心衰竭及冠心症照護團隊
個別必修課程:
護理師/導管室技術師/體循師

認識基本心電圖

高雄長庚
冠狀動脈照護團隊

初訂 2019.02.01

Rate

- Normal : 60 ~ 100 bpm

  Bradicardia : < 60 bpm

  Tachycardia : > 100 bpm
- EKG Monitor : 15X

  Complete EKG : 6X
- Small box : 0.04 sec

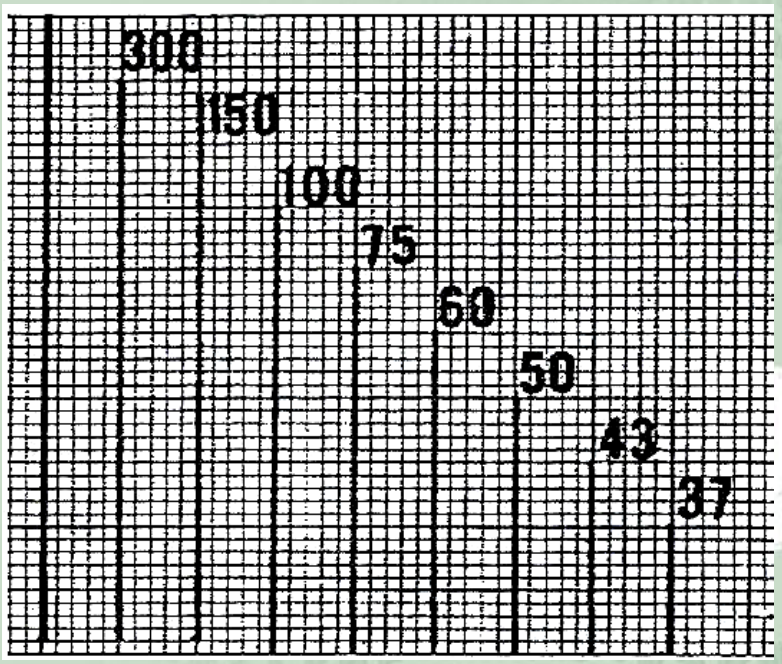
  Large box : 0.2 sec

Rate

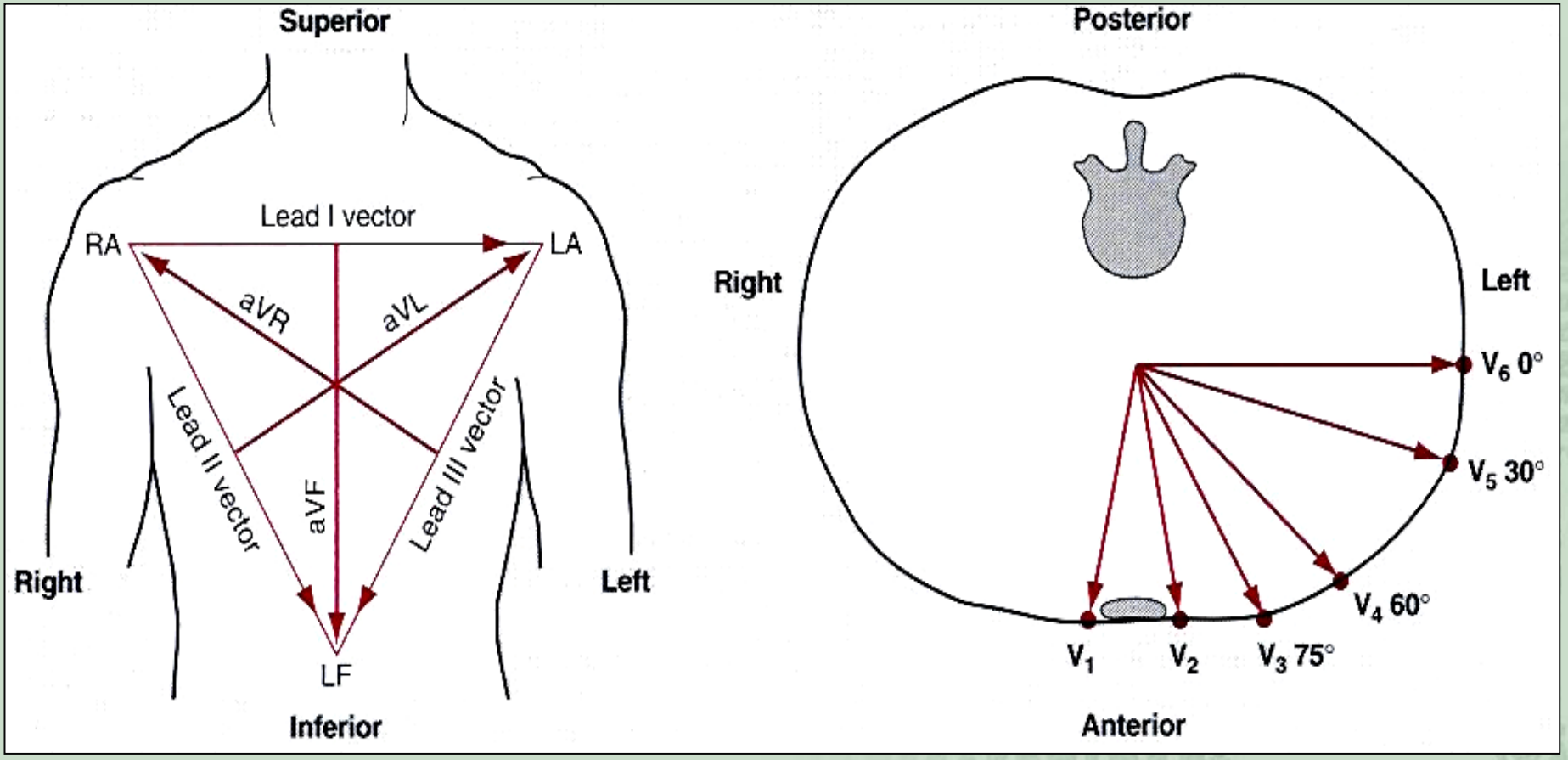
- 300 Rule:

TABLE 31-1
DETERMINING HEART RATE ON THE ECG

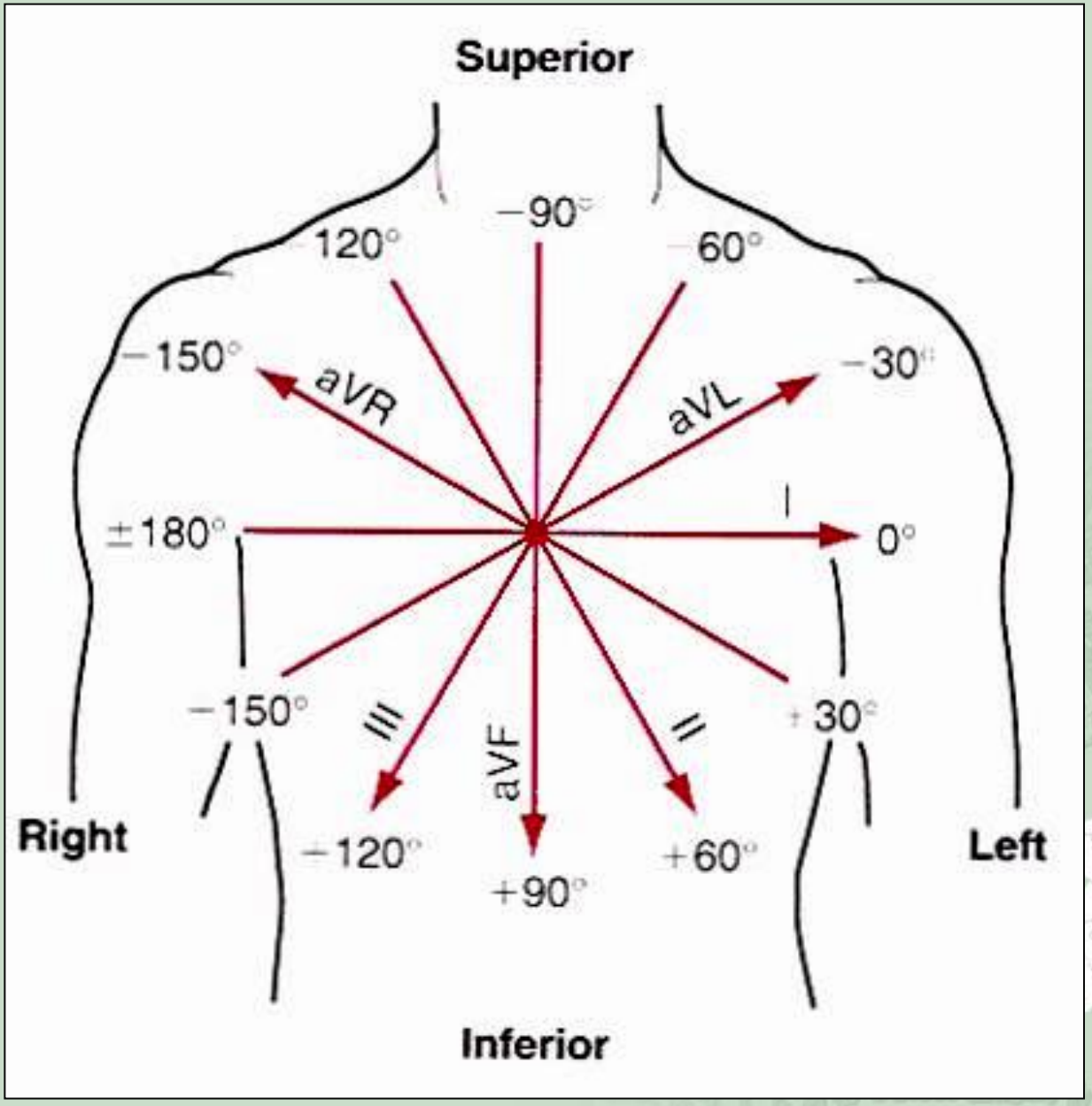
R-R Distance (# of boxes)	Heart Rate (bpm)
1	300
2	150
3	100
4	75
5	60
6	50



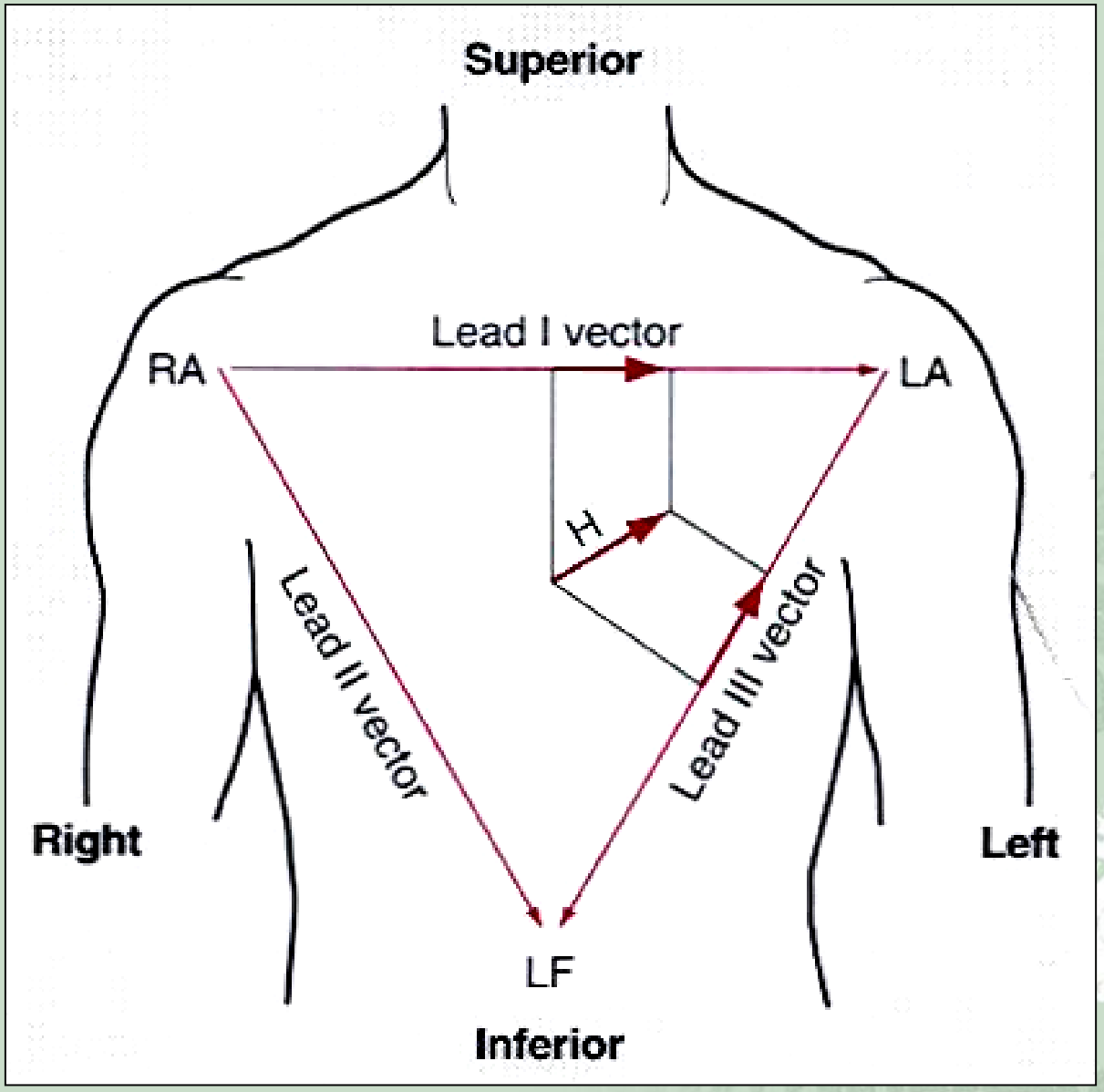
Axis


Superior
Lead I vector
RA
LA
aVR
aVL
Lead II vector
aVF
Lead III vector
Right
Left
LF
Inferior
Posterior
Right
Left
V6 0°
V5 30°
V4 60°
V1
V2
V3 75°
Anterior

