کلیاتی در مورد تفسیر الکتروکاردیوگرام

و

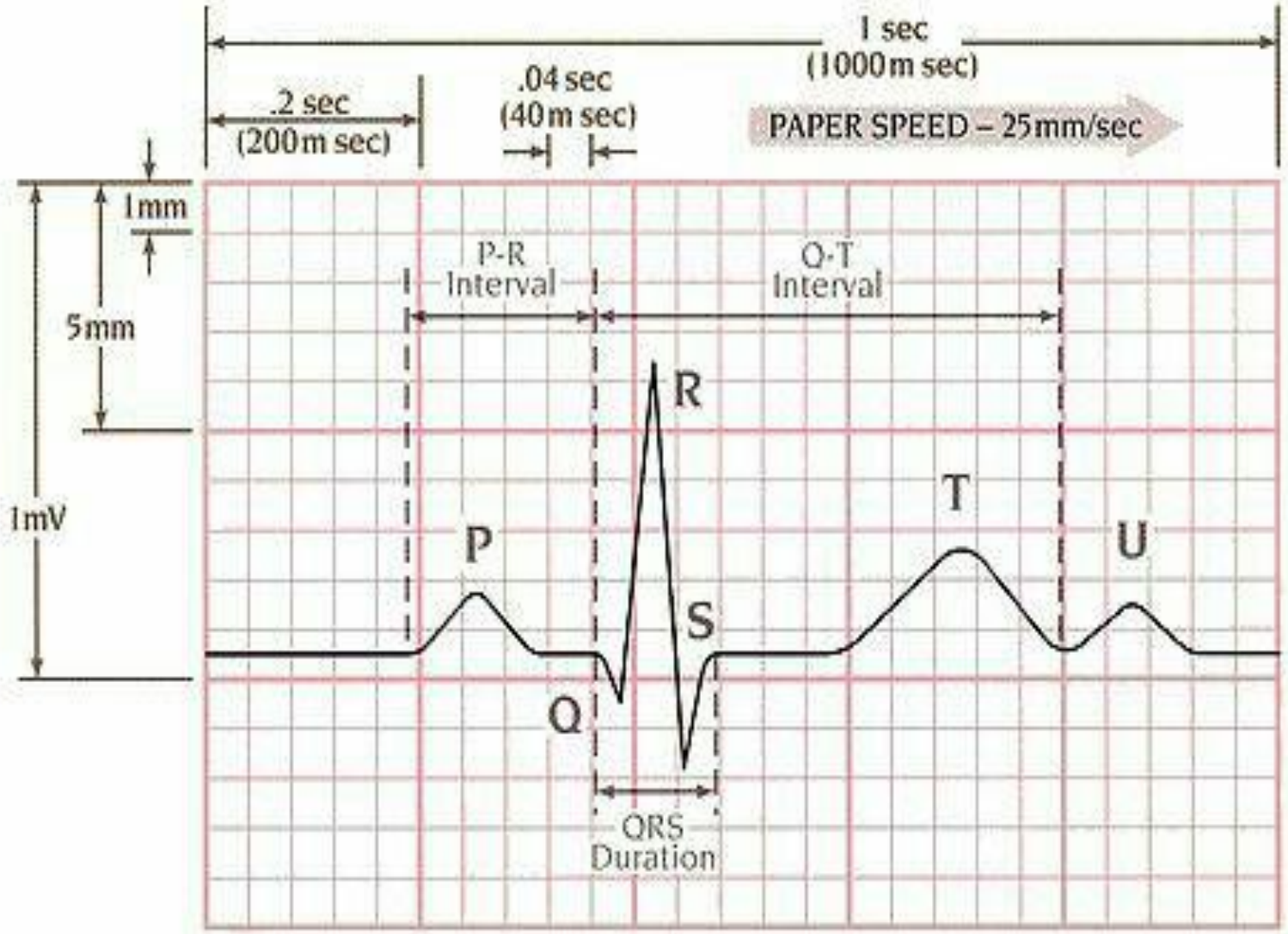
آریتمی های شایع

دانشگاه علوم پزشکی جندی شاپور اهواز
مرکز آموزشی – درمانی آیت ا... طالقانی (ره) اهواز

بهار ۱۳۹۷

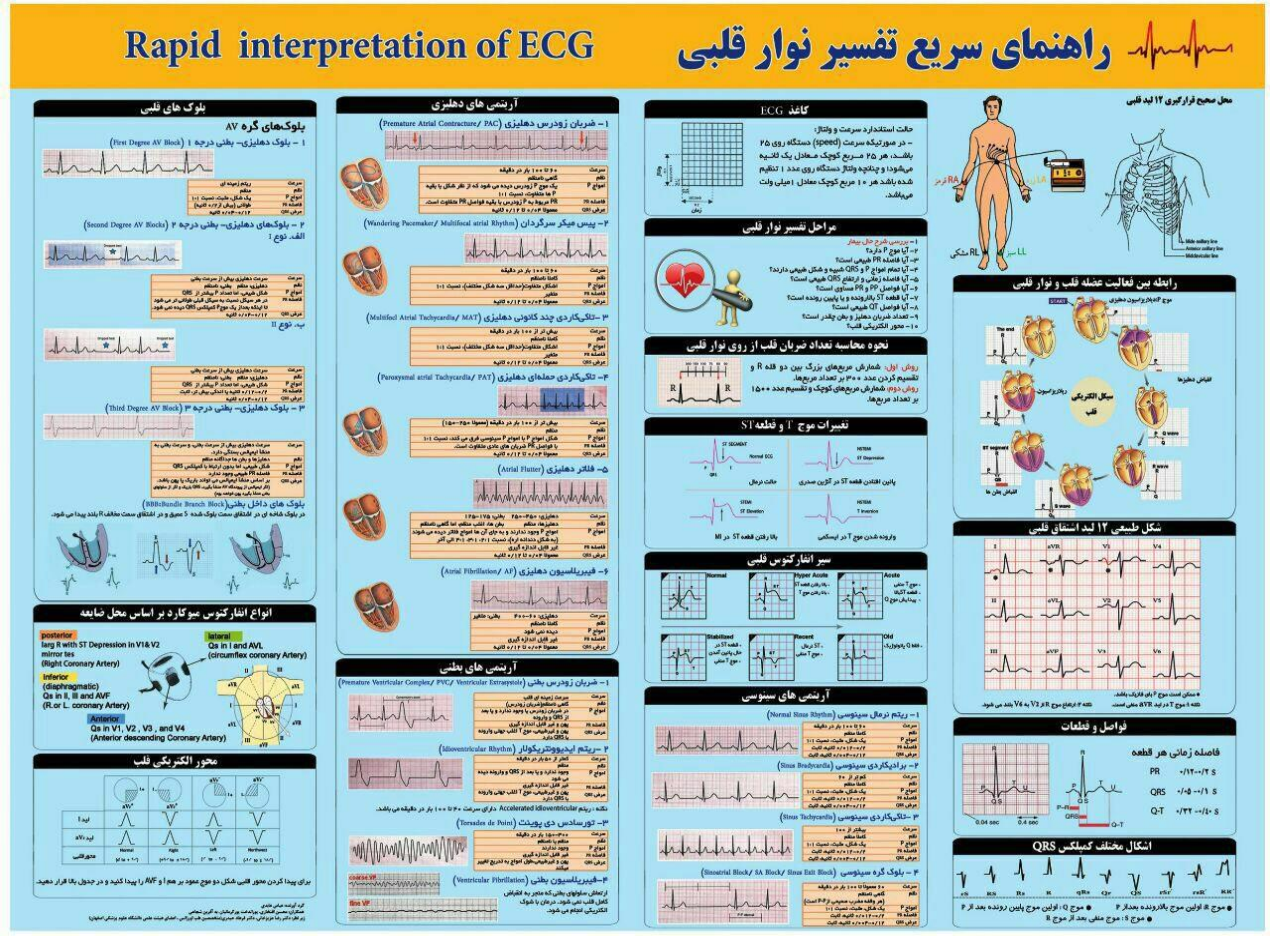
شهرام خمیسی - کارشناس ارشد پرستاری

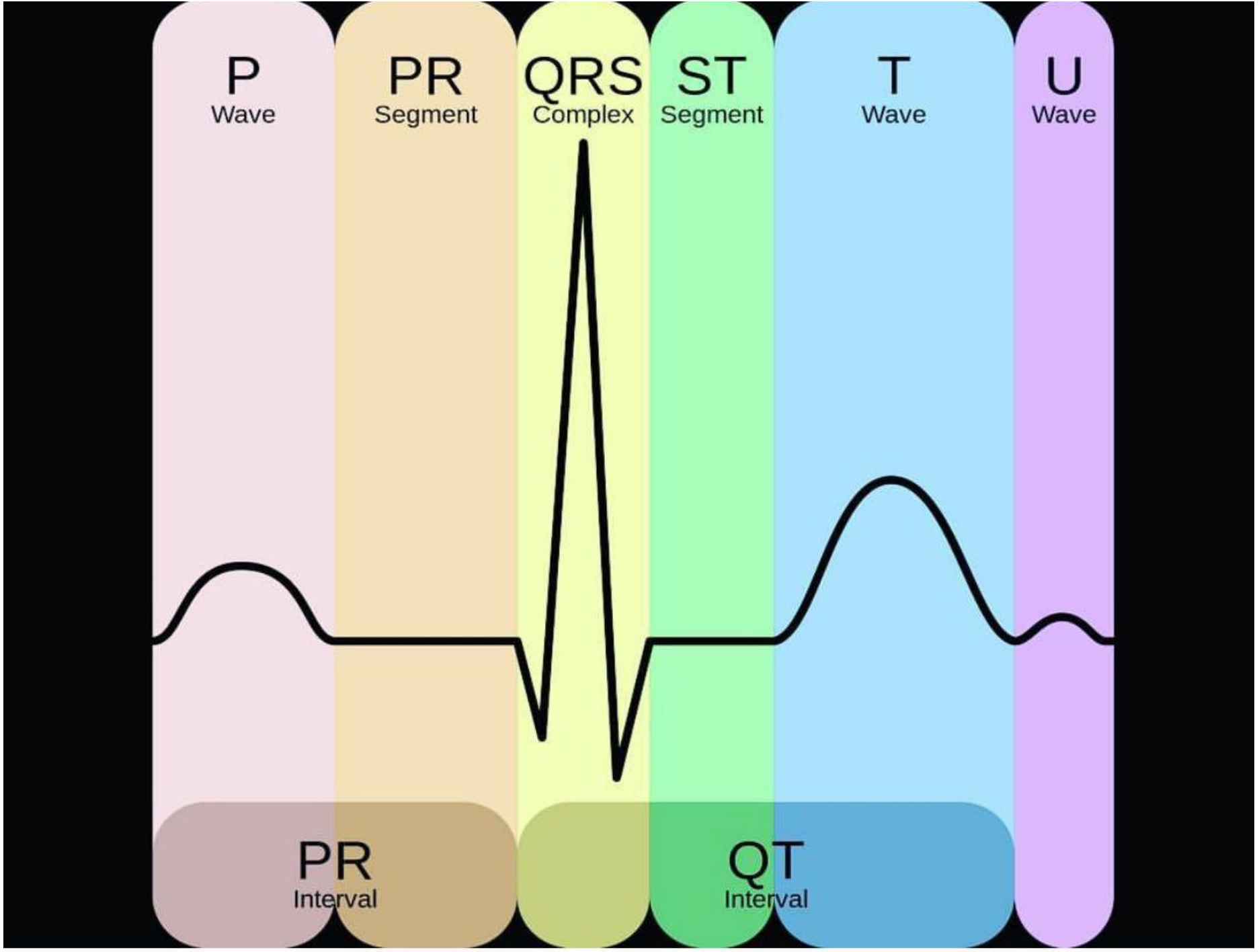
Shahram khamissi
MSN – BSN



VERTICAL AXIS	1 Small Square	= 1mm (0.1mV)	
	1 Large Square	= 5mm (0.5mV)	
	2 Large Squares	= 1mV	

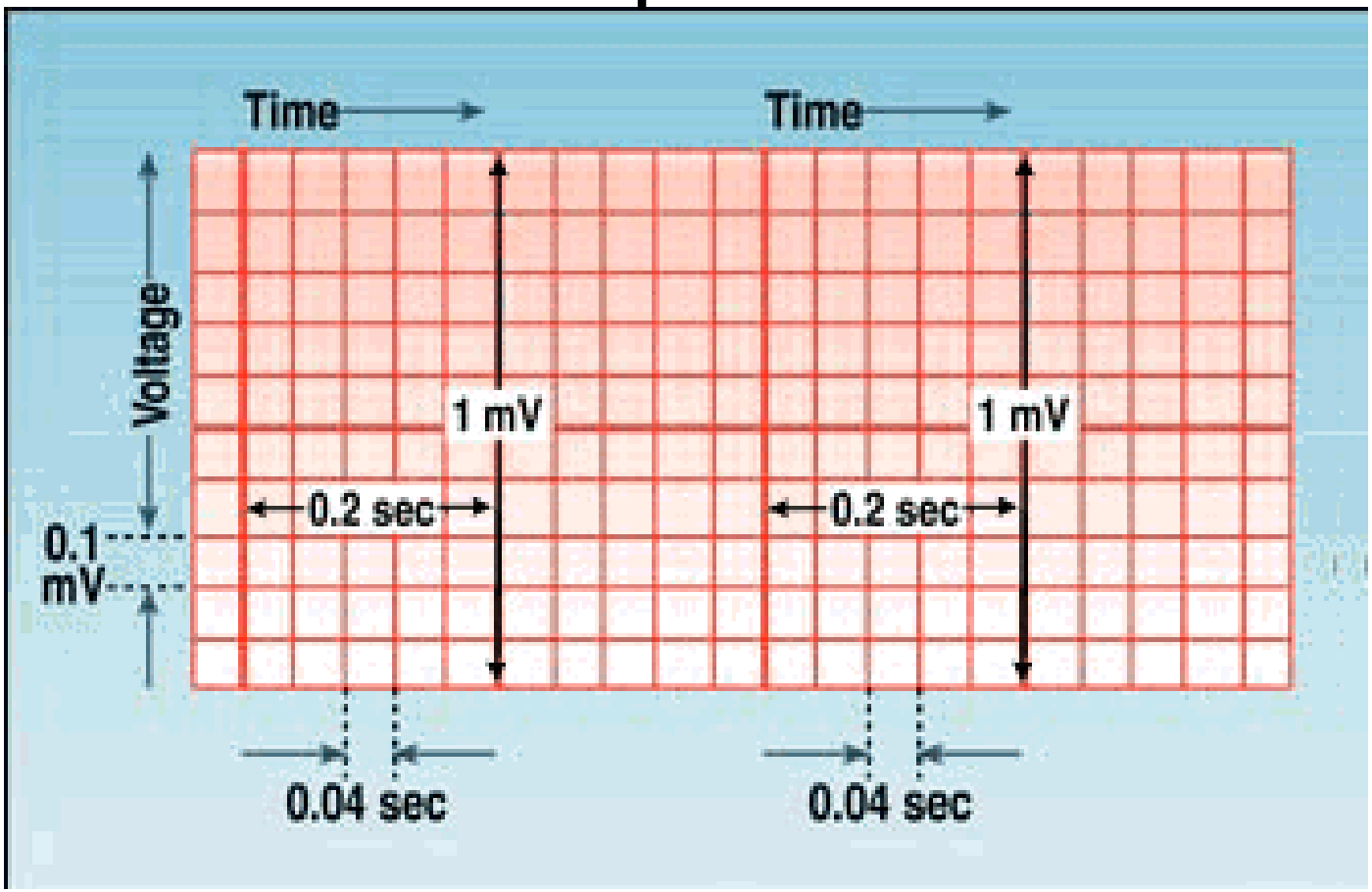
HORIZONTAL AXIS	1 Small Square	= .04 sec (40 m sec)
	1 Large Square	= .2 sec (200 m sec)
	5 Large Squares	= 1 sec (1000 m sec)


راهنمای سریع تفسیر نوار قلبی
Rapid interpretation of ECG
محل صحیح قرارگیری ۱۲ لید قلبی
RA قرمز
LA زرد
RL مشکی
LL سبز
کاغذ ECG
حالت استاندارد سرعت و ولتاژ:
– در صورتیکه سرعت (speed) دستگاه روی ۲۵ باشد، هر ۲۵ مربع کوچک معادل یک ثانیه می‌شود و چنانچه ولتاژ دستگاه روی عدد ۱ تنظیم شده باشد هر ۱۰ مربع کوچک معادل ۱ میلی ولت می‌باشد.
زمان
مراحل تفسیر نوار قلبی
۱– بررسی شرح حال بیمار
۲– آیا موج P دارد؟
۳– آیا فاصله PR طبیعی است؟
۴– آیا تمام امواج P و QRS شبیه و شکل طبیعی دارند؟
۵– آیا فاصله زمانی و ارتفاع QRS طبیعی است؟
۶– آیا فواصل PP و PR مساوی است؟
۷– آیا قطعه ST بالارونده و یا پایین رونده است؟
۸– آیا فاصله QT طبیعی است؟
۹– تعداد ضربان دهلیز و بطن چقدر است؟
۱۰– محور الکتریکی قلب؟
نحوه محاسبه تعداد ضربان قلب از روی نوار قلبی
روش اول: شمارش مربع‌های بزرگ بین دو قله R و تقسیم کردن عدد ۳۰۰ بر تعداد مربع‌ها.
روش دوم: شمارش مربع‌های کوچک و تقسیم عدد ۱۵۰۰ بر تعداد مربع‌ها.
تغییرات موج T و قطعه ST
ST SEGMENT
Normal ECG
حالت نرمال
پایین افتادن قطعه ST در آنژین صدری
بالا رفتن قطعه ST در MI
وارونه شدن موج T در ایسکمی
ST Depression
ST Elevation
T inversion
سیر انفارکتوس قلبی
Normal
Hyper Acute
Acute
Stabilized
Recent
Old
آریتمی های سینوسی
۱– ریتم نرمال سینوسی (Normal Sinus Rhythm)
۲– برادیکاردی سینوسی (Sinus Bradycardia)
۳– تاکیکاردی سینوسی (Sinus Tachycardia)
۴– بلوک گره سینوسی (Sinoatrial Block/ SA Block/ Sinus Exit Block)
رابطه بین فعالیت عضله قلب و نوار قلبی
START
The end
سیکل الکتریکی قلب
انقباض دهلیزها
انقباض بطن ها
ST segment
Q wave
R wave
S wave
شکل طبیعی ۱۲ لید اشتقاق قلبی
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
فواصل و قطعات
فاصله زمانی هر قطعه
PR ۰/۱۲–۰/۲ s
QRS ۰/۰۵–۰/۱ s
Q-T ۰/۳۲–۰/۴ s
0.04 sec
0.4 sec
اشکال مختلف کمپلکس QRS
rS
RS
Rs
R
qRs
Qr
QS
rSr'
rsR'
RR'
● موج R: اولین موج بالارونده بعد از P
● موج Q: اولین موج پایین رونده بعد از P
● موج S: موج منفی بعد از موج R
بلوک های قلبی
بلوک‌های گره AV
۱ – بلوک دهلیزی– بطنی درجه ۱ (First Degree AV Block)
۲ – بلوک‌های دهلیزی– بطنی درجه ۲ (Second Degree AV Blocks)
الف. نوع I
ب. نوع II
۳ – بلوک دهلیزی– بطنی درجه ۳ (Third Degree AV Block)
بلوک های داخل بطنی (BBB:Bundle Branch Block)
در بلوک شاخه ای در اشتقاق سمت بلوک شده S عمیق و در اشتقاق سمت مخالف R بلند پیدا می شود.
انواع انفارکتوس میوکارد بر اساس محل ضایعه
posterior
larg R with ST Depression in V1& V2
mirror tes
(Right Coronary Artery)
lateral
Qs in I and AVL
(circumflex coronary Artery)
Inferior
(diaphragmatic)
Qs in II, III and AVF
(R.or L. coronary Artery)
Anterior
Qs in V1, V2 , V3 , and V4
(Anterior descending Coronary Artery)
محور الکتریکی قلب
لید I
لید aVF
محور قلبی
Normal
Right
Left
Northwest
برای پیدا کردن محور قلبی شکل دو موج عمود بر هم I و AVF را پیدا کنید و در جدول بالا قرار دهید.
آریتمی های دهلیزی
۱ – ضربان زودرس دهلیزی (Premature Atrial Contracture/ PAC)
۲– پیس میکر سرگردان (Wandering Pacemaker/ Multifocal atrial Rhythm)
۳ –تاکیکاردی چند کانونی دهلیزی (Multifocal Atrial Tachycardia/ MAT)
۴– تاکیکاردی حمله‌ای دهلیزی (Paroxysmal atrial Tachycardia/ PAT)
۵– فلاتر دهلیزی (Atrial Flutter)
۶– فیبریلاسیون دهلیزی (Atrial Fibrillation/ AF)
آریتمی های بطنی
۱– ضربان زودرس بطنی (Premature Ventricular Complex/ PVC/ Ventricular Extrasystole)
۲ –ریتم ایدیوونتریکولار (Idioventricular Rhythm)
نکته: ریتم Accelerated idioventricular دارای سرعت ۴۰ تا ۱۰۰ بار در دقیقه می باشد.
۳– تورسادس دی پوینت (Torsades de Point)
۴–فیبریلاسیون بطنی (Ventricular Fibrillation)
coarse VF
fine VF
ارتعاش سلولهای بطنی که منجر به انقباض کامل قلب نمی شود. درمان با شوک الکتریکی انجام می شود.


P
Wave
PR
Segment
QRS
Complex
ST
Segment
T
Wave
U
Wave
PR
Interval
QT
Interval

ECG Graph Paper

Y- Axis Amplitude in mill volts



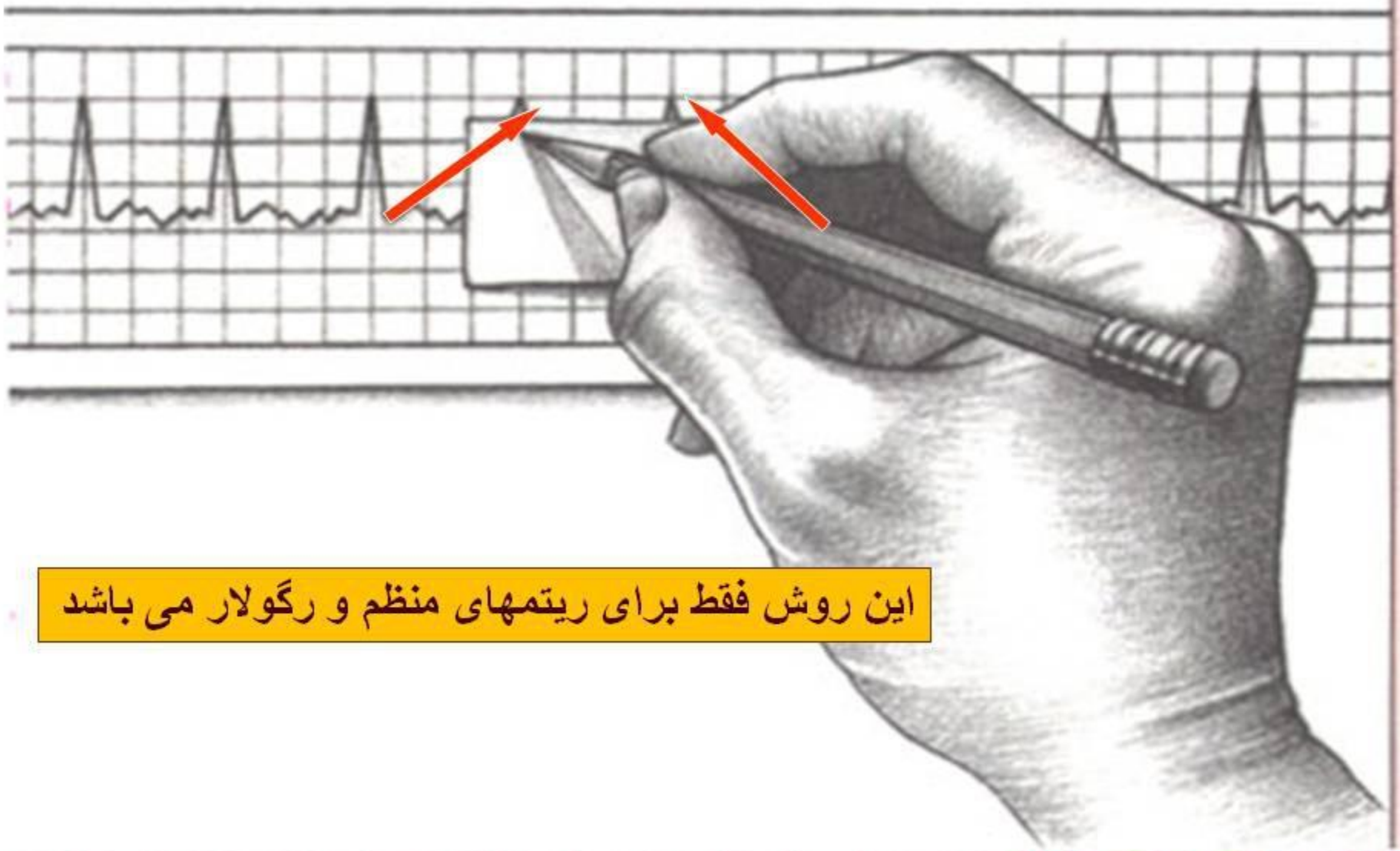
X- Axis time in seconds

Rate Determination

No. of Big Boxes	R – R Interval	Rate Cal.	Rate
One	0.2 sec	60 ÷ 0.2	300
Two	0.4 sec	60 ÷ 0.4	150
Three	0.6 sec	60 ÷ 0.6	100
Four	0.8 sec	60 ÷ 0.8	75
Five	1.0 sec	60 ÷ 1.0	60
Six	1.2 sec	60 ÷ 1.2	50
Seven	1.4 sec	60 ÷ 1.4	43
Eight	1.6 sec	60 ÷ 1.6	37

روش اول

$$\text{تعداد ضربانات قلبی} = \frac{300}{\text{تعداد خانه های بزرگ}}$$



این روش فقط برای ریتمهای منظم و رگولار می باشد

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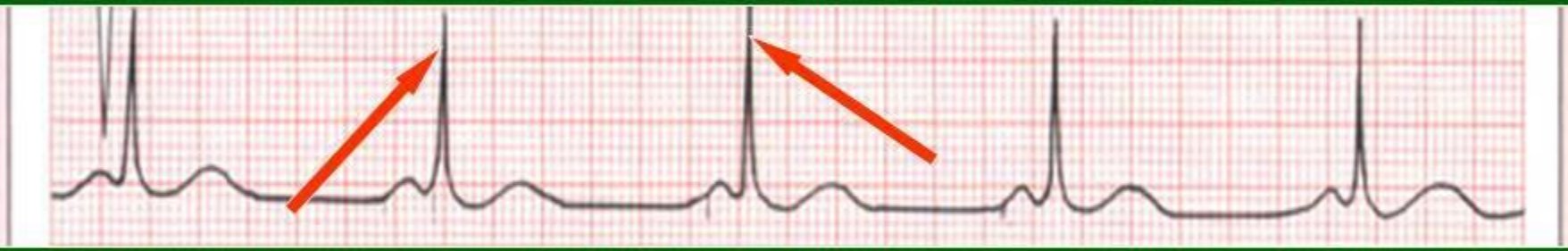
روش دوم

$$\text{تعداد ضربانات قلبی} = \frac{1500}{\text{تعداد خانه های کوچک}}$$

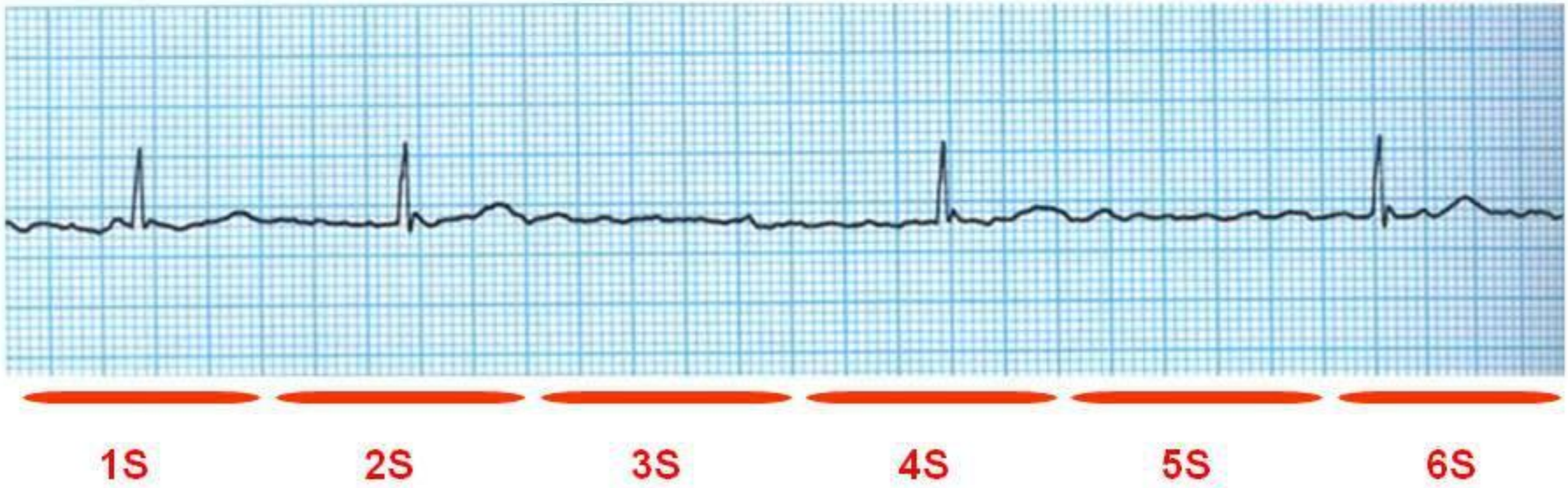


این روش فقط برای ریتمهای منظم و رگولار می باشد

روش چهارم

10 × تعداد کمپلکس QRS در 6 ثانیه = تعداد ضربانات قلبی

این روش فقط برای ریتمهای نا منظم و ایررگولار می باشد



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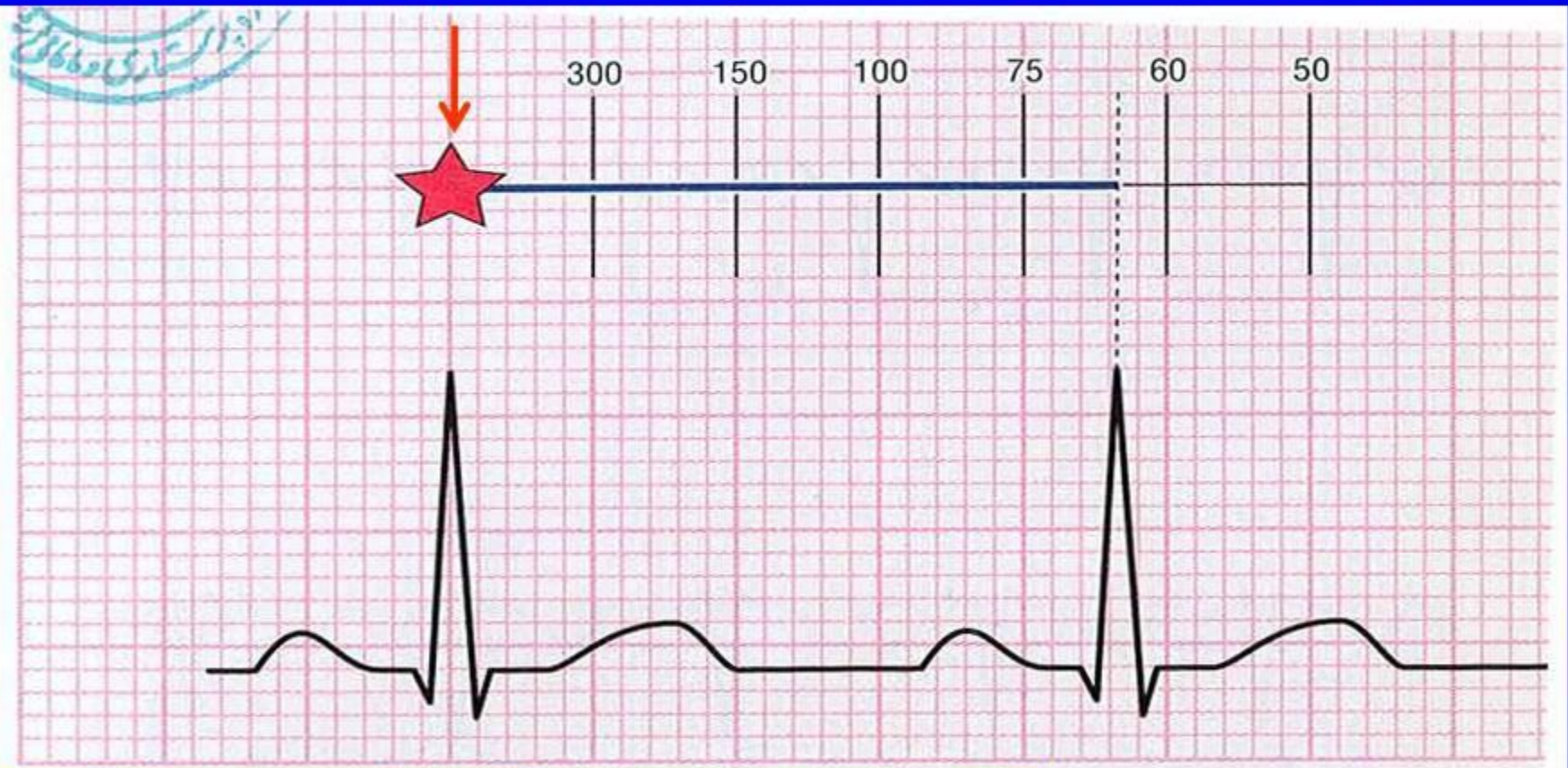
روش سوم

300 - 150 - 100 - 75 - 60 - 50



این روش فقط برای ریتمهای منظم و رگولار می باشد

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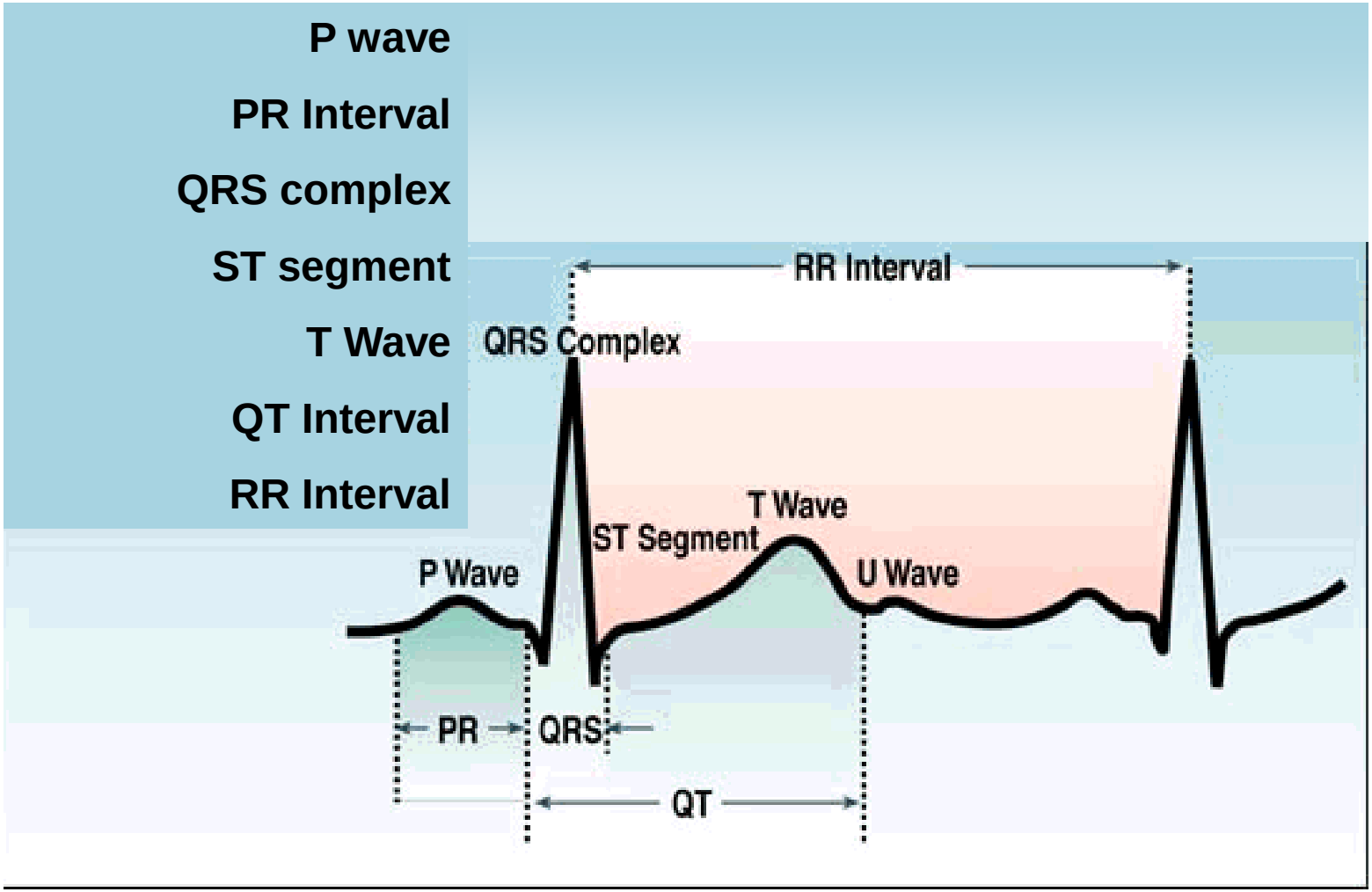
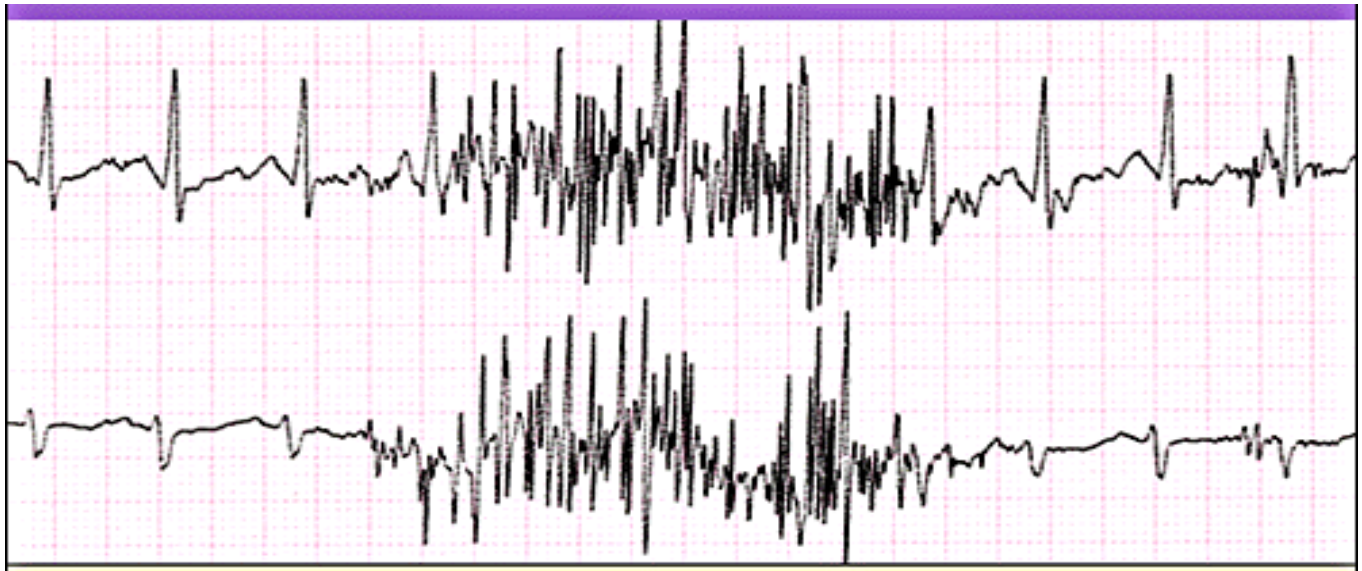
ECG Complex


P wave
PR Interval
QRS complex
ST segment
T Wave
QT Interval
RR Interval
RR Interval
QRS Complex
T Wave
ST Segment
P Wave
U Wave
PR
QRS
QT

Muscle Tremor



Electrical interference caused by the patient's tensed muscles.

- Limb movements cause baseline fluctuations
- Tense muscles cause tremor of baseline
- Hairy chest interferes with proper contact of chest leads – better to shave the area if needed.
- Reassurance, starting recording a few minutes after the leads are placed – reduce muscle tension

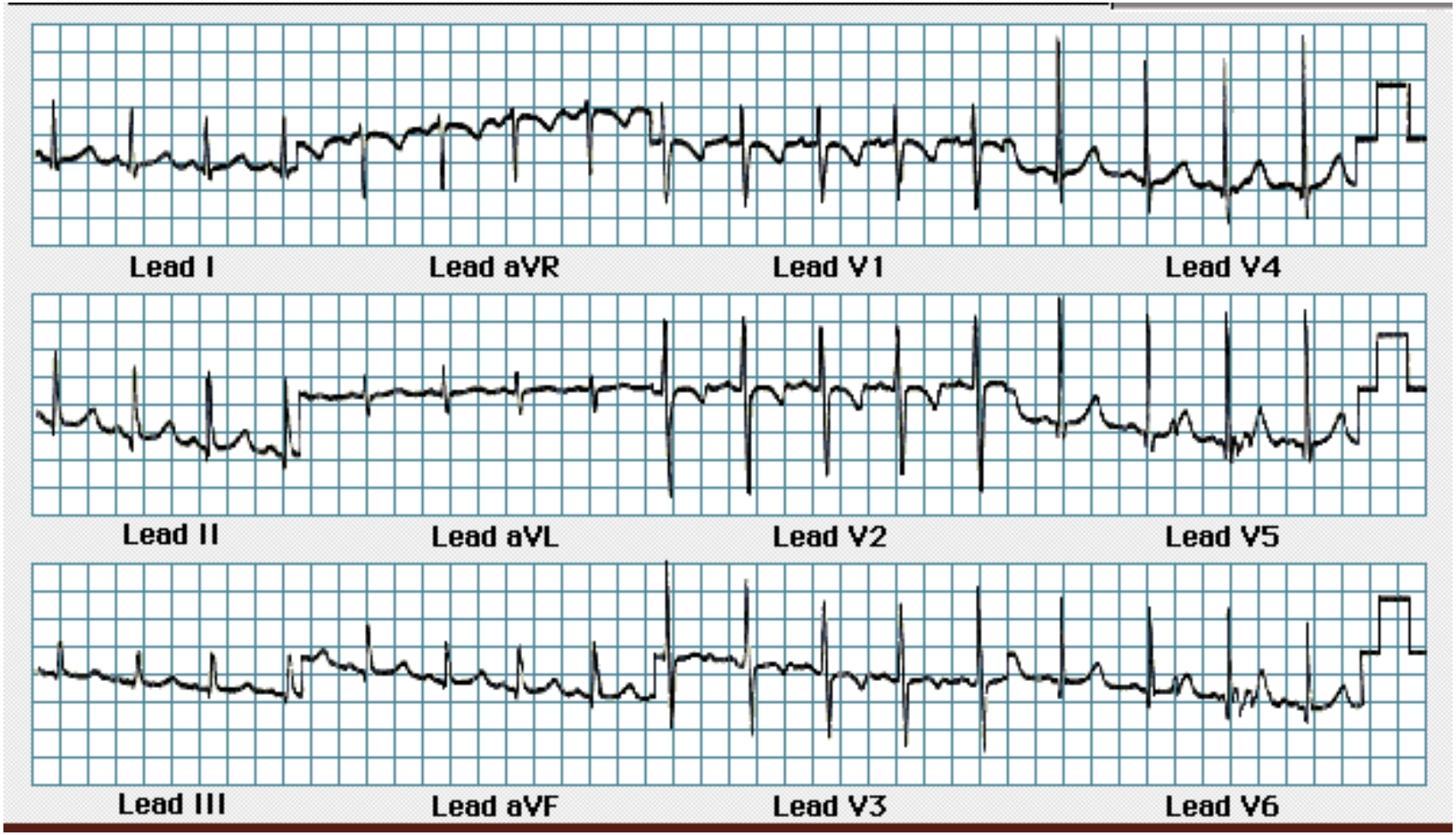
Normal ECG

- Standardization – 10 mm (2 boxes) = 1 mV
- Double and half standardization if required
- Sinus Rhythm – Each P followed by QRS, R-R constant
- P waves – always examine for in L2, V1, L1
- QRS positive in L1, L2, L3, aVF and aVL. – Neg in aVR
- QRS is < 0.08 narrow, Q in V5, V6 < 0.04, < 3 mm
- R wave progression from V1 to V6, QT interval < 0.4
- Axis normal – L1, L3, and aVF all will be positive
- ST Isoelectric, T waves ↑, Normal T↓ in aVR,V1, V2

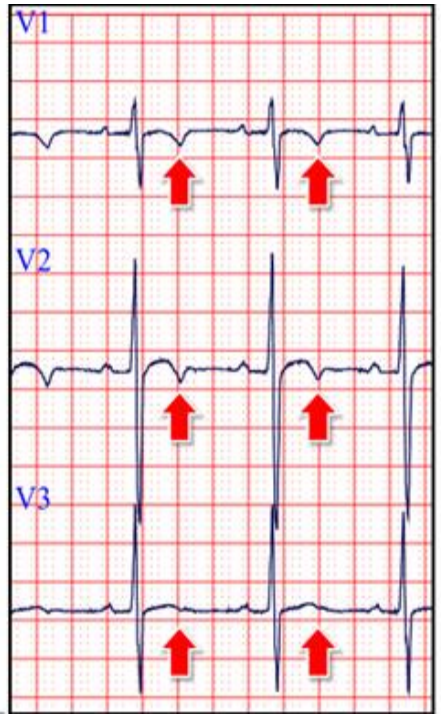
Pediatric ECG

- This is the ECG of a 6 year old child
- Heart rate is 100 – Normal for the age
- See V1 + V5 R >> 35 – Not LVH – Normal
- T↓ in V1, V2, V3 – Normal in child
- Base line disturbances in V5, V6 – due to movement by child

Pediatric ECG



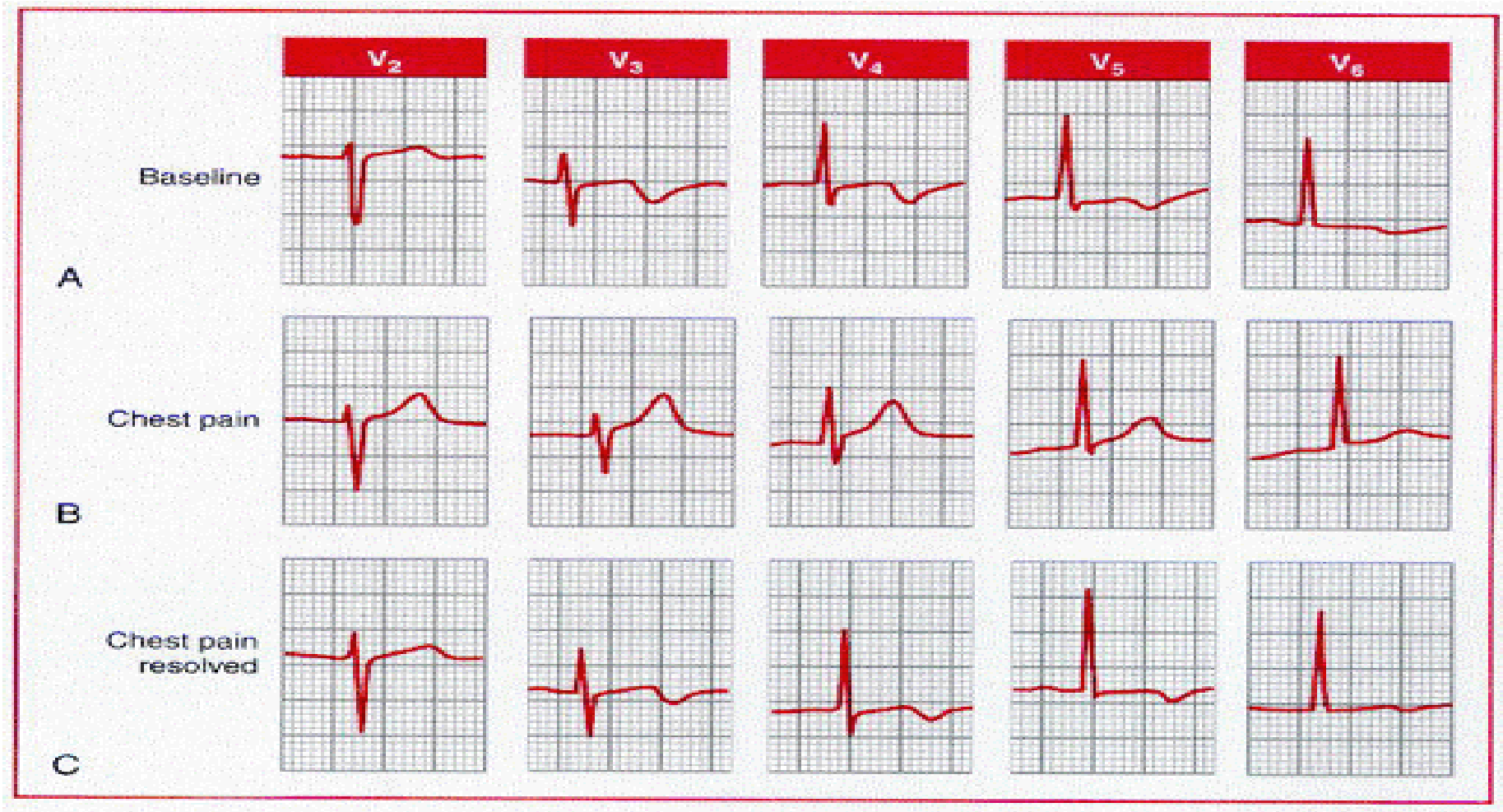
Persistent Juvenile Pattern

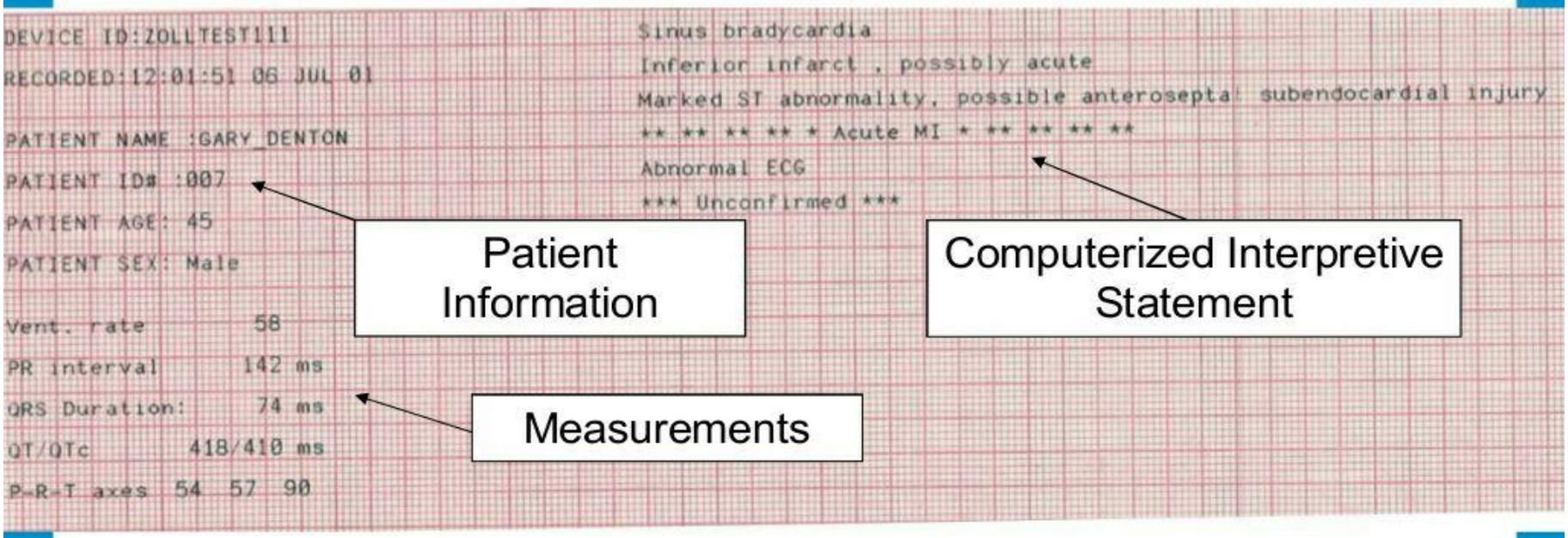


Normal Variations in ECG

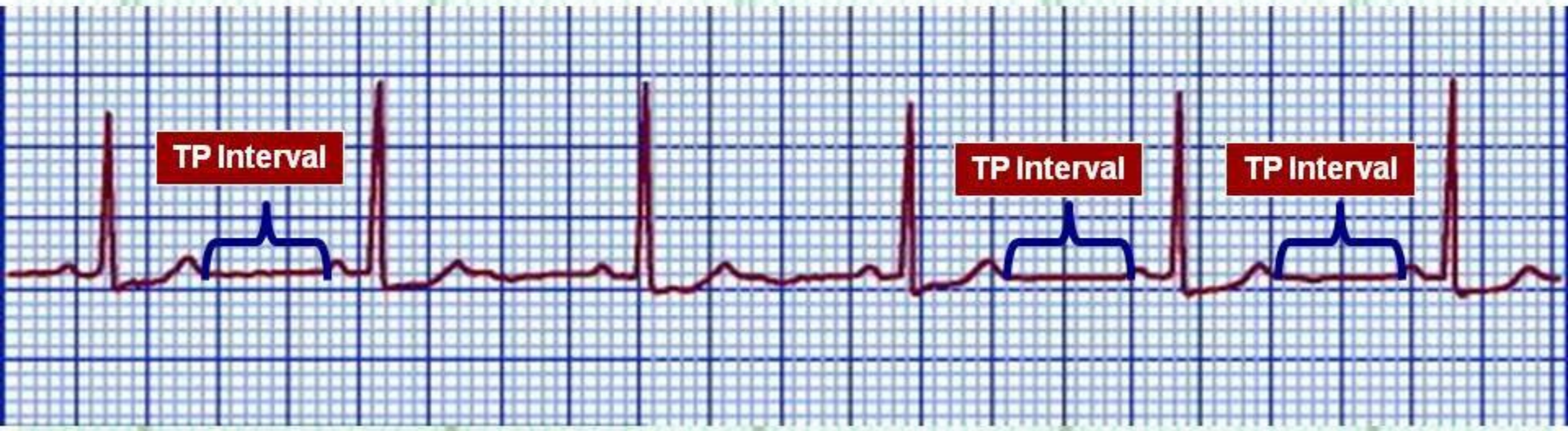
- May have slight left axis due to rotation of heart
- May have high voltage QRS – simulating LVH
- Mild slurring of QRS but duration < 0.09
- J point depression, early repolarization
- T inversions in V2, V3 and V4 – Juvenile T ↓
- Similarly in women also T↓
- Low voltages in obese women and men
- Non cardiac causes of ECG changes may occur

Pseudo Normalization



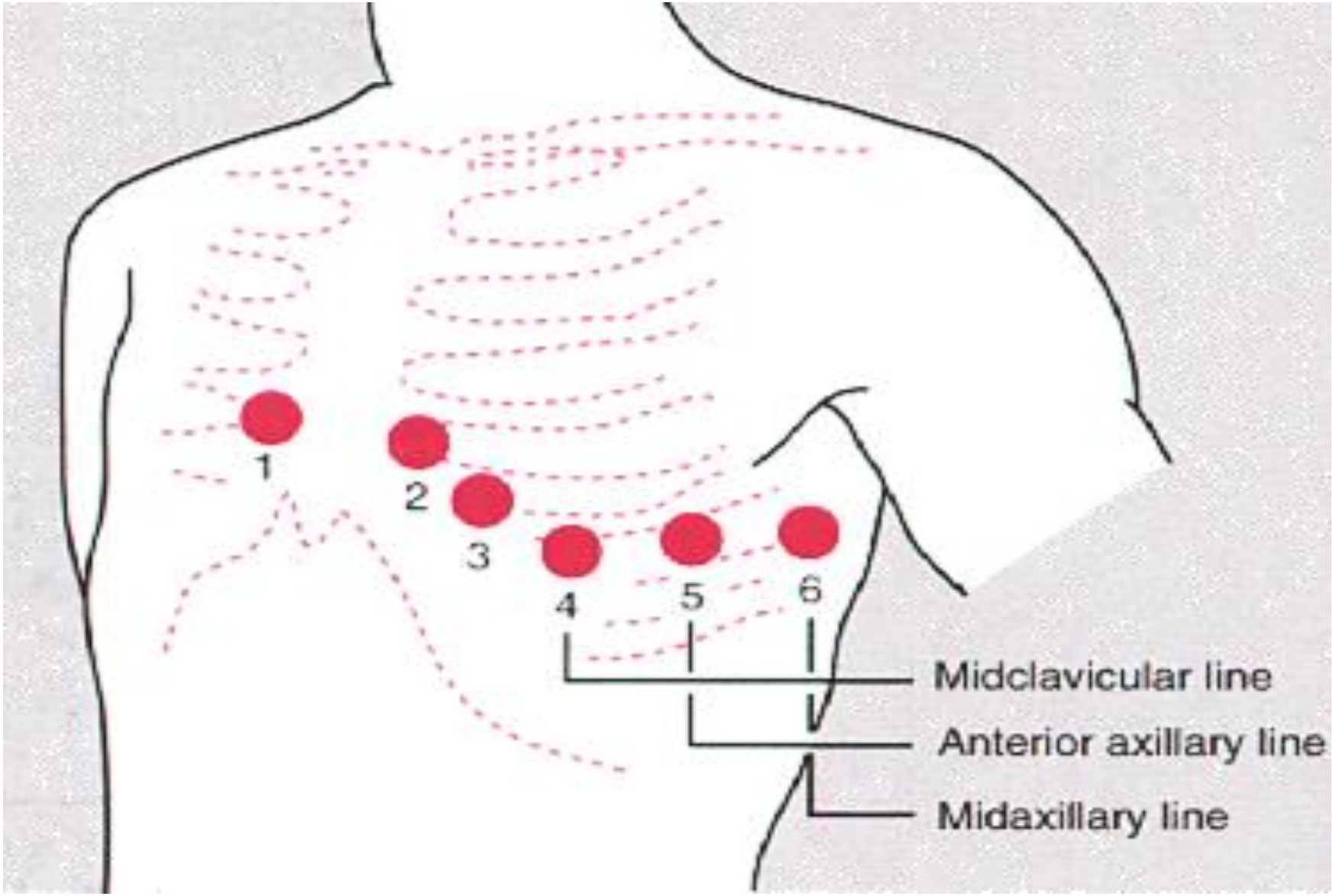

DEVICE ID:ZOLLTEST111
RECORDED:12:01:51 06 JUL 01
PATIENT NAME :GARY_DENTON
PATIENT ID# :007
PATIENT AGE: 45
PATIENT SEX: Male
Vent. rate 58
PR interval 142 ms
QRS Duration: 74 ms
QT/QTc 418/410 ms
P-R-T axes 54 57 90
Sinus bradycardia
Inferior infarct , possibly acute
Marked ST abnormality, possible anterosepta! subendocardial injury
** ** ** ** * Acute MI * ** ** ** **
Abnormal ECG
*** Unconfirmed ***
Patient Information
Computerized Interpretive Statement
Measurements

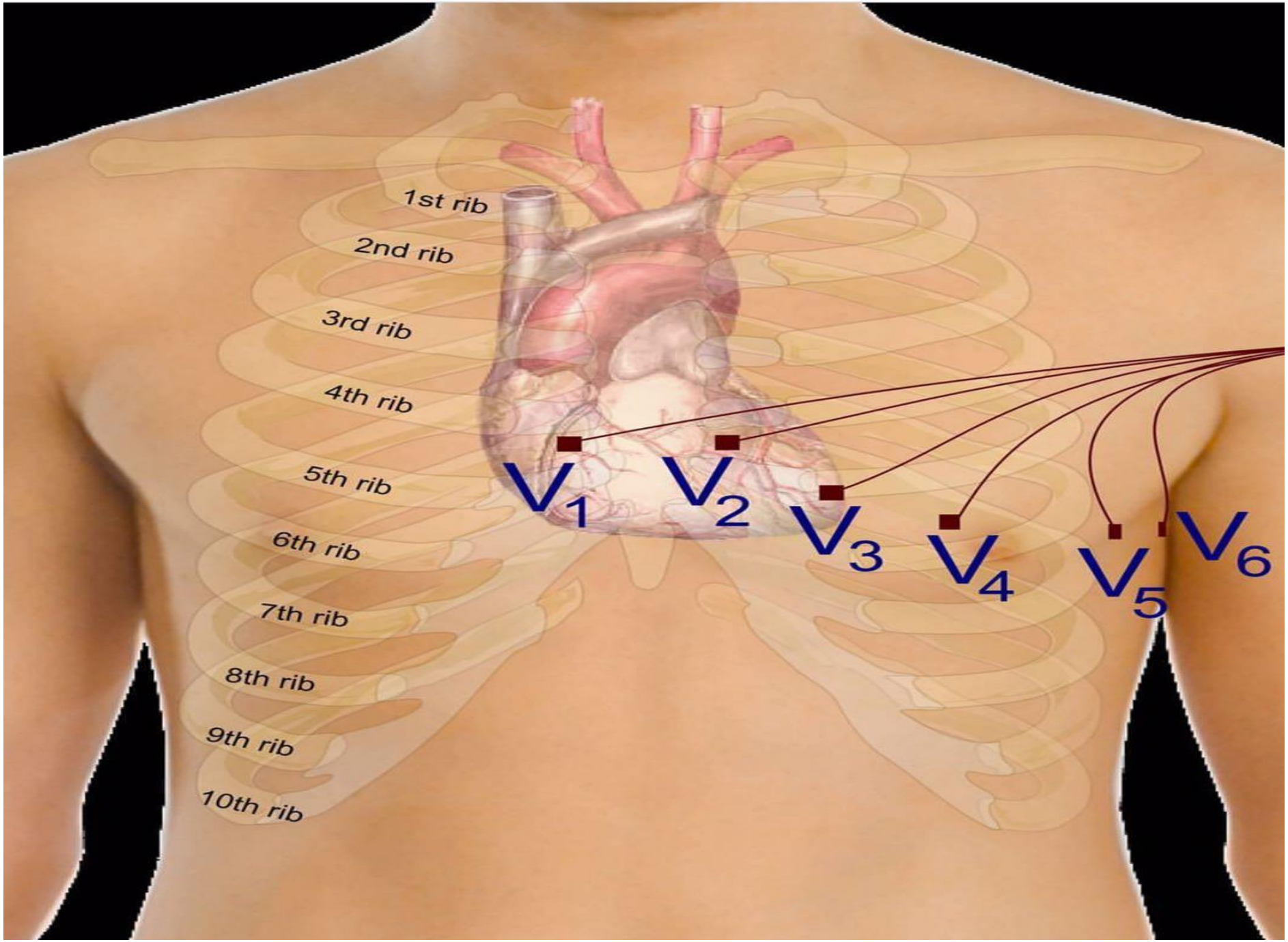
Isoelectric Line (TP Interval)



Lead Placement

- V1 - Right parasternally, 4th ICS
- V2 - Left parasternally, 4th ICS
- V3 - Between V2 and V4
- V4 - 5th ICS, mid clavicular line
- V5 - Between V4 and V6
- V6 - Left mid-axillary line, (level with V4)


1
2
3
4
5
6
Midclavicular line
Anterior axillary line
Midaxillary line


1st rib
2nd rib
3rd rib
4th rib
5th rib
6th rib
7th rib
8th rib
9th rib
10th rib
V1
V2
V3
V4
V5
V6

Posterior Lead Placement

V7 - Post axillary line, level with V4

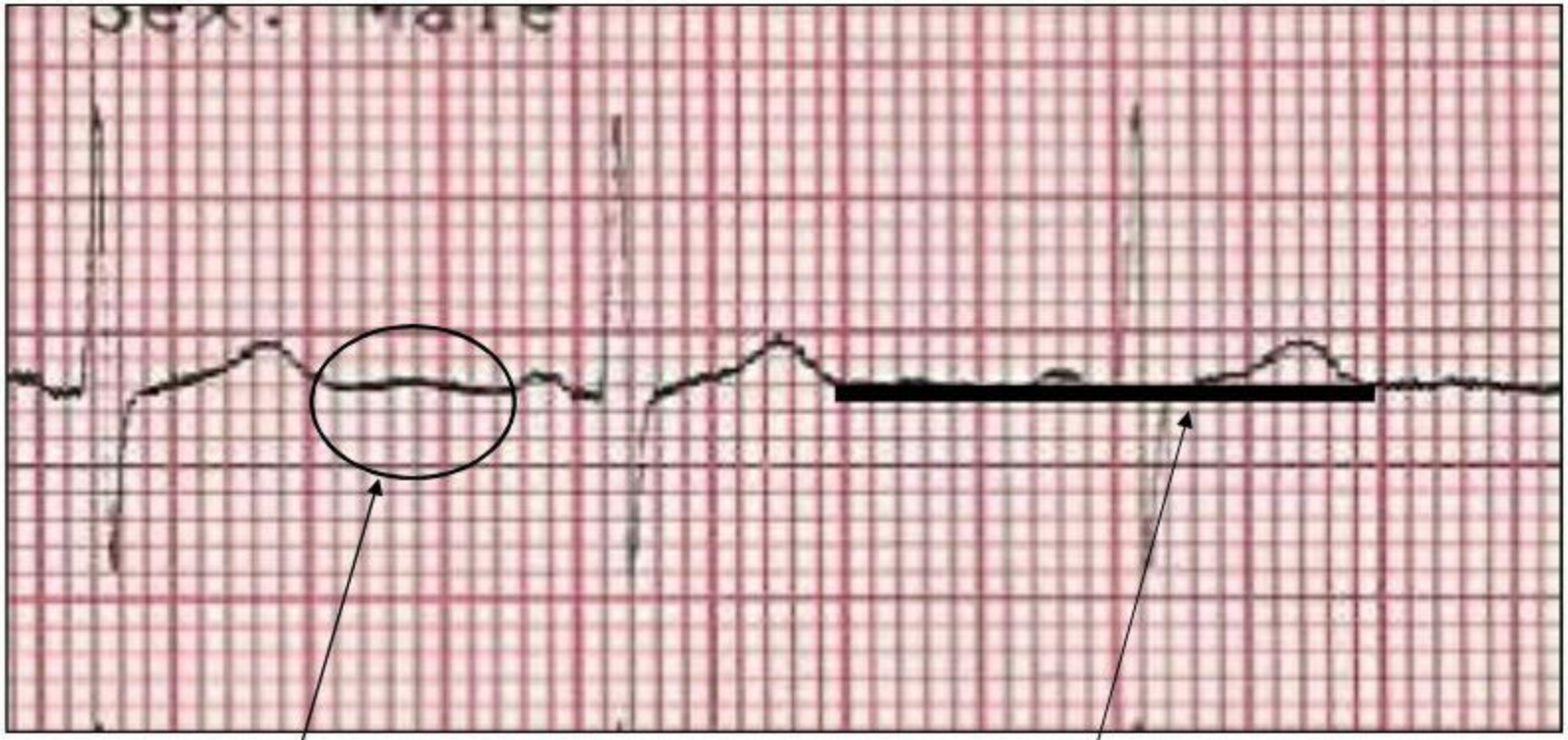
V8 - Mid scapular line, level with V4

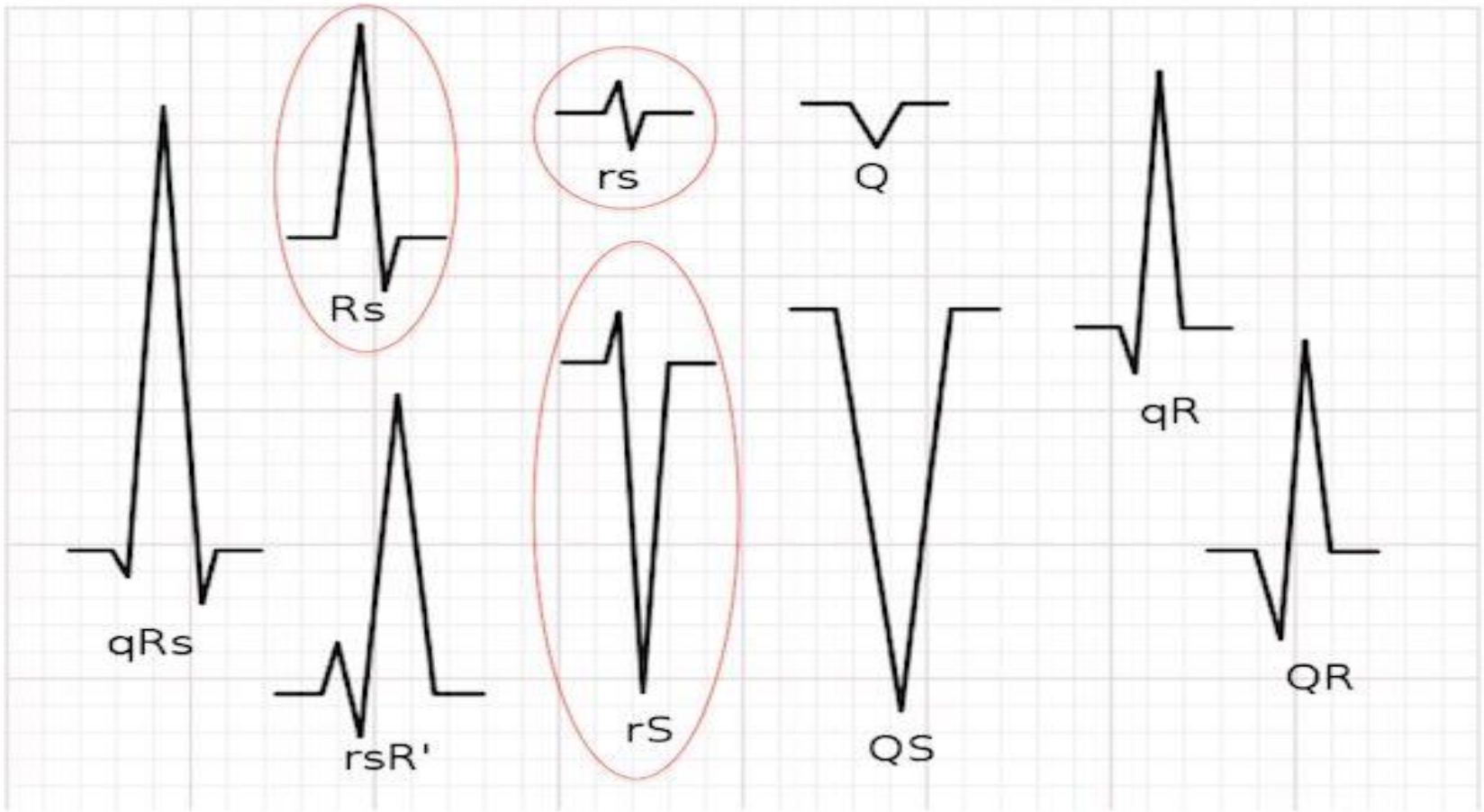
V9 - Left paravertebral area, level with V4

Specialty Leads

- Posterior Wall - V7-V9
- Right Ventricle - V3R-V6R

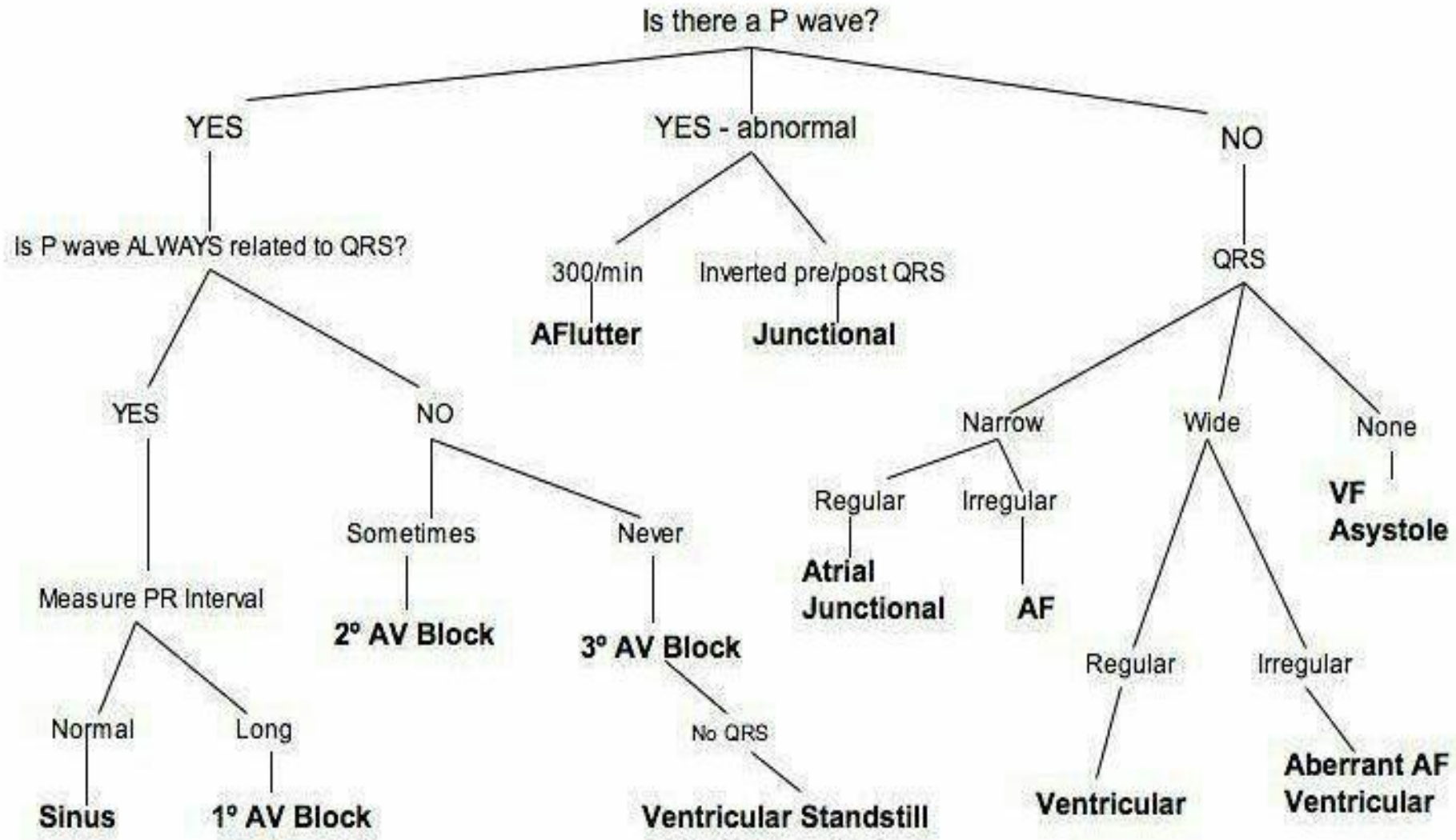
T-P Segment




rs
Q
Rs
qRs
rsR'
rS
QS
qR
QR

ECG INTERPRETATION FLOWCHART

Elizabeth Gherardin Box Hill Hospital 1999



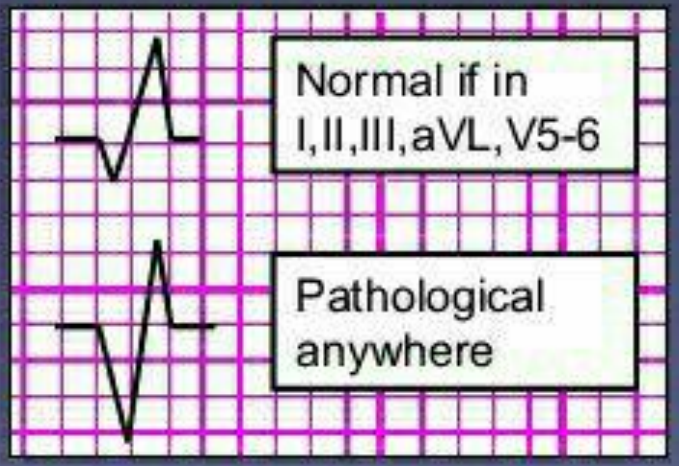
The Q wave

Are there any pathological Q waves?

- A Q wave can be pathological if it is:
  - Deeper than 2 small squares (0.2mV)

  and/or
  - Wider than 1 small square (0.04s)

  and/or
  - In a lead other than III or one of the leads that look at the heart from the left (I, II, aVL, V5 and V6) where small Qs (i.e. not meeting the criteria above) can be normal



Normal Q Waves

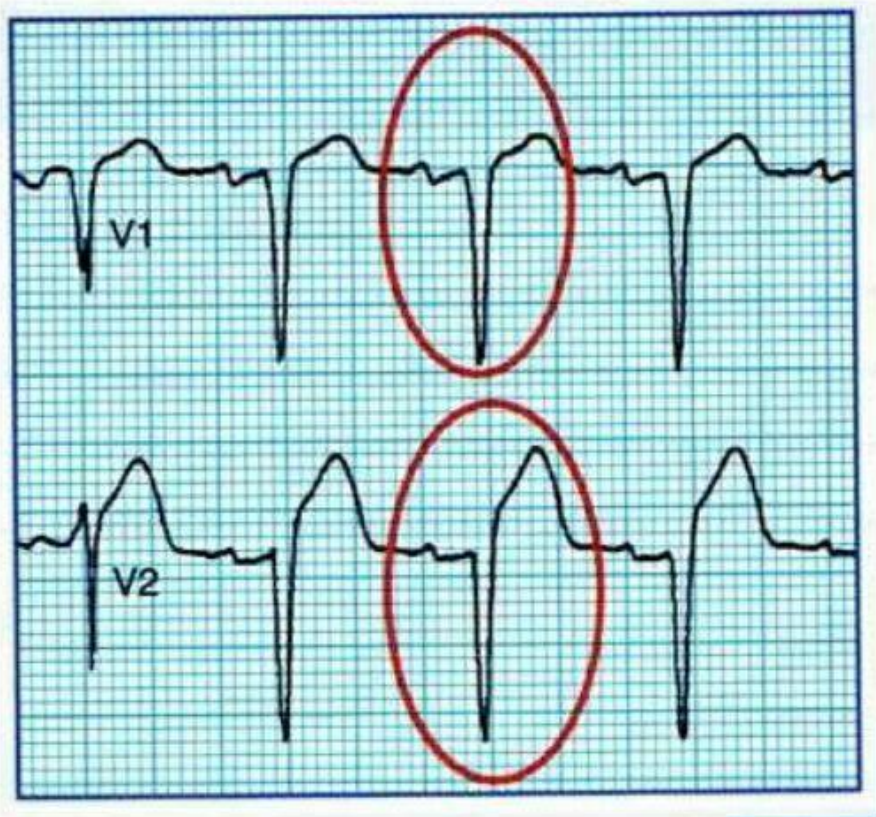
- The normal Q wave in lead I is due to septal depolarization
- It is small in amplitude – less than 25% of the succeeding R wave, or less than 3 mm
- Its duration is < 0.04 sec or one small box
- It is seen in L1 and some times in V5, V6

Pathological Q wave

- The pathological Q wave of infarction in the respective leads is due to dead muscle
- It is deep in amplitude – more than 25% of the succeeding R wave, or more than 4 mm
- Its duration is > 0.04 sec or > 1 small box
- It is seen in Leads facing the infarcted muscle mass

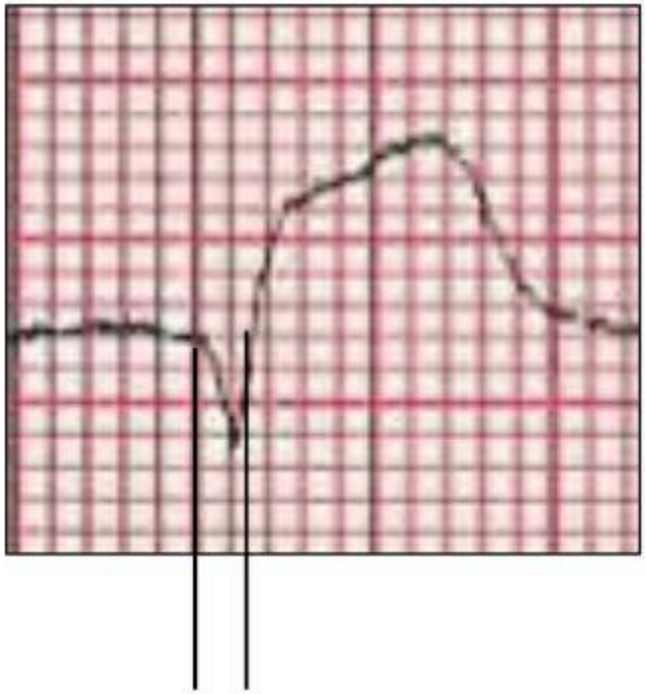
Pathological Q Wave

- Signifies infarction, or death of the tissue
- Indicators of an Infarct
  - Pathological Q wave
  - >40 ms, or 0.04 sec wide, or 1/3 of R wave height
  - Indicates on-going or permanent damage



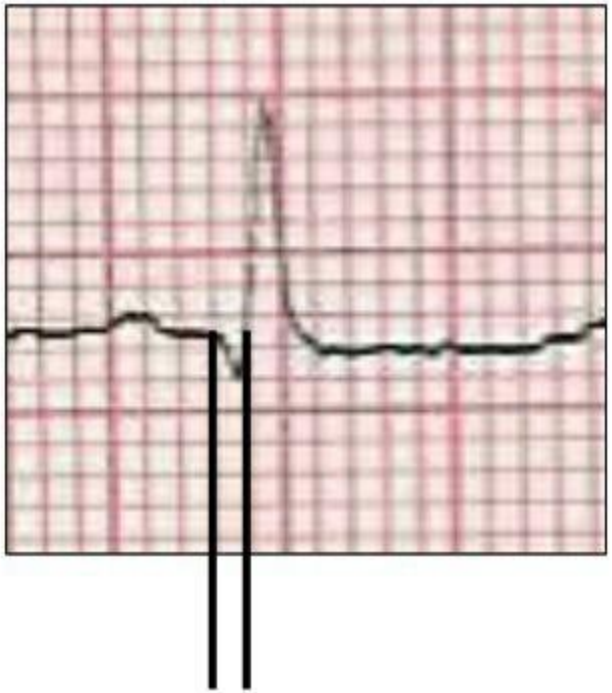
Q Waves

Pathological



> 1mm

Physiological



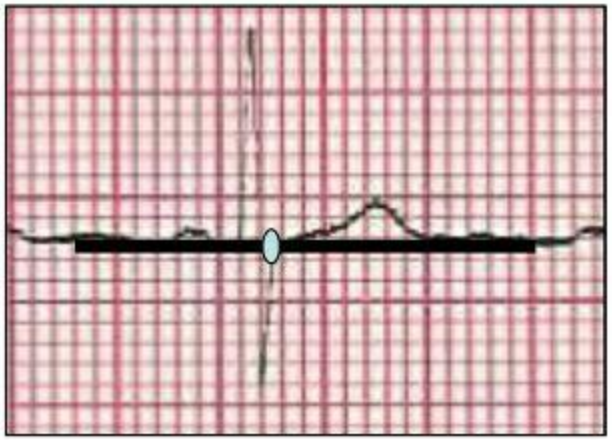
< 1mm

ST Segment

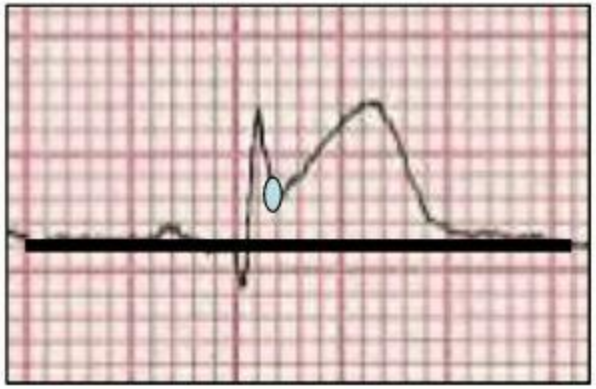
- The ST segment is normally level with the T-P segment rather than the PR segment

- Examine every lead for ST segment elevation of 1 mm or more.

