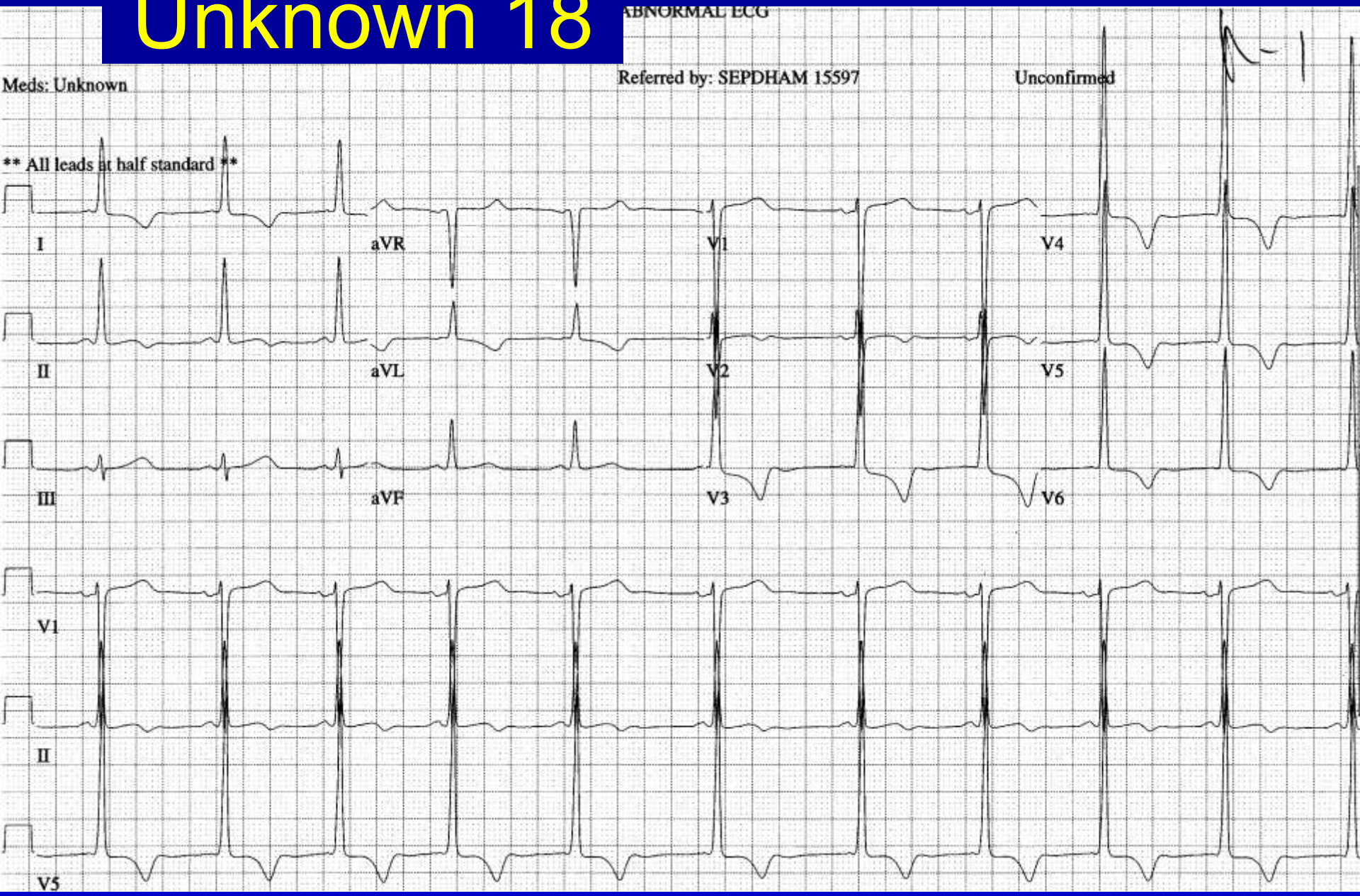


64 year old woman with LAE and LVH by Sokolow-Lyon or Cornell and dramatic repolarization abnormality, QRS duration 0.07. Voltage lower 4 years later with QRS duration 0.11.

Unknown 18

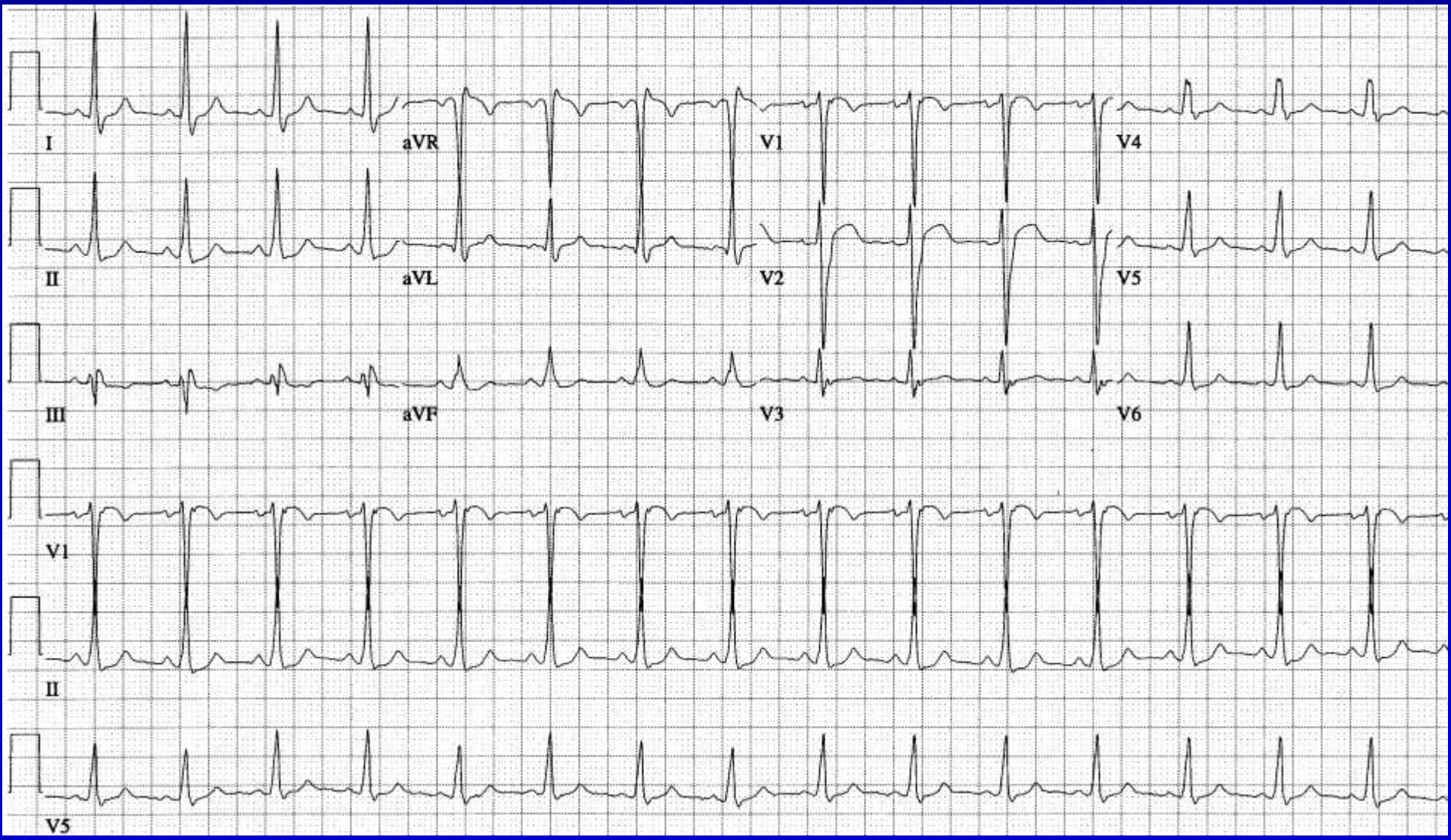

ABNORMAL ECG
Meds: Unknown
Referred by: SEPDHAM 15597
Unconfirmed
** All leads at half standard **
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5