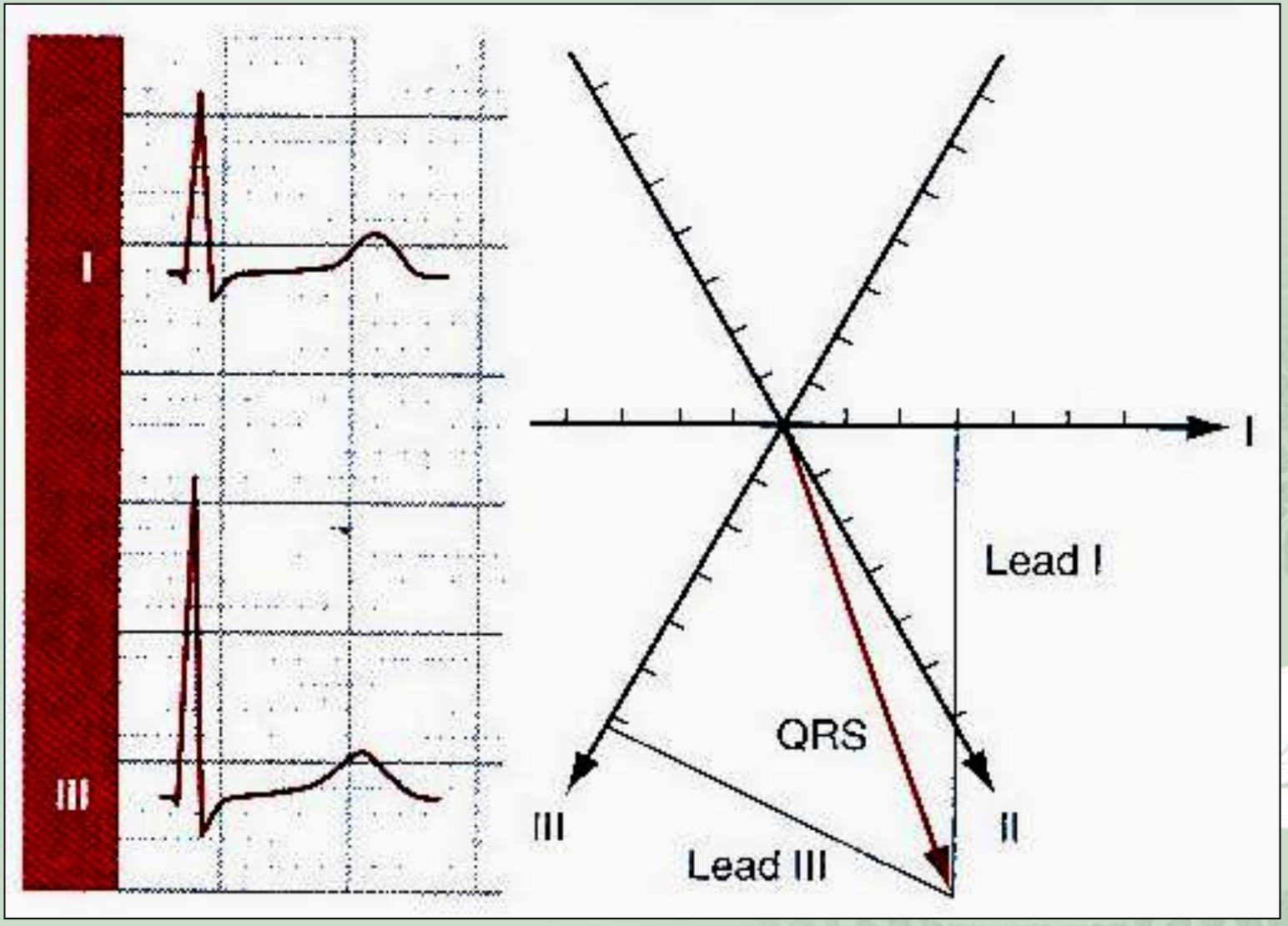
Axis


Superior
−90°
120°
60°
−150°
aVR
aVL
−30°
±180°
I
0°
−150°
III
aVF
II
+30°
Right
−120°
+60°
Left
+90°
Inferior

Axis


Superior
Lead I vector
RA
LA
H
Lead II vector
Lead III vector
Right
Left
LF
Inferior

Axis



Morphology

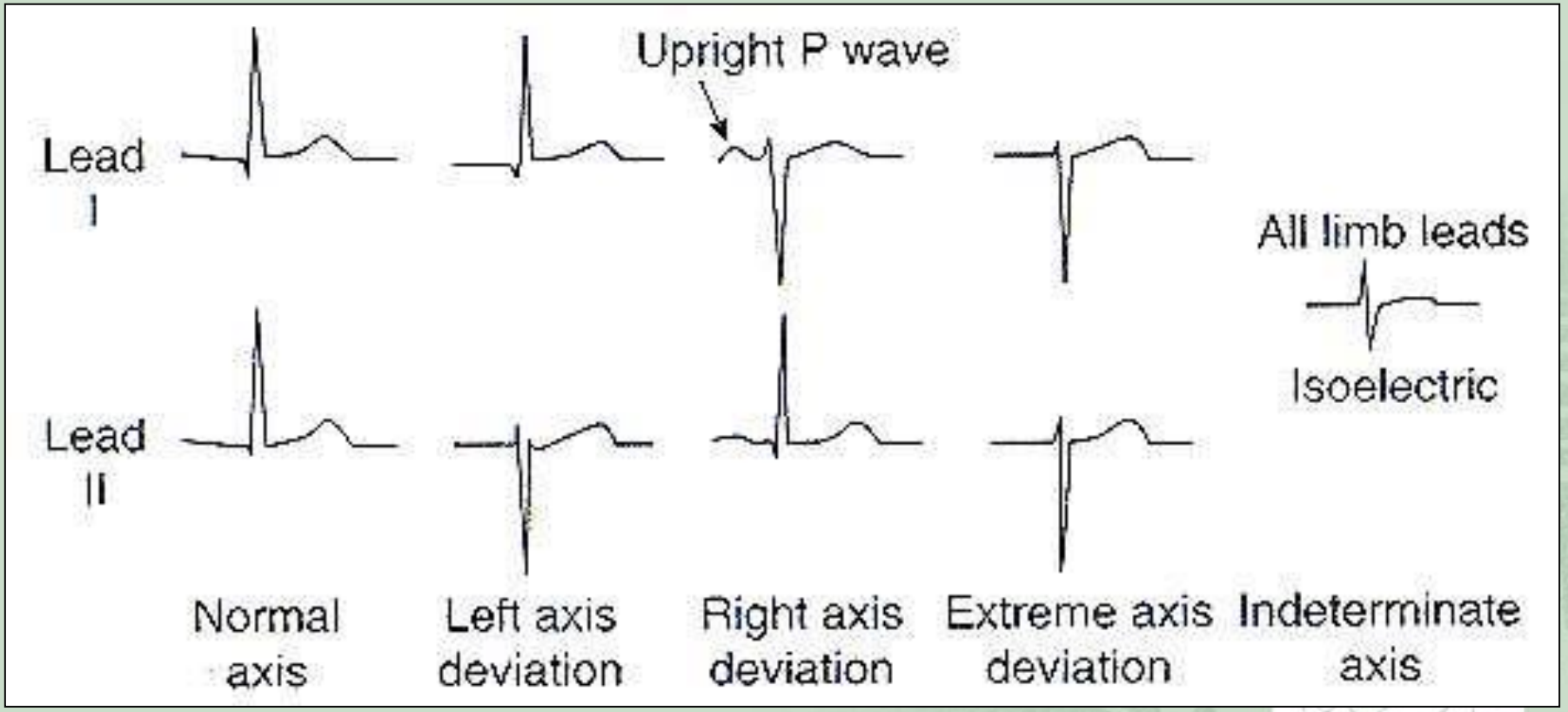
- Axis – RAD, LAD
- P wave morphology – RAE , LAE
- QRS complex morphology
  - LVH, RVH
  - LBBB, RBBB
  - LAFB, LPFB
  - Bifascular/Trifascular block
  - Ventricular pre-exicitation

Axis

- Definition : Spatial orientation of an electric vector
-

	Lead I	Lead II
Normal : -30 ~ +90	+	+
LAD : -30 ~ -90	+	-
RAD : +90 ~ +180	-	+
EAD : -90 ~ -180	-	-

Axis


Upright P wave
Lead I
Lead II
All limb leads
Isoelectric
Normal axis
Left axis deviation
Right axis deviation
Extreme axis deviation
Indeterminate axis

Axis - LAD

- LAD : -30 ~ -90
- Causes : Normal variant
- LVH, LAFB, LBBB
  Inferior wall MI,
  Ventricular preexcitation
  (posteroseptal accessory pathway)
  Elevated diaphragm
  (Obesity, pregnancy, ascites)

Axis - RAD

- RAD : +90 ~ +180
- Causes : Normal variant
  RVH, LPFB
  Cor pulmonale, COPD
  Anterolateral MI
  Ventricular preexcitation
  (free wall accessory pathway)
  Dextrocardia
  Childhood
  Limb lead reversal

Axis - Extreme

- Indeterminate (Northwest) axis : -90 ~ +180
- Causes : Normal variant

  COPD

  RVH

Anterosuperior axis

- Defines whether the apex of the heart faces the left arm (horizontal) or the left foot (vertical),
- Horizontal heart leads to a QRS complex in aVL that resembles that in leads V5 and V6
- Vertical heart leads to a QRS complex in lead aVF that resembles that in leads V5 and V6

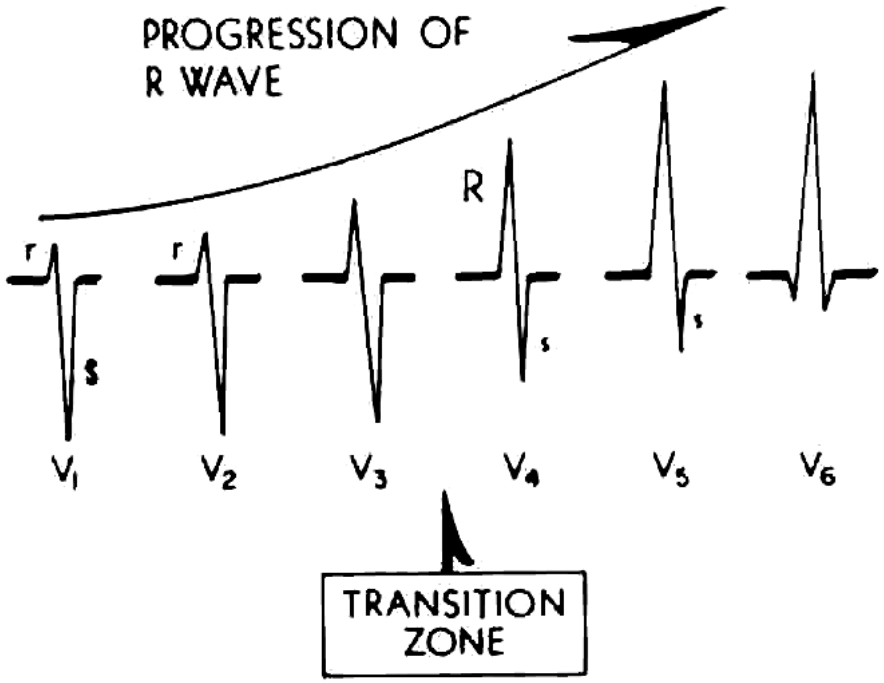
Longitudinal Axis

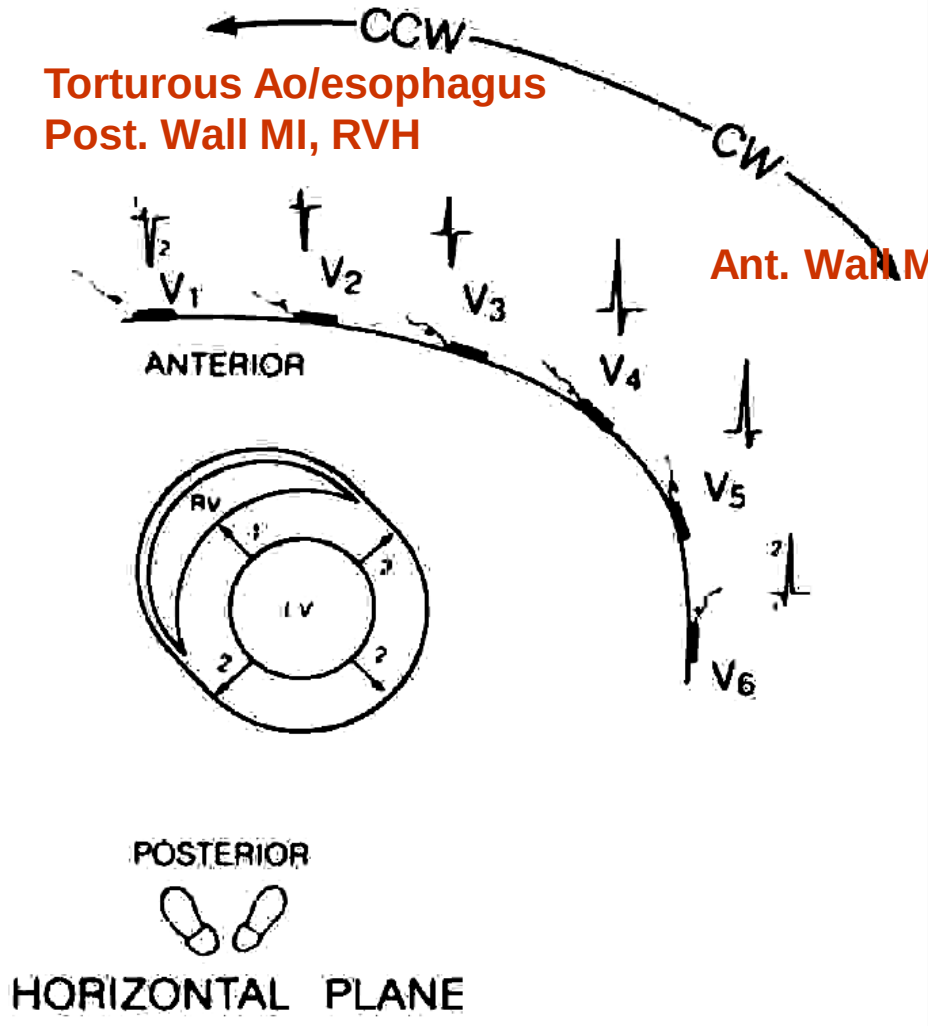
- apex-to-base direction, which defines whether the left ventricle is anterior or posterior
- Clockwise rotation results in a more posterior position of the left ventricle, and the RV QRS complex (rS) is displaced to the left precordial leads.
- Counterclockwise rotation results in a more anterior shift of the left ventricle, and the LV QRS (qR) pattern is observed in the right precordial leads, mimicing RVH