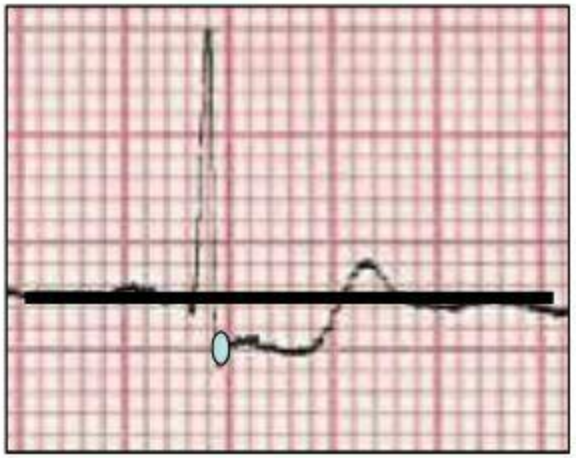
ST Segment



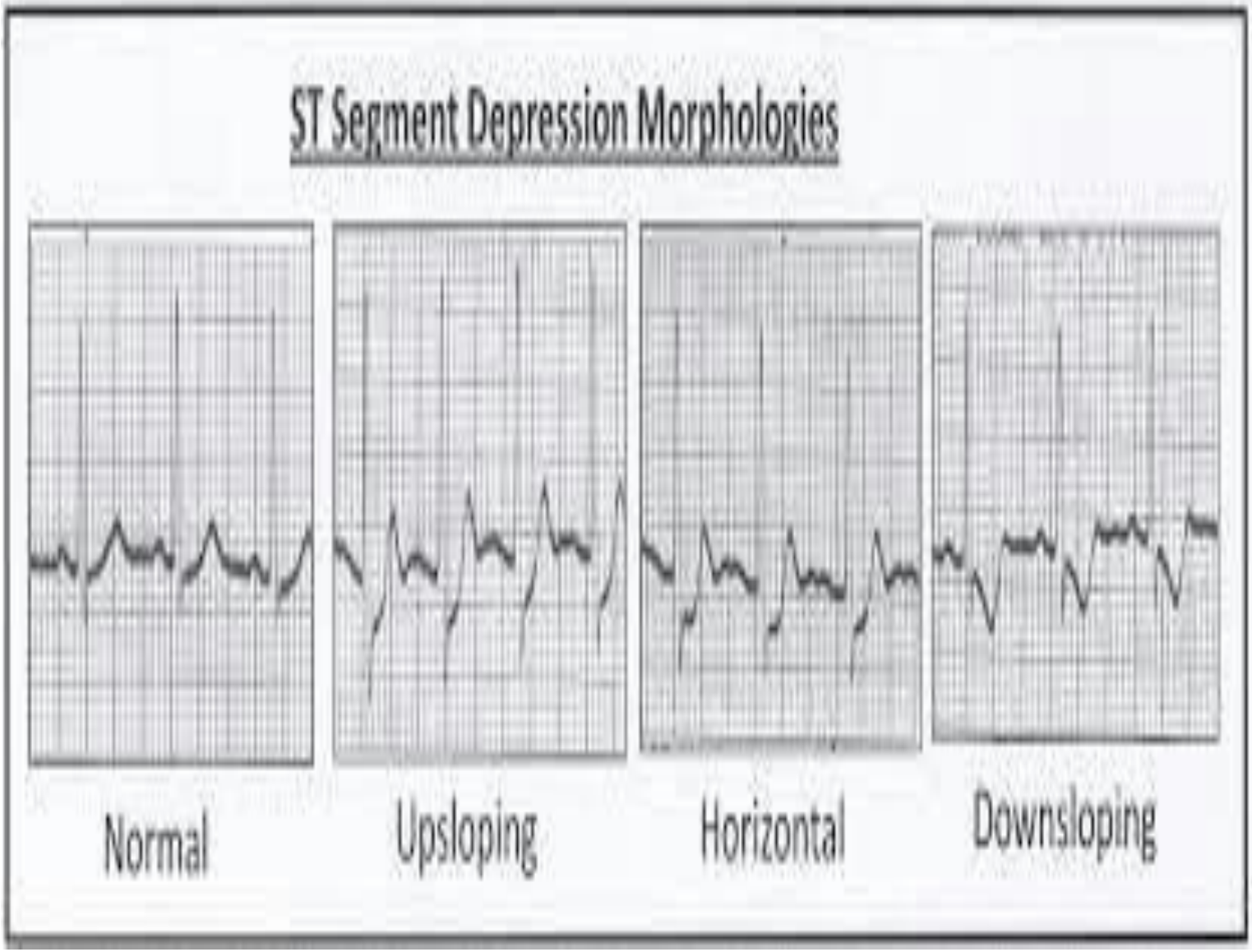
Isoelectric

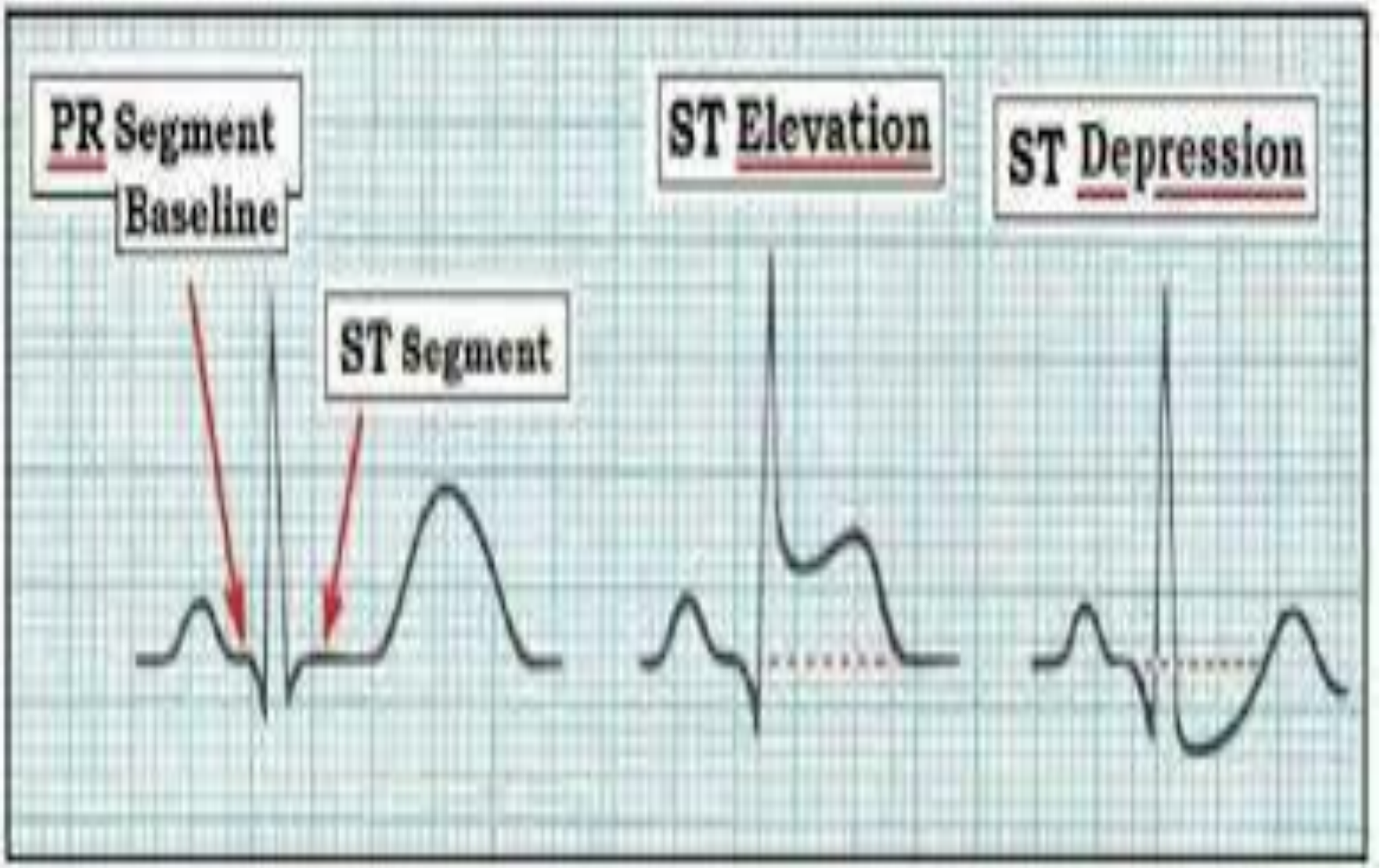


Elevated



Depressed

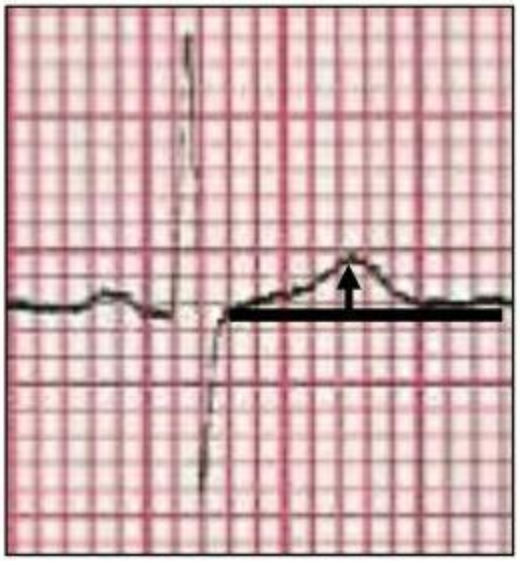

ST Segment Depression Morphologies
Normal
Upsloping
Horizontal
Downsloping


PR Segment
Baseline
ST Segment
ST Elevation
ST Depression

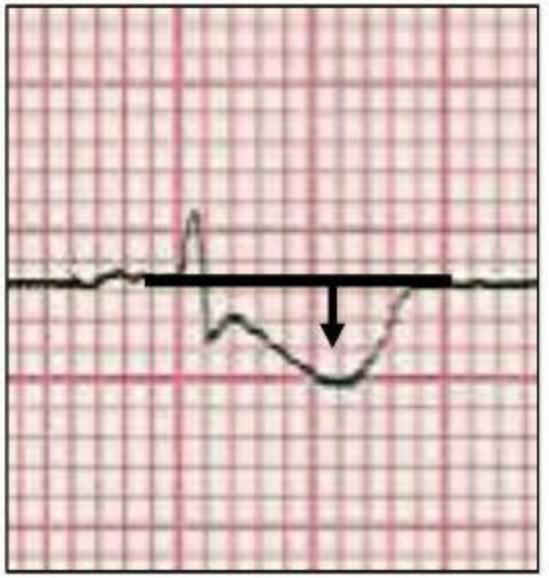
T Wave

- Large wave form following QRS Complex
- Represents Ventricular Repolarization
- Should be upright in most leads (III and V1)
- Look for consistent morphology

T Waves

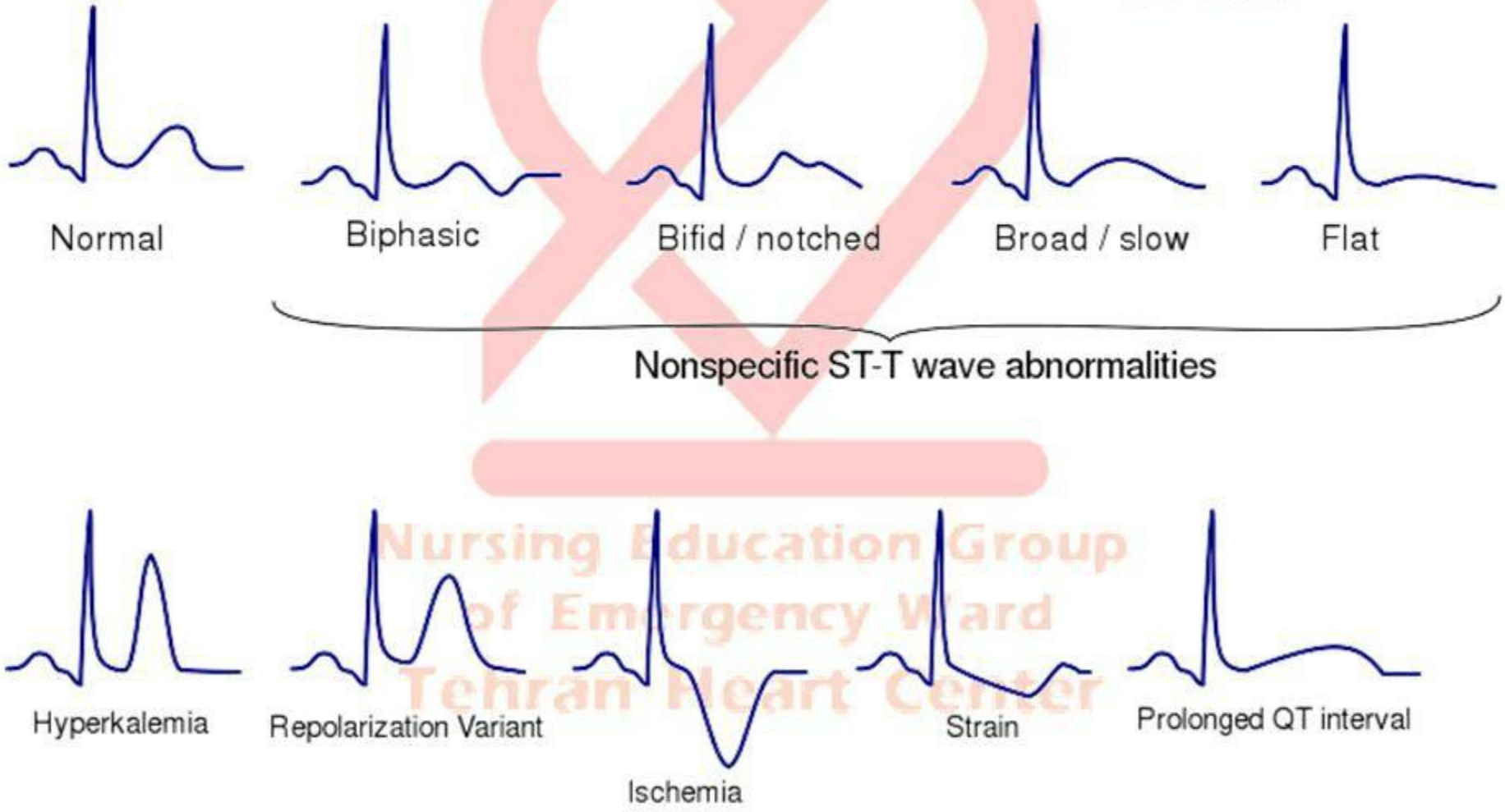


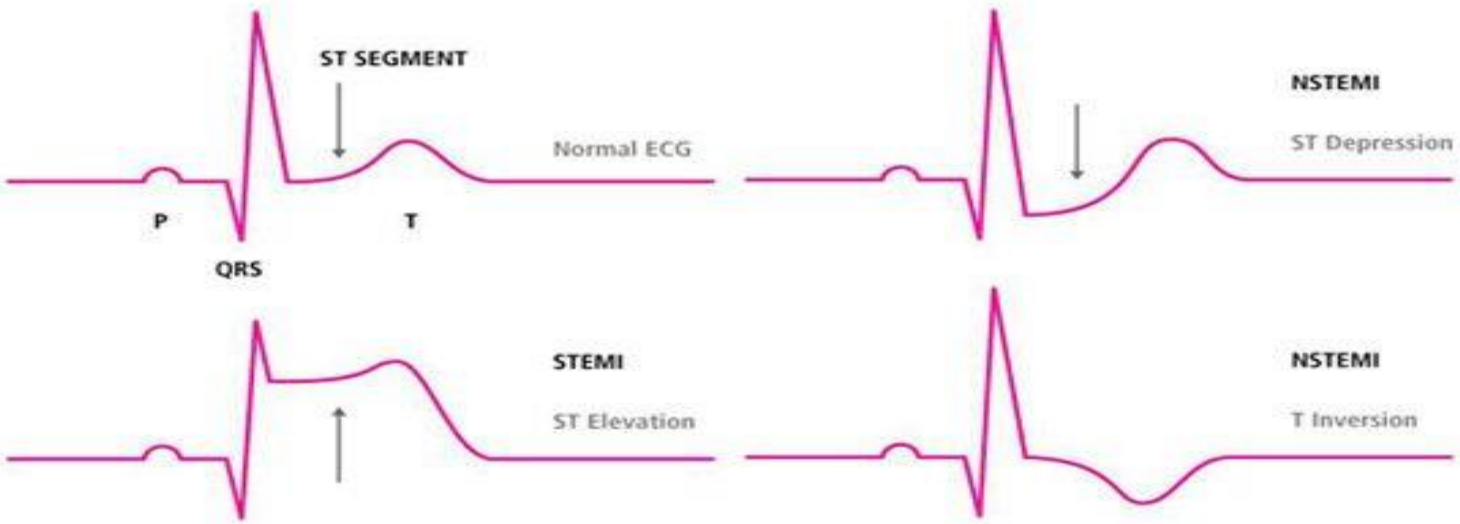
Upright

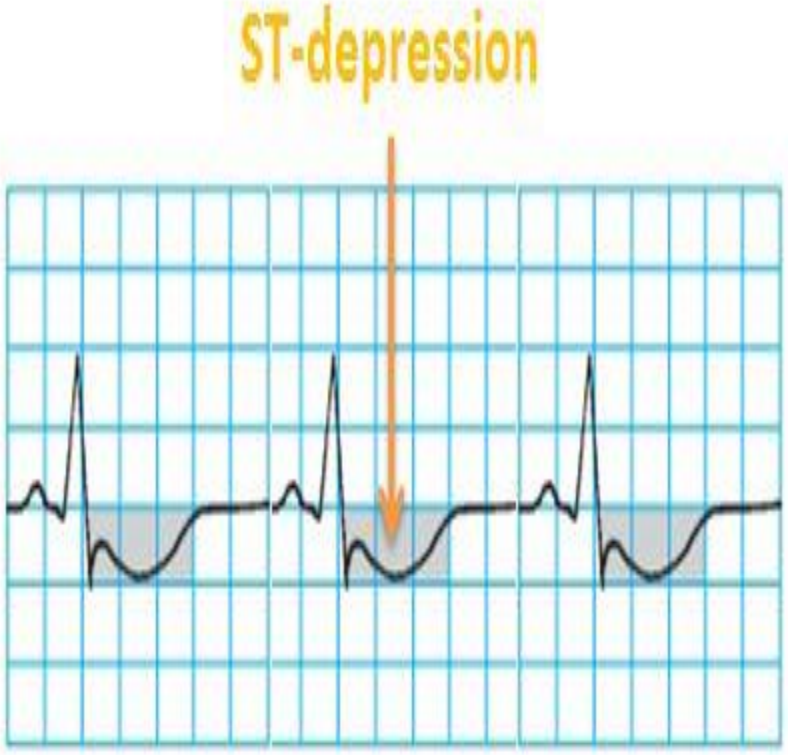


Inverted

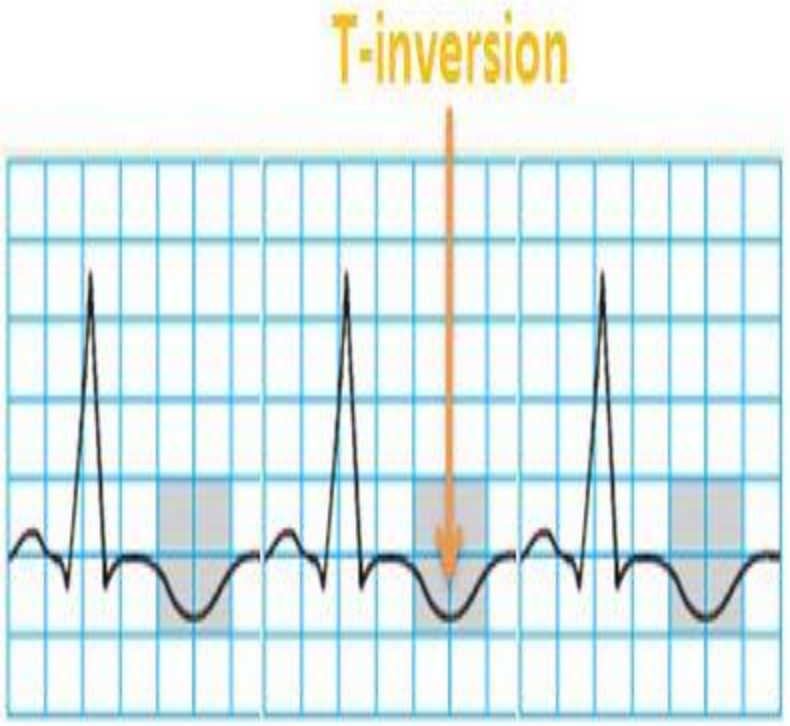
T wave morphology



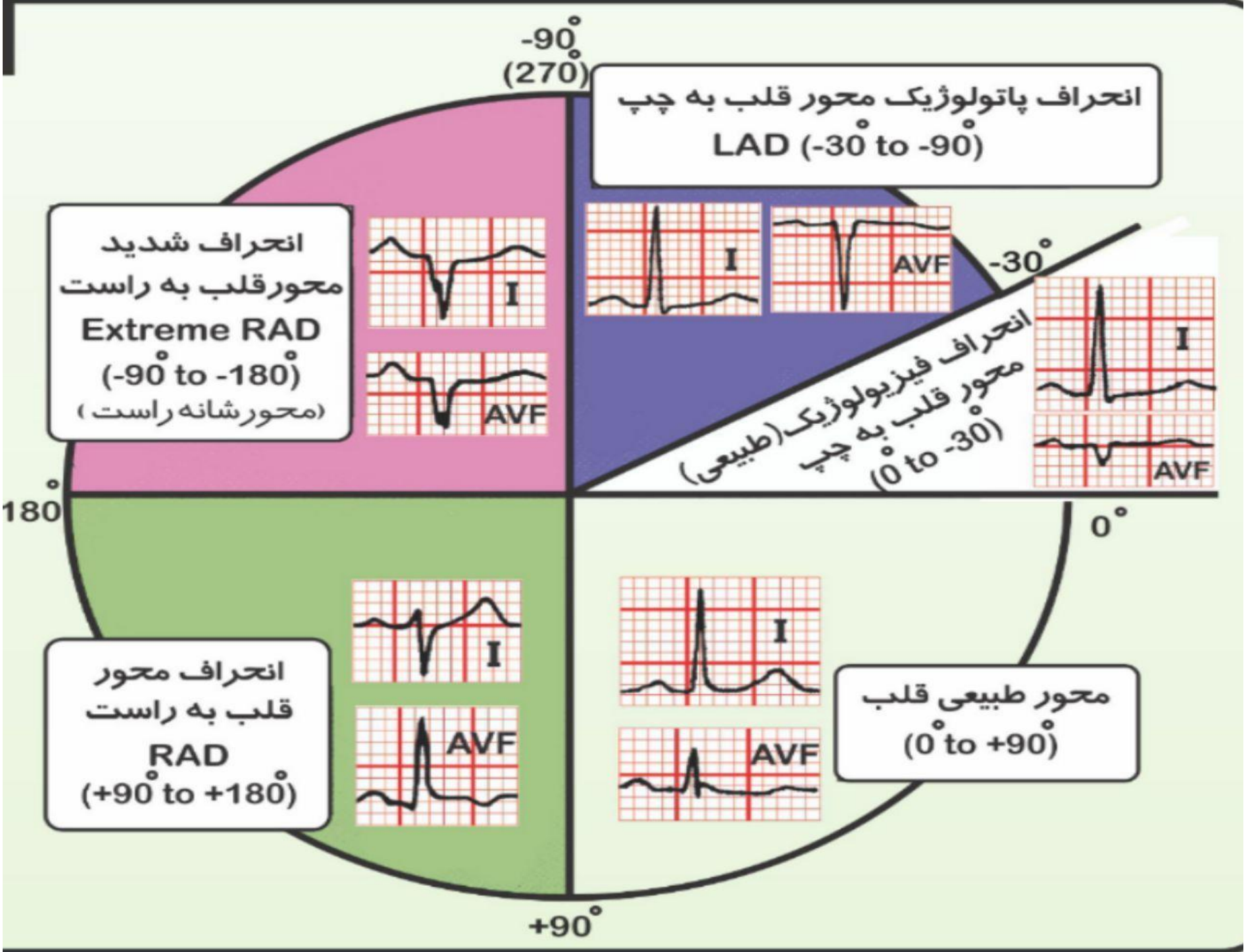


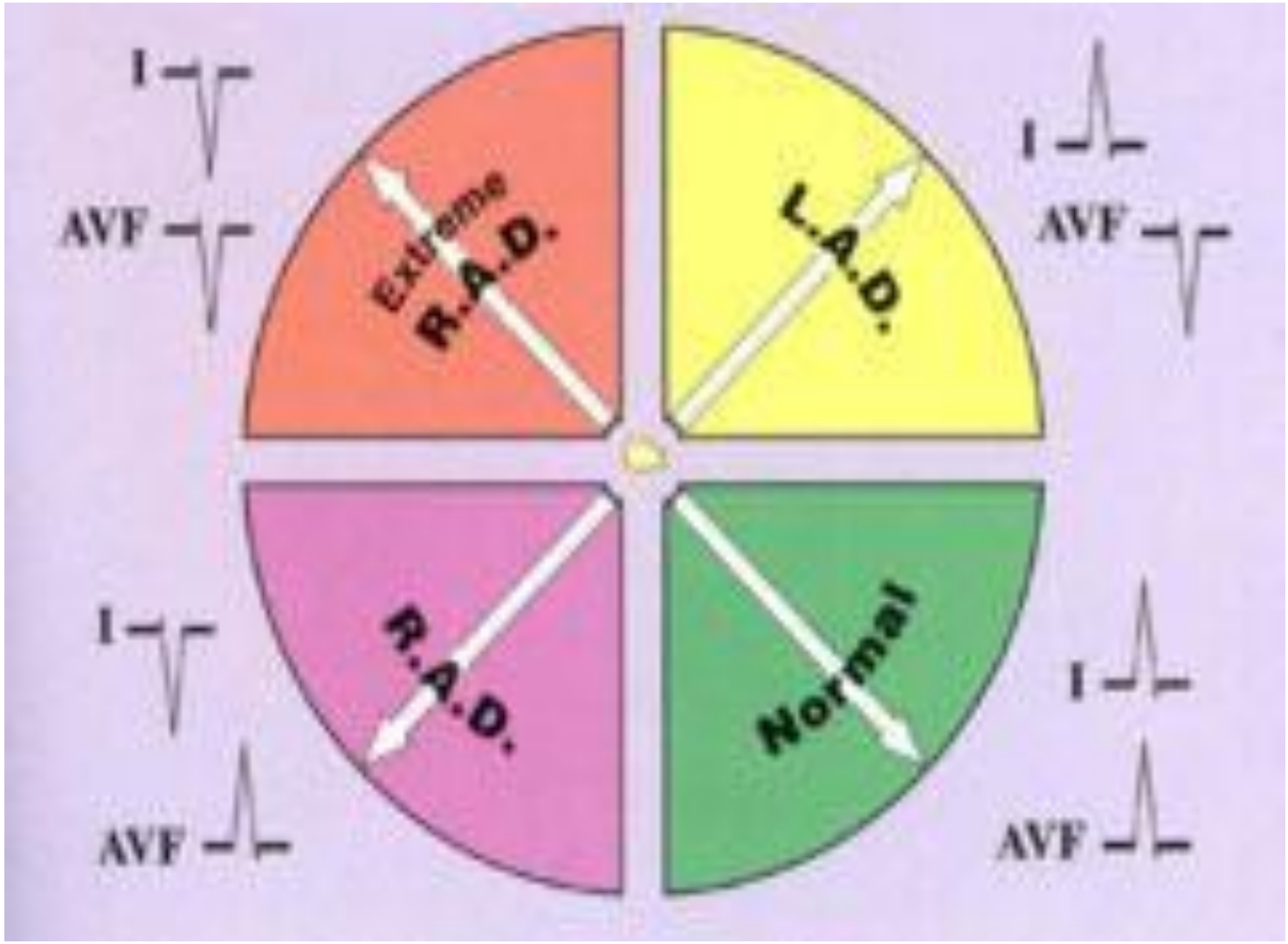


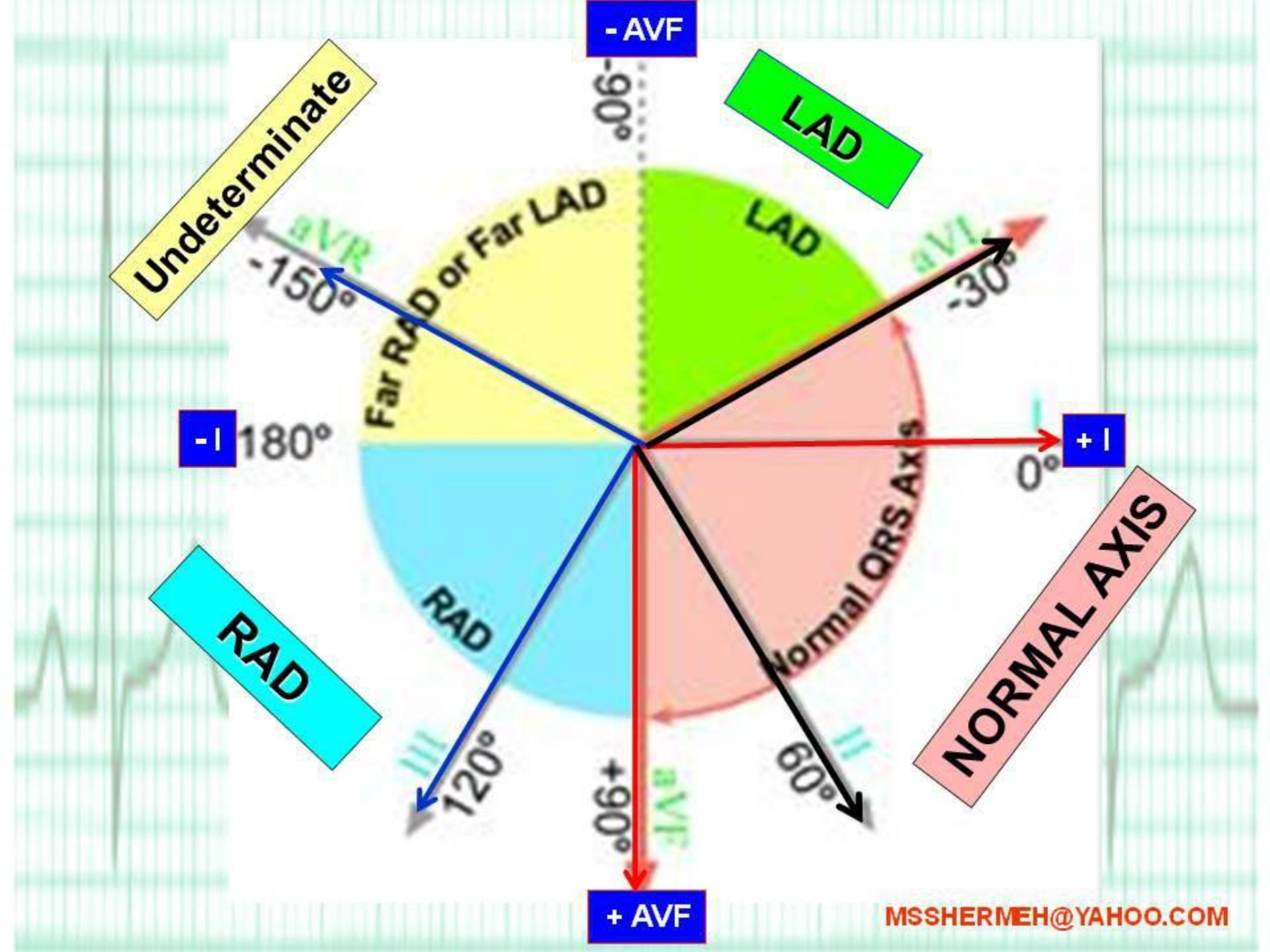
ST-segment depression in nstemi

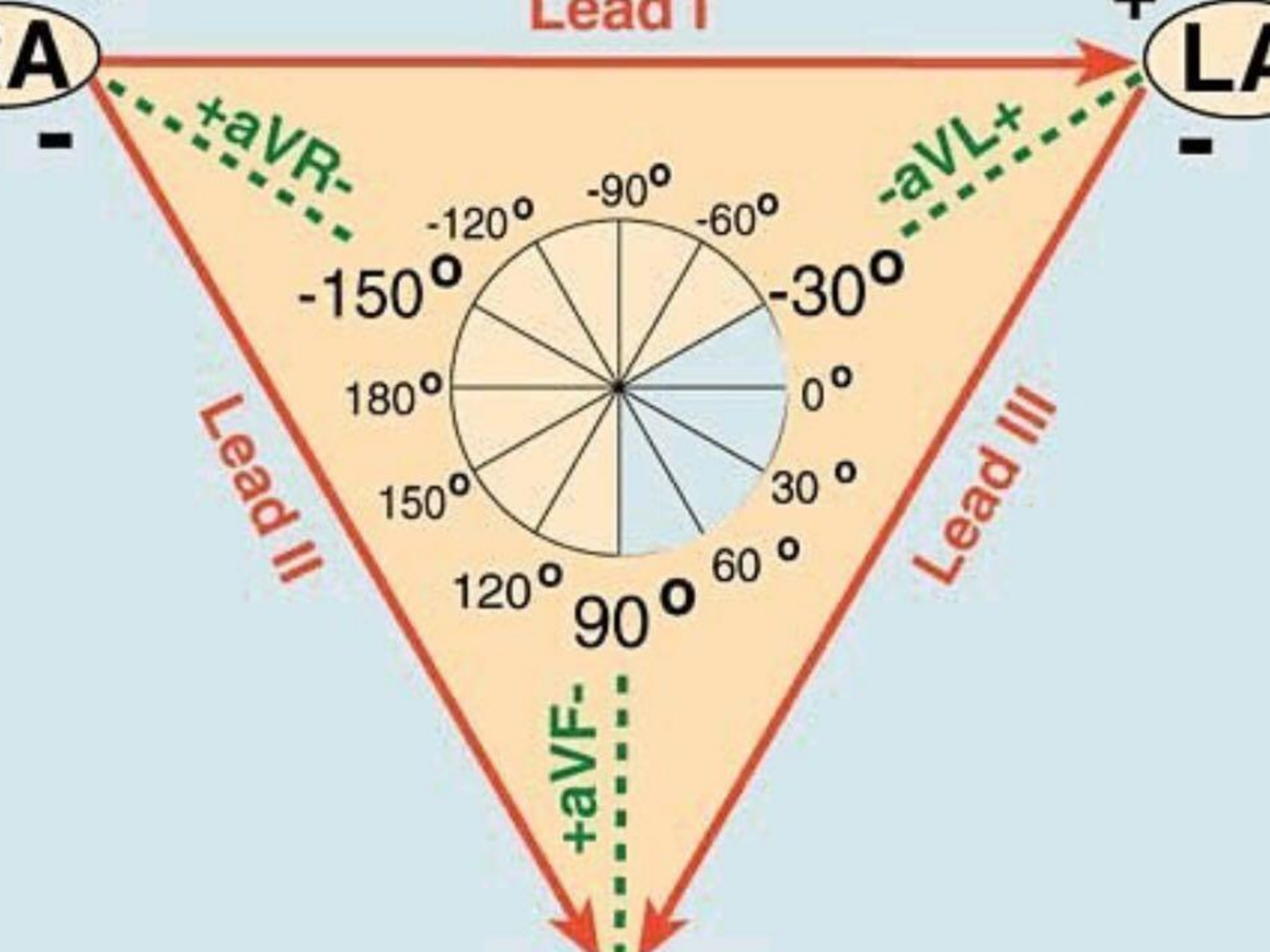


T-wave inversion in nstemi


-90°
(270°)
انحراف پاتولوژیک محور قلب به چپ
LAD (-30° to -90°)
انحراف شدید
محورقلب به راست
Extreme RAD
(-90° to -180°)
(محورشانه راست)
I
AVF
-30°
انحراف فیزیولوژیک(طبیعی)
محور قلب به چپ
(0° to -30°)
180°
0°
انحراف محور
قلب به راست
RAD
(+90° to +180°)
محور طبیعی قلب
(0° to +90°)
+90°


I
AVF
Extreme
R.A.D.
I
AVF
L.A.D.
I
AVF
R.A.D.
I
AVF
Normal

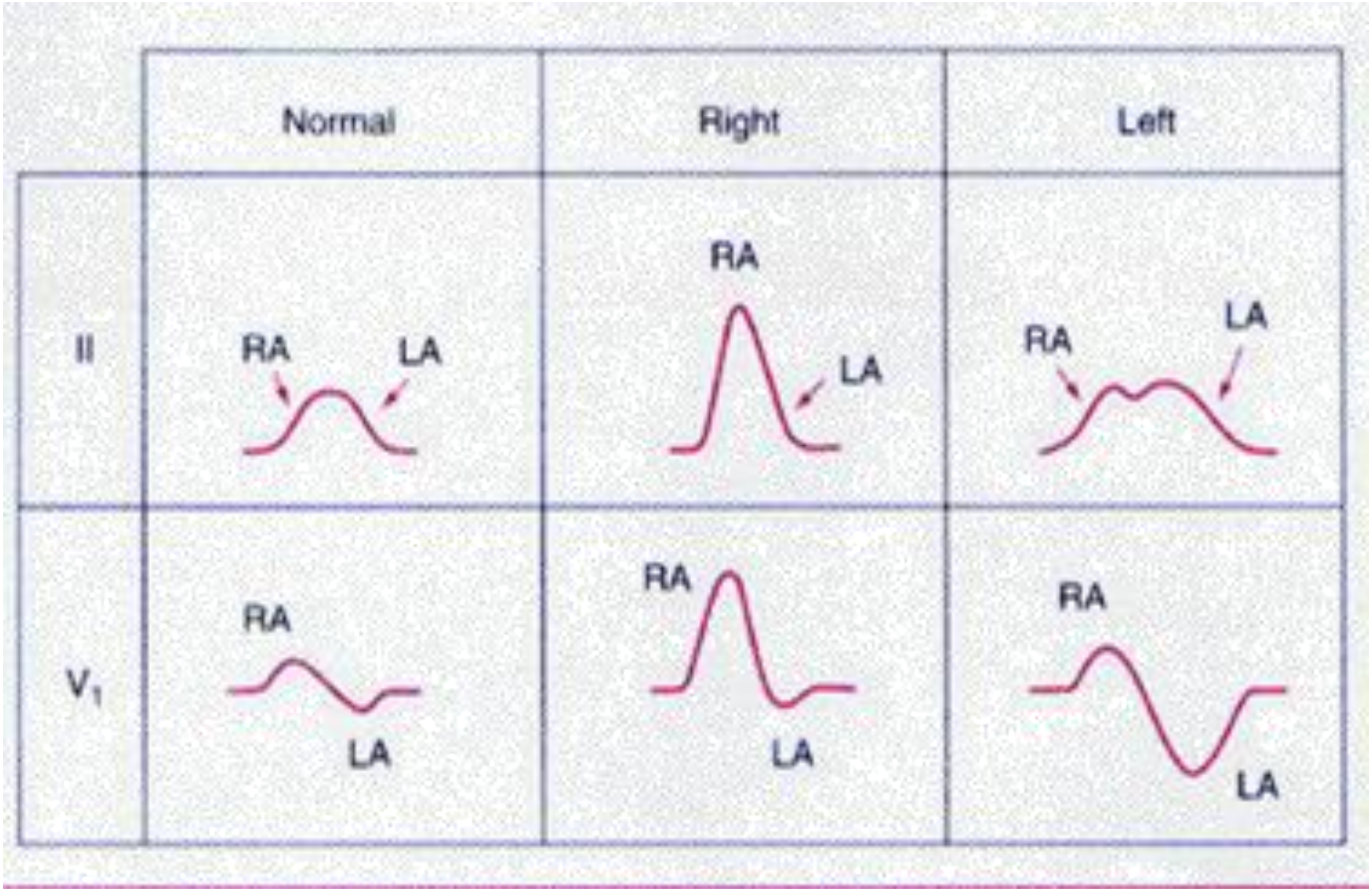

- AVF
-90°
LAD
Undeterminate
aVR
-150°
Far RAD or Far LAD
LAD
aVL
-30°
-I
180°
+ I
0°
Normal QRS Axis
RAD
RAD
III
120°
60°
II
+90°
aVF
NORMAL AXIS
+ AVF
MSSHERMEH@YAHOO.COM


Lead I
RA
LA
-
+
-
+aVR-
-aVL+
-120°
-90°
-60°
-150°
-30°
180°
0°
150°
30°
120°
60°
90°
Lead II
Lead III
+aVF-

Axis Determination

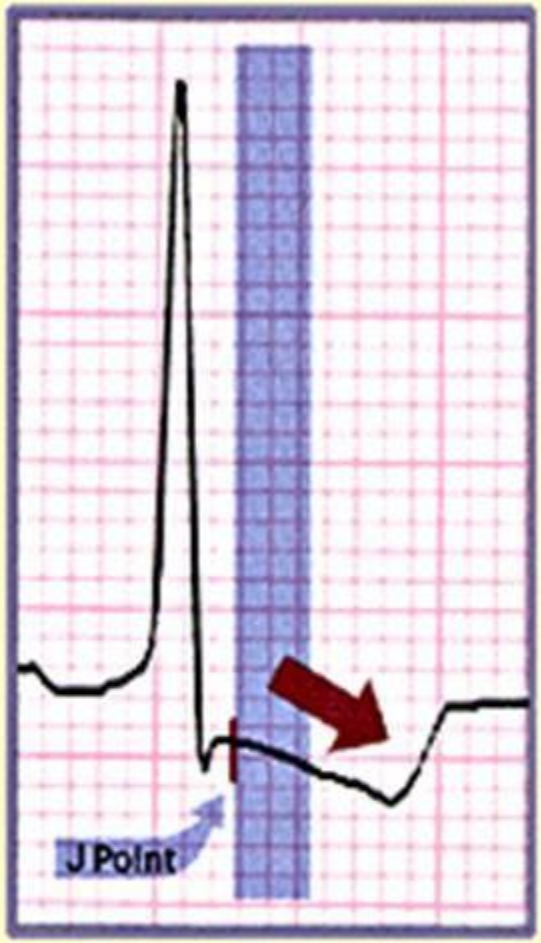
Axis	L_I	L_{III} or aVF	TIP
Normal	Positive	Positive	Both Up
Right	Negative	Positive	Meet
Left	Positive	Negative	Leave
Indeterminate	Negative	Positive	Meet

Atrial Waves

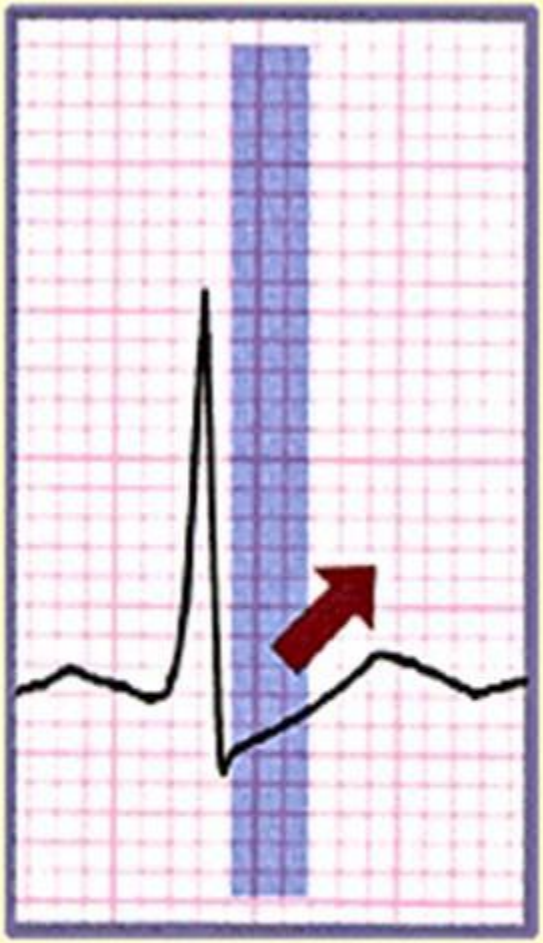


Atrial Ectopics

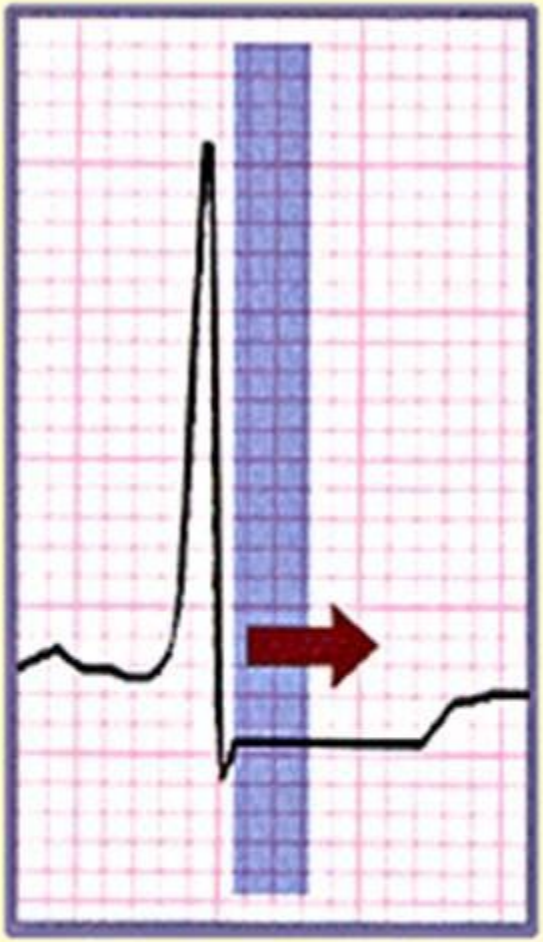
- Note the premature (ectopic) beats marked as
- APC (Atrial Premature Contractions)
- These occurred before the next expected QRS complex (premature)
- Each APC has a P wave preceding the QRS of that beat – So impulse has originated in the atria
- The QRS duration is normal < 0.08, not wide



Downsloping ST



Upsloping ST

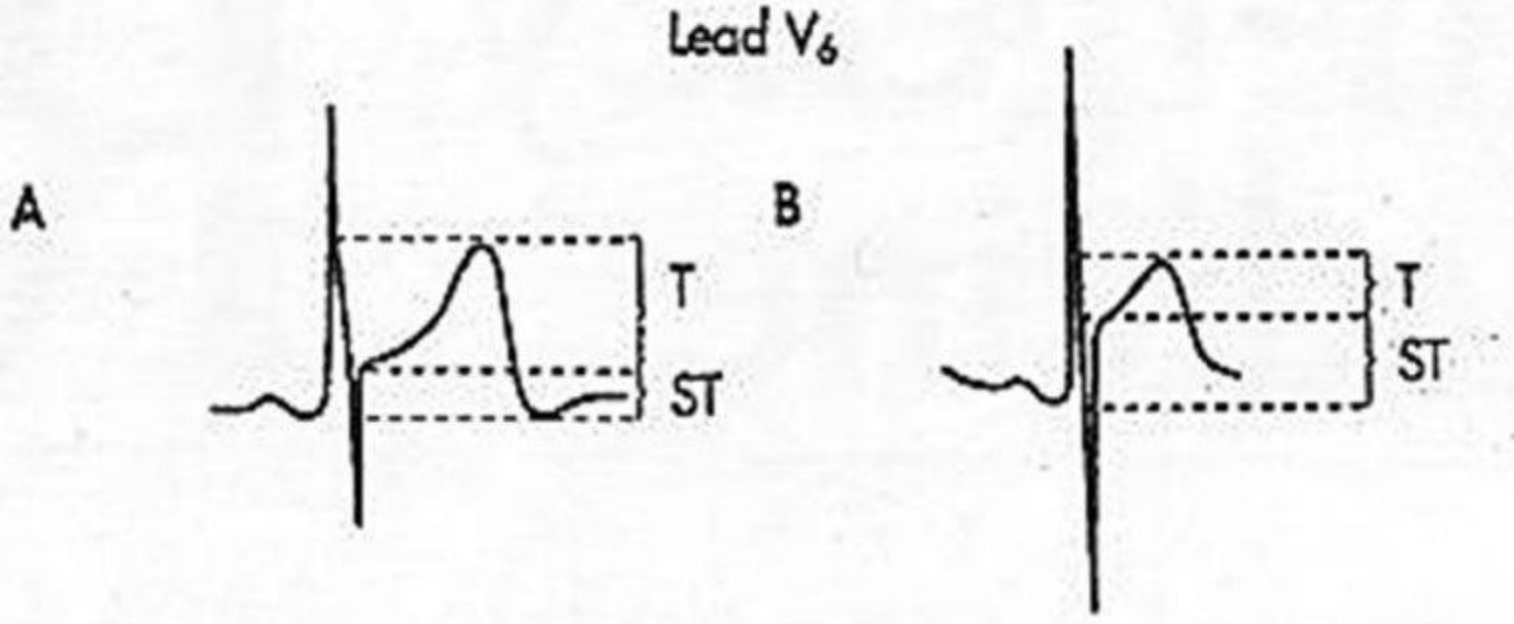


Horizontal ST

The J point occurs at the end of the QRS comples.
The ST segment begins at the J point and extends to a user defined interval

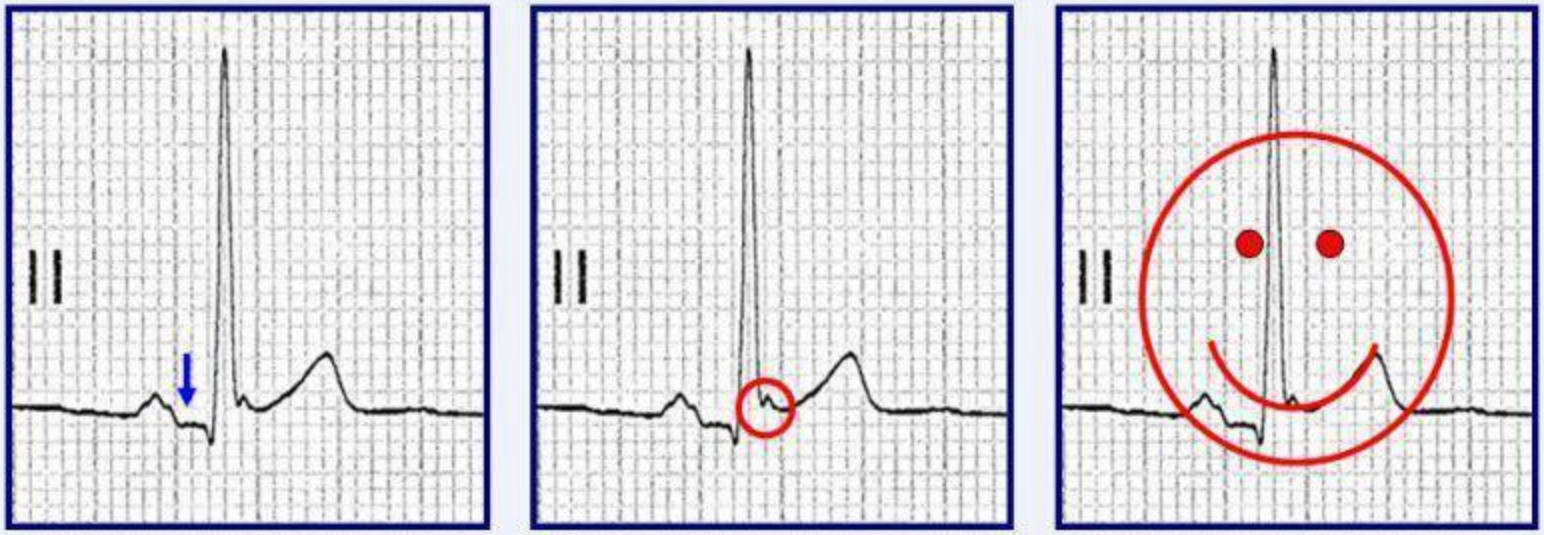
ST Segment Depression

BER vs Pericarditis



Benign early repolariztion with ST / T ratio less than 0.25.

Acute pericarditis with ST / T ratio greater than 0.25.



A. B. C.

ems12lead.com

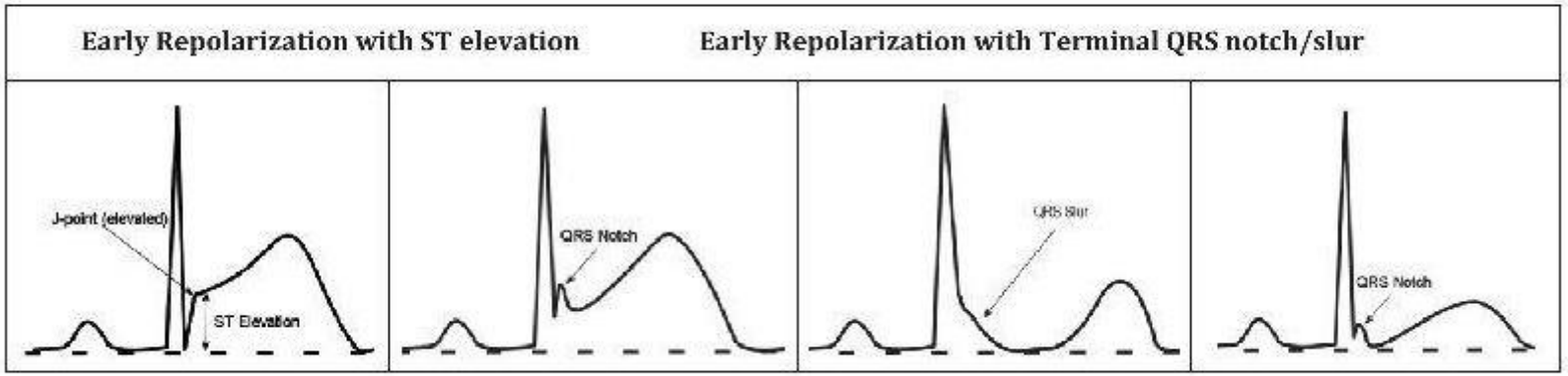
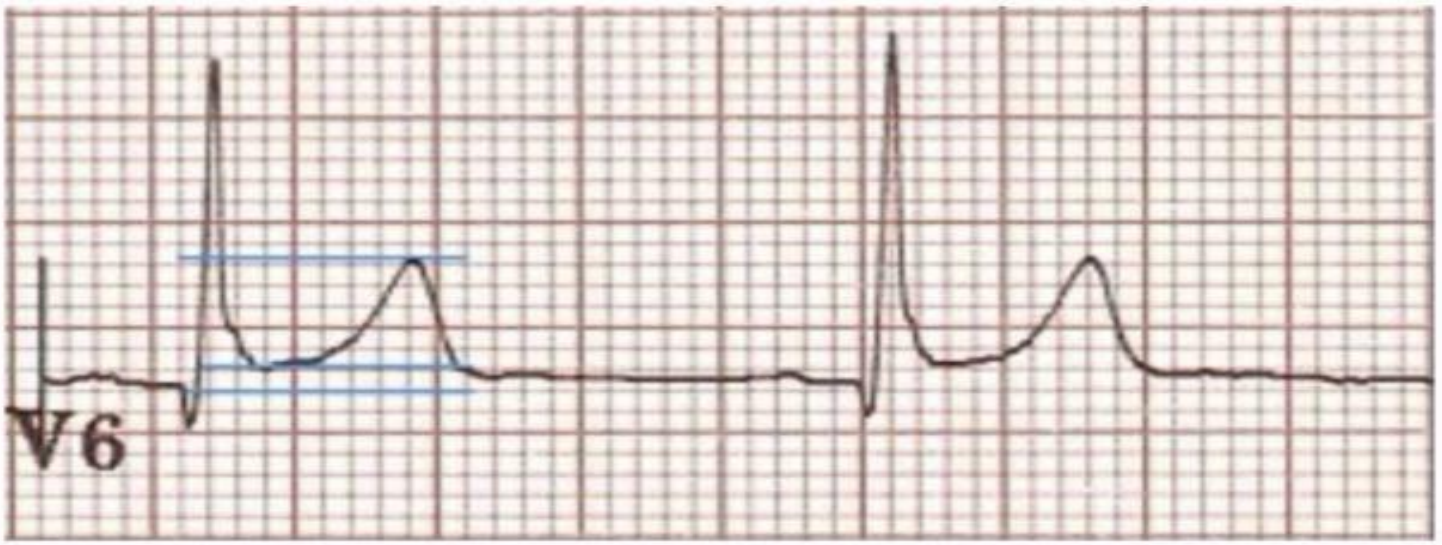


Figure 1. Examples of early repolarization pattern.

Example 1: Benign Early Repolarisation



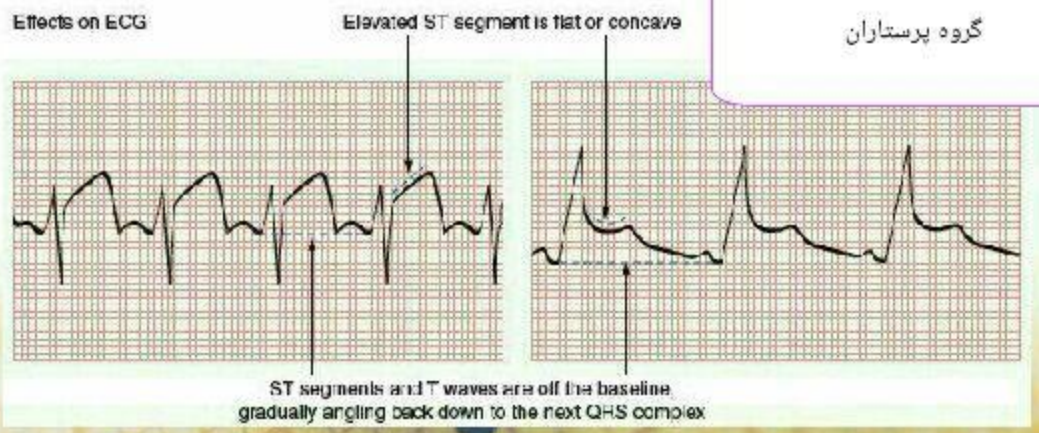
- ST segment height = 1 mm
- T wave height = 6 mm
- ST / T wave ratio = 0.16
- The ST / T wave ratio < 0.25 is consistent with BER.

Indicative Lead Groups

- Inferior Wall - II, III, and aVF
- Septal Wall - V1, V2
- Anterior Wall - V3, V4
- Lateral Wall - V5, V6, I, and aVL

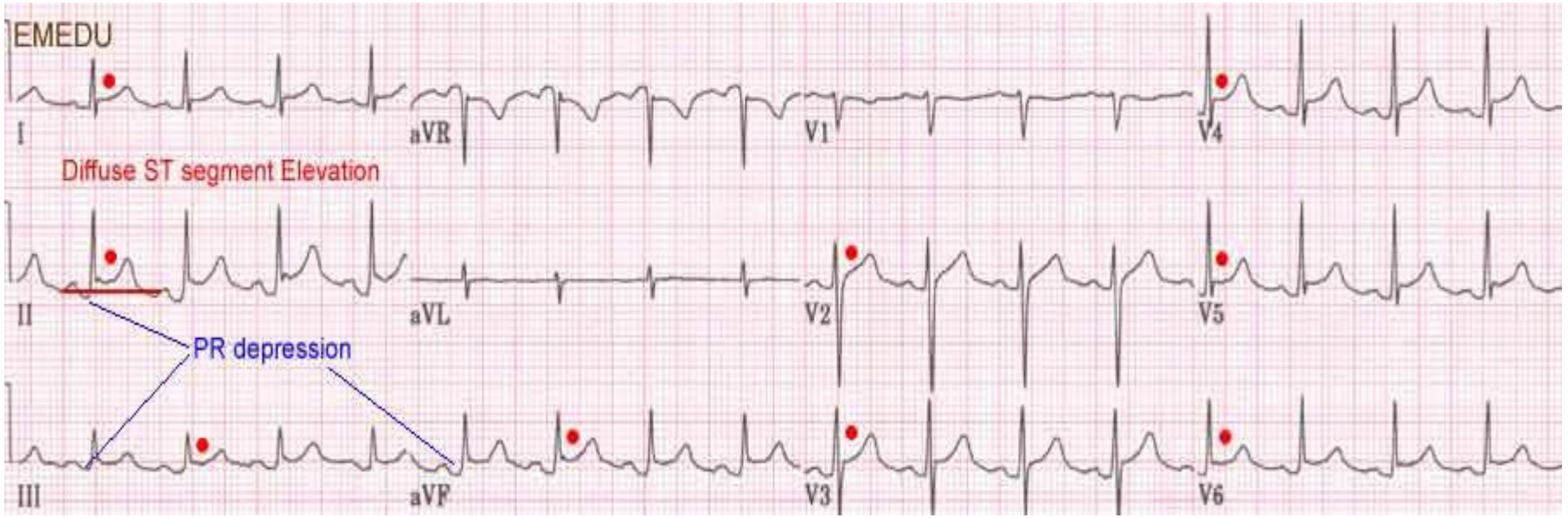
ECG Changes in Pericarditis

- T wave initially upright and elevated but then during recovery phase it inverts
- ST segment elevated and usually flat or concave

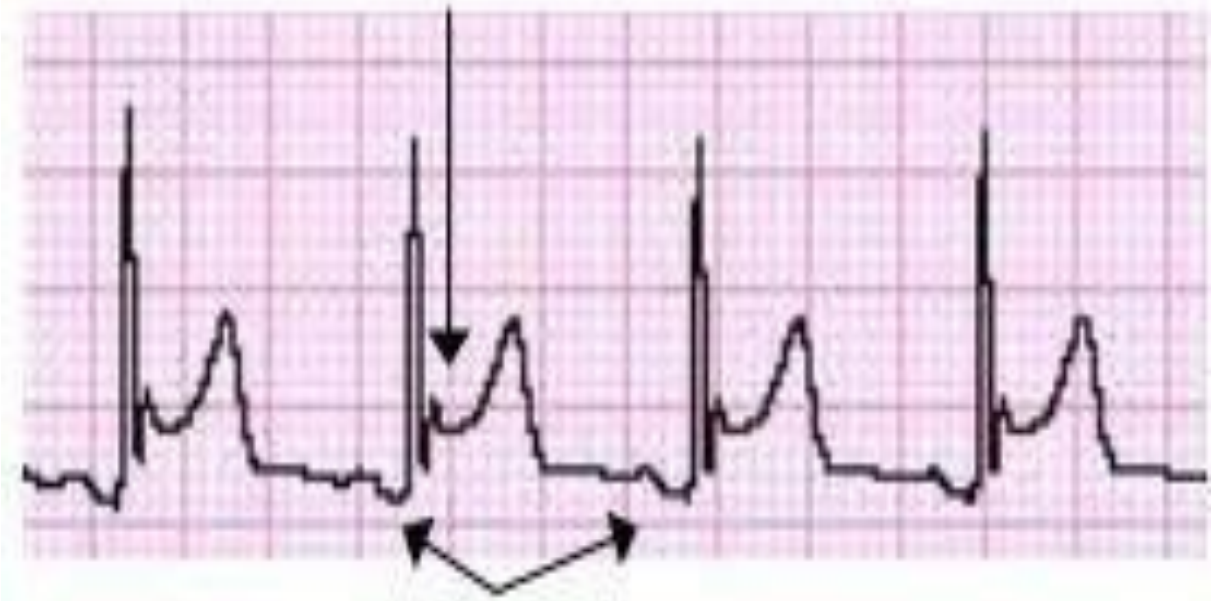


گروه پرستاران

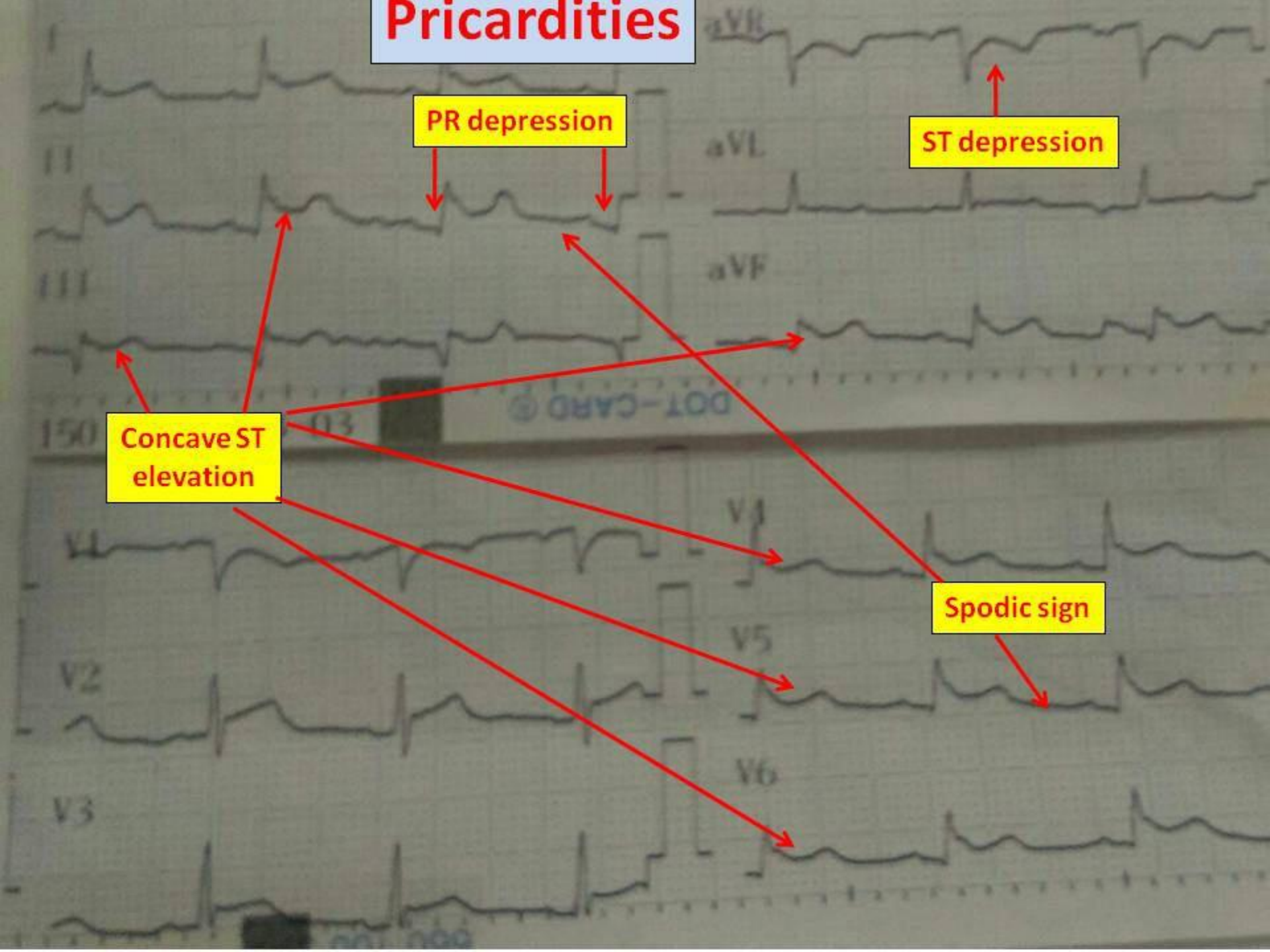
Pericarditis



Concave-up ST elevation



PR segment depression

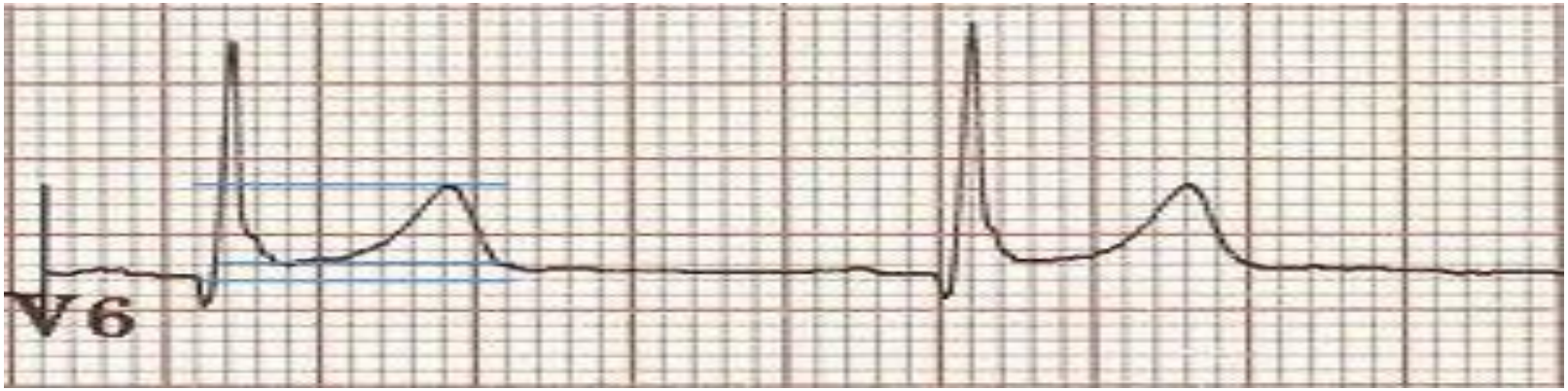

Pricardities
PR depression
ST depression
Concave ST elevation
Spodic sign

Differenitation Between Pericarditis & Early Repolarization

!The vertical height of the ST segment elevation (from the end of the PR segment to the J point) is measured and compared to the amplitude of the T wave in V6.

- A ratio of > 0.25 suggests pericarditis
- A ratio of < 0.25 suggests BER

ST segment height = 1 mm
T wave height = 6 mm
ST / T wave ratio = 0.16
The ST / T wave ratio < 0.25 is consistent with BER.

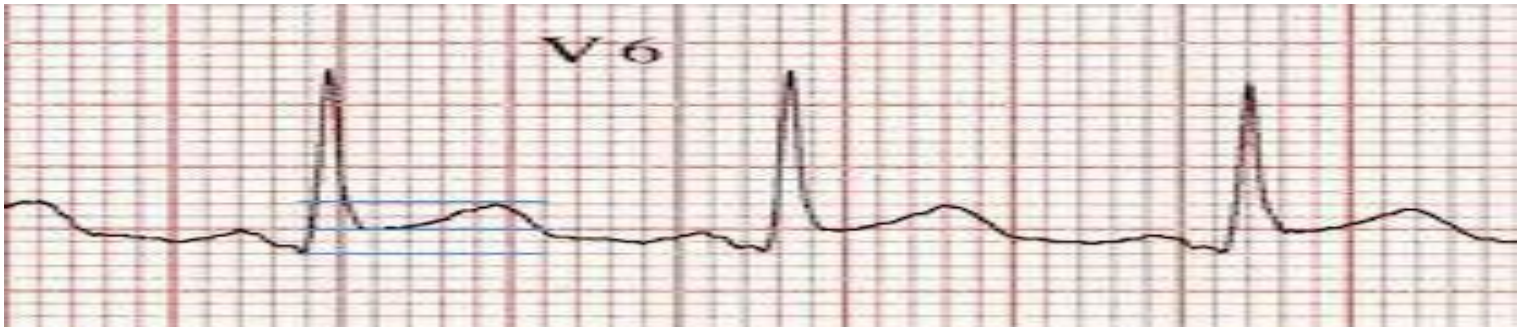


ST segment height = 2 mm

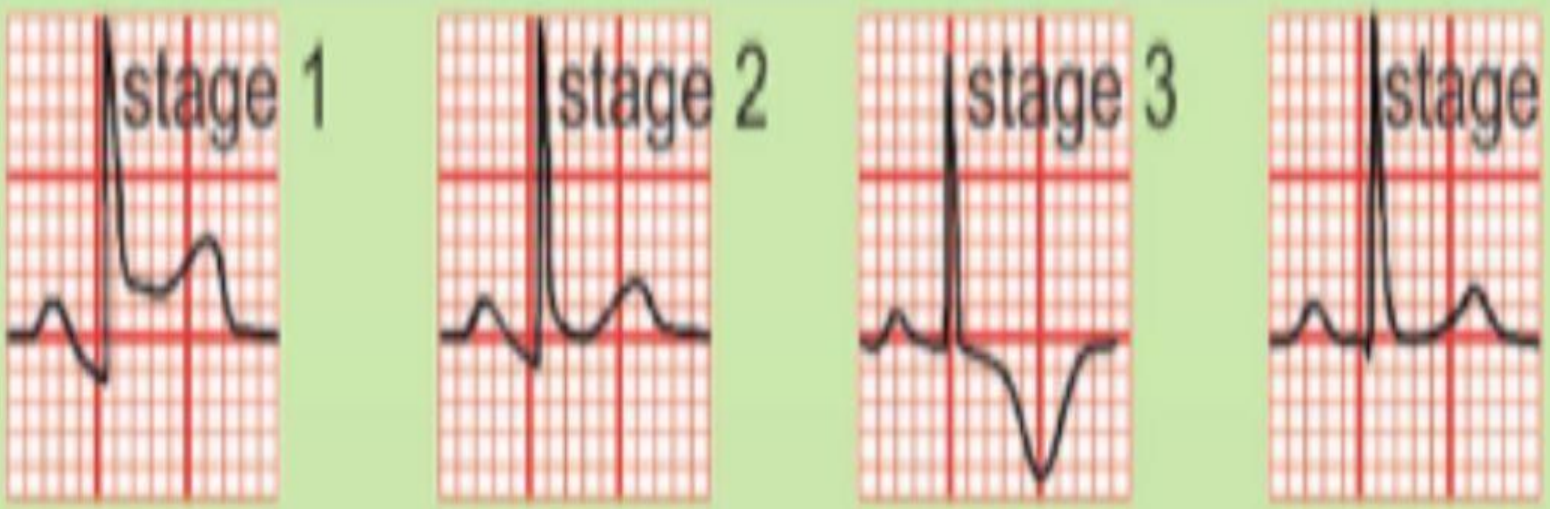
T wave height = 4 mm

ST / T wave ratio = 0.5

The ST / T wave ratio > 0.25 is consistent with pericarditis.

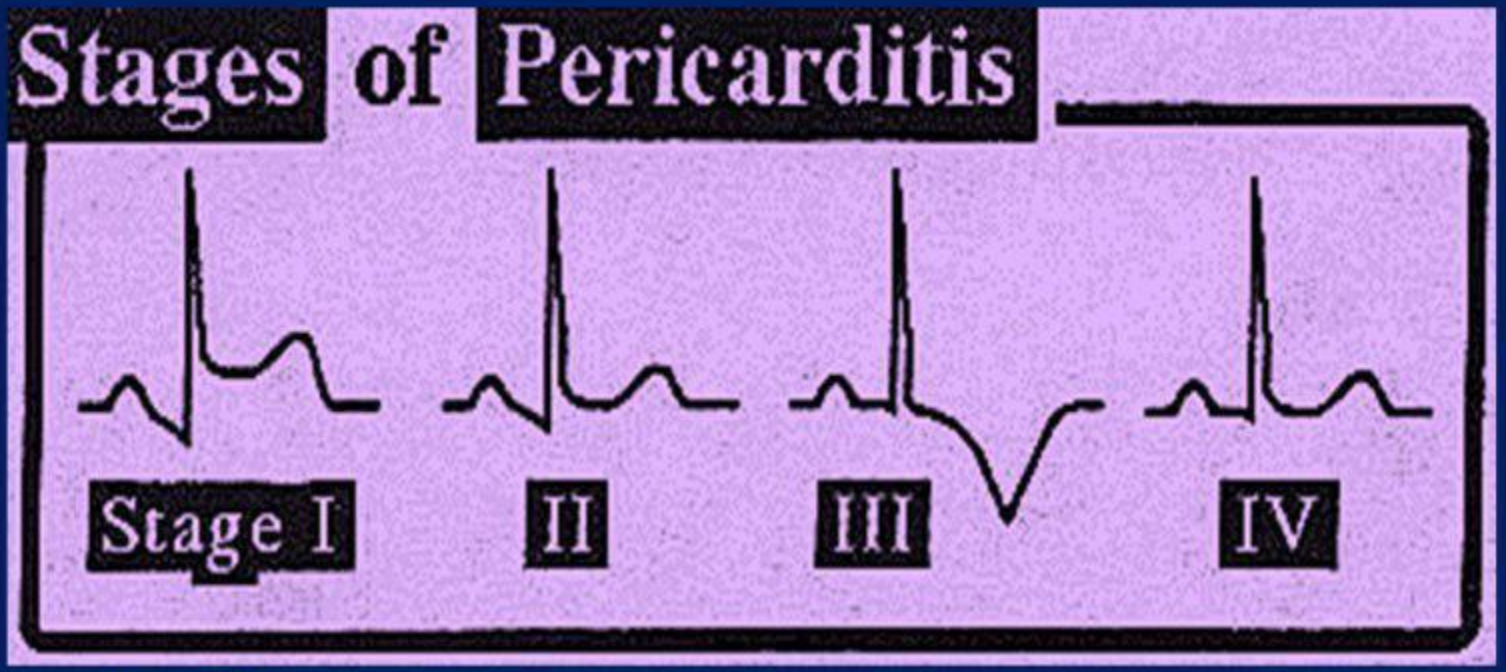


پریکاردیت Pericarditis



مشخصات : قطعه ST به شکل کفه‌ای بالا رفته و فاصله P تا Q دپرس می شود، سپس یکبار طبیعی شدن، بعد منفی شدن T و نهایتاً طبیعی شدن کلی شکل می گیرد.

Stages of Pericarditis



- **Stage I**
  everything is UP (i.e., ST elevation in almost all leads - see below)
- **Stage II**
  Transition (i.e., "pseudonormalization").
- **Stage III**
  Everything is DOWN (inverted T waves).
- **Stage IV**
  Normalization

Wide QRS tachycardia

- **Features for differentiation :**

- History and physical examination:

1. Previous MI , angina , CHF – have 95% for diagnosing **VT**
2. Pts with VT are older than **SVT-A** (> 35 yrs)
3. **SVT-A** often have previous episode(>3years)
4. Pts with **SVT-A** are hemodynamic stable.
5. Termination of WCT with physical manoeuvres and medications

Wide QRS tachycardia

1. **QRS duration :**

- > 160 ms with LBBB , >140 ms with RBBB - **VT**

Exceptions:

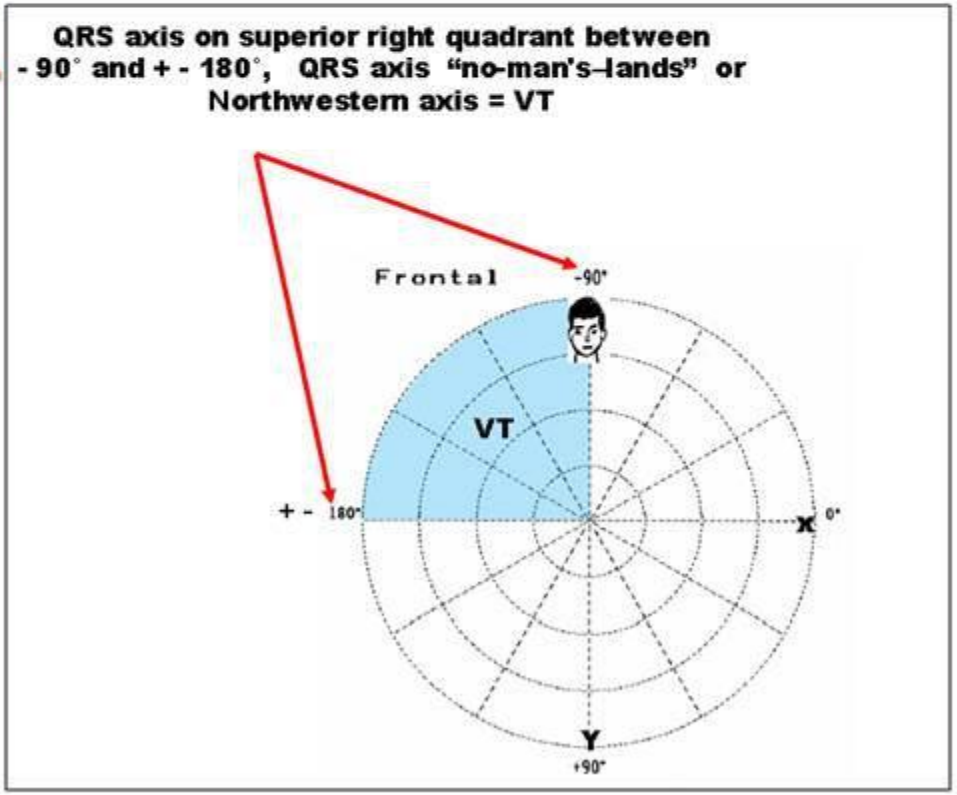
a. Anti arrythmitic drugs non specifically prolong QRS duration.
b. Pts with structurally normal heart may have VT with QRS duration of 120-140ms.(<140ms in12% , < 120 ms in 4%)
c. QRS duration also depend site of origin of VT , septal VT

QRS duration has sensitivity of 70%

Wide QRS tachycardia

2. QRS axis :

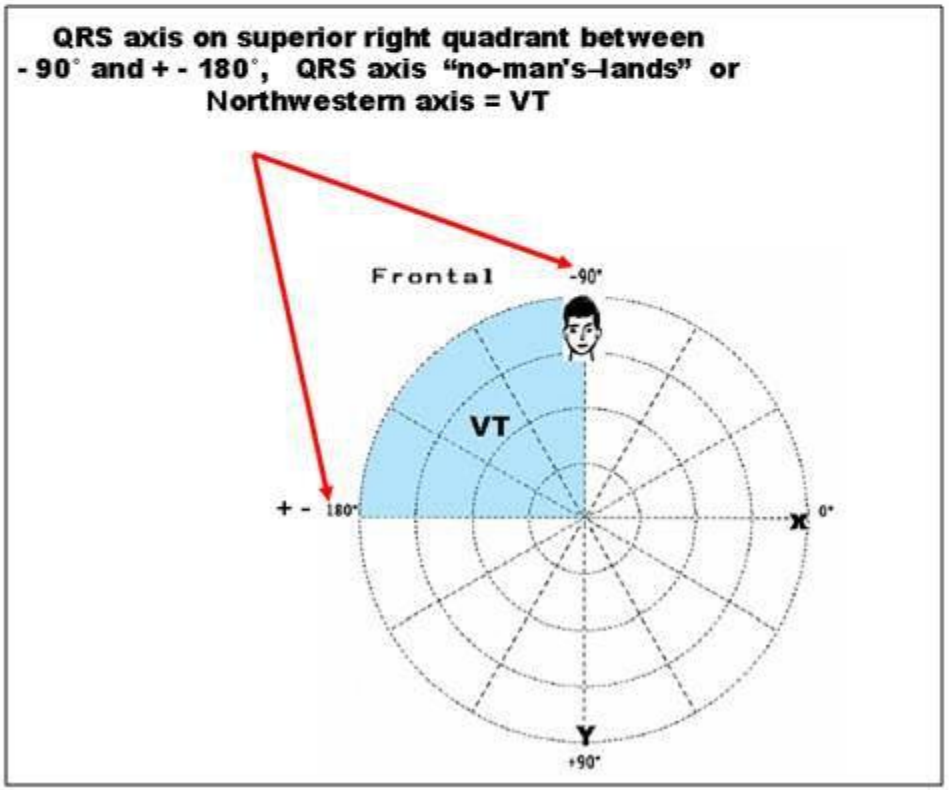
- Frontal plane axis of -90 to +180 --- **VT**
- RBBB with LAD --- **VT**
- LBBB with RAD --- **VT**



Wide QRS tachycardia

3. Concordant QRS in limb leads :

- The presence of predominantly negative QRS complexes in leads I,II,III is suggestive of **VT**
- Highly specific for **VT**

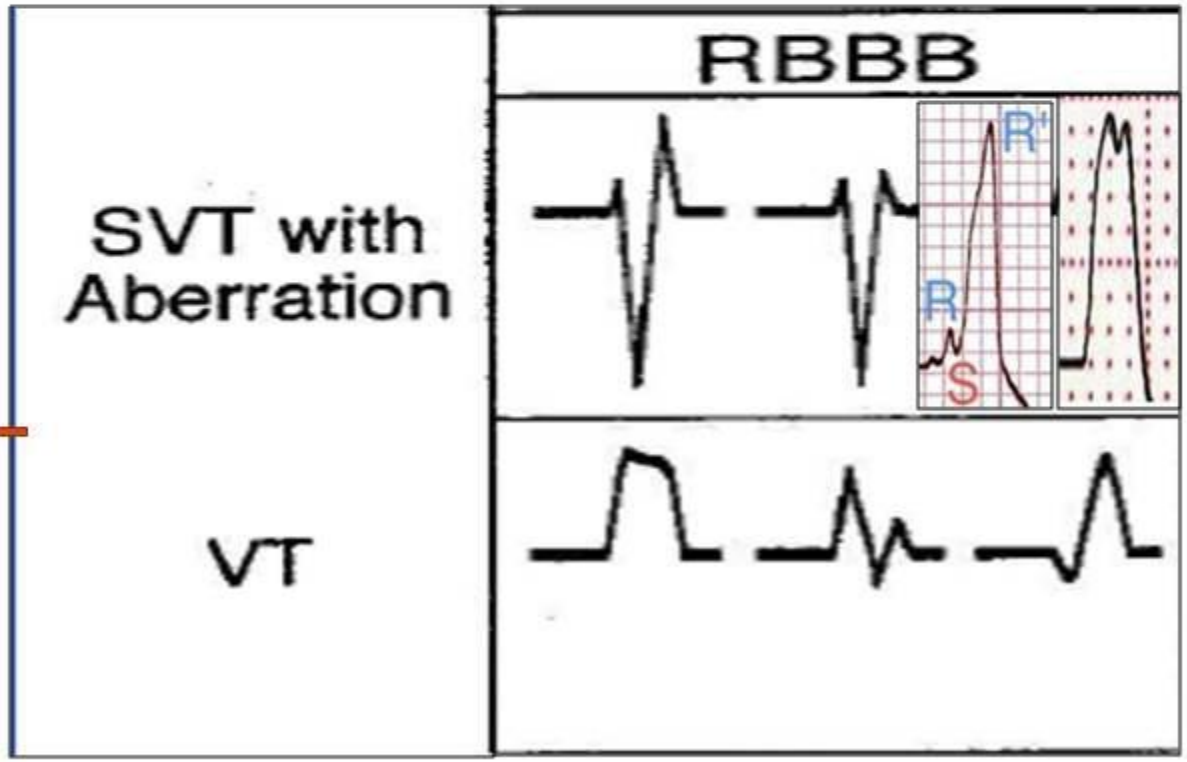


Wide QRS tachycardia

5. RBBB – V1 :

- rSR,rSr , rR , rsr patterns consistent ---- **SVT**
- R>30ms with any negative QRS , qR --- **VT**

Sensitivity – 30-80%
Specificity - 84-95%

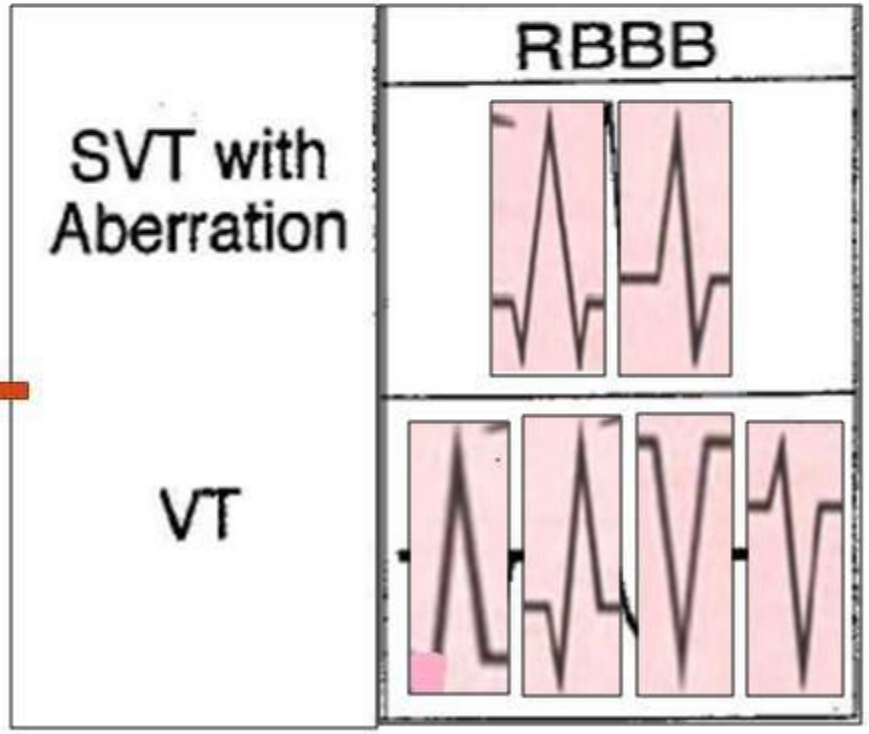


Wide QRS tachycardia

5. RBBB – V6 :

- qRs , Rs , R/S >1 --- **SVT–A**
- R , QR , QS , R/S < 1 --- **VT**

Sensitivity – 30-60%
Specificity - 80-100%



Wide QRS tachycardia

6. Q wave presence :

- Q during WCT --- suggest old MI --- **VT most likely**.