Unknown 18

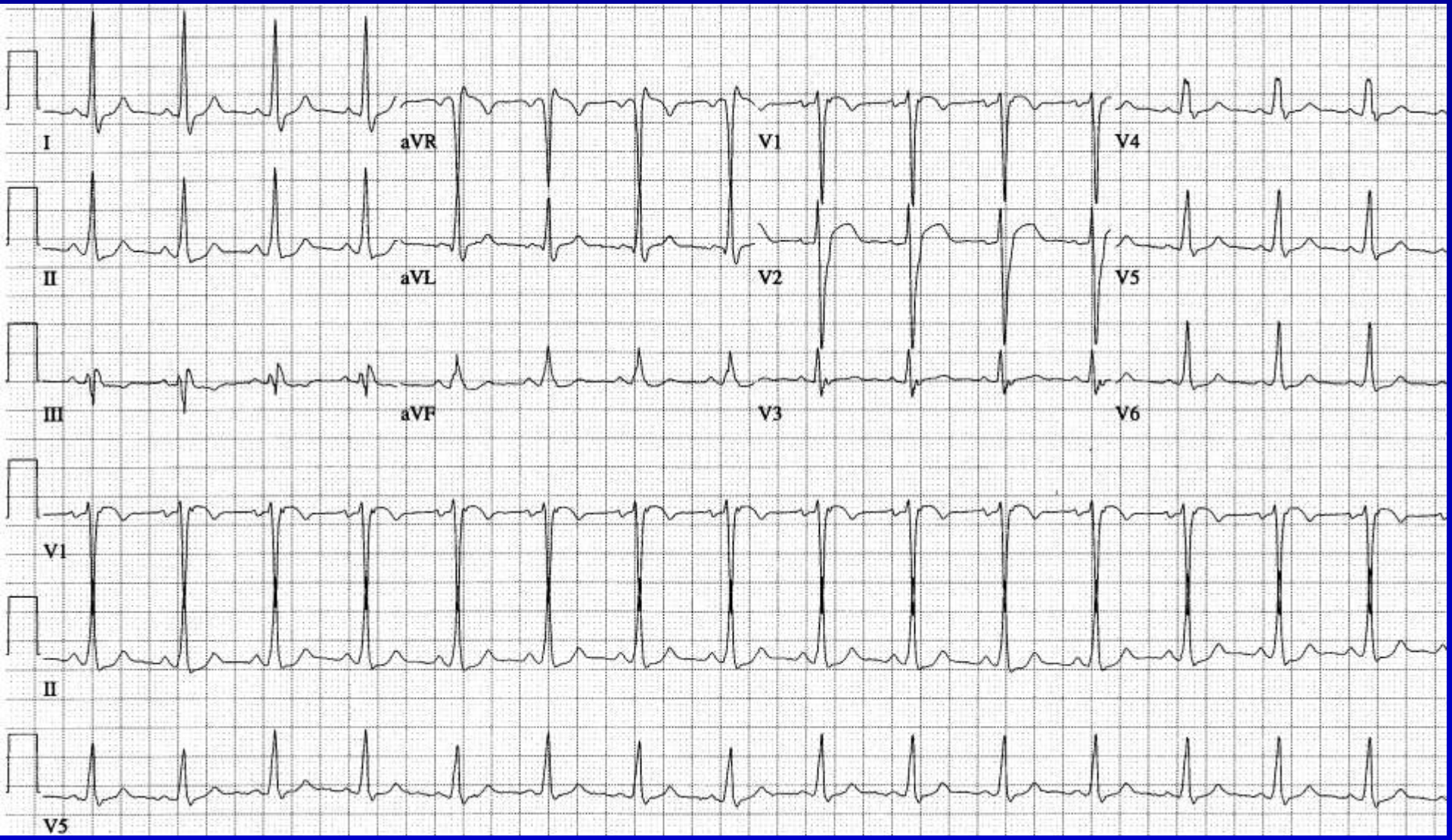


Half standard. World class LVH. 44 year old man.