Clockwise vs. Counter Clockwise

P wave

- Represents atrial depolarization and is a measure of the





Atrial Depolarization

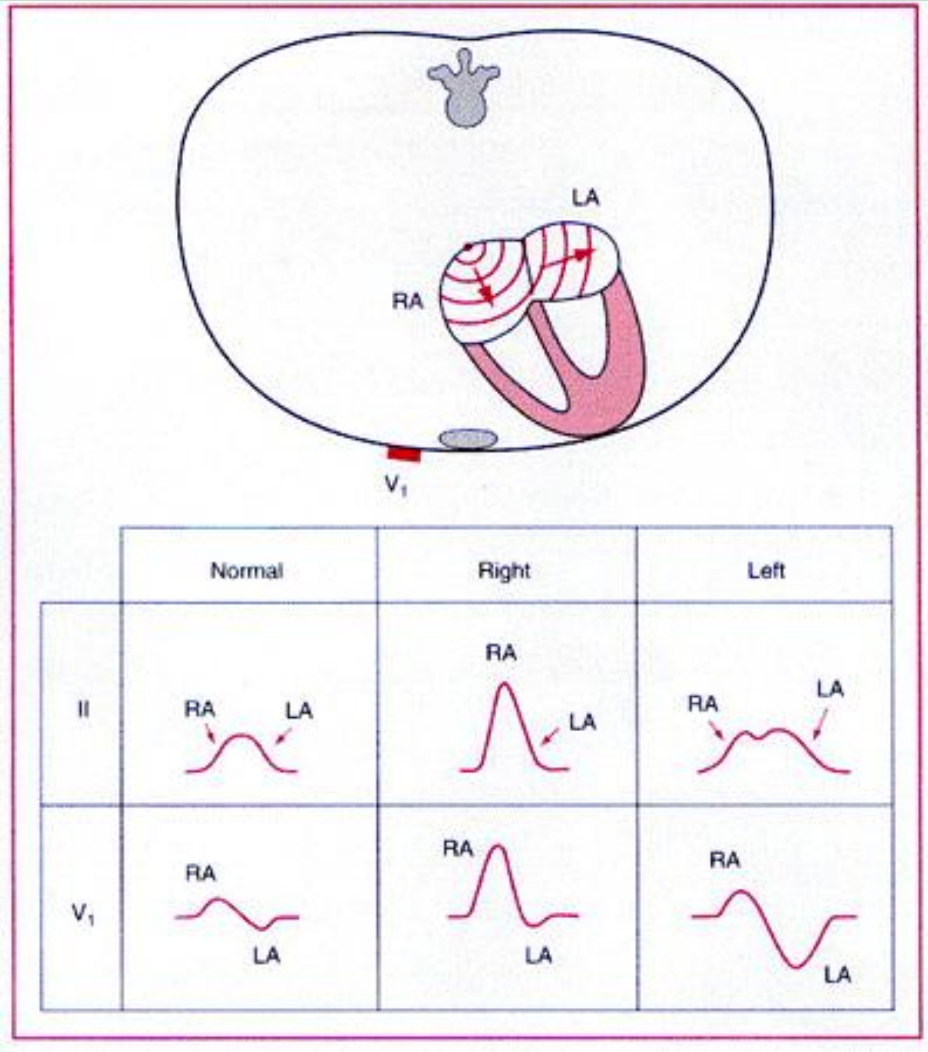
- **RA: SA node —> inferior and anterior.**

  Initial positive deflection of P wave in II & V1

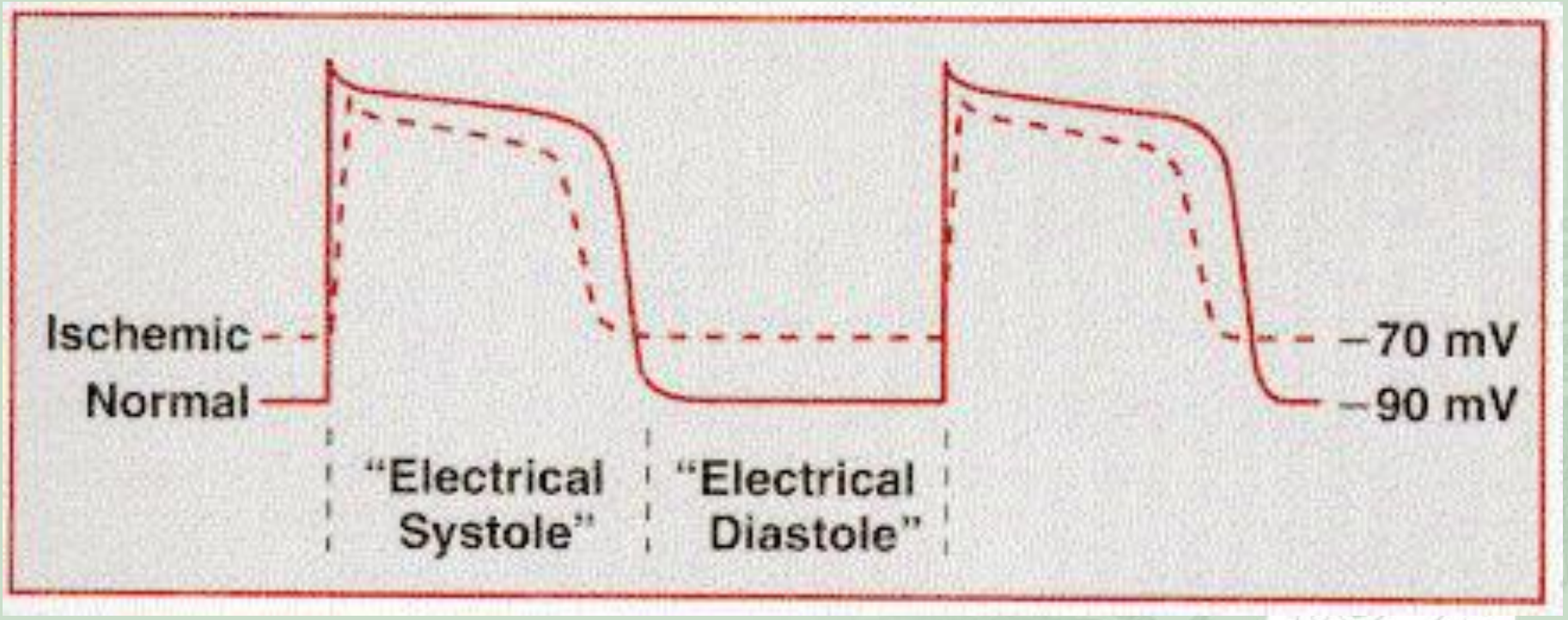
- **LA: High interatrial septum —> inferior and posterior.**