Exceptions :

1. Pts with DCMP will have Q wave during VT that are not present during baseline.
2. PSEUDO Q wave with retrograde p wave deforming QRS can be seen in SVT-A
3. Preexcited tachycardia with posterior AV connection can have Q wave in inferior leads

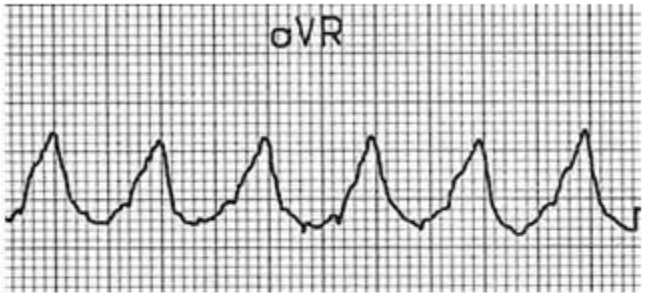
Wide QRS tachycardia

7. AV dissociation :

- The most specific ECG finding for **VT** .
- Clues for AV dissociation:

1. Clinically by cannon A waves , variable intensity of S1 , Variation in SBP unrelated to respiration.
2. AV dissociation
3. AV ratio of less than 1
4. 2:1 VA block(d/t retrograde conduction)
5. Variation in QRS amplitude during WCT
6. Fusion & capture beats
7. Recording separate atrial electro gram (oesophageal/transvenous)
8. Echo (evaluating RA contraction in relation to ventricular)

Wide QRS tachycardia

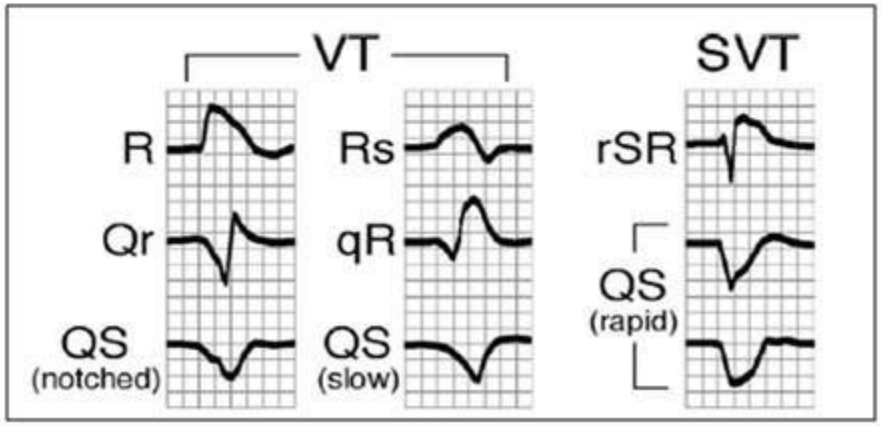


9. AVR changes :

1. initial '**r**' wave in **AVR**
2. initial '**r**' or '**q**' wave of > 40ms duration
3. **notch** in descending limb of negative onset and predominantly negative QRS
4. Vi/Vt ≤ 1

All the above features are indicative of VT

Sensitivity – 96.7%
Specificity – 99%

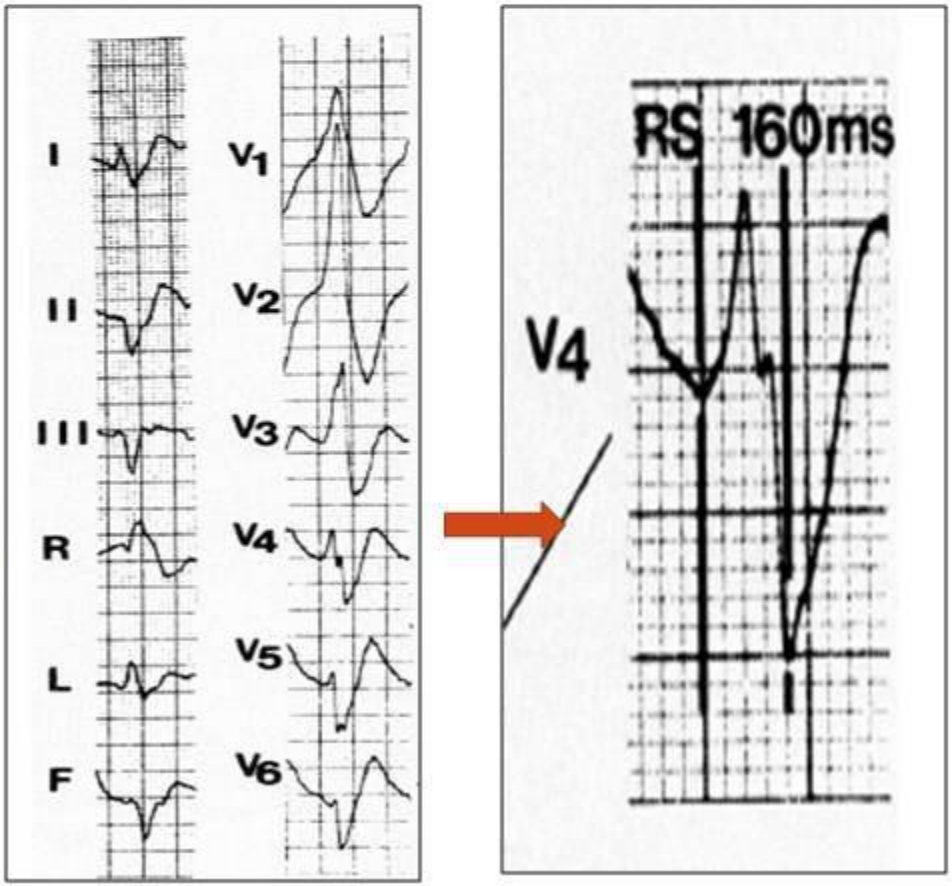


Wide QRS tachycardia

4. Pericordial RS duration criteria :

- If concordant QRS complexes are absent with RS complex (**R wave to nadir of S wave** > 100 ms).----**VT**

Sensitivity – 66%
Specificity - 98%



Wide QRS tachycardia

3. Concordant QRS in chest leads:

- Concordant QRS in chest leads is diagnostic of **VT** uncommon in SVT-A.
- Exceptions:
  - Positive concordance (ventricular activation begins left posteriorly) seen in VT originating in Lt post wall or SVT using a left posterior accessory pathway for AV conduction.
  - If no additional criteria for WPW are absent don't consider it because of low incidence(<6%)

Specificity of 90%, Sensitivity of 20%

Wide QRS tachycardia

7. AV dissociation :

- The most specific ECG finding for **VT** .
- Clues for AV dissociation:

1. Clinically by cannon A waves , variable intensity of S1 , Variation in SBP unrelated to respiration.
2. AV dissociation
3. AV ratio of less than 1
4. 2:1 VA block(d/t retrograde conduction)
5. Variation in QRS amplitude during WCT
6. Fusion & capture beats
7. Recording separate atrial electro gram (oesophageal/transvenous)
8. Echo (evaluating RA contraction in relation to ventricular)

Wide QRS tachycardia

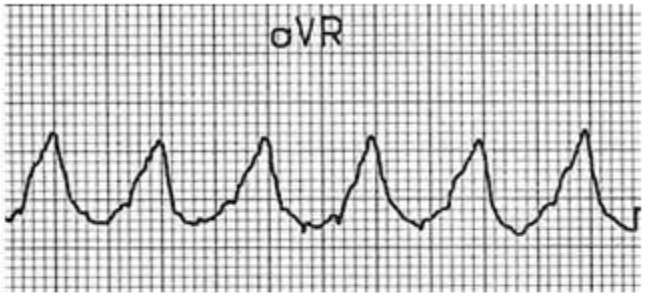
6. Q wave presence :

- Q during WCT --- suggest old MI --- **VT most likely**.

Exceptions :

1. Pts with DCMP will have Q wave during VT that are not present during baseline.
2. PSEUDO Q wave with retrograde p wave deforming QRS can be seen in SVT-A
3. Preexcited tachycardia with posterior AV connection can have Q wave in inferior leads

Wide QRS tachycardia



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All the above features are indicative of VT

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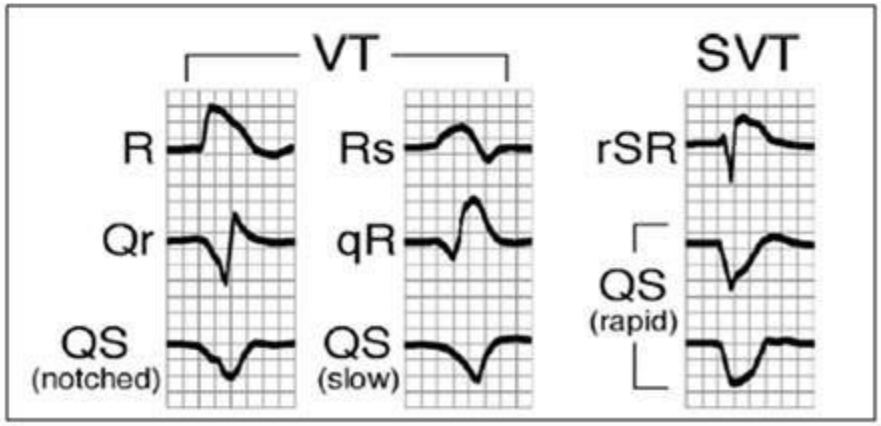
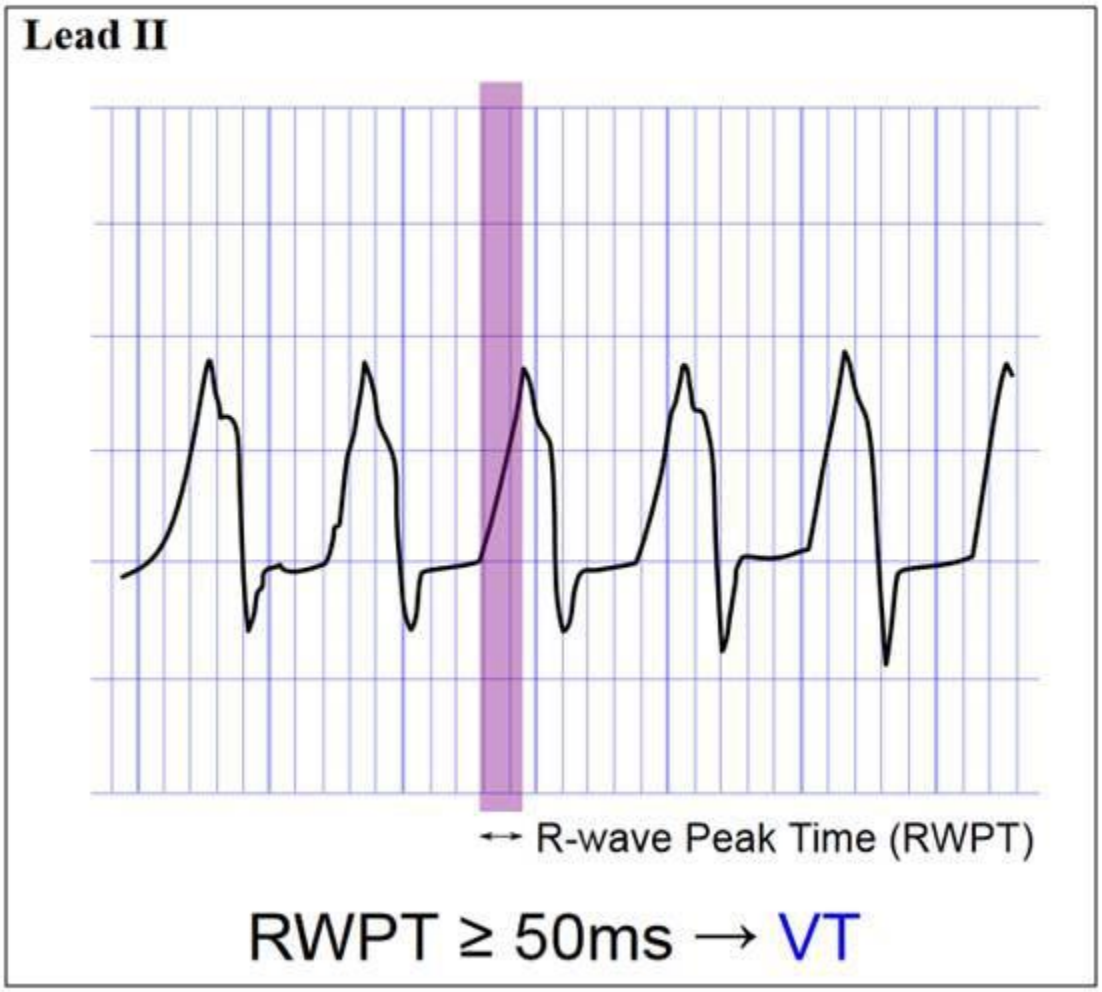


Wide QRS tachycardia

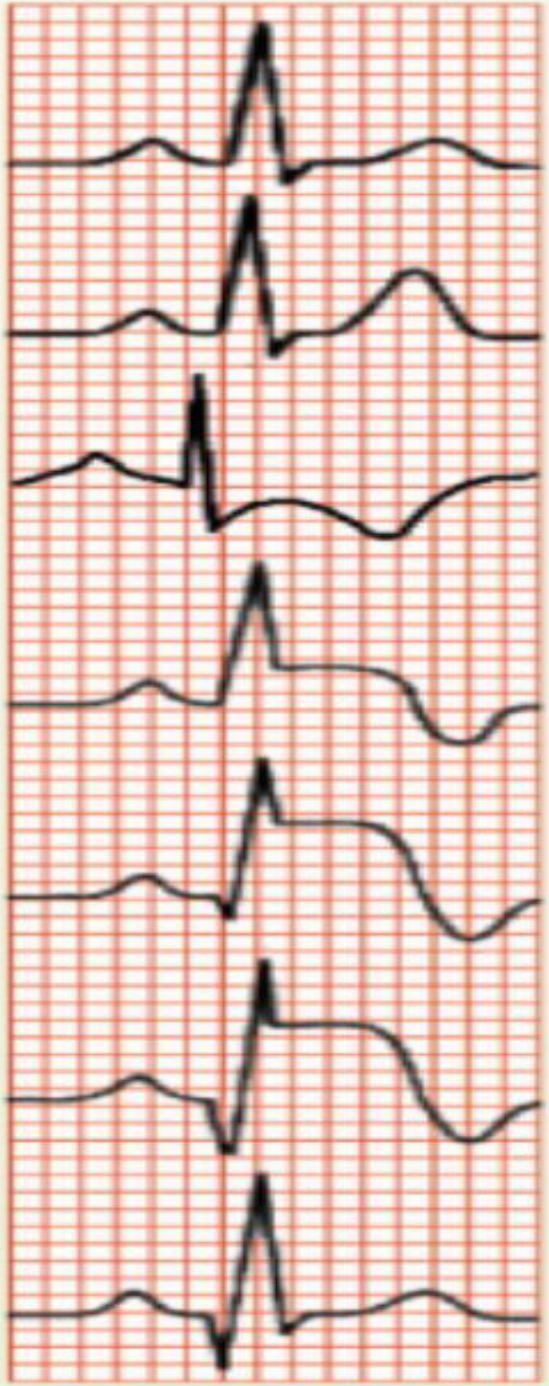
10. Lead II R-wave-peak-time (RWPT) criterion : Pavas criteria



RWPT > or =50 ms at DII is a **simple and highly sensitive** criterion that discriminates VT from SVT in patients with wide QRS complex tachycardia.

Sensitivity and specificity of 97%

Heart Rhythm. 2010 Jul;7(7):922-6. Epub 2010 Mar 4.



Normal

Hyperacute
T wave

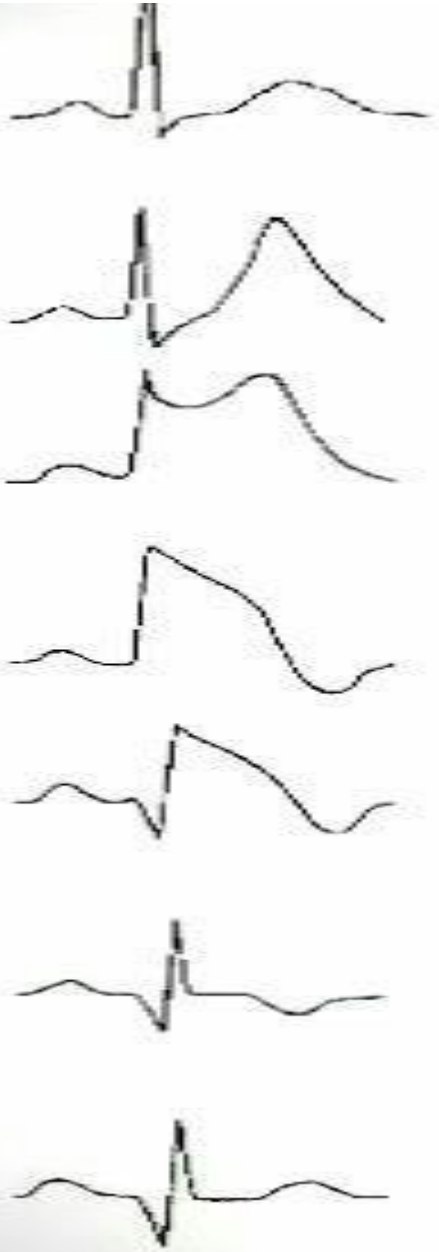
T wave invertion
or ST depression

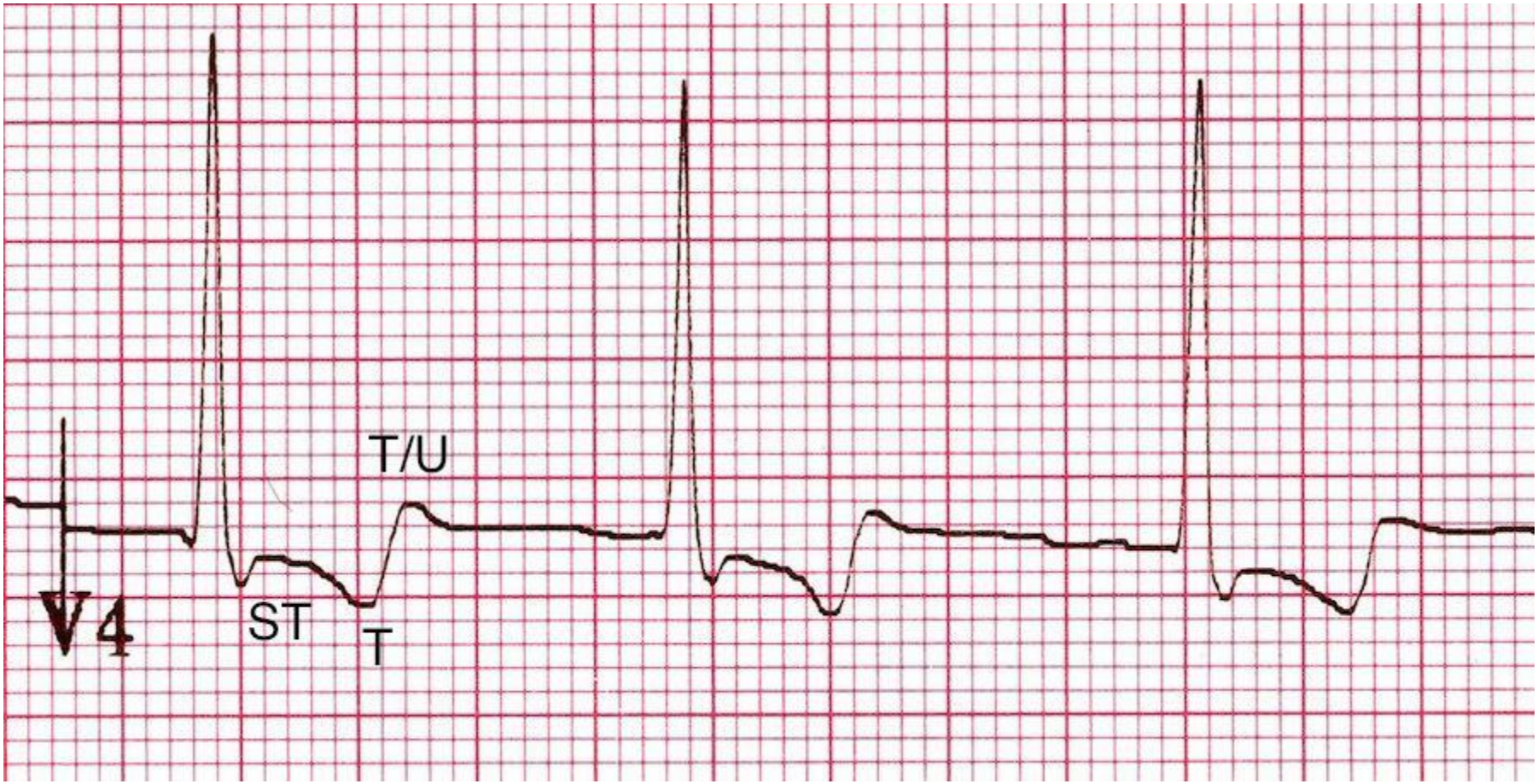
ST Elevation
non-Q wave

ST Elevation
Q wave

development

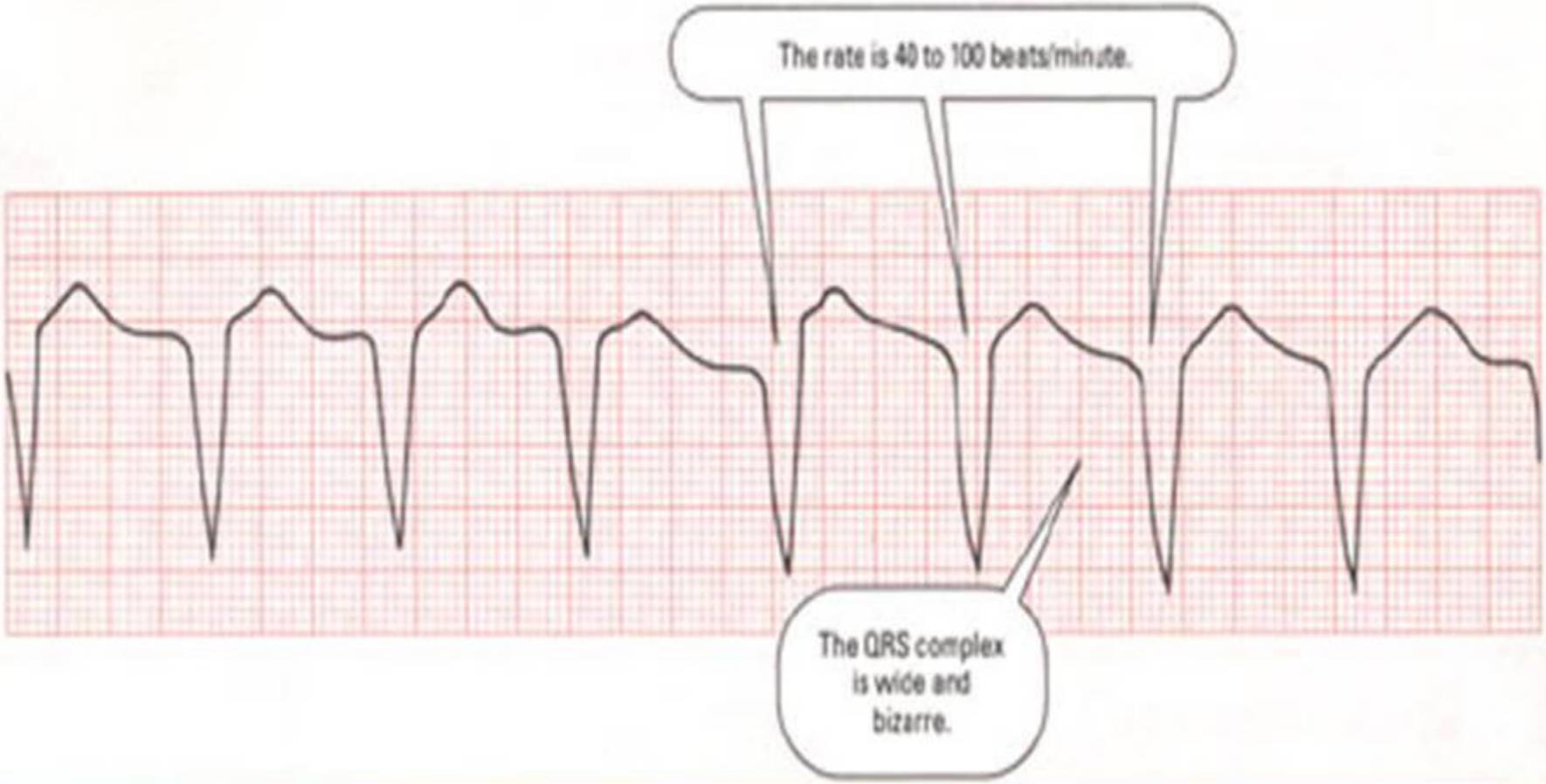
ST and T wave
normalization


Before
Minutes afterwards
Hours afterwards
Days afterwards
Weeks afterwards


T/U
V4
ST
T

Accelerated idioventricular rhythm

An accelerated idioventricular rhythm has the same characteristics as an idioventricular rhythm except that it's faster. The rate shown here varies between 40 and 100 beats/minute.



Causes of chest pain

- Cardiovascular
  - A.C.S.
  - Pericarditis
  - Aortic dissection
  - Aortic stenosis
- Pulmonary
  - Pulmonary embolism
  - Pleurisy
  - Pneumothorax
  - Pneumonia
- Pediatrics
  - Kawasaki disease
  - Hypertrophic cardiomyopathy
  - Congenital heart disease
- Gastrointestinal
  - Esophageal reflux
  - Esophageal spasm
  - Esophageal rupture
  - Peptic ulcer disease
  - Gallbladder disease
  - Pancreatitis
- Chest Wall Pain
  - Herpes Zoster
  - Costochondritis
  - Cervical radiculopathy
  - Rib fracture
  - Anxiety

- IHD
  - stable Angina
  - ACS
    - Unstable Angina
    - Myocardial Infarction
      - ST Elevation MI
      - New LBBB
      - Non ST Elevation MI

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Stable vs. Unstable Angina

Stable Angina	Unstable Angina
Episodic	Severe and of New onset
Crescendo - Decrescendo	Crescendo pattern
Occurs on exertion, relieved by rest	Occurs at rest
Lasts 2-5 mins	Lasts > 10 min

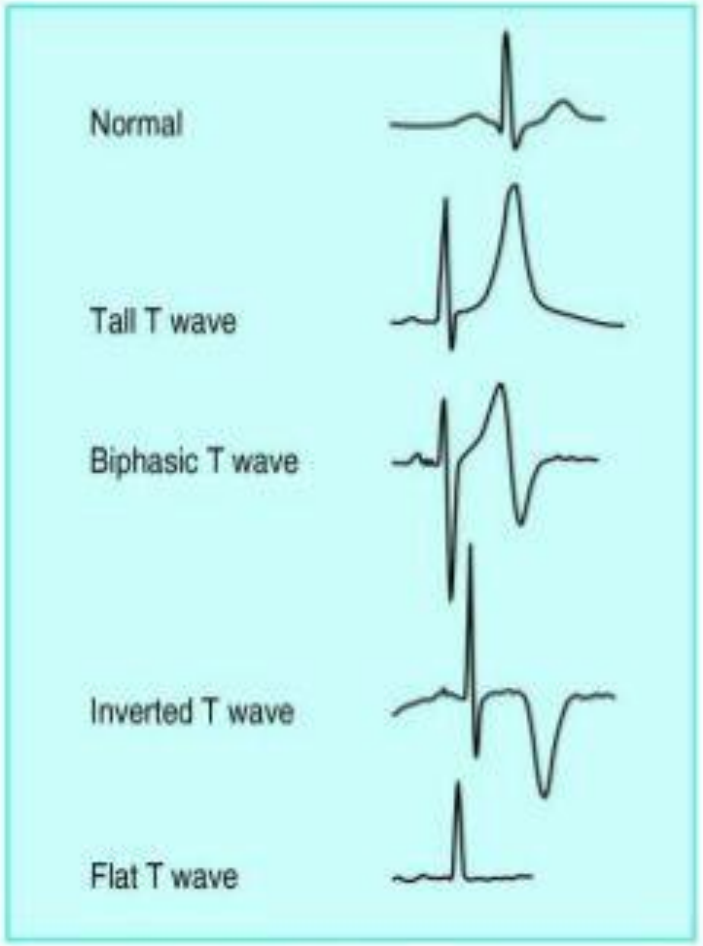
VARIANT ANGINA (VA)

- Temporary increase in coronary vascular tone (vasospasm) causing a marked, but transient reduction in luminal diameter[1]
- Patients are predominantly younger women[1]
- It has been associated with vasospastic disorders such as Raynaud's phenomenon and migraine headaches.[1]

1. Keller KB, Lemberg L. Prinzmetal's angina. Am J Crit Care. 2004;13(4):350-4

ECG effects of myocardial ischemia

- T waves may become
  - Flattened
  - Inverted
  - Tall or
  - Biphasic

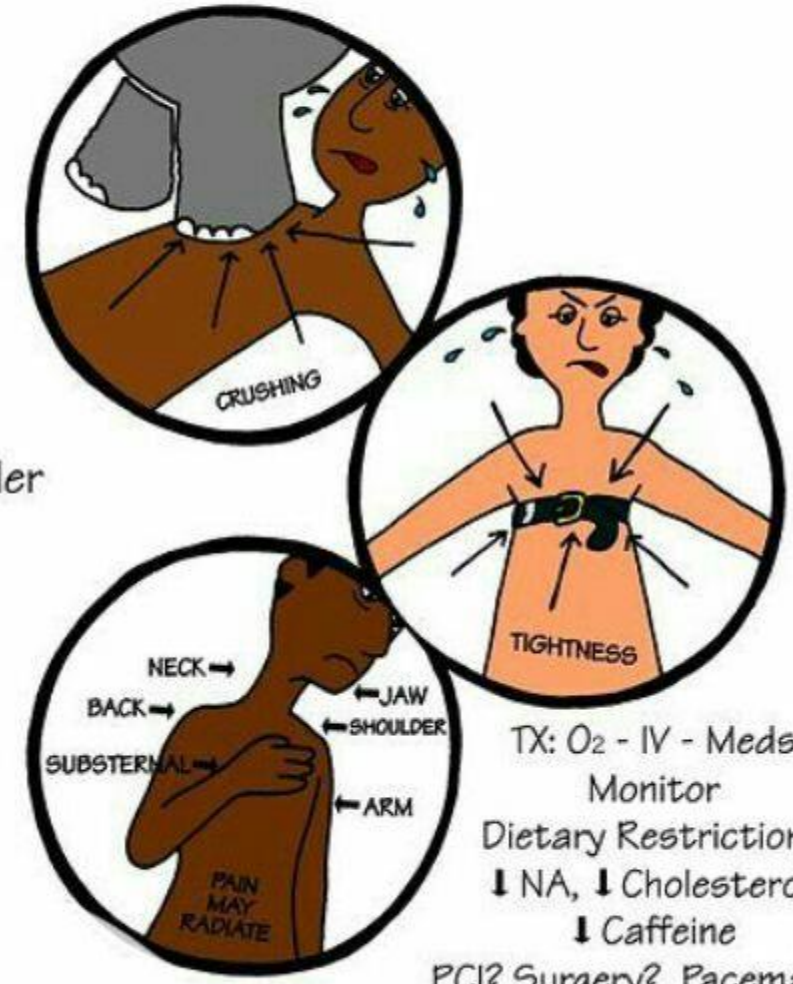


- MYOCARDIAL INFARCTION (MI) -

- CORONARY OCCLUSION -

- HEART ATTACK -

- Pain:
  - Sudden Onset
  - Substernal
  - Crushing
  - Tightness
  - Severe
  - Unrelieved by Nitro
  - May Radiate To: Back
    - Neck
    - Jaw
    - Shoulder
    - Arm
- Dyspnea
- Syncope (⬇ BP)
- Nausea
- Vomiting
- Extreme Weakness
- Diaphoresis
- Denial is Common
- ⬆ HR



TX: O_2 - IV - Meds
Monitor
Dietary Restrictions
⬇ NA, ⬇ Cholesterol,
⬇ Caffeine
PCI? Surgery? Pacemaker?

Subendocardial VS Transmural

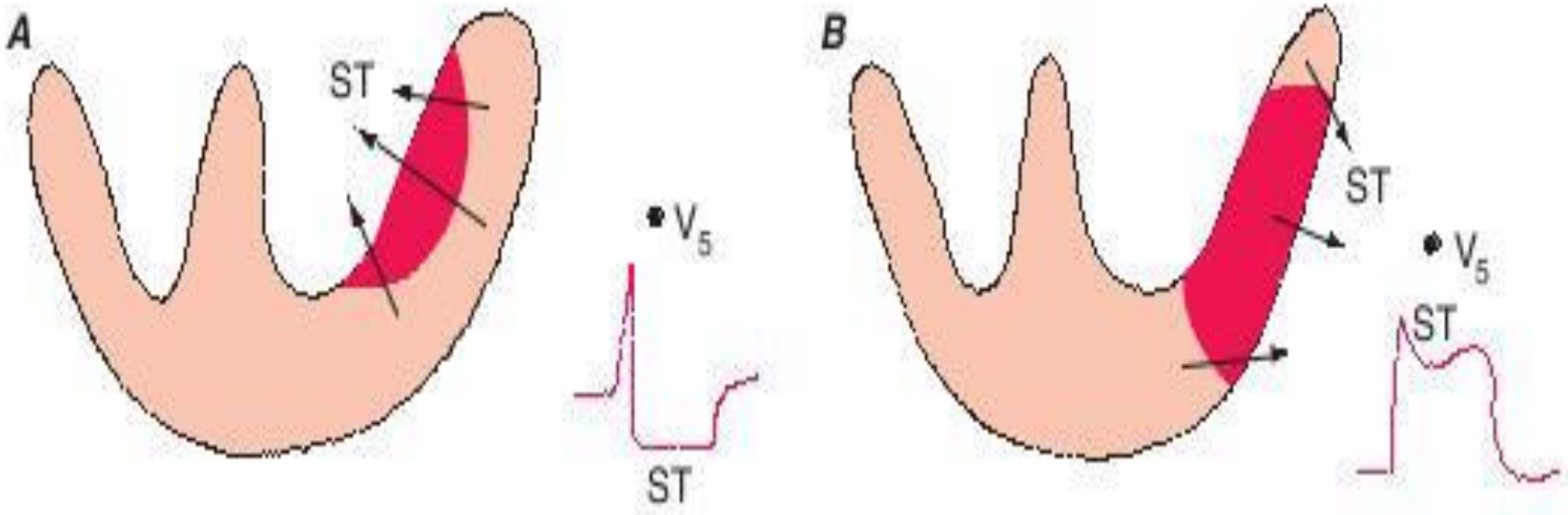
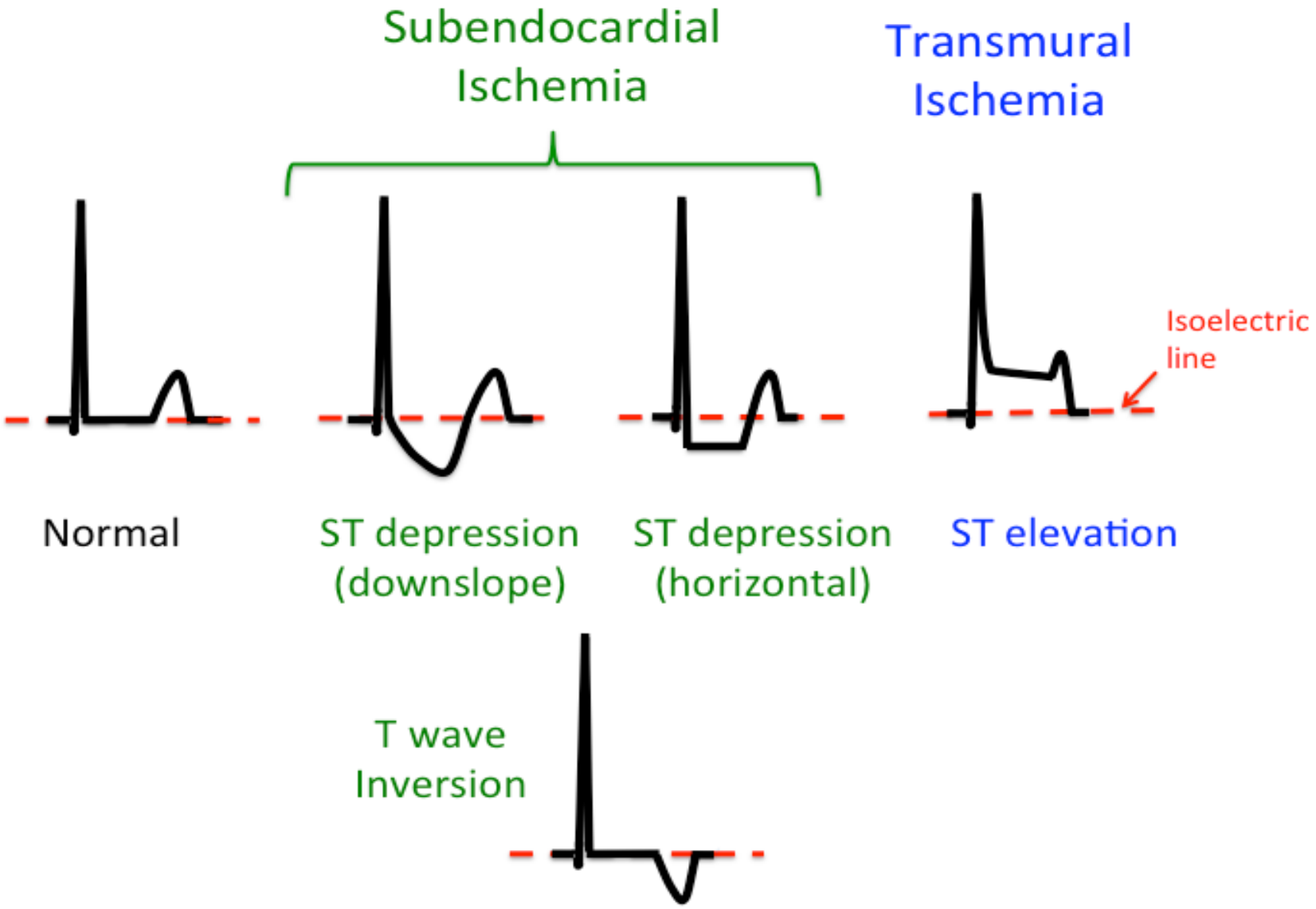
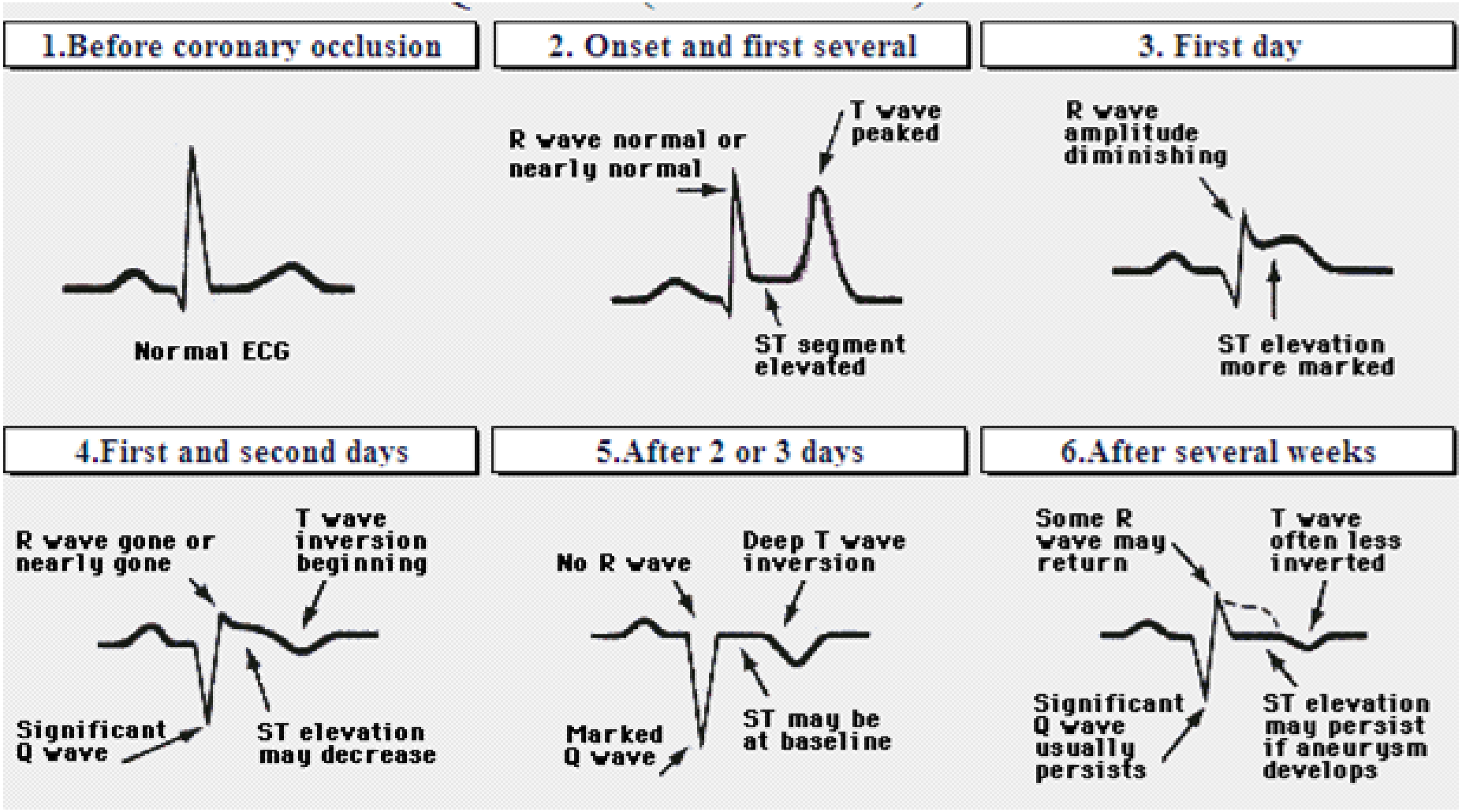


FIGURE 221-11 Acute ischemia causes a current of injury. With predominant subendocardial ischemia (***A***), the resultant ST vector will be directed toward the inner layer of the affected ventricle and the ventricular cavity. Overlying leads therefore will record ST depression. With ischemia involving the outer ventricular layer (***B***) (transmural or epicardial injury), the ST vector will be directed outward. Overlying leads will record ST elevation.


Subendocardial Ischemia
Transmural Ischemia
Isoelectric line
Normal
ST depression (downslope)
ST depression (horizontal)
ST elevation
T wave Inversion

Serial ECG changes of MI



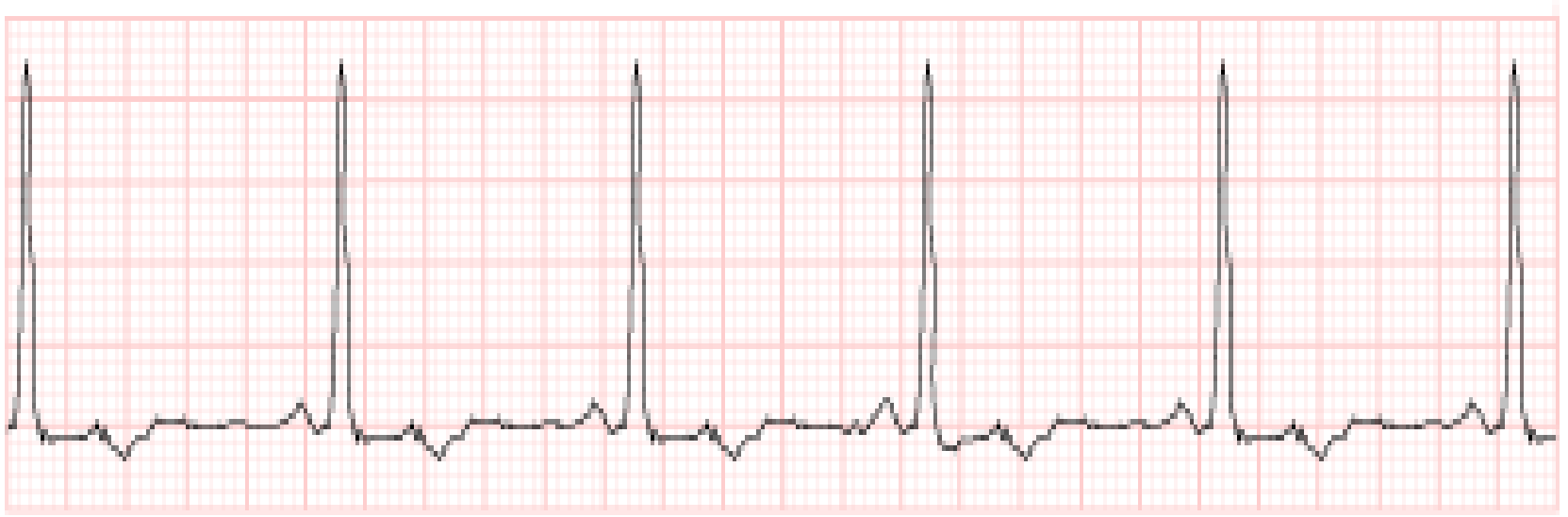
ST Segment Elevations

I. Myocardial ischemia/infarction
   A. Noninfarction, transmural ischemia (Prinzmetal's angina pattern)
   B. Acute myocardial infarction
   C. Post–myocardial infarction (ventricular aneurysm pattern)
II. Acute pericarditis
III. Normal variant ("early repolarization" pattern)
IV. Left ventricular hypertrophy (LVH)/left bundle branch block (LBBB) (V_1–V_2 or V_3 and other leads with QS or rS waves, only)
V. Brugada pattern (right bundle branch block (RBBB)–like pattern with ST elevations in right precordial leads)
VI. Other (rarer)
   A. Myocardial injury (non-infarction)
      1. Myocarditis (ECG may resemble myocardial infarction or pericarditis patterns)
      2. Tumor invading the left ventricle
      3. Trauma to the ventricles
   B. Hypothermia (J waves/Osborn waves)
   C. Hyperkalemia (usually localized to V_1 and V_2)

ST Segment Depressions

- I. **Myocardial ischemia or**
- **infarction**
- A. Acute subendocardial
- ischemia
- B. B. Reciprocal change with
- acute transmural ischemia
- II. **Abnormal noncoronary**
- **patterns**
- A. LVH or RVH: “strain”
- pattern)
- B. Secondary ST-T changes
- 1. LBBB or RBBB
- 2. WPW
- C. Drugs (e.g., digitalis)
- D. Metabolic conditions (e.g.,
- hypokalemia)
- E. Miscellaneous
- (e.g.,cardiomyopathy)
- III. **Physiologic and normal**
- **variants***

T Wave Inversion



- **Symmetrical & at least 2mm**
- **Ischemia: T Wave inversion**

Types of Angina

- Chronic Stable Angina – Dynamic occlusion + Micro vascular dysfunction – Progressive
- Micro vascular Angina – No flow limiting stenosis – Angio normal – less severe IHD
- Unstable Angina – Dynamic occlusion + Micro vascular dysfunction + Active Thromb
- Prinzemetal Angina – Occlusive spasm, No Micro vascular dys, No thrombus – ST ↑

Micro Vascular Angina

- Normal Coronary blood flow by angiogram
- No significant CAD in epicardial blood vessels
- Cardiac micro circulation is at fault
- Poor collateral connections – younger age
- More common in women – Syndrome X
- ECG or TMT show ST - T changes repeatedly
- Patient will be symptomatic for IHD

Unstable Angina

- Presence of one or more of the three features,
- Crescendo Angina- more severe, prolonged, or frequent. Decrease in exercise capacity
- New onset (1 month) & brought on by minimal exertion. Not relieved by Nitrates
- Angina at rest as well as with minimal exertion. There are 3 classes – 1 to 3
- This may progress to NSTEMI or STEMI

Prinzemetal Angina

- Transient ST-segment elevation during chest pain due to coronary vasospasm – variant angina
- ECG with ST ↑. Becomes normal soon, No Q wave
- Intermittent chest pain
  - – often repetitive, usually at rest, early morning
- Other vasospasms - syncope, Raynaud's, migraine
- βblockers contraindicated. CCB, α blockers Rx.

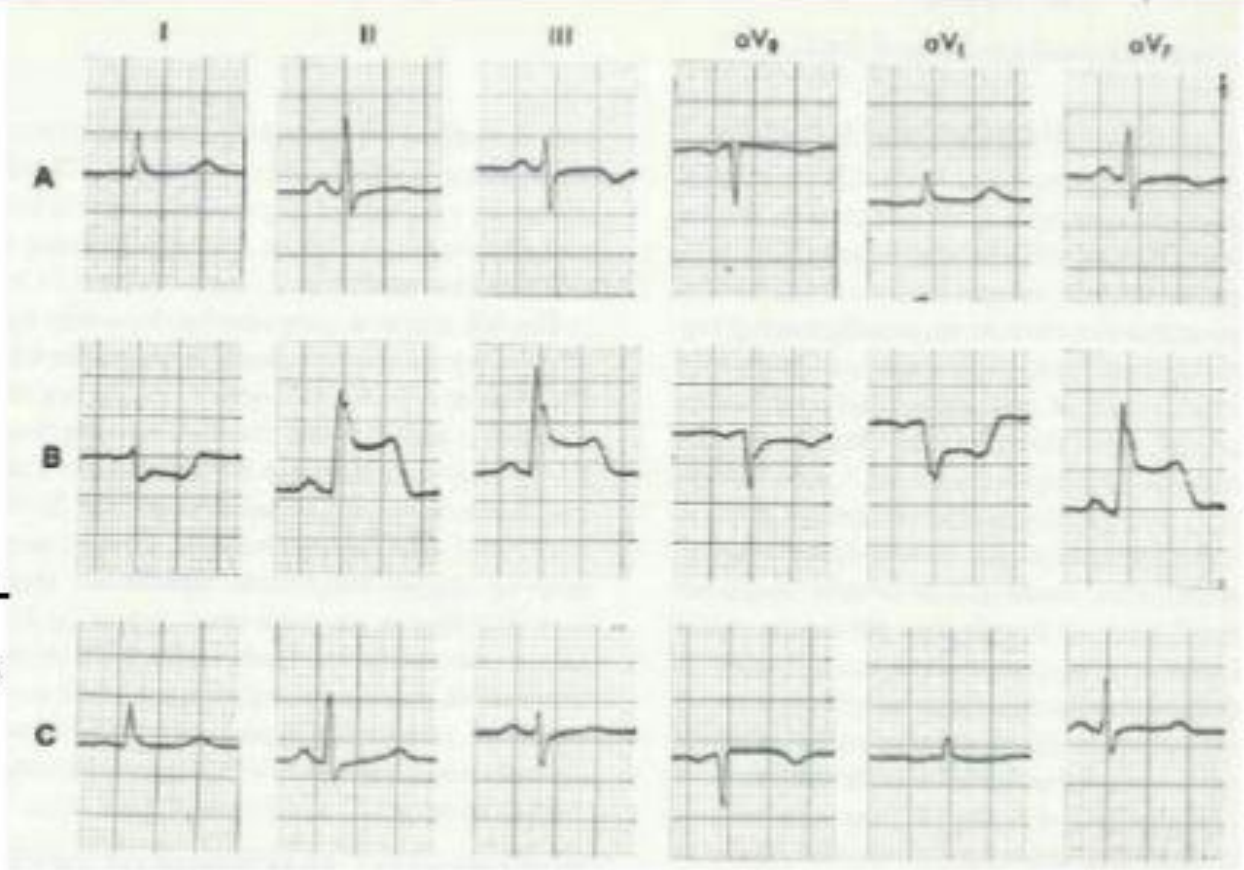
Prinzmetal's angina with transient ST elevation

Patient with history of exertional and rest angina

A. Baseline resting ECG shows non-specific inferior ST-T changes

B. With chest pain, ST elevations in II, III, aVF and reciprocal ST depression in I and aVL

C. Return of ST segments to baseline after nitroglycerin

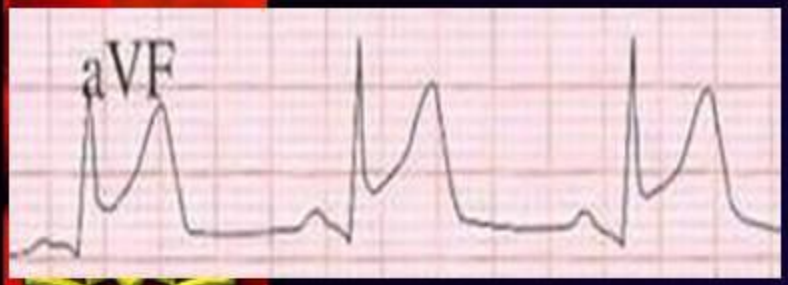


Management of ACS

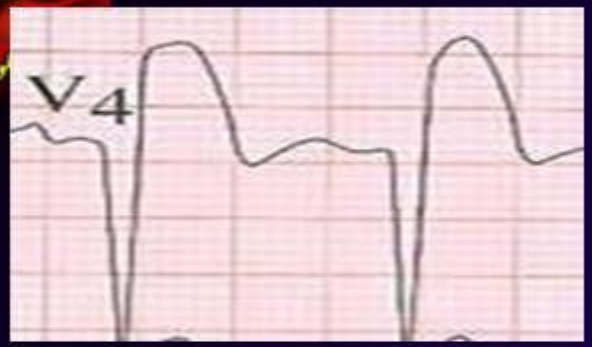
IHD type	Drug Rx.	Hep. /LMH	ICU Care	Lytic Rx	P PTCA
Stable Angina	A+B	No	No	No	No
Unstable Angina	A+B+C	Heparin	No / Yes	No	No
NSTEMI	A+B+C+G	LMH	YES	No	No
STEMI or QWMI	A+B+C+G	LMH	YES	YES	YES

A = Aspirin, B = Beta-blocker, C = Clopidogrel, G = GPIIb/IIIa Inhibitor

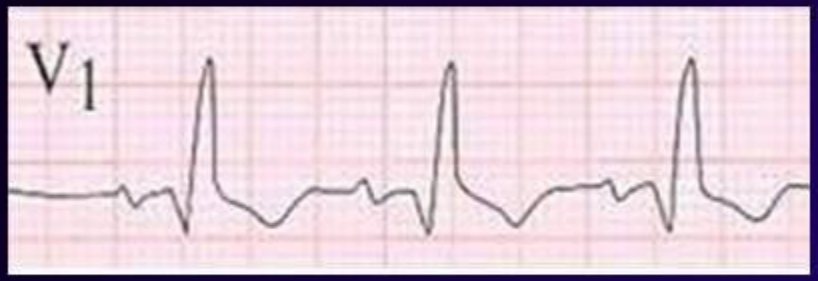
مراحل MI در ECG



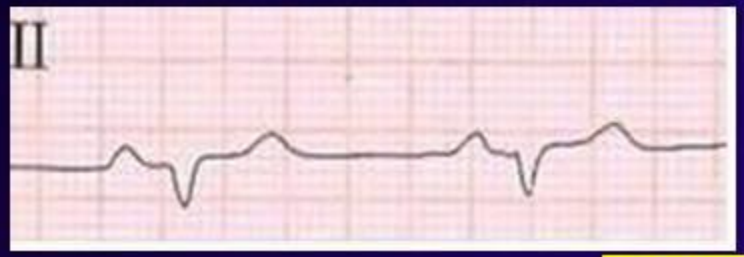
Hyper acute
T Tall (over 50% of R)



Acute
Q , ST elevation, T invert



Recent
Q , T invert



Old
Q wave

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Evolution of Acute MI

A – Normal ST segment and T waves

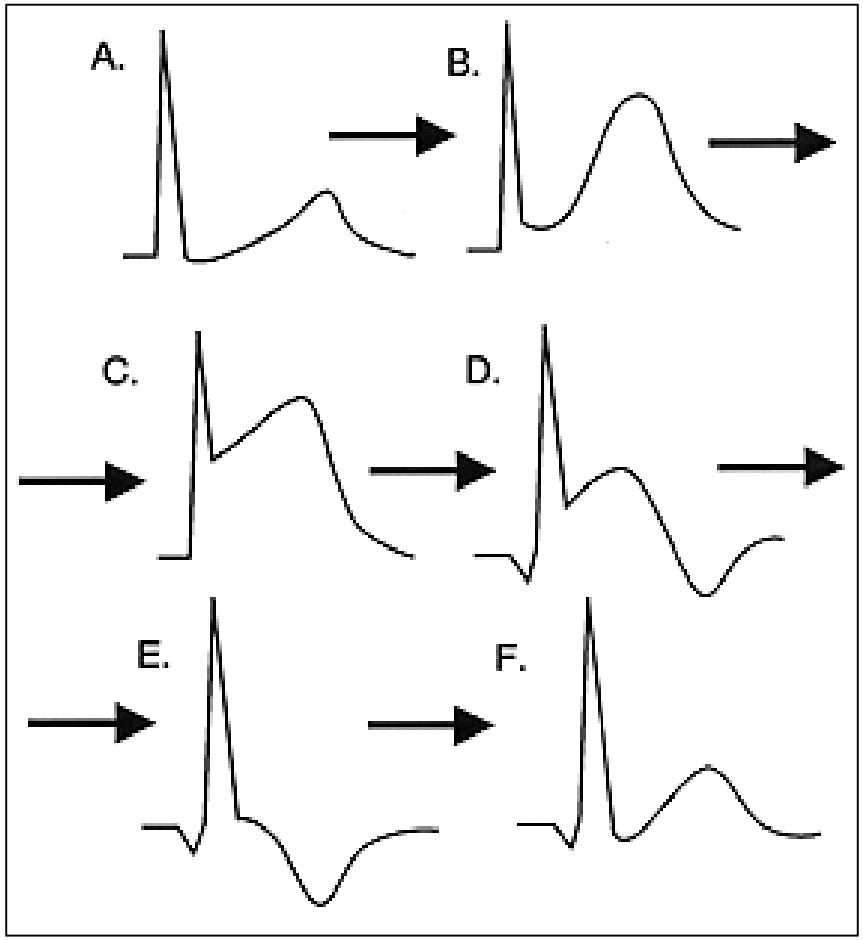
B – ST mild ↑ and prominent T waves

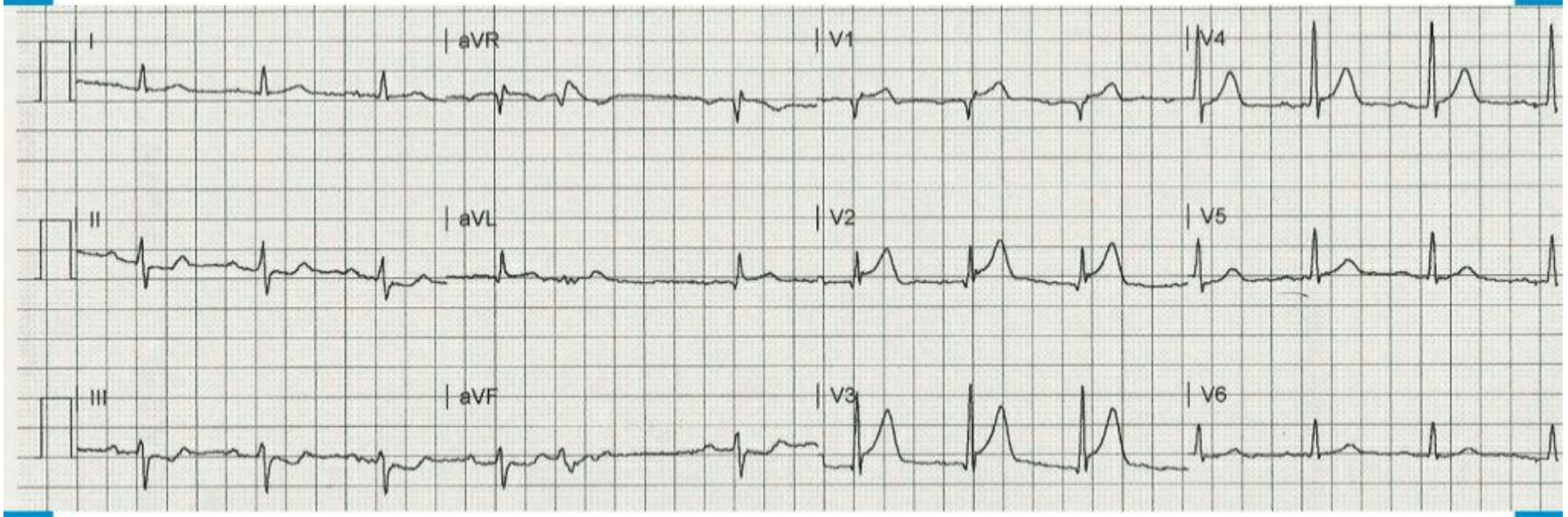
C – Marked ST ↑ + merging upright T

D – ST elevation reduced, T ↓ , Q starts

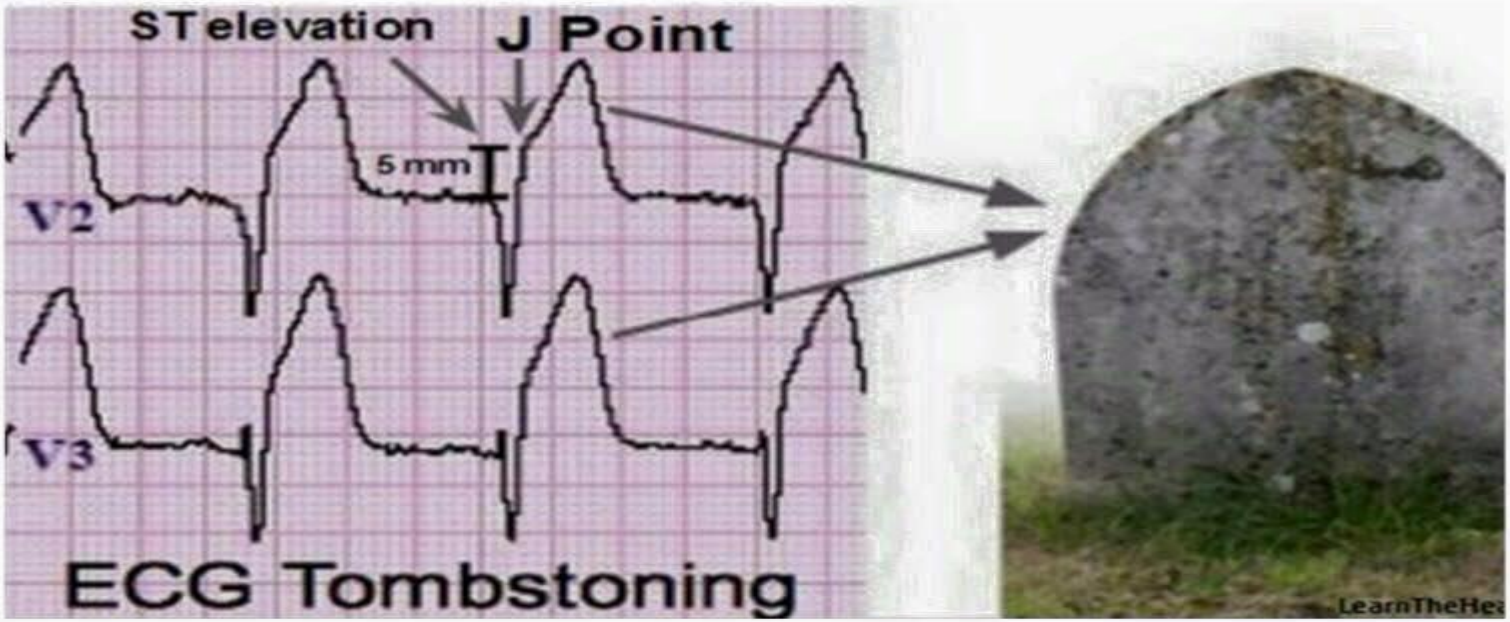
E – Deep Q waves, ST segment returning to baseline, T wave is inverted

F – ST became normal, T Upright, Only Q+



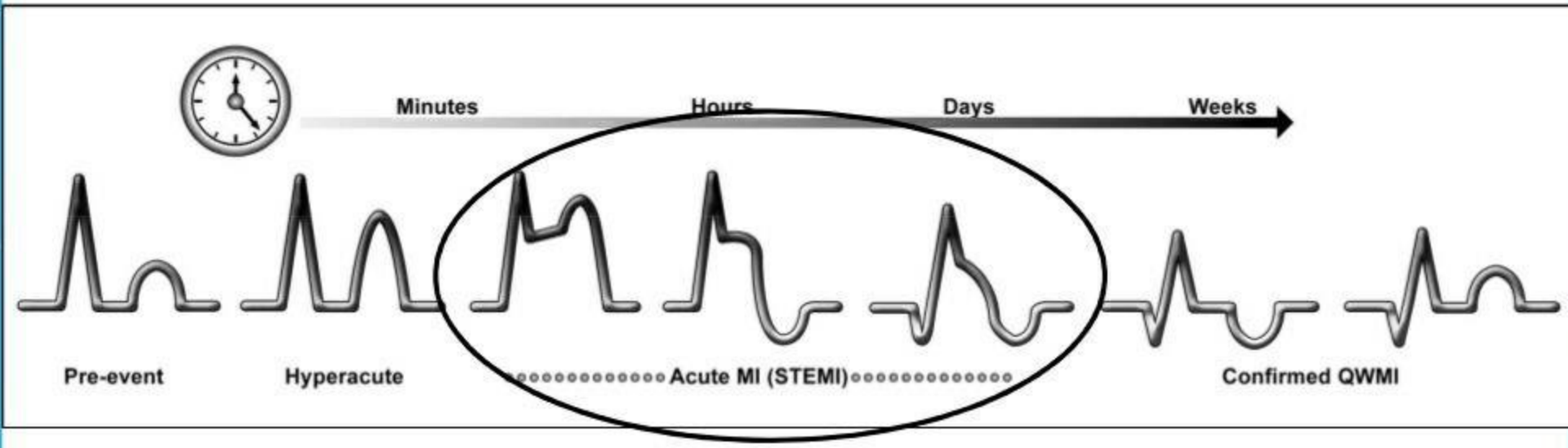


ST Segment Elevation in Leads:	**V1-V4, (aVL?)**
ST Segment Depression in Leads:	**II, III, aVF, V4, V5**
T Wave Inversion in Leads:	**III**
Pathological Q Waves in Leads:	**V1**
Interpretation/Localization:	**Acute Anteroseptal MI**

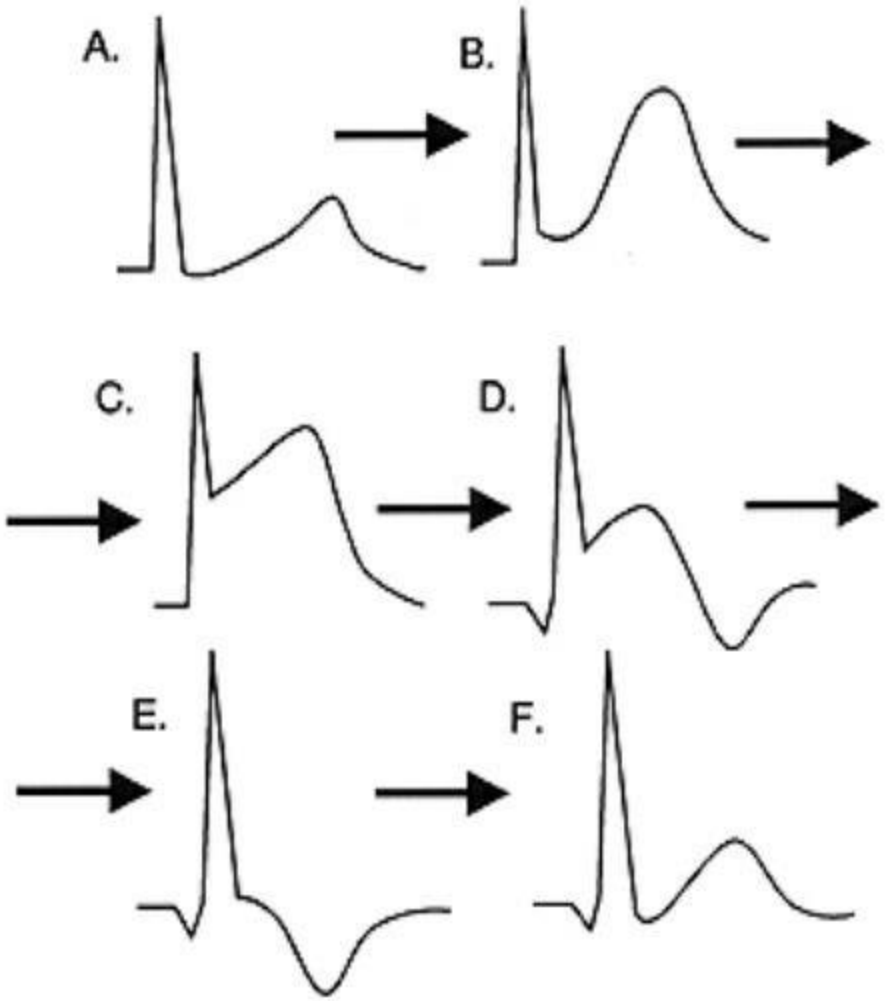


نشانه tombstone signe(سنگ قبر) چیست ؟

این نشانه در بیمارانی اطلاق می شود که دچار انفارکتوس و بدنبال آن RBBB شده اند یعنی اگر MI باعث RBBB شده باشه حتما MI extensive هست که به RBBB کشیده شده است و بیمار نخواهد توانست بهار سال آینده رو ببیند .


Minutes
Hours
Days
Weeks
Pre-event
Hyperacute
Acute MI (STEMI)
Confirmed QWMI

Evolution of STEMI



A. Normal

B. Hyper-acute T-waves: Minutes to Hours

C. ST-Elevation: 0-12 Hours

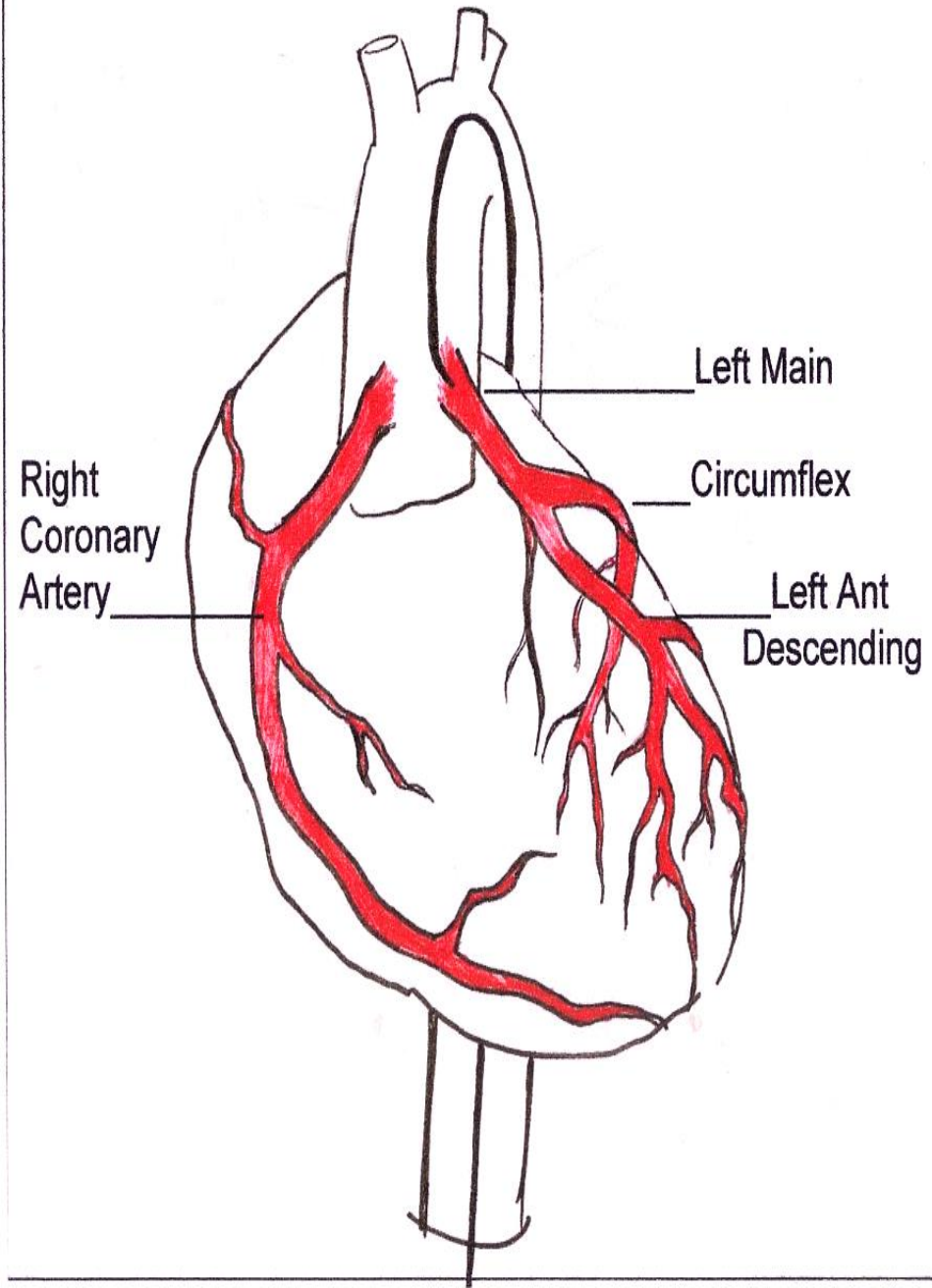
D. Q-Wave Development: 1-12 Hours

E. ST-Elevation with T-wave inversion: 2-5 days

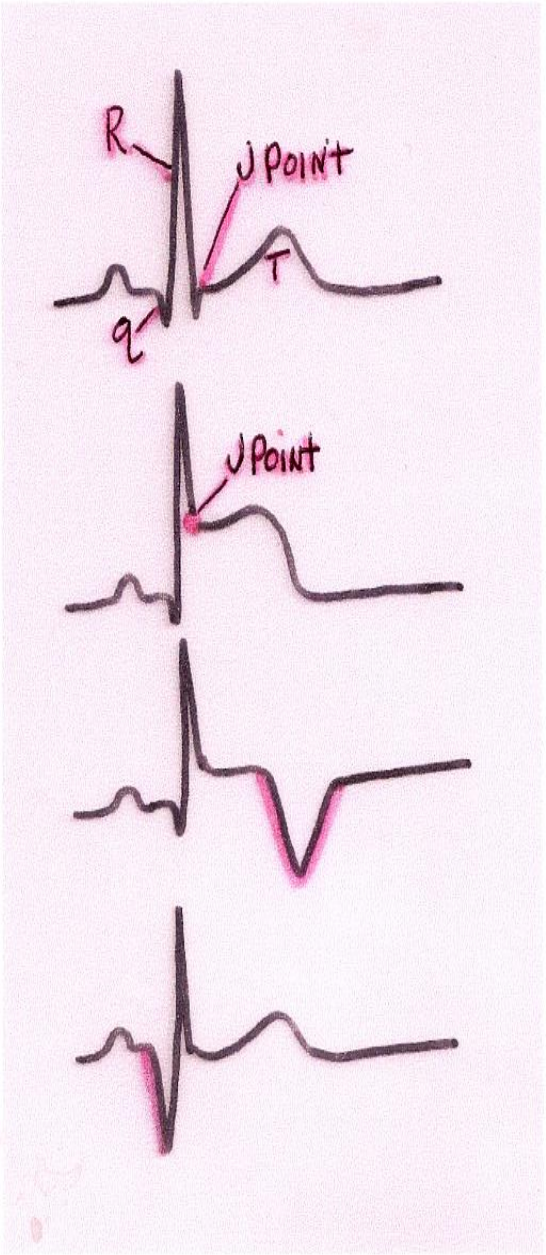
F. T-Wave recovery: weeks to months

Evolution of Acute MI

Coronary Arteries



ECG Signs of M.I.