Unknown 19

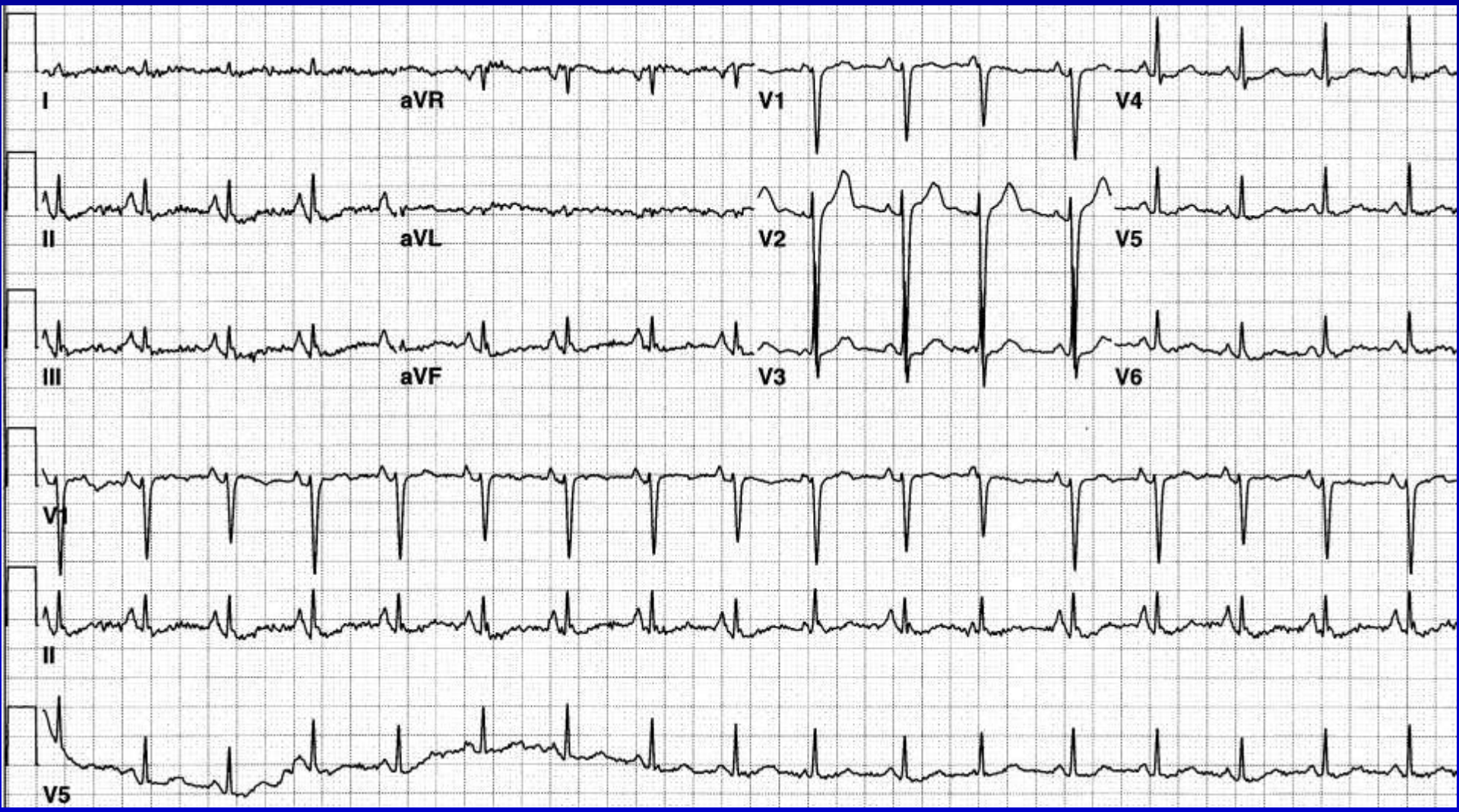


Unknown 19

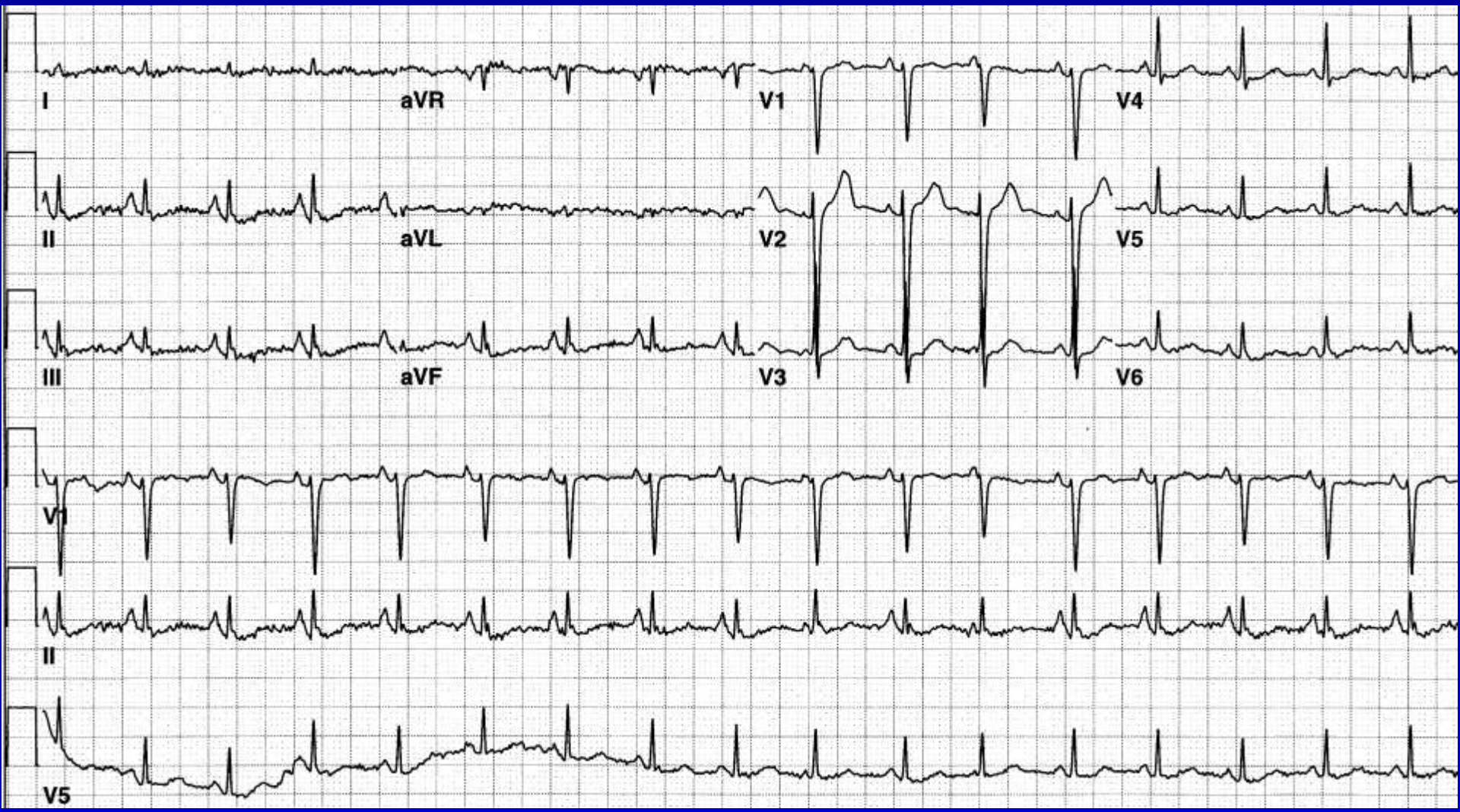


35 year old woman, echo 8 months prior mild LVH and mildly low EF. LVH by Sokolow-Lyon, but less than 40 years old. QRS 0.12, R peak time abnormal in V5