  Terminal deflection of the P wave - positive in II

  negative in V1

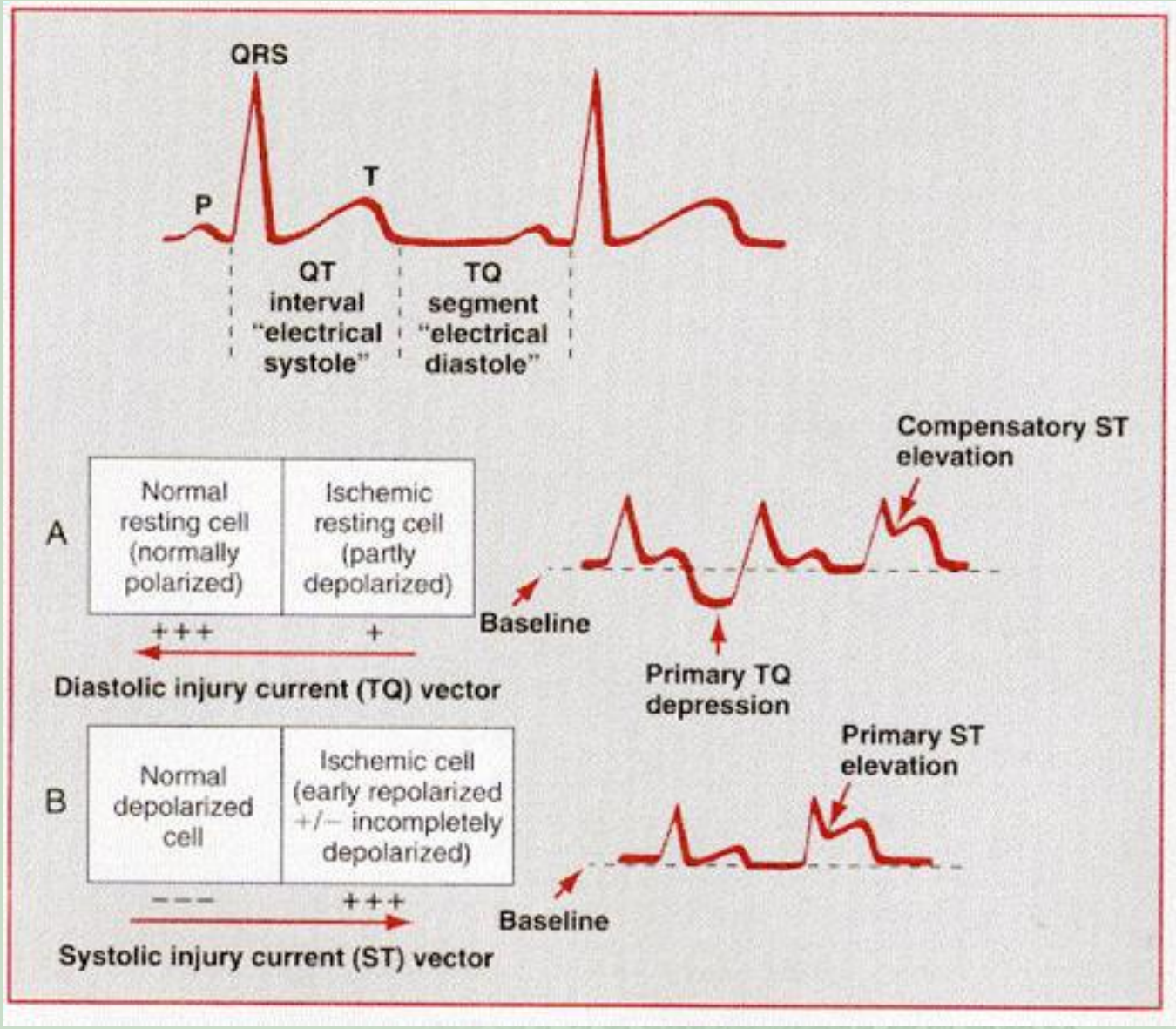


Myoischemia – mechanism of injury current



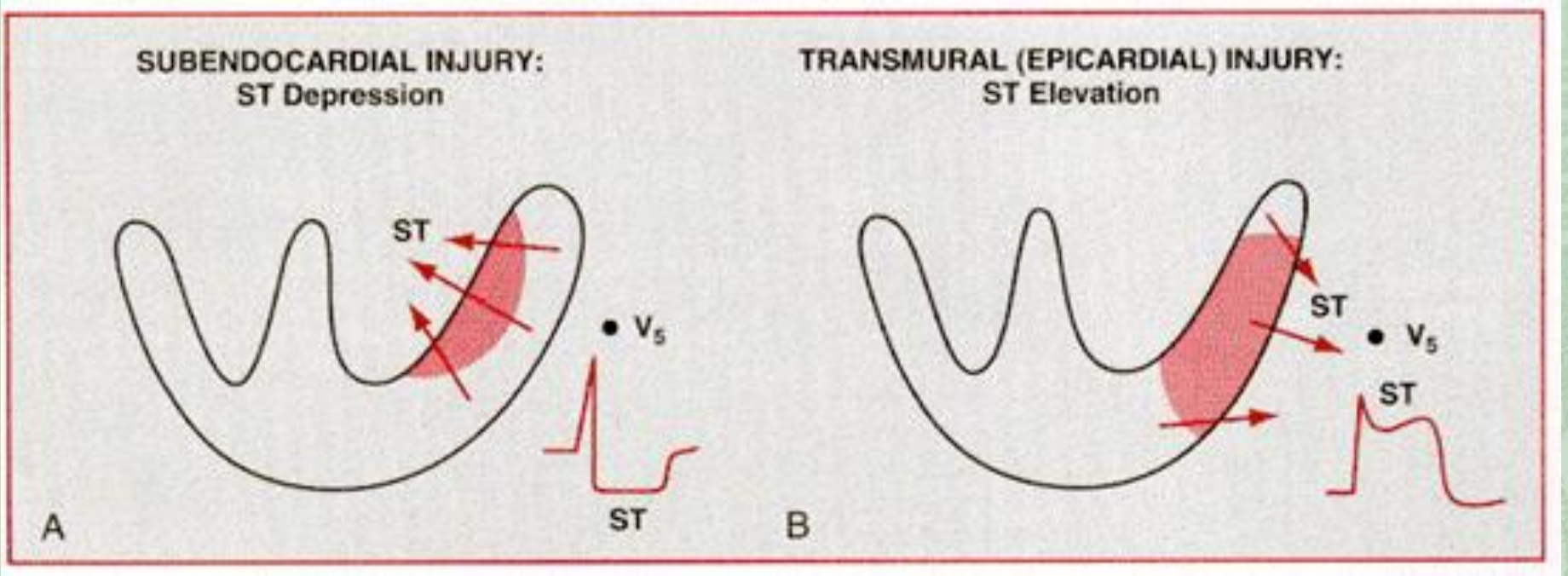
Myoischemia - mechanism

Injury Current

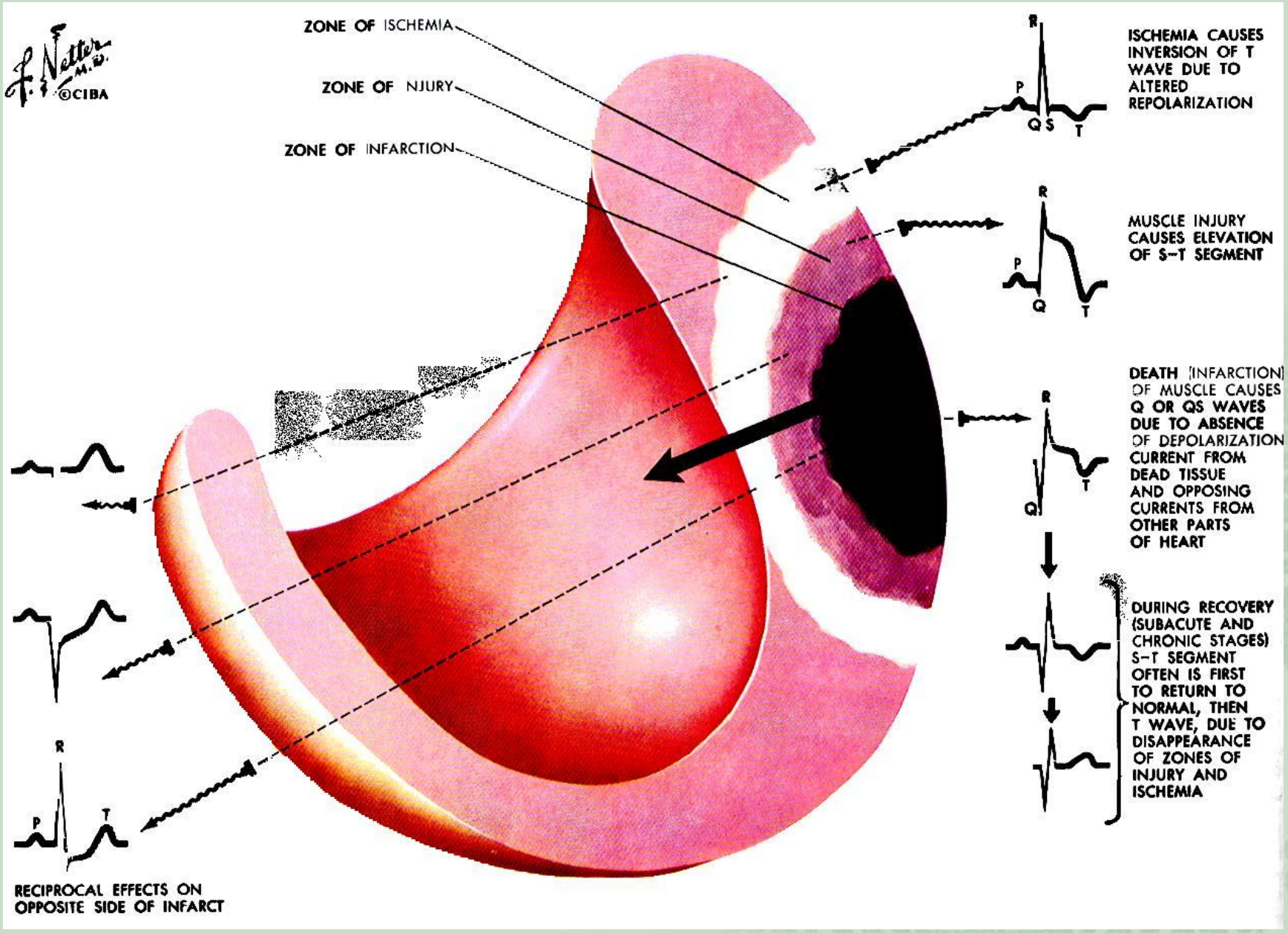


Myoischemia - extent

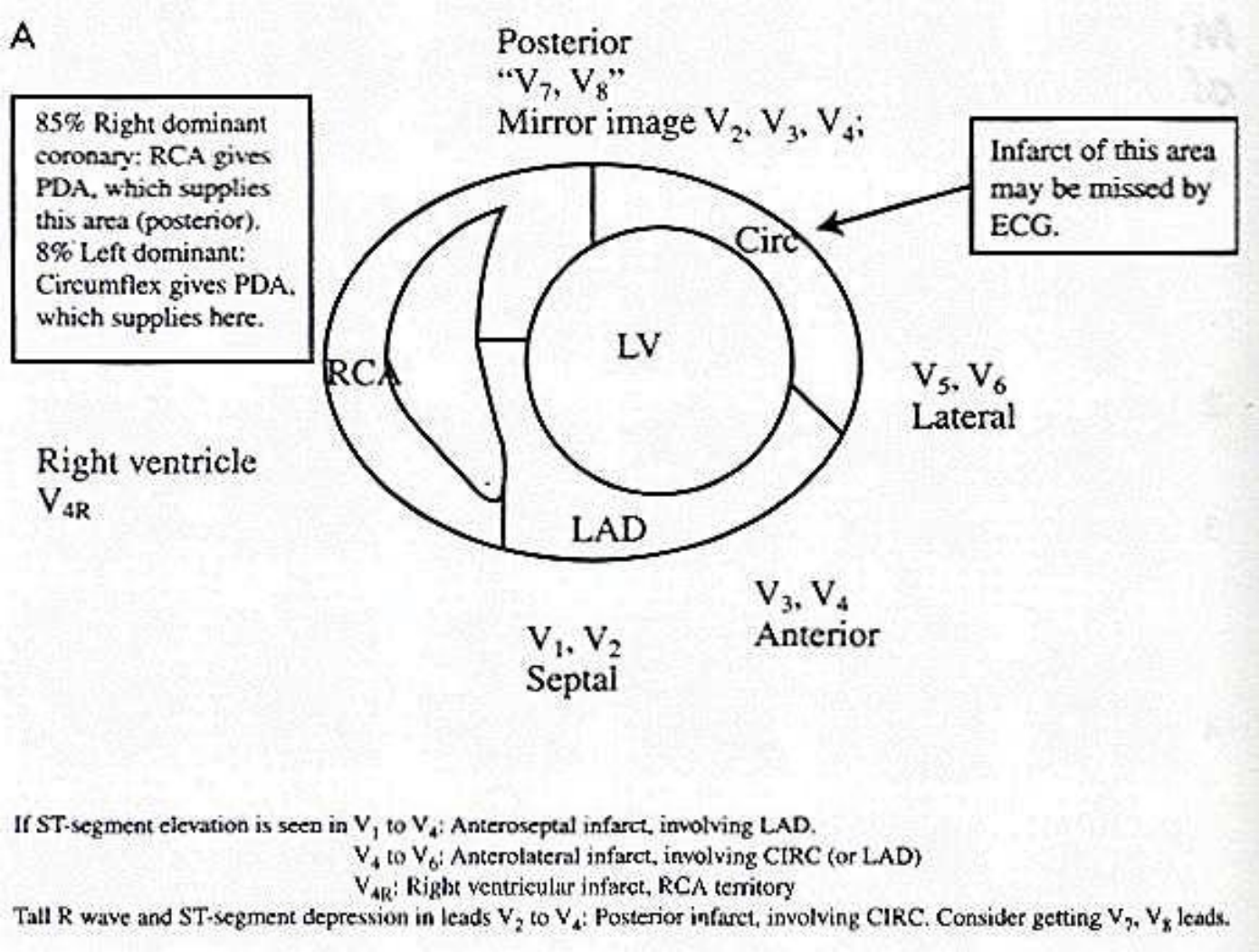
- Subendocardial injury vs Transmural injury



Myoischemia



Myocardial Infarction



Myocardial Infarction

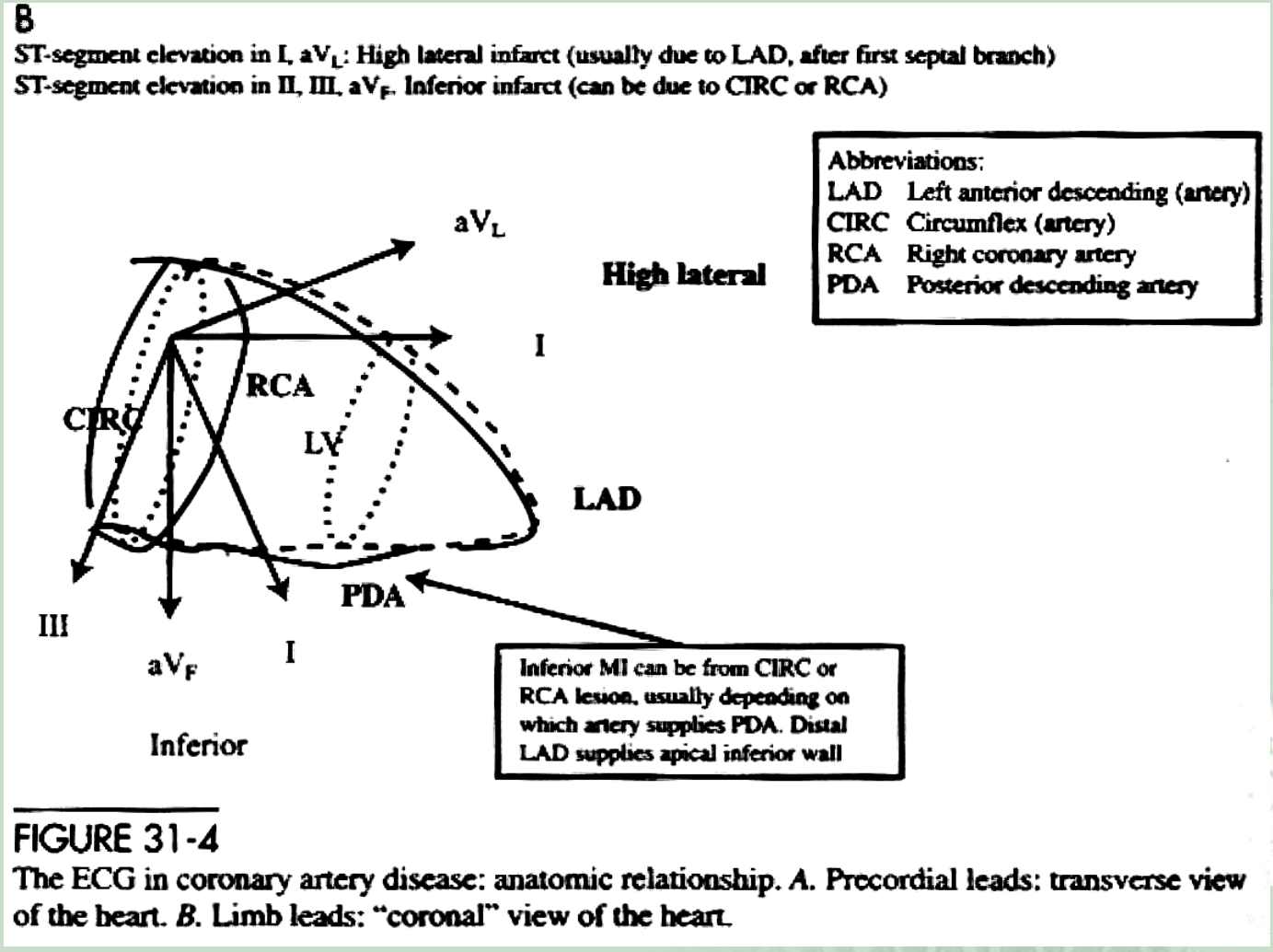
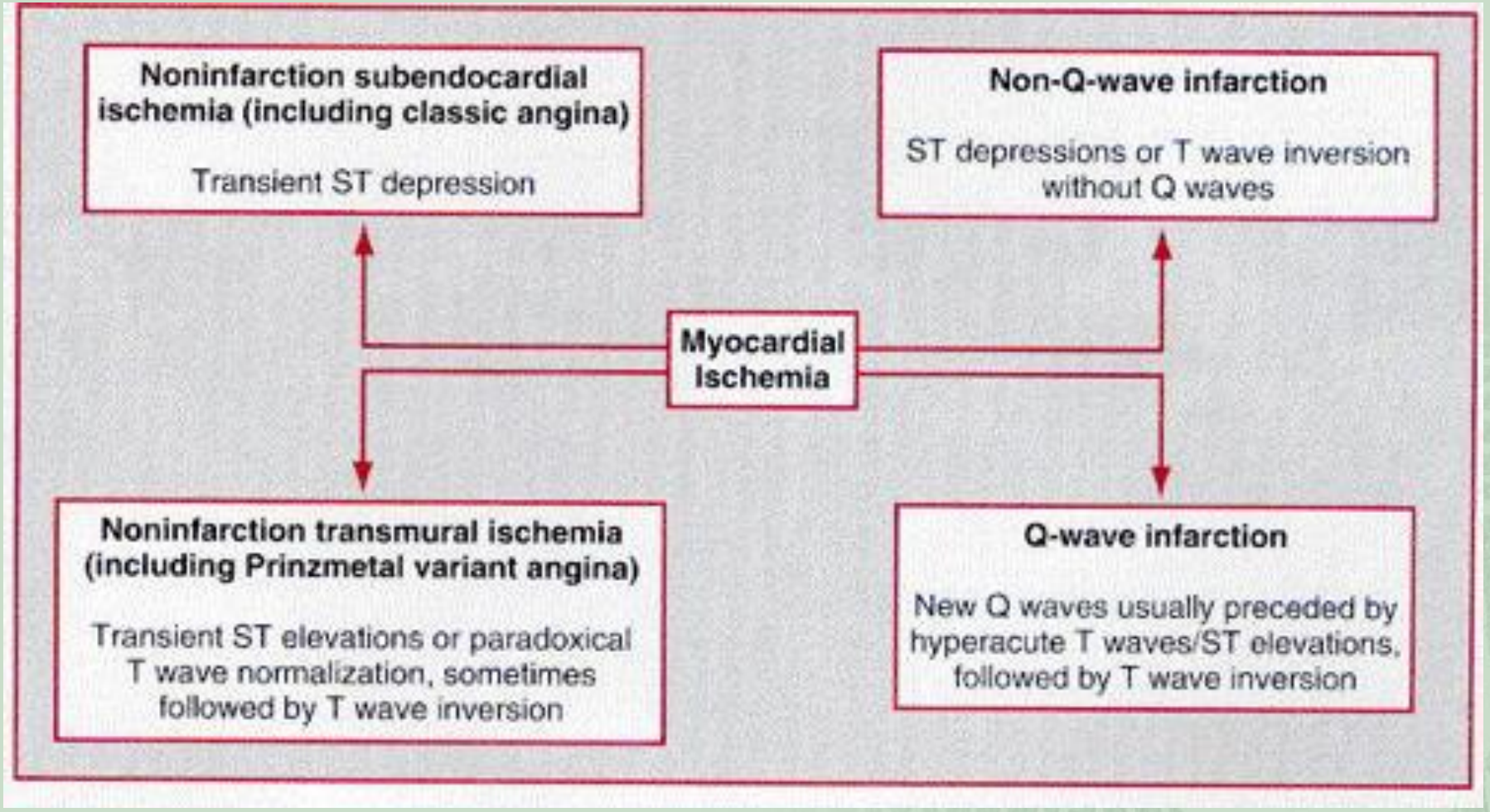