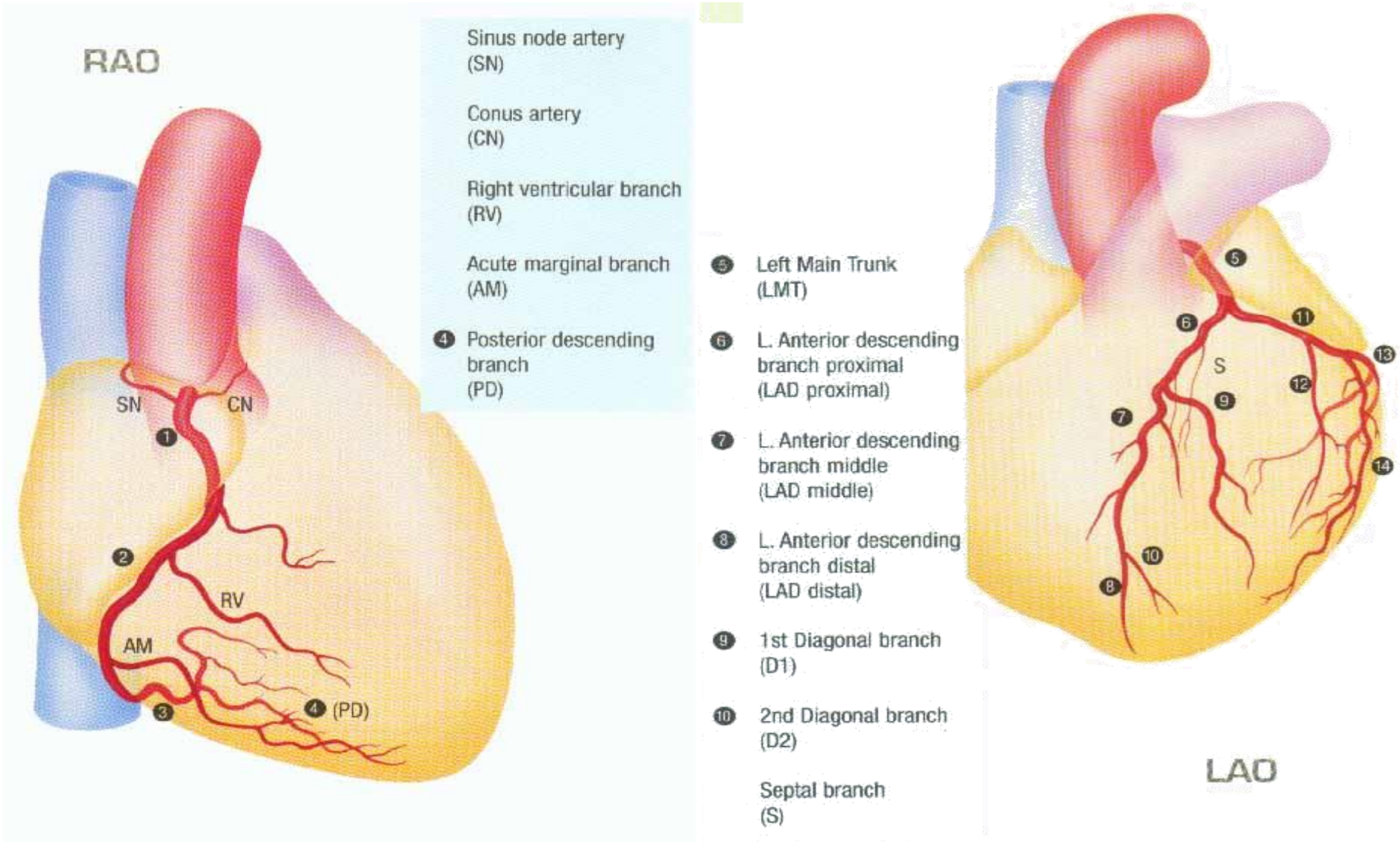
Normal

ST Segment Elevation
Acute Injury

T Wave Inversion
Ischemia

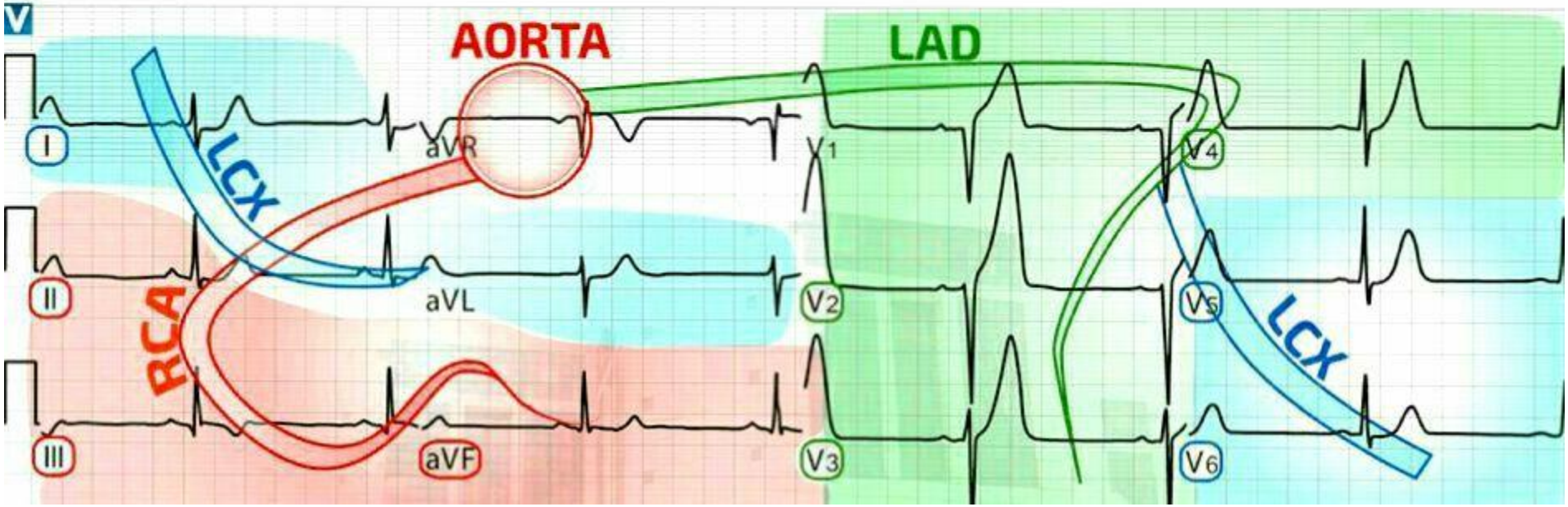
Pathological Q Wave
Necrosis

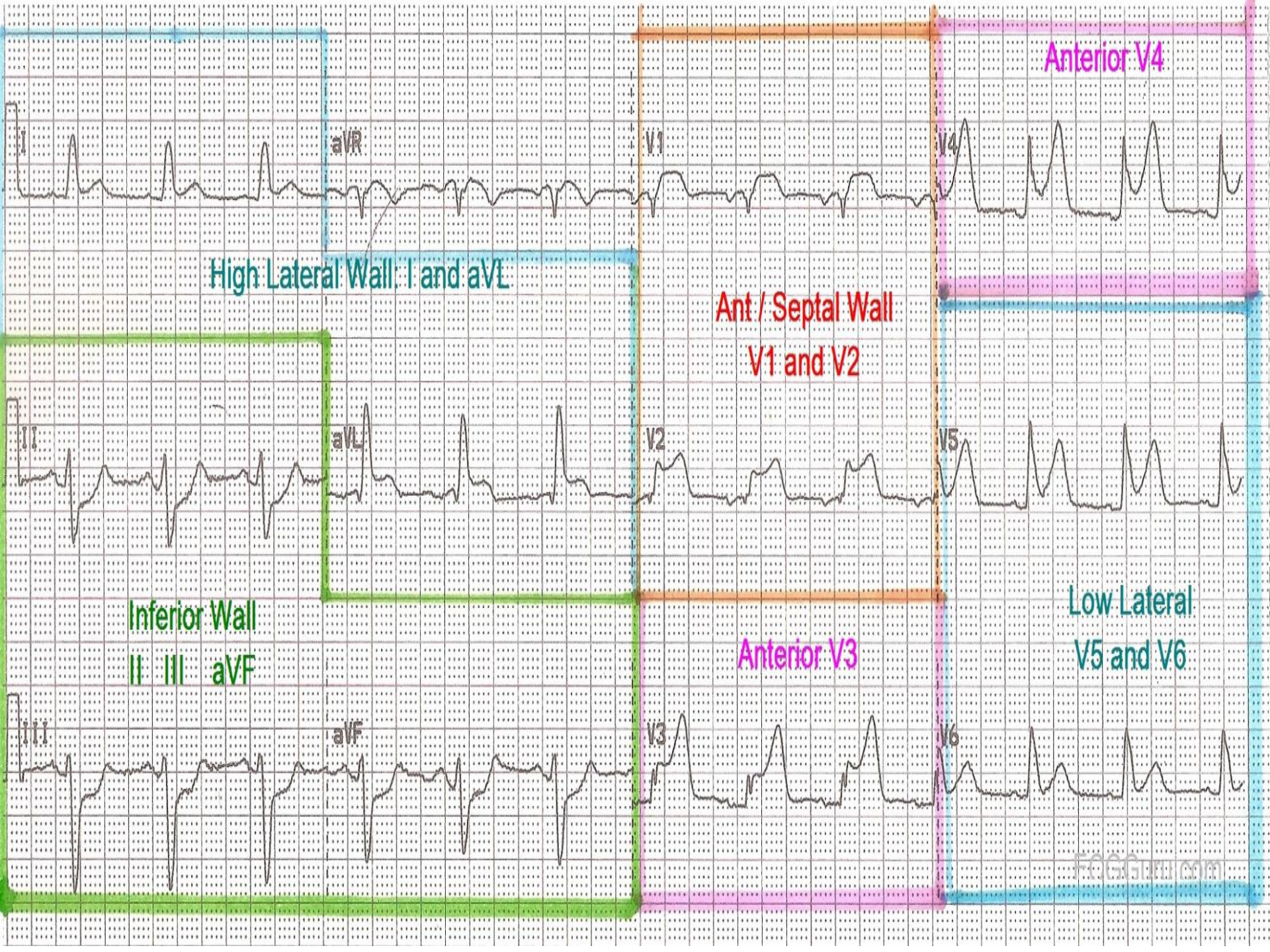
Blood Supply of Heart

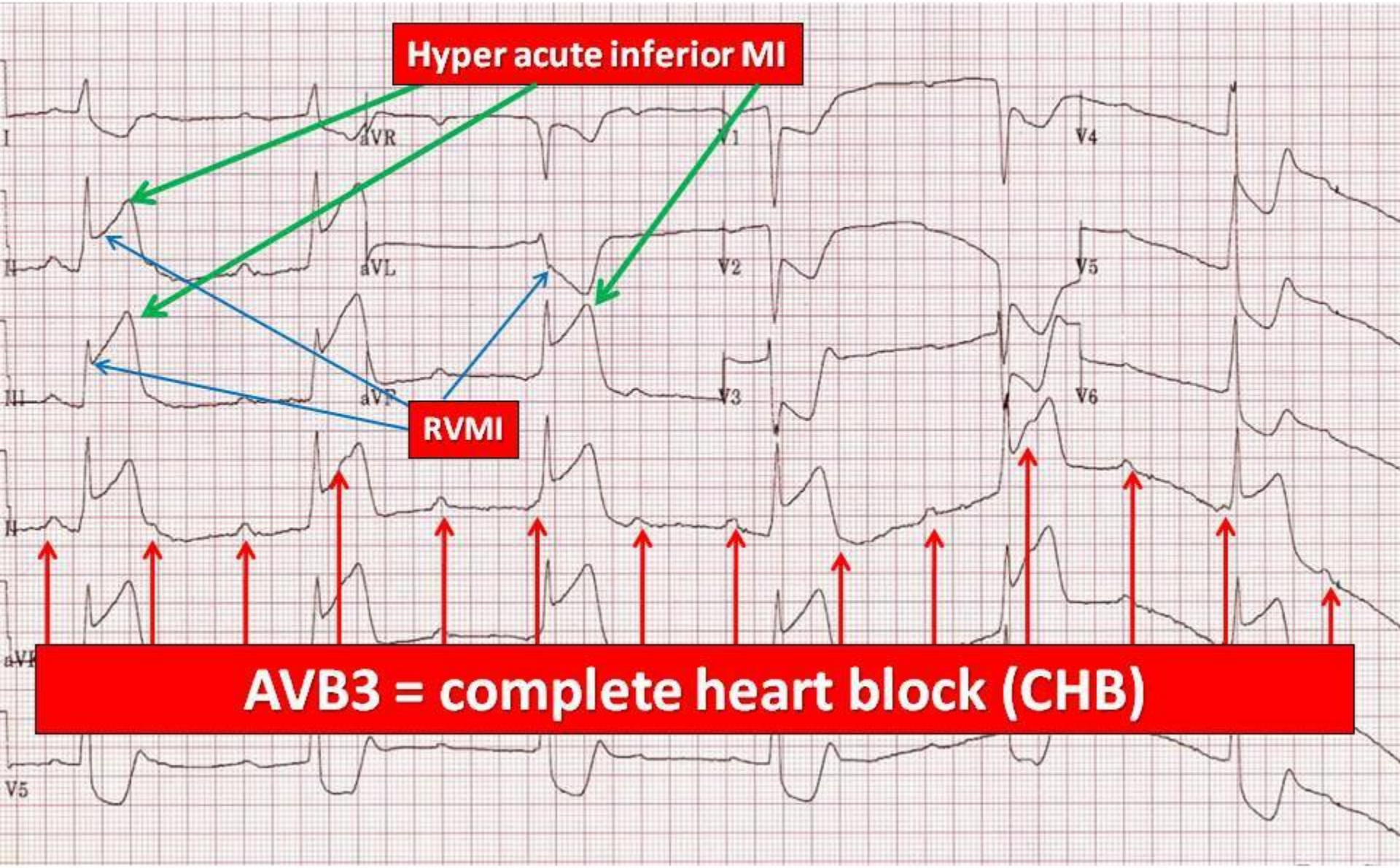


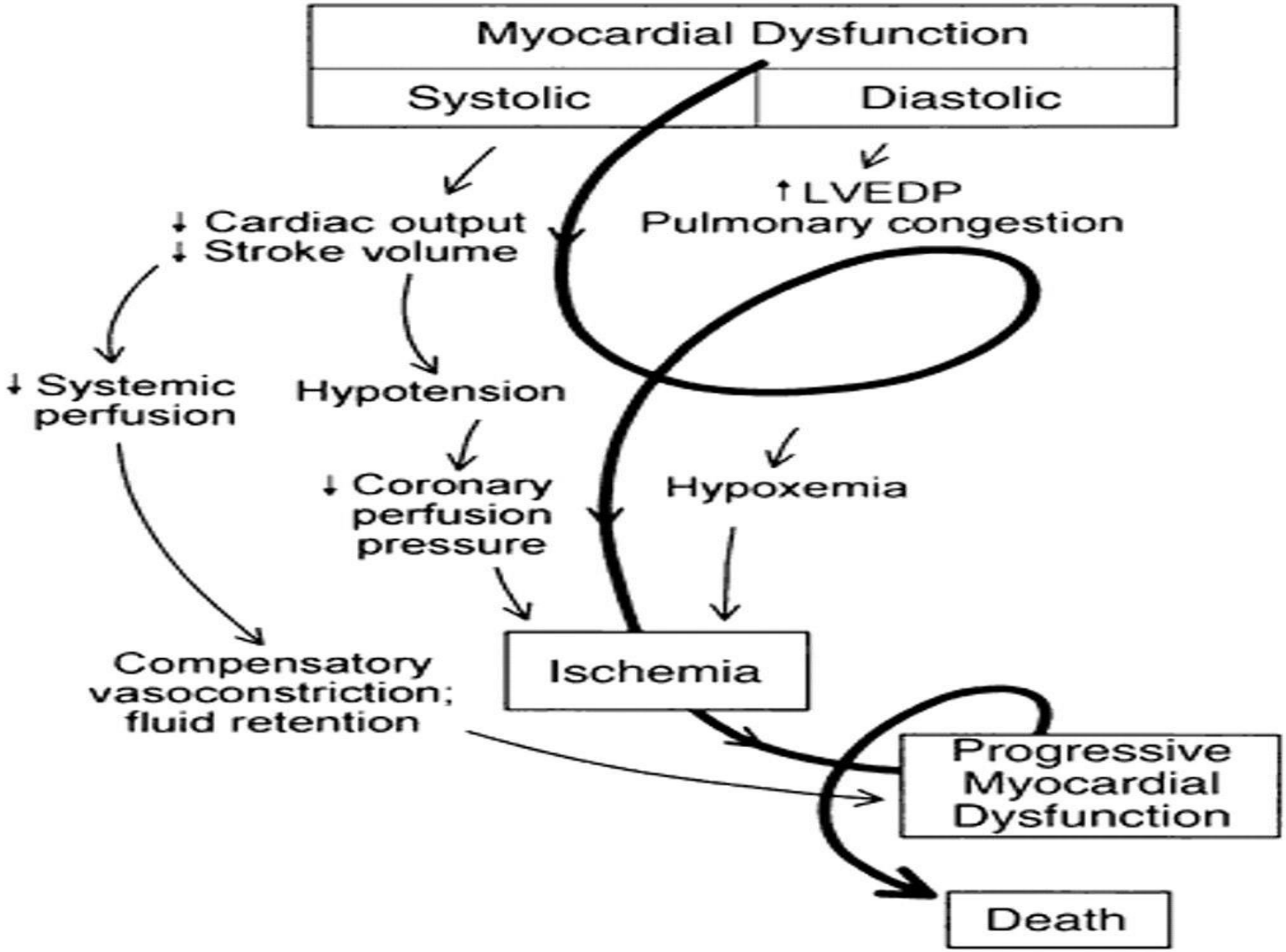
AREA INVOLVED	LEADS ADJACENT	ARTERY INVOLVED
Septal	V1-V2	LAD
Anterior	V2, V3 and V4	LAD
Anteroseptal	V1-V4	LAD
Lateral	I, aVL and V5-V6	LCX
Anterolateral	V2-V6, I and aVL	LCA (LAD + LCX)
Inferior	II, III and aVF	RCA
Posterior	Reciprocal changes V1-V3	RCA

Nursing Education Group
Tehran Heart Center




I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
High Lateral Wall I and aVL
Ant / Septal Wall
V1 and V2
Anterior V4
Inferior Wall
II III aVF
Anterior V3
Low Lateral
V5 and V6
FOAMed.com

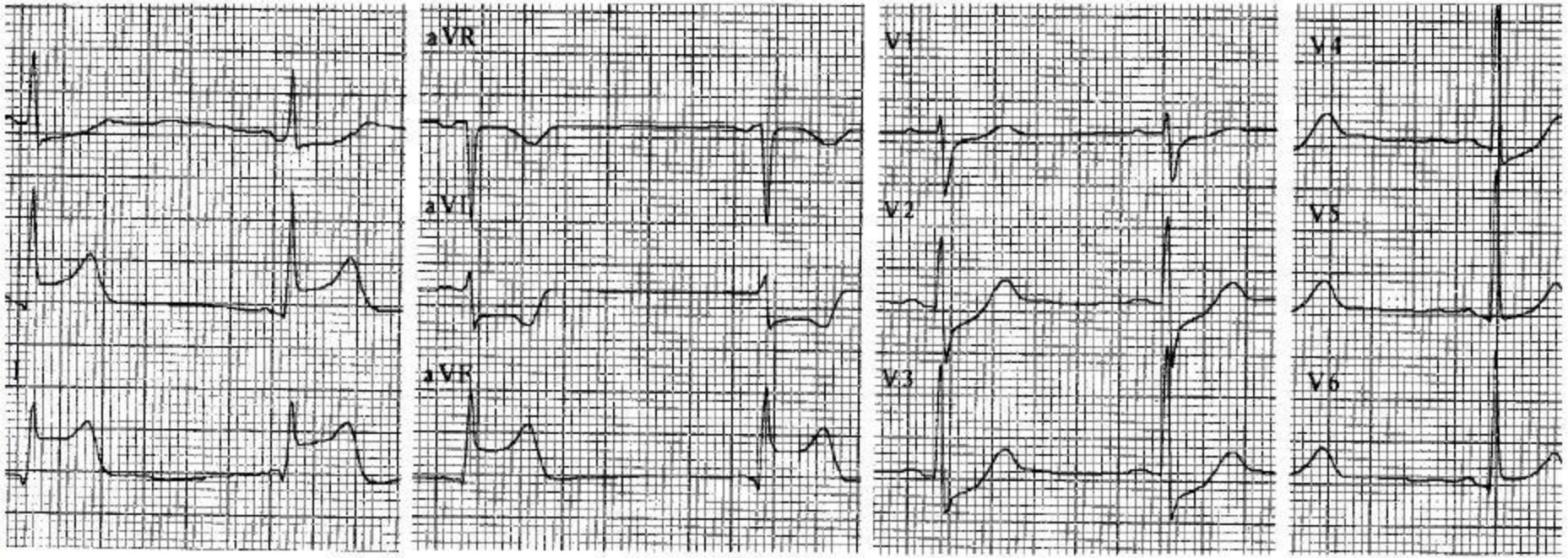

Hyper acute inferior MI
RVMI
AVB3 = complete heart block (CHB)
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
aVF
V5


Myocardial Dysfunction
Systolic
Diastolic
↓ Cardiac output
↓ Stroke volume
↑ LVEDP
Pulmonary congestion
↓ Systemic perfusion
Hypotension
↓ Coronary perfusion pressure
Hypoxemia
Compensatory vasoconstriction; fluid retention
Ischemia
Progressive Myocardial Dysfunction
Death

ST ↑in ant. &inf. leads

Leads	*RCA (proximal RCA)*	*LAD*
V_1 to V_{3-4}	Usually ST↑ ($V_1 > V_{3-4}$)*	Usually ST↑ ($V_{3-4} > V_1$)
Inferior leads	Usually ST↑ greater than that in precordial leads	ST↑ usually much lesser than that in precordial leads
I and aVL	ST depresssion (usually the sum ≥ 5 mm)	Usually not ST depression especially in I

ECG: First Priority



Then make a destination decision…

…STEMI = PCI Center

علائم بالینی انفارکتوس بطن راست

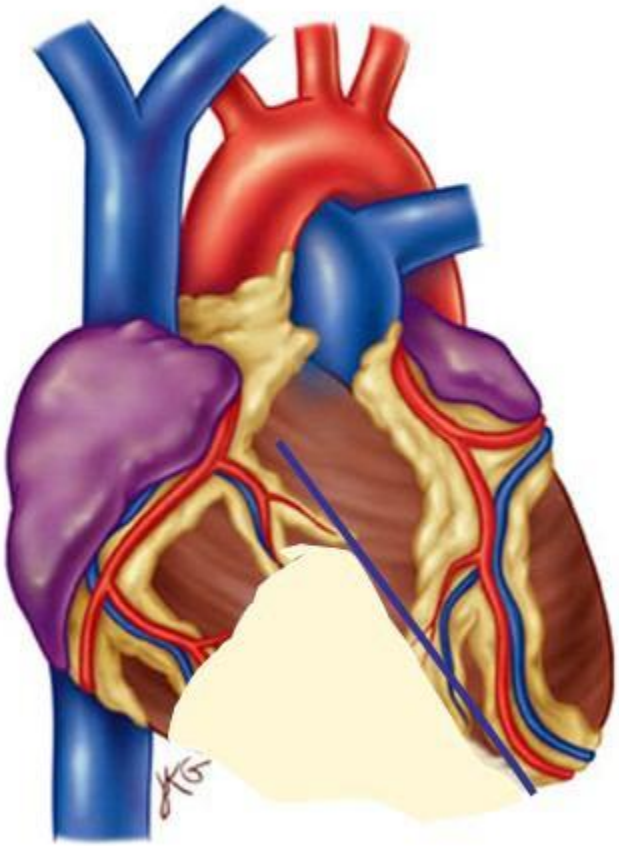
این علائم که پرستار باید دقیقا مراقب بروز آنها در بیمار باشد عبارتند از :

- اتساع ورید ژیگولار : در هنگام دم ، فشار ورید ژیگولار افزایش می یابد (علامت کاسمال) و ممکن است نبض پارادوکس ایجاد می شود .
- هپاتومگالی و ادم محیطی
- طولانی شدن صدای S2 در سمع و وجود گالوپ S3 و S4 بطن راست در ناحیه LSB به دلیل دیلاتاسیون و کاهش کمپلیانس بطن راست .
- سمع سوفل سیستولیک ناشی از نارسایی تریکوسپید در LSB
- هایپوتانسیون به دلیل کاهش برونده قلب و بروز شوک کاردیوژنیک
- وجود پوست سرد و مرطوب همراه با سیانوز
- اولیگوری
- تغییر در سطح هوشیاری

RV infarction

- STE >1mm V3R and V4R
- STE V1 > V2
- High degree AV block

Clinical Triad of RVI



- Hypotension
- Jugular vein distention
- Dry lung sounds

اقدامات درمانی اورژانس هنگام بروز انفارکتوس بطن راست

هدف از این اقدامات از بین بردن علائم نارسایی پمپ خون توسط بطن راست است این اقدامات عبارتند از :

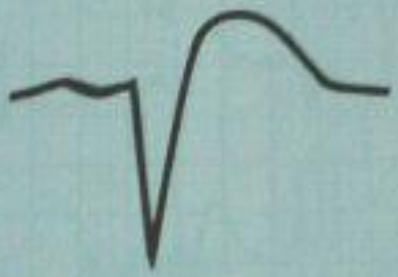
- تجویز سرم نرمال سالین به میزان ۲۰۰ml/h به منظور فرستادن حجم خون کافی به طرف قلب چپ و دستیابی به حداکثر پیش بار .
- مانیتورینگ فشار وج مویرگهای ریوی (PCWP) جهت کنترل حجم مایع انفوزیون شده و پیشگیری از صدمه به دهلیز چپ . بدین منظور باید فشار وج کاپیلرهای ریوی بین ۱۸ – ۱۵ میلی متر جیوه حفظ شود و هرگز از ۲۰ میلی متر جیوه تجاوز نکند .
- تجویز دارو های اینوتروپ مثبت نظیر دوپامین و دوبوتامین . **دوپامین** با دوز ۲-۵ µg/kg/min موجب دیلاتاسیون عروق کلیه و ایجاد دیورز می شود با دوز ۵-۱۰ µg/kg/min گیرنده های دوپامینرژیک را تحریک کرده ، قدرت انقباضی قلب را تقویت می کند و با دوز بالاتر از ۱۰ µg/kg/min موجب انقباض عروقی و تاکی کاردی می شود . در این میان به دلیل آنکه **دوبوتامین** با دوز ۲,۵-۱۰ µg/kg/ min بدون ایجاد انقباض عروقی شدید و تاکی کاردی ، همان اثر اینوتروپ را روی قلب دارد ، داروی انتخابی اول به حساب می آید .
- در صورت بروز هایپوتانسیون ، بلوک های قلبی و کاهش برون ده قلب ، می توان از پیس میکر موقت استفاده کرد .

Oh Boy!

- ST elevation in the septal, anterior and lateral leads
- V1 - V6, I and aVL
- Acute anterior-lateral myocardial infarction

Value of ST – T changes in V4R in acute infero posterior MI

ST↗ ≥ 1 mm
POS T-WAVE

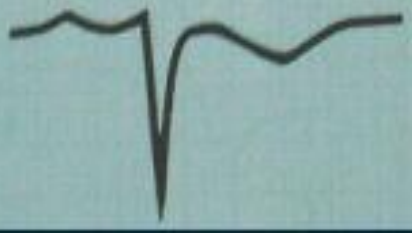


PROXIMAL OCCLUSION RCA
(RVMI)

NO ST↗:
POS T-WAVE

DISTAL OCCLUSION RCA

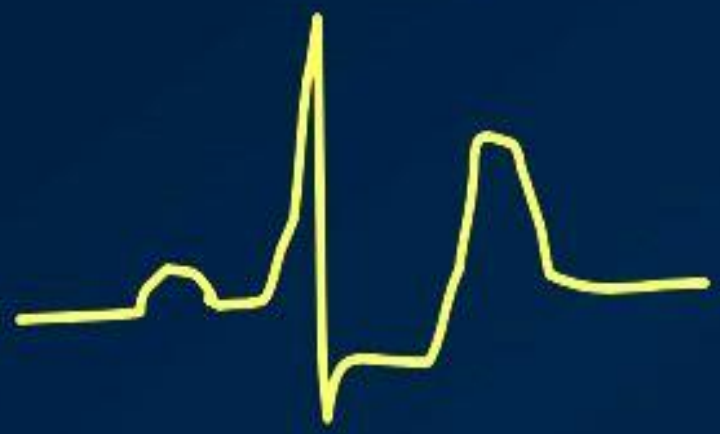
NEG T-WAVE



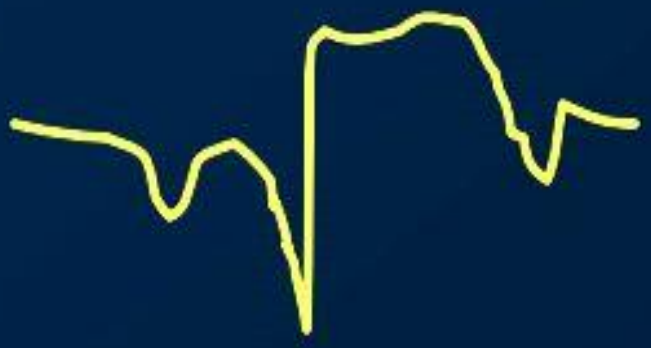
OCCLUSION CX

True PWMI

- ST depression in V1, R/S >1, and upright T wave

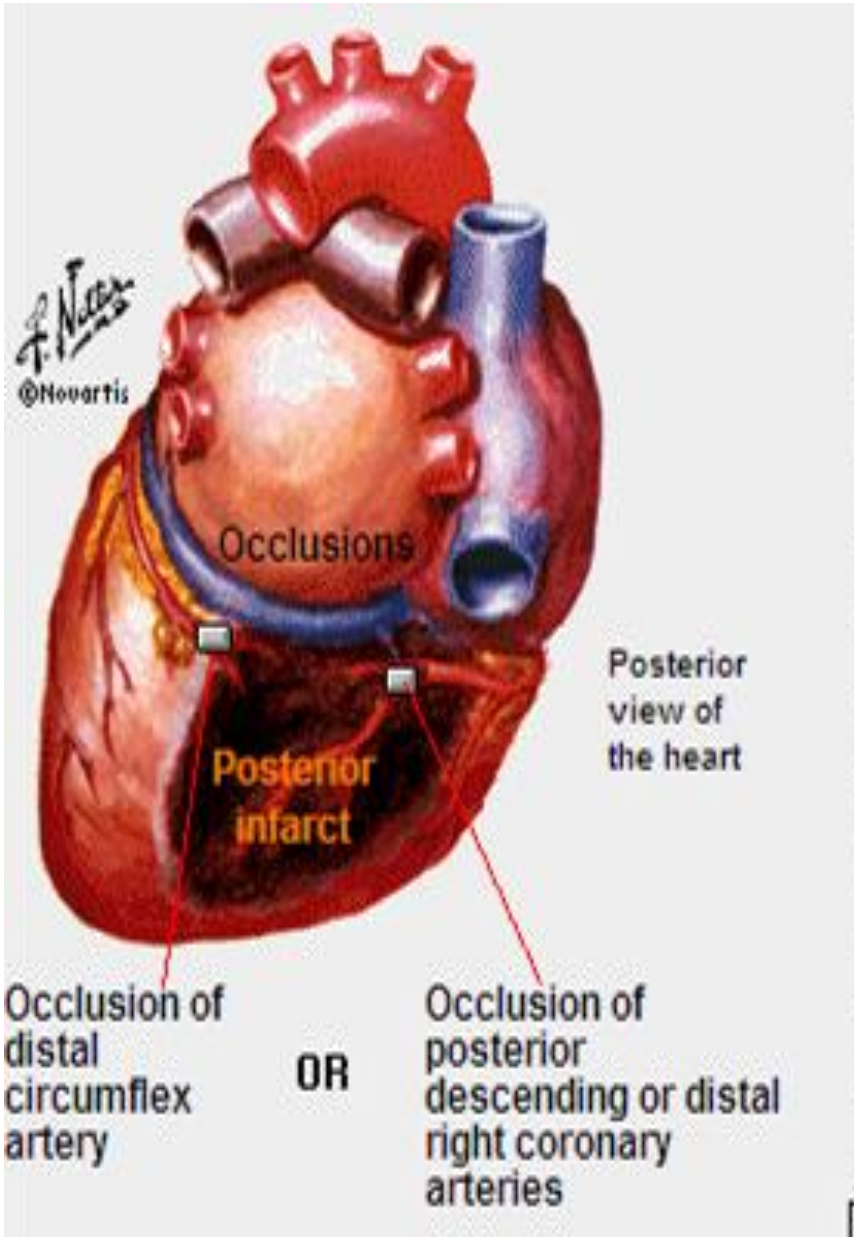


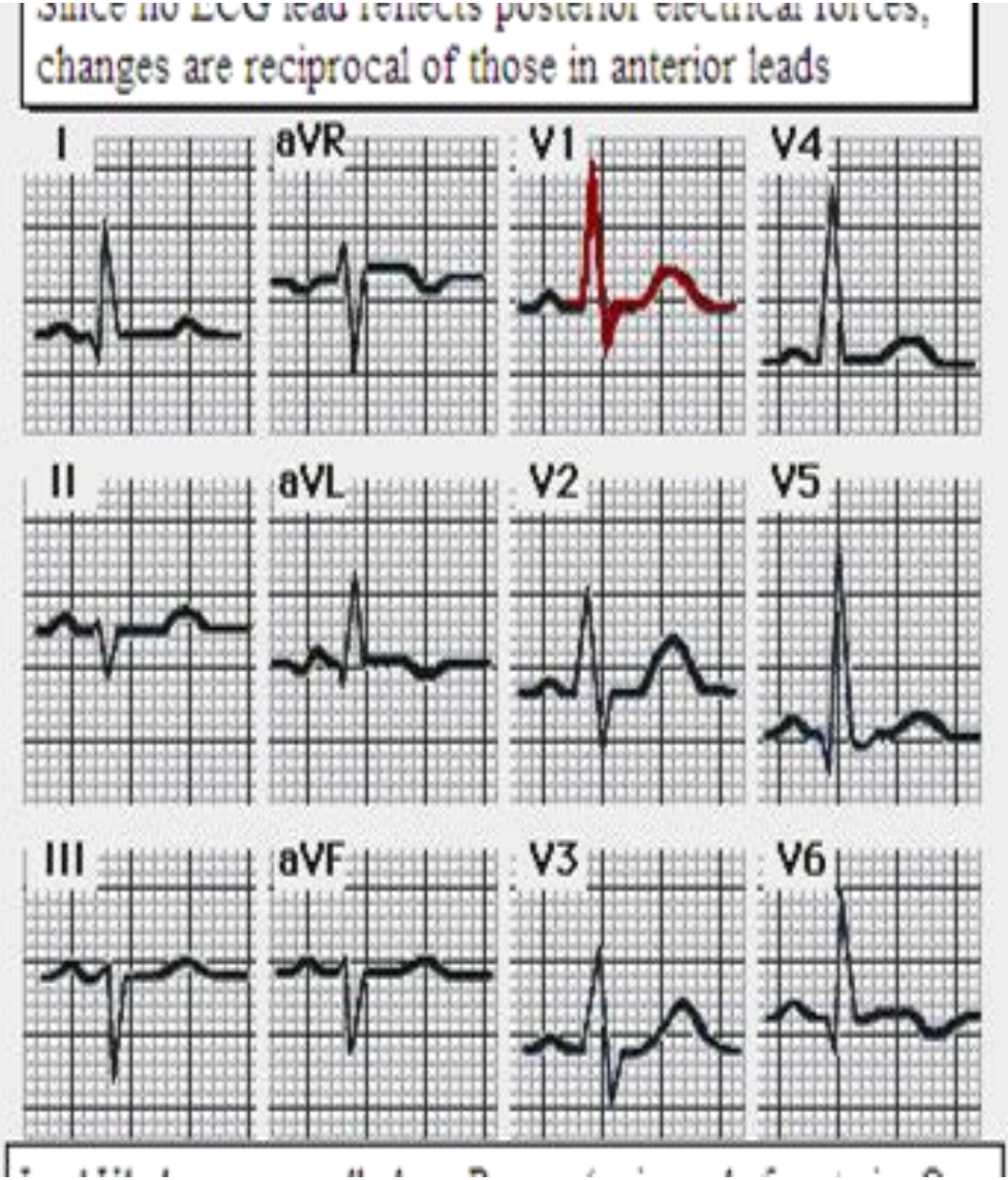
V1



V9

Acute True Posterior MI





Acute True Posterior MI

- Due to occlusion of the distal Left circumflex artery or posterior descending or distal right coronary artery
- Mirror image changes or reciprocal changes in the anterior precardial leads
- Lead V1 shows unusually tall R wave (it is the mirror image of deep Q)
- V1 R/S > 1, Differential Diagnosis - RVH

Acute True Posterior MI

- V2, V3 show tall R waves, Even V1 shows R
- V2, V3, V1 leads R/s ratio is >> 1
- These R waves are the mirrored MI – Qs
- These leads show deep ST depression
- This ST↓is in fact the mirrored ST↑of MI
- The same leads show sharp T waves
- These are the mirrored T inversions of MI

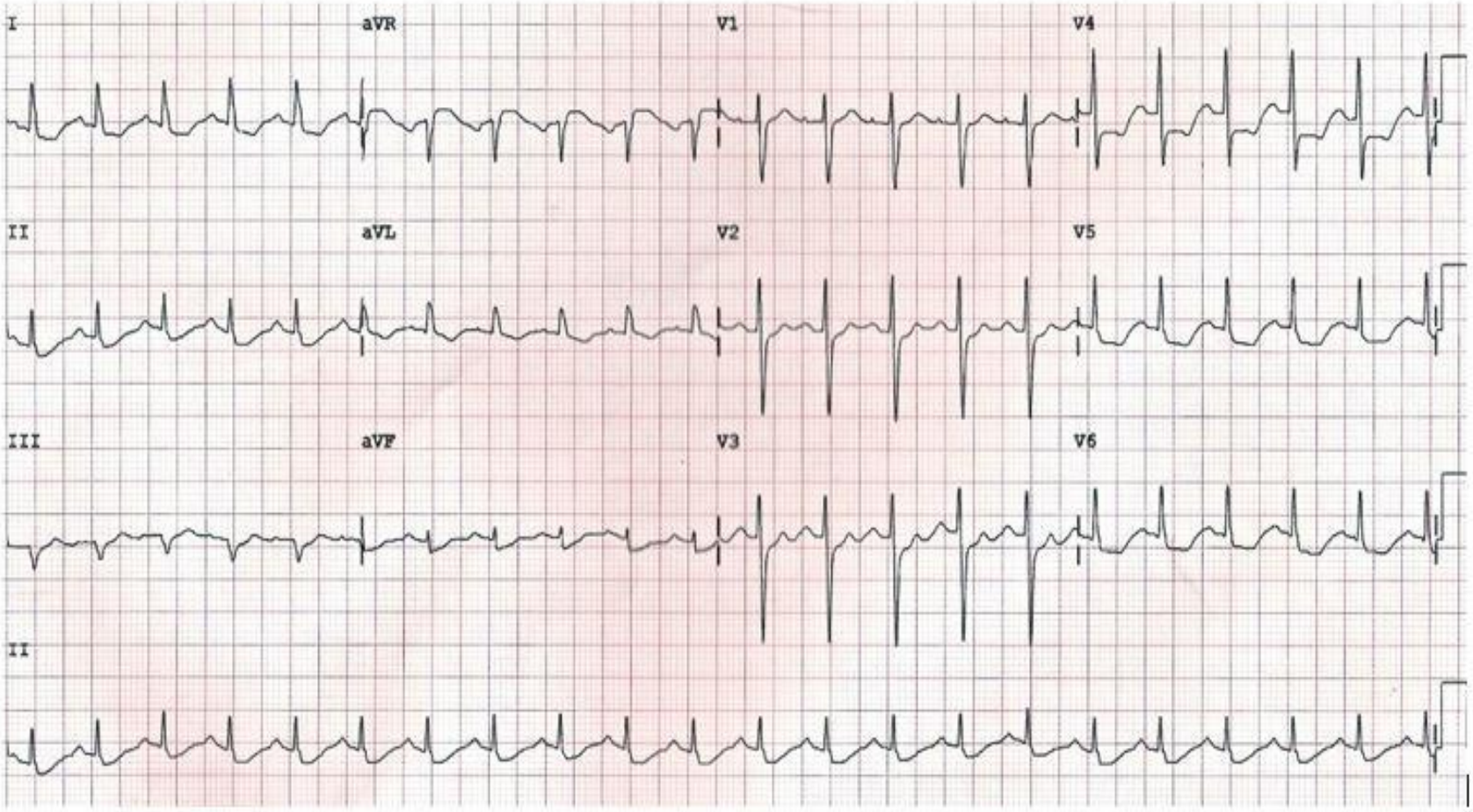
Inferio-Posterior MI

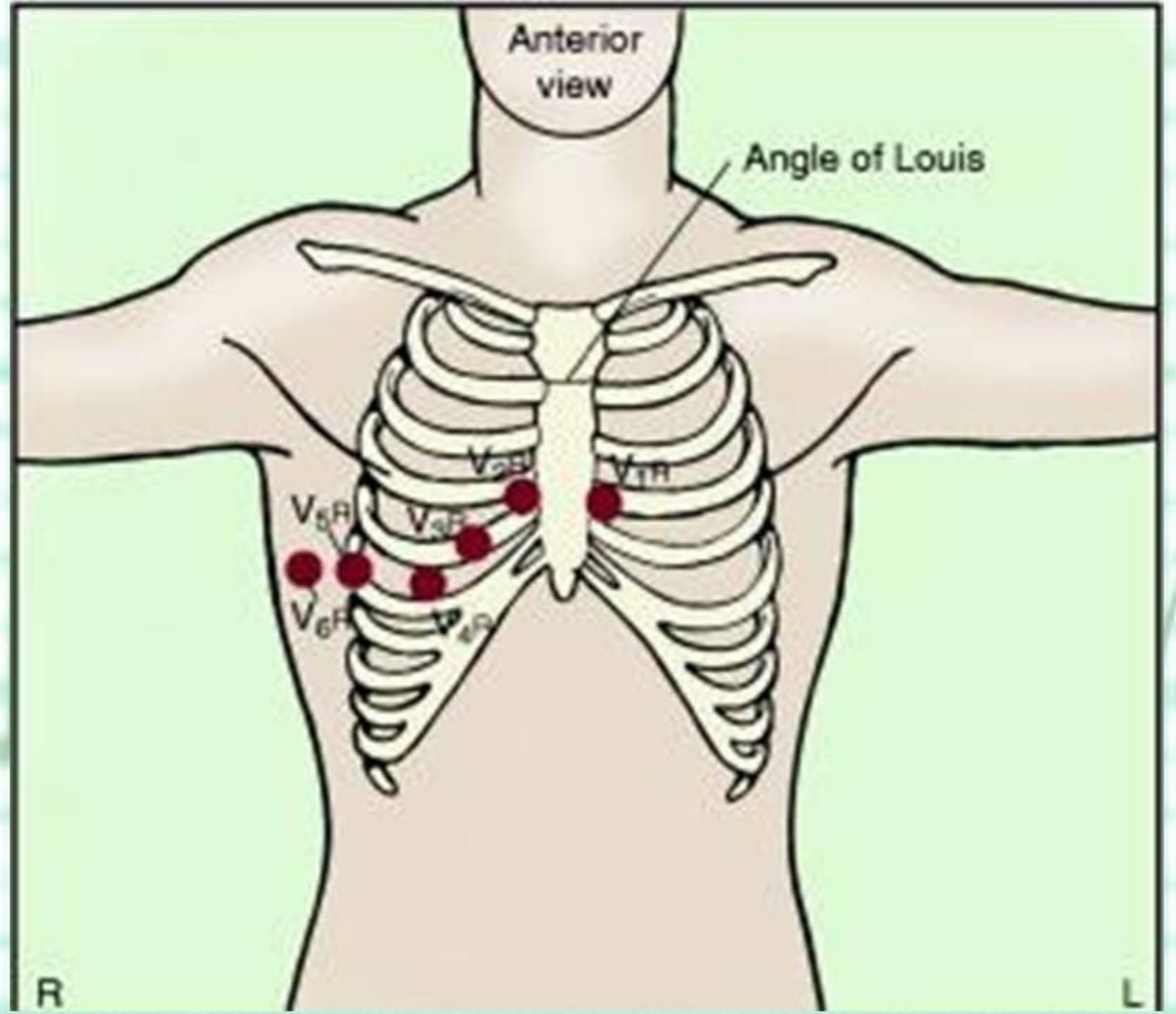
- V1, V2 show tall R waves
- V1, V2 leads R/s ratio is >> 1
- These R waves are the mirrored MI – Qs
- This ST↓ is in fact the mirrored ST ↑ of MI
- The T ↓are the mirrored T inversions of MI
- L2, L3 and aVF show gross ST ↑ - Inferior MI
- V4R, V5R show ST elevations – RV – MI too.

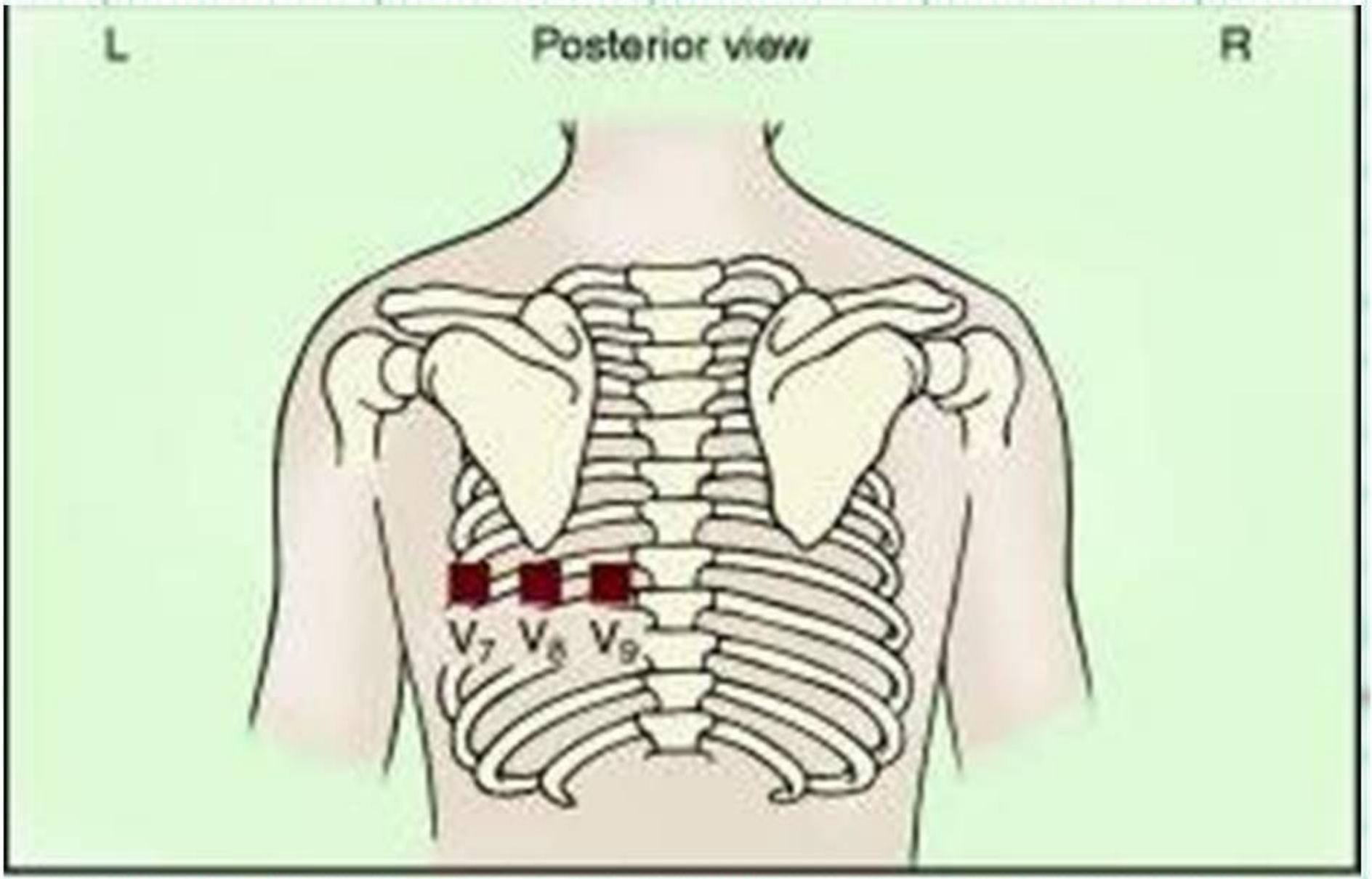
ST Elevation in aVR – LMCA occlusion?

This ECG demonstrates the classical pattern of left main coronary artery (LMCA) occlusion:

- **Widespread horizontal ST depression, most prominent in leads I, II and V4-6**
- **ST elevation in aVR ≥ 1mm**
- **ST elevation in aVR ≥ V1**




Anterior view
Angle of Louis
V_{2R}
V_{1R}
V_{5R}
V_{3R}
V_{6R}
V_{4R}
R
L


L
Posterior view
R
V7 V8 V9

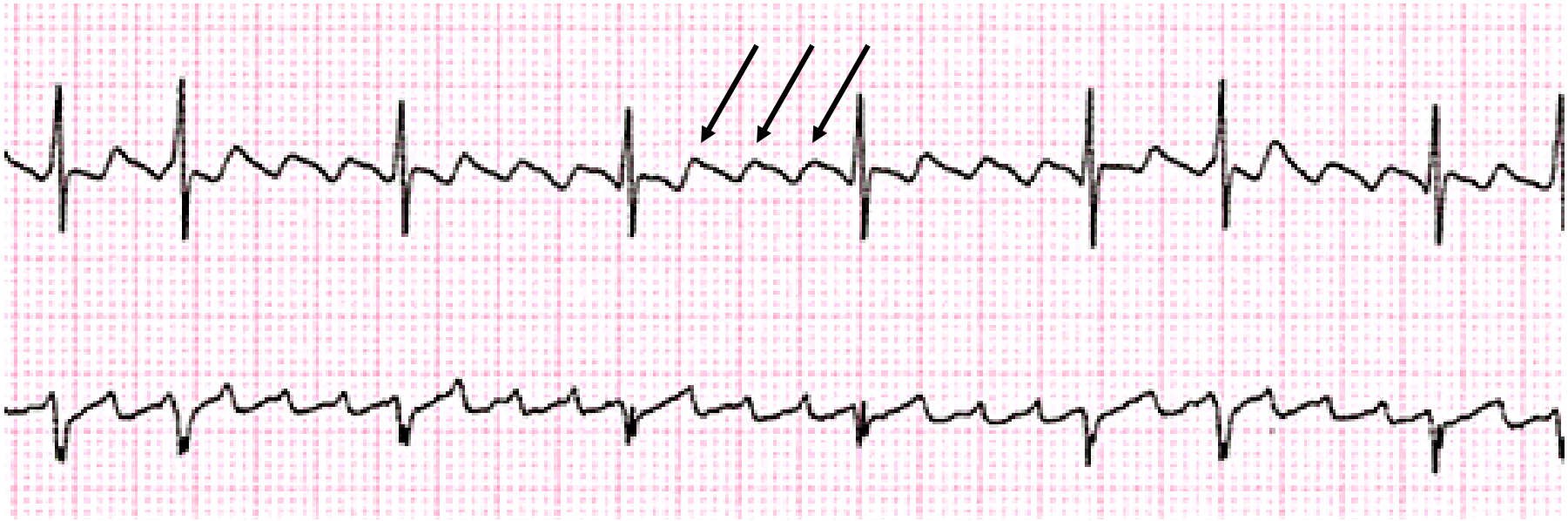
Reciprocal Changes

Inferior: II,III,aVF ⟶ Anterior: V1-V6, aVL, I

Septal: V1, V2 ⟶ Lateral: V5,V6, I, aVL

Early Anterior: V1-V3 ⟶ Posterior: V7-V9

Atrial Flutter

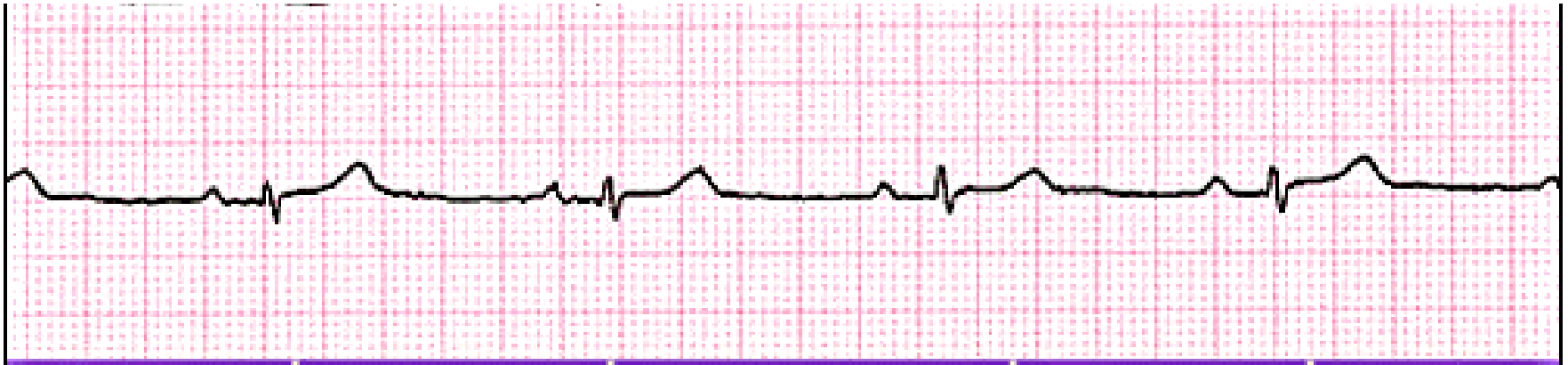


Rhythm	P wave	PR interval	Heart rate QRS in sec
A: 220-430 bpm V: <300 bpm	Regular or variable	Sawtoothed appearance	N/A	<.12

Myxedema

- Note the ECG changes
- Bradycardia – HR of 55 per minute
- Low voltages of all complexes
  - Less than 5 mm Limb leads
  - Less than 8 mm chest leads
- DD of low voltage complexes
- Pericardial effusion, Constrictive pericarditis
- Severe Emphysema
- Pneumothorax or left sided pleural effusion

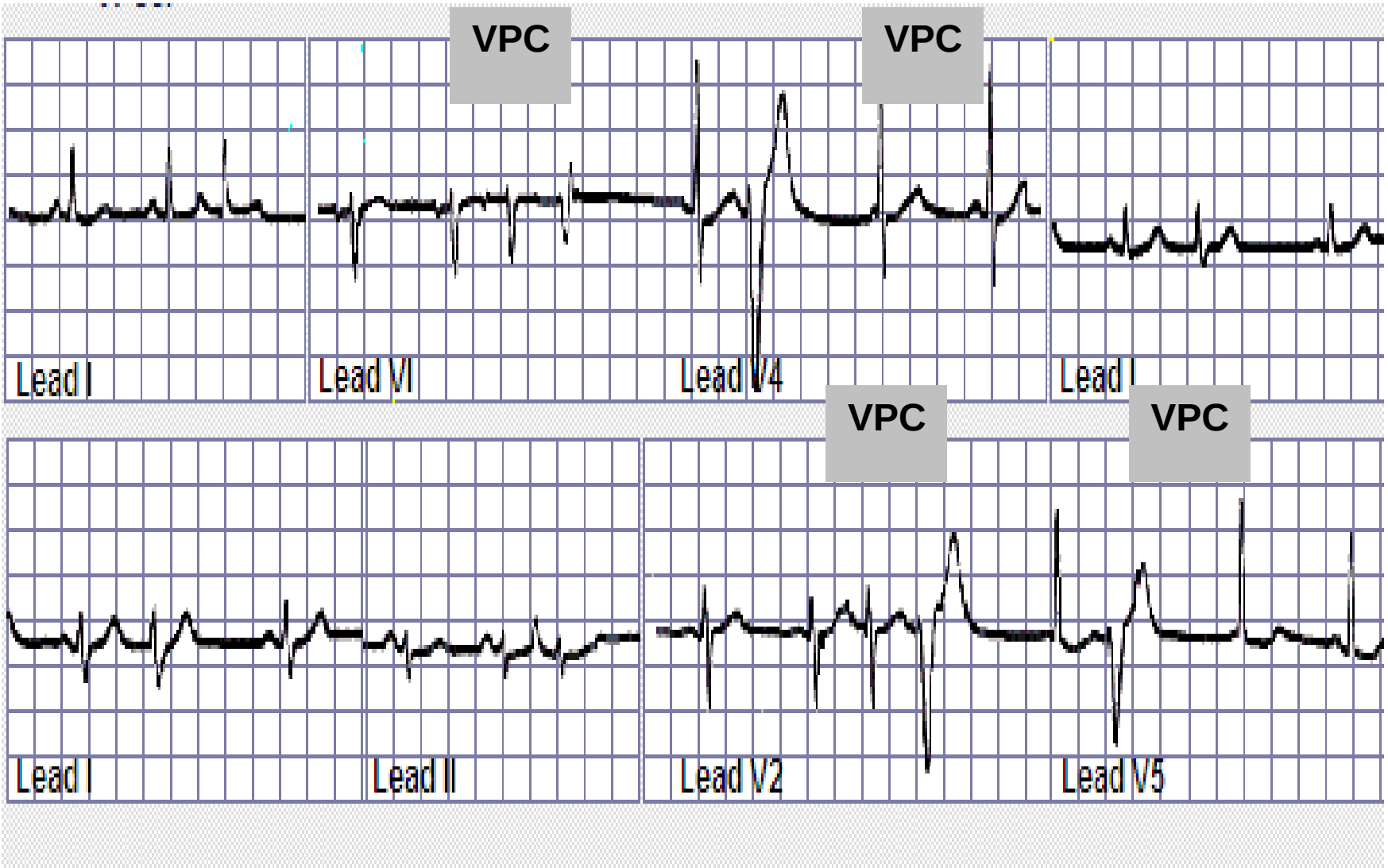
Myxedema



Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
<60 bpm	Regular	Before each QRS, identical	.12 to .20	<.12

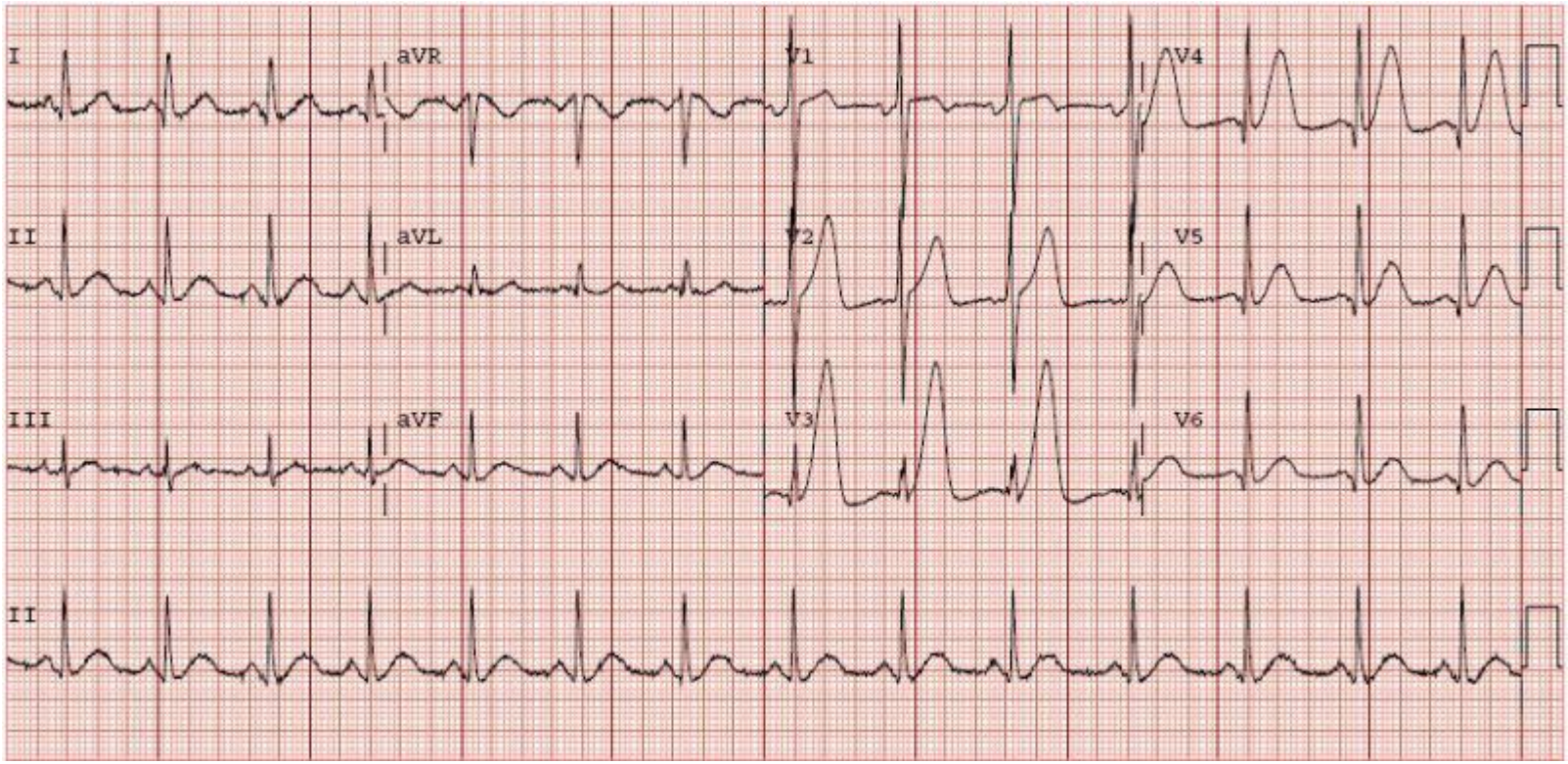
- Note ECG changes of Atrial Flutter
- The heart rate is regular or variable
- Atrial rate is 300 per minute
- All P waves are not conducted to ventricles
- The R-R intervals very depending on the AV conduction ratio
- The QRS is narrow – < 0.12 sec
- The P waves have a ‘saw toothed’ appearance called ‘F’ waves

Ventricular Ectopics

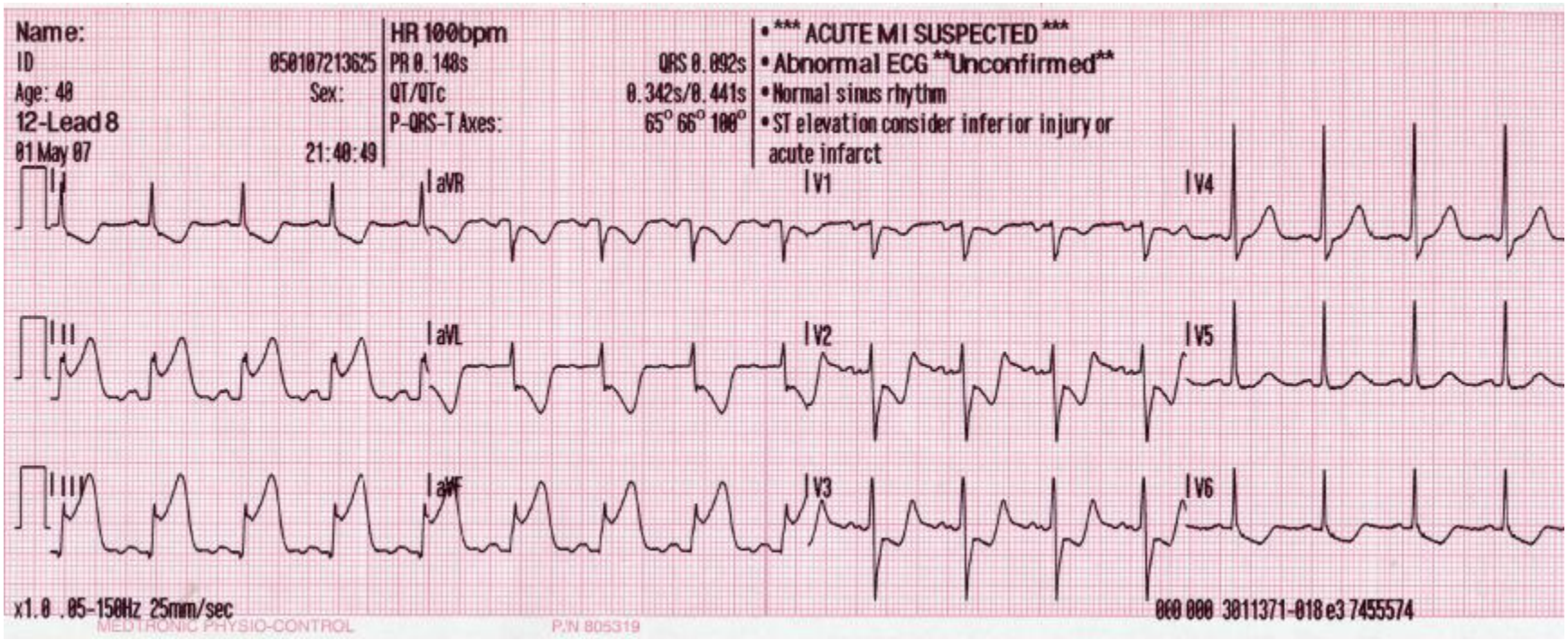


- Note the premature (ectopic) beats marked as VPC (Ventricular Premature Contractions)
- These occurred before the next expected QRS complex (premature)
- Each VPC has no definite P wave preceding the QRS of that beat – So impulse has originated in the ventricles
- The QRS complexes are wide with abnormal duration of > 0.12 and their shapes are bizarre

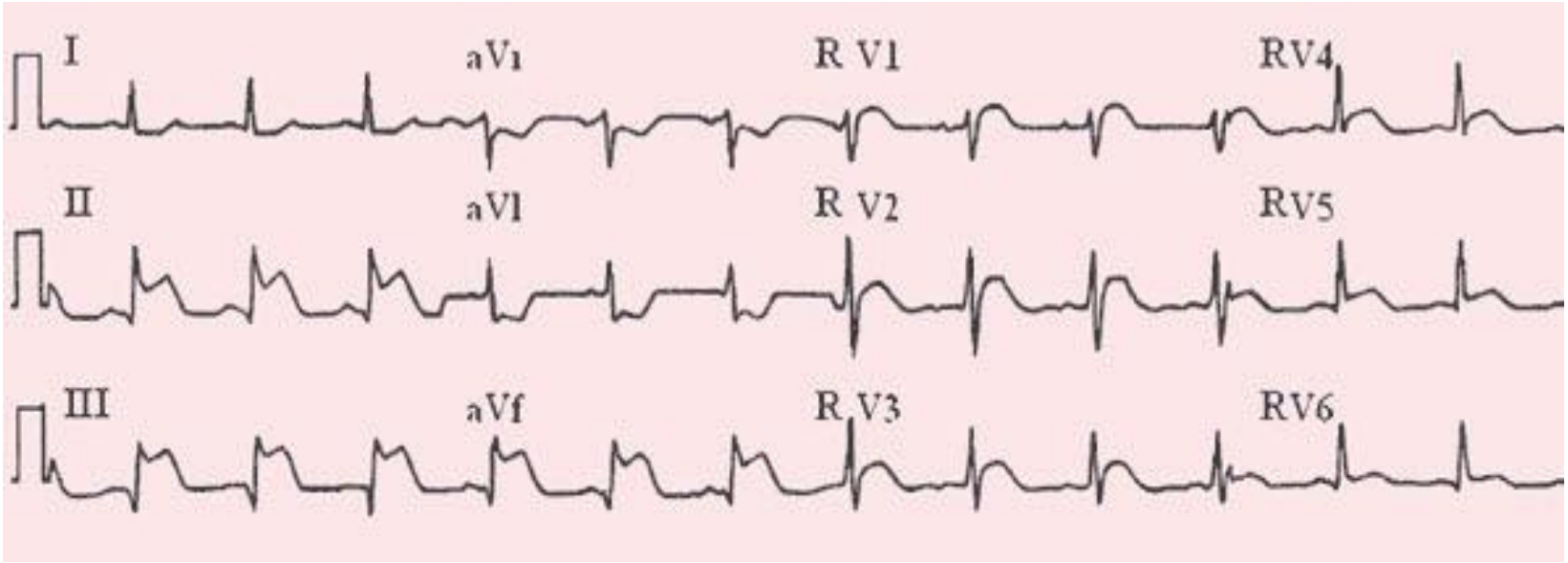
Hyperacute MI



Inferior MI


Name:
ID 050107213625
Age: 48 Sex:
12-Lead 8
01 May 07 21:40:49
HR 100bpm
PR 0.148s QRS 0.092s
QT/QTc 0.342s/0.441s
P-QRS-T Axes: 65° 66° 100°
*** ACUTE MI SUSPECTED ***
Abnormal ECG **Unconfirmed**
Normal sinus rhythm
ST elevation consider inferior injury or acute infarct
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
x1.0 .05-150Hz 25mm/sec
MEDTRONIC PHYSIO-CONTROL
P/N 805319
000 000 3011371-018 e3 7455574

RV MI



with ST segment depression (Fig. 2.19). However, it may rarely be the sole abnormality. Figure 2.32 was recorded in a 50-year-old man with stable angina. It shows isolated U wave inversion in leads V_2 and V_3. Subsequent coronary angiography showed critical stenosis of the proximal left anterior descending artery. In ischaemic heart disease patients showing U wave inversion in the mid- or left praecordial leads, the coronary arteries which are stenosed are nearly always **the left main coronary artery or the left anterior descending artery**.[11]

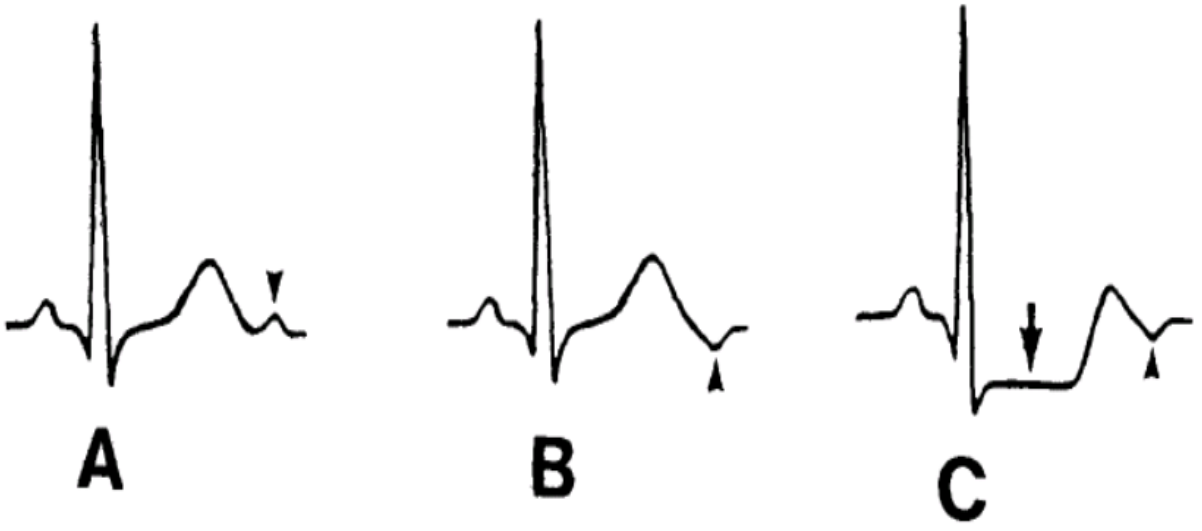
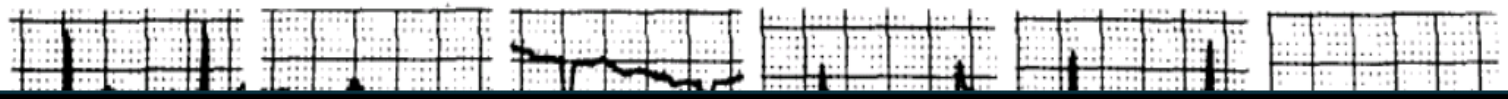


Fig. 2.31 Diagram showing: (A) Normal upright U wave (arrowhead). (B) Isolated U wave inversion (arrowhead). (C) U wave inversion (arrowhead) associated with ST segment depression (arrow).



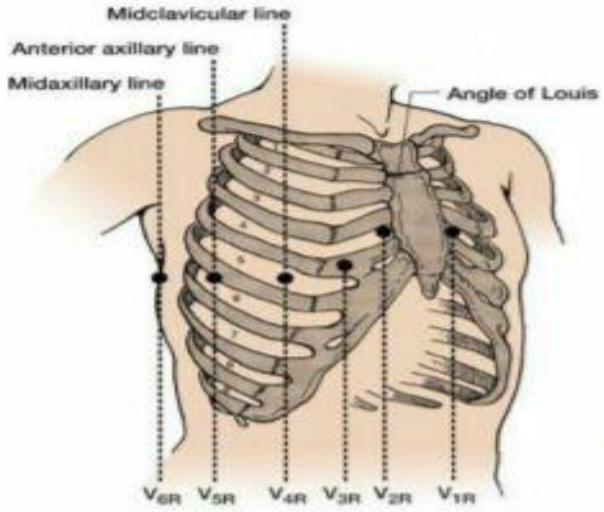
Right-Sided ECG

Recommended Clinical Practice

To detect right ventricular STEMI associated with occlusion of the right coronary artery, obtain a right-sided ECG. [1-3] **[Level A Recommendation]**

When a 15-lead &/or 18-lead ECG machine is not available, manipulation of the leads from a standard 12-lead ECG machine allow additional areas of the heart to be imaged.[4-5]

- Indications of a RV wall infarction may include:[4-7]
  - ST elevation in the inferior leads, II, III, and aVF[4-6]
    - ST elevation that is greatest in lead III is especially significant[5,8-9]
  - ST elevation in V_1 (considered to be the only precordial lead that faces the RV on the standard 12-lead ECG)[4-6,8]
  - Other findings may include: right bundle branch block, second- and third- degree atrioventricular blocks, ST segment elevation in lead V_2 50% greater than the magnitude of ST segment depression in lead aVF[5,8]
  - Hypotension and clear lung fields[6,10]
- Place ECG electrodes (stickers) as follows[4] (Figure 1):



Right-sided ECG Electrode Placement

V_1R: 4th intercostal space, left sternal border

V_2R: 4th intercostal space, right sternal border

V_3R: halfway between V2R and V4R, on a diagonal line

V_4R: 5th intercostal space, right midclavicular line

V_5R: right anterior axillary line, same horizontal line as V4R and V6R

V_6R: right mid-axillary line, same horizontal line as V5R and V6R

Arm and leg electrodes remain unchanged from standard 12-lead ECG

Figure 1 used with permission from Barbara J. Drew, RN, PhD, FAAN, FAHA [Drew, B. J., & Ide, B. (1995). Right ventricular infarction. *Progress in Cardiovascular Nursing, 10*, 46.]

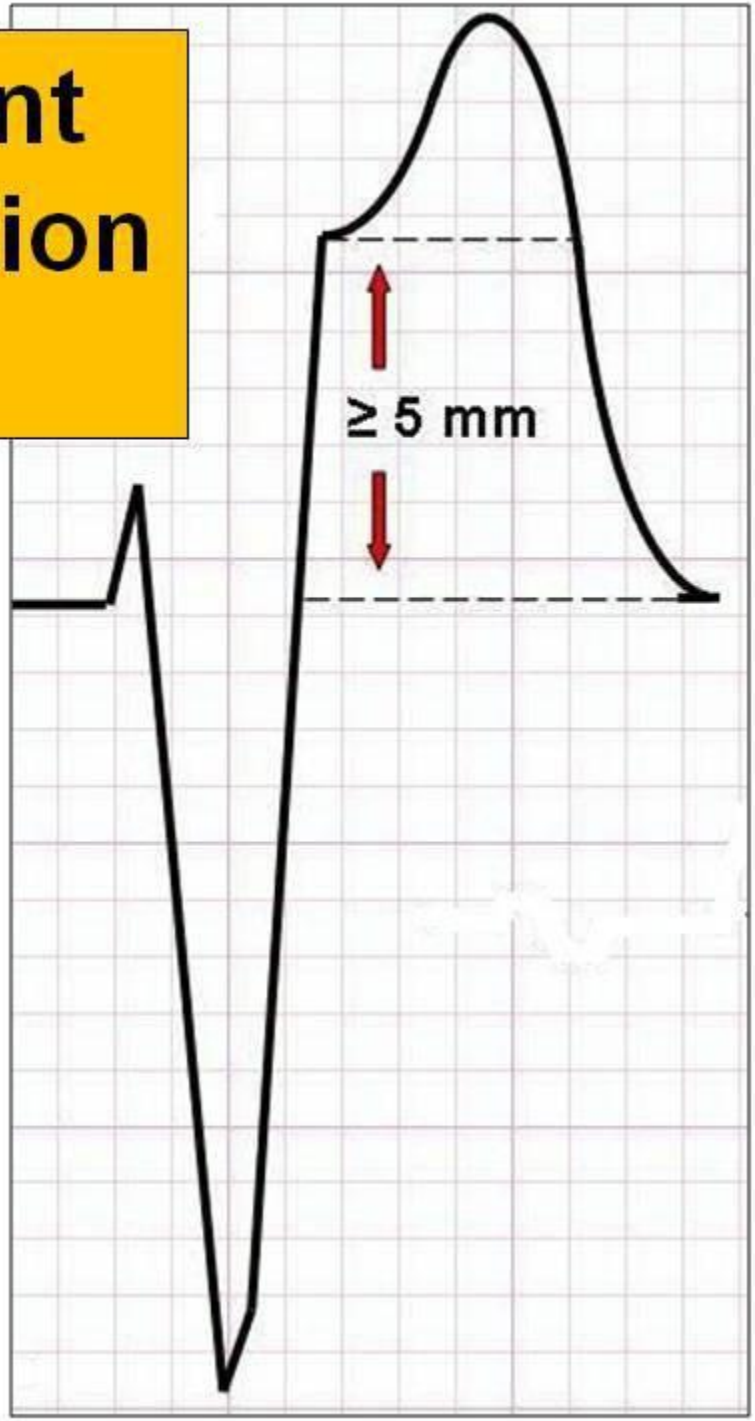
- Place ECG lead cables as follows (using a 12-lead machine):
  - A right-sided ECG is a "mirror reflection" of the standard left sided 12-lead ECG. Begin with lead cable V_1 and attach it to electrode V_1R, continue connecting lead cables to electrodes in sequence until lead cable V_6 is connected to electrode V_6R
  - Arm and leg electrodes and lead cables remain unchanged from the standard 12-lead ECG placement

Sgarbossa Criteria

ST **Elevation** ≥ 1 mm and concordant with QRS complex	**Score 5 points** Odds Ratio (OR) 25.2
ST Depression ≥ 1 mm in V1, V2, V3	**Score 3 points** OR 6.0
ST **Elevation** ≥ 5 mm and discordant with QRS complex	**Score 2 points** OR 4.3

Odds Ratio: a measure of the degree of association; for example, the odds of exposure among the cases compared with the odds of exposure among the controls (www.cebpas.it/ebm/tools/glossary.htm)

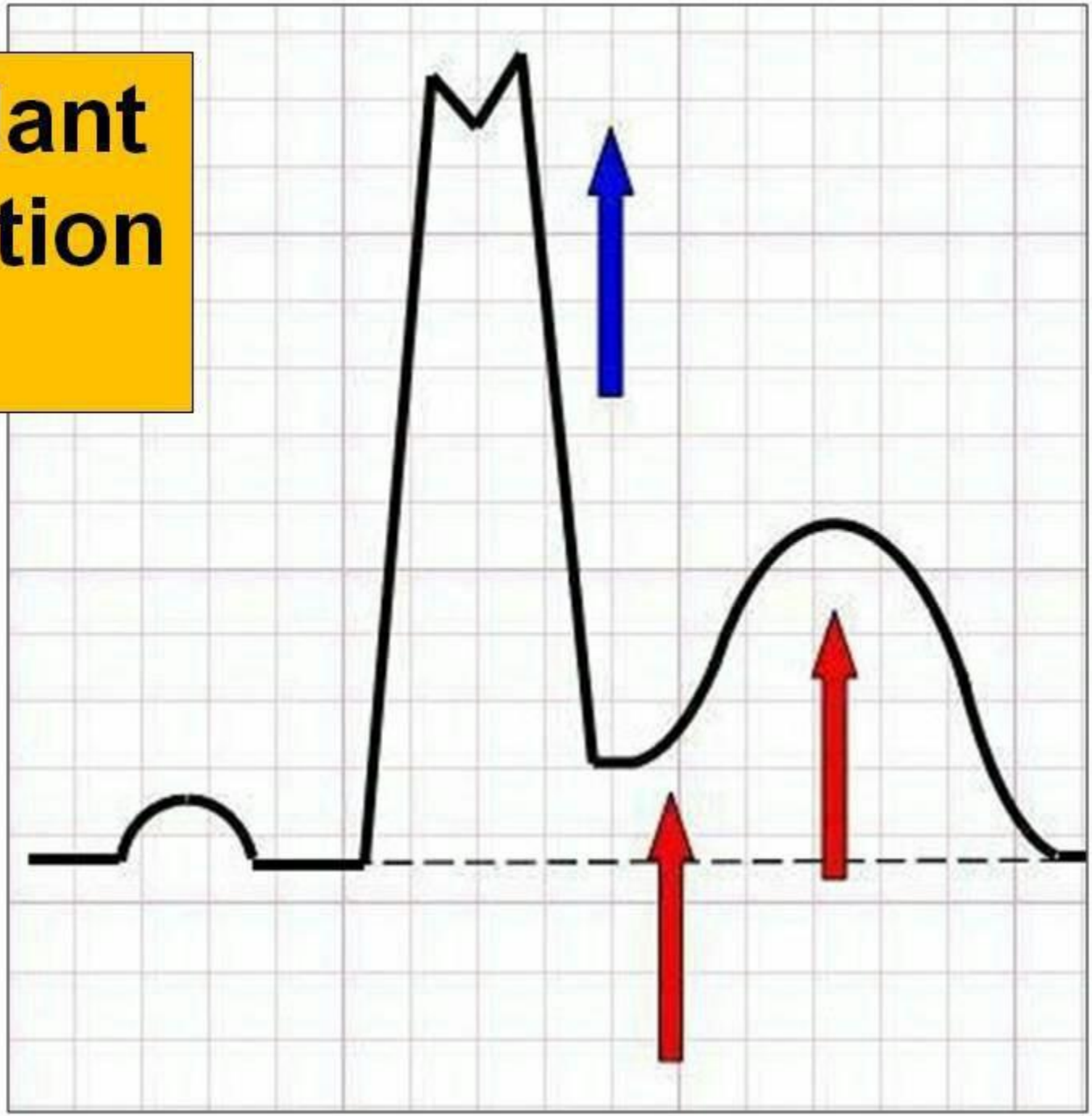
Discordant ST elevation v1,v2,v3

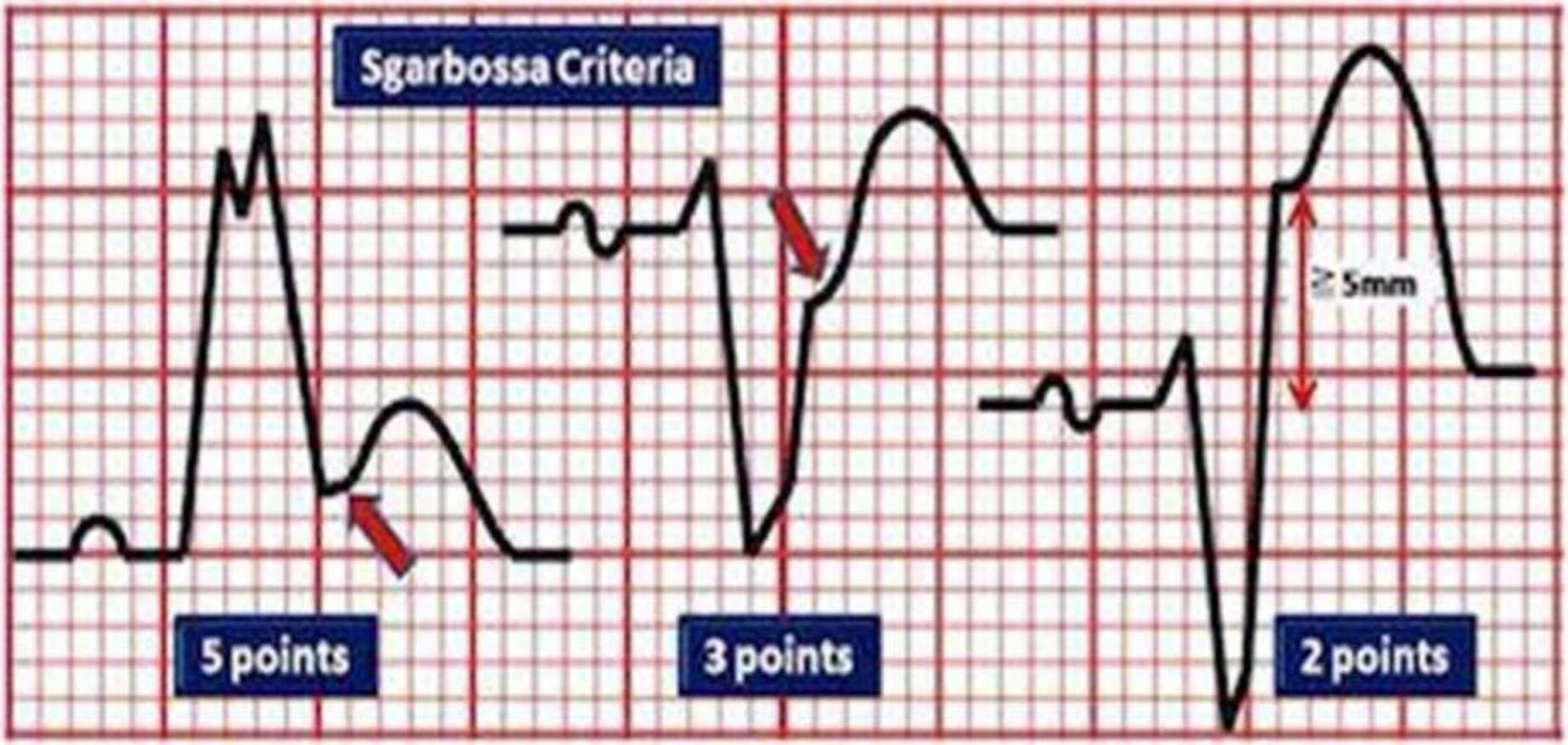


2 Point

Concordant ST elevation v5,v6

5 Point





Sgarbossa ECG Criteria for LBBB	
Concordant STE ≥1mm	5 points
STD ≥1mm in V1 – V3	3 points
Discordant STE ≥5mm	2 points

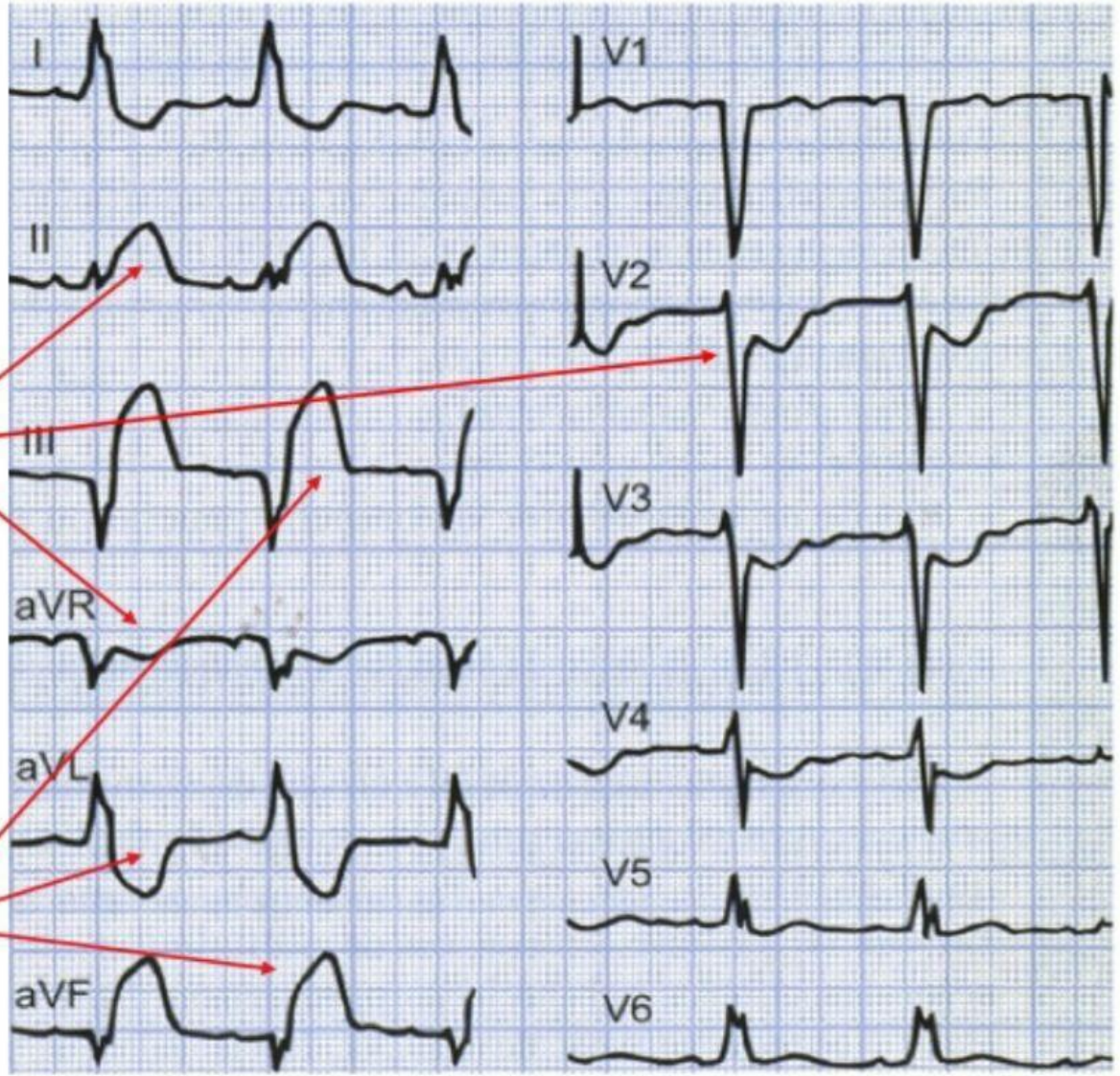
Critères de Sgarbossa

(infarctus et BBG)