Unknown 20

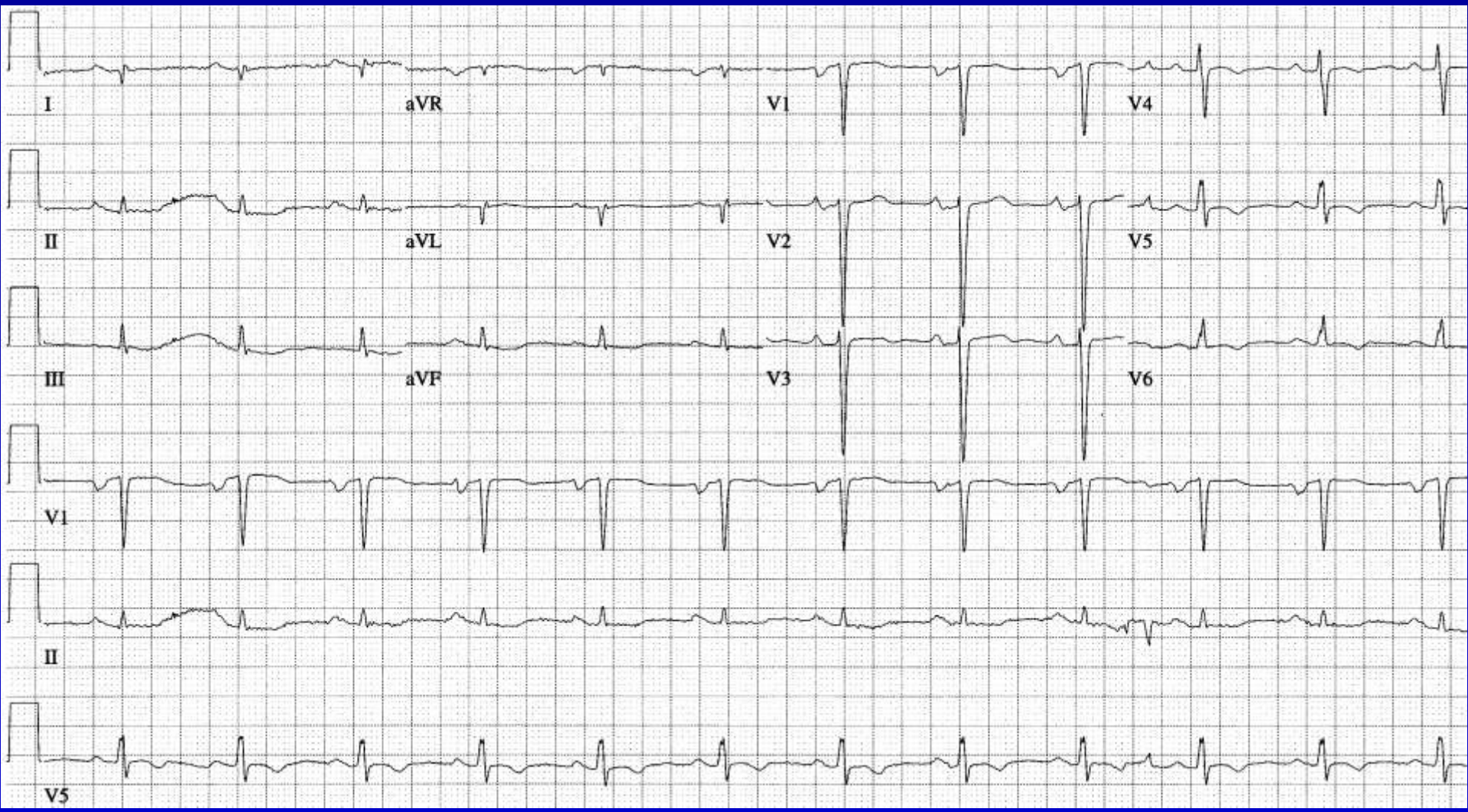


Unknown 20

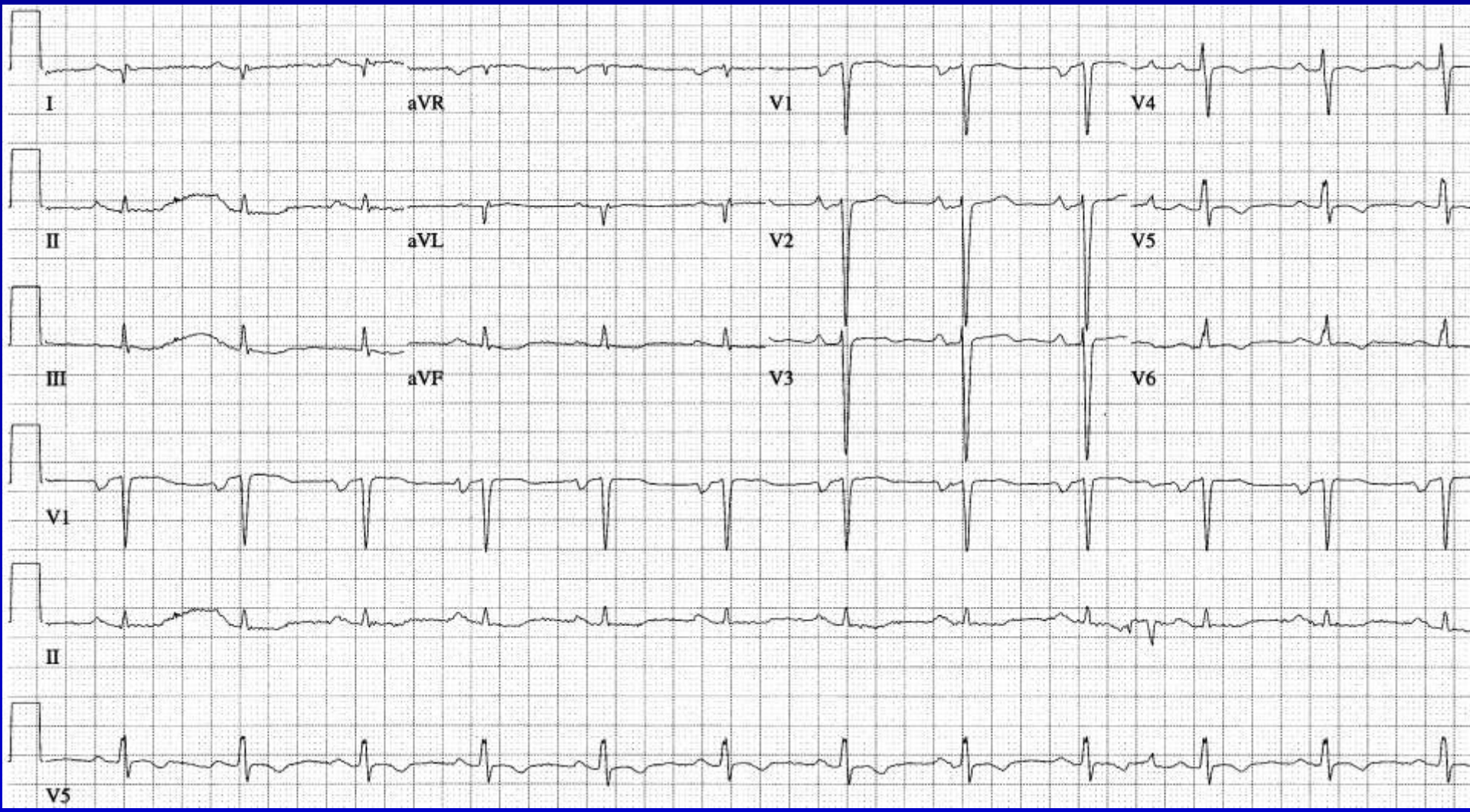


90 year old woman, peaked P in II, rightward QRS axis without RVH, RAE in V1, two PAC's, QRS duration 0.07, T flattening. ?True?