FIGURE 31-4

The ECG in coronary artery disease: anatomic relationship. *A.* Precordial leads: transverse view of the heart. *B.* Limb leads: "coronal" view of the heart.

Myoischemia - different pattern of ischemia



AMI

- When the resulting intracoronary thrombus is only partially occlusive, ST depressions or T-wave inversions or both develop, potentially resulting in a “non-Q” MI.
- When the thrombus is completely occlusive, ST elevations typically occur.

  75% - deep Q waves

  25% - no deep Q wave

  but reduction of the R wave height or

  notching of the QRS complex
- Only half of pathologically “transmural” infarcts are associated with Q waves on the ECG

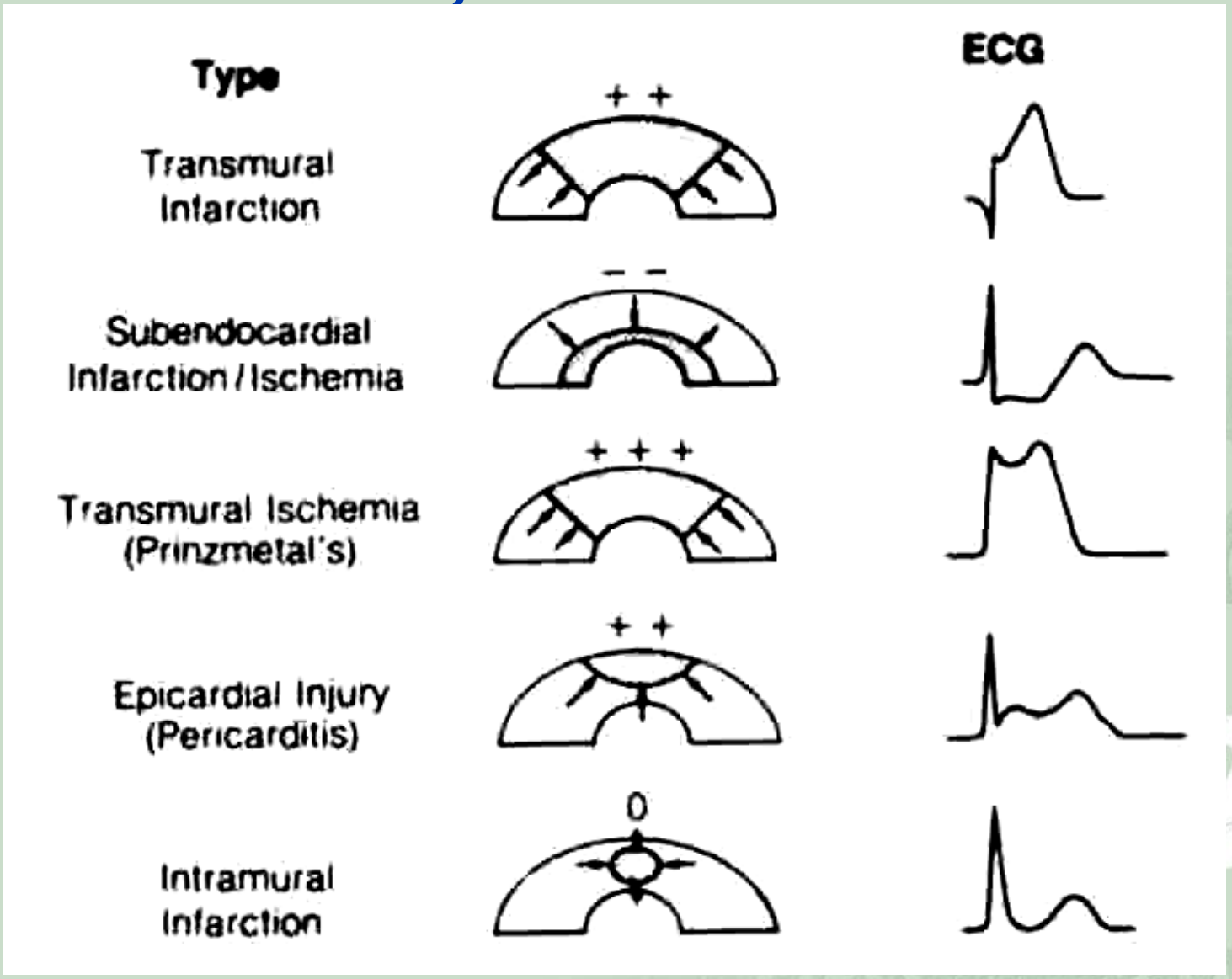
AMI

- There were no differences in the rates of recurrent ischemic events between Q and non-Q MI
- Differentiation between these two types of infarction is important when considering initial management.

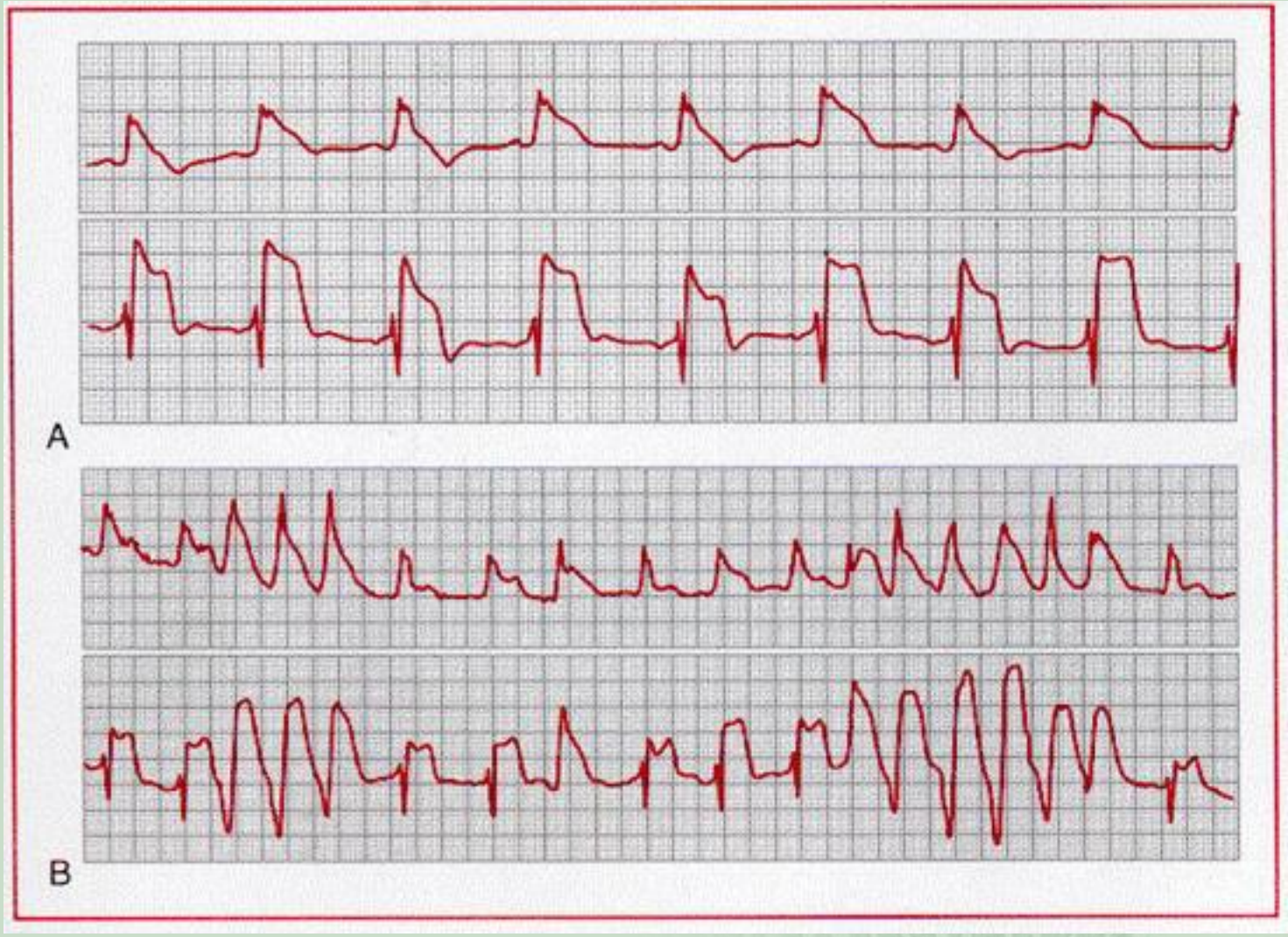
  => ST segment elevation MI are candidates for reperfusion therapy (thrombolysis or catheter-based),

  => non-ST-segment elevation MI or unstable angina fare better with initial vigorous anti-ischemic, antithrombin, and antiplatelet therapies

Different EKG Patterns of Myoischemia



Prinzmetal Angina



Myocardial Infarction

TABLE 7–1. ACUTE TRANSMURAL MI. The characteristic ECG changes and their evolution.

Phase	Time	ST Segment	T Waves	R or Q Waves
Initial	First several hours	Elevated	Accentuated	Not yet
Early	First day	Elevated	Inverting	Q waves form
Mid	First days	Elevated or normalizing	Inverted	Q waves formed
Late	After first days	Elevated or normal	Variable	Q waves persist Loss of R waves persists

Myocardial Infarction

TABLE 7–2. ACUTE TRANSMURAL MI. The characteristic ECG changes and their location.

Territory	Artery Usually Responsible	ST Elevation in Contiguous Leads	Amount of ST Elevation
Anterior	LAD, diagonal, proximal marginal	Two leads of V_{1-4}	2 mm
Apical			2 mm
Inferior	PDA Origin: RCA 90%, LCx 10%	Two of: II, III, aVF	1 mm
Lateral	LCx	V_{5-6}	2 mm
High lateral	LCx, distal RCA	I, aVL	2 mm
Posterior	RCA or LCx	ST depression in V_1, V_2	2 mm
Right ventricular	Acute marginal branch of RCA	V_4R	1 mm

Myocardial Infarction

Anatomy of myocardial infarction

Infarction area	ECG leads involved	Coronary artery	Branch
Extensive anterior	I, AVL, V_1–V_6	Left, LM	LAD, LCX
Anteroseptal	V_1–V_3	Left	LAD
Anterolateral	I, AVL, V_4–V_6	Left	LCX
Inferior	II, III, AVF	Right 80%* Left 20%	PDA
True posterior	V_1–V_2 (reciprocal)	Variable: Left Right	 LCX PL

Abbreviations: LAD = Left anterior descending artery, LCX = Left circumflex artery; LM = Left main artery; PDA = Posterior descending artery; PL = Posterolateral branches.

*Refers to percentage of patients.