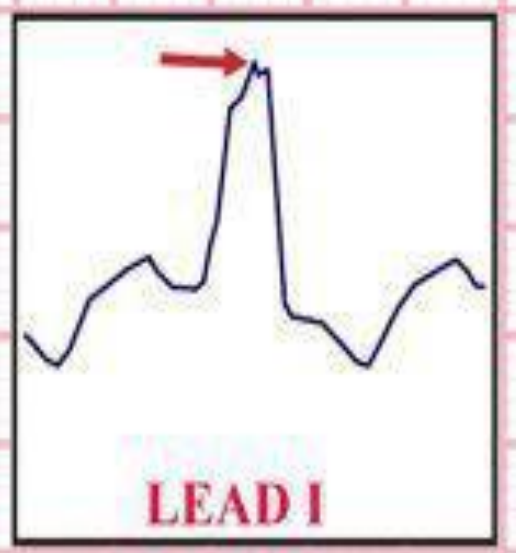
Concordance du ST

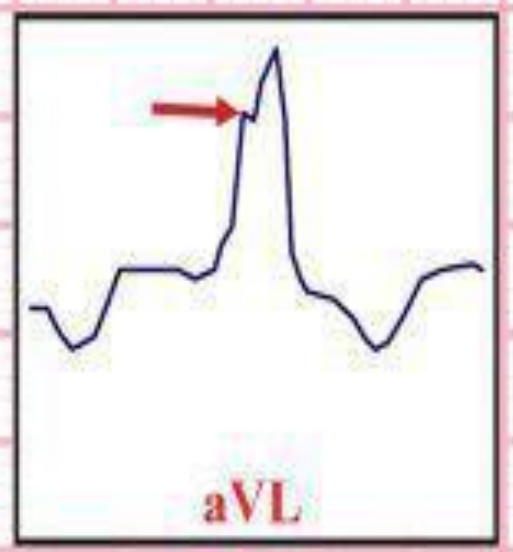
IDM visible dans 20% BBG

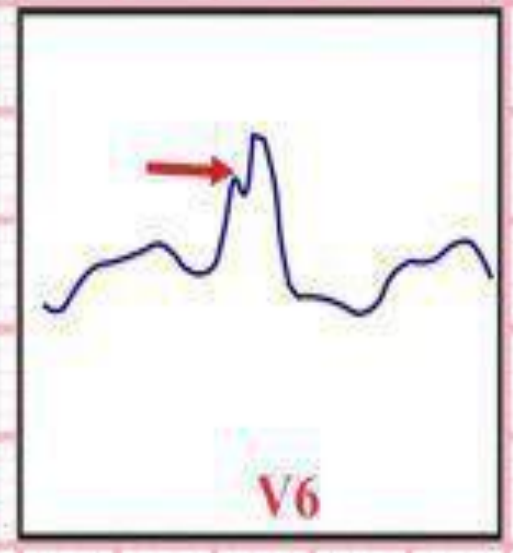
Discordance du ST > 5 mm



Chapman's Sign

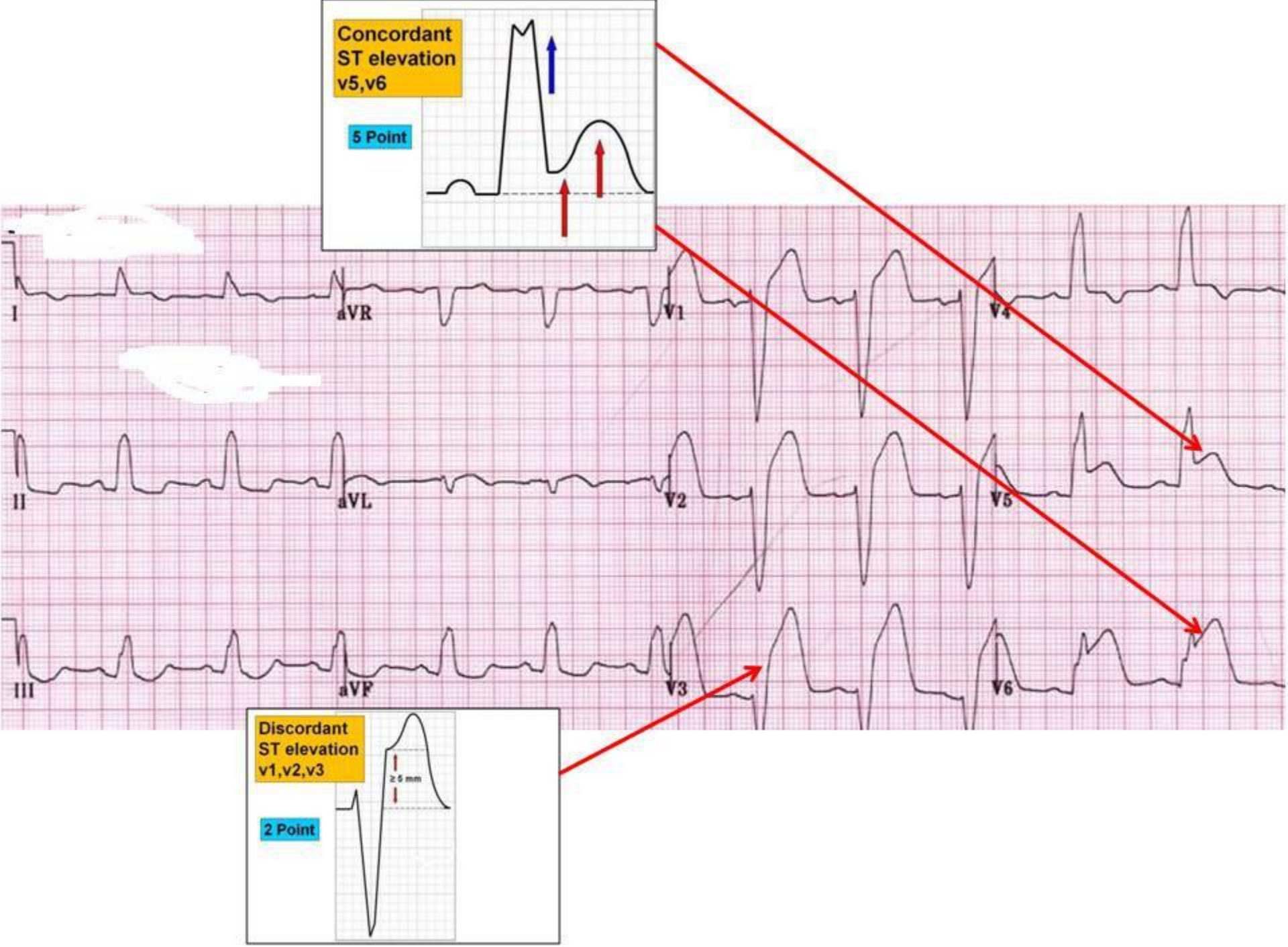


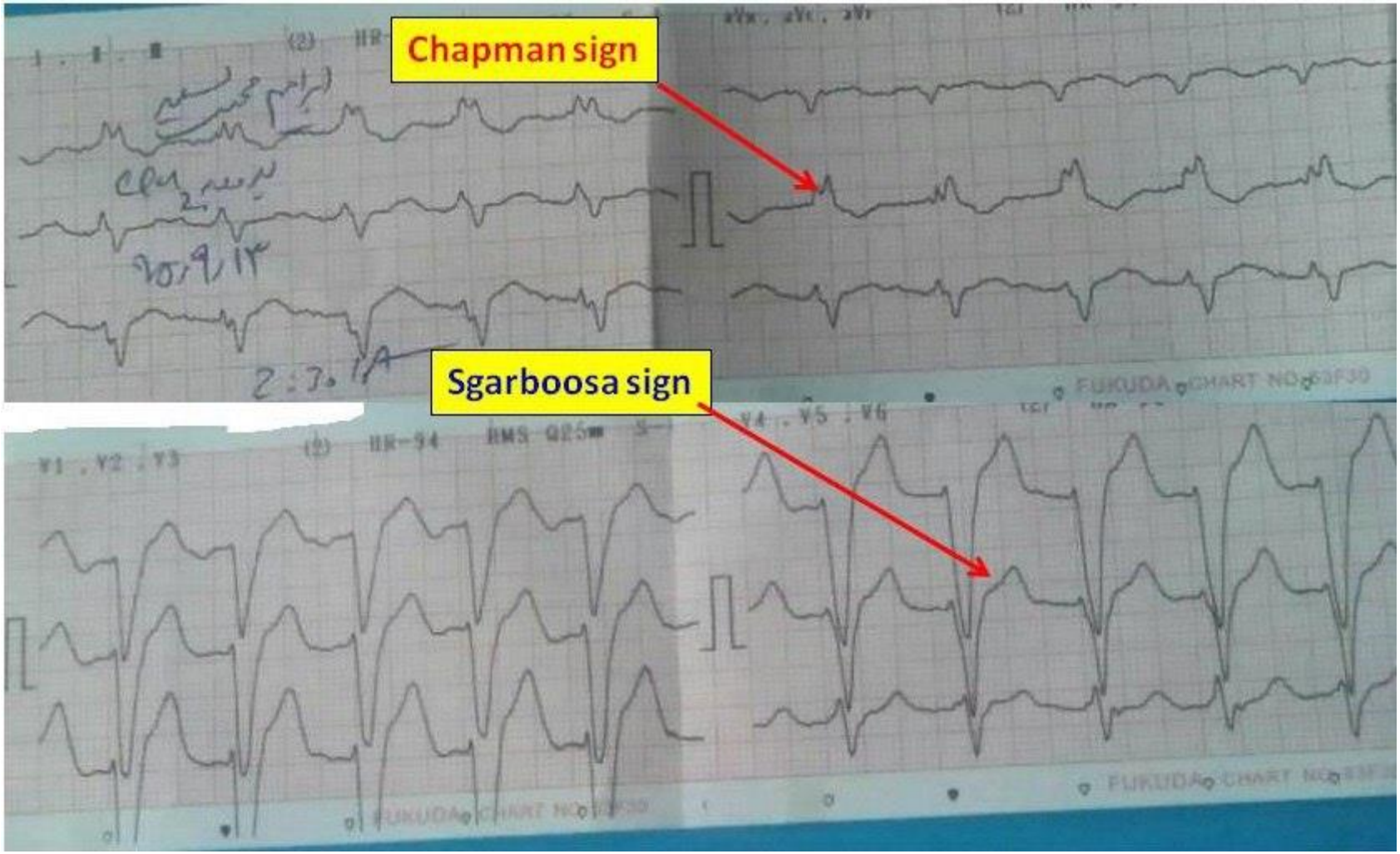


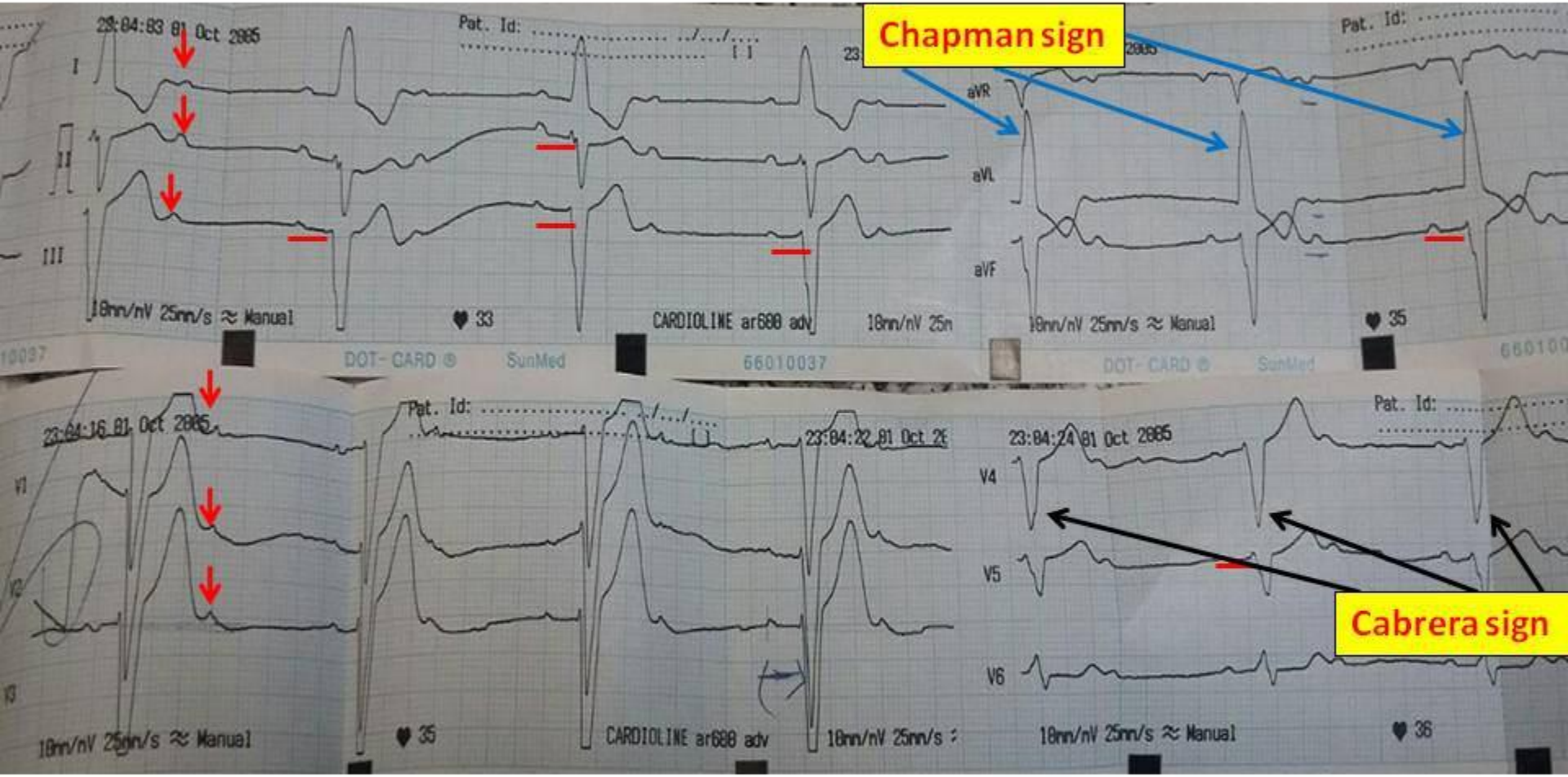


Chapman's sign is used to diagnose an acute myocardial infarction in the setting of LBBB and consists of a notch in the upslope of the R wave in lead I, aVL, or V6.

*** This has a low sensitivity, but a high specificity.**


Concordant
ST elevation
v5,v6
5 Point
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
Discordant
ST elevation
v1,v2,v3
≥ 5 mm
2 Point


Chapman sign
Sgarboosa sign


Chapman sign
Cabrera sign
I
II
III
aVR
aVL
aVF
V4
V5
V6
Pat. Id:
10mm/mV 25mm/s ≈ Manual
CARDIOLINE ar600 adv
DOT-CARD ®
SunMed
66010037

LVH

Strain pattern

- strain pattern
- repolarization abnormality
- ST segment depression with asymmetric biphasic or inverted T waves with prominent R wave

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Prominent T wave inversion
More than 5mm

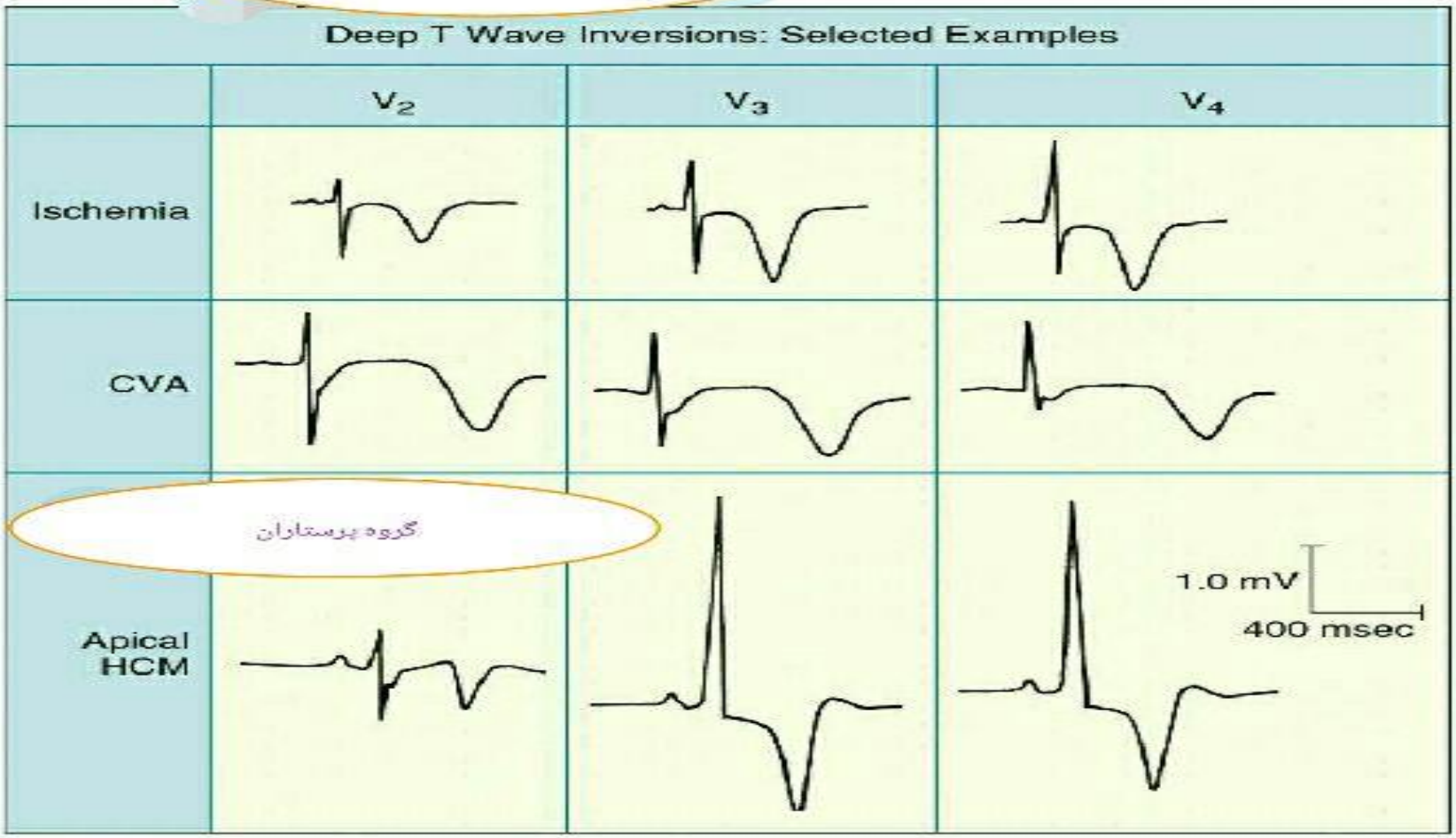


FIGURE 12-46 Deep T wave inversion can have various causes. In the *middle* tracing, note the marked QT prolongation in conjunction with the cerebrovascular accident (CVA) T wave pattern, caused here by subarachnoid hemorrhage. Apical hypertrophic cardiomyopathy (HCM) and takotsubo syndrome are other causes of deep T wave inversion that can be mistaken for ischemia from acute/evolving or chronic obstructive coronary disease. *(From Goldberger AL: Deep T wave inversions. ACC Curr J Rev 5:28, 1996.)*

CVA (mostly Hemorrhagic)

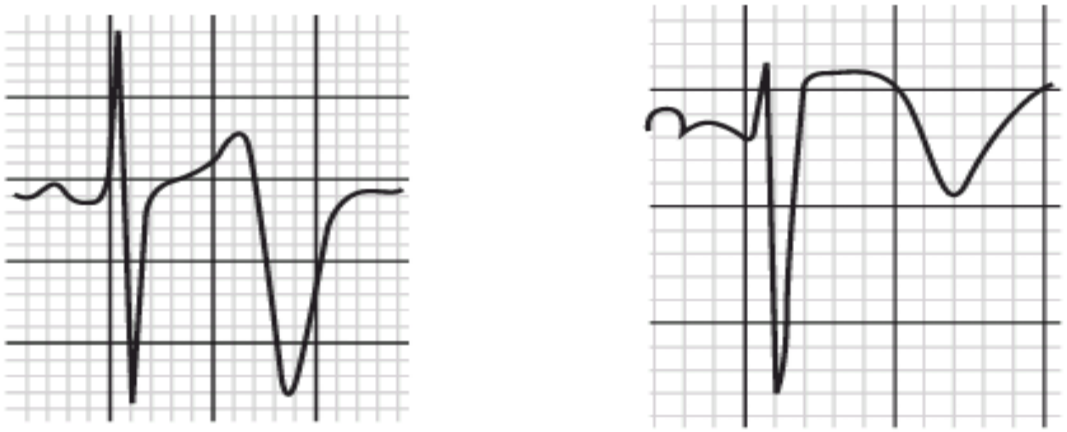


FIGURE 13-9 • ST segment elevation in intracranial hemorrhage. These tracings are from two patients with intracranial hemorrhage and marked ST segment/T wave abnormality. Note the ST segment elevation.

LV Aneurism

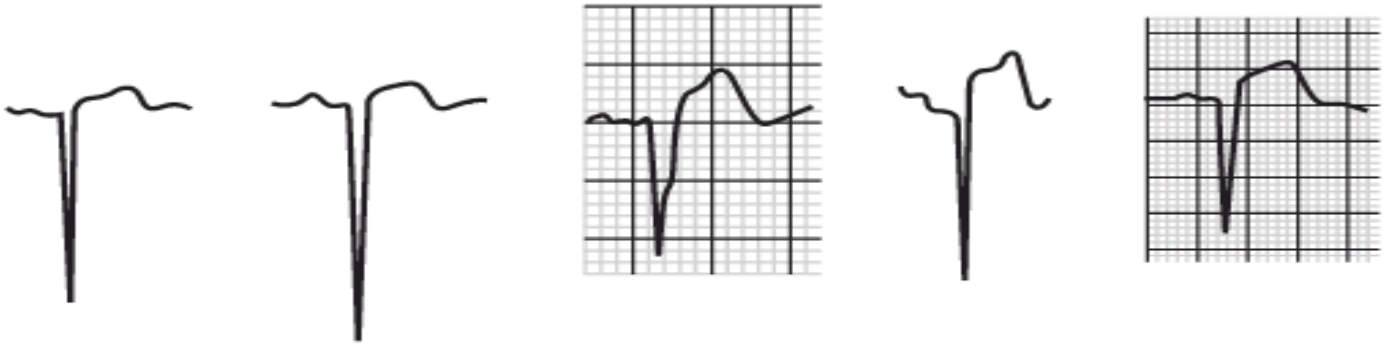
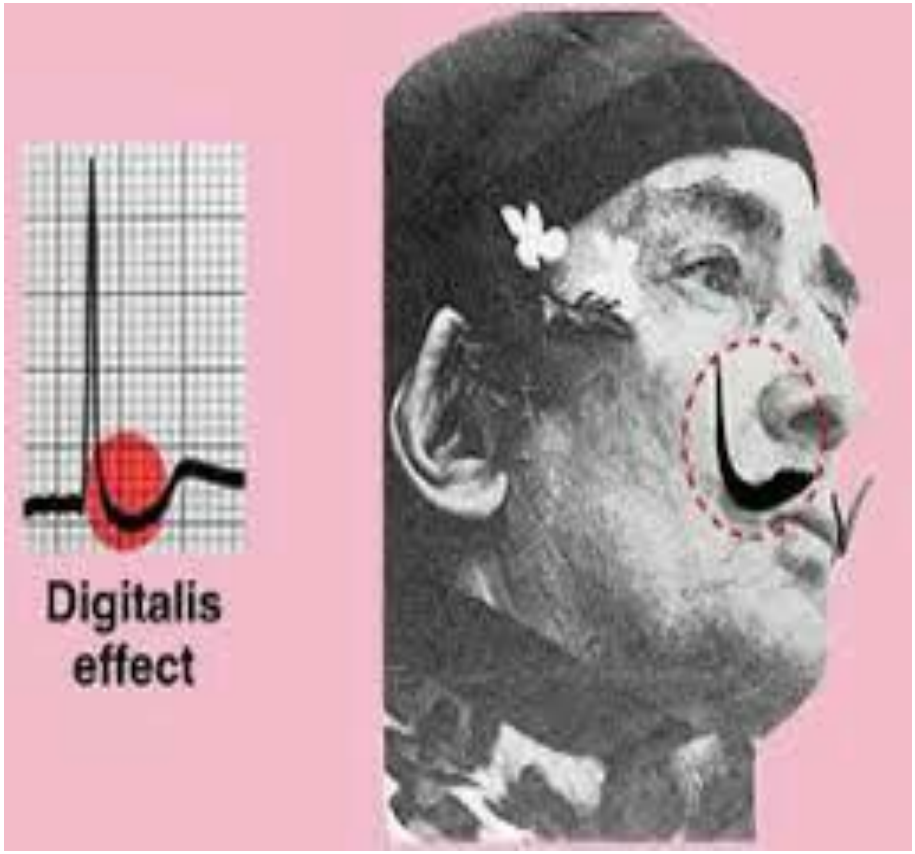


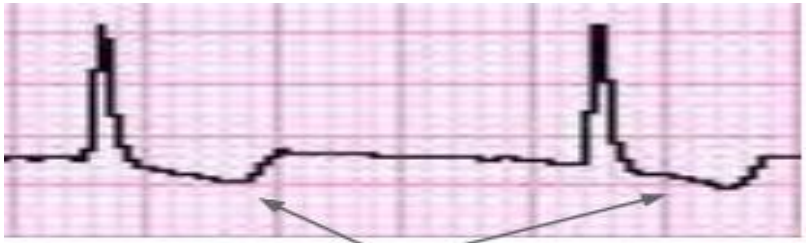
FIGURE 13-8 • Left ventricular aneurysm–related ST segment elevation. Note the varying magnitudes, ranging from minimal to maximal, and morphologies, including concave and convex varieties, of the ST segment elevation.

Digoxin toxicity

Reference range for Digoxin

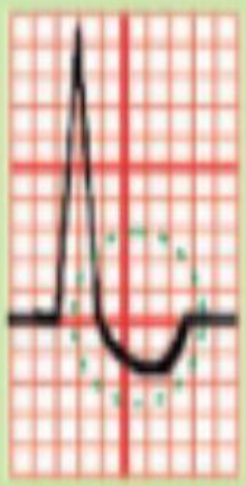
- 0.8-2 ng/mL (1.2-2 nmol/L)
- Half-life: 36 hours
- Toxic level: more than 2 ng/mL (2 nmol/L)





"Reverse check" or "reverse tick" sign from digoxin effect.

مسمومیت با دیژیتال Dig.Toxicity



دپرسیون ST با طرح خاص
(معروف به سبیل سالوادور)

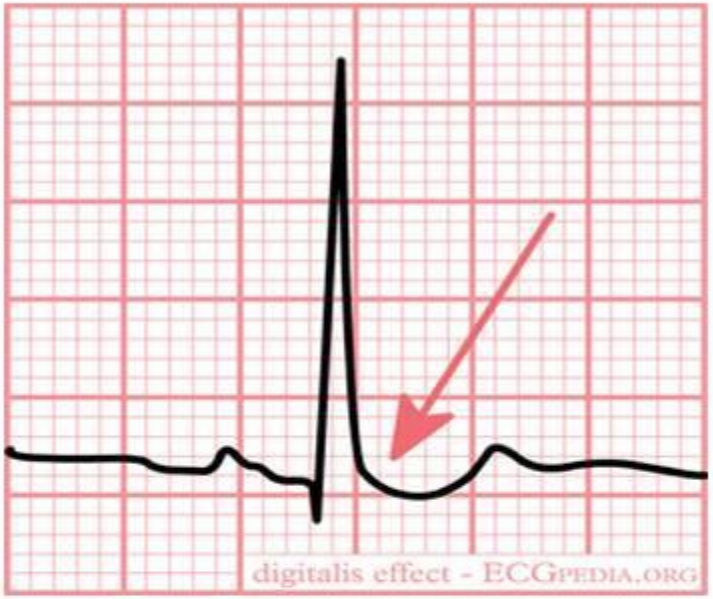
مشخصات: قطعه ST افت و موج T معکوس می‌گردد.

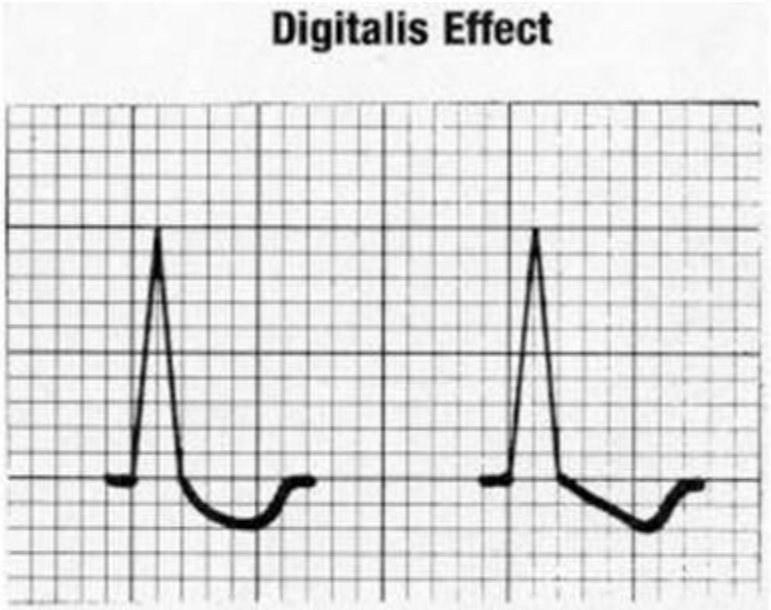
Digoxin toxicity

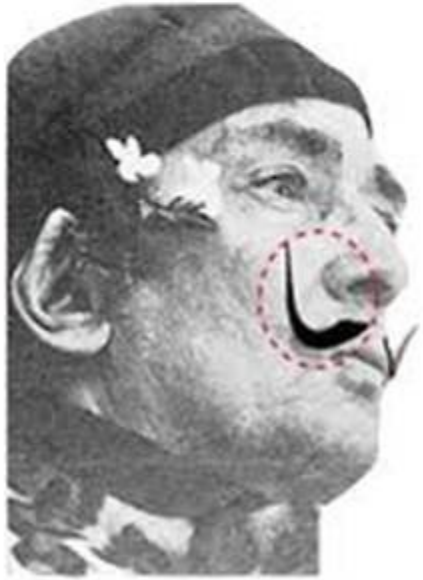
Symptoms in Acute and Chronic Digoxin Overdose

Acute Toxicity	Chronic Toxicity
■ GI: Nausea, vomiting, and abdominal pain • Absence of nausea and vomiting hours after exposure make acute digoxin toxicity unlikely ■ CV: Dysrhythmias • Hypotension secondary to dysrhythmia ■ CNS: Lethargy, confusion, weakness	■ Symptoms tend to be more insidious ■ GI symptoms less pronounced ■ Neuro symptoms more pronounced • Delirium, confusion, drowsiness, hallucinations ■ CV: dysrhythmias • Hypotension secondary to dysrhythmia ■ Visual changes such as alterations in color vision • Scotomas or blindness

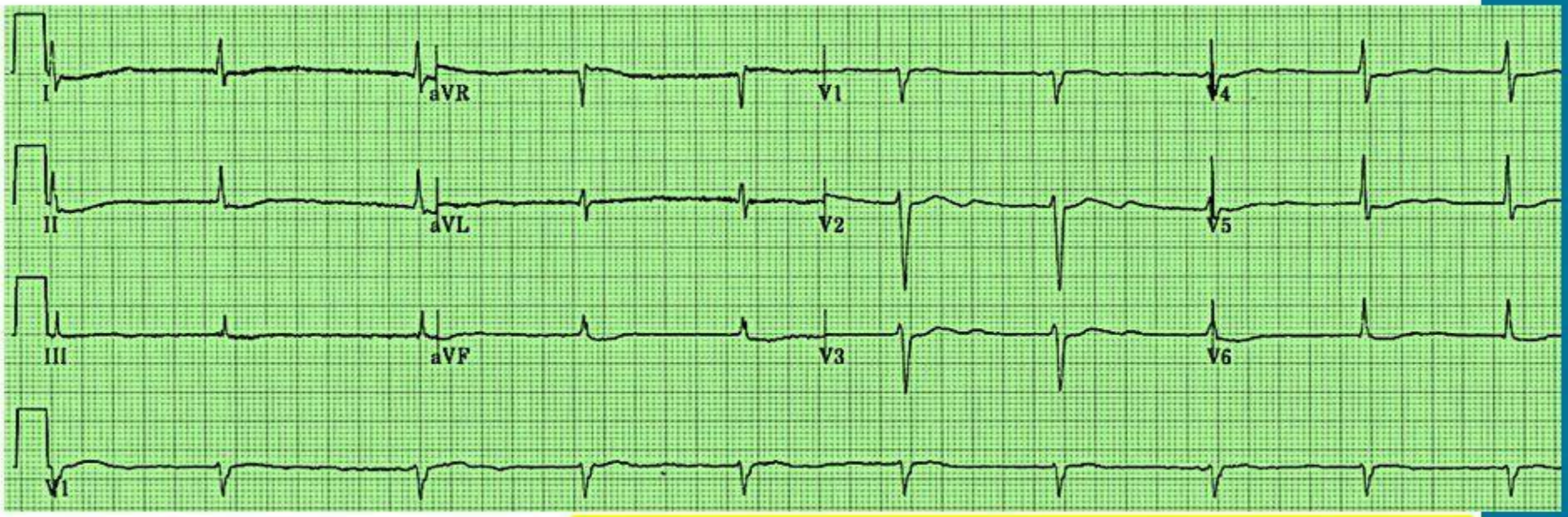
Digoxin toxicity





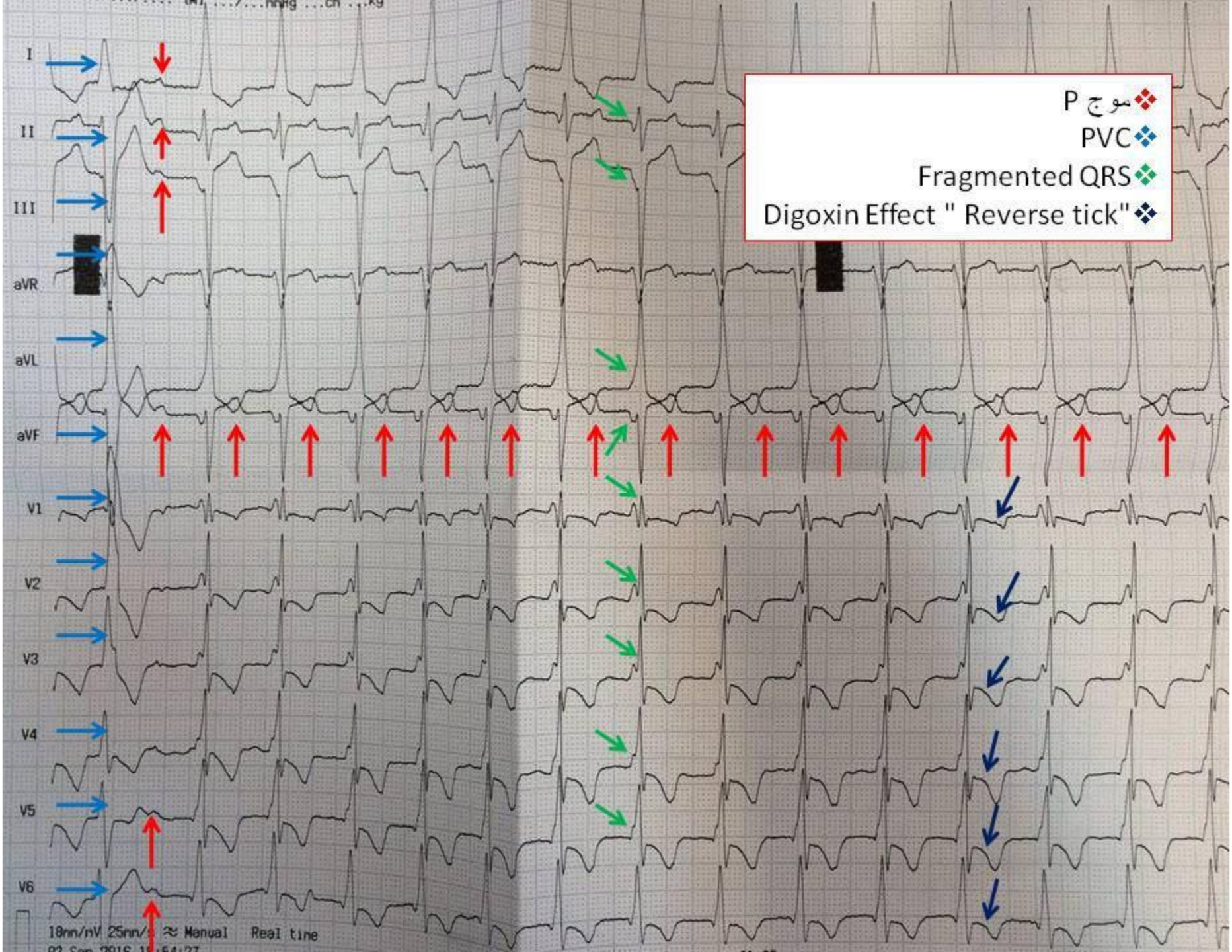


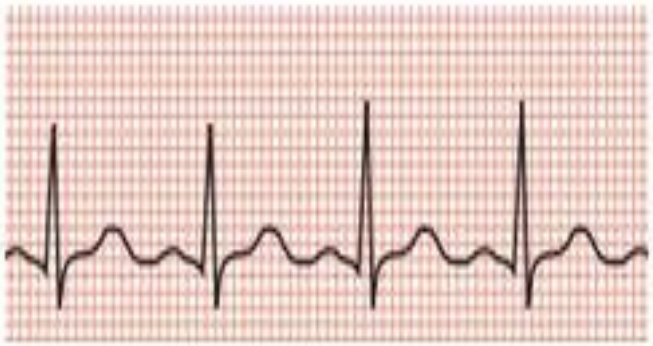
Digitalis Effect



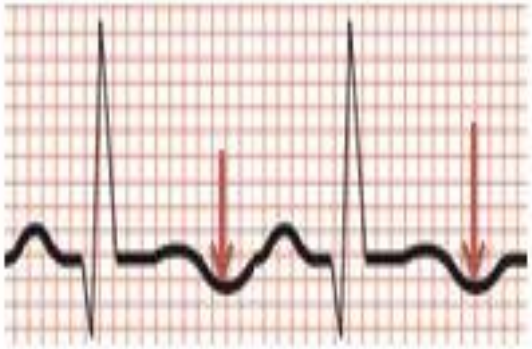
Digoxin Effect with Evidence of Toxicity

- ECG Findings
- ● ST segment shortening and depression leading to a“scooped” appearance
- ● QT interval shortening
- ● PR interval prolongation
- ● Decreased T wave amplitude
- ● Premature ventricular complexes are the most commondysrhythmia
- ● Bradydysrhythmias, various heart blocks, especially with findings consistent with increased automaticity (atrialtachycardia with block, atrial fibrillation with slow ventricular response, accelerated junctional rhythms)
- ● Bidirectional ventricular tachycardia may rarely be seen


P موج
PVC
Fragmented QRS
Digoxin Effect " Reverse tick"
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
10mm/mV 25mm/s ≈ Manual Real time



Normal ECG
in unstable angina

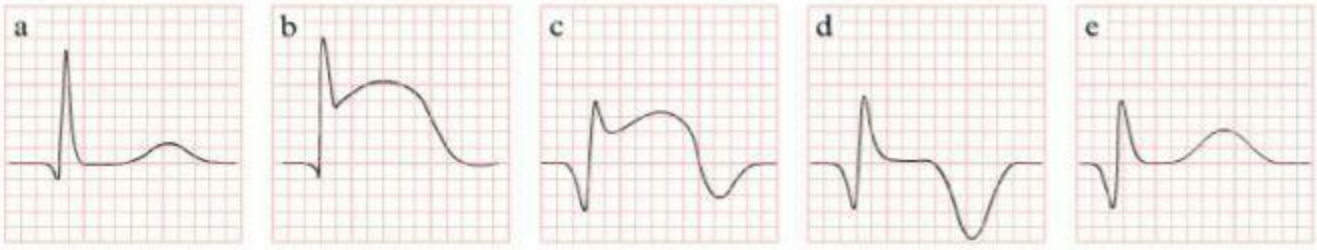


T-wave inversion
in unstable angina

ST-segment depression
in unstable angina

ECG manifestations of ischemia

a) Normal
b) ST elevation
c) Pathologic Q Wave
d) T wave inversion
e) normalisation with persistent Q wave



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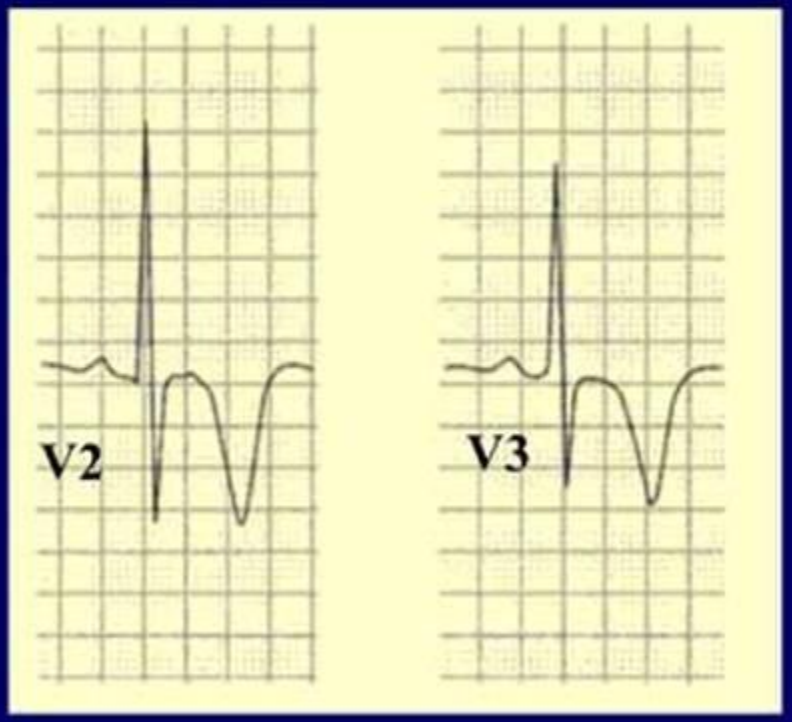
Also:
Change in heart axis, ST depression, R wave decrease
Widened QRS complex, QTc prolongation

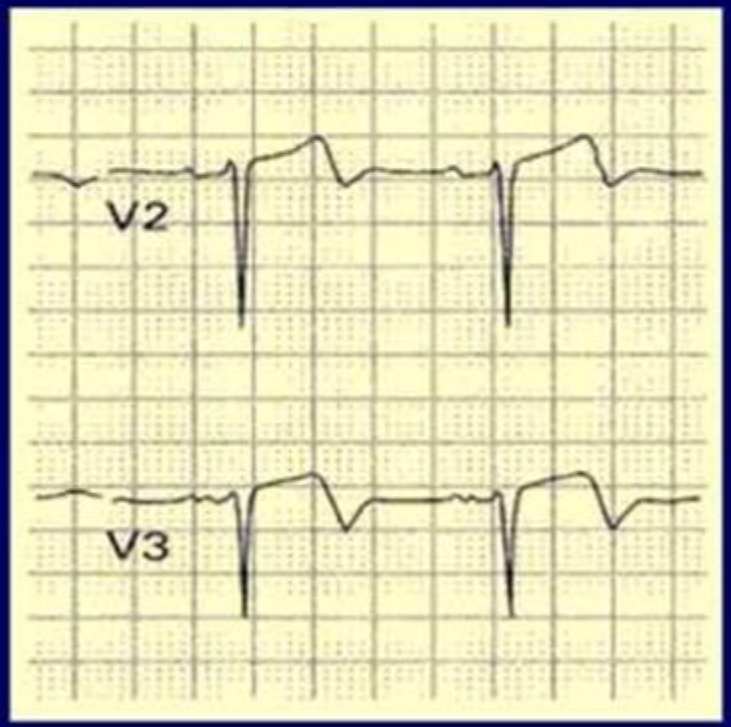
Wellens syndrome

There are 2 types of Wellens syndrome:

- 1- Symmetric deeply inverted T waves in V2 and V3
- 2- Biphasic T waves in V2 and V3 (less common)



Type 1



Type 2

Table 1 :Wellens' syndrome criteria

r history of chest pain
ing chest pain: EKG is normal or with mild ST elevation or depression, or minal negative deflection of the T wave in V1 and V2
diac enzymes are normal or mildly elevated
pathologic precordial Q-waves
loss of precordial R-waves
eply inverted or biphasic T-waves in V2 and V3, possibly V1, V4, V5 and/or en pain free

Wellens Syndrome

- وجود درد سینه ای آنزینی
- سطح آنزیم های قلبی طبیعی یا کمی بالا
- بدون موج Q و تغییرقطعه ST
- طبیعی بودن موج Rدر لیدهای سینه ای
- بروز Ant MI در ۷ ساعت آینده
- انسداد حاد ابتدا شریان LAD
- 1980 توسط آقای Wellens
- Primary PCI یا ترومبولیتیک تراپی

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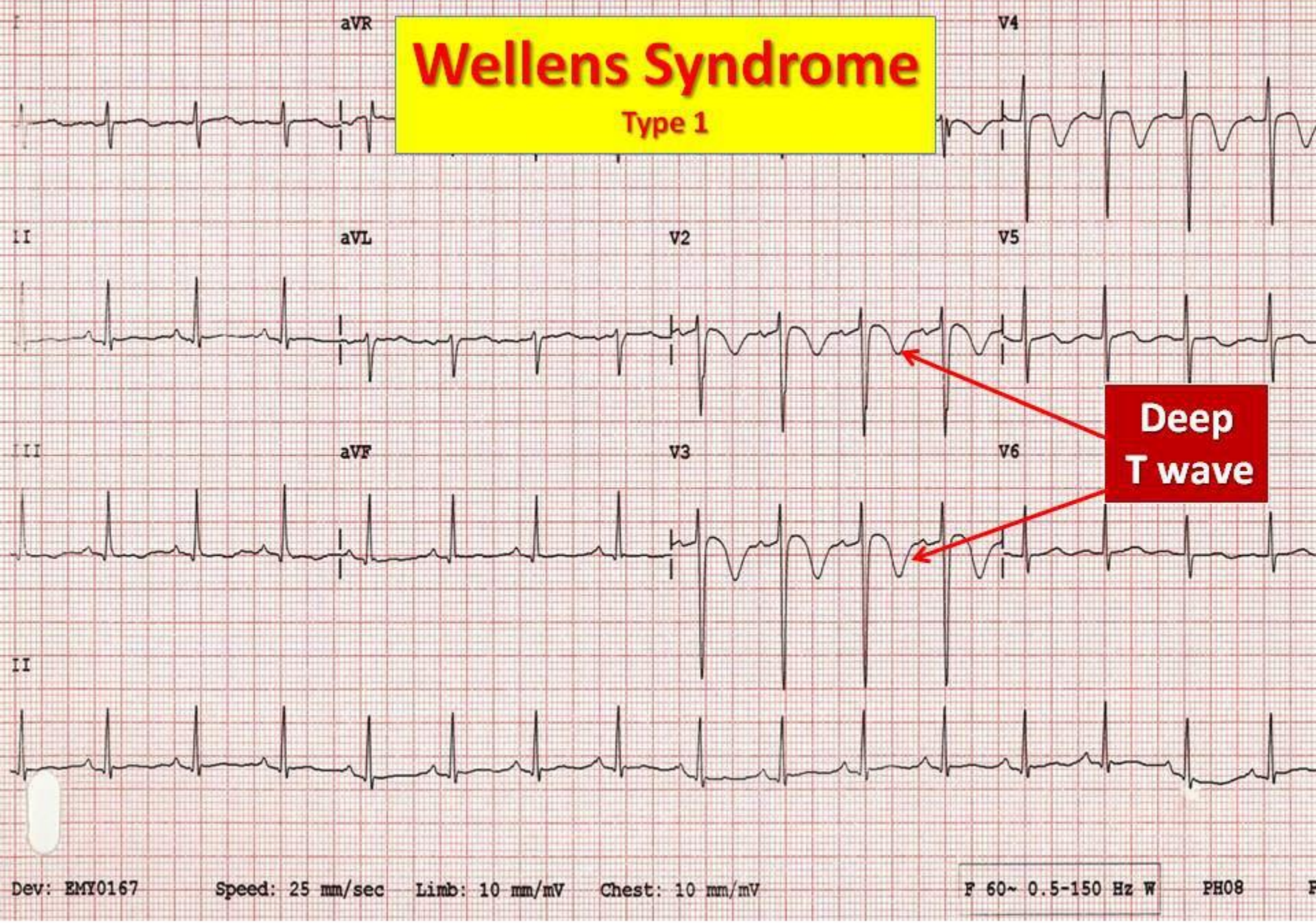
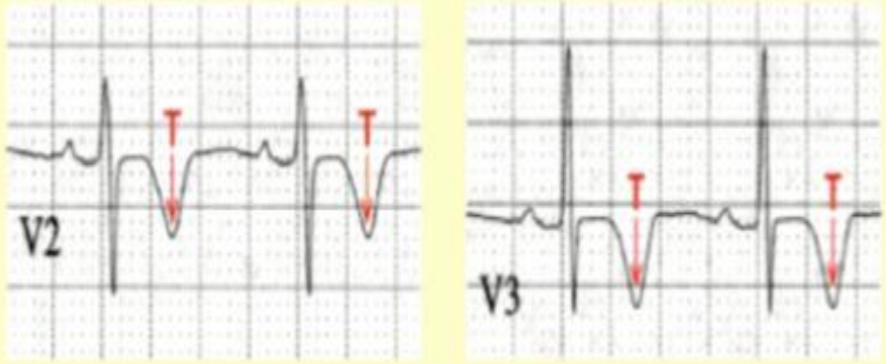

Wellens Syndrome
Type 1
aVR
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
Deep
T wave
Dev: EMY0167
Speed: 25 mm/sec
Limb: 10 mm/mV
Chest: 10 mm/mV
F 60~ 0.5-150 Hz W
PH08

Clinical Significance

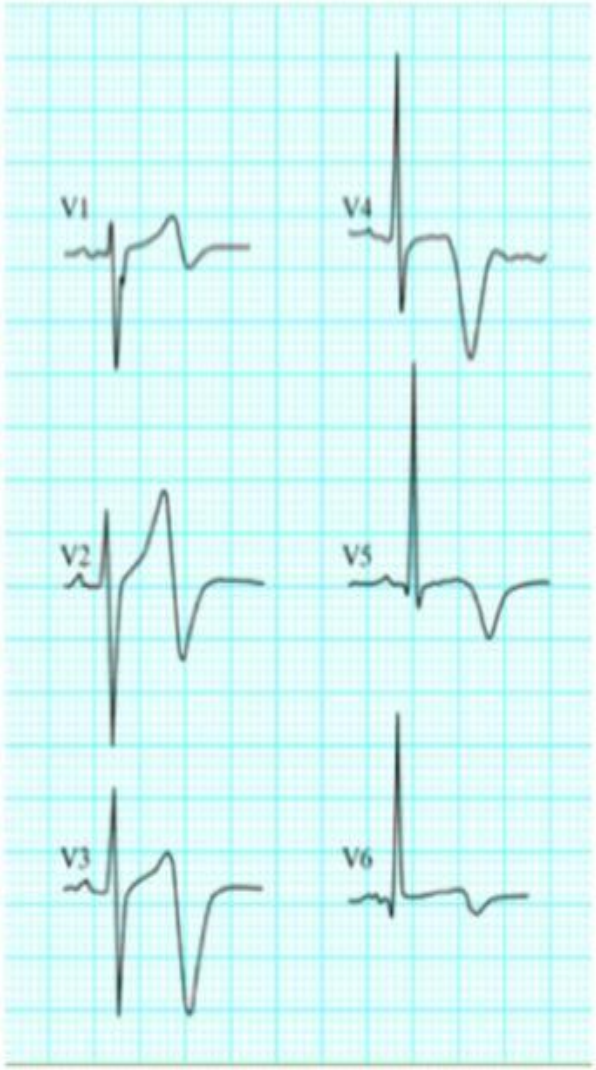
- Wellens' syndrome is a pattern of **deeply inverted or biphasic T waves in V2-3**, which is highly specific for a **critical stenosis of the left anterior descending artery** (LAD).
- Patients may be pain free by the time the ECG is taken and have normally or minimally elevated cardiac enzymes; however, they are at extremely **high risk for extensive anterior wall MI** within the next few days to weeks.
- Due to the critical LAD stenosis, these patients usually require invasive therapy, do poorly with medical management and may suffer MI or cardiac arrest if inappropriately stress tested.

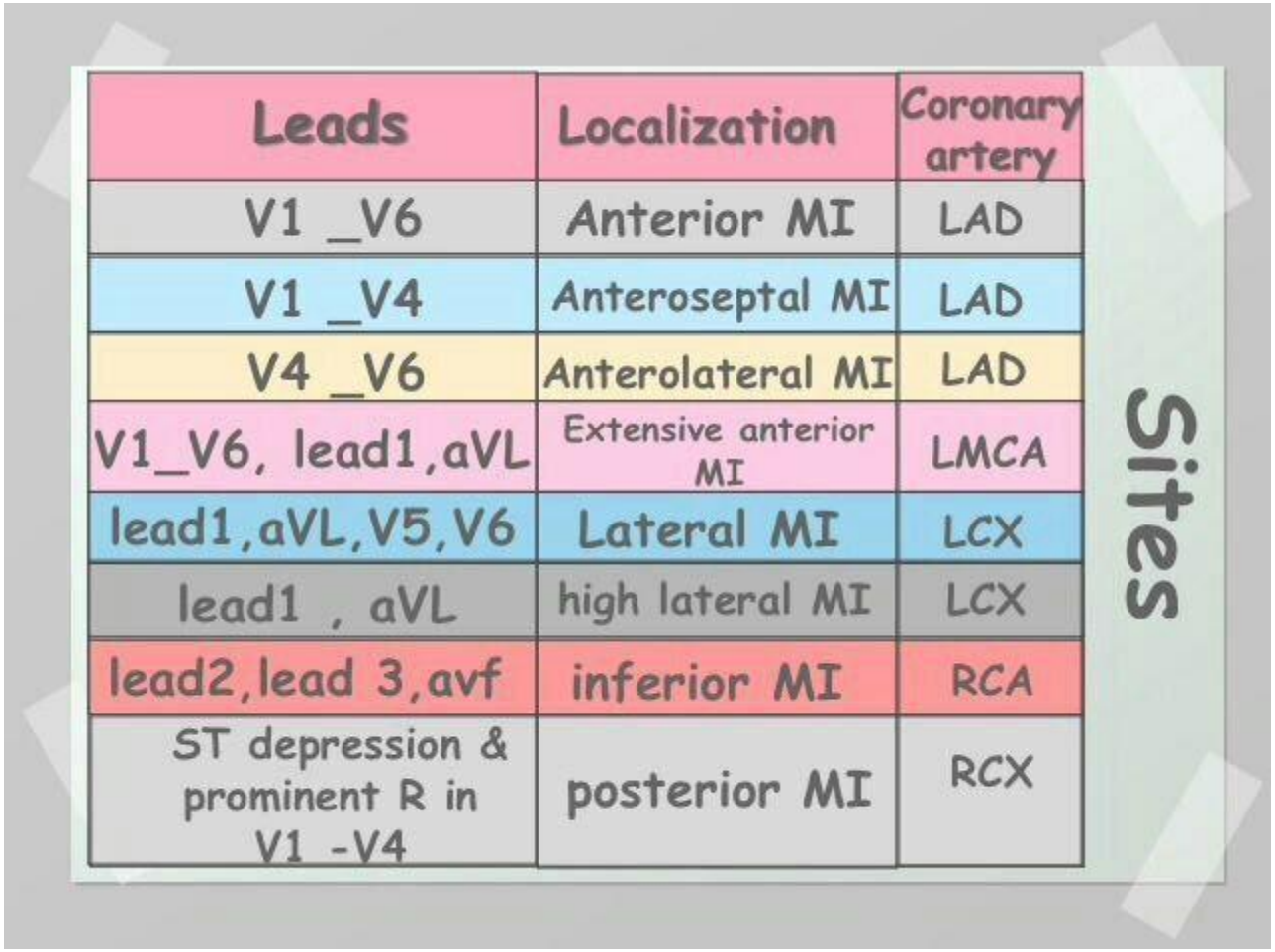
Type I (75% of Cases)

Wellens Syndrome



Type II (25% of Cases)




Leads
Localization
Coronary artery
V1 _V6
Anterior MI
LAD
V1 _V4
Anteroseptal MI
LAD
V4 _V6
Anterolateral MI
LAD
V1_V6, lead1,aVL
Extensive anterior MI
LMCA
lead1,aVL,V5,V6
Lateral MI
LCX
lead1 , aVL
high lateral MI
LCX
lead2,lead 3,avf
inferior MI
RCA
ST depression & prominent R in V1 -V4
posterior MI
RCX
Sites

معیارهای تشخیص RVMI در ECGسمت چپ به روایت شمروث

1- S>1mm در لید V1

2- در لید V2 اگر ST depression مساوی یا کمتر از 50 درصد ST elevation لید avf باشد

3- ST elevation در لیدهای V1 تا V6 ولی با این معیار که میزان الویشن از V1به سمت V6کاهش میابد یعنی الویشن در V1از همه بلند تر و در V6از همه کوتاهتر است و Qهم وجود ندارد

(در Ant MIمیزان الویشن از V1تا V6افزایش می یابد و Qپاتولوژیک داریم)

4-در V1، ST elevation و در V2، ST depression داشته باشیم

How to spot right ventricular infarction

The first step to spotting RV infarction is to suspect it… in all patients with inferior STEMI!

In patients presenting with inferior STEMI, right ventricular infarction is suggested by the presence of:

- **ST elevation in V1** - the only standard ECG lead that looks directly at the right ventricle.
- **ST elevation in lead III > lead II** - because lead III is more "rightward facing" than lead II and hence more sensitive to the injury current produced by the right ventricle.

Other useful tips for spotting right ventricular MI

If the magnitude of ST elevation in V1 exceeds the magnitude of ST elevation in V2.

- If the ST segment in V1 is isoelectric and the ST segment in V2 is markedly depressed.
- ***NB. The combination of ST elevation in V1 and ST depression in V2 is highly specific for right ventricular MI.***

Right ventricular infarction is confirmed by the presence of ST elevation in the right-sided leads (V3R-V6R). Right-sided leads

Inferior MI +Posterior M.I.

Lateral / Infero Lateral / Baso Lateral MI not postero inferior MI.

↓

Proximal RCA (posterior+inferior) + RV infarct

OR

LCX Posterior+Inferior MI

↓

ST ↑ II,III,aVF,aVL,I

ST ,tall R V1,V2,V3,

ST ↑ II>III

↓

ST ↑ II,III,aVF

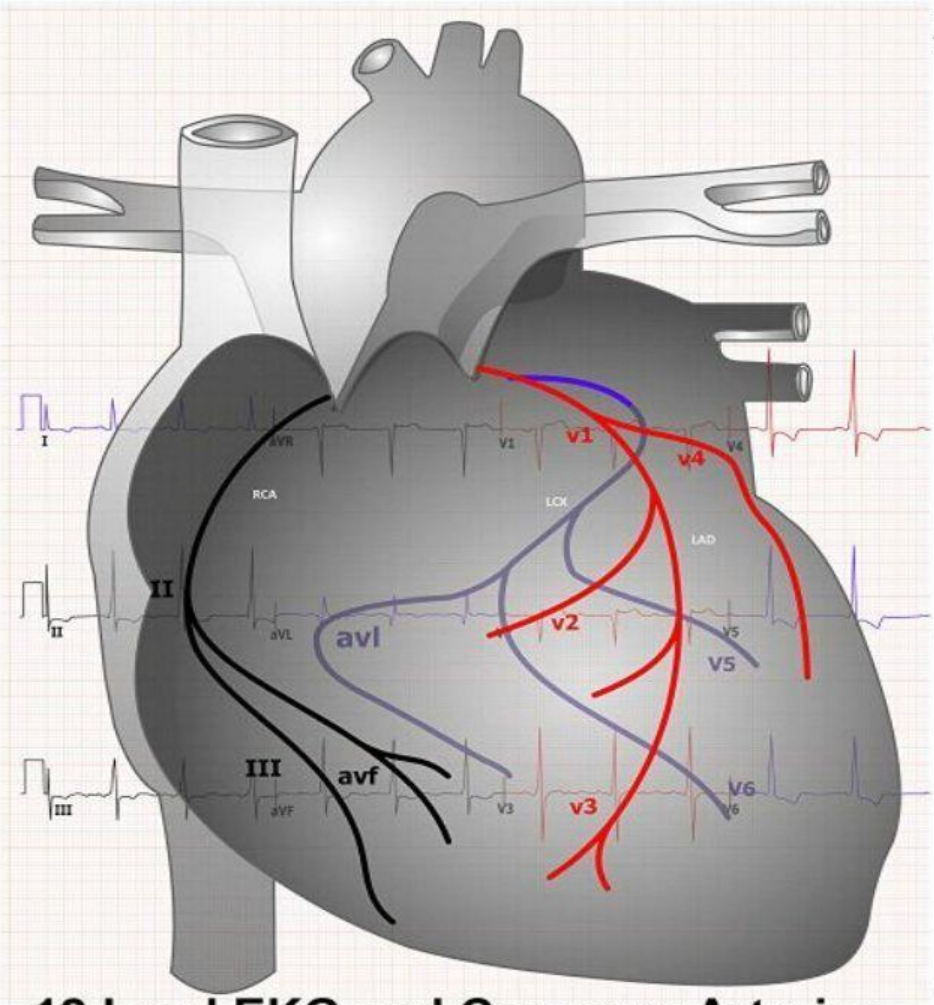
ST ↓ I,aVL

↑

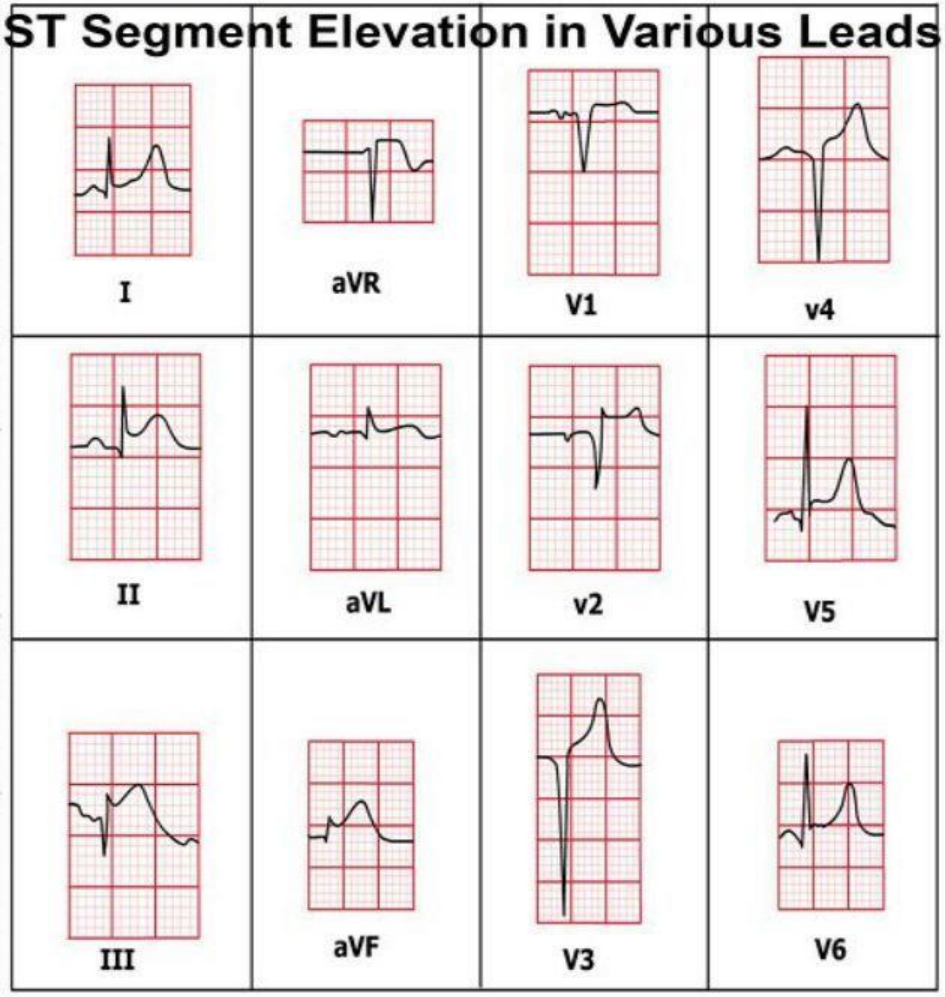
↑ ST V3R,V4R

ST III>II

Dr. UZMA ANSARI



12 Lead EKG and Coronary Arteries



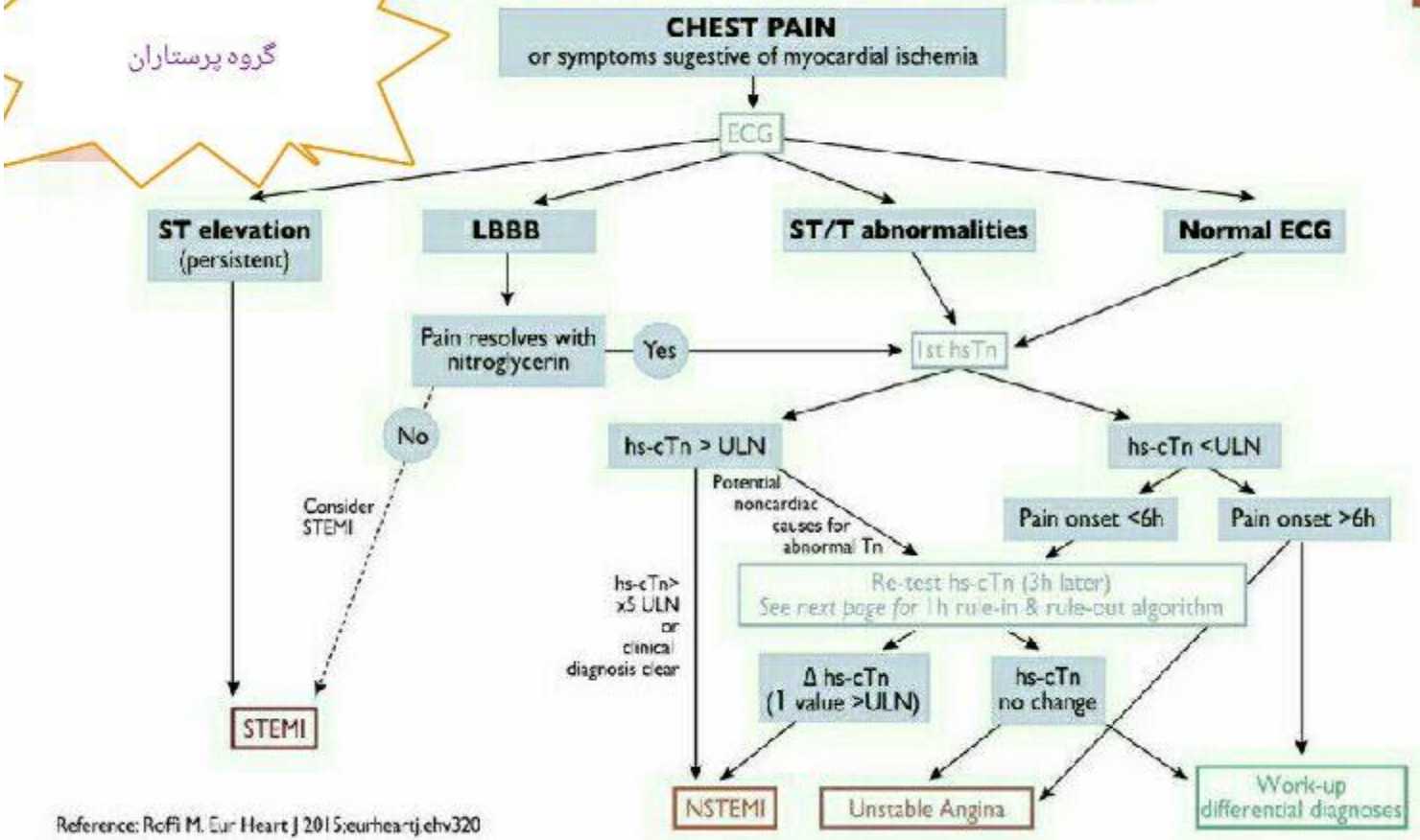
Coronary Circulation and 12 Lead EKG

Leads	Anatomical area	Coronary artery
I, aVL, V5, V6	Lateral wall	Circumflex artery
II, III, aVF	Inferior wall	Right coronary artery
V1, V2	Septum	Left anterior descending artery
V3, V4	Anterior wall	Left anterior descending artery

گروه پرستاران

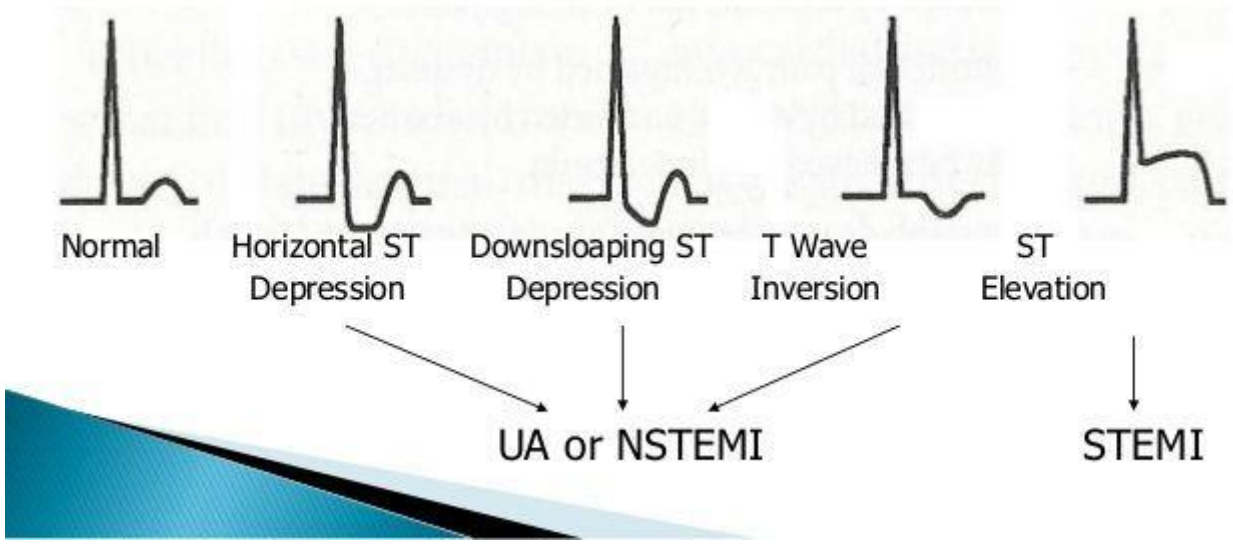
ACUTE CORONARY SYNDROMES: Diagnosis (1)

2.1
p.24



Reference: Roffi M. Eur Heart J 2015;eurheartj.ehv320

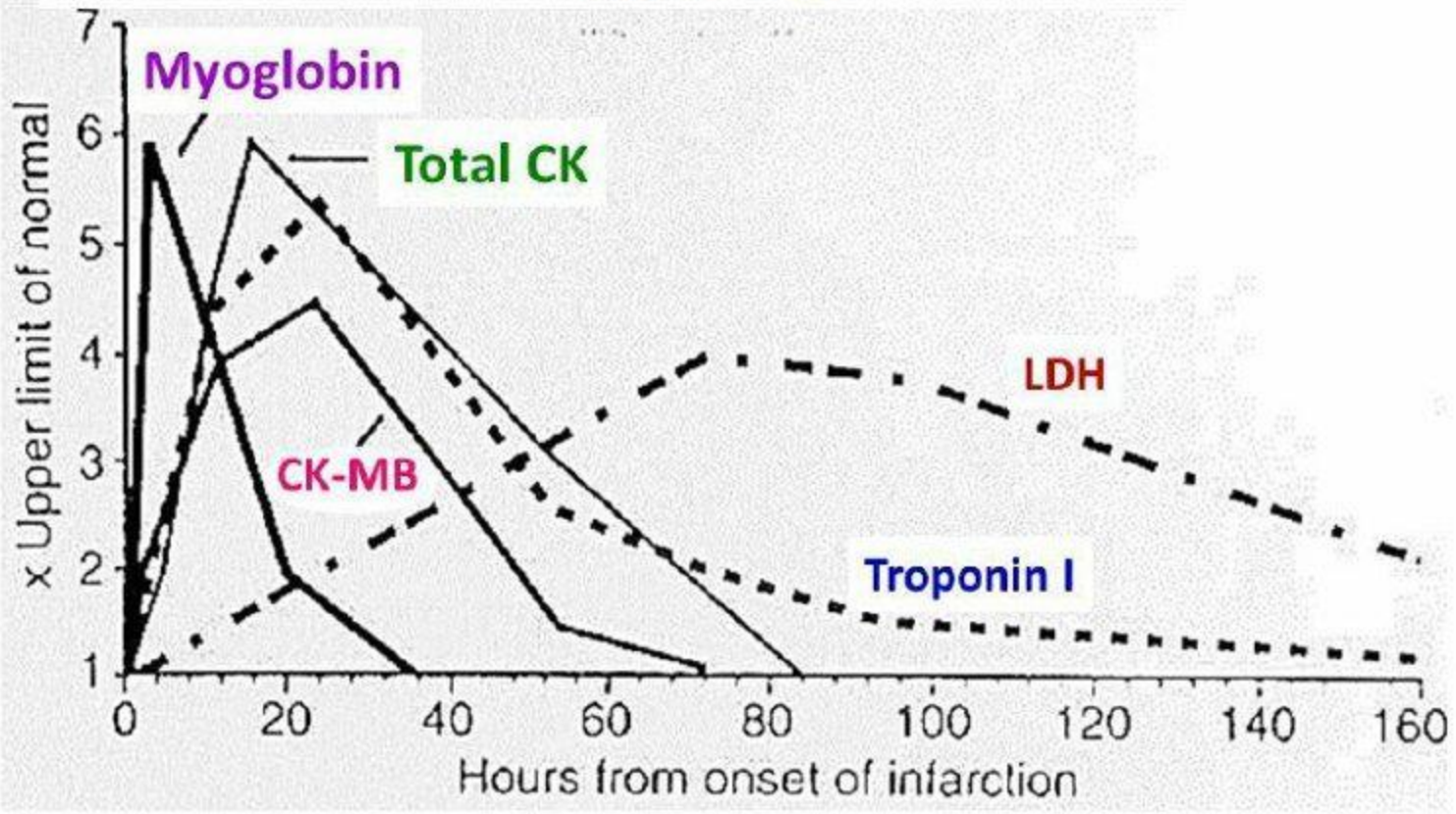
ACS – ECG findings



Location	ST ↑	ST ↓
Anterior	I, aVL, V1-6	III and aVF
Lateral	I, aVL, V5-6	II, III, aVF
Inferior	II, III, aVF	I and aVL
Right Ventricle	V1 and $V4_R$ III > II	I and aVL

Lead I Lateral	aVR	V1 Septal	V4 Anterior
Lead II Inferior	aVL Lateral	V2 Septal	V5 Lateral
Lead III Inferior	aVF Inferior	V3 Anterior	V6 Lateral

Biomarkers Released in Blood after Myocardial Infarction



تغییرات آنزیمی در MI

آنزیم قلبی	افزایش اولیه (ساعت)	رسیدن به حداکثر مقدار (ساعت)	برگشت به حد نرمال
CPK کل	۳-۶	۲۴ -۳۶	۳ روز
CK- MB	۴-۸	۱۲-۲۴	۳-۴ روز
میوگلوبین	۱-۳	۴-۱۲	۱۲ ساعت
تروپونین T یا I	۳-۴	۴-۲۴	۱-۳ هفته

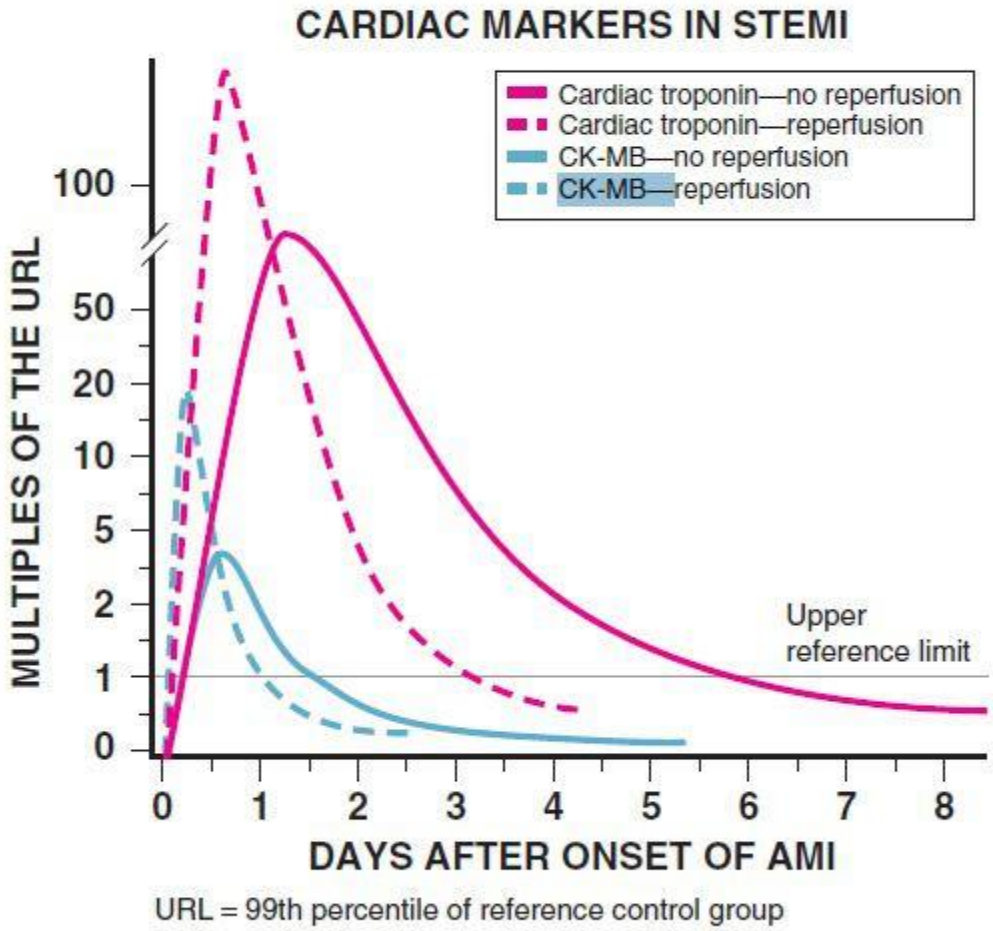
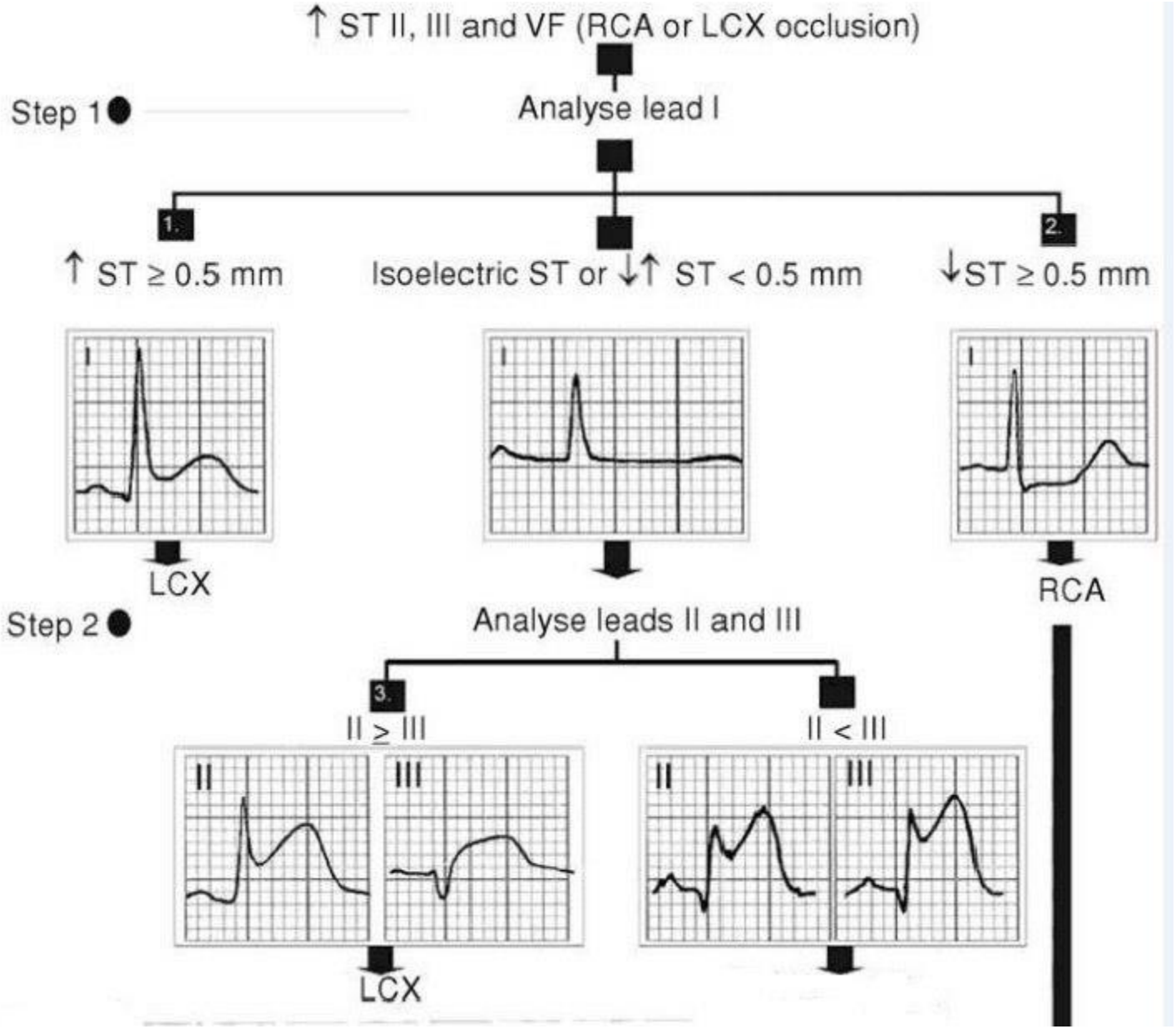


FIGURE 51-17 The kinetics of the release of CK-MB and cardiac troponin in


↑ ST II, III and VF (RCA or LCX occlusion)
Step 1
Analyse lead I
1.
↑ ST ≥ 0.5 mm
Isoelectric ST or ↓↑ ST < 0.5 mm
2.
↓ST ≥ 0.5 mm
I
I
I
LCX
RCA
Step 2
Analyse leads II and III
3.
II ≥ III
II < III
II
III
II
III
LCX

معیارهای شناسایی الگو The de Winter ECG

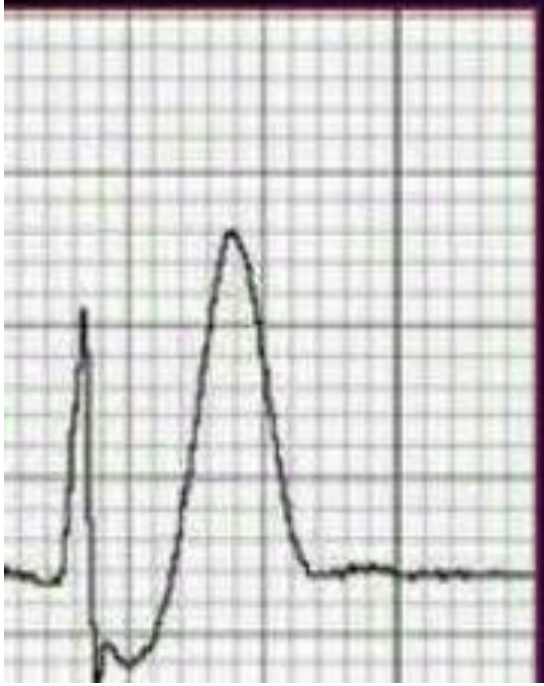
۱- موج Tall T برجسته در لیدهای سینه ای

۲- وجود Upsloping ST depression بیشتر از ۱ میلیمتر در لیدهای

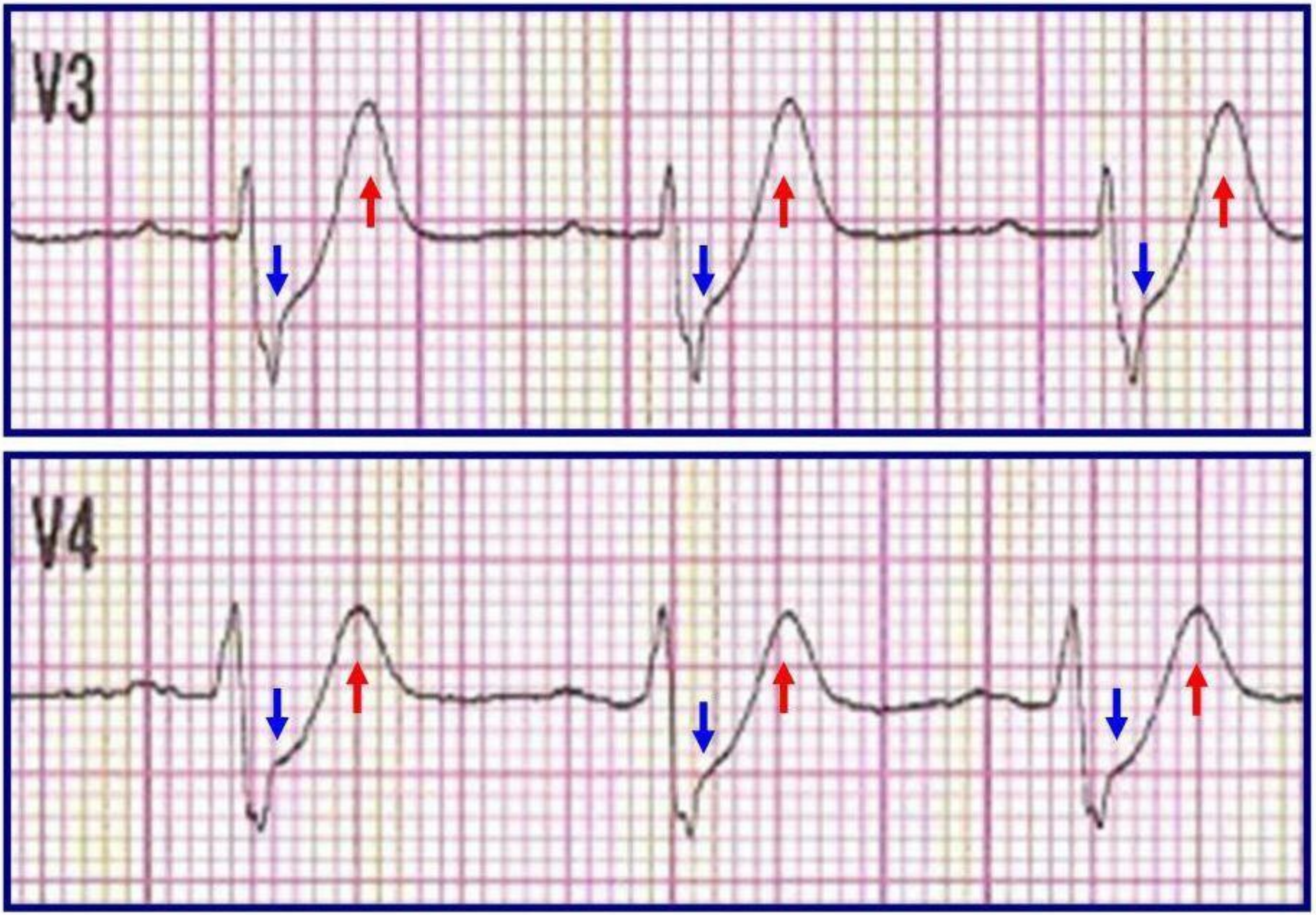
۳- عدم حضور STEدر لیدهای سینه ای

۴- وجود STEبیشتر از نیم میلیمتر در لید AVR

۵- وجود آمدن نرمال مورفولوژی STE در ادامه



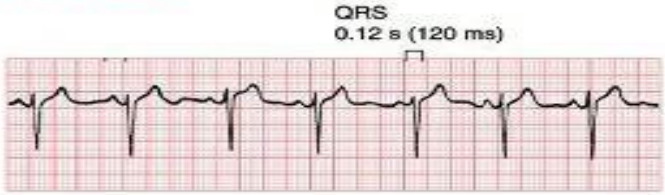
De Winter ST/T-Waves



منظور از تاکی‌کاردی از نوع Narrow QRS چیست؟

- Narrow QRS Complex Tachycardia به این معناست که QRS Duration در آن کمتر از 3 خانهٔ کوچک (کمتر از 0.12 ثانیه یا 120 msec) می‌باشد.
- لازم به ذکر است که در ECG هر خانهٔ کوچک معادل 0.04 sec یا 40 msec می‌باشد.