Unknown 21

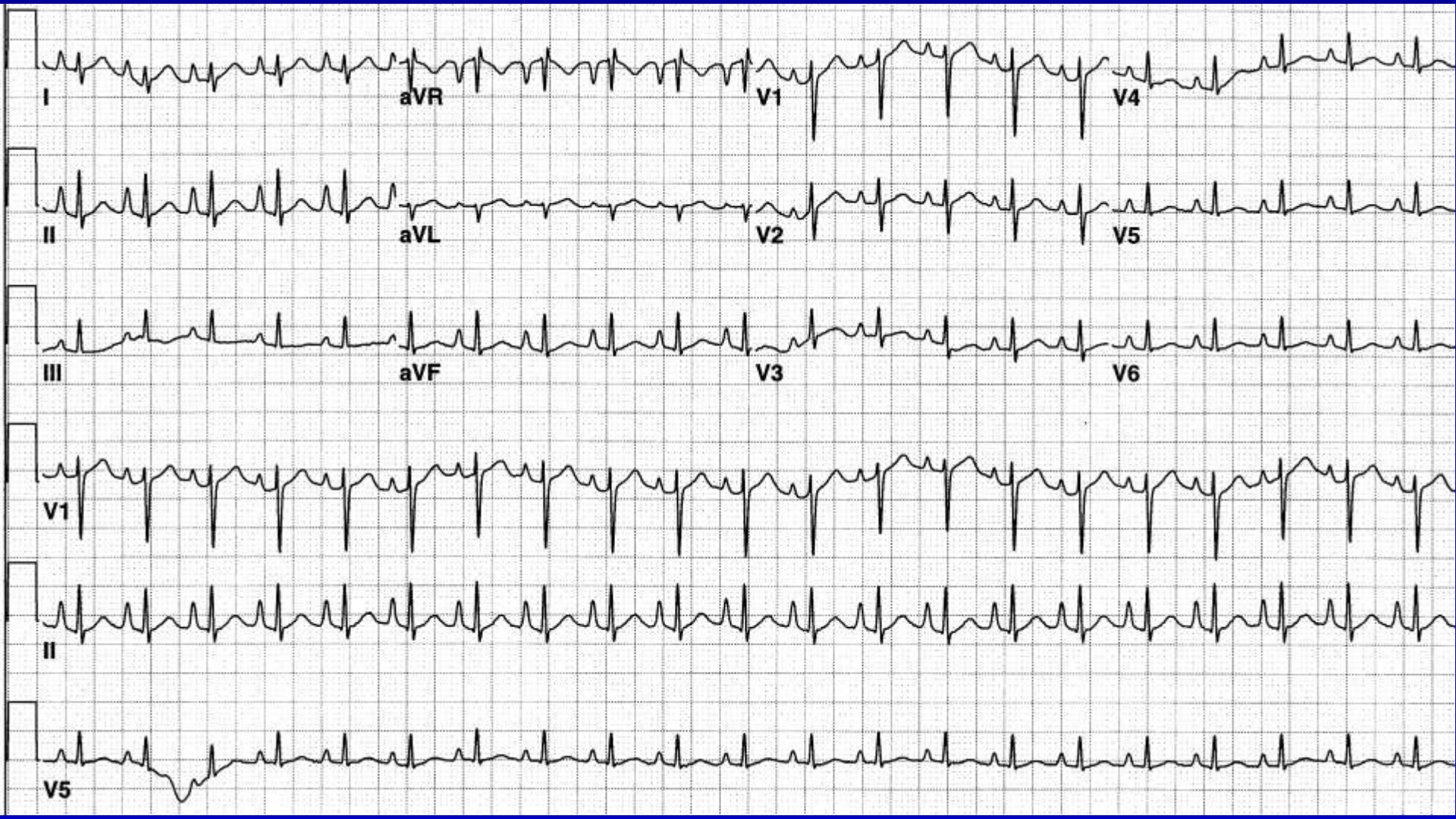


Unknown 21

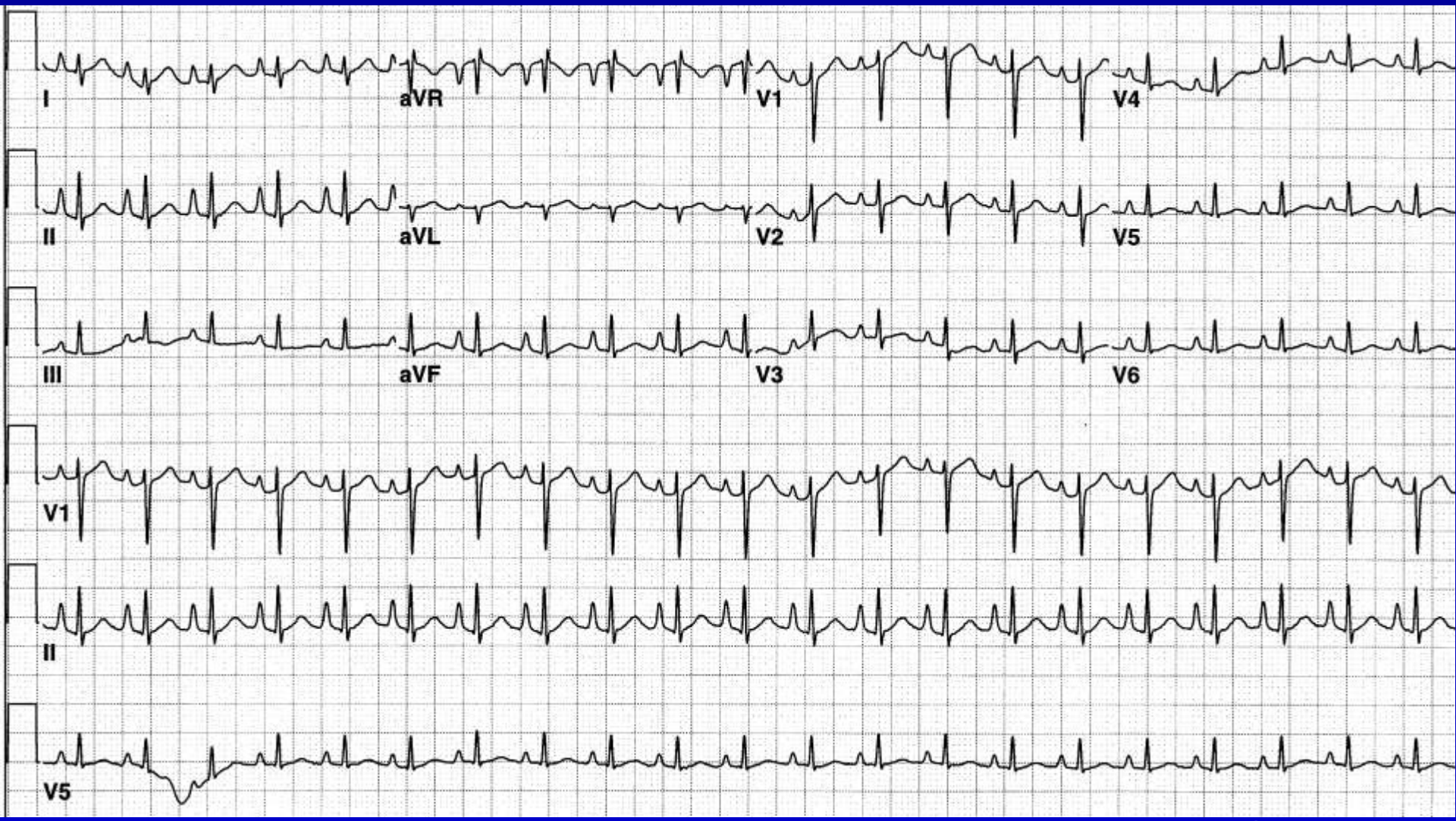


45 year old man, with lateral wall MI and consequent RAD and large voltage suspicious for LVH and clearcut LAE and nonspecific ST and T probably from LVH, maybe anterior MI too