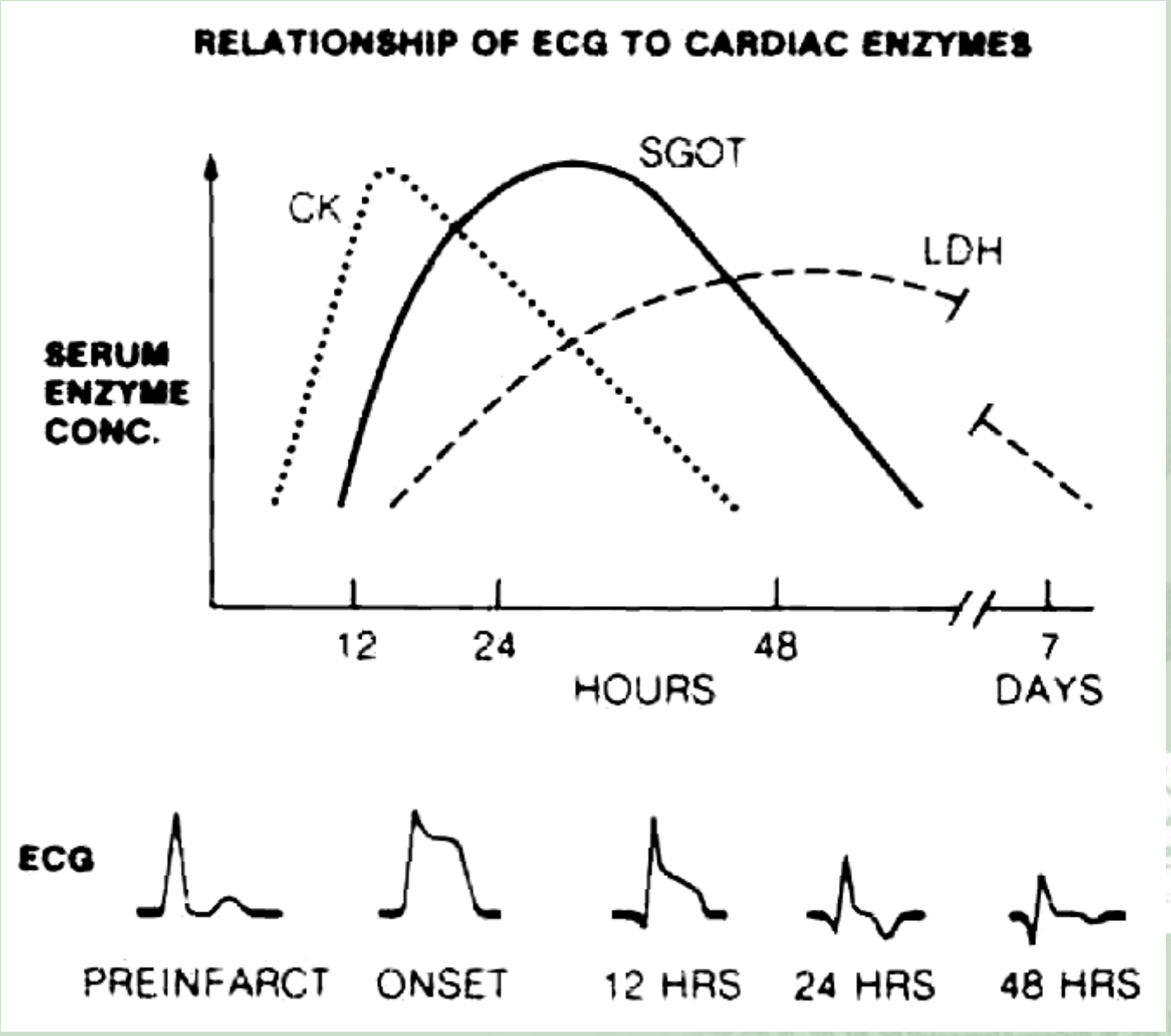
Myocardial Infarction

TABLE 7–3. ACUTE TRANSMURAL MI LOCALIZATION.

Territory	ECG Lead	ECG Criteria[1]
Anterior	V_2	Any Q, or R ≤ 1 mm and > 10 ms, or R in V_2 ≥ R in V_1
	V_3	Any Q, or R ≤ 2 mm and > 20 ms
Apical	V_5	Q ≥ 30 ms or Q ≥ $\frac{1}{2}$ R height
	V_6	Q ≥ 30 ms or Q ≥ $\frac{1}{3}$ R height
High lateral	I, aVL	Either I or aVL: Q ≥ ms
Inferior	II, III, aVF	II or aVF: Q ≥ 30 ms III: Q depth > $\frac{1}{3}$ R height
Posterior	V_1 V_2	R ≥ 40 ms or ≥ 6 mm R ≥ 50 ms or ≥ 15 mm

[1] *In the absence of LVH, LAFB, LBBB, RBBB, RVH, COPD, W-P-W, the criteria listed are ≥ 93% specific.*

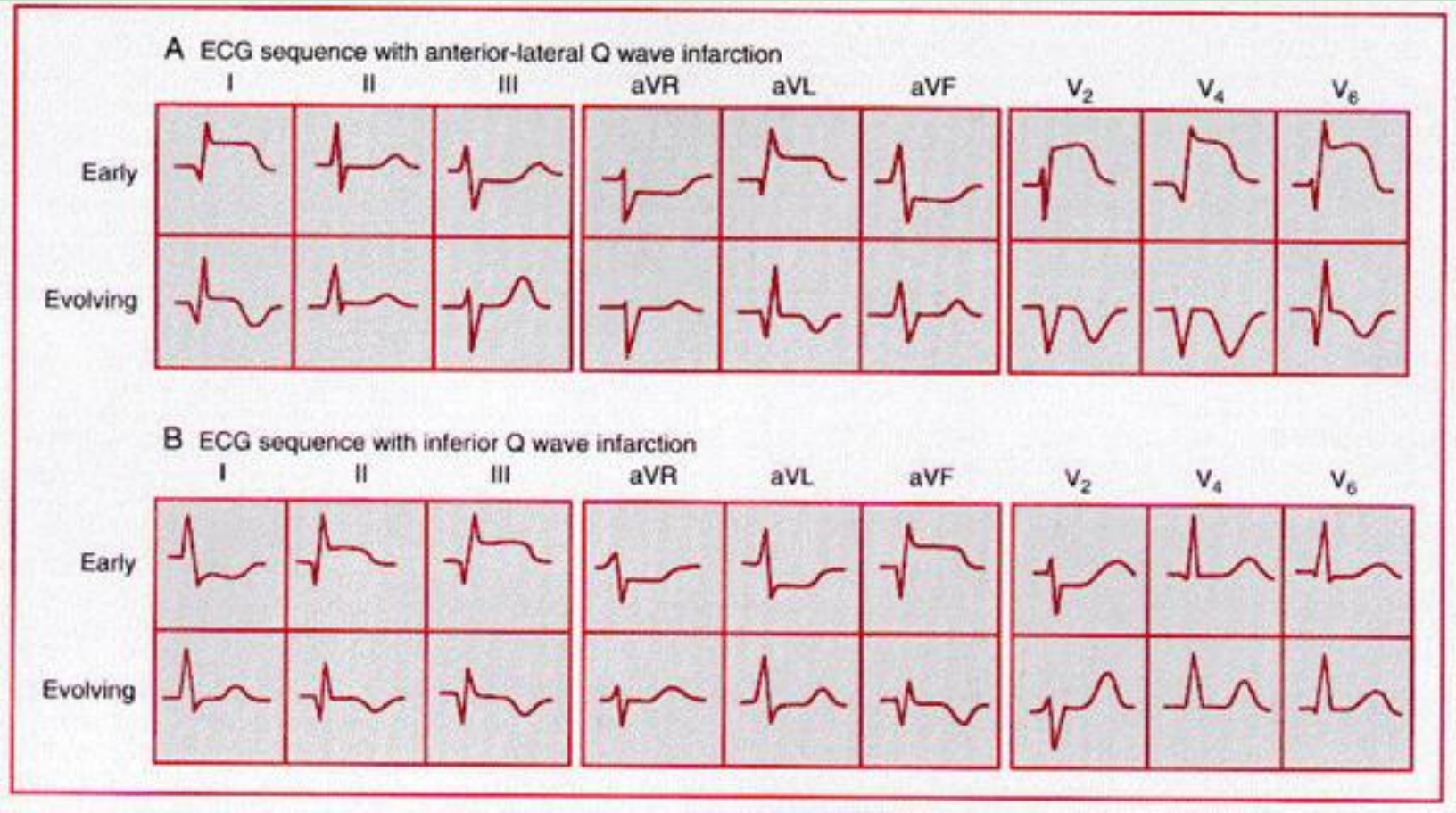
Cardiac Enzyme & EKG



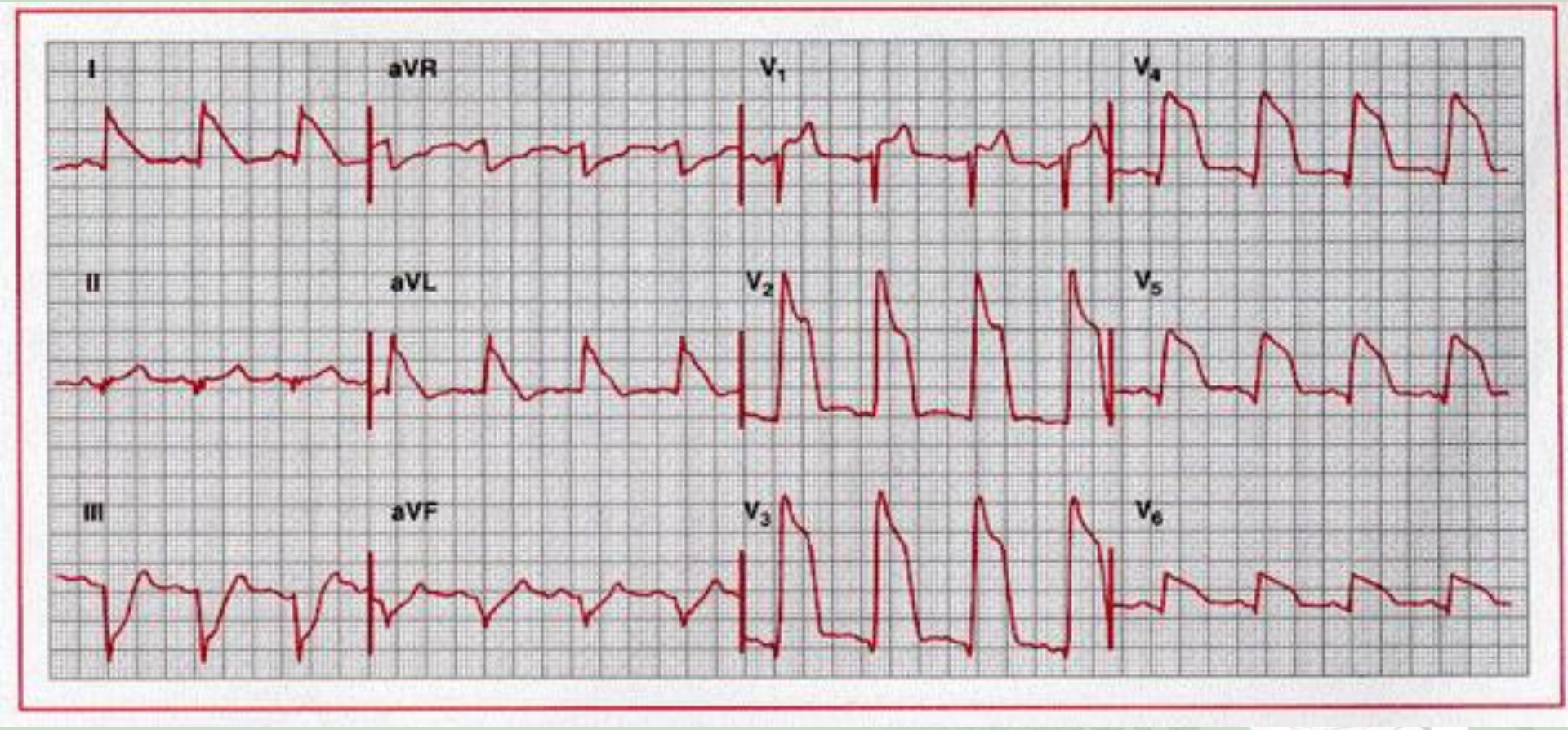
Rhythm Abnormality in MI

- VT/Vf
- PAF
- AIVT
- AV block
- Sinus bradicardia (Bezold-Jarisch Reflex)
- Sinus tacycardia