Normal QRS Complex



پس QRS Duration در AVNRT، کمتر یا مساوی 3 خانهٔ کوچک در EKG است.

Rhythm و Rate در AVNRT چگونه است؟

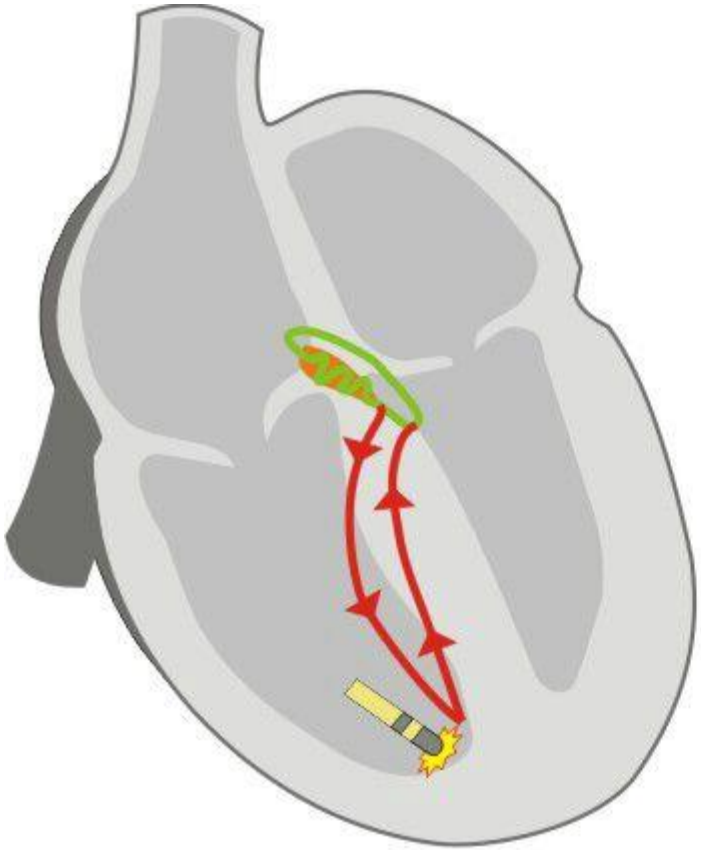
- ریتم در PSVT منظّم است.

Braunwald's: معمولاً QRS Rate بین **150** beats/min تا **250** است.

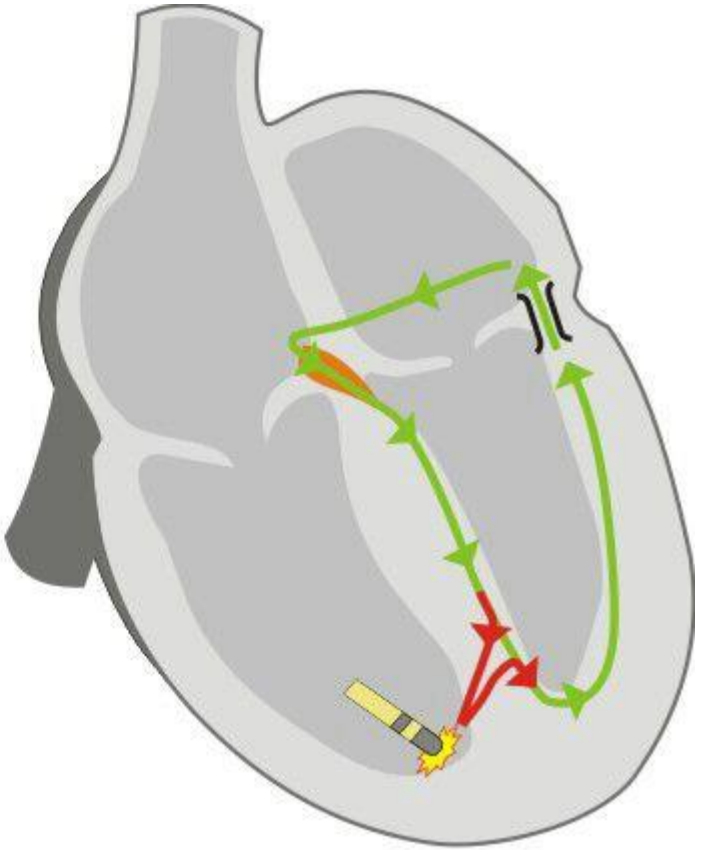
- البته در رفرانس‌های گوناگون و البتّه معتبر، بازه‌های مختلفی بیان شده است.
- در بعضی رفرانس‌ها بین 120 تا 250 – 200 هم ذکر شده است.
- به طور ناشایع Rate ممکنست به کمتر از 110 هم برسد.
- گاهی، بویژه در اطفال ممکنست Rate به بالاتر از 250 هم برسد.
- سرعت تا 300 هم ممکنست برسد و تفسیر را با مشکل مواجه کند.

Types of SVT

- PSVT (PAT, AVNRT ,AVRT)
- Accessory pathway tachycardia (WPW)
- Atrial tachycardia
- Atrial fibrillation
- Atrial flutter
- Junction Tachycardiardia
- Sinus Tachycardiardia



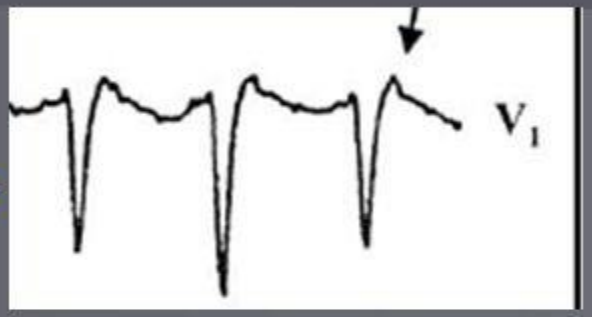
AVNRT

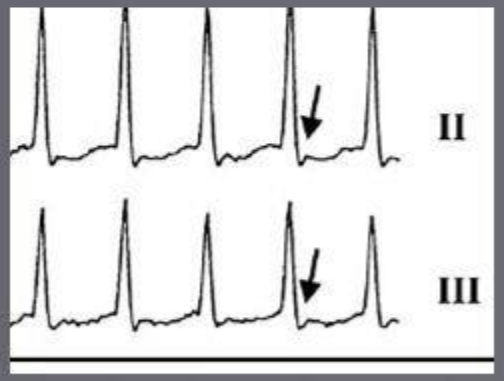


AVRT

AVNRT vs. AVRT: Can you tell them apart

- Helpful ECG findings
  - Pseudo R' in V1
  - Pseudo S in II, III, aVF
    - specific (but not sensitive) for AVNRT
  - ST elevation in aVR
  - RP >100 ms
  - ST depression ≥2mm
    - Suggest (not highly specific or sensitive) AVRT

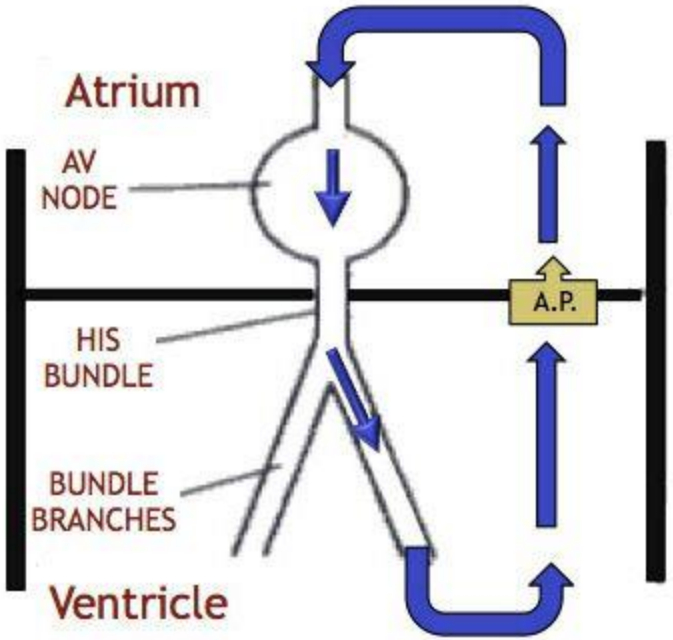


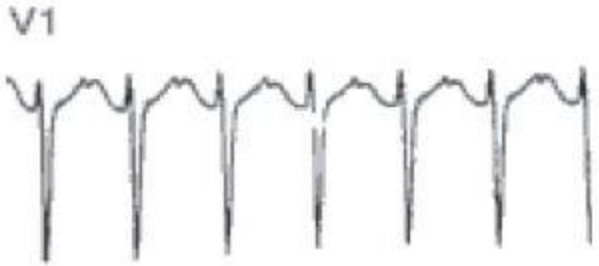


Bottom line = 12-lead lacks 100% accuracy but important to look because AVRT more serious Dx

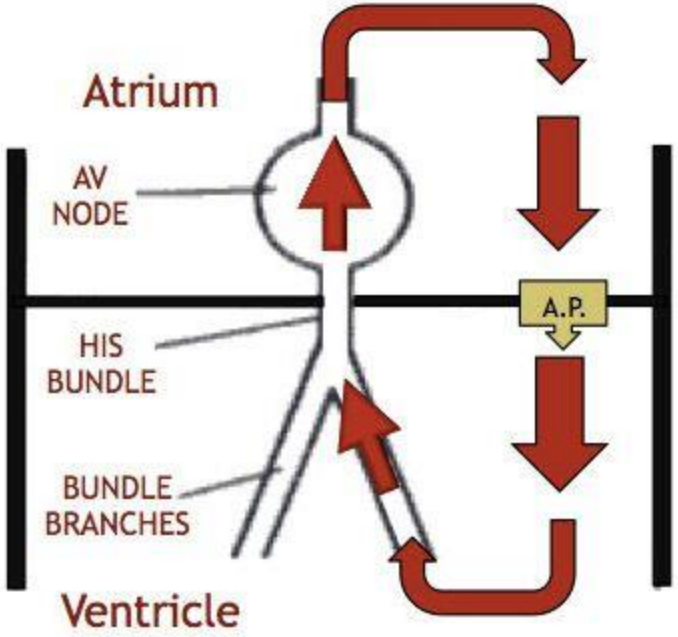
See Adam Osters talk July 22, 2004 for more detailed explanation

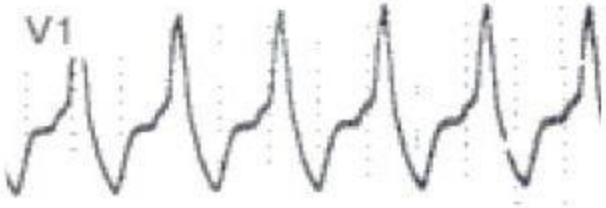
Orthodromic Narrow Tachycardia

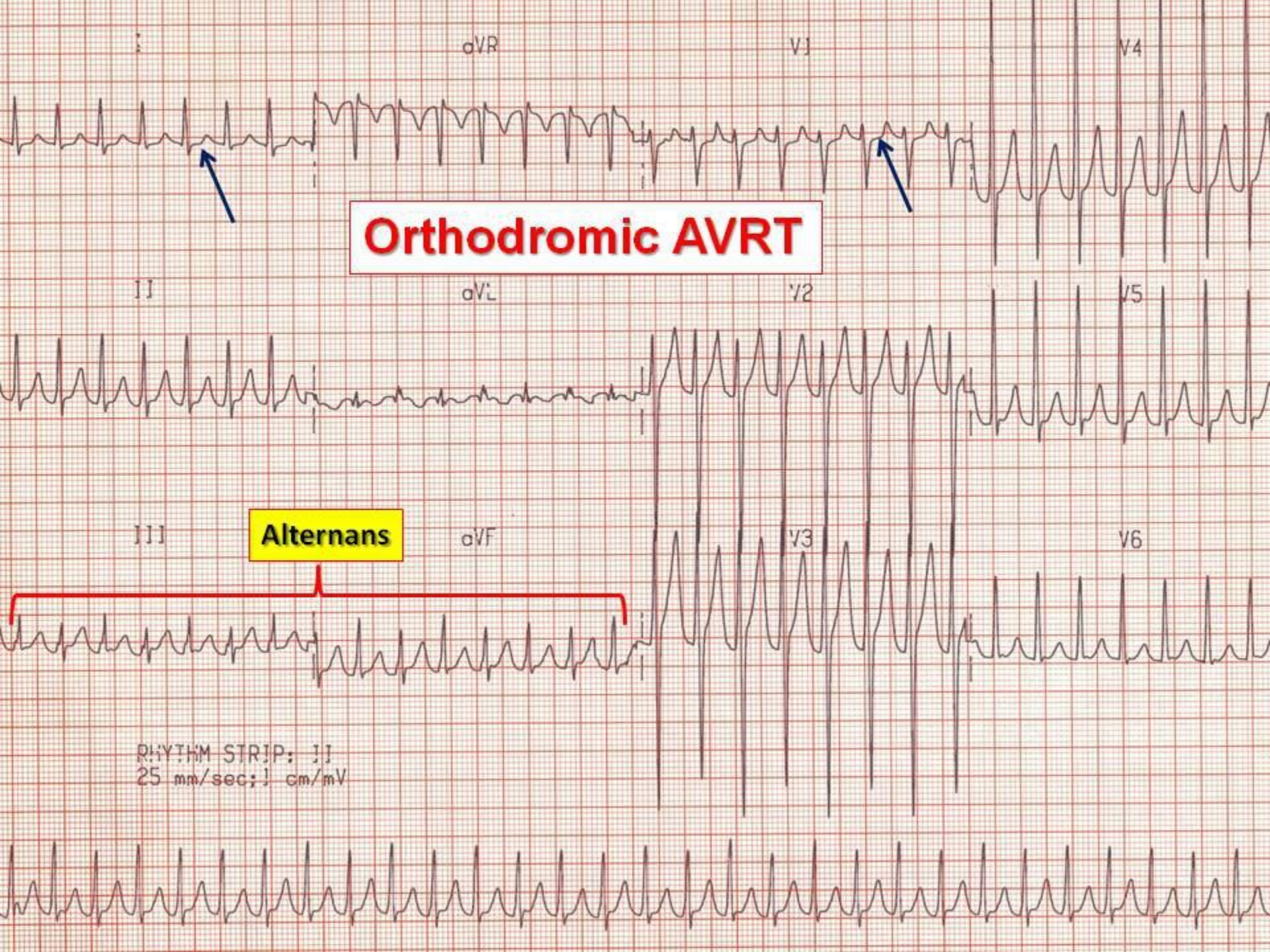


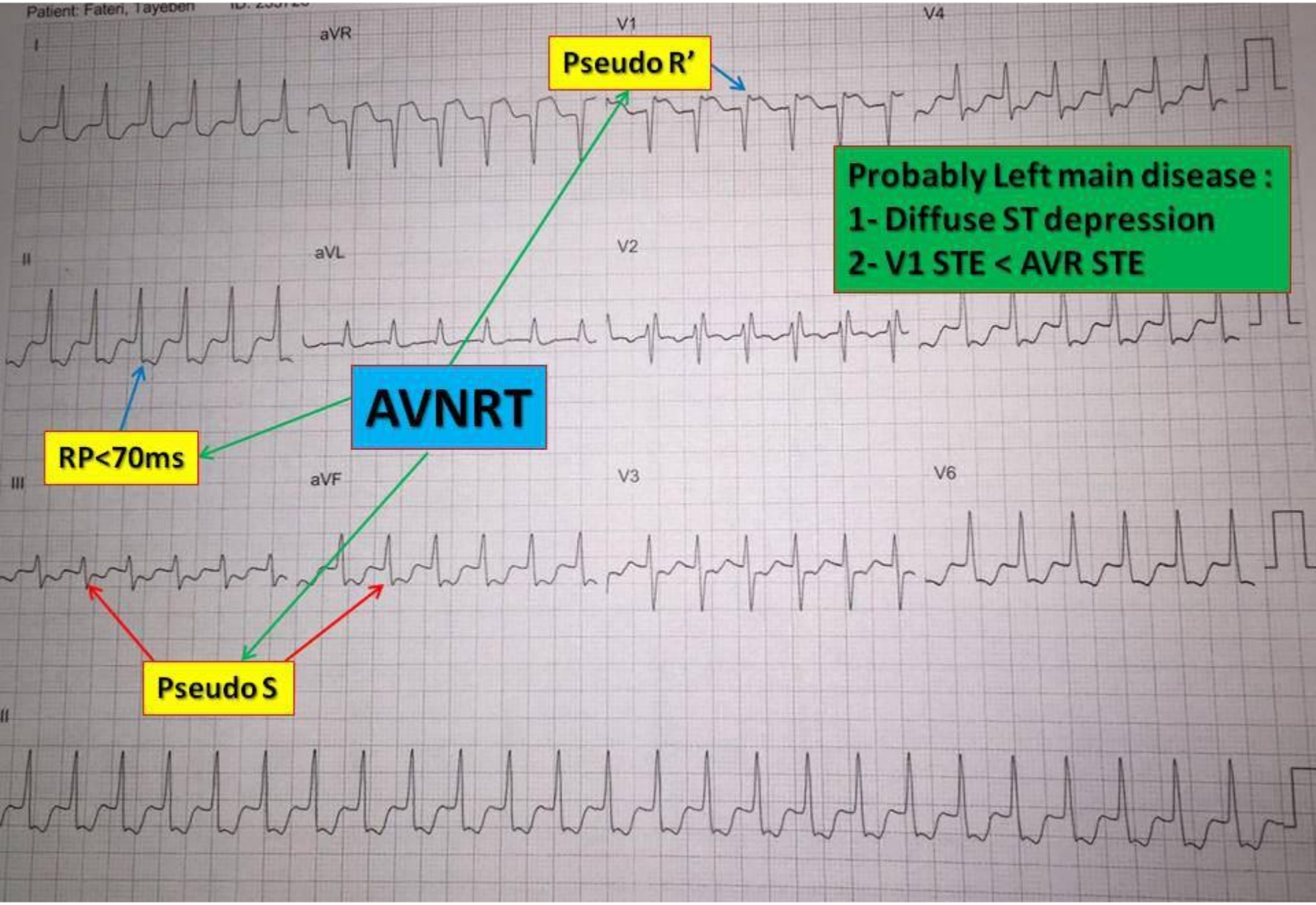


Antidromic Wide Tachycardia

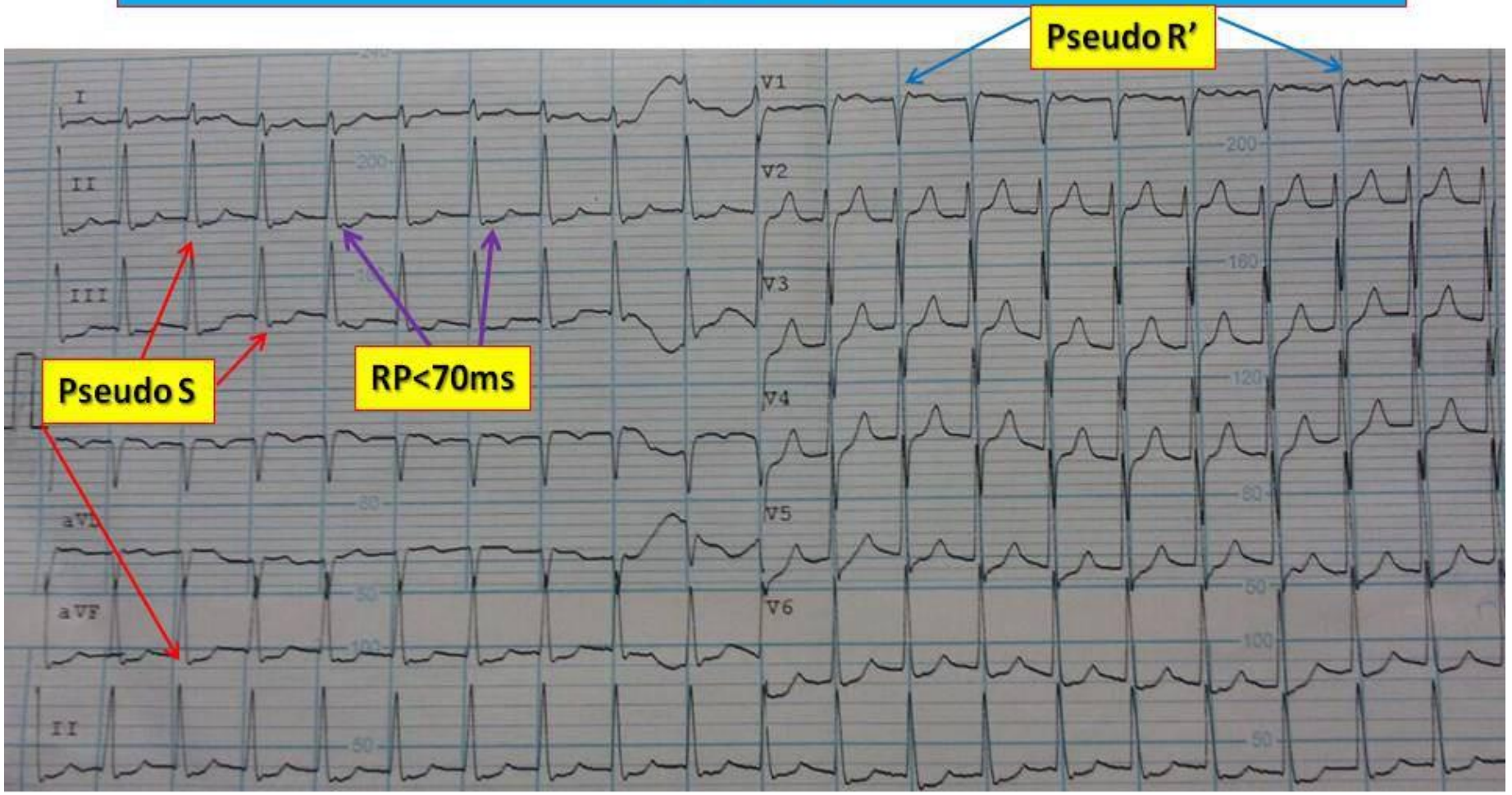




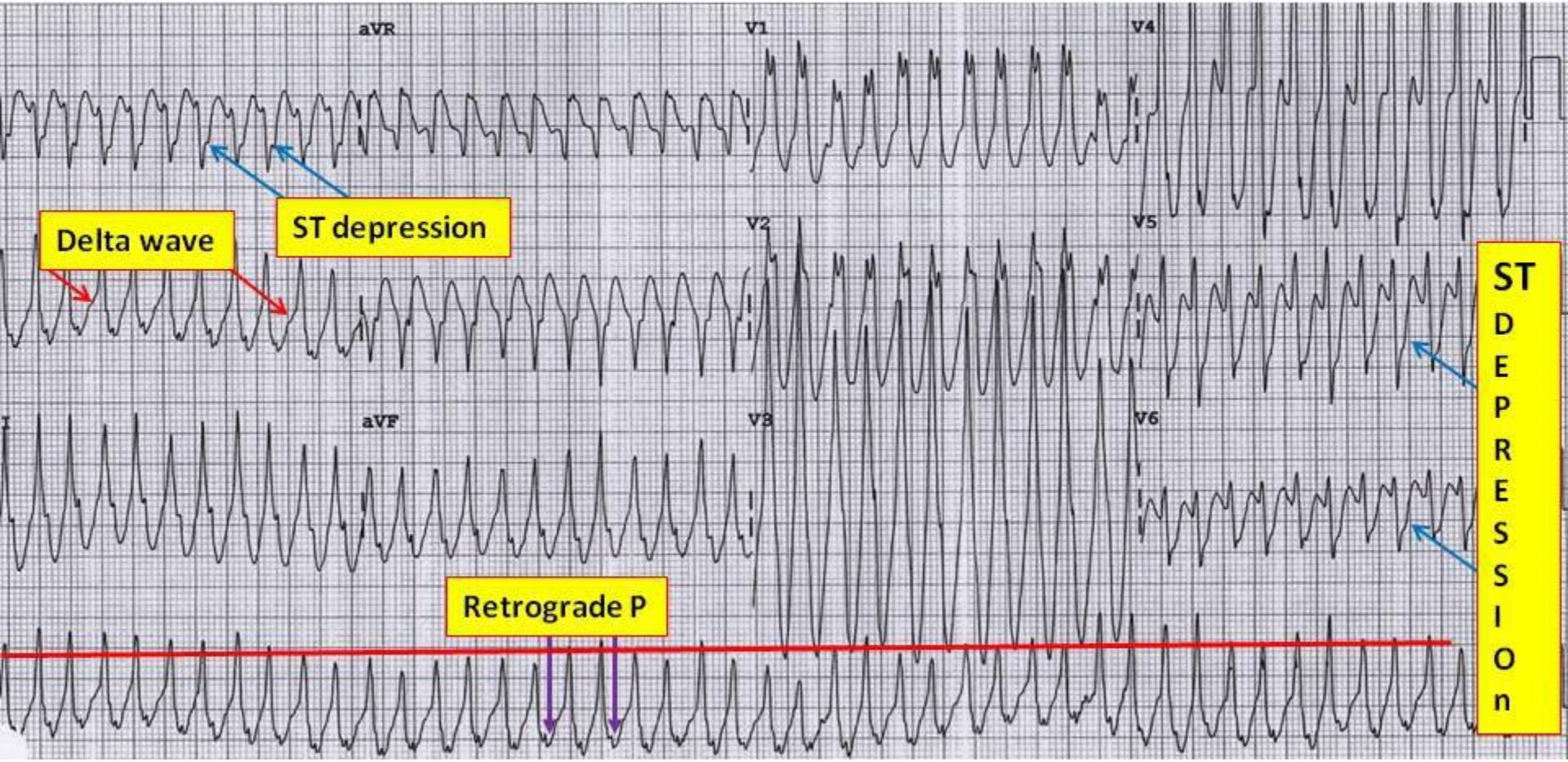

aVR
V1
V4
Orthodromic AVRT
II
aVL
V2
V5
III
Alternans
aVF
V3
V6
RHYTHM STRIP: II
25 mm/sec; 1 cm/mV


Patient: Fateri, Tayeben
I
aVR
V1
V4
Pseudo R'
Probably Left main disease :
1- Diffuse ST depression
2- V1 STE < AVR STE
II
aVL
V2
AVNRT
RP<70ms
III
aVF
V3
V6
Pseudo S
II

Atrioventricular nodal re-entry tachycardia
AVNRT



Atrioventricular re-entry tachycardia
AVRT



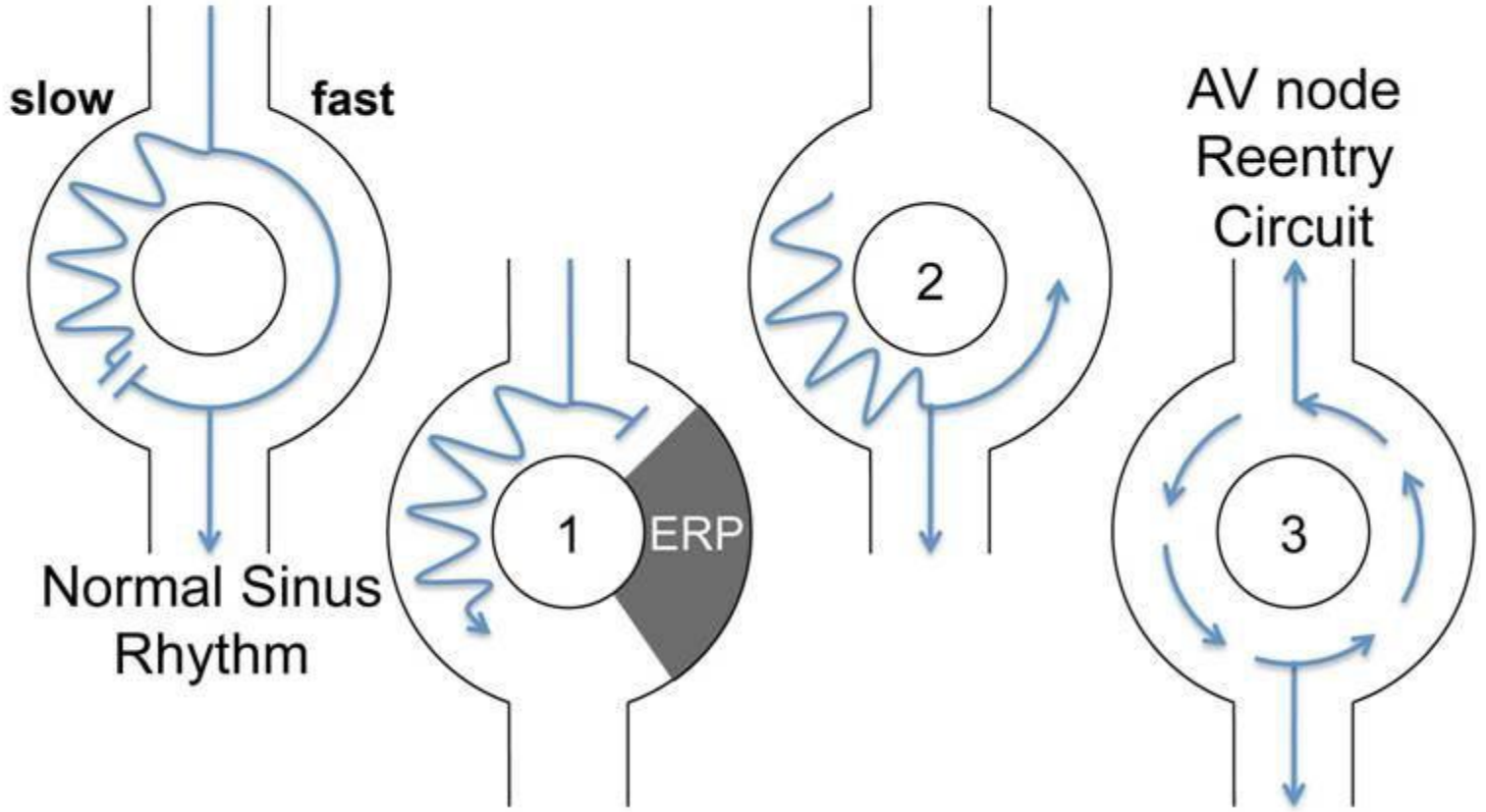
ECG Findings AVNRT

- Regular tachycardia ~140-280 bpm.
- QRS complexes usually narrow (< 120 ms)
- ST-segment depression may be seen with or without CAD.
- QRS alternans.
- P waves if visible retrograde in II, III, AVF.
- P waves may be visible after the QRS complex, or very rarely visible before the QRS complex.
- Pseudo R' wave may be seen in V1 or V2.
- Pseudo S waves may be seen in leads II, III , AVF.

Mechanism of AVNRT

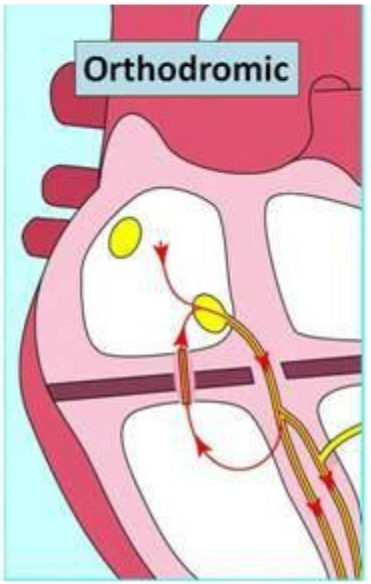
The **slow pathway** (*alpha*)

The **fast pathway** (*beta*)



ECG finding of AVRT (orthodromic)

- Rate usually 200 – 300 bpm
- P waves may be hidden in QRS complex or retrograde
- QRS Complex usually <120 ms unless existing bundle branch block
- QRS Alternans
- T wave inversion common
- ST segment depression



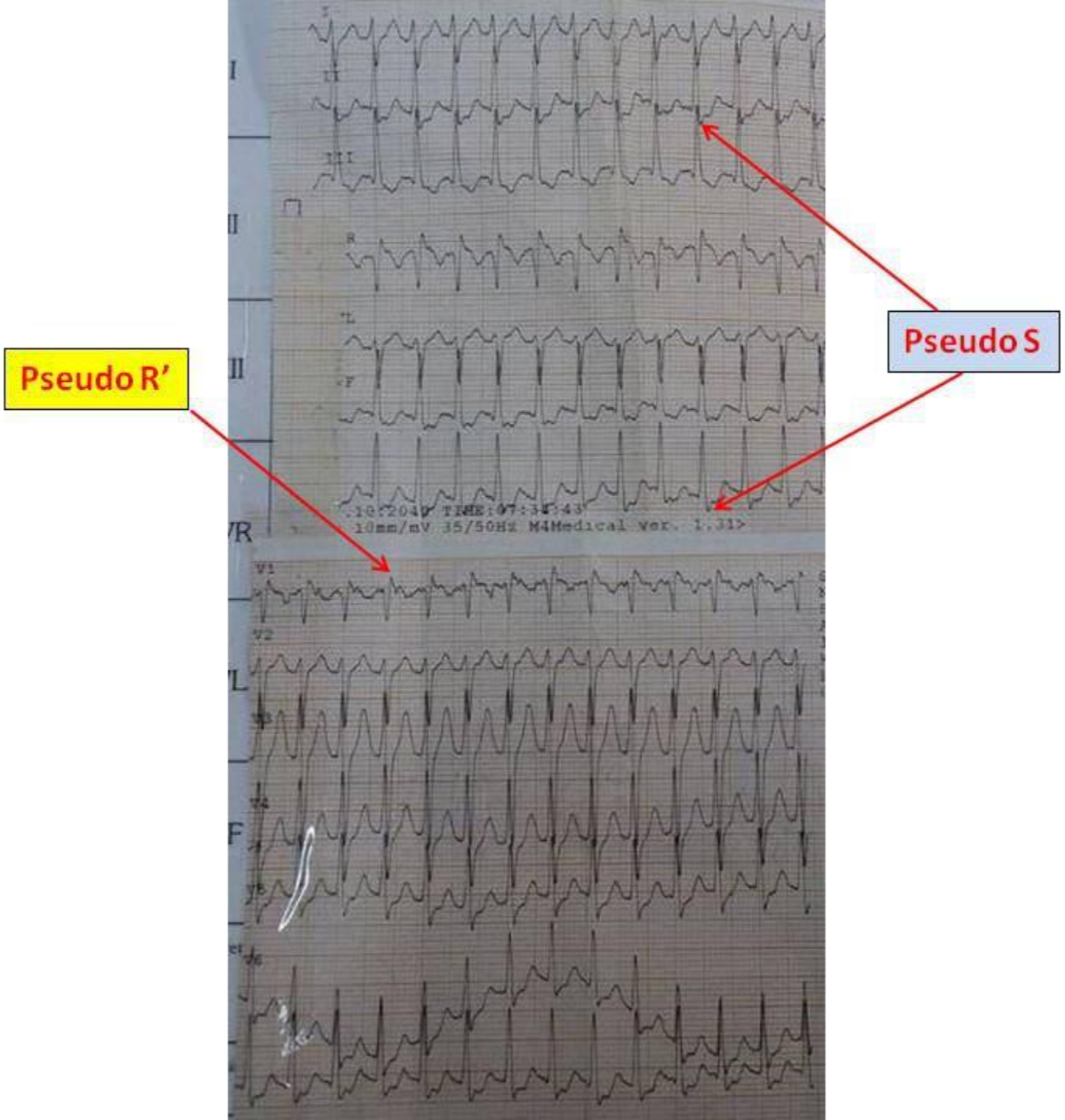
Treatment of orthodromic AVRT

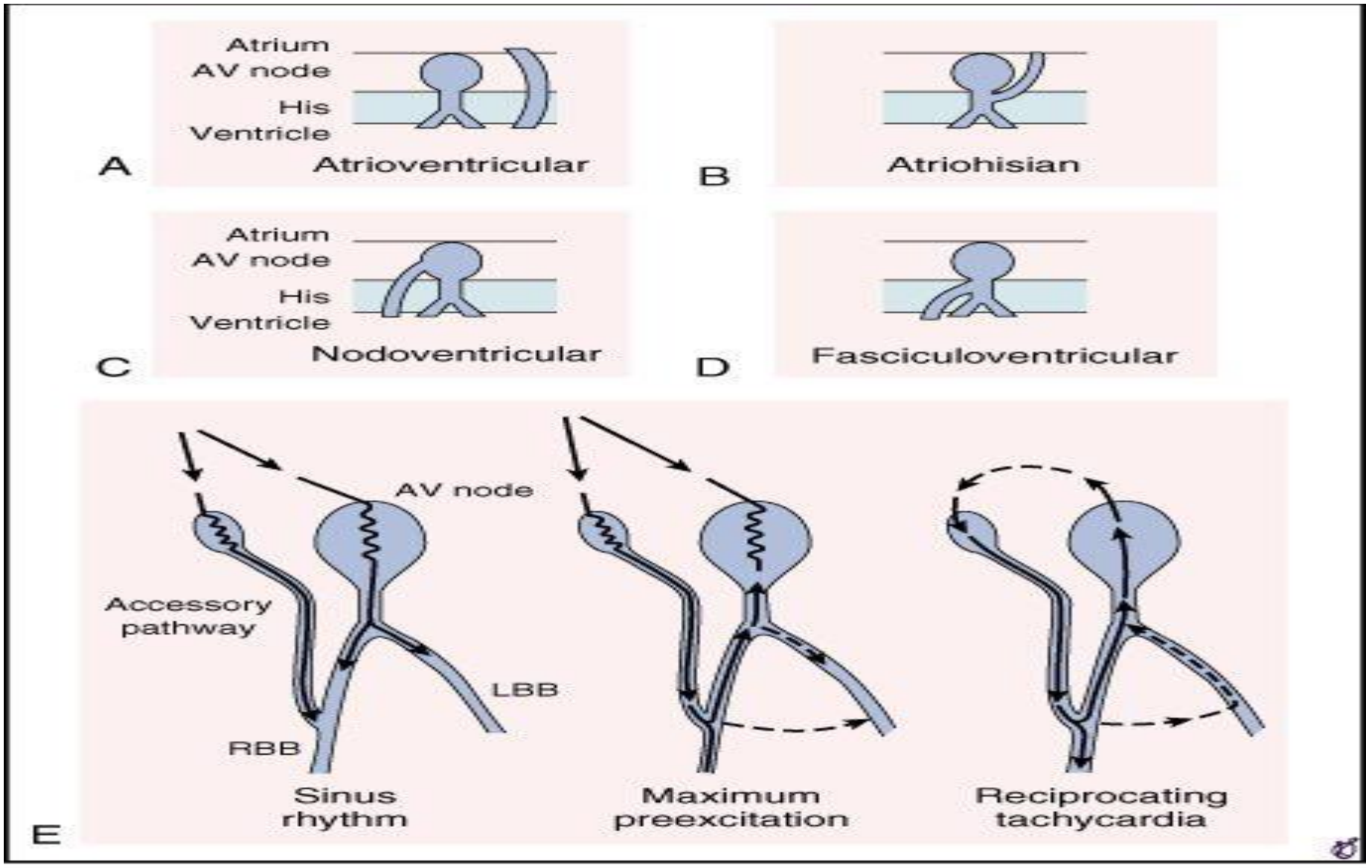
- Treatment of AVRT is based on the presence of hemodynamic instability e.g. hypotension, altered mental state, or pulmonary edema.
- In hemodynamic stable
  - vagal maneuvers
  - adenosine
  - calcium-channel blockers
  - DC Cardioversion if non-responsive to medical therapy.
- In hemodynamic unstable , urgent synchronized DC Cardioversion.

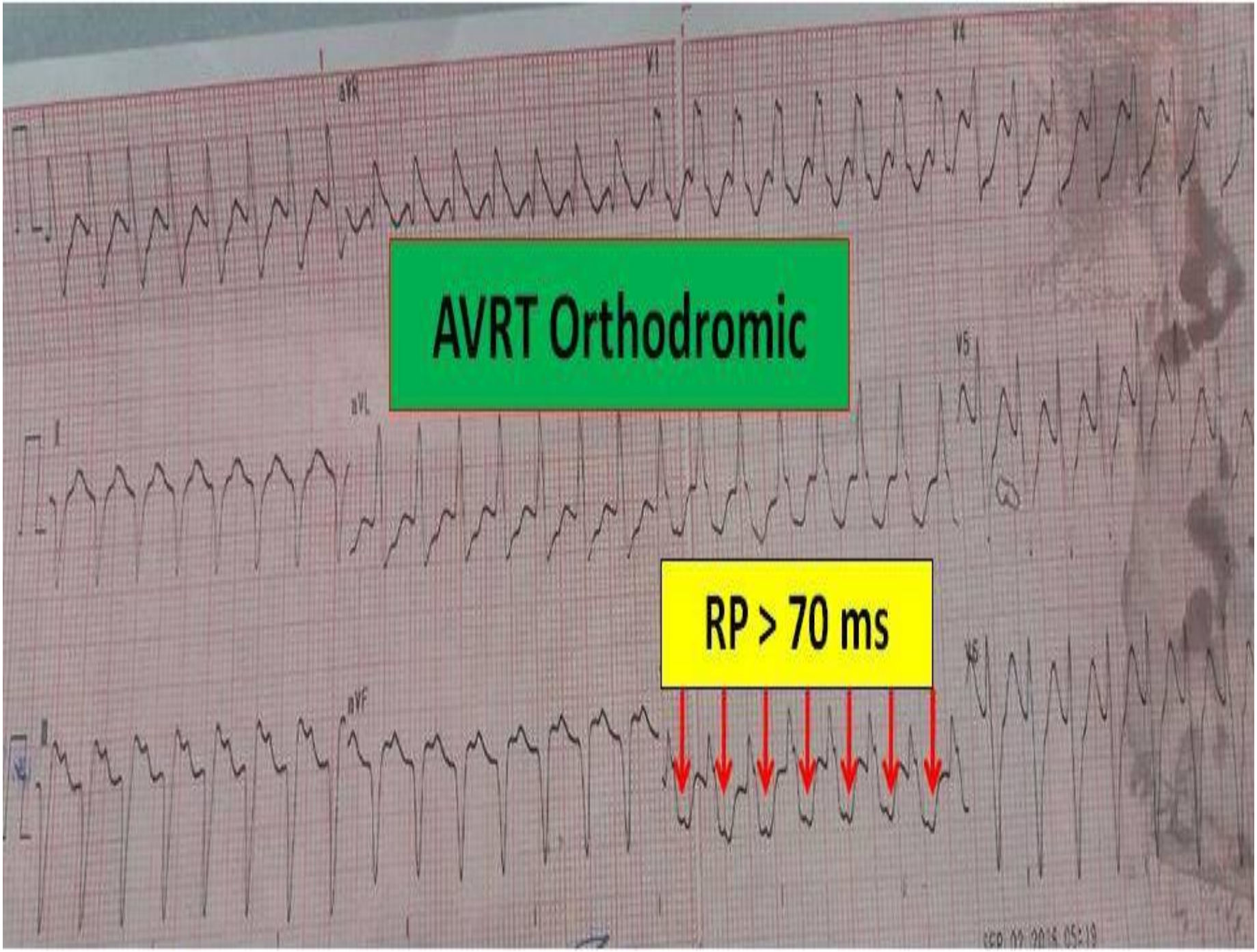
AVNRT ECG Recognition

- Regular or irregular because of varying conduction through the AV node.
- Rate: 170-250 bpm
- Conduction ratio: usually 1:1, uncommonly 2:1
- Typical:
  - The retrograde P wave is seen within, or in close proximity to the terminal portion of the QRS complex (Short RP)
  - Pseudo s wave
  - Presence of a notch in lead aVL is a sensitive and specific predictor of a diagnosis of AVNRT*
- Atypical:
  - The retrograde P wave occurs late, within or following the T wave (Long RP).

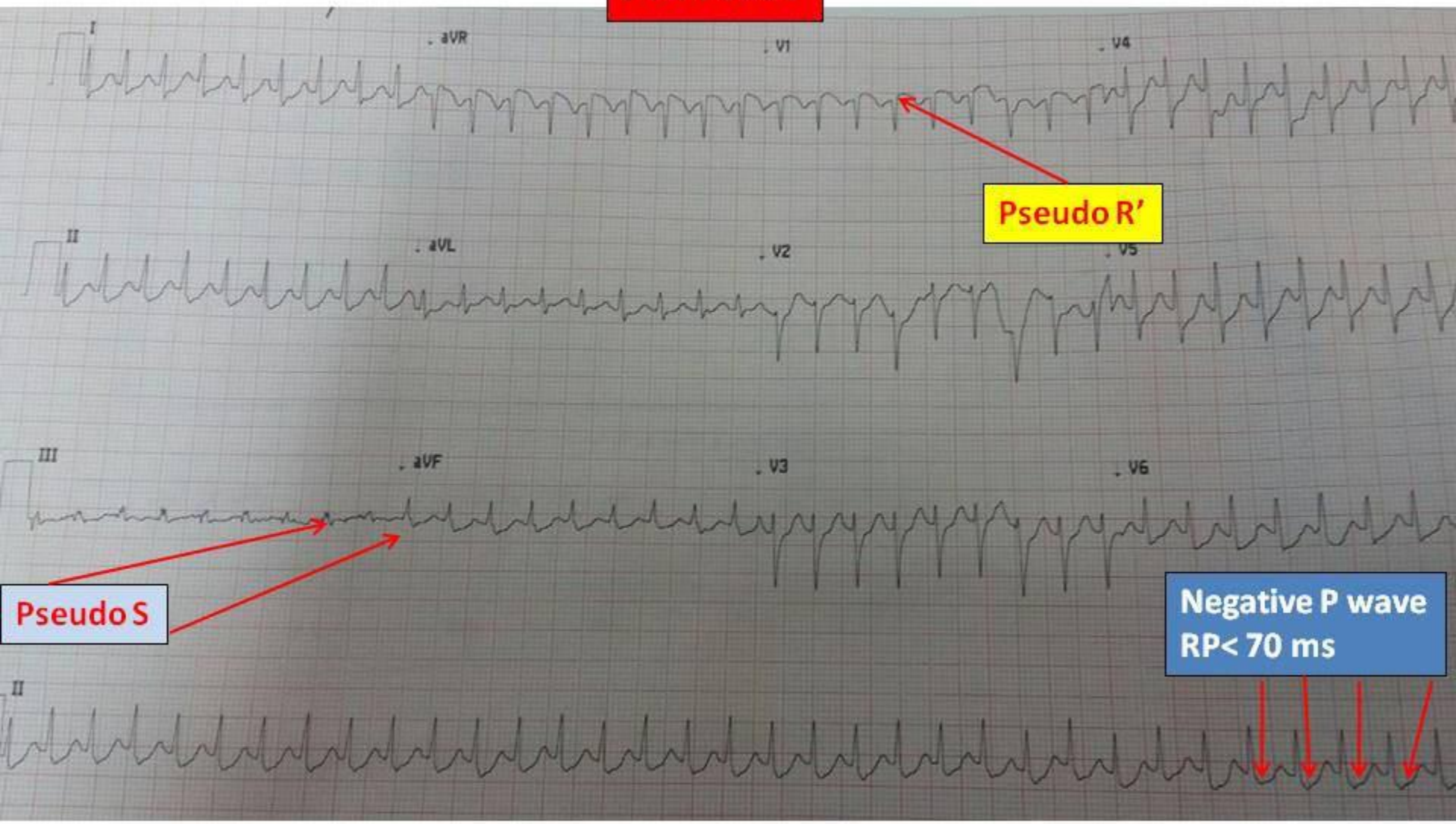
*Utility of the aVL lead in the electrocardiographic diagnosis of atrioventricular node reentrant tachycardia. Darío Di Toro, et al. Europace (2009) 11, 944–948


Pseudo R'
Pseudo S




AVRT Orthodromic
RP > 70 ms

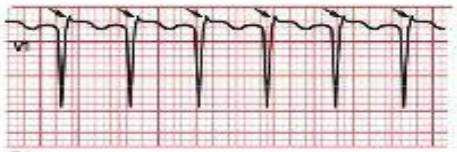
AVNRT

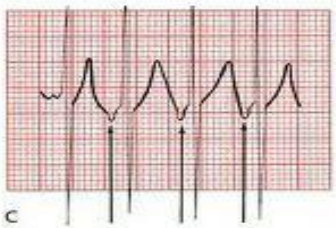

I
aVR
V1
V4
Pseudo R'
II
aVL
V2
V5
III
aVF
V3
V6
Pseudo S
Negative P wave
RP< 70 ms
II

✓ در این Box، خلاصه‌ای از یافته‌های ECG در Typical AVNRT را با هم مرور می‌کنیم.

ECG Findings:

1. ابتدا با دیدن یک تاکی‌کاردی کاملاً منظم – با Narrow QRS (کمتر از 3 خانه کوچک) – که سرعتی معمولاً بین 150 تا 250 b/min دارد؛ به AVNRT مشکوک می‌شویم.
2. **حال به دنبال موج P می‌گردیم تا ببینیم موج P در ECG قابل تشخیص هست یا نه؛ که در اکثریت موارد هم موج P در ECG مشاهده نمی‌کنیم.**
3. امّا اگر موج P قابل رؤیت باشد؛ (در 30 درصد موارد)، به دو صورت می‌توانیم آن را ببینیم:
   a. **در حالت شایع‌تر** یک دندانه یا بریدگی (Notching) کوچک در قسمت انتهایی QRS در لید V_1 می‌بینیم (Pseudo r' wave) که سبب بوجود آمدن الگوی **RSR'** در لید V_1 و یا **Pseudo S wave** در لیدهای تحتانی **II – III – aVF** می‌شود.
   b. **به صورت غیر شایع** ممکنست موج P بلافاصله قبل یا بلافاصله بعد از QRS دیده شود.
4. به طور شایعی ممکنست بویژه در Rate بالا، **ST Depression** ببینیم؛ که لزوماً نشاندهندهٔ ایسکمی میوکارد نیست. (نویسنده: بویژه در فرد جوان بدون مشکل زمینه‌ای)
5. باز هم به طور شایعی ممکنست بعد از خاتمهٔ AVNRT، در نوار قلب **Inverted T wave** در لیدهای **Anterior** و **Inferior** ببینیم که باز هم نشاندهندهٔ CAD نیست.





امّا اگر مانند ECG بیمار اوّل، ST Depression مشاهده کردید، به اشتباه نیفتید...

- در AVNRT با سرعت بالا، بطور شایع ممکنست ST Depression دیده شود، حتّی بدون وجود CAD و وجود آن لزوماً نشاندهندهٔ ایسکمی میوکارد نیست. (نویسنده: بویژه در فرد جوان و بدون مشکل (مینه‌ای)
- در 25% تا 50% بیماران، Significant ST Depression در حین تاکی‌کاردی در AVNRT دیده می‌شود که لزوماً پیشگو کنندهٔ ایسکمی نیست.

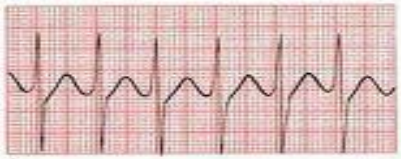
ممکنست بعد از خاتمهٔ اپیزود حمله T wave Inversion هم مشاهده شود...

- در حدود 40 % بیماران بعد از خاتمهٔ اپیزود حمله Newly T wave Inversion معمولاً در لیدهای Anterior و Inferior دیده می‌شود. (T wave Inversion After Termination)
- این T inversion ممکنست بلافاصله بعد از خاتمهٔ تاکی‌کاردی دیده شود، یا تا 6 ساعت بعد از خاتمهٔ آن ظهور پیدا کند و تا مدّت زمان متغیری باقی بماند. (به طور متوسط 34 ساعت)
- این یافته به Rate و یا Duration تاکی‌کاردی بستگی ندارد.
- گرچه این T Inversion به خاطر رپولاریزاسیون غیرنرمال ظاهر می‌شود، ولی بعلّت بیماری عروق کرونری نیست، بلکه ممکنست بعلّت تغییرات غلظت یونی در نتیجهٔ Rate بالا باشد.

معمولاً P دیده نمی‌شود؛ چونکه توسط QRS پوشیده شده است. (شکل A)

بسیاری اوقات موج P توسط QRS پوشیده شده و به هیچ عنوان در ECG دیده نمی‌شود.

- اگر هم موج P دیده شود، معمولاً بصورت inverted P در لیدهای تمتانی aVF III II مشاهده می‌شود. (چونکه دهلیزها دارند به طور غیرطبیعی و از سمت گره AV بصورت رتروگرید دپولاریزه می‌شوند).



NO P wave

A

امـــــــا بهترین شانس شما برای دیدن موج P چیست؟

- بهترین شانس، دیدن Pseudo R' wave در لید V_1 است.
- به این معنا که ممکنست که یک دندانه یا بریدگی (Notching) کوچک در قسمت انتهایی QRS ببینیم که نشاندهندهٔ Superimposed شدن P روی QRS است. (Pseudo R' wave)
- این حالت سبب بوجود آمدن Small R wave در قسمت انتهایی کمپلکس QRS و تولید Pseudo R' wave و الگویی به نام RSR' در لید V_1 و همچنین Pseudo S wave در لیدهای تمتانی (II – III – aVF) می‌شود. (شکل B)

وجود الگوی **RSR'** در لید V_1 یا وجود **Pseudo S wave** در لیدهای **II – III – aVF** به نفع Typical AVNRT می‌باشد.

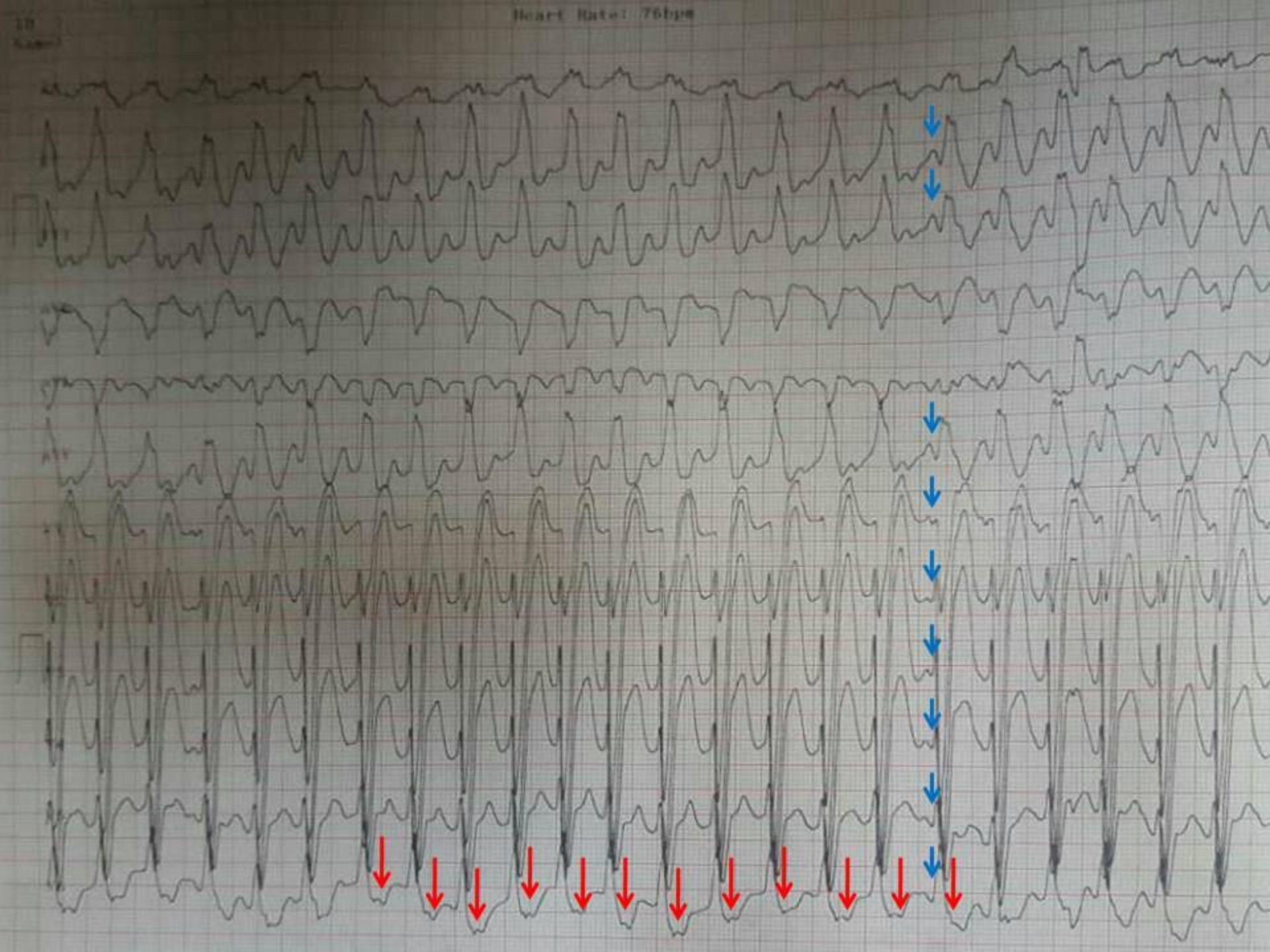
البتّه RSR' Pattern گاهی فقط با مقایسه با ریتم سینوسی قبلی بیمار قابل تشخیص است.

- بطور ناشایع‌تری، ممکنست موج P معکوس، بلافاصله قبل یا بلافاصله بعد از QRS دیده شود. (شکل C)

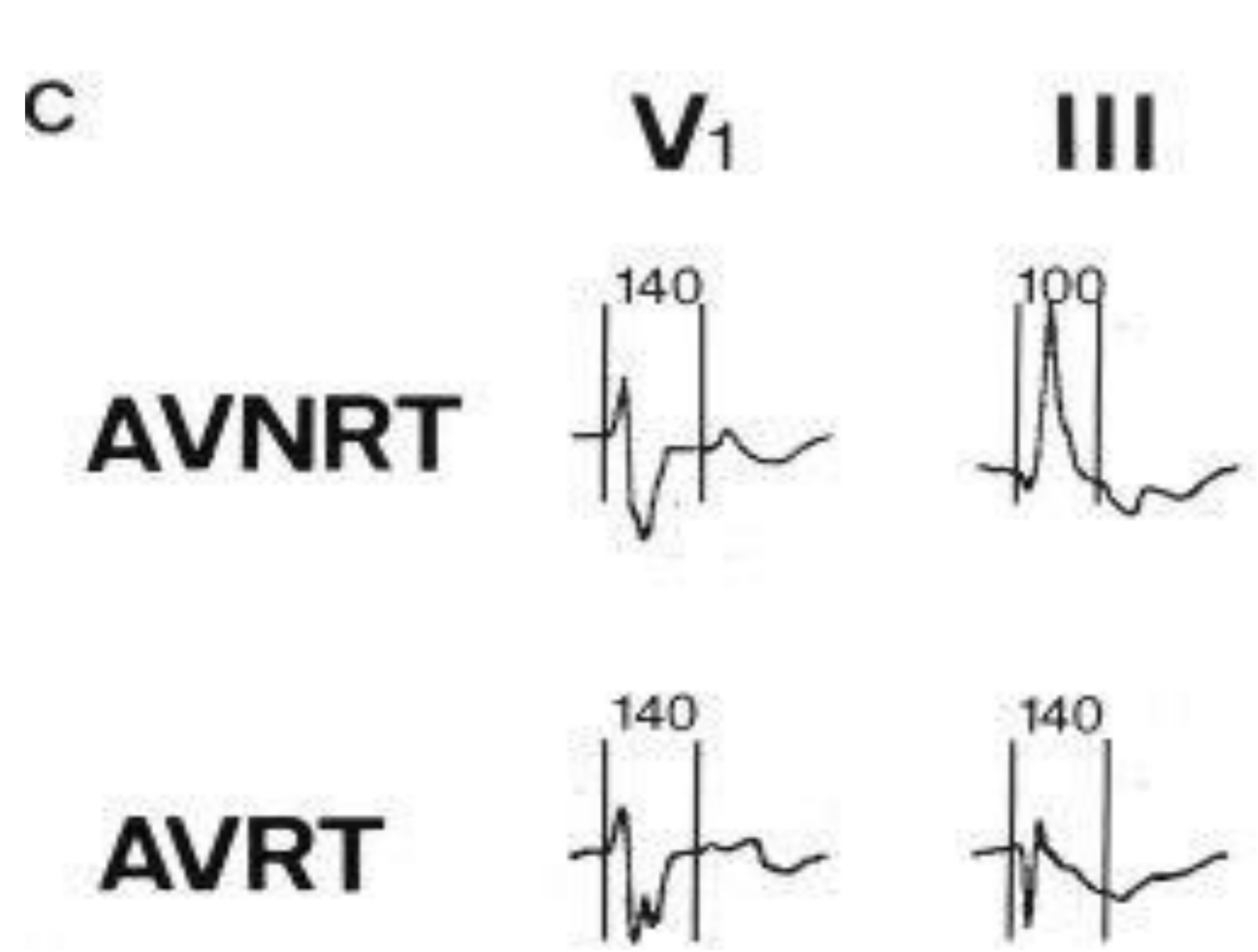
AVNRT ECG Recognition

- Regular or irregular because of varying conduction through the AV node.
- Rate: 170-250 bpm
- Conduction ratio: usually 1:1, uncommonly 2:1
- Typical:
  - The retrograde P wave is seen within, or in close proximity to the terminal portion of the QRS complex (Short RP)
  - Pseudo s wave
  - Presence of a notch in lead aVL is a sensitive and specific predictor of a diagnosis of AVNRT*
- Atypical:
  - The retrograde P wave occurs late, within or following the T wave (Long RP).

*Utility of the aVL lead in the electrocardiographic diagnosis of atrioventricular node reentrant tachycardia. Darío Di Toro, et al. Europace (2009) 11, 944–948


Heart Rate: 76bpm

C



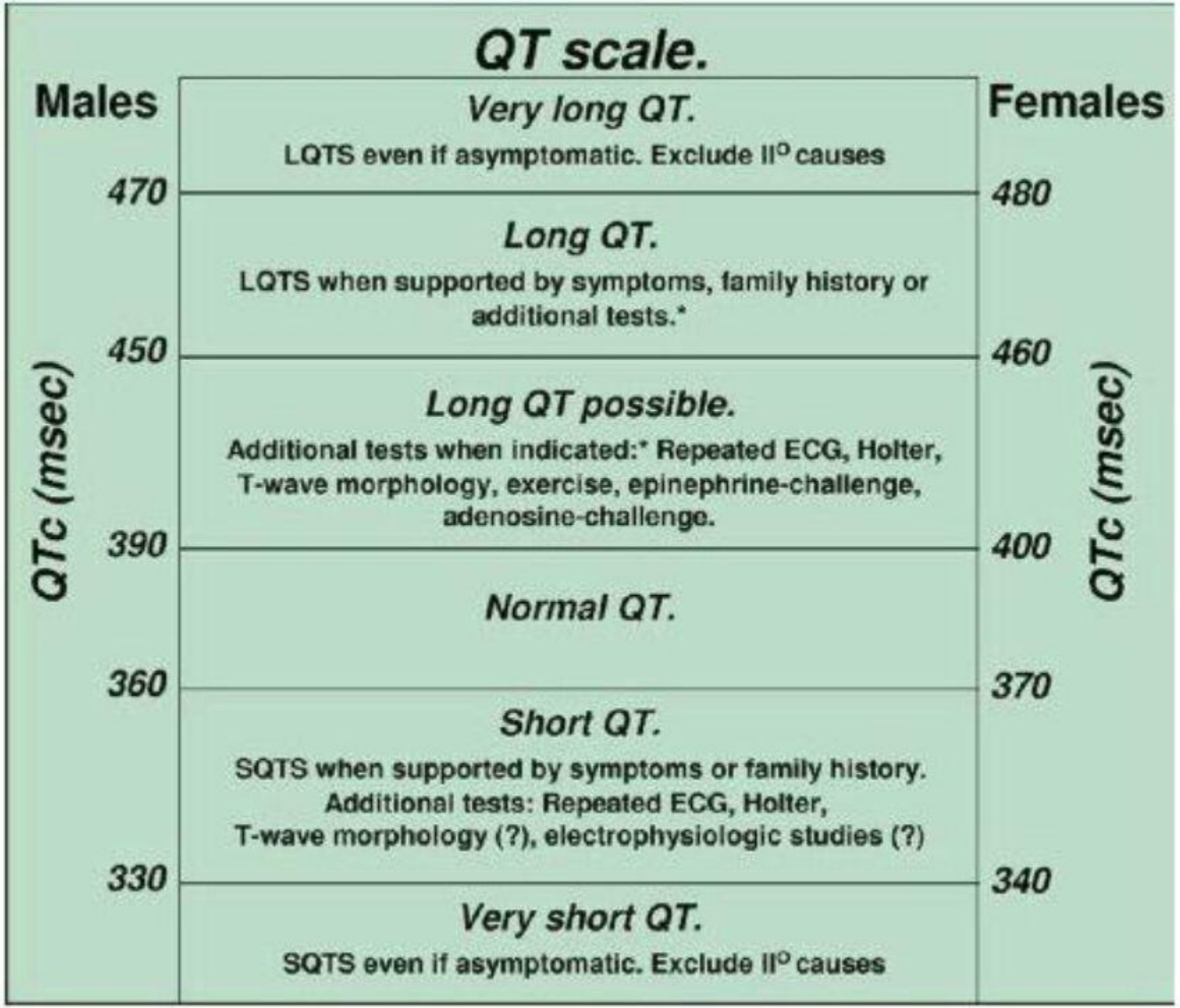
Causes of long QT_C interval

Congenital (at least six genetic mutations identified)

- Romano-Ward syndrome (autosomal dominant)
- Jervell and Lange-Nielsen syndrome (cardiac abnormality - autosomal dominant & associated deafness - autosomal recessive)

Acquired

- drugs
- cardiac pathology (heart failure, ischaemia, myocarditis)
- electrolyte abnormality (hypokalaemia, hypomagnesaemia)
- cerebrovascular disease (subarachnoid haemmorhage, ischaemic stroke)
- severe bradycardia (especially complete heart block)
- hyperthyroidism/hypothyroidism


QT scale.
Males
Females
Very long QT.
LQTS even if asymptomatic. Exclude II° causes
470
480
Long QT.
LQTS when supported by symptoms, family history or additional tests.*
450
460
Long QT possible.
Additional tests when indicated:* Repeated ECG, Holter, T-wave morphology, exercise, epinephrine-challenge, adenosine-challenge.
390
400
Normal QT.
360
370
Short QT.
SQTS when supported by symptoms or family history.
Additional tests: Repeated ECG, Holter,
T-wave morphology (?), electrophysiologic studies (?)
330
340
Very short QT.
SQTS even if asymptomatic. Exclude II° causes
QTc (msec)
QTc (msec)

Long-QT Syndrome

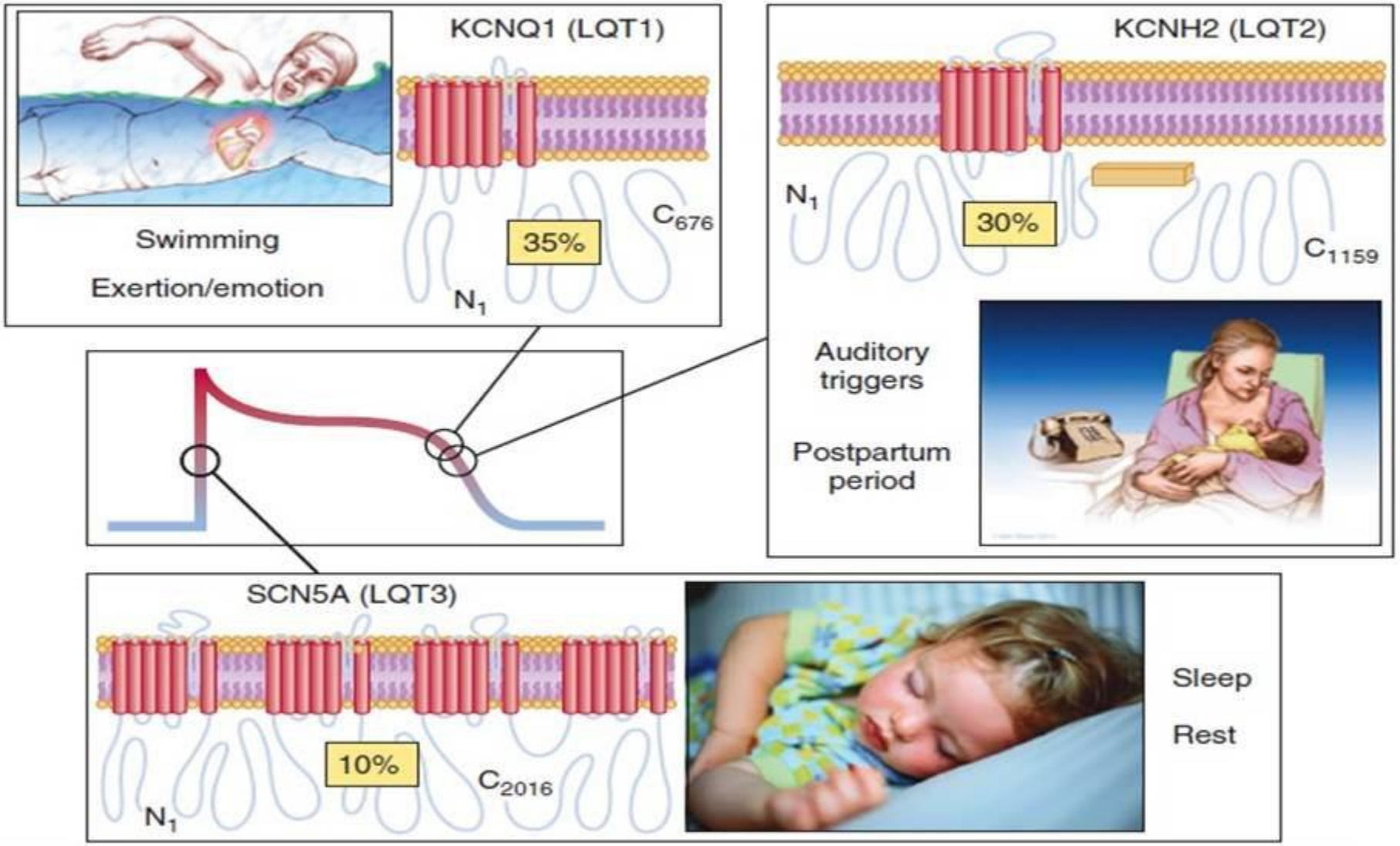
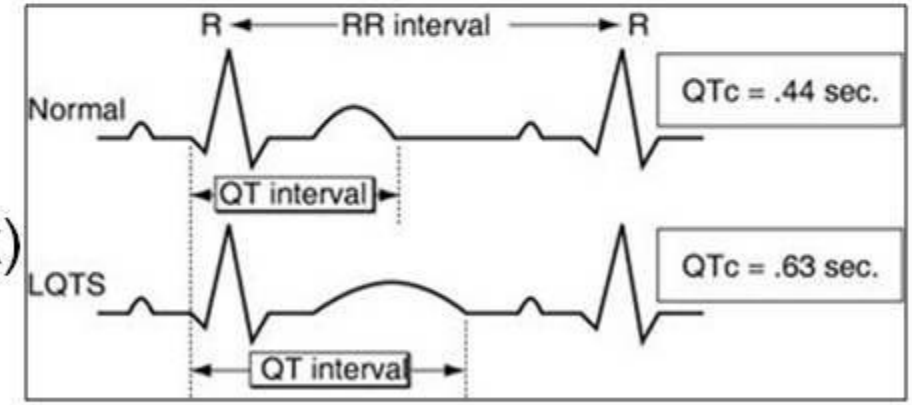


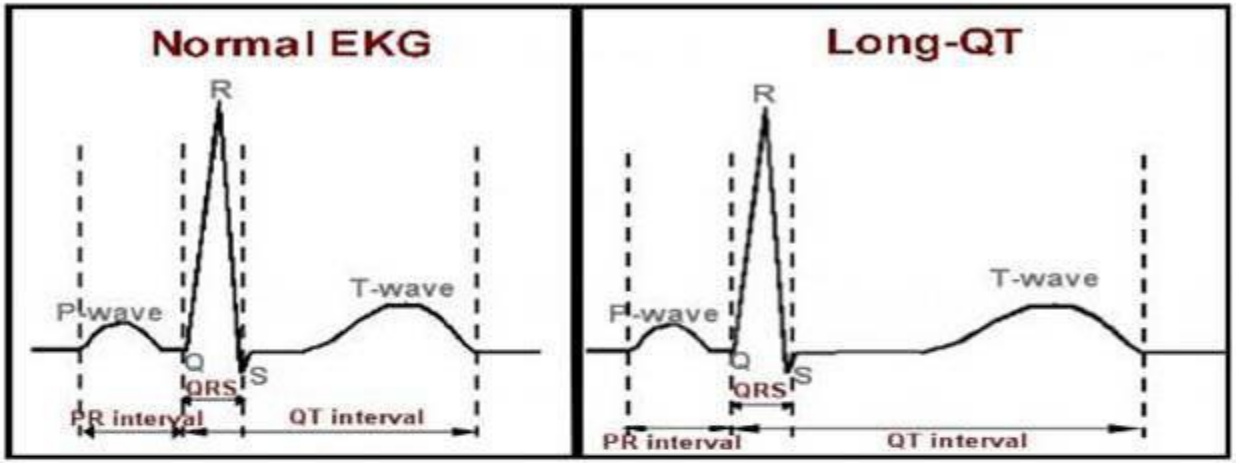
FIGURE 32-3 Genotype-phenotype correlations in LQTS. Seventy-five percent of cases of clinically strong LQTS are due to mutations in three genes (*KCNQ1*, 35%; *KCNH2*, 30%; and *SCN5A*, 10%) encoding for ion channels that are critically responsible for orchestration of the cardiac action potential. Genotype-phenotype correlations have been observed, including swimming/exertion/emotion and LQT1, auditory triggers/postpartum period and LQT2, and sleep/rest and LQT3.

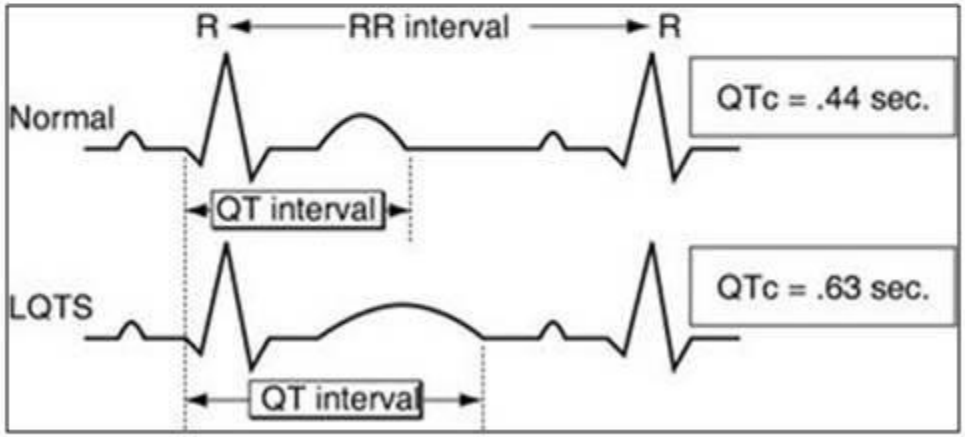
Long-QT Syndrome

- QT prolongation (450- 470 msec)
- increased risk for syncope, seizures, and SCD in the setting of a structurally normal heart and otherwise healthy individual.
- The incidence of LQTS may exceed 1 in 2500 Persons
- This repolarization abnormality is triggers such as:
  - exertion
  - swimming
  - emotion
  - Auditory stimuli (alarm clock)
  - postpartum period

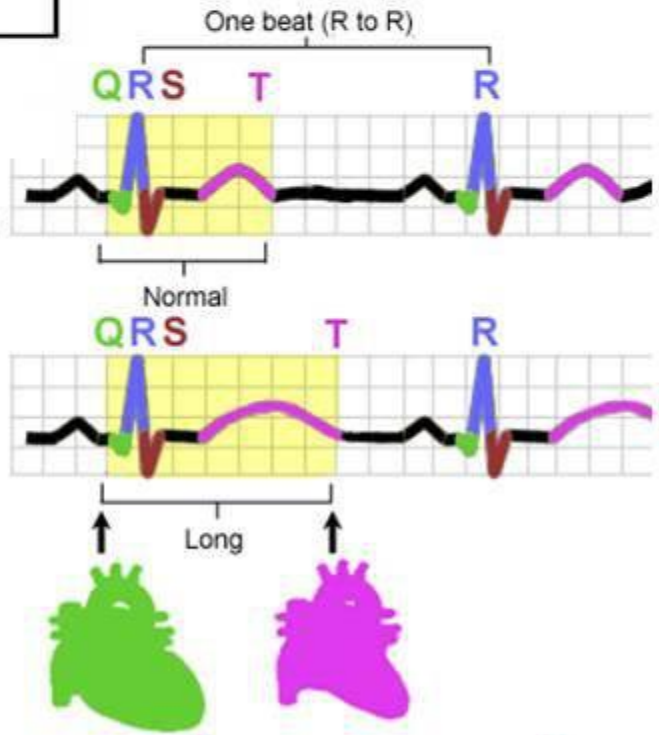


Long-QT Syndrome





Each section of an electrocardiogram (ECG or EKG) is referred to by a letter name: Q, R, S, and T.