Unknown 22

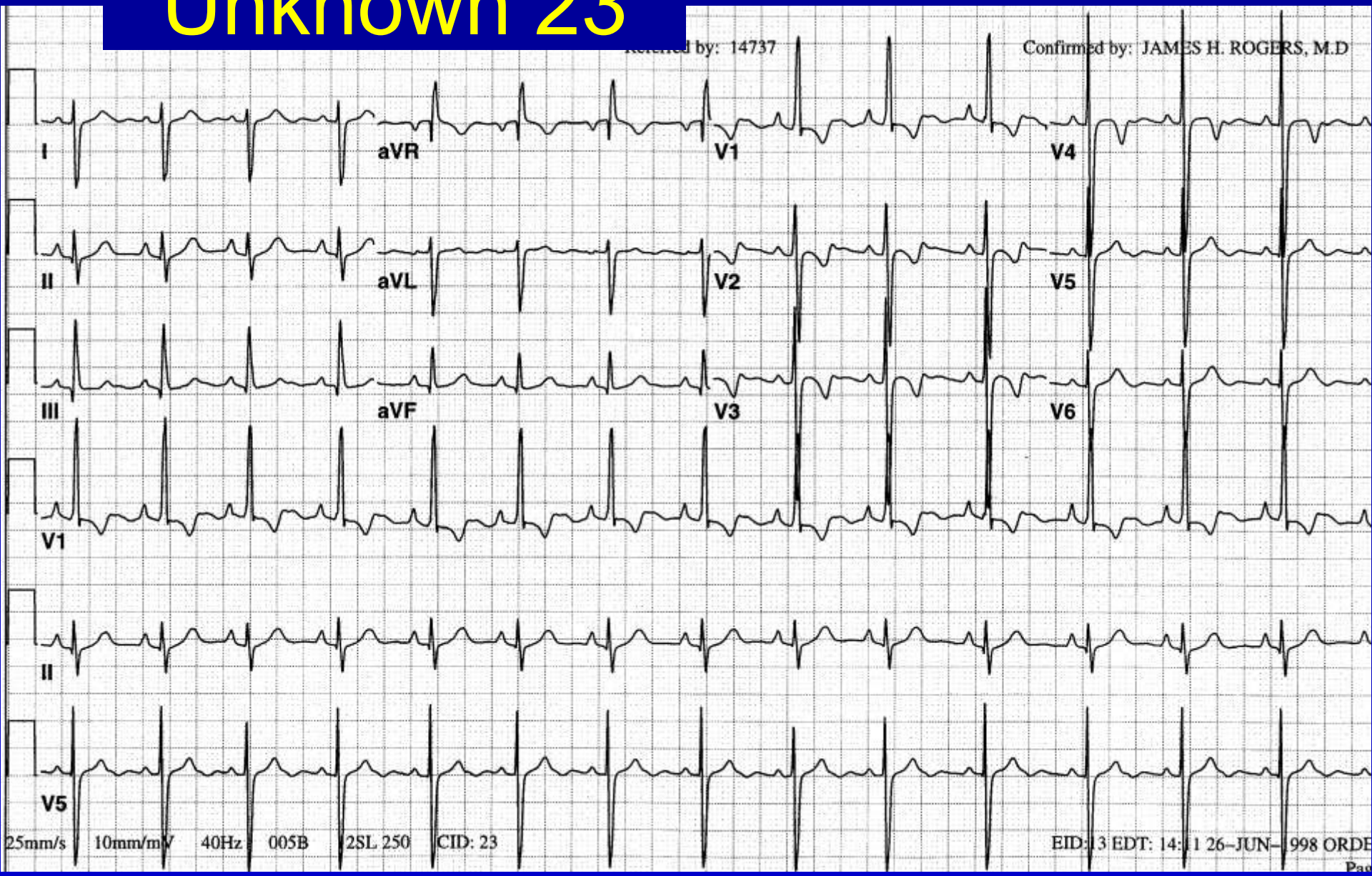


Unknown 22

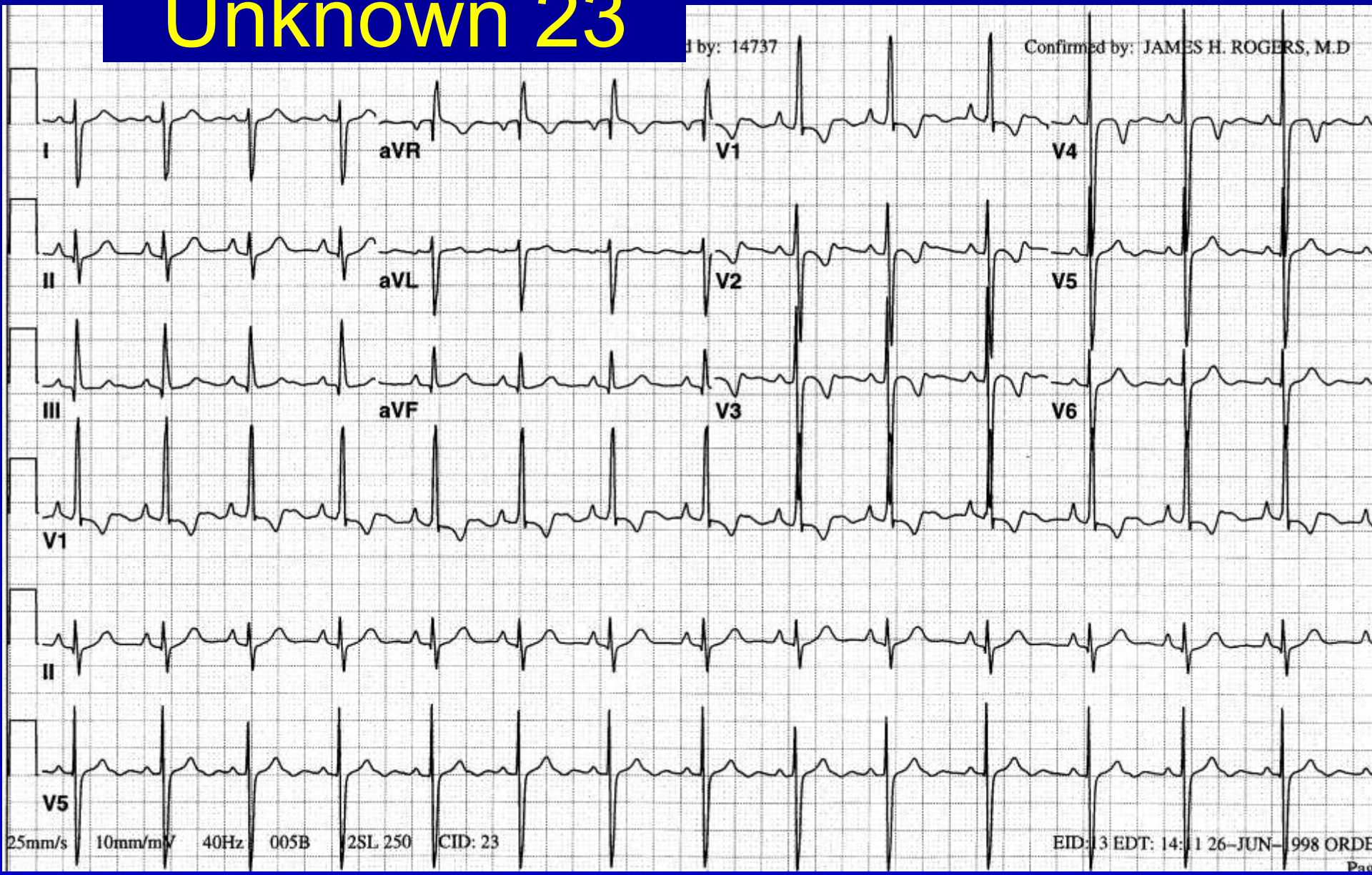


42 year old woman, dramatic RAE, and axis about 80 and no criteria for RVH. Sinus tachycardia. RAE more with pressure and LAE more with volume load (no RAE in ASD)

Unknown 23

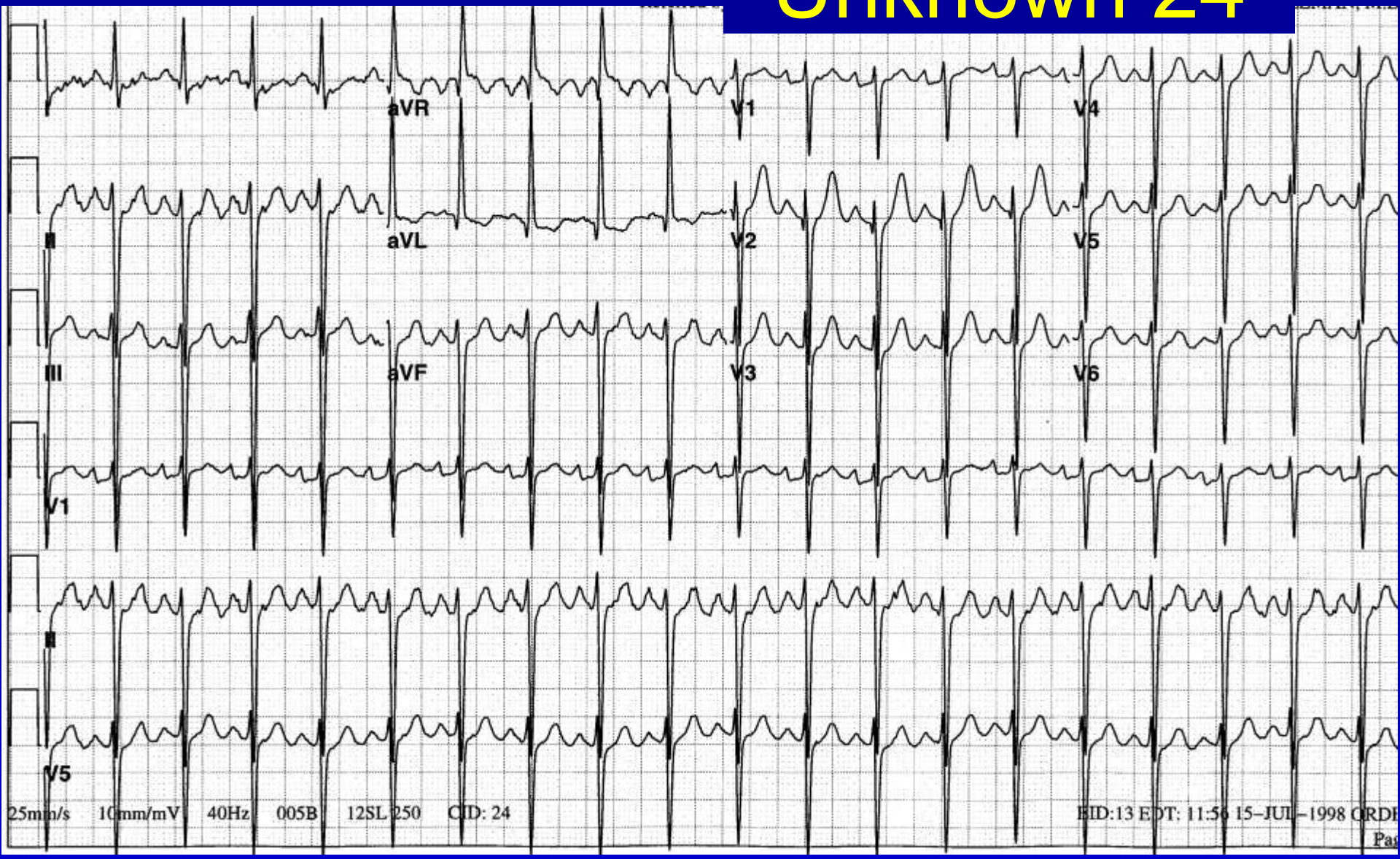

Confirmed by: JAMES H. ROGERS, M.D
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
25mm/s
10mm/mV
40Hz
005B
2SL 250
CID: 23
EID:13 EDT: 14:11 26-JUN-1998 ORDE

Unknown 23

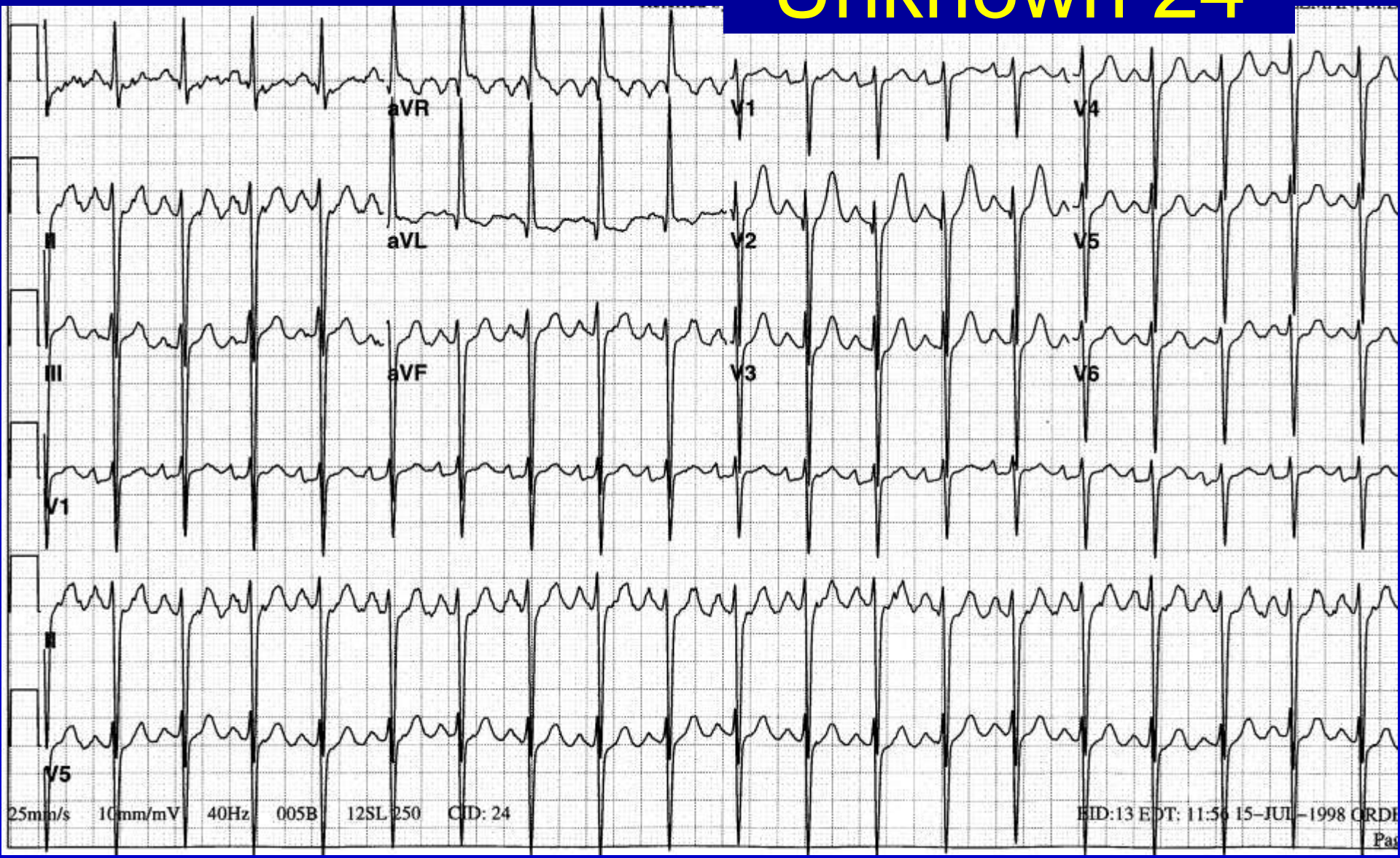


10 year old boy; pediatric cardiologist diagnosed RAE, RAD and RVH and the T inversion in V4 is abnormal but not in V1-3.