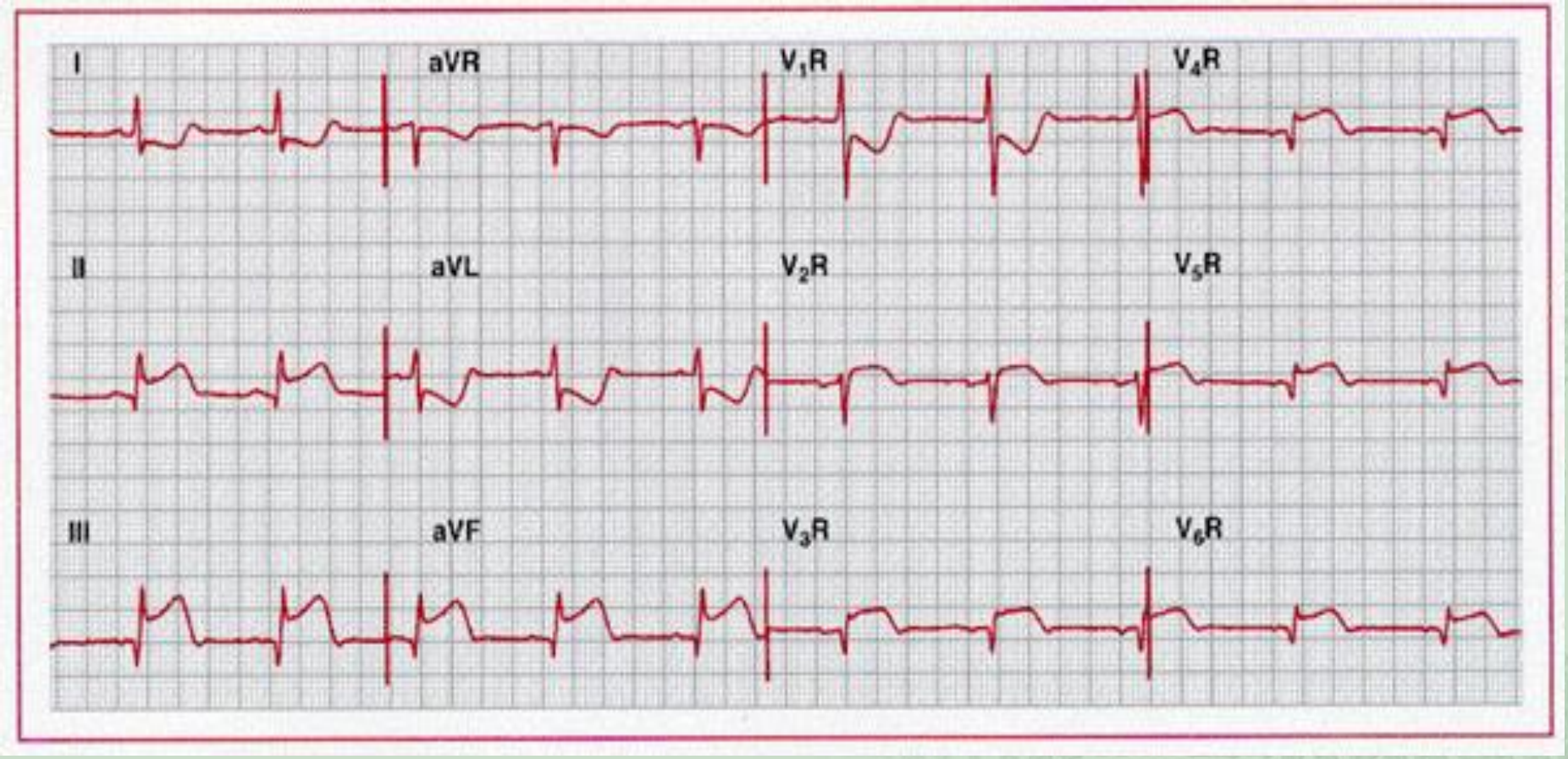
EKG Evolution of AMI



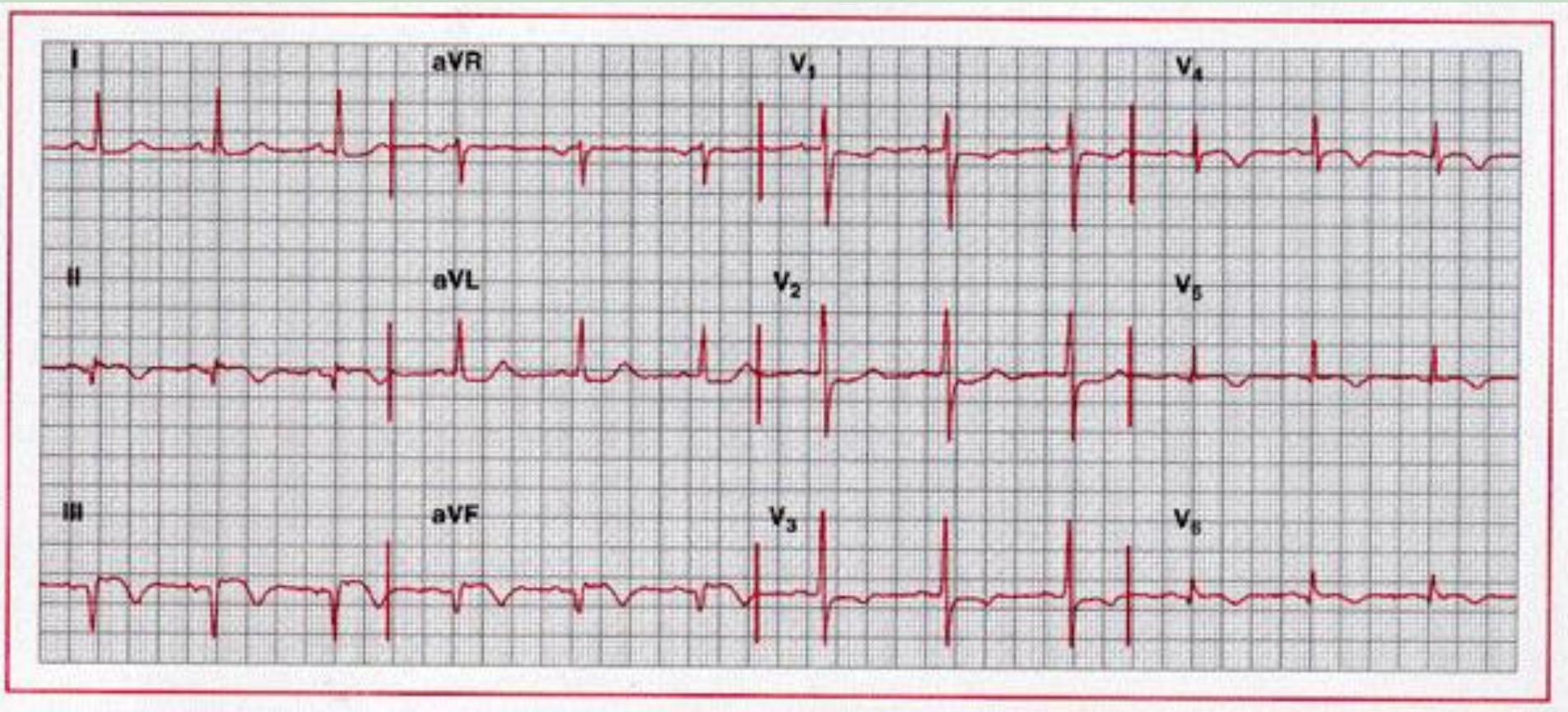
Ant. Wall MI with Hyperacute T Wave



Inferior Wall AMI with RV infarction



Post.inferior wall MI



Myoischemia – AMI & LBBB

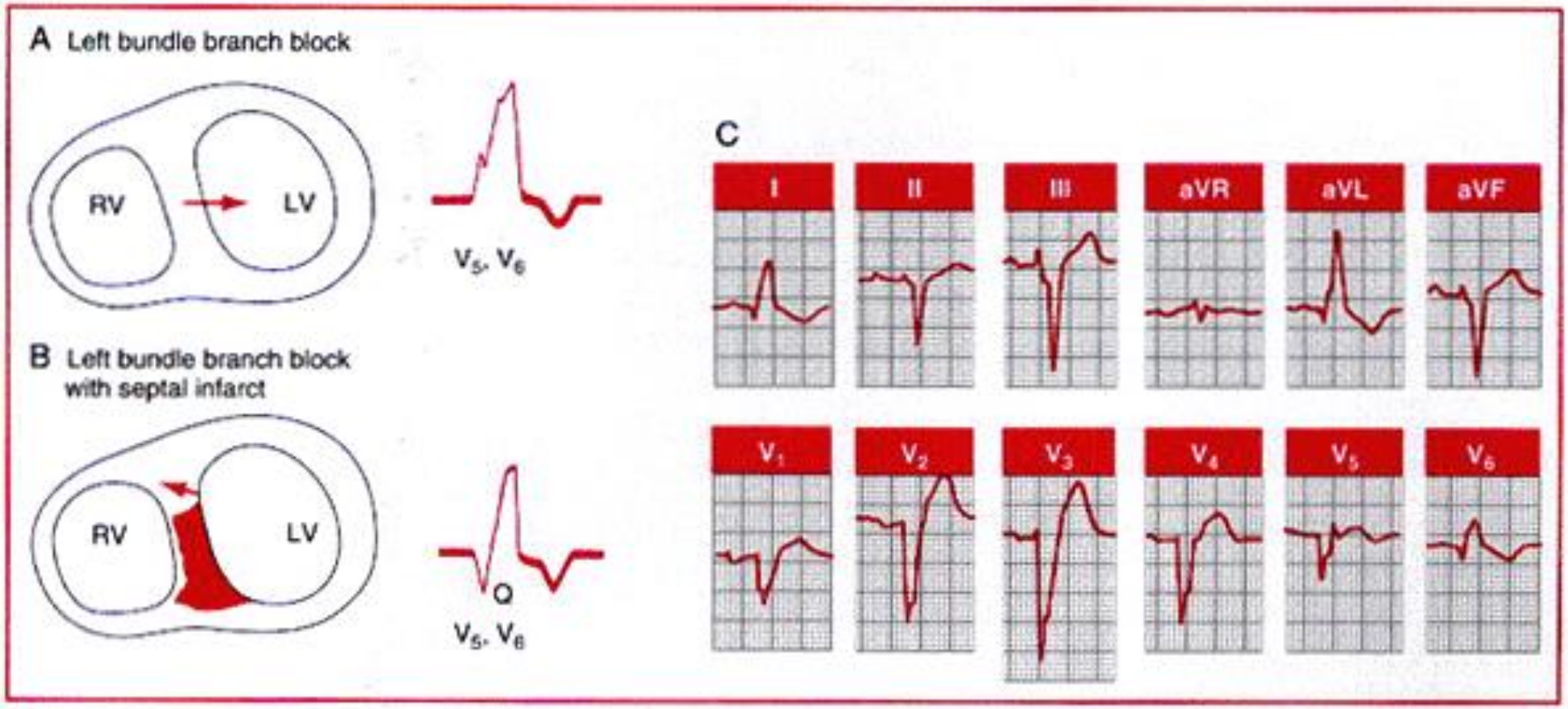
EKG criteria for AMI in presence of LBBB :

Criterion	Score
ST segment elevation > 1 mm concordant with QRS	5
ST segment depression > 1 mm in lead V1, V2 or V3	3
ST segment elevation > 5 mm discordant with QRS	2

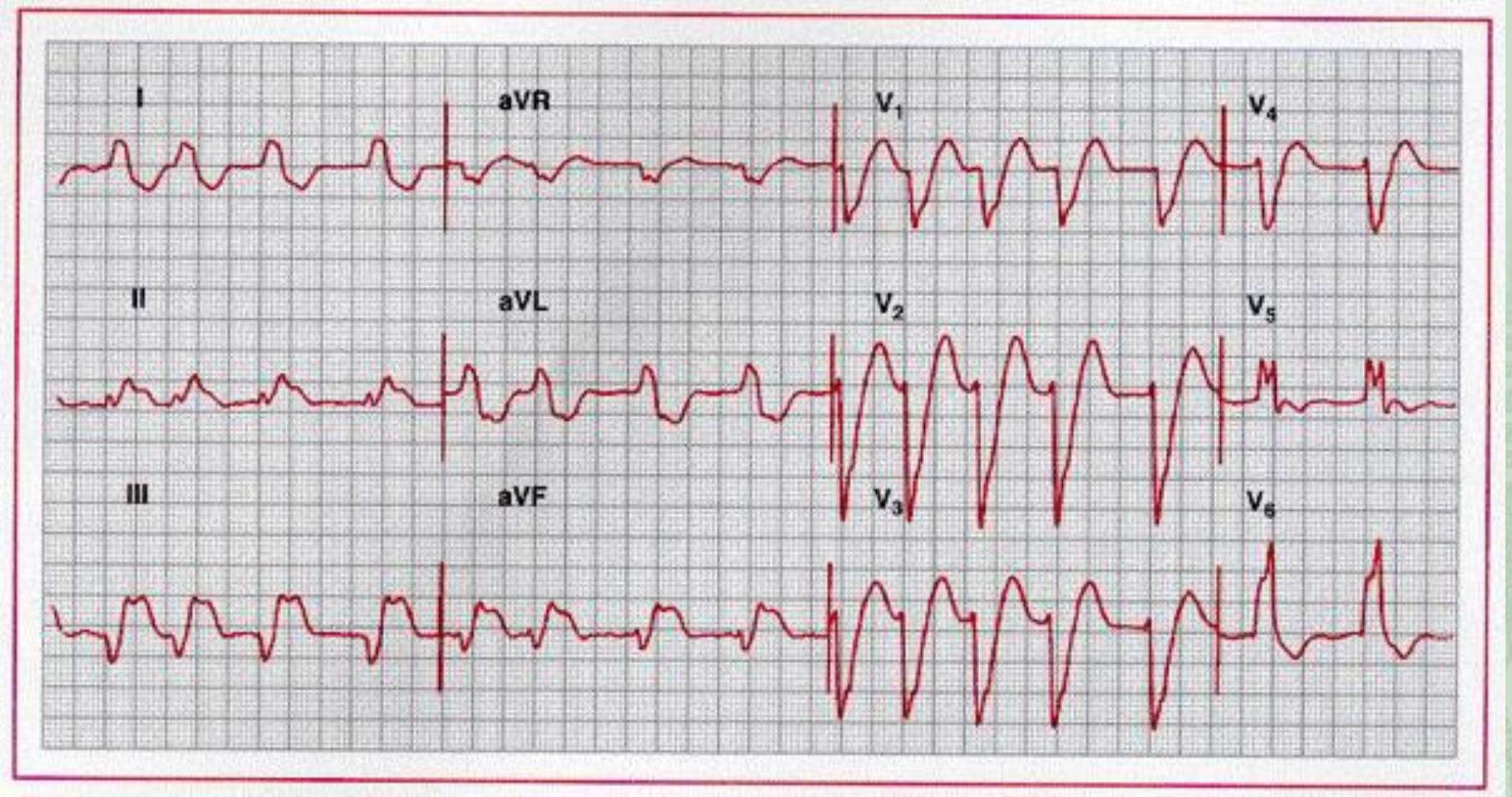
Score > 3 => specificity > 90% and 88% positive predict value