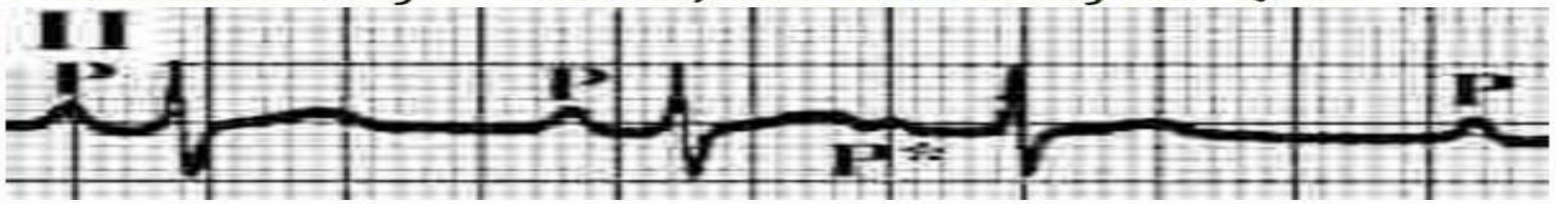


ECG II Rhythms and Traces

Premature Atrial Contraction (PAC)

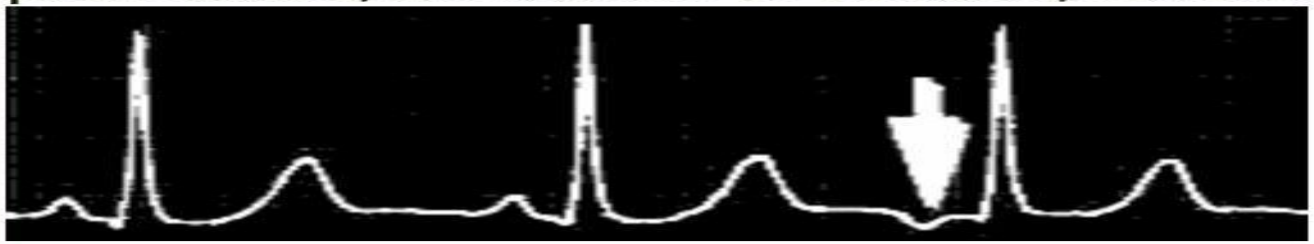
Atrial Premature Depolarization (APD)

Preceded by P wave, followed by nl QRS



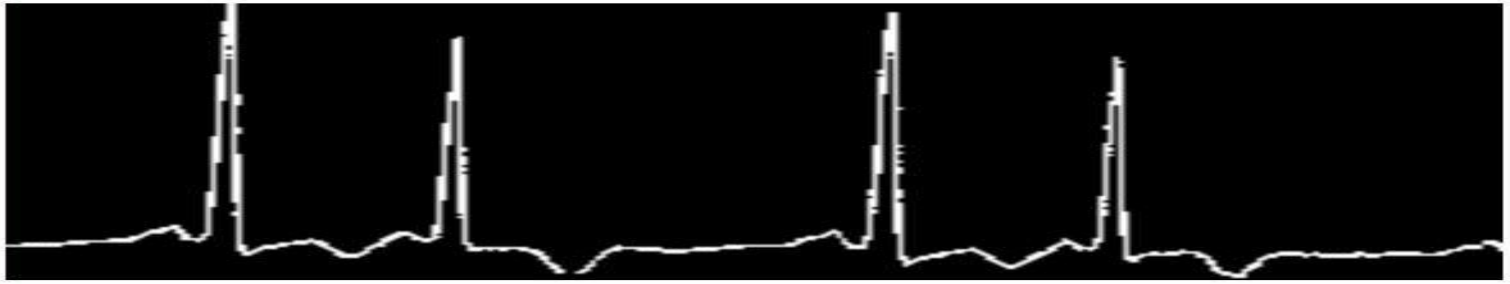
Premature Atrial Contraction

Aberrantly conducted PAC. Bizarre QRC. P wave is premature, abnormal or hidden, followed by nl QRC

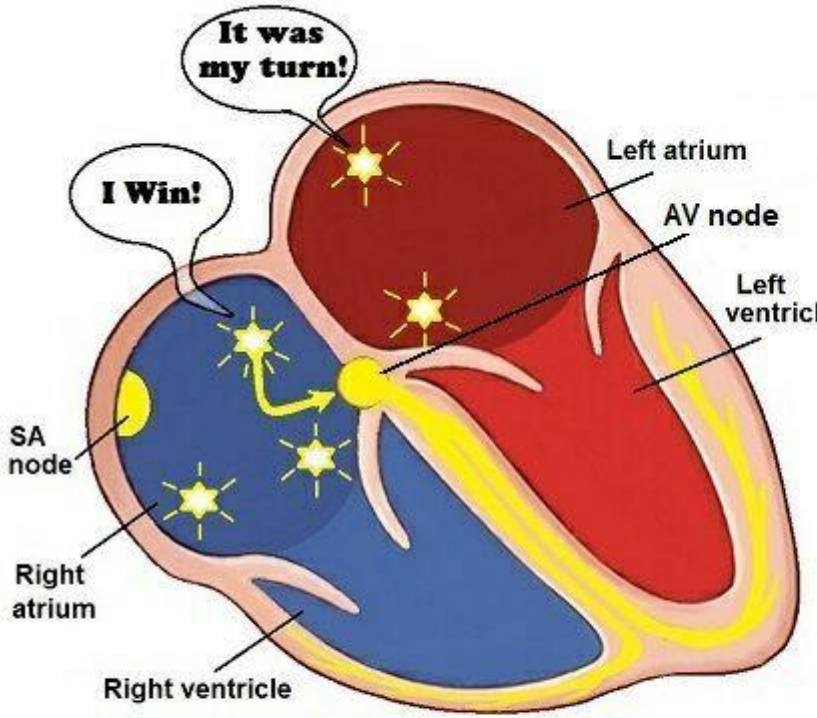


Atrial Bigeminy

(every other beat is a PAC) P wave is premature, abnormal or hidden, irregular rhythm. PR interval <.20s / QRS <.12s

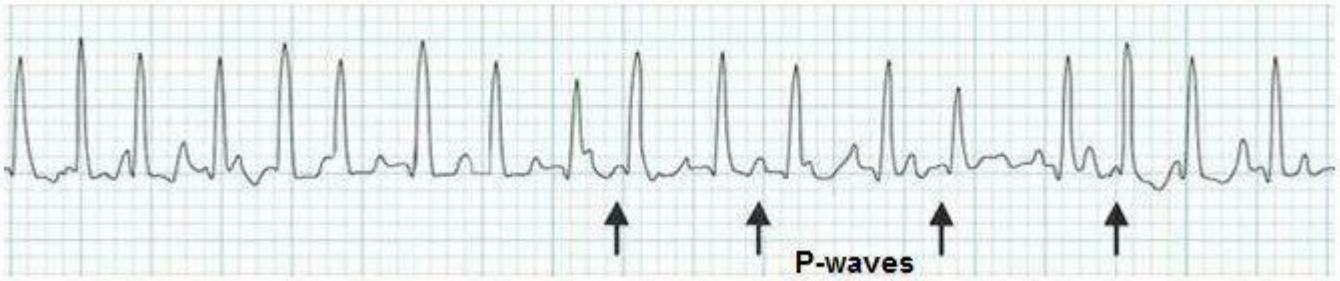


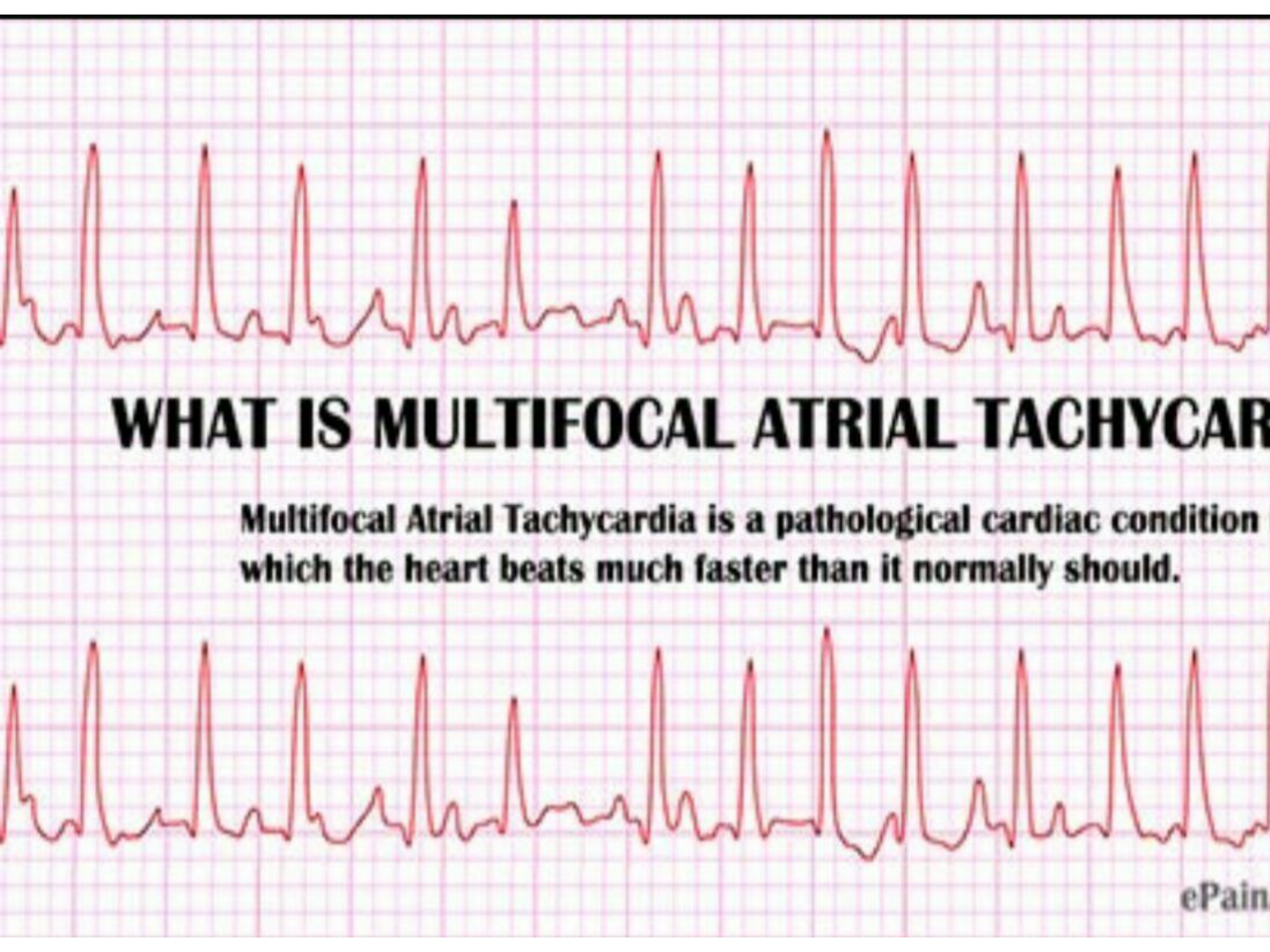
What's up with Multifocal Atrial Tachycardia ?



1. The SA node is not pacing the heart
2. Several groups of excitable cells in the atria compete to pace the heart
3. MAT has at least three or more different shaped P-waves.
4. MAT is an irregular rhythm above 100 bpm
5. MAT has irregular P-R, R-R and P-P intervals

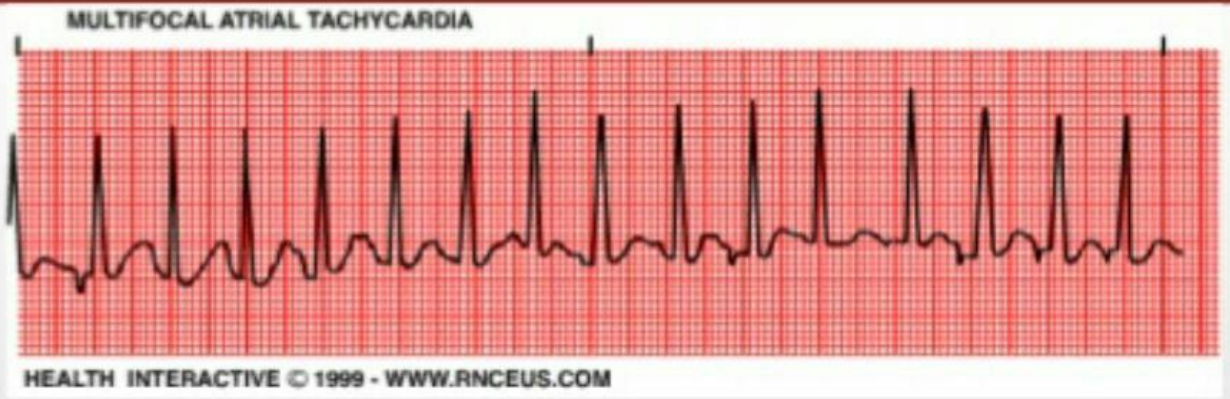
Definition:
A rapid, irregular atrial rhythm arising from multiple ectopic foci within the atria.




WHAT IS MULTIFOCAL ATRIAL TACHYCAR
Multifocal Atrial Tachycardia is a pathological cardiac condition
which the heart beats much faster than it normally should.
ePain

Multifocal Atrial Tachycardia (MAT)

(Rapid Wandering Pacemaker)

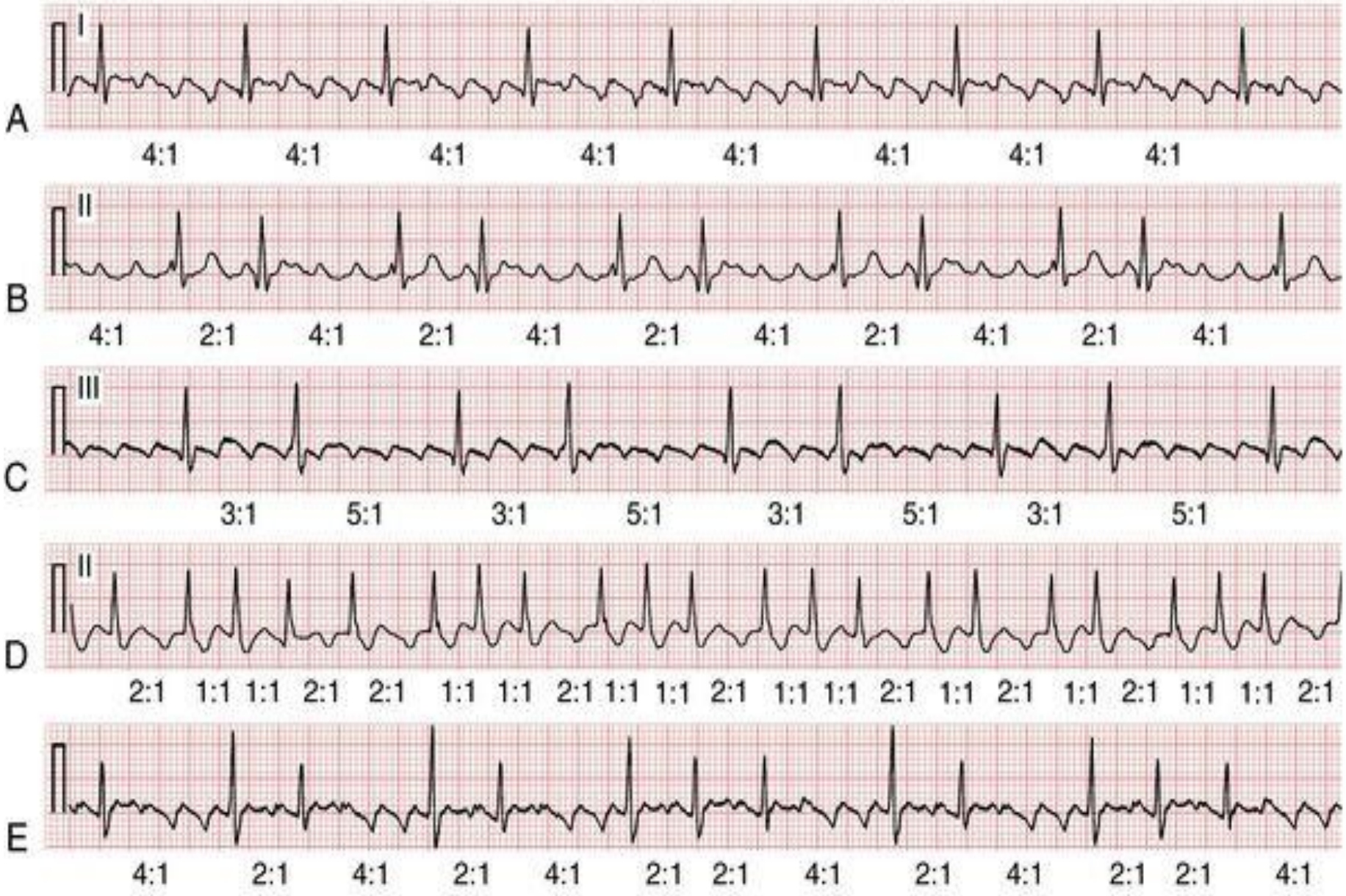


- Similar to wandering pacemaker (≤ 100)
- MAT rate is >100
- Usually due to pulmonary issue
  - COPD
  - Hypoxia, acidotic, intoxicated, etc.
- Often referred to as SVT by EMS
  - Recognize it is a tachycardia and QRS is narrow

📖 فلوتر دهلیزی (Atrial flutter) :

- ✔ نوعی آریتمی منظم دهلیزی با ریت ۲۶۰ تا ۳۰۰ ضربه در دقیقه است .
- ✔ در خط ایزوالکتریک لیدهای تحتانی حالت دندانه.اره.ای (Saw-tootch appearance) ایجاد می شود .
- ✔ اگر از هر ۲ انقباض دهلیـــزی یکی وارد بطن شود (2:1) ریت بطنـــی ۱۳۰ تا ۱۵۰ ضربه در دقیقه است اگر (3:1) باشد ریت بطنی حدود ۷۰ در دقیقه است .

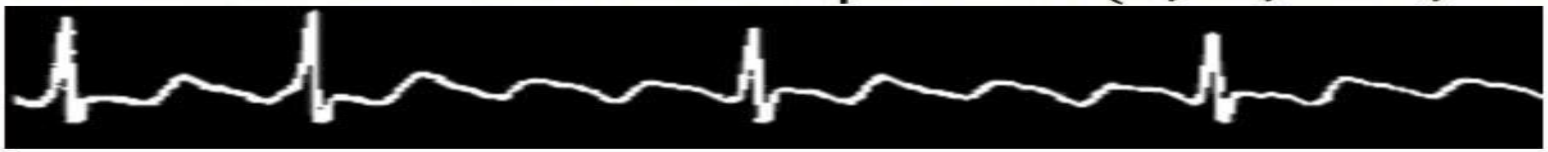
Atrial Flutter with Variable AV Block



ECG III Rhythms and Traces

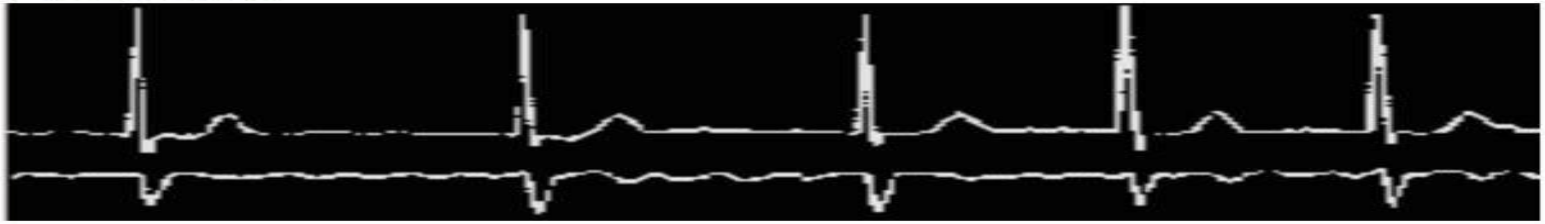
Atrial Flutter

Atrial rate 240-320/min. Ventricular rate varies with AV block 2:1. Saw tooth pattern (II, III, aVF, V1)



Atrial Fibrillation (AF)

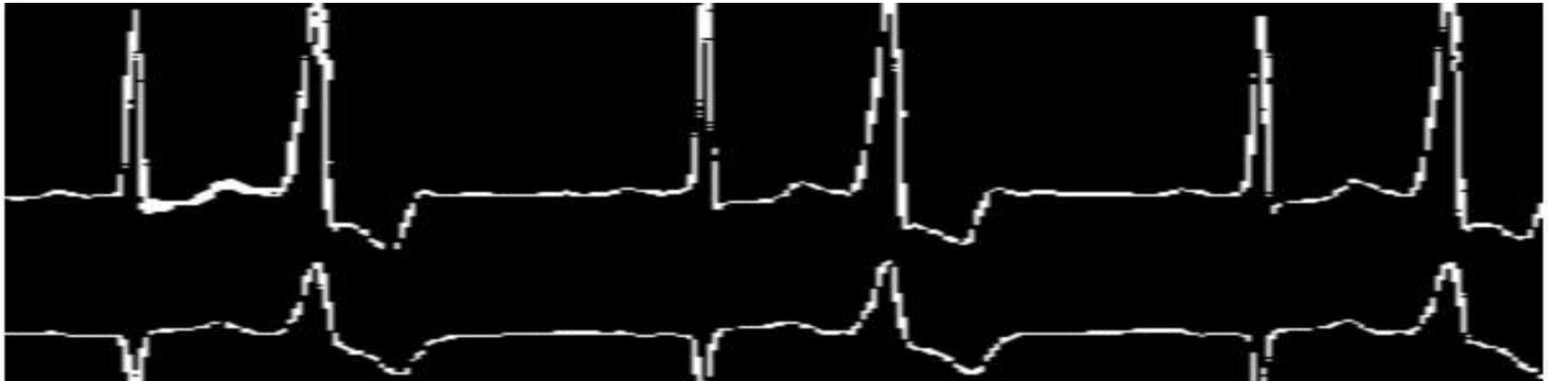
Highly irregular. No P wave. Vent. response: 160-200.min



-geminy

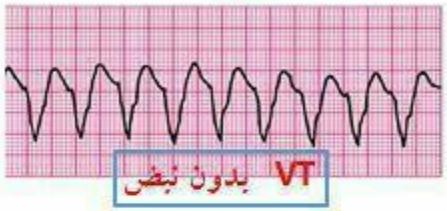
Bigeminy (coupled beat)

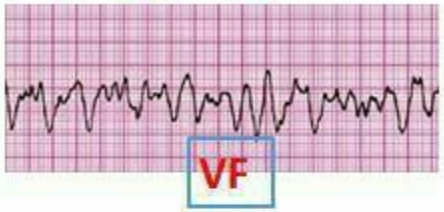
Sinus beat → PVC → PVC.



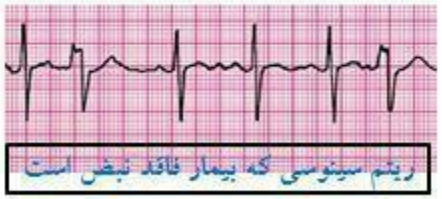
@emt_ems

ریتمهایی که دراحیاءشناخت آنها ضروریست



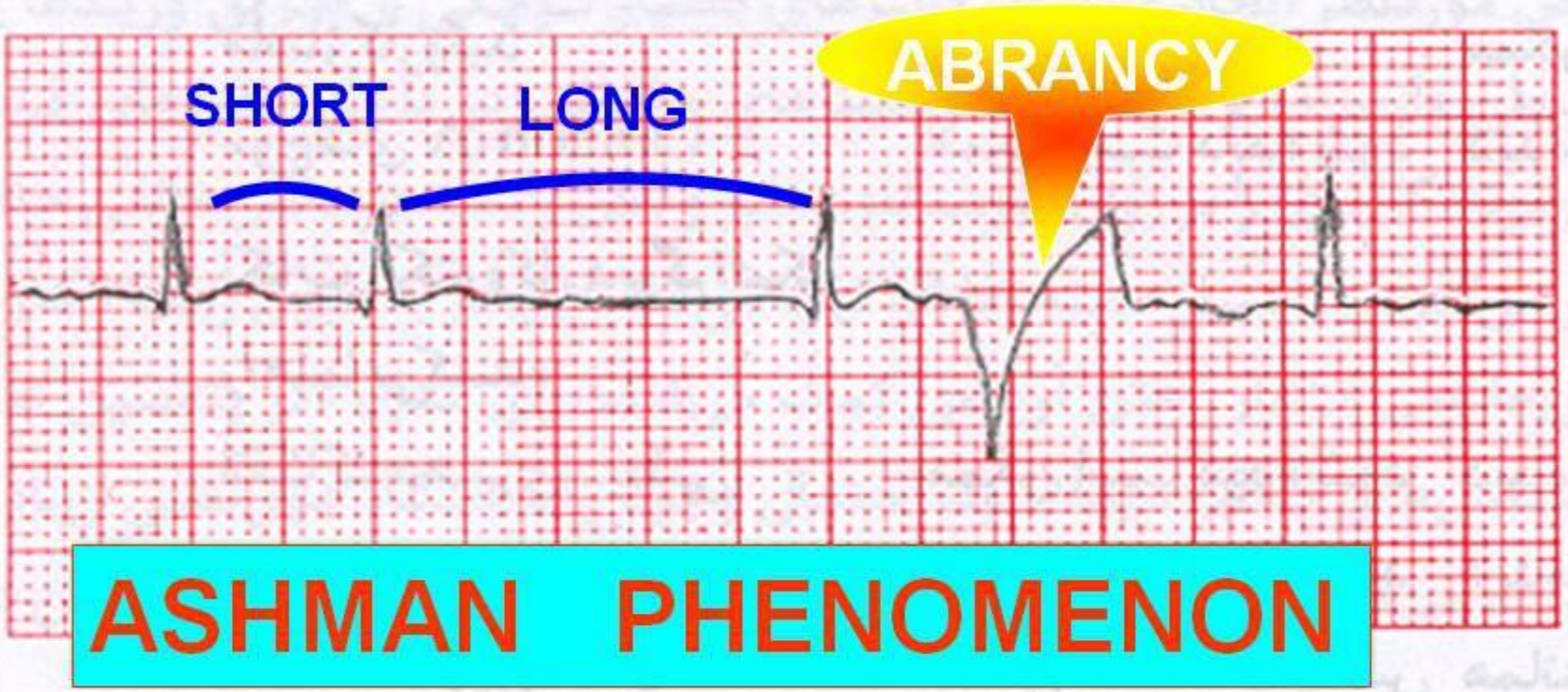


در ریتم های بالا اقدام اول بعد از مونیتورینگ شوک دادن به بیمار با بالاترین ژول انتخابی است



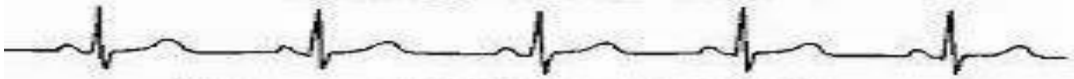
در ریتم های بالا اقدام اول بعد از مونیتورینگ،شروع ماساژ قلبی و تزریق اپی انفرین می باشد

@EMT_EMS

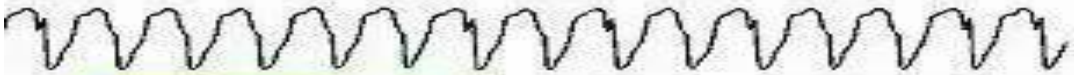


پدیدهٔ Ashman چهارمین ضربان به PVC شباهت دارد، اما می‌تواند یک ضربان فوق بطنی نیز باشد که در مسیری انحرافی هدایت شده است. به فیبریلاسیون دهلیزی زمینه‌ای توجه کنید و همچنین به فاصلهٔ کوتاهی که قبل از ضربان دوم وجود دارد، و فاصلهٔ بلندی که قبل از ضربان سوم وجود دارد. همهٔ اینها زمینه را برای بروز پدیدهٔ Ashman آماده می‌سازند.

COMMON EKG RHYTHMS



Normal Sinus Rythm



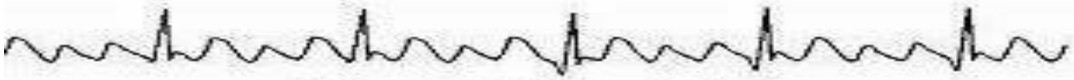
Ventricular Tachycardia

SHOCK

Ventricular Fibrillation

SHOCK

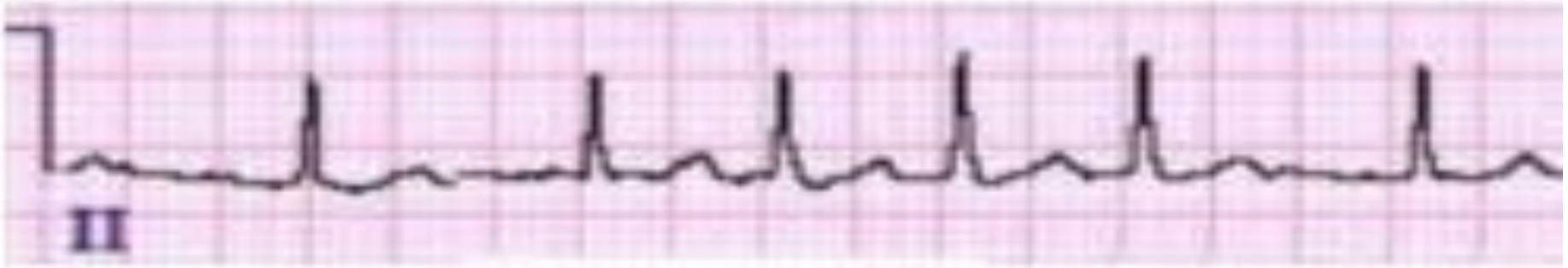
Premature Ventricular Contraction



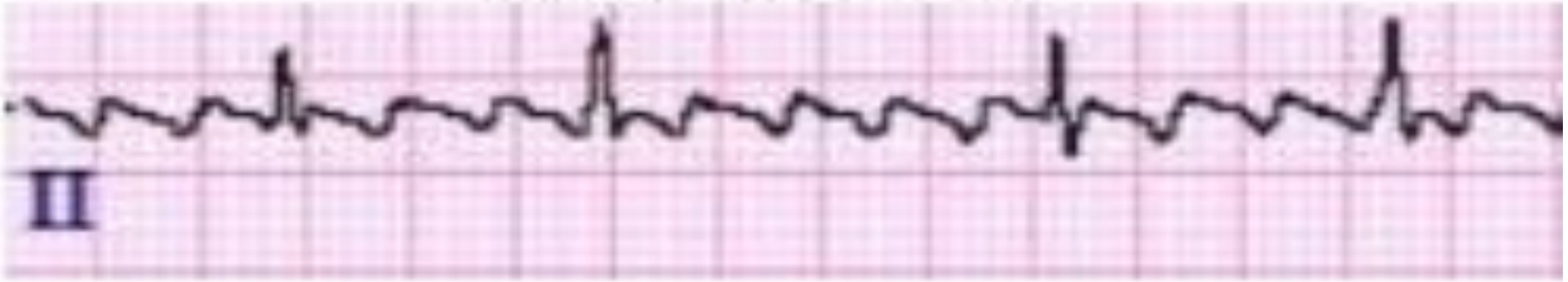
Atrial Flutter

Atrial Fibrillation

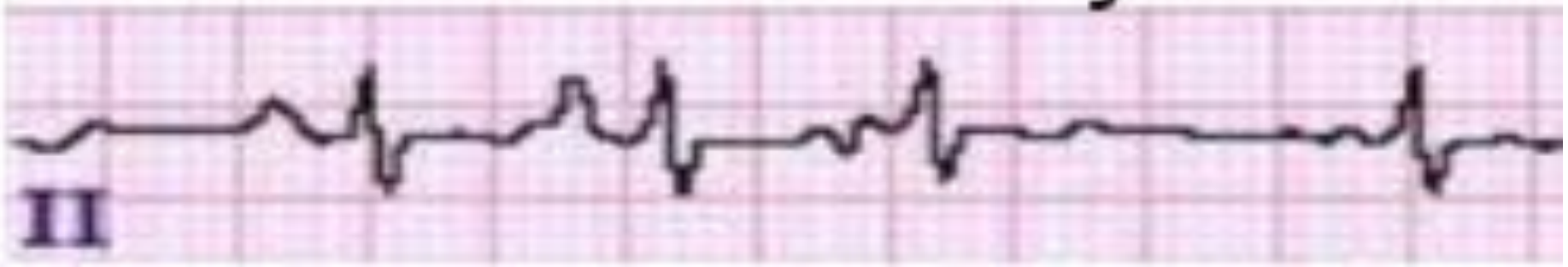
Atrial fibrillation



Atrial flutter

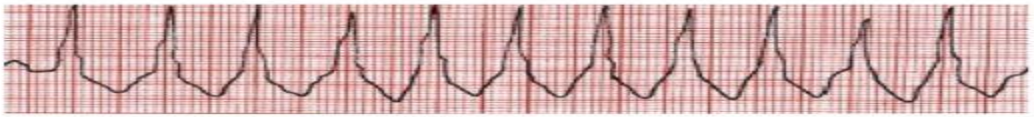


Multifocal atrial tachycardia

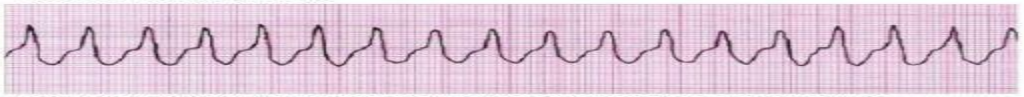


آریتمی هایی که در ایست قلبی و ریوی باید بدانیم

تاکی کاردی بطنی

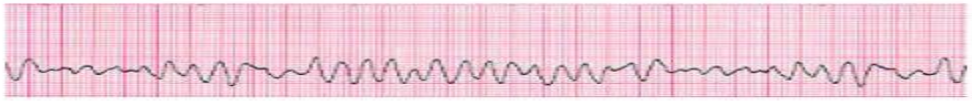


Ventricular tachycardia

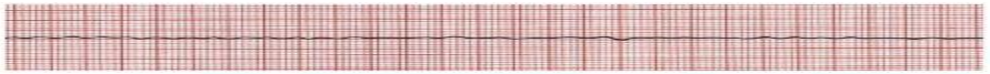


fast heart rhythm, that originates in one of the ventricles- potentially life-threatening arrhythmia because it may lead to ventricular fibrillation, asystole, and sudden death.
Rate=100-250bpm

فیبریلاسیون بطنی

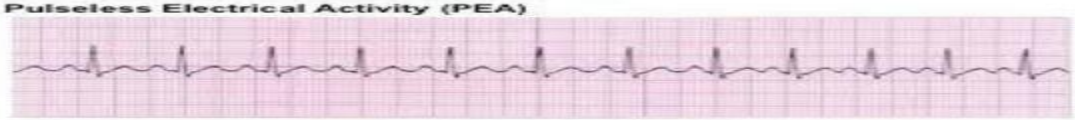


آسیستول



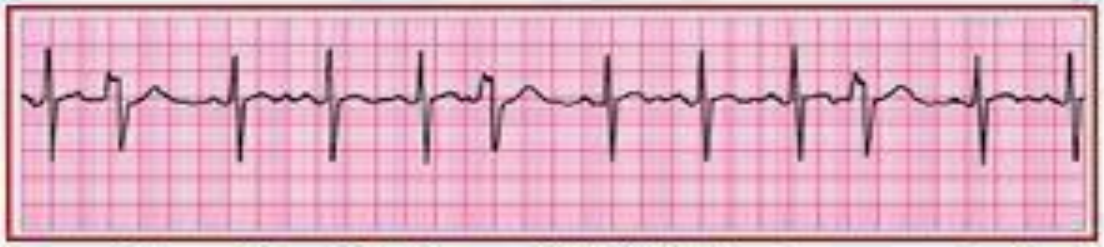
PEA

Pulseless Electrical Activity (PEA)



Not an actual rhythm. The absence of a palpable pulse and myocardial muscle activity with the presence of organized muscle activity (excluding VT and VF) on cardiac monitor. Pt is clinically dead.

فعالیت الکتریکی بدون نبض



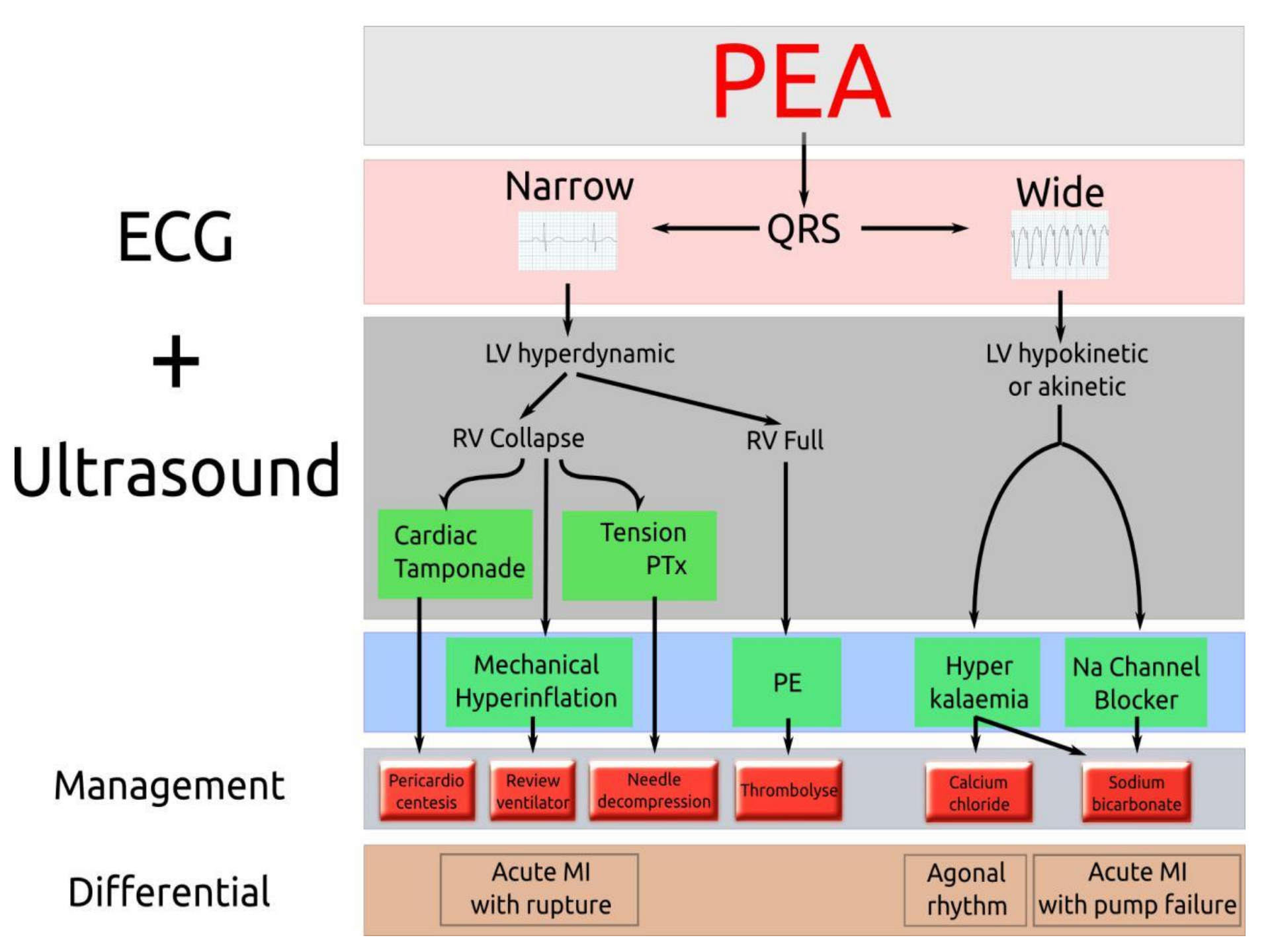
مفهوم کلی : فعالیت الکتریکی قلبی نرمال بوده ولی پمپاژقلبی صورت نگرفته وفاقد نبض میباشدوممکن است بعلت اتفکاک الکترومکانیکی قلب باشد

اقدامات : انجام CPR (۵چرخه) ،IV access ، اکسیژن تراپی و دارودرمانی ورفع علت زمینه ای

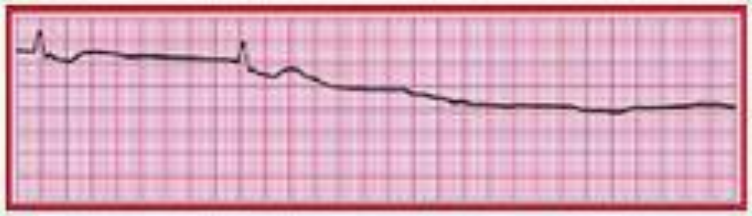
دارودرمانی : اپی نفرین

احتمالات: هیپوولمی-تامپوتادپریکارد-پنوموتوراکس فشاری -اسیدوز-هیپوکسمی-آمبولیهای بزرگ ریوی، اتفارکتوس وسیع قلبی ،مسمومیت بابیکربنات ،سه حلقه ای ها،دیژیتال،کلسیم وکلسیم بلوکروگلوکاگون

نکته : دراین نوع ریتم انقباض قلبی ممکن است موجود باشد ویا نباشد


PEA
ECG
+
Ultrasound
Narrow
QRS
Wide
LV hyperdynamic
LV hypokinetic
or akinetic
RV Collapse
RV Full
Cardiac
Tamponade
Tension
PTx
Mechanical
Hyperinflation
PE
Hyper
kalaemia
Na Channel
Blocker
Management
Pericardio
centesis
Review
ventilator
Needle
decompression
Thrombolyse
Calcium
chloride
Sodium
bicarbonate
Differential
Acute MI
with rupture
Agonal
rhythm
Acute MI
with pump failure

آسیستول



مفهوم کلی: عدم وجود هرگونه فعالیت قلبی ، که شانس بقا ۰/۱ درصد میباشد

اقدامات : انجام CPR تا۵چرخه ،IV access ، اکسیژن تراپی ودارودرمانی

نکته: درصورت شک بین ریتمVF وآسیستول بجای انجام دفیبریلاسیون فشردن قفسه سینه بمدت ۲دقیقه توصیه میشود.

دارودرمانی : اپی نفرین

نکته : دراین نوع ریتم قلبی قبل ازهرگونه اقدام جهت مطمئن بودن ازریتم نشان داده شده بایستی اتصالات چک گردیده ودر۲لید دیگرنیز آسیستول تایید گردد

نکته : دستگاه ضربان ساز دیگر دردرمان آسیستول جایی ندارد.

نکته : درکودکان آسیستول وفعالیت الکتریکی بدون نبض نسبت به VF شایعتراست

- **VT management:**

Unstable:
Shock

stable

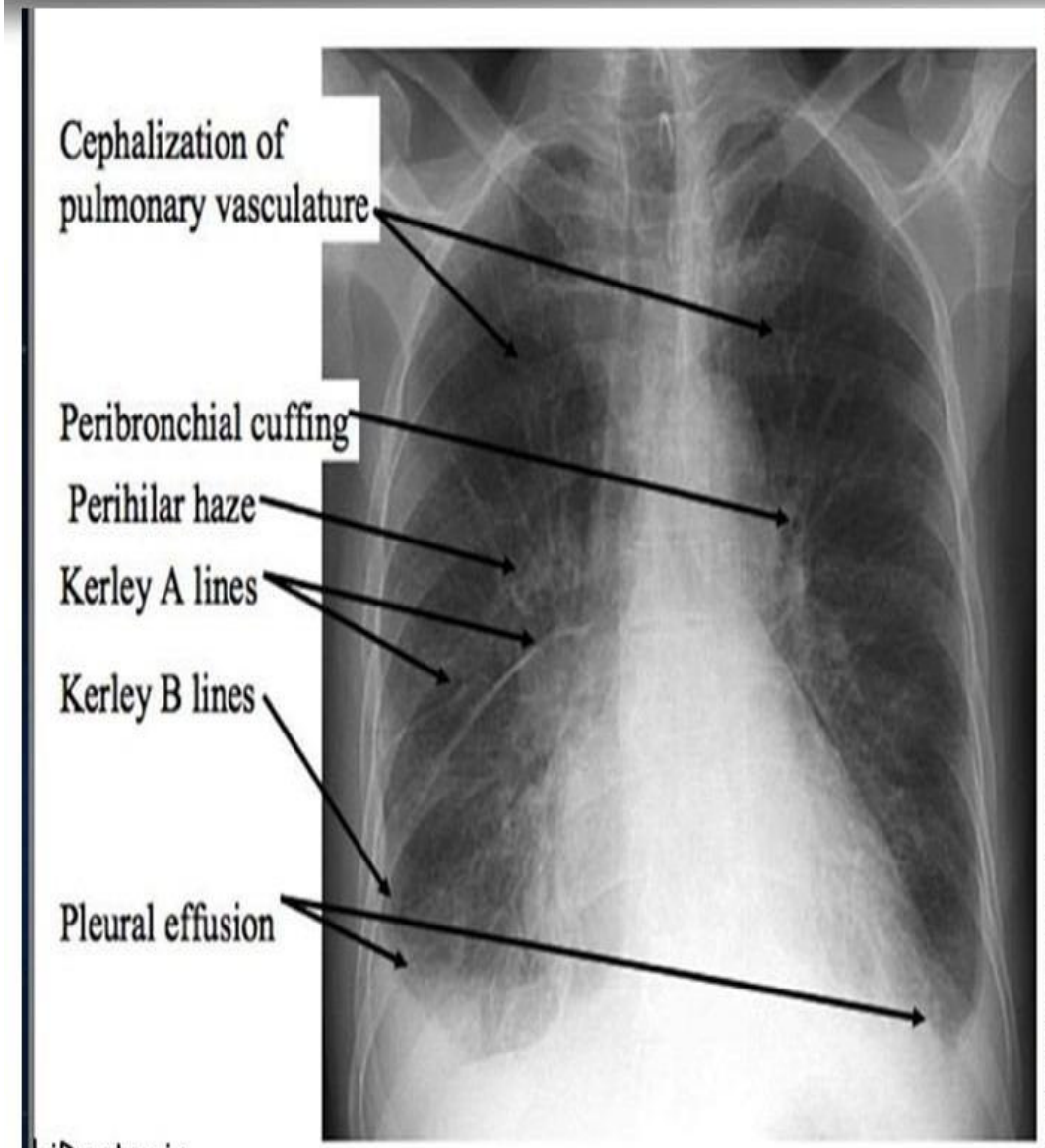
Amiodaron
initial doses 150mg stat (15 mg/min for 10 minutes), followed by 1 mg/min for 6 hours and then 0.5 mg/min for the remaining 18 hours
⊙ we can repeated loading dose another 150mg as needed

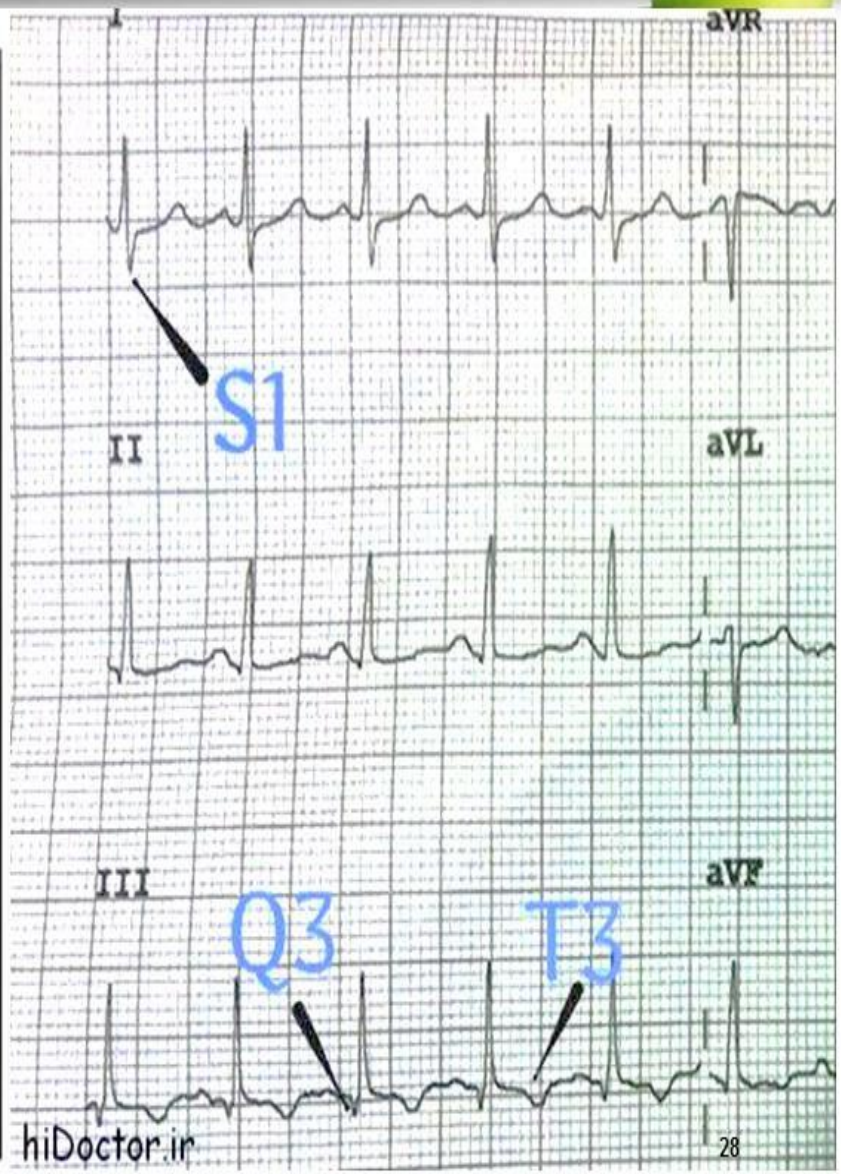
lidocaine:
1 to 1.5 mg/kg; repeat with 0.5 to 0.75 mg/kg every 5 to 10 minutes as necessary (maximum cumulative dose: 3 mg/kg). Follow with continuous infusion of 1 to 4 mg/minute (or 14 to 57 mcg/kg/minute).

Procainamide
Loading dose: Infuse 20 to 50 mg/minute or 100 mg every 5 minutes until arrhythmia controlled, hypotension occurs, QRS complex widens by 50% of its original width, or total of 17 mg/kg is given.
Follow with a continuous infusion of 1 to 4 mg/minute.

- @DRMVP

آمبولی ریه





چون ترومبوز در DVT کوچکتر است). در موارد شک بالینی که به DVT، در بیش از ۹۰٪ موارد DVT وجود نداشته و D-dimer منفی است. در بیش از ۹۵٪ کسانی که PTE ندارند، D-dimer منفی است.

۲- ABG : به تشخیص آمبولی کمک زیادی نمی‌کند. ABG معمولاً نشاندهندهٔ آلکالوز تنفسی در بیمار است (به علت تاکی‌پنه) اغلب هم PO_2 و هم PCO_2 کاهش دارد.

۳- EKG نیز به تشخیص کمک زیادی نمی‌کند. شایعترین یافته در آن تاکیکاردی سینوسی است. ممکنست در درصد کمی از بیماران right axis deviation و RBBB مشاهده شود. به ندرت الگوی تیپیک S1Q3T3 ایجاد می‌شود (S عمیق در لید I، Q پاتولوژیک و T معکوس در لید III) در لیدهای V1-V4 نیز ممکن است T invert دیده شود.

۴- CXR اغلب در آمبولی ریه نرمال است. در تنگی نفس ناگهانی و CXR نرمال، در قدم اول باید آمبولی رد شود.

علائم زیر ممکنست در گرافی دیده شوند:

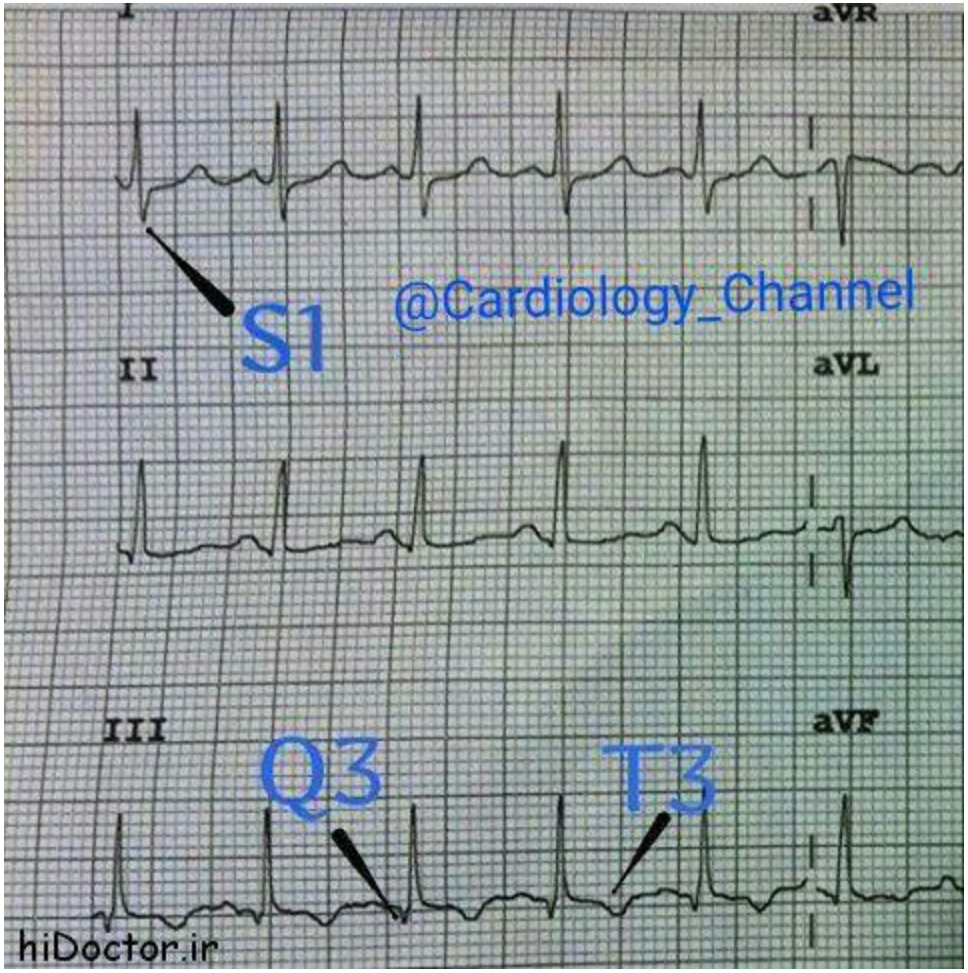
- علامت **Westermark** (اولیگمی در قسمتی از ریه)
- علامت **Humpton** (یک wedge consolidation که قاعده آن بر روی پلور یا دیافراگم است)
- علامت **Palla** (بزرگتر شدن شریان پولمونر راست)

In a patient who has suffered a pulmonary embolus there may be transient non-specific changes in the patient's ECG:

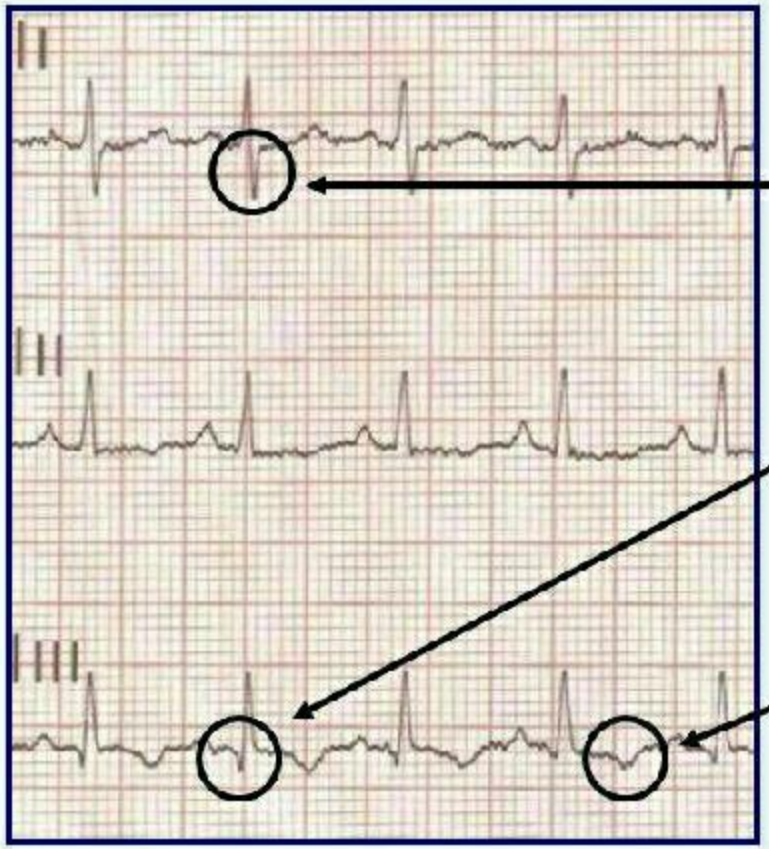
- often no changes are seen on the ECG
- classical changes are S1, Q3, T3

The detailed changes are as follows:

- tall R waves in V1
- P pulmonale (peaked P waves) best seen in the inferior leads
- there may be right axis deviation and clockwise rotation
- atrial arrhythmias may occur
- there may be T wave inversion in the leads V1, V2, V3
- possible right bundle branch block
- there may be a shift of transition point to the left, so that the R wave equals the S wave in V5 or V6 rather than V3 or V4
- in this condition there is often a Q wave in lead 3, resembling an inferior infarction
- right ventricular strain is very occasionally seen, causing an S wave in lead I, Q wave and inverted T wave in lead III
    - right ventricular strain pattern on ECG is associated with adverse short-term outcome and adds incremental prognostic value to echocardiographic evidence of right ventricular dysfunction in patients with acute pulmonary embolism and normal blood pressure (1)


aVR
S1
@Cardiology_Channel
II
aVL
III
aVF
Q3
T3
hiDoctor.ir

S1Q3T3



S-waves in lead I

Q-waves in lead III

Inverted
T-waves in lead III

ems12lead.com

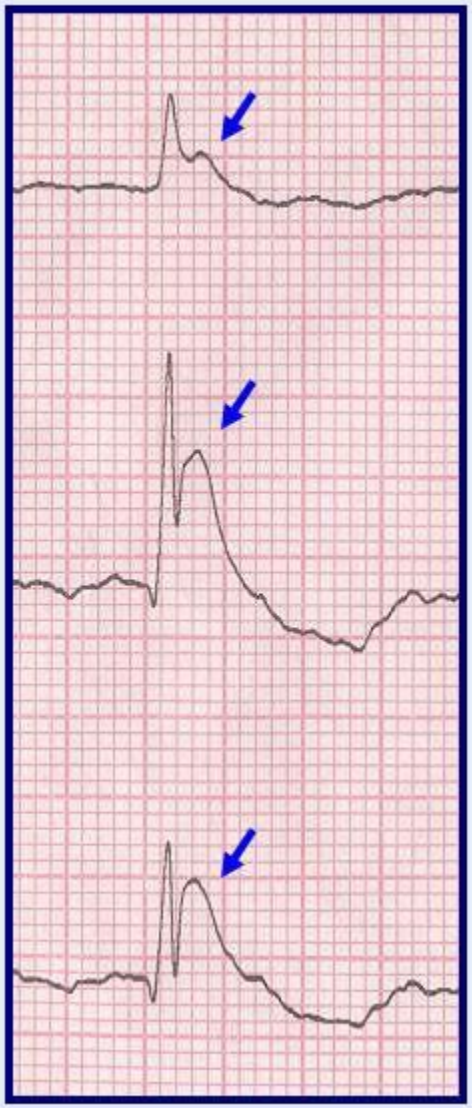
CLASSIFICATION	PRESENTATION	THERAPY
Massive PE	Systolic blood pressure <90 mm Hg or poor tissue perfusion or multisystem organ failure plus right or left main pulmonary artery thrombus or "high clot burden"	Thrombolysis or embolectomy or inferior vena caval filter plus anticoagulation
Submassive PE	Hemodynamically stable but moderate or severe right ventricular dysfunction or enlargement	Addition of thrombolysis, embolectomy, or filter remains controversial
Small to moderate PE	Normal hemodynamics and normal right ventricular size and function	Anticoagulation

Web Table 3 Approved thrombolytic regimens for pulmonary embolism

Streptokinase	250 000 IU as a loading dose over 30 minutes, followed by 100 000 IU/h over 12–24 hours
	Accelerated regimen: 1.5 million IU over 2 hours
Urokinase	4400 IU/kg as a loading dose over 10 min, followed by 4400 IU/kg per hour over 12–24 hours
	Accelerated regimen: 3 million IU over 2 hours
rtPA	100 mg over 2 hours; or
	0.6 mg/kg over 15 minutes (maximum dose 50 mg)

IU = international units; rtPA = recombinant tissue plasminogen activator.

Osborne Waves or "J-Waves"



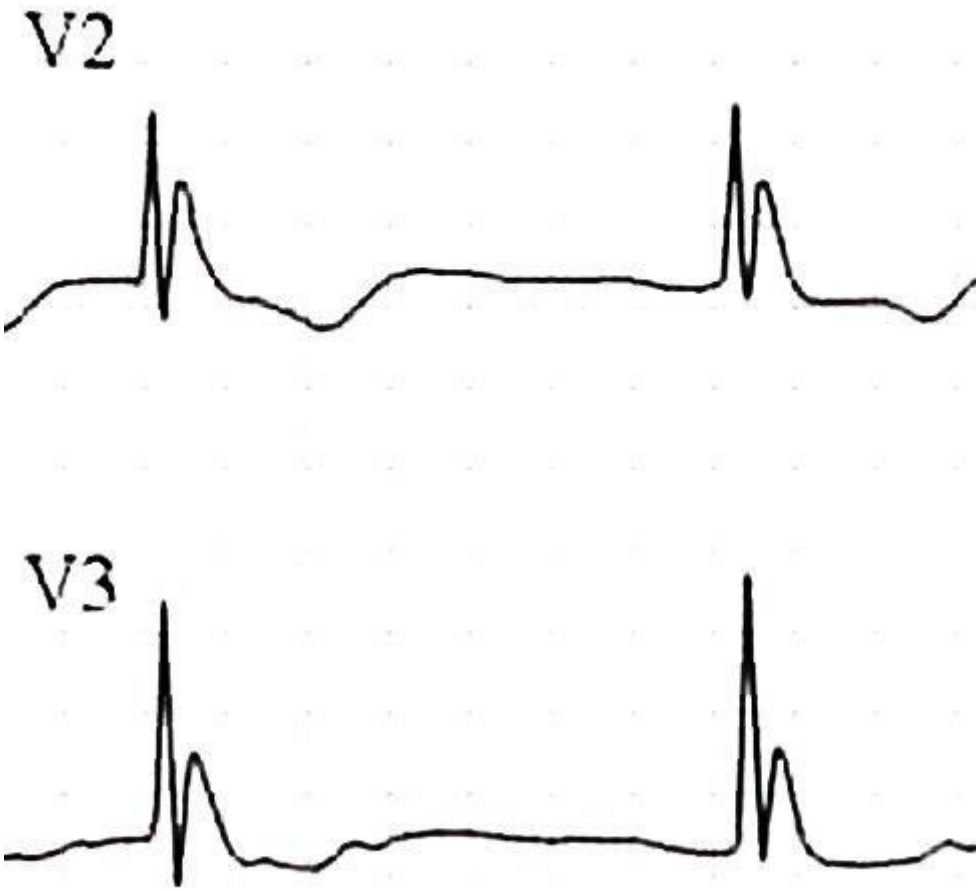
Here we see the Osborn waves of severe hypothermia (blue arrows).

The rhythm is atrial fibrillation.

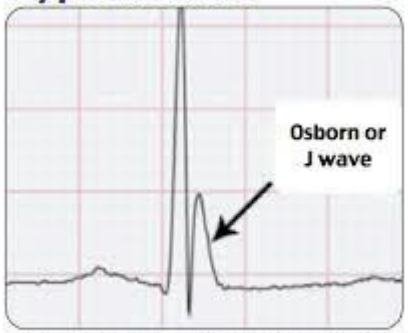
Bradycardia is present.

The QT/QTc is prolonged.

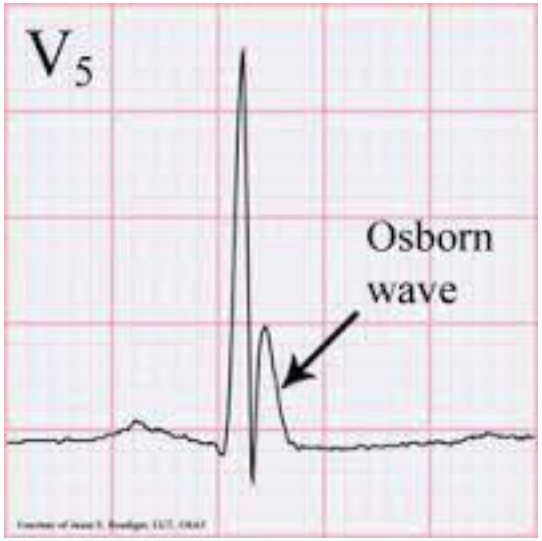
The patient's core temperature was measured at 76°F (24°C).

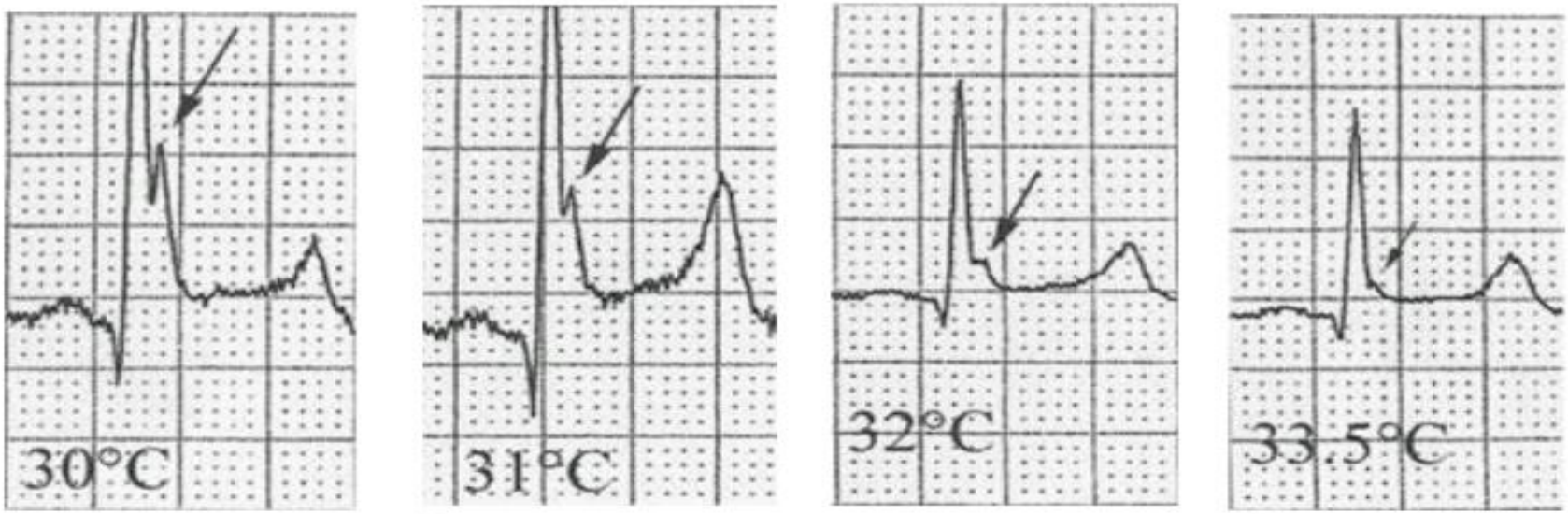

V2
V3

Hypothermia



- Size of wave correlates with degree of hypothermia
- Typically appears below 32°C
- Usually resolves with warming
- No prognostic value

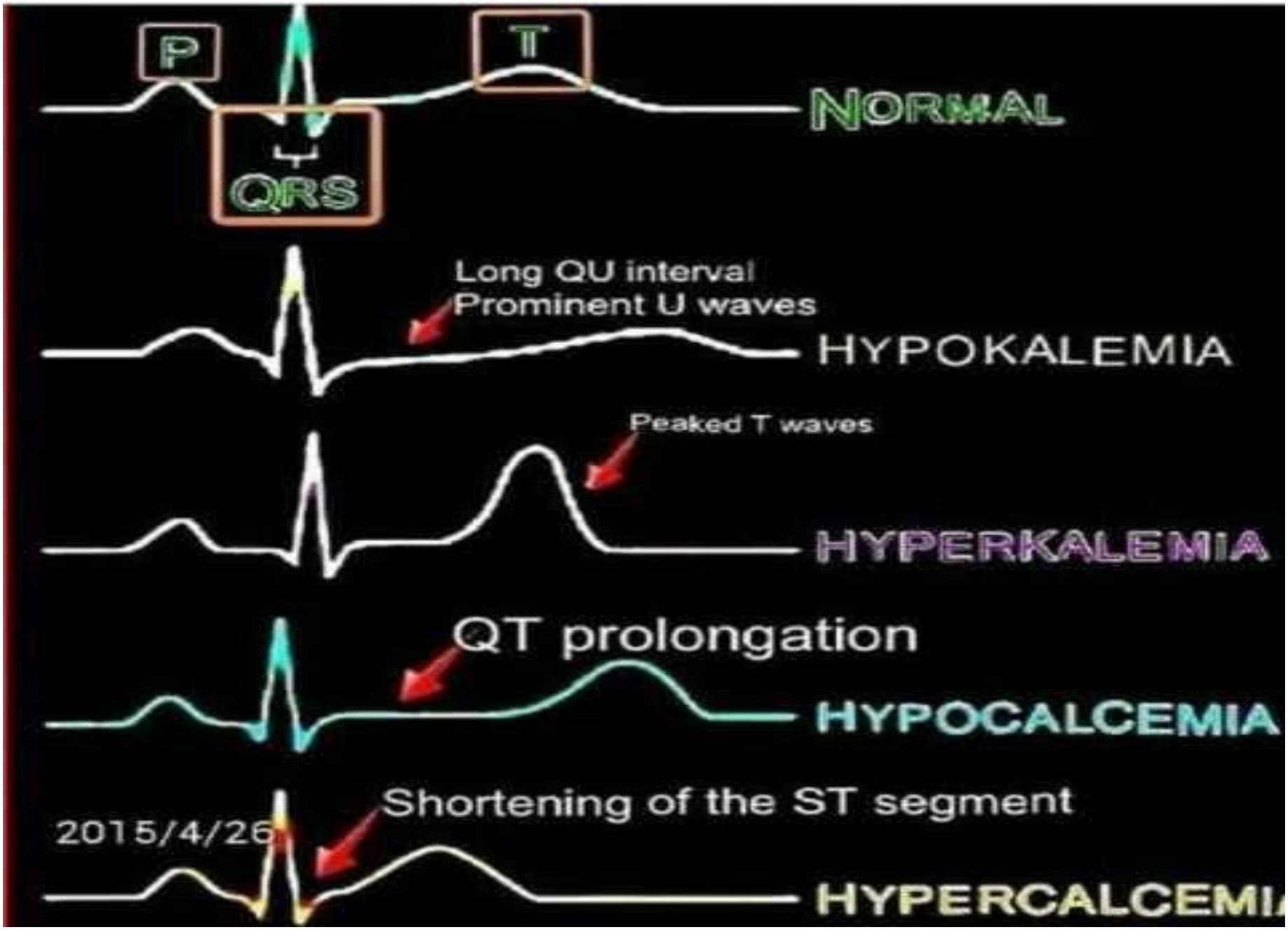




ECGs 3.23a–3.23d

ECG monitor lead: Osborn wave in a 53-year-old patient during open heart surgery. The Osborn wave and ST depression gradually diminishes during increasing body temperature.

a. **Temperature 30 °C.**
b. **Temperature 31 °C.**
c. **Temperature 32 °C.**
d. **Temperature 33.5 °C.**


P
T
QRS
NORMAL
Long QU interval
Prominent U waves
HYPOKALEMIA
Peaked T waves
HYPERKALEMIA
QT prolongation
HYPOCALCEMIA
Shortening of the ST segment
2015/4/26
HYPERCALCEMIA

Effects of hyperkalemia on the ECG

6.5 mEq/L> **Serum potassium** > 5.5 mEq/L

(Tall T waves)

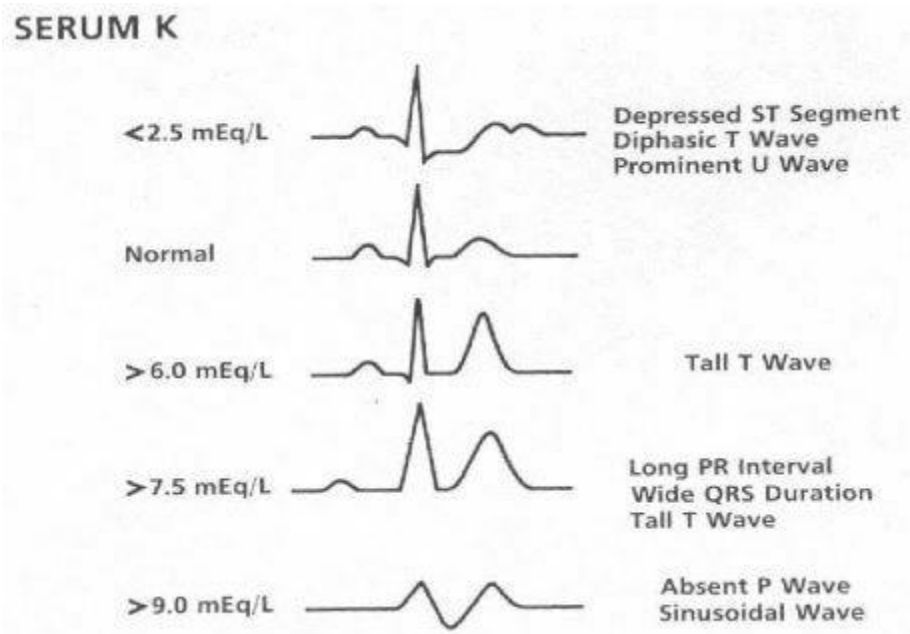
7 mEq/L> **Serum potassium** > 6.5 mEq/L

1. P wave widens and flattens
2. PR segment lengthens
3. P waves eventually disappear

9 mEq/L> **Serum potassium** > 7 mEq/L

1. Prolonged QRS interval
2. High-grade AV block
3. bundle branch blocks, fascicular blocks
4. Sinus Bradycardia or slow AF

SERUM K


<2.5 mEq/L
Depressed ST Segment
Diphasic T Wave
Prominent U Wave
Normal
>6.0 mEq/L
Tall T Wave
>7.5 mEq/L
Long PR Interval
Wide QRS Duration
Tall T Wave
>9.0 mEq/L
Absent P Wave
Sinusoidal Wave

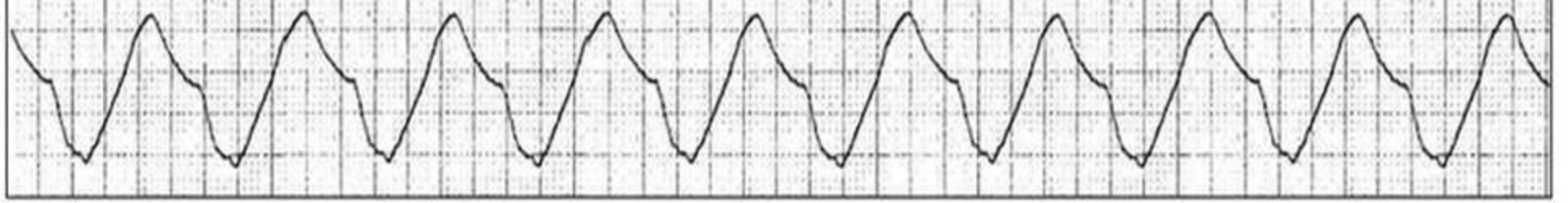
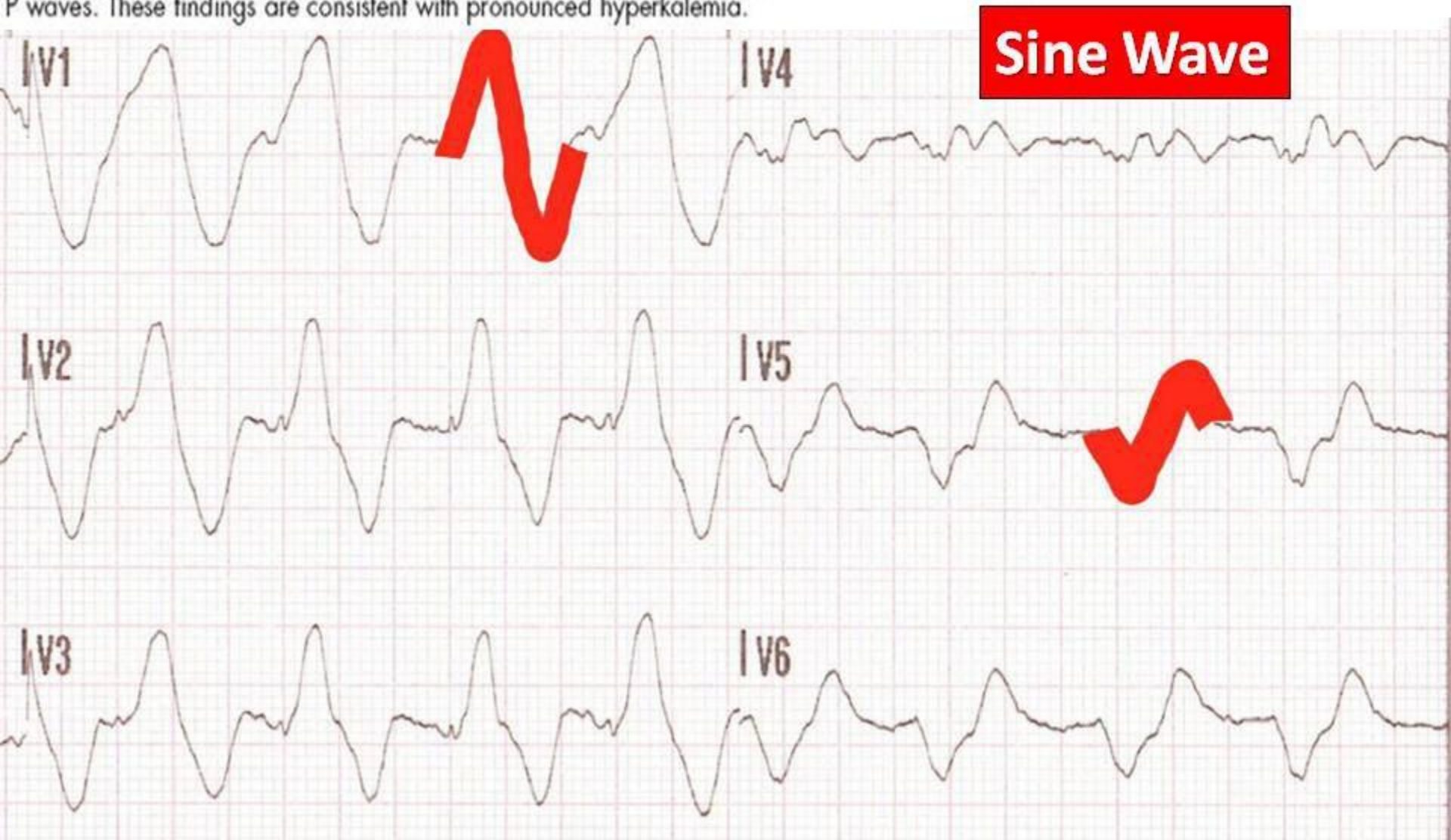


Figure 1 – This ECG rhythm strip shows a wide-complex bradycardic rhythm with sine-wave configuration and the absence of discernible P waves. These findings are consistent with pronounced hyperkalemia.



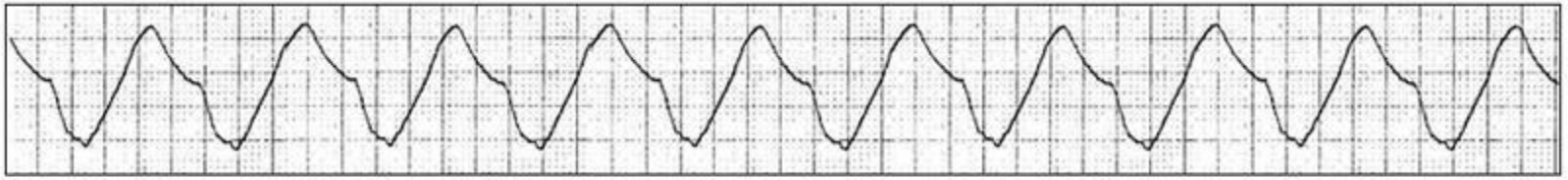


Figure 1 – This ECG rhythm strip shows a wide-complex bradycardic rhythm with sine-wave configuration and the absence of discernible P waves. These findings are consistent with pronounced hyperkalemia.