Unknown 24

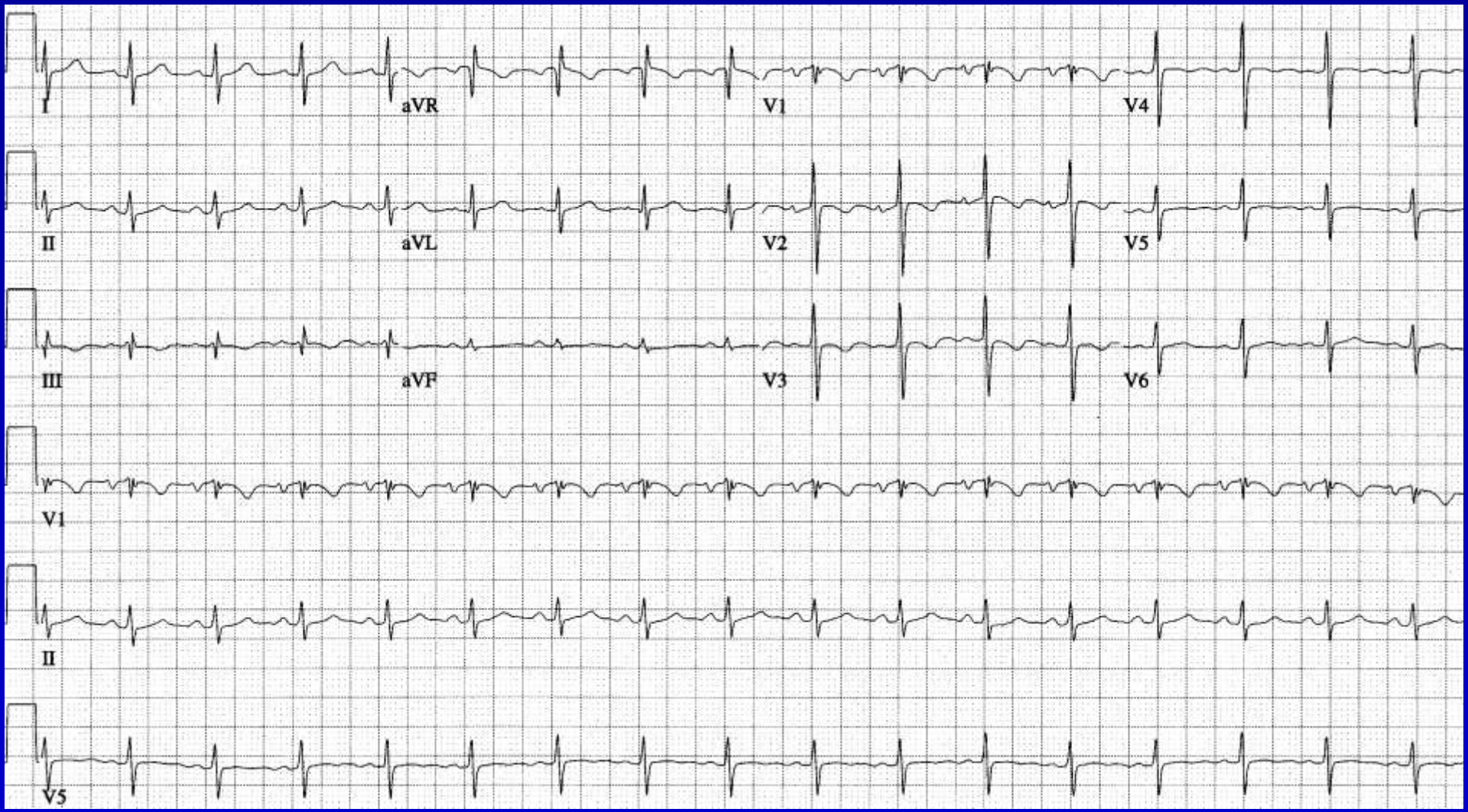

aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
25mm/s 10mm/mV 40Hz 005B 12SL 250 CID: 24
EID:13 EDT: 11:56 15-JUL-1998

Unknown 24

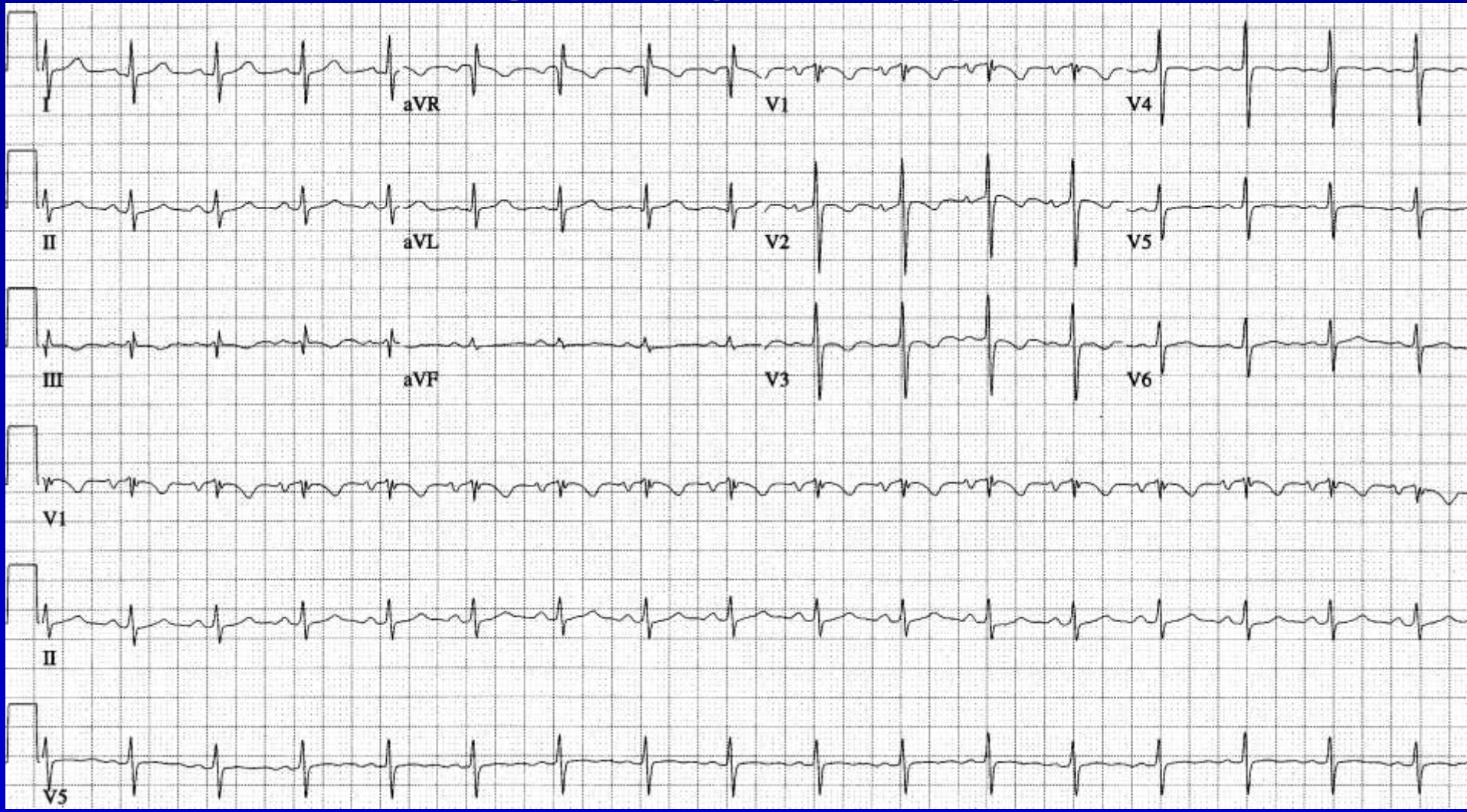


76 year old man, with sinus tachycardia, RAE, LVH, AFB, doesn't add up well.