NEJM 1996;334:481-487

Septal Infarct with LBBB



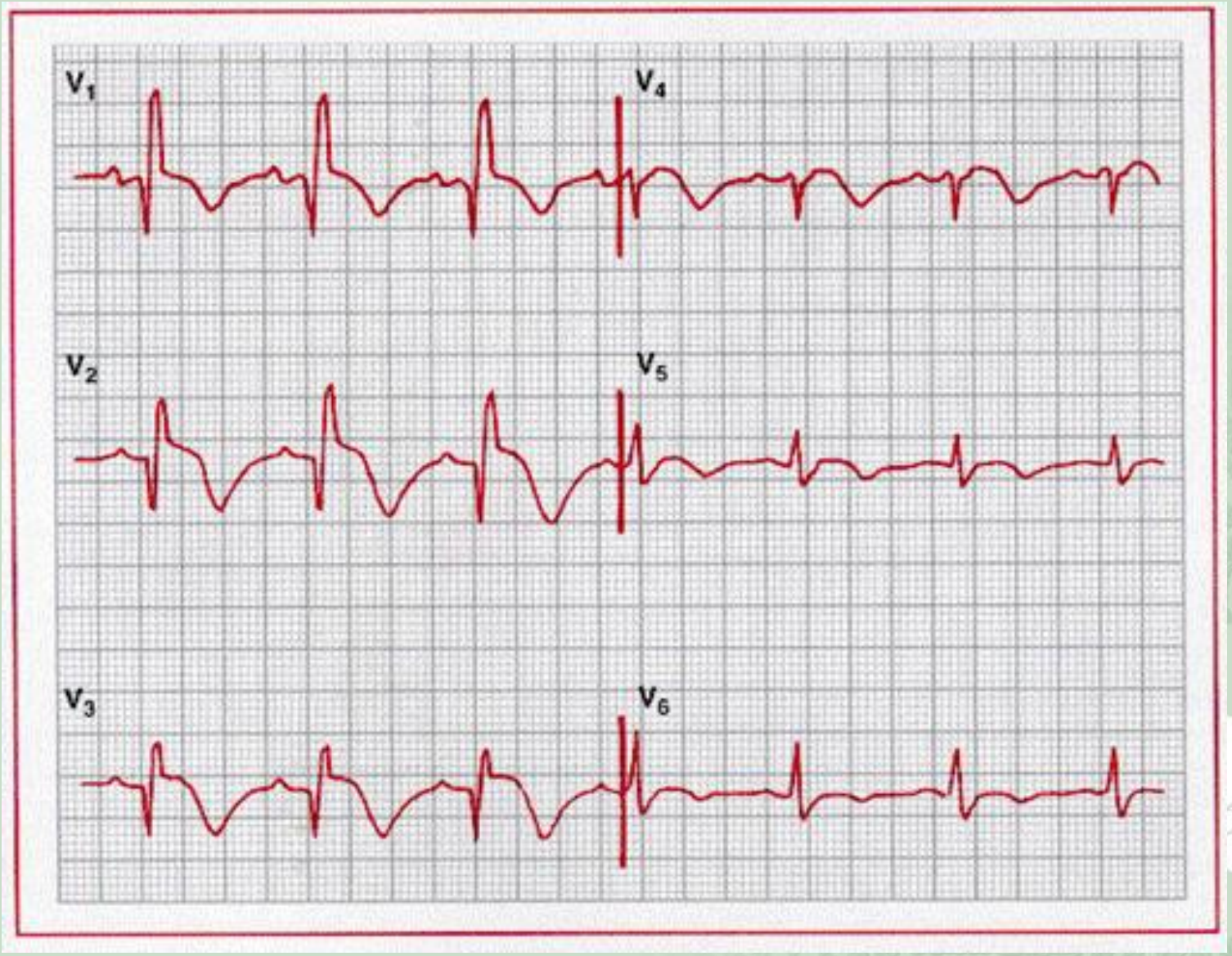
Inf. Wall AMI & CLBBB



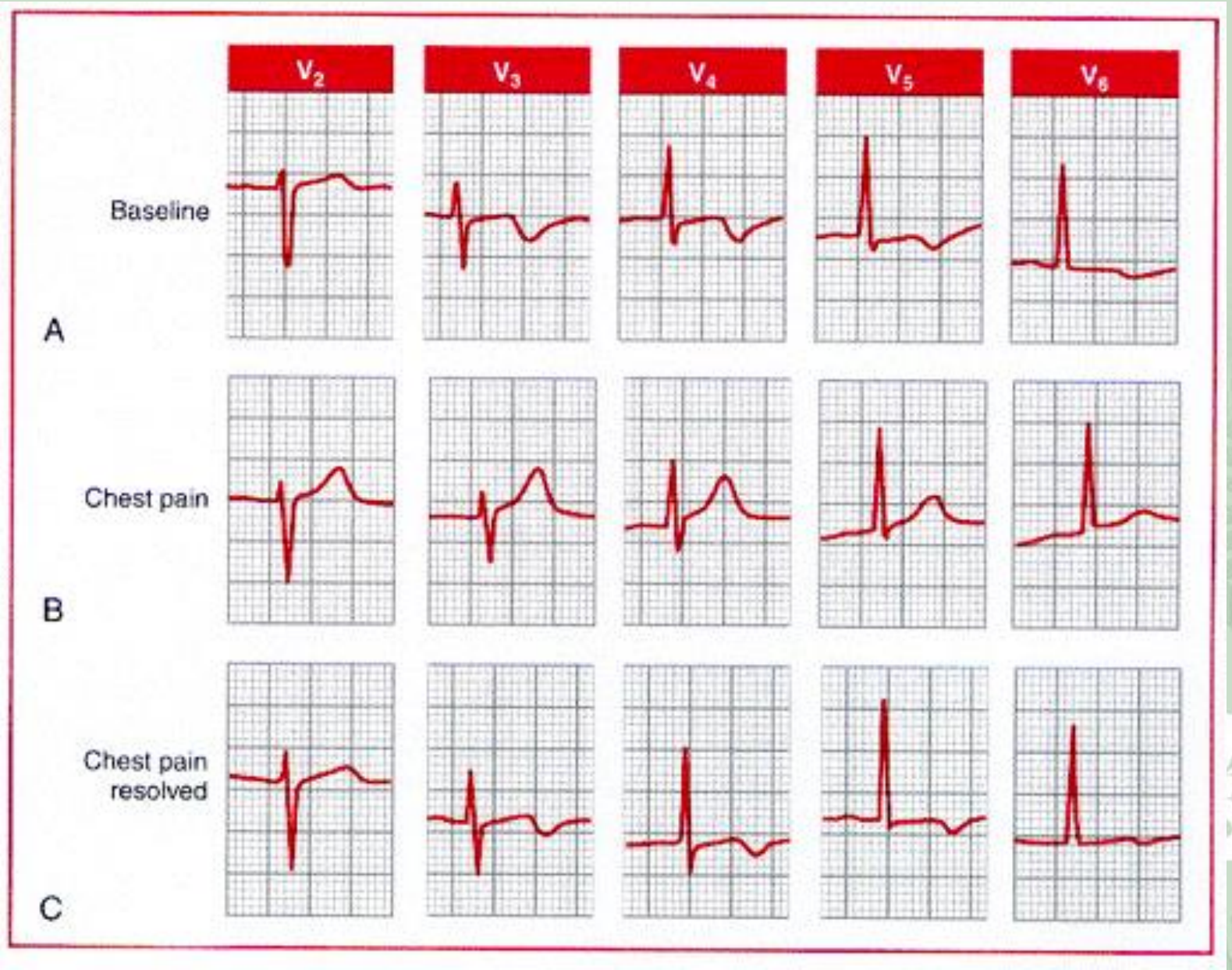
Myoischemia – AMI & RBBB

- Pseudonormalization of original inverted T wave in V1-V4 lead

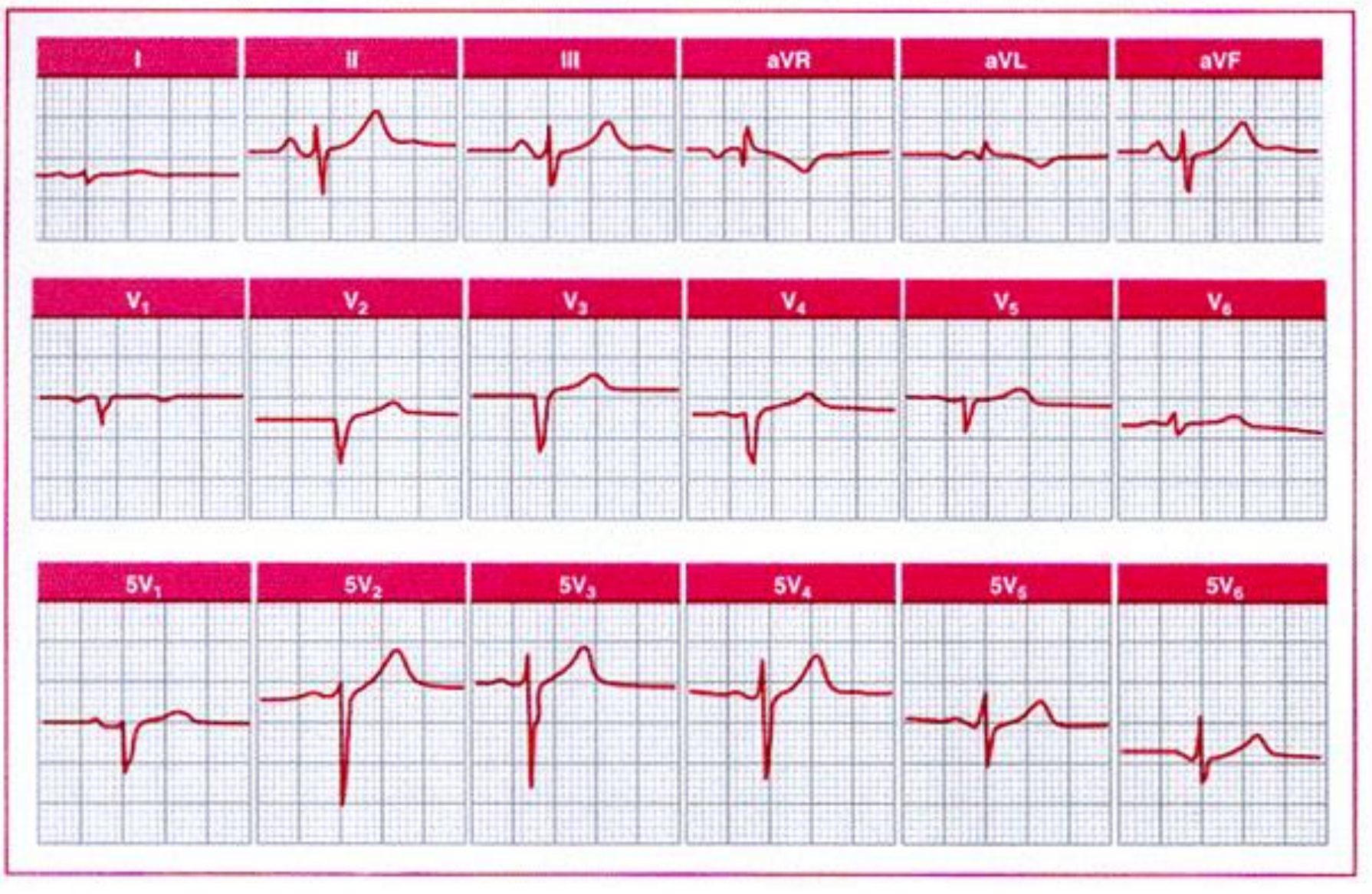
Ant. Wall MI & CRBBB



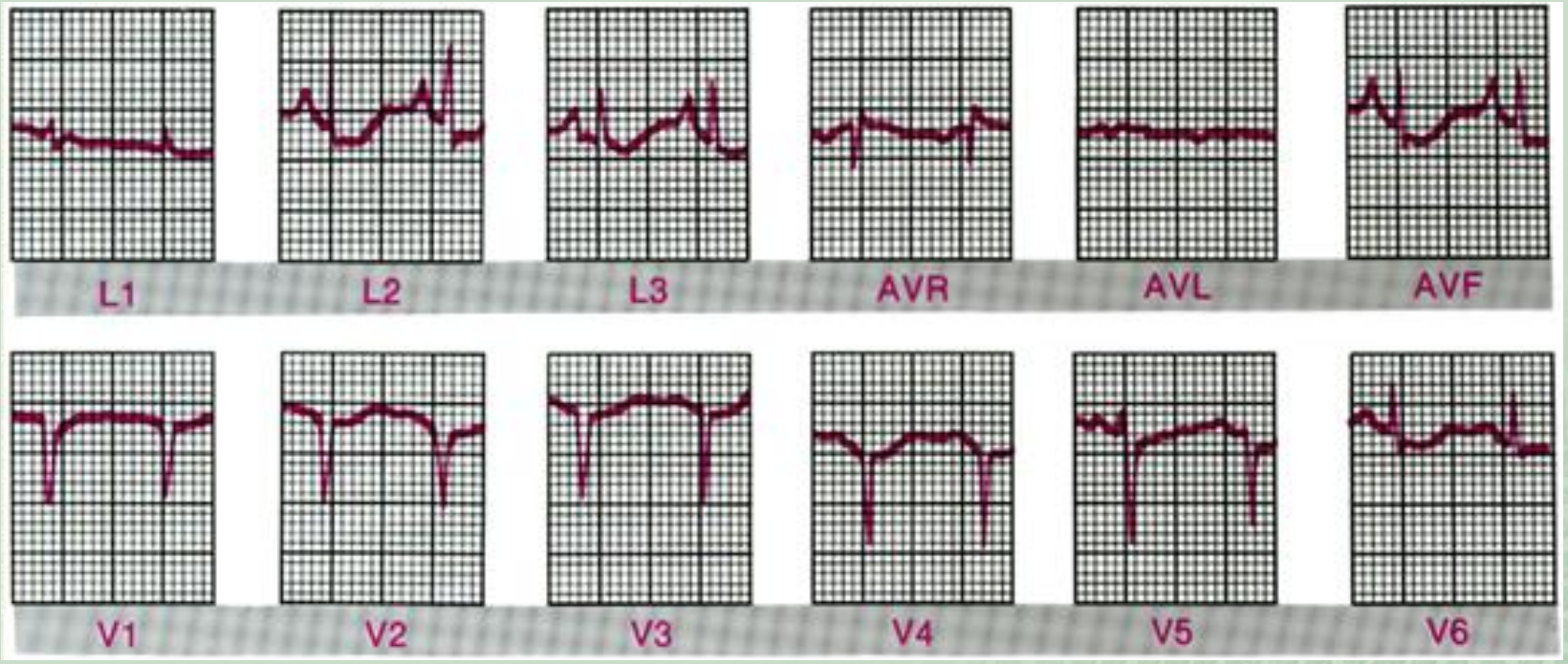
Myoischemia - – Pseudo-normalization



Psuedoinfarction



COPD



- Pointed tall T in Lead II => RAE
- RAD
- CWR
- PRWP at V1-V5 => mimic anteroseptal MI
- Psudo-ST depression at V6 => atrial repolarization

THANK YOU!

感謝聆聽