Unknown 25

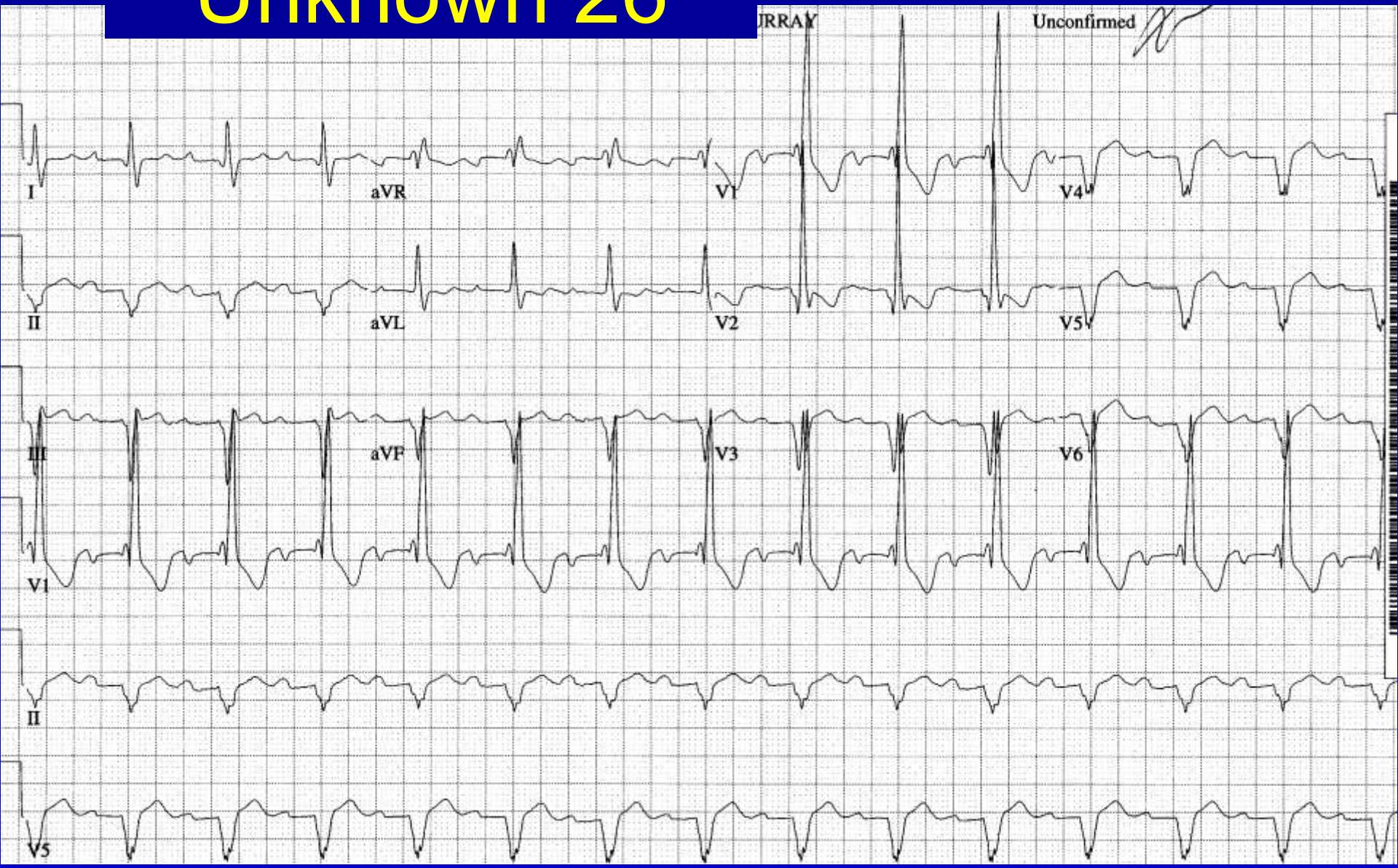


Unknown 25

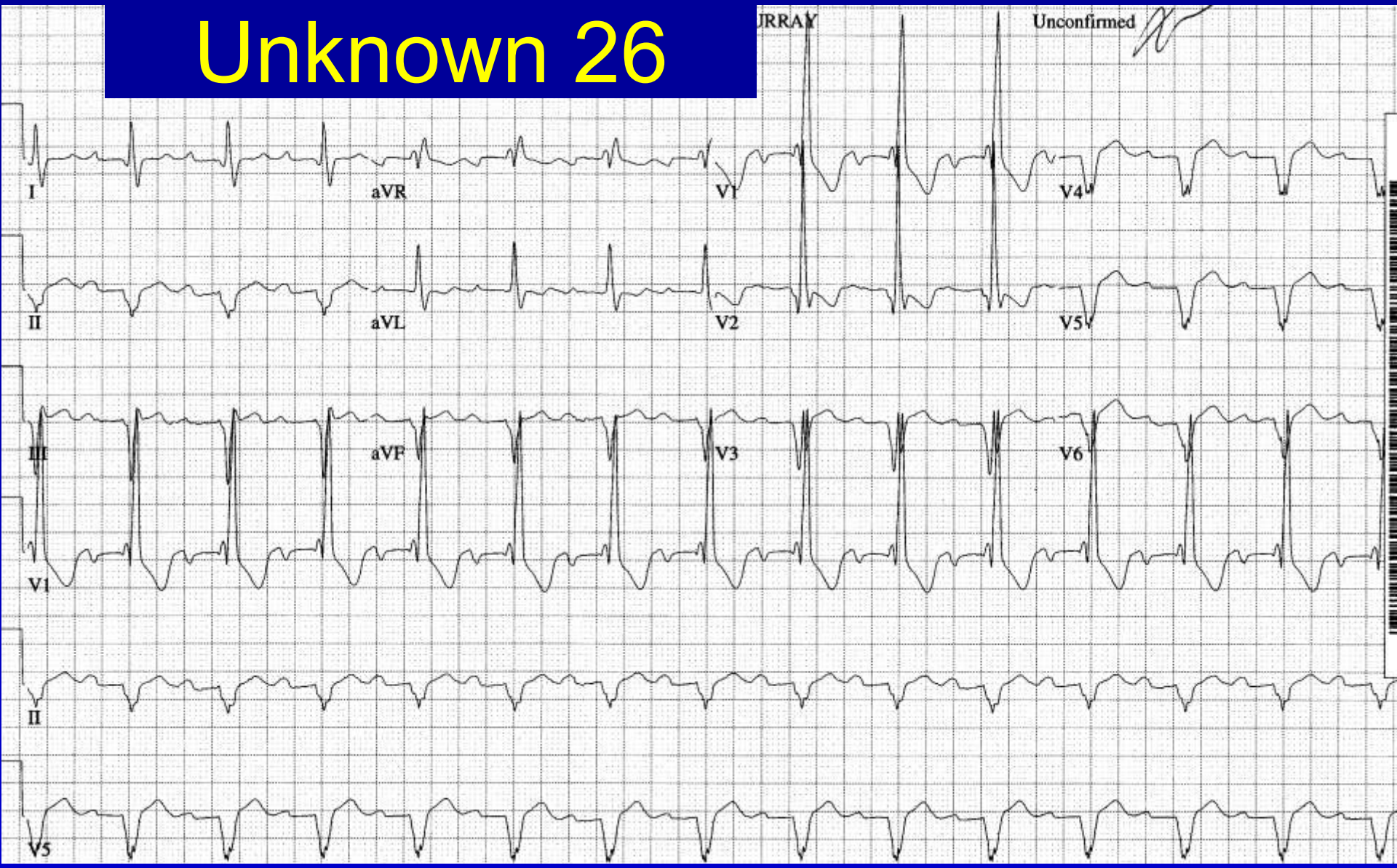


74 year old woman in MICU, echo normal EF, mild LVH, normal LA size, mild RAE. The significant S in I with indeterminate axis is new from one day before.

Unknown 26



Unknown 26



54 year old man, first degree AV block, LAE, IVCD, possible anterolateral MI and inferior MI and RBBB and RVH, no anterior hemiblock since terminal R in aVF