هایپرکالمی Hyperkalemia

- بررسی، کنترل وحمایت ABCDE
  - Airway
  - Breathing
  - Circulation
  - Disability
  - Exposure
- گرفتن ECG ۱۲ لیدی و مانیتورینگ ریتم قلبی درصورت K > 6.5
- مستسنی کردن موارد هایپرکالمی کاذب
- انجام درمان های تجربی آریتمی درصورت شک به هایپرکالمی

شدید

K > 6.5 mmol/L

نیاز به درمان اورژانسی

متوسط

K = 6-6.4 mmol/L

درمان براساس وضعیت بالینی، ECG و سرعت افزایش

خفیف

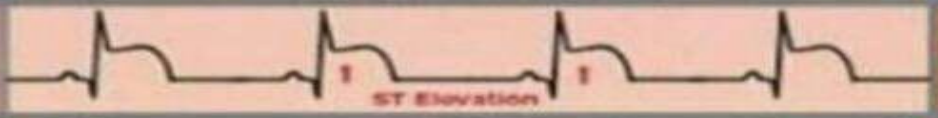
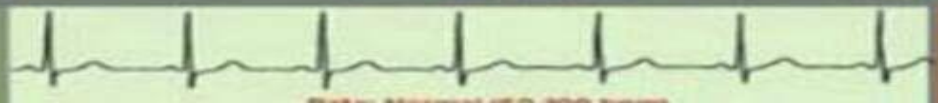
K = 5.5-5.9 mmol/L

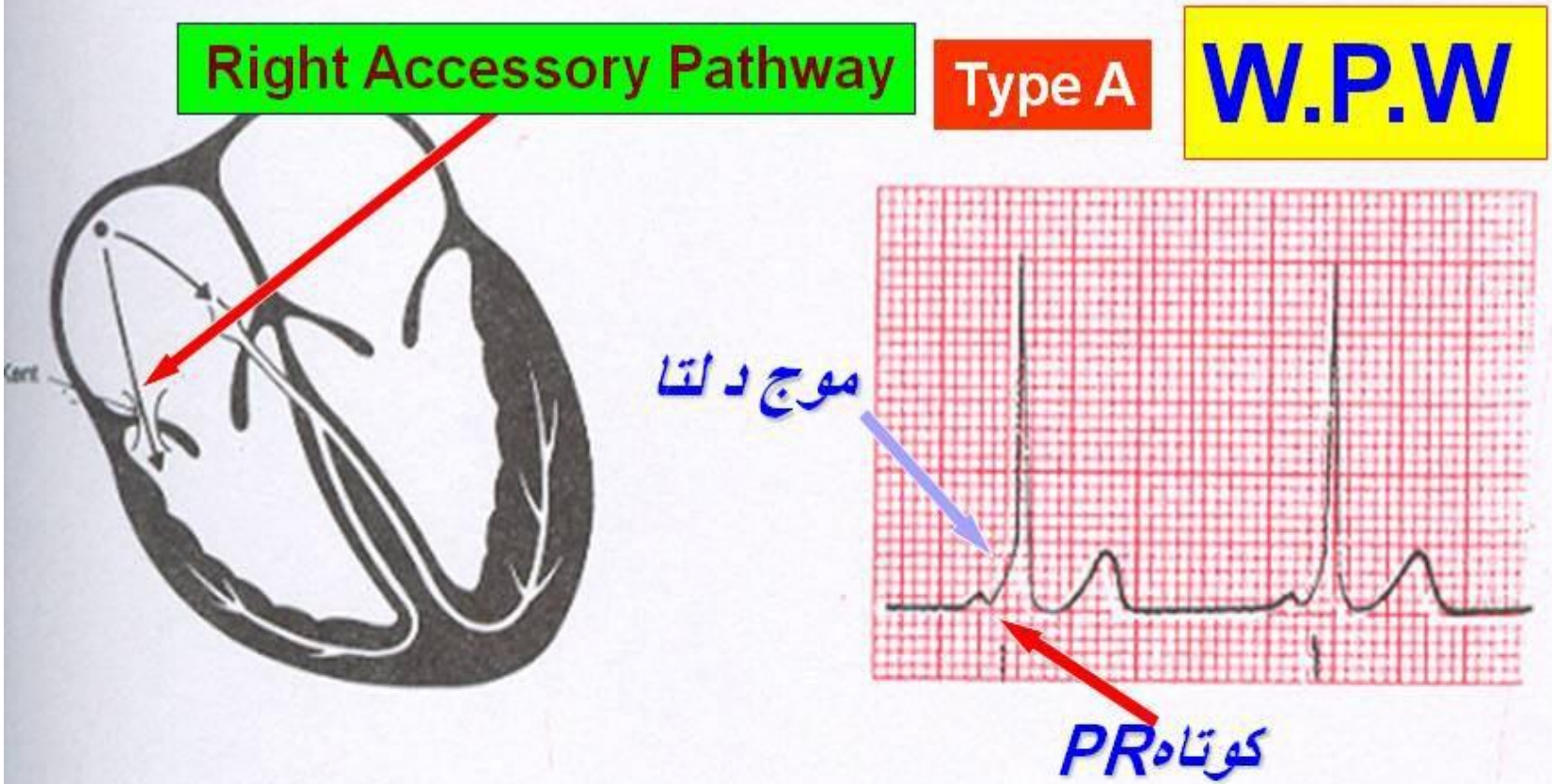
درنظر داشتن علل و نیاز درمان

درخواست کمک حرفه ای

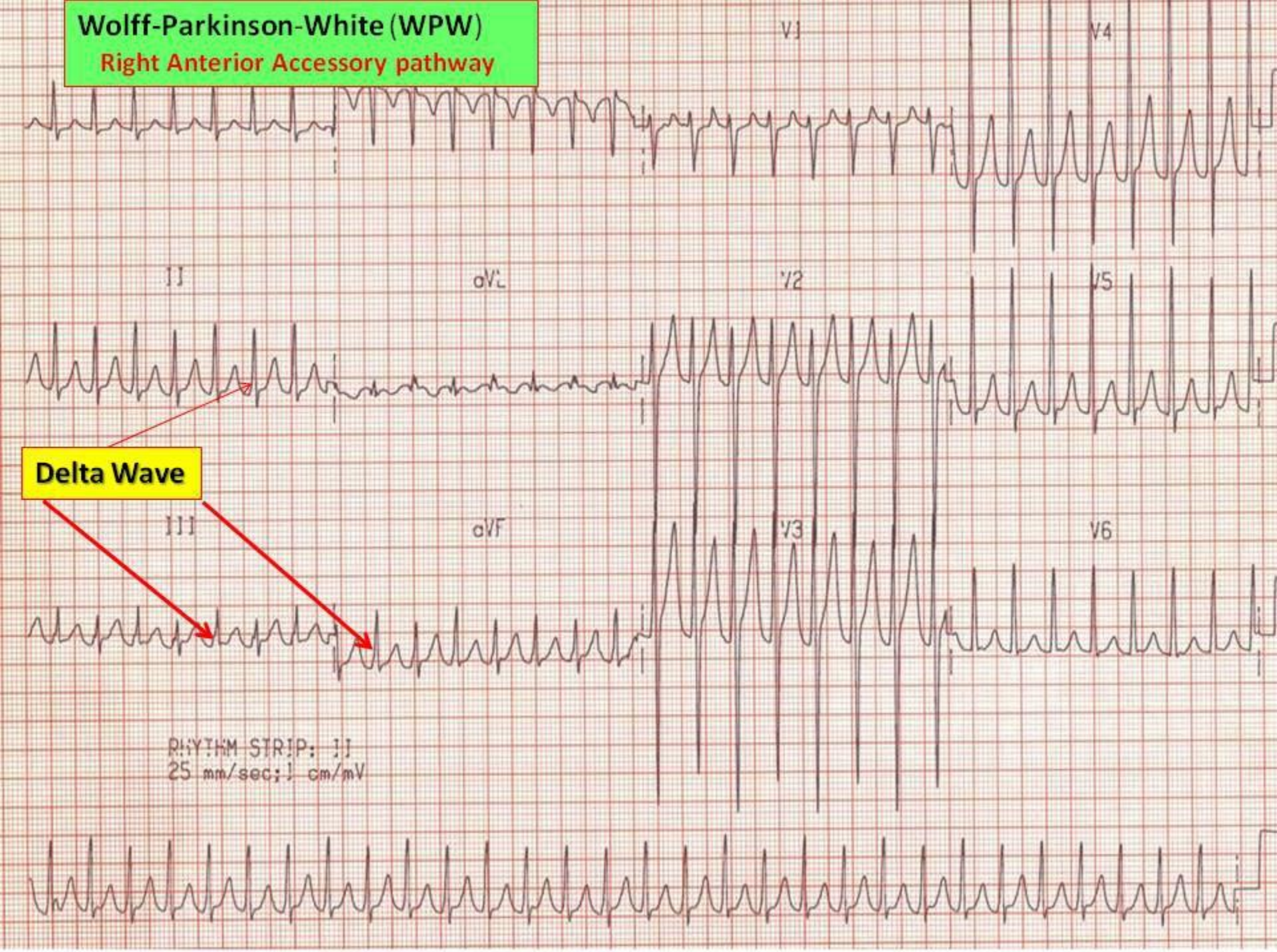
آیا تغییرات ECGوجود دارد؟

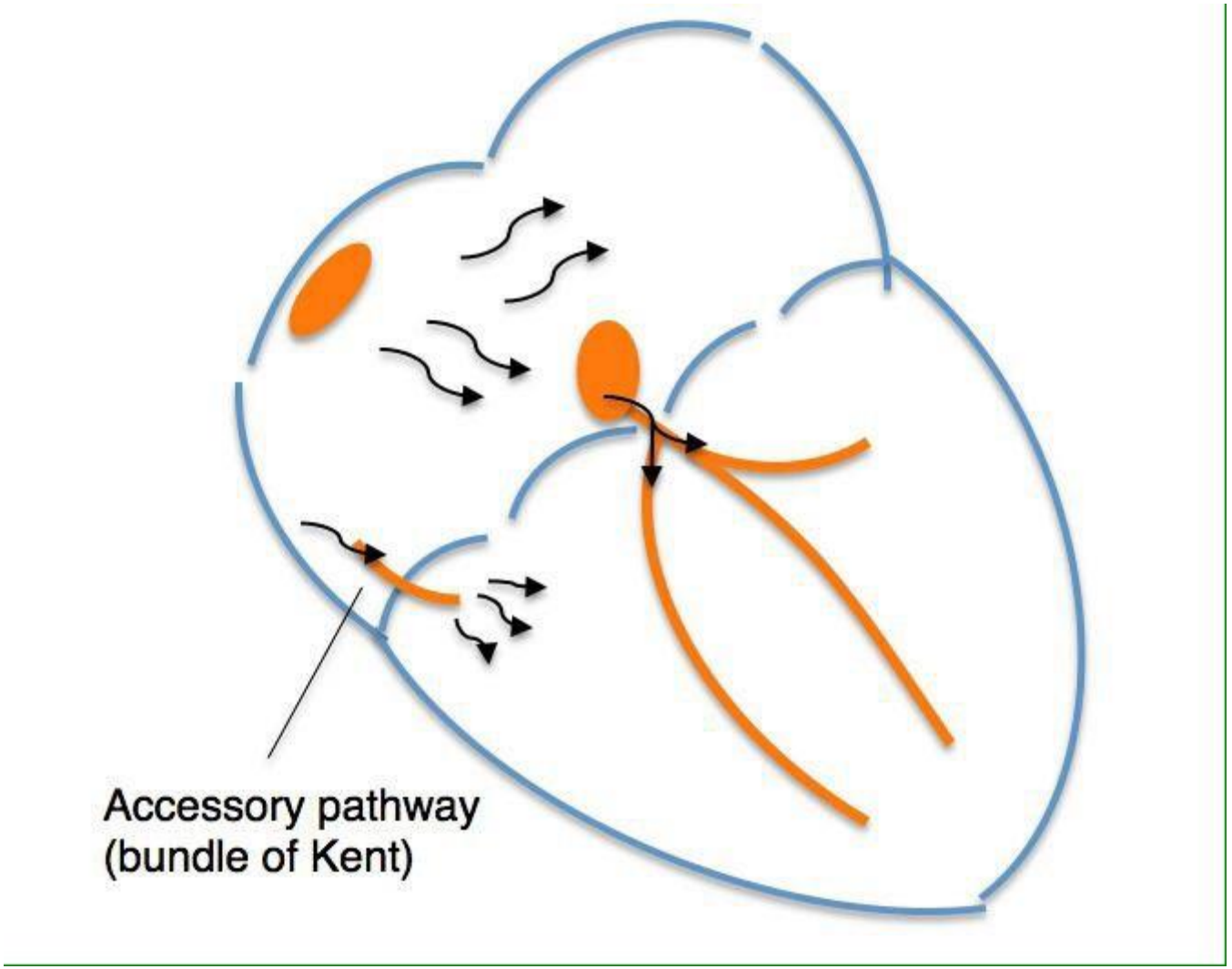
- موج T بلند
- QRS پهن
- برادیکاردی
- P فلت یا غایب
- Sine-wave
- VT

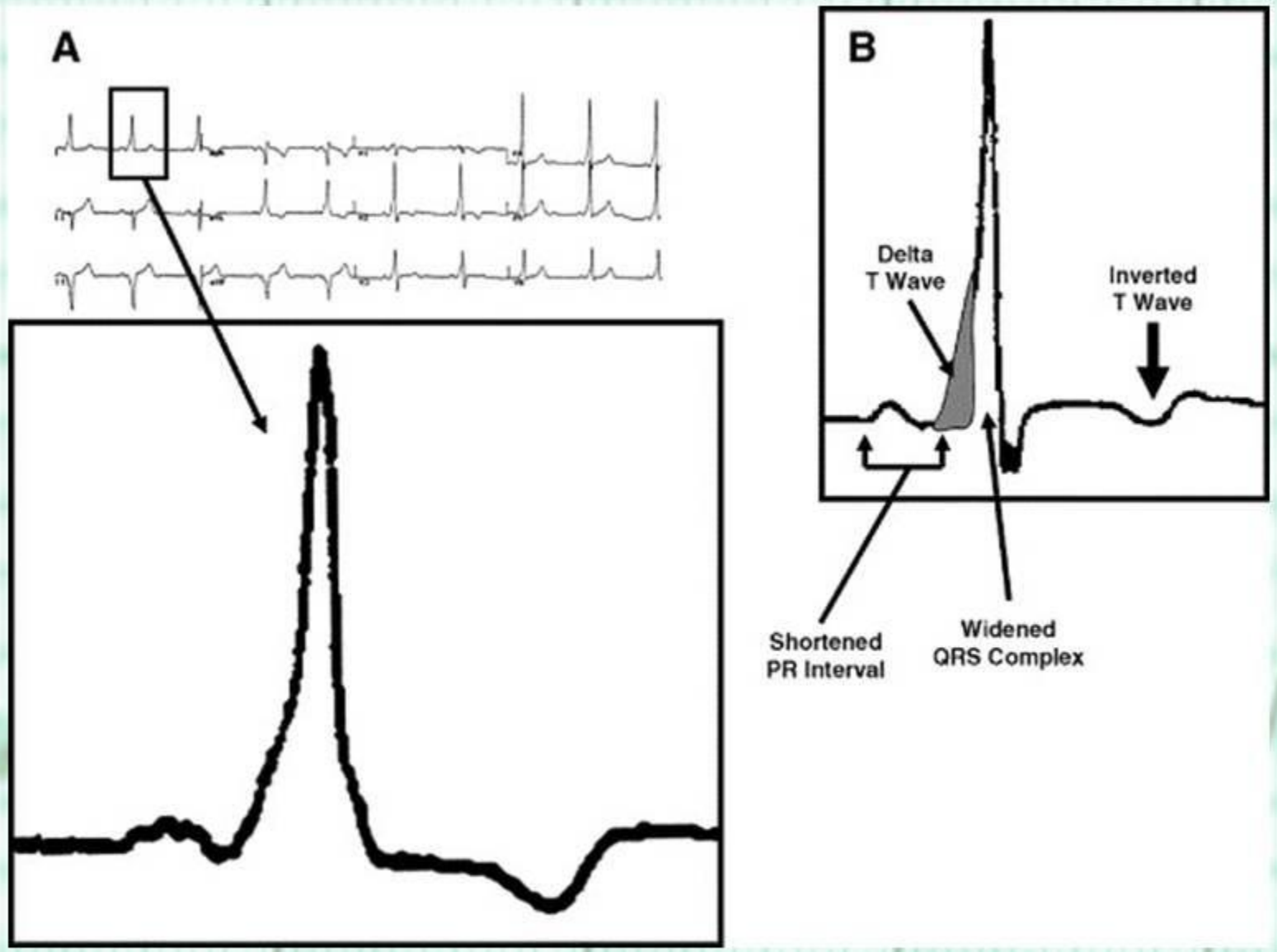
Common & Formal Rhythm Names	6 Second Rhythm Strip	Identifiers
SHOCKABLE		
V-Fib Ventricular Fibrillation		Irregular, No P Wave, No QRS
V-Tach Ventricular Tachycardia		Regular, No P Wave, Wide QRS
Torsade de Pointes Type Of Ventricular Tachycardia		Irregular, No P Wave, Wide QRS
*Synchronized Cardioversion possible for SVT if medication ineffective.		
SVT* Supraventricular Tachycardia		Regular, P Wave Hidden, Normal QRS
STEMI ST Elevation Myocardial Infarction		Reg or Irreg, P Wave, ST Elevated
A-Fib Atrial Fibrillation		Irregular, No P Wave, Normal QRS*
A-Flutter Atrial Flutter		Reg or Irreg, No P Wave, Normal QRS
PVC Premature Ventricular Contraction		Irregular, No P Wave, Wide QRS
Sinus Brady Sinus Bradycardia		Regular, P Wave, Normal QRS
Sinus Tach Sinus Tachycardia		Regular, P Wave, Normal QRS
NSR Normal Sinus Rhythm		Regular, P Wave, Normal QRS

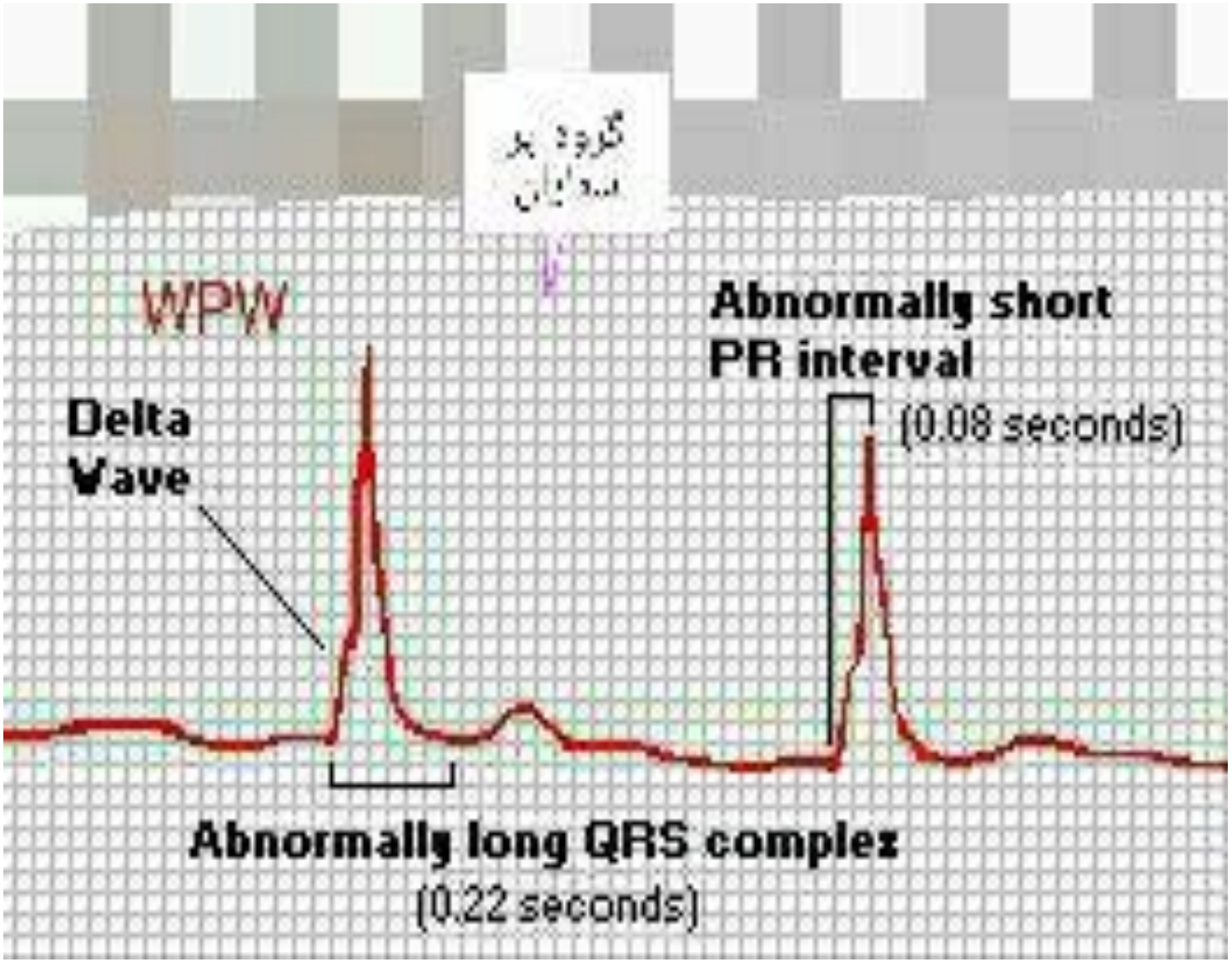


سندرم ولف ـ پارکینسون ـ وایت (WPW). جریان الکتریکی بر اثر تأخیر طبیعی صورت گرفته در گر
متوقف می‌شود، اما در دستهٔ کنت بدون مانع طی مسیر می‌کند. EKG کوتاه شدن فاصلهٔ PR و مو
را نشان می‌دهد.


Wolff-Parkinson-White (WPW)
Right Anterior Accessory pathway
V1
V4
II
aVL
V2
V5
Delta Wave
III
aVF
V3
V6
RHYTHM STRIP: II
25 mm/sec; 1 cm/mV


Accessory pathway
(bundle of Kent)

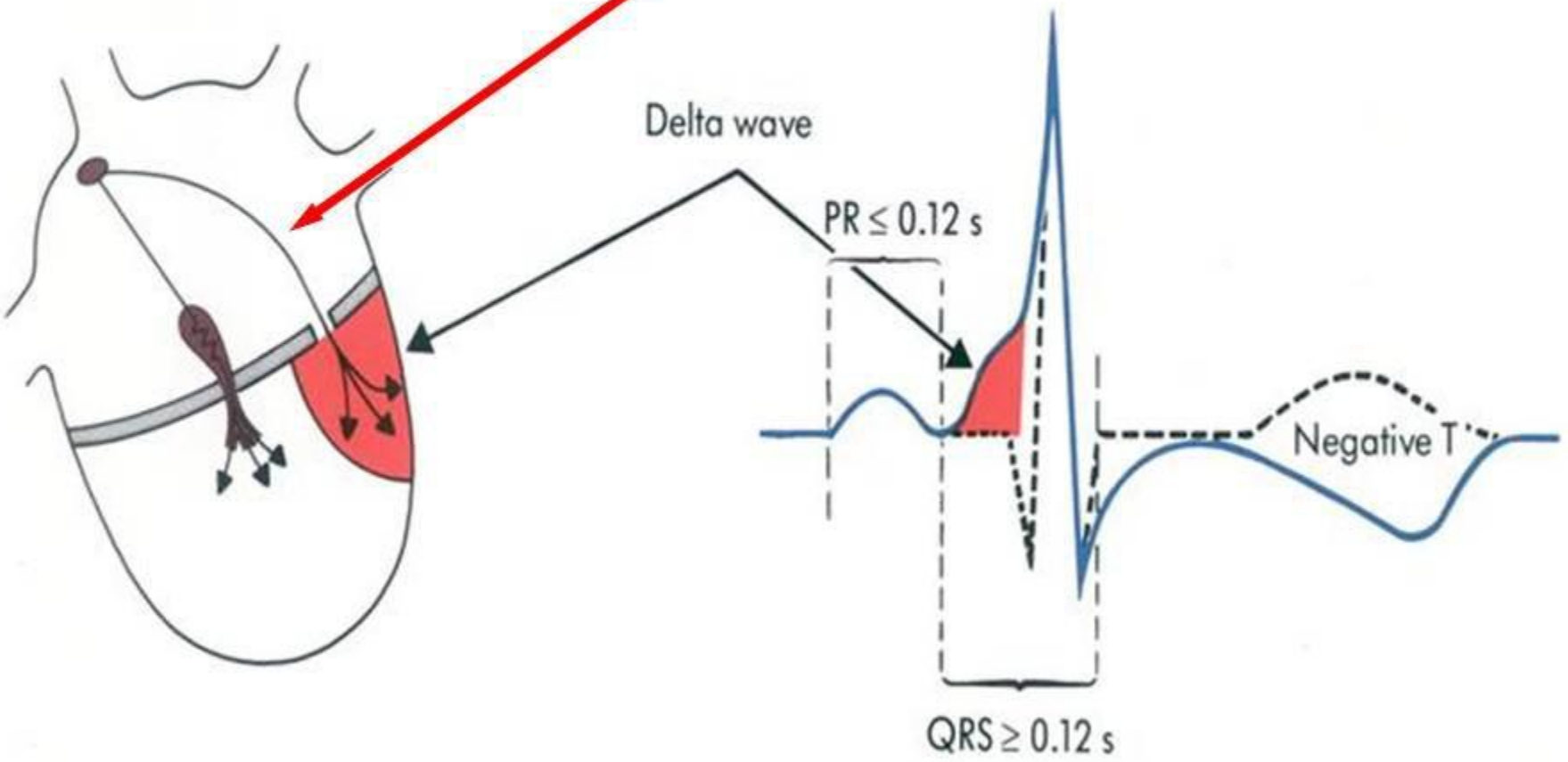



WPW
Abnormally short
PR interval
(0.08 seconds)
Delta
Wave
Abnormally long QRS complex
(0.22 seconds)

WPW SYNDROME

Type B

Left Accessory Pathway



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Type of delta wave polarity

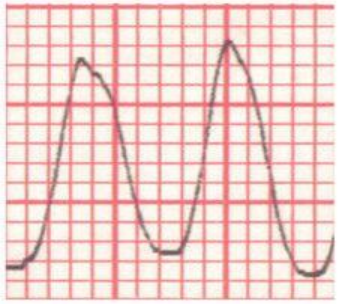
A) Positive delta wave
B) Isoelectric delta wave
C) Over pre-excitation delta wave
D) Negative delta wave

1- Adopted from ; Martin Eisenberger, Europace (2010) 12, 119 – 123
2- https://drsvenkatesan.com

Dominant R wave in aVR

- Poisoning with sodium-channel blocking drugs (e.g. TCAs)
- Dextrocardia
- Incorrect lead placement (left/right arm leads reversed)
- Commonly elevated in ventricular tachycardia (VT)

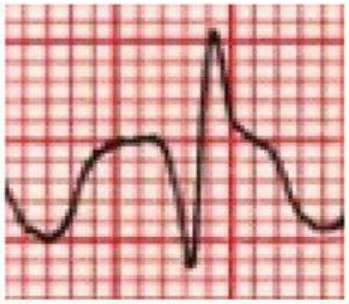
In lead aVR



Initial R-wave

Yes → VT

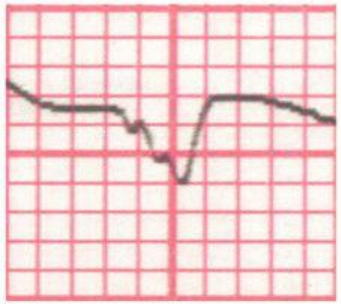
No



Initial r or q > 40 ms

Yes → VT

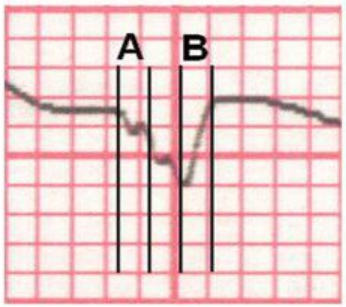
No



Notch on the descending limb of a negative onset and predominantly negative QRS

Yes → VT

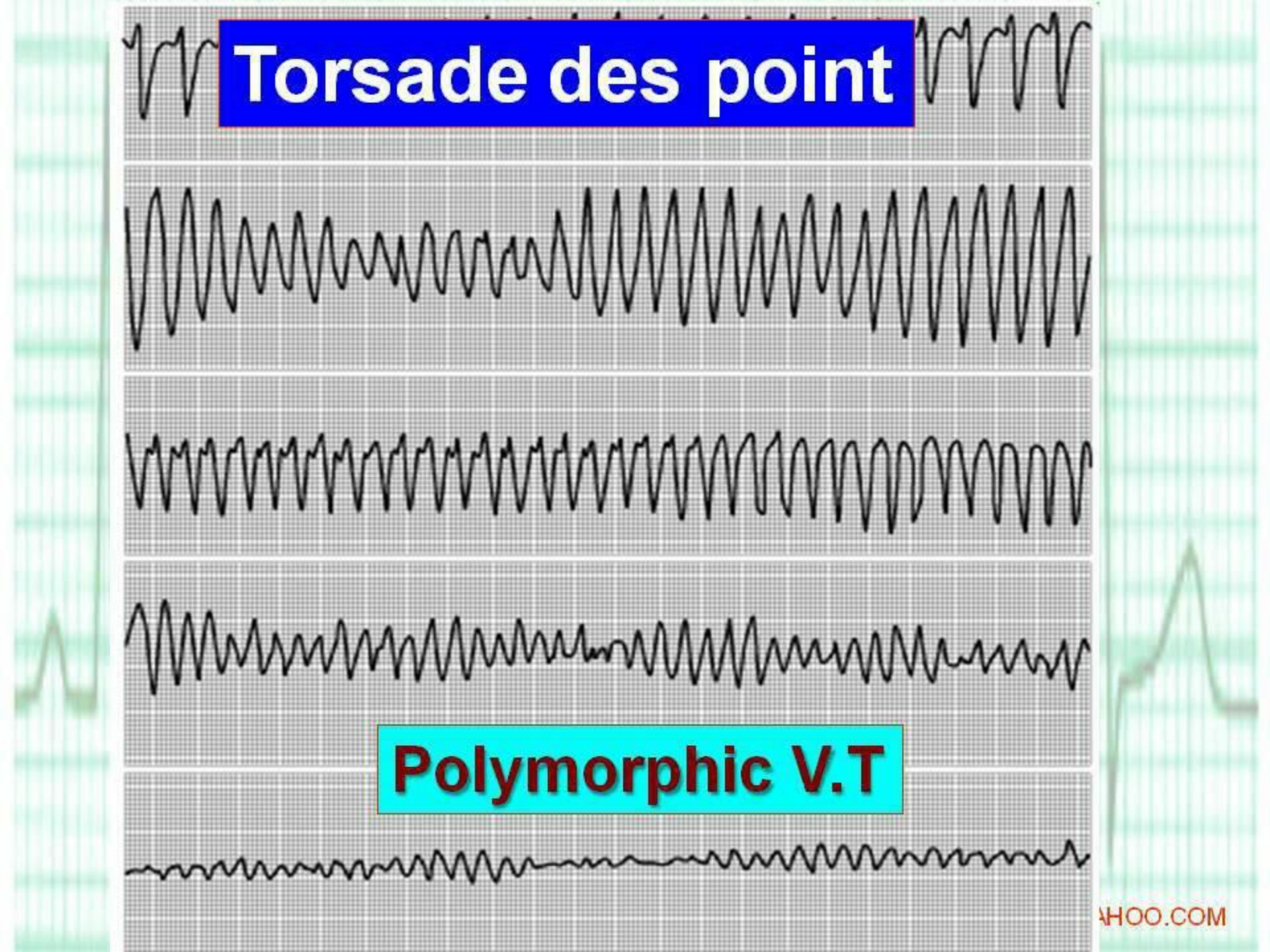
No



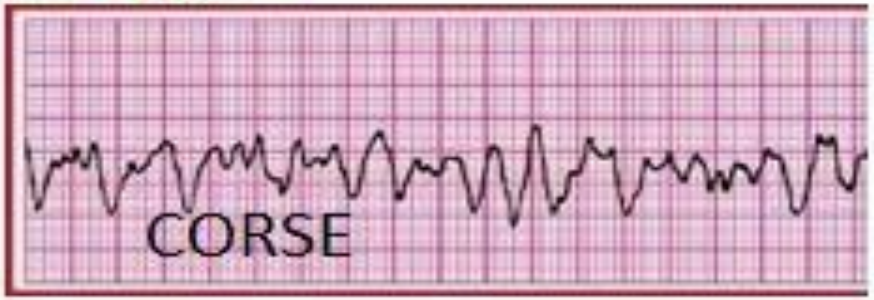
$V_i/V_t \leq 1$

Yes → VT

No → SVT


Torsade des point
Polymorphic V.T
AHOO.COM

فیبریلاسیون بطنی



مفهوم کلی: آشفتگی کامل قلبی درنتیجه فعالیت مناطق متعددقلبی باسرعت زیاد بدون دخالت هرگونه فعالیت سازمان یافته میباشد این ریتم شایعترین ریتم قابل شوک دادن دربزرگسالان است.

- این ریتم درکودکان ممکن است بیان کننده مسمومیت دارویی، اختلالات الکترولیتی وبیماریهای مادرزادی قلبی باشدد.
- دراین نوع ریتم برون ده قلبی وجود ندارد

اقدامات : استفاده ازدفیبریلاتور، IV access ، اکسیژن تراپی وداروودرمانی وCPR

بزرگسالان : مونوفازیک۳۶۰ وبای فازیک با ۲۰۰- ۱۲۰ ژول شروع میشود درفیبریلاسیون دهلیزی بای فازیک۱۲۰ ژول ومونوفازیک ۲۰۰ژول میباشد

کودکان : درشوک اول 2J/KG وبدنبال آن4J/KG ودرشوکهای بعدی تا10J/KG وحتی تا اندازه بزرگسالان هرکدام که کمتر باشد ادامه می یابد.

داروودرمانی: اپی تفرین پس ازشوک دوم و آمیوداروون ولیدوکائین پس ازشوک سوم

Cardioversion

- Alive, unstable, tachyarrhythmia
- Unstable?
  - Signs of low cardiac output: systolic hypotension < 90 mmHg, altered mental status
  - Excessive rates >150/min
  - Chest pain
  - Heart failure
- Synchronized cardioversion is shock delivery that is timed (synchronized) with the QRS complex

Factor	V-Tach	SVT with Aberrancy
Age	>50	<35
History	MI, CHF, CABG, MVR	MVR, WPW
Cannon A Waves	Present	Absent
Arterial Pulse	Variation	No variation
First heart sound	Variable	Not variable
Fusion Beats	Present	Absent
AV dissociation	Present	Absent
QRS	>0.14sec	<0.14sec
Axis	Extreme LAD (< -30)	Normal or slightly abnl
Vagal Maneuvers	No response	Slows or terminates
QRS morphology	V1 - R or qR	V1 - rsR'
(RBBB-like pattern)	V6 - rS	V6 - R(slurredS)

نکات مهم و دوزها در تاکیکاردی با نبض بزرگسالان

- **کاردیوورژن سینکرونایز**

دوز های توصیه شده برای شروع : (با روشن کردن دکمهٔ sync دستگاه دفیبریلاتور)

- باریک و منظم : ۵۰ تا ۱۰۰ ژول • باریک و نامنظم : ۱۲۰ تا ۲۰۰ژول بای فازیک یا ۲۰۰ژول مونوفازیک
- پهن و منظم : ۱۰۰ژول • پهن و نا منظم : دوز دفیبریلاسیون(غیرسینکرونایز)

- **دوز IV آدنوزین :** دوز اول 6mg به صورت تزریق سریع وریدی و به دنبال آن تزریق 20cc N/S

دوزدوم درصورت نیاز 12 mg

- **دوز IV کلسیم کانال بلاکر ها :**
  - **وراپامیل** با دوز 2.5-5mg بلوس درعرض ۲دقیقه وتکرارآن هر۱۵تا ۳۰دقیقه 5-10mg تا سقف دوز 20 mg
  - **دیلتیازم** با دوز 15-20mg درعرض۲ دقیقه و در صورت عدم تأثیر 25-30mg درعرض ۱۵ دقیقه و دوز انفوزیون 5-15 mg/hour
- **دوز IV ایندرال :** 0.1mg/kg با تقسیم در ۳ دوز مساوی و هر ۲ تا ۳ دقیقه به صورت آهسته و رقیق شده تزریق می شود.(نکته : سرعت تزریق ایندرال نباید از 1mg/min بیشتر باشد ، ضمنا تجویز بتابلاکرها در بیماران ریوی و CHF باید با احتیاط صورت گیرد.)

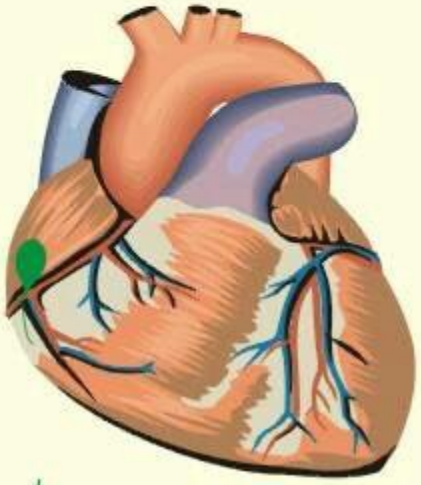
- **انفوزیون آنتی آریتمی برای تاکیکاردی پایدار باQRS پهن :**
  - **دوزIV پروکائین آمید :** 20-50mg/min تا ازبین رفتن آریتمی ، ایجاد هیپوتانسیون ، افزایش بیش از ۵۰% زمان QRS یا سقف دوز 17mg/kg
  - **دوزIV آمیودارون :** دوز اول 150mg درعرض ۱۰دقیقه

وتکرار درصورت برگشت VT و بدنبال آن انفوزیون 1mg/min برای ۶ ساعت اول

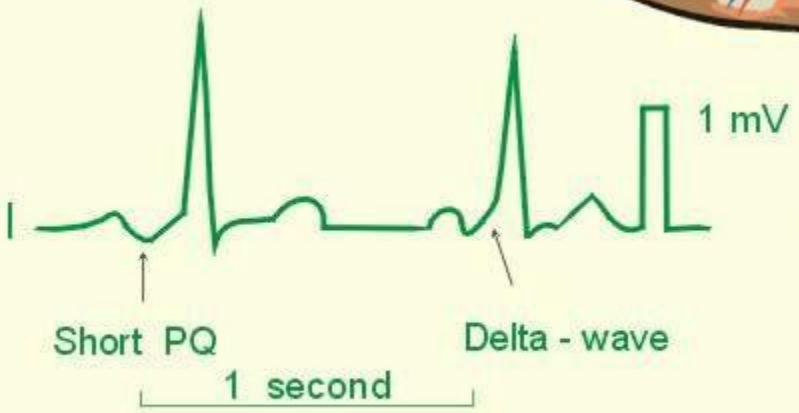
- **دوز IV سوتالول :** 100mg(1.5mg/kg) در عرض ۵ دقیقه و پرهیز از تجویز آن در صورت QT طولانی

Defibrillation	Cardioversion
Not synchronised	Synchronised on the R wave
For cardiac arrest	For periarrest tachyarrhythmias (unstable)
Higher energy joules	Lower energy joules
No escalating energy for next shock	Escalate for next shock (100 - 200 - 300 - 360J)

WPW- Block & Long Qt-syndrome



WPW - Block



The Long Q T - Syndrome

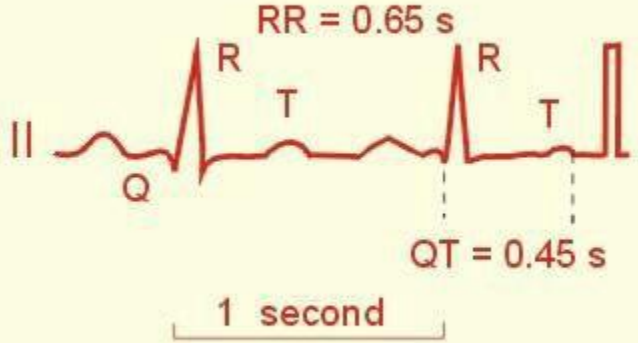


Fig. 11-14

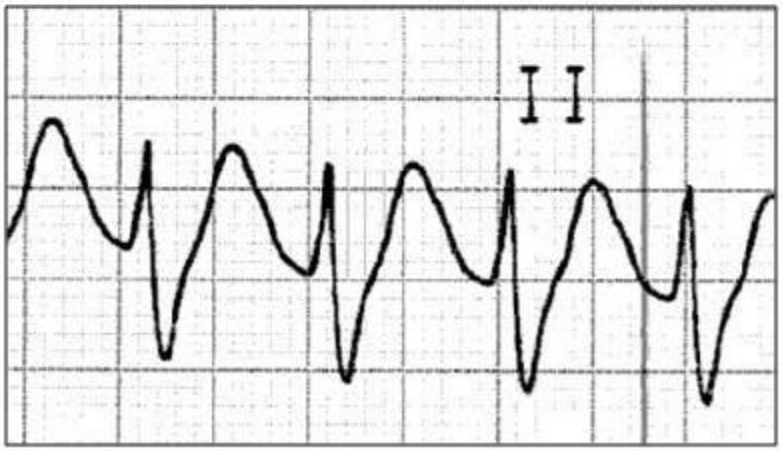
KMc

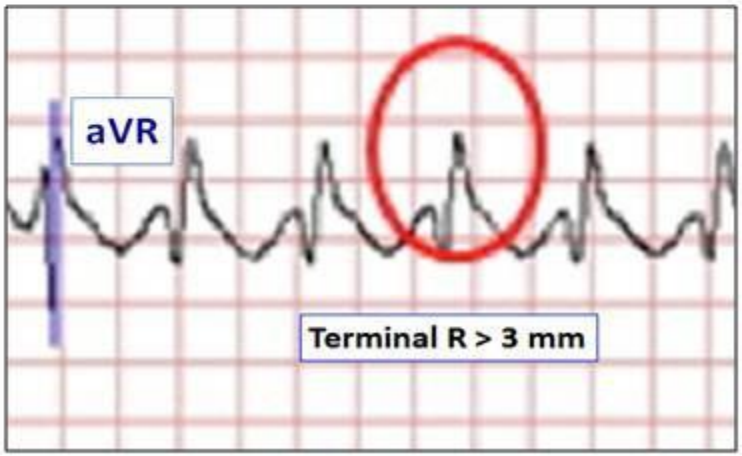
Tricyclic Overdose (Sodium-Channel Blocker Toxicity)

- **sodium-channel blocking** medications such as:
  - Tricyclic antidepressants (= **most common**)
  - Type Ia antiarrhythmics (quinidine, procainamide)
  - Type Ic antiarrhythmics (flecainide, encainide)
  - Local anaesthetics (bupivacaine, ropivacaine)
  - Antimalarials (chloroquine, hydroxychloroquine)
  - Dextropropoxyphene
  - Propranolol
  - Carbamazepine
  - Quinine

ECG Findings Tricyclic Overdose

- Interventricular conduction delay (IVCD) — QRS > 100 ms in lead II
- Right axis deviation (RAD)
- Terminal R wave > 3 mm in AVR
- R/S ratio > 0.7 in AVR





Clinical Features of Tricyclic Overdose

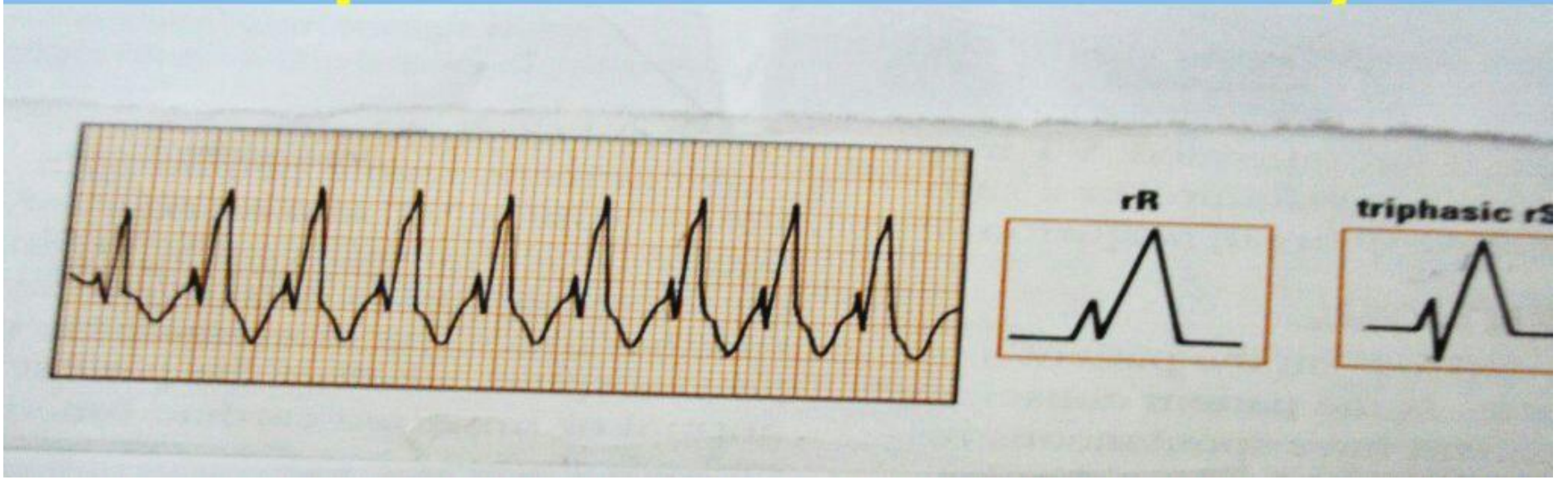
In overdose, the tricyclics produce rapid onset (within 1-2 hours) of:

- Sedation and coma
- Seizures
- Hypotension
- Tachycardia
- Broad complex Dysrhythmia
- Anticholinergic syndrome

In V_1 or MCL_1 If wide and positive QRS

If the QRS complex is triphasic similar to a RBBB

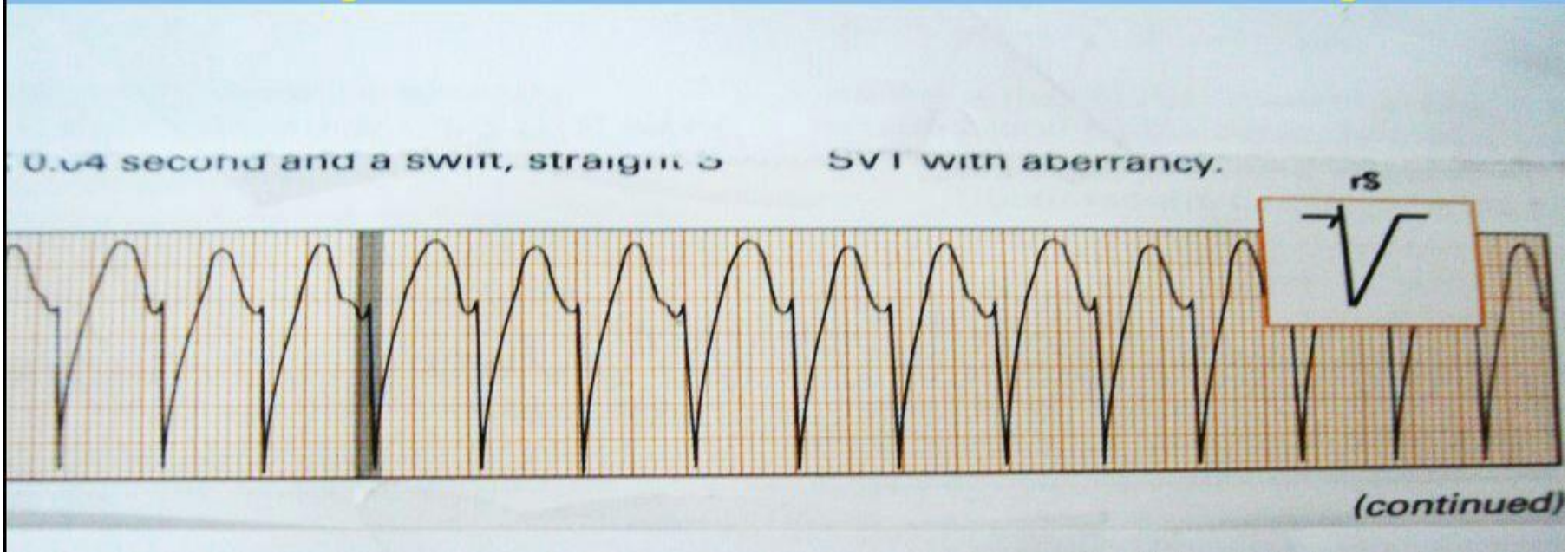
Suspect SVT with aberrancy



In V_1 or MCL_1 If wide and negative QRS

- If the QRS complex has an R wave < 0.04 s and swift and straight on the downstroke

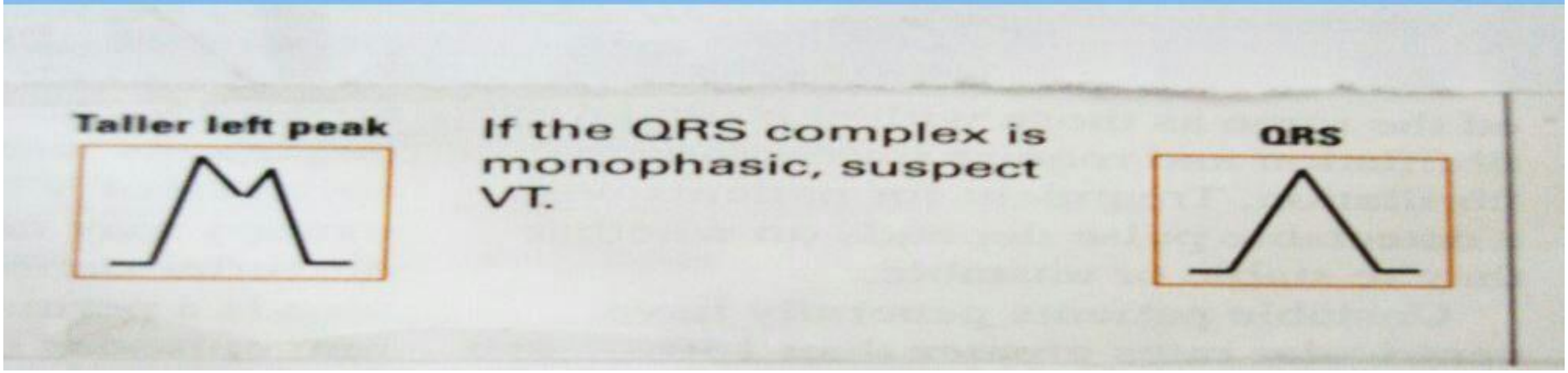
Suspect SVT with aberrancy



In V_1 or MCL_1 If wide and positive QRS

If the QRS complex is tall and shaped like rabbit ears with the left peak taller than the right and monophasic QRS

Suspect VT



انواع S.A. BLOCK

- درجه 1
- درجه 2
  - Type 1
  - Type 2
- درجه 3

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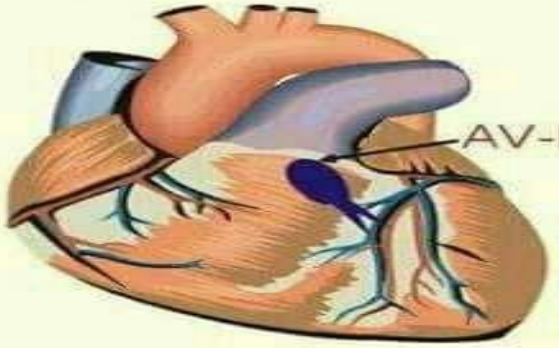
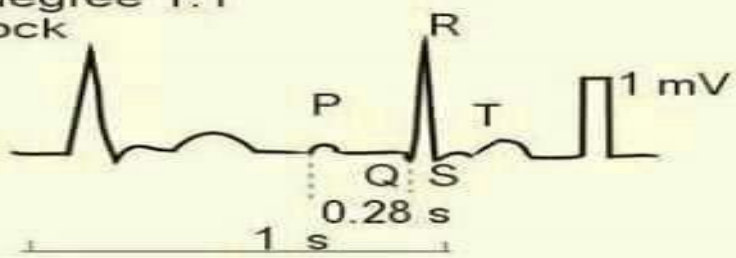
Four Types of AV-block

(PQ interval > 0.2 s)

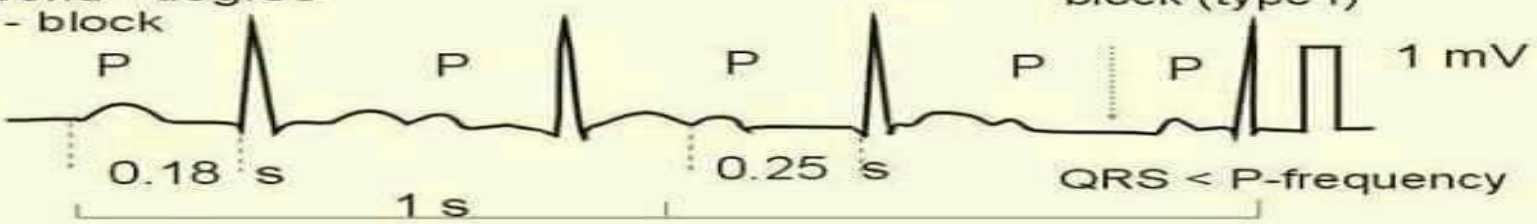


First - degree 1:1
AV - block



Second - degree
AV - block

Wenchebach
block (type I)



Second - degree
AV - block

Mobitz II block (no warning)

Third - degree
AV - block

Complete AV - block (Adam Stokes disease
in AV or His-bundle

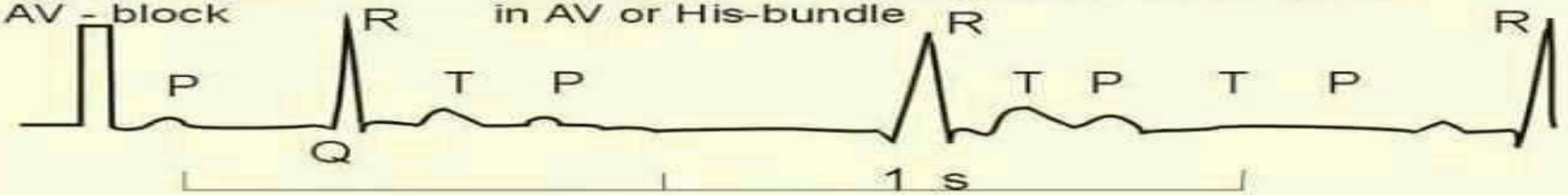


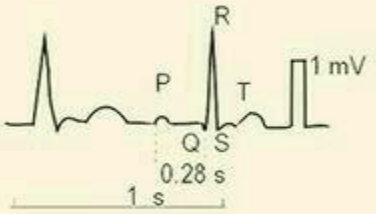
Fig. 11-12

KMc

The Heart Block Poem

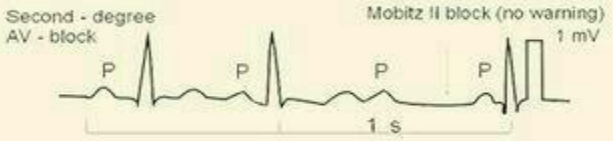
by the Princeton Surgical Group & nursesLabs

If the **R** is far from **P**,
then you have a **FIRST DEGREE**.

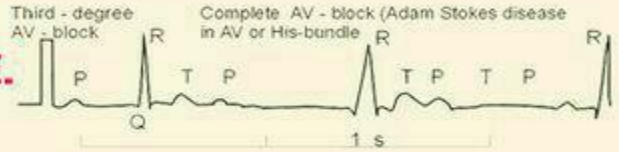


Longer, longer, longer, drop!
Then you have a **WENKEBACH**.

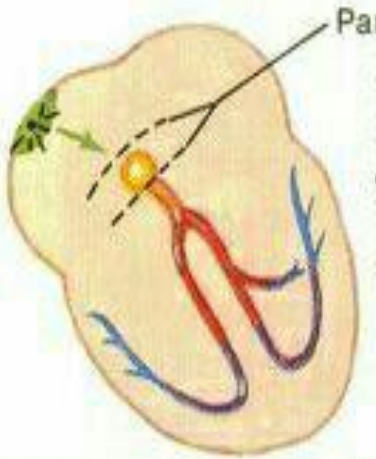
If some **P**s don't get through,
then you have **MOBITZ II.**

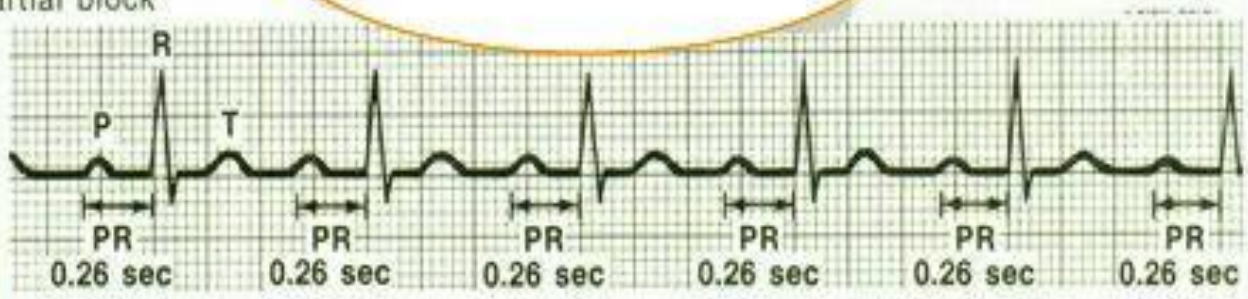


If **P**s and **Q**s don't agree,
then you have a **THIRD DEGREE.**



1° AV Block





Rhythm: regular

Rate: (that of underlying rhythm)

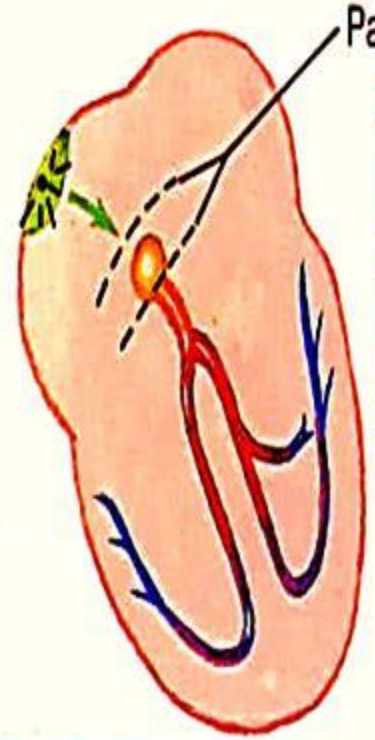
PRI is > than .2 seconds

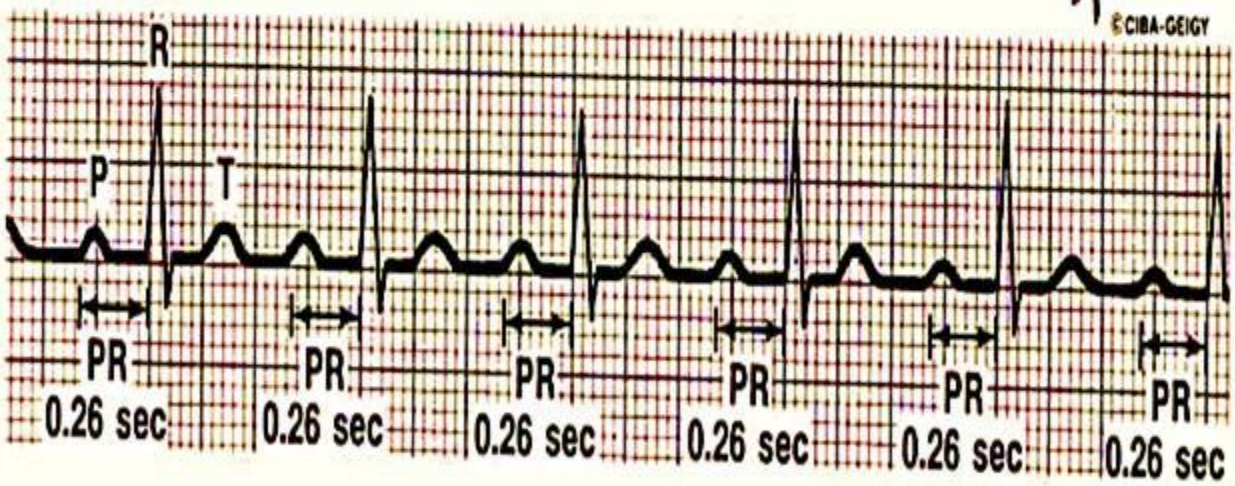
QRS: usually normal

AVB I

D. Fixed but prolonged PR interval

First-degree AV block





F. Netter M.D.

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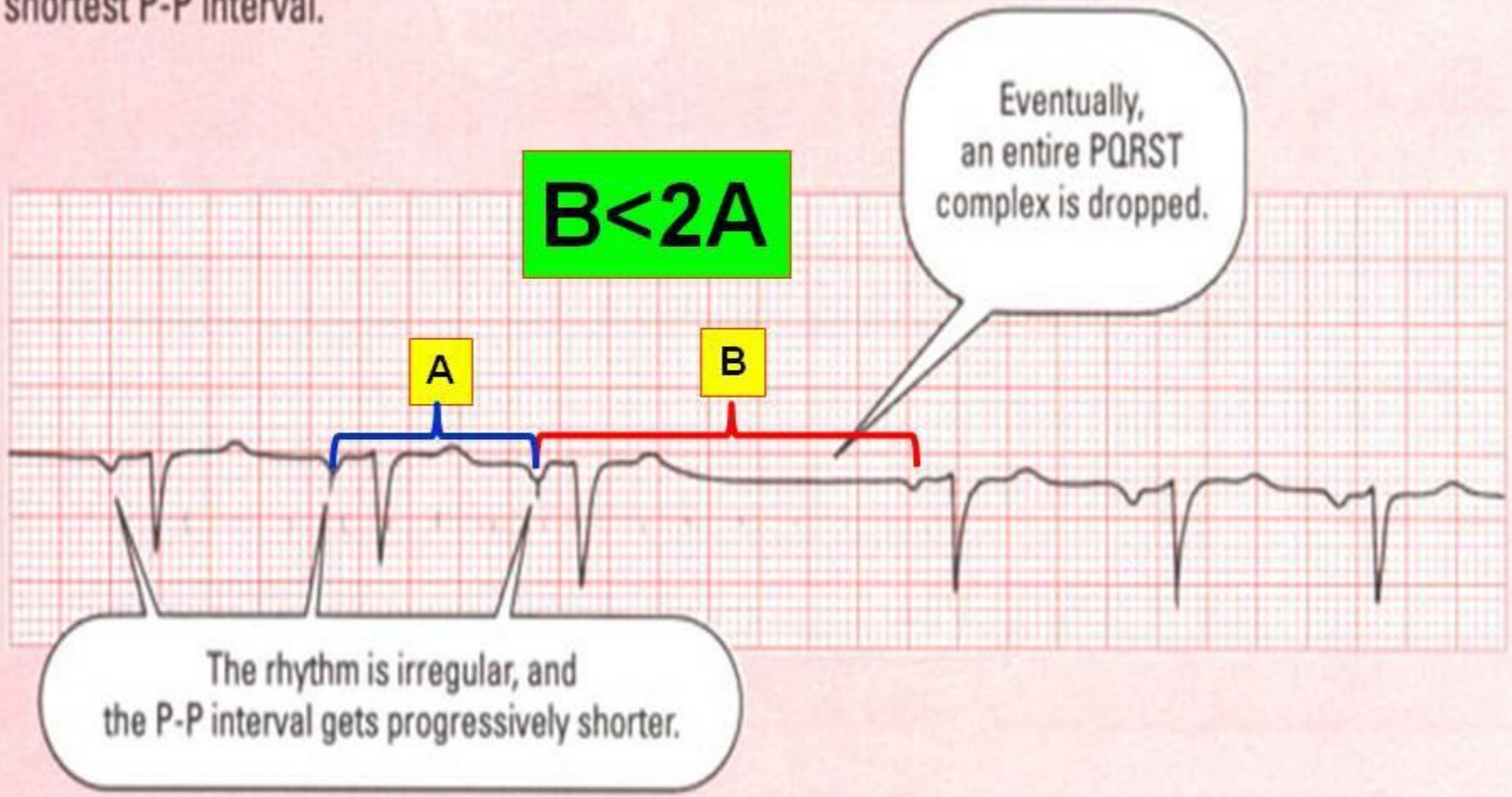
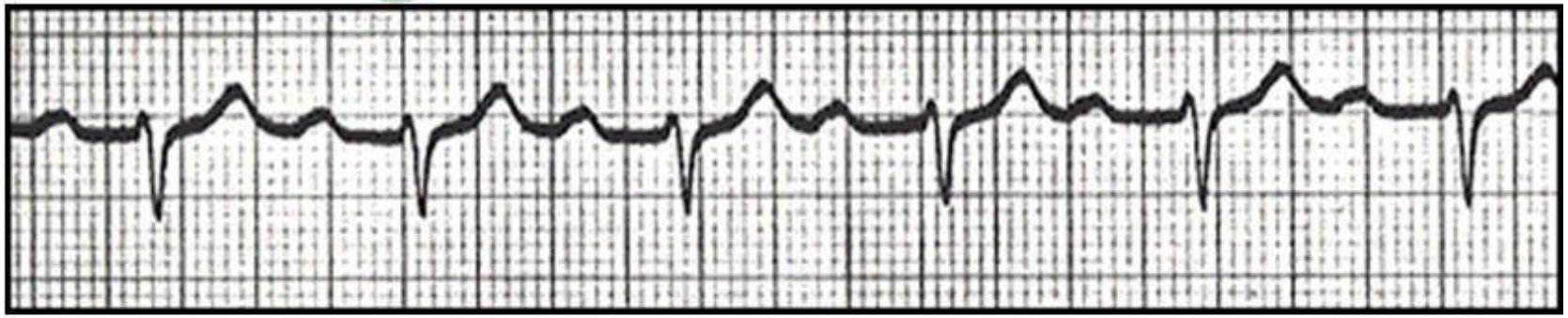
P wave precedes each QRS complex but PR interval, although uniform, is >0.2 second (>5 small boxes)

Second-degree type I block

In this type of SA block, conduction time between the sinus node and the surrounding atrial tissue becomes progressively longer until an entire cycle is dropped. The pause is less than twice the shortest P-P interval.

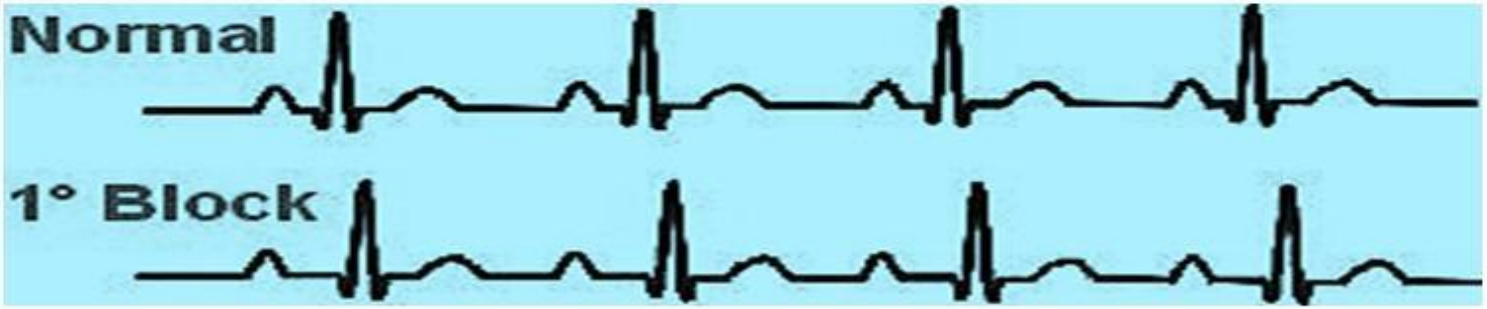


1st Degree AV Block

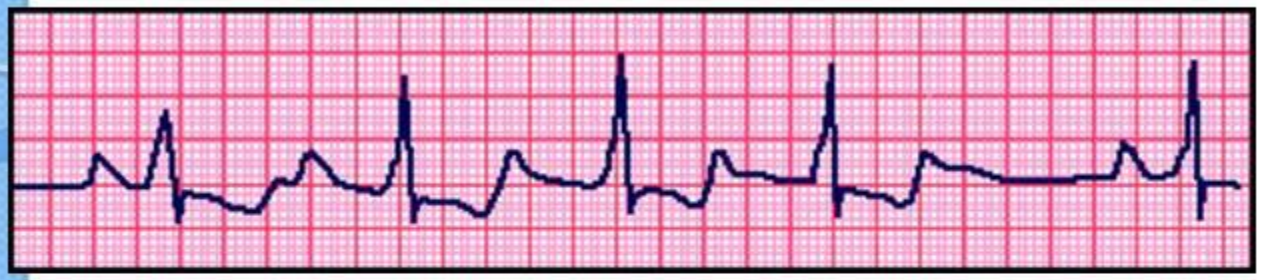


EKG Characteristics: Prolongation of the PR interval, which is constant

All P waves are conducted



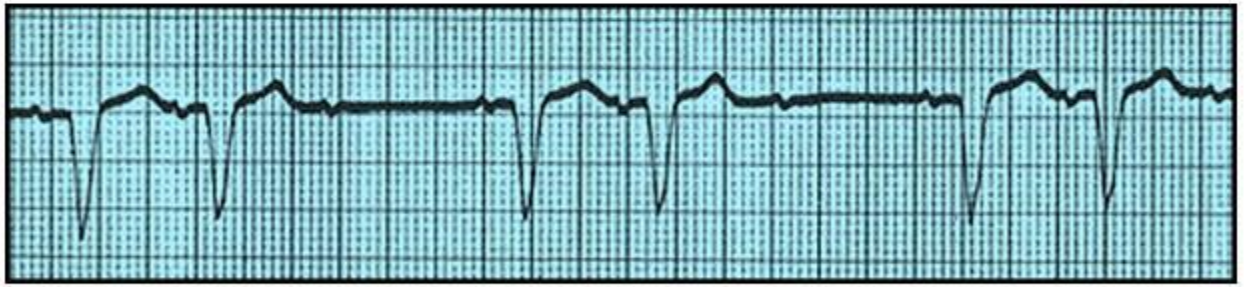
2nd Degree AV Block



Type 1
(Wenckebach)

EKG Characteristics: Progressive prolongation of the PR interval until a P wave is not conducted.

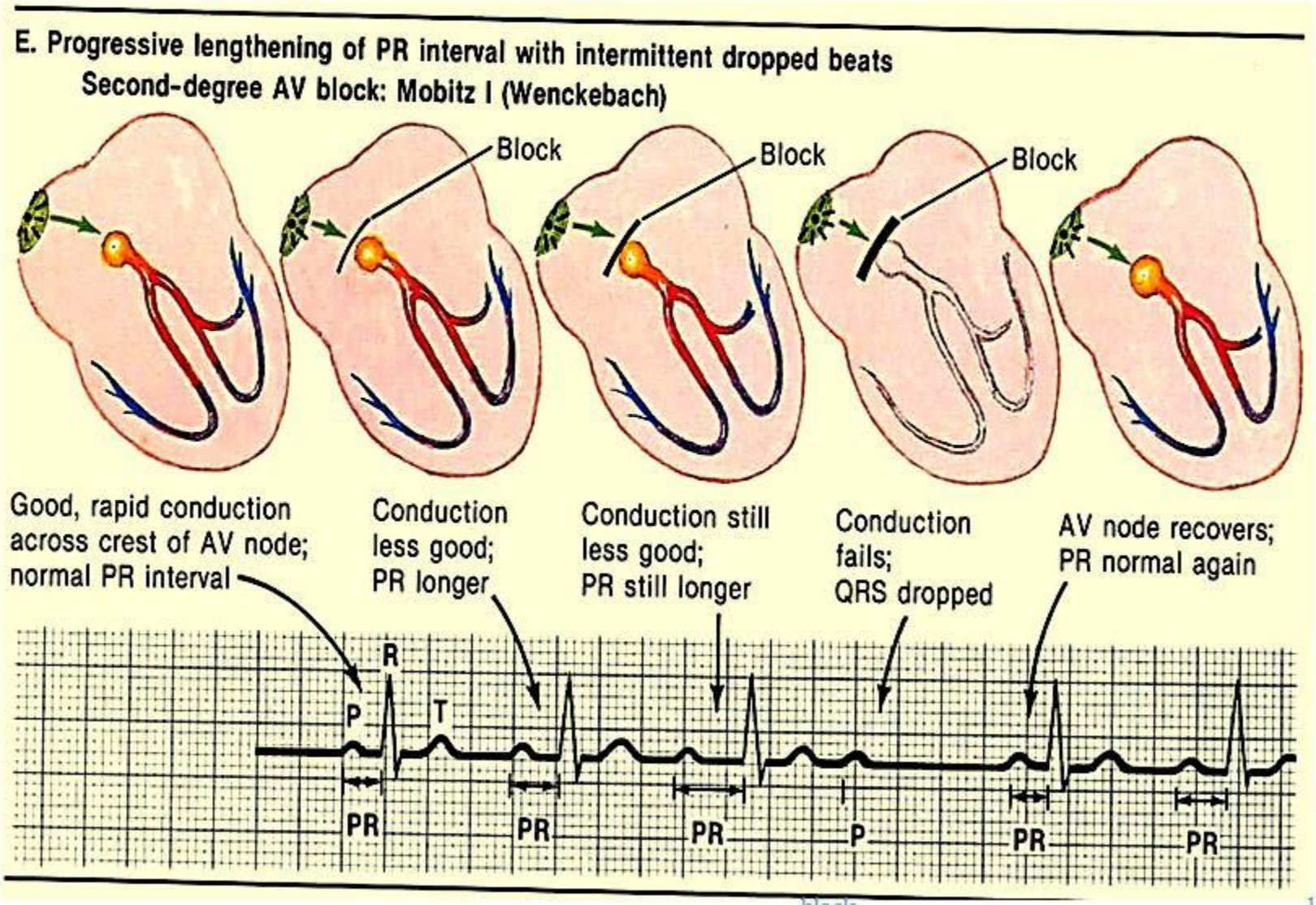
As the PR interval prolongs, the RR interval actually shortens



Type 2

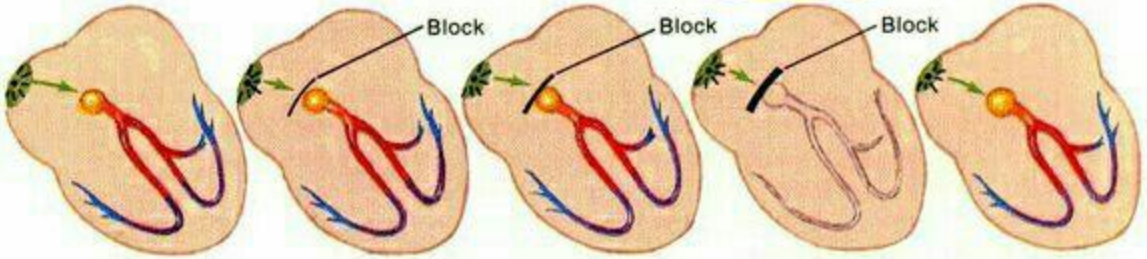
EKG Characteristics: Constant PR interval with intermittent failure to conduct

بلوك درجه II (ونكباخ)

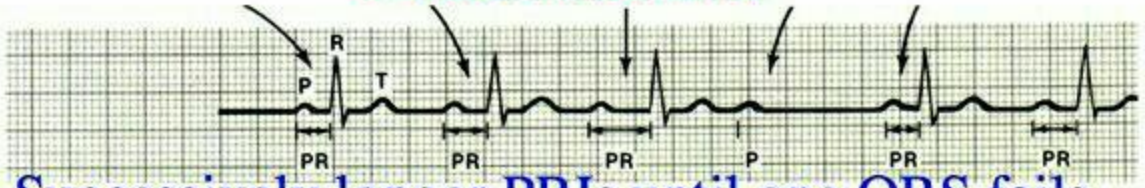


2° AV Block Mobitz I

گروه پرستاران



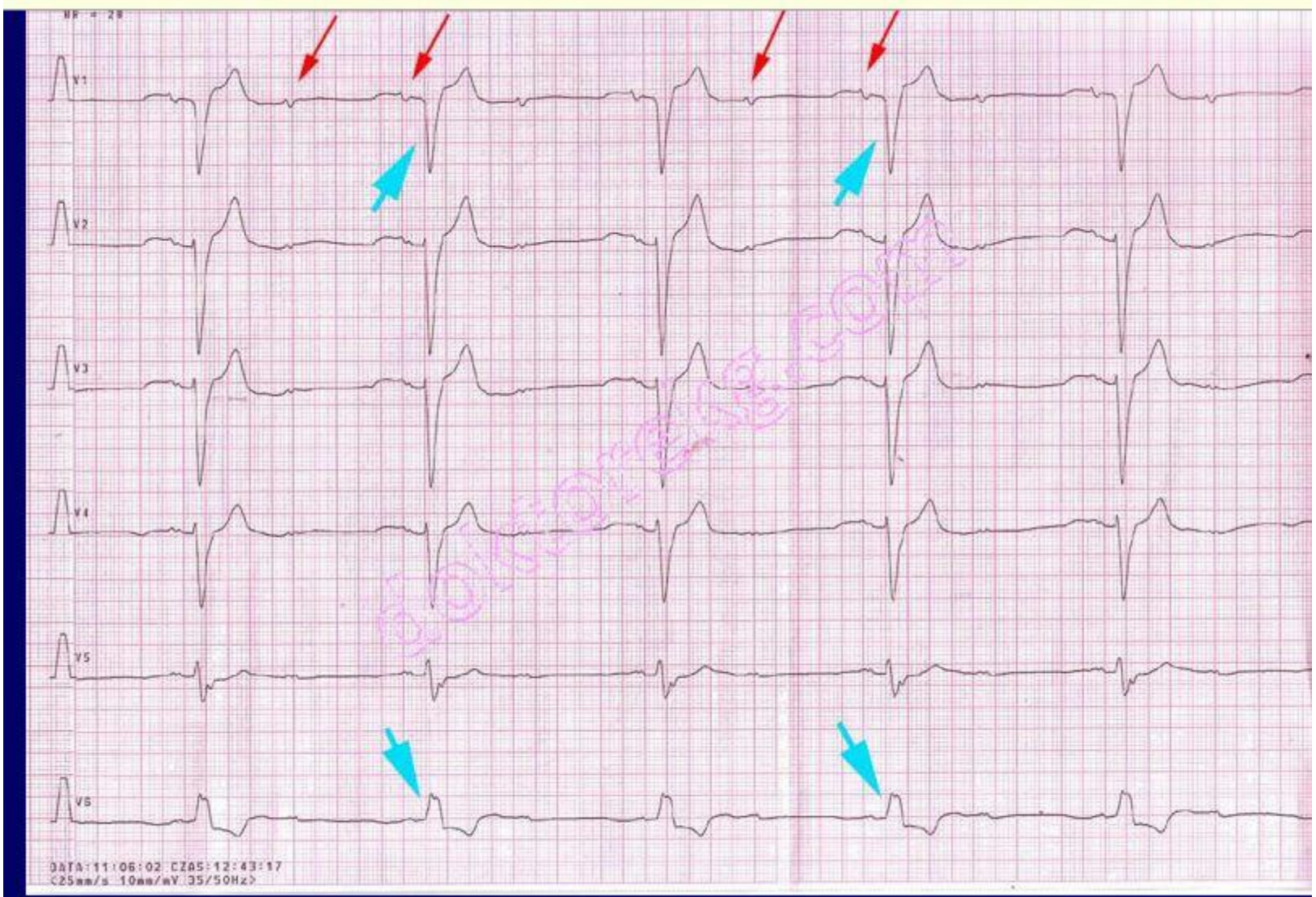
P Waves look Similar!



Successively longer PRIs until one QRS fails

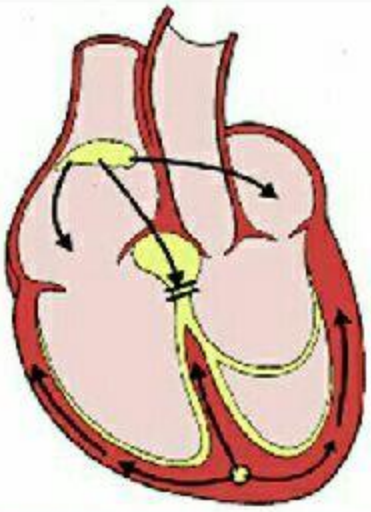
Rhythm (ventricular) is often irregular

Atrial rhythm is ~ **regular,** QRS is normal

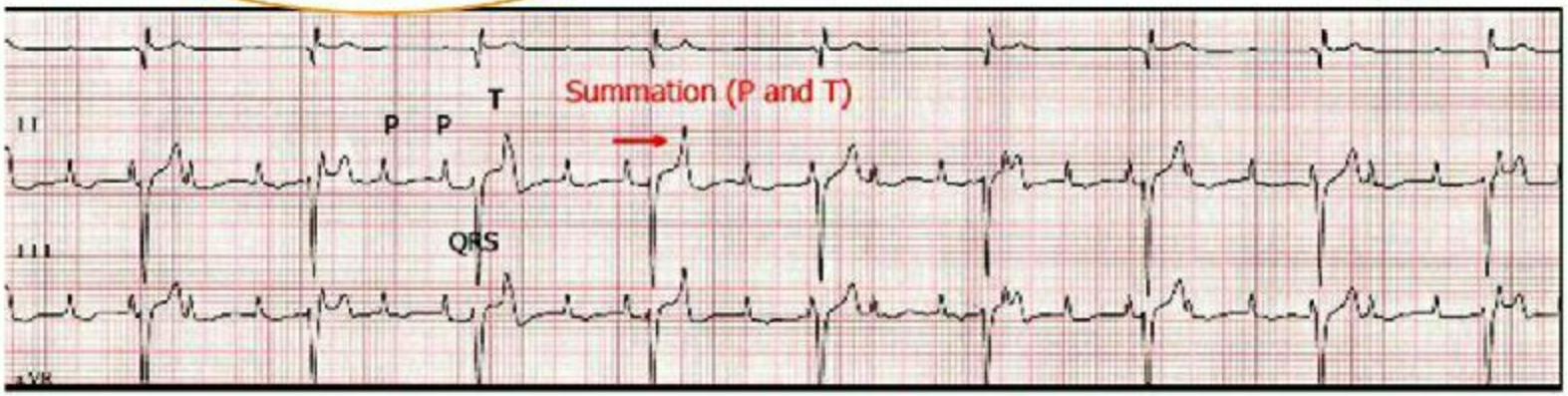


ECG 2. Mobitz Type 2 second degree AV block. This is a 2:1 block and the presence of wide QRS complex supports the diagnosis of Mobitz type 2 block. Of every 2 P wave s, only 1 is conducted to the ventricles.

Third degree AV block or complete AV block



گروه پرستاران



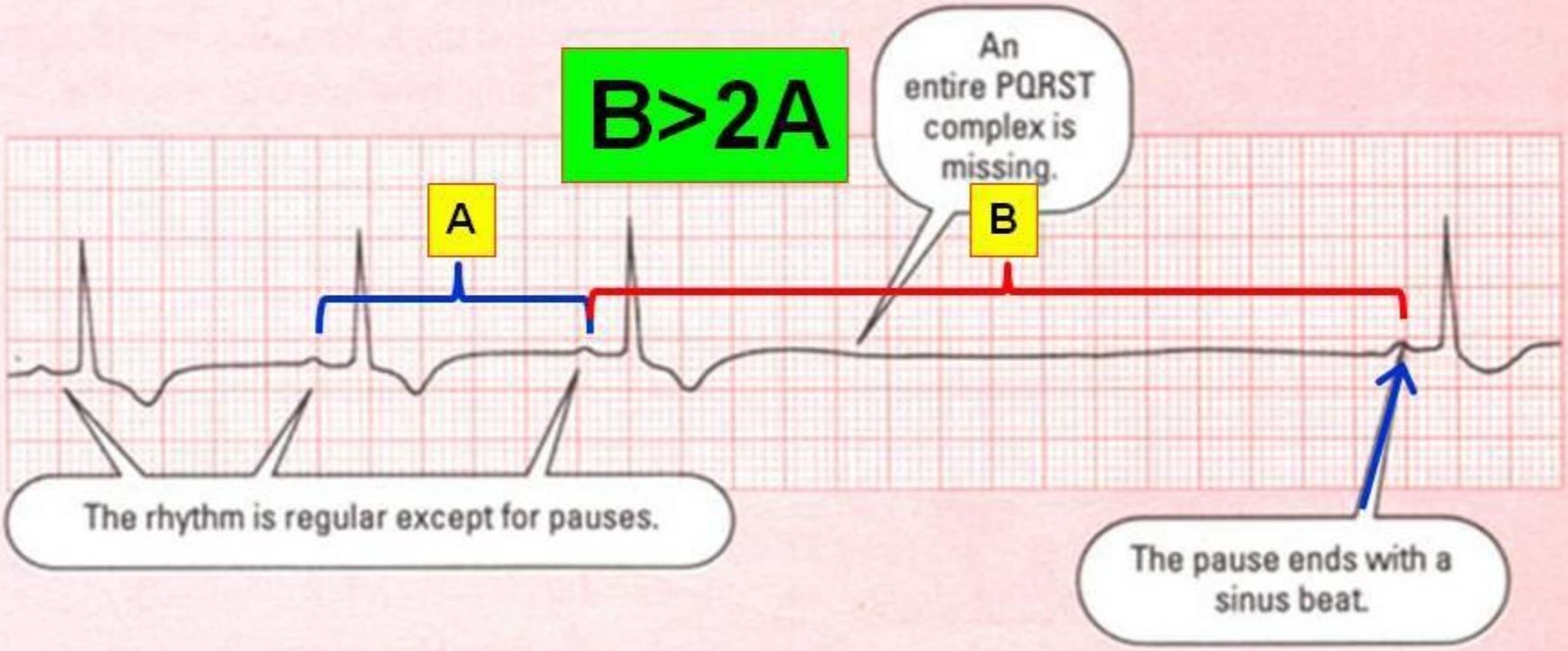
*None of P waves conduct to ventricles (P-P and QRS-QRS are independent)
*Slow, regular ventricular escape rhythm

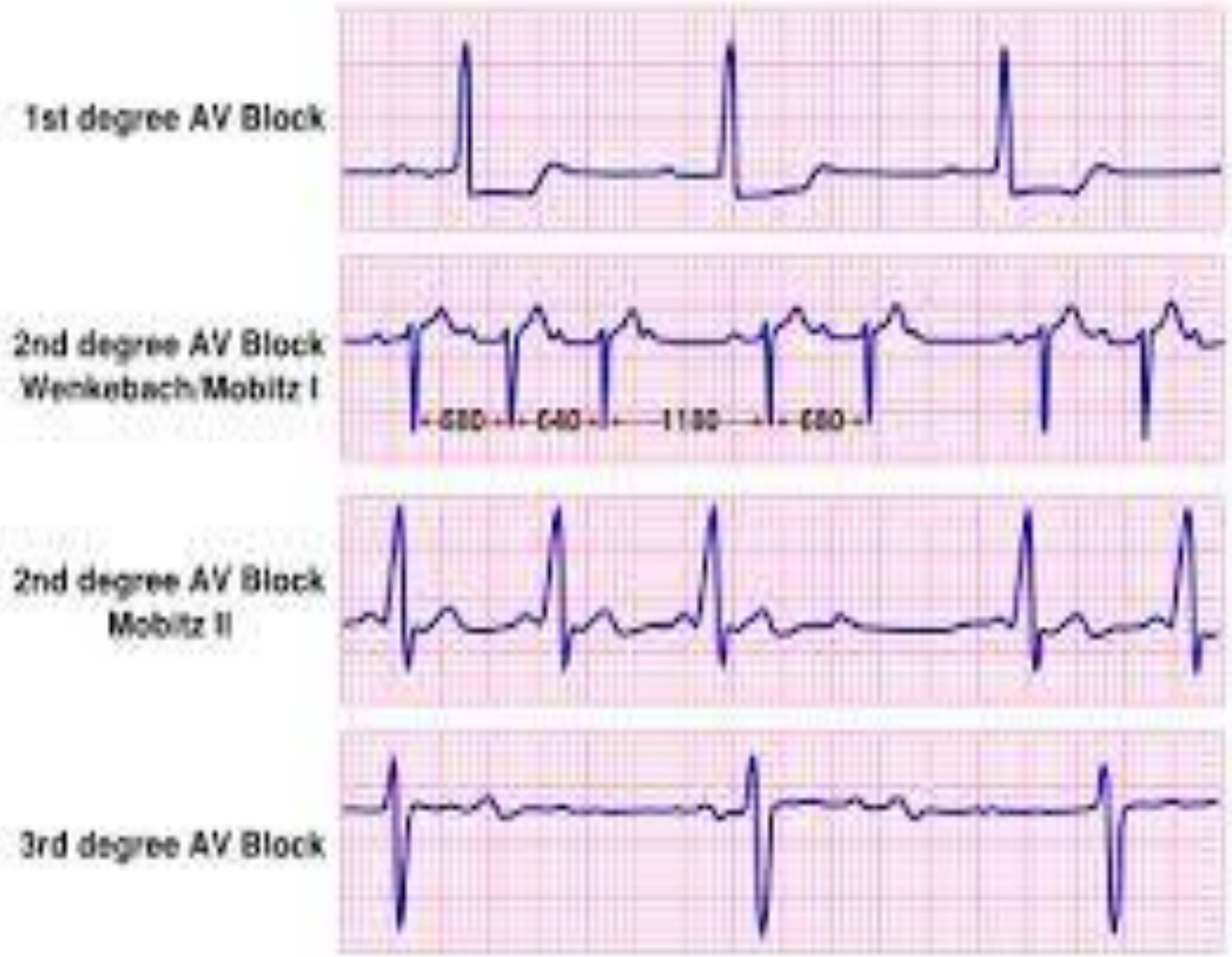
Third-degree S.A. block

In this arrhythmia, some impulses are blocked, causing long sinus pauses. The pause isn't a multiple of the sinus rhythm. On an ECG, third-degree SA block looks similar to sinus arrest but results from a different cause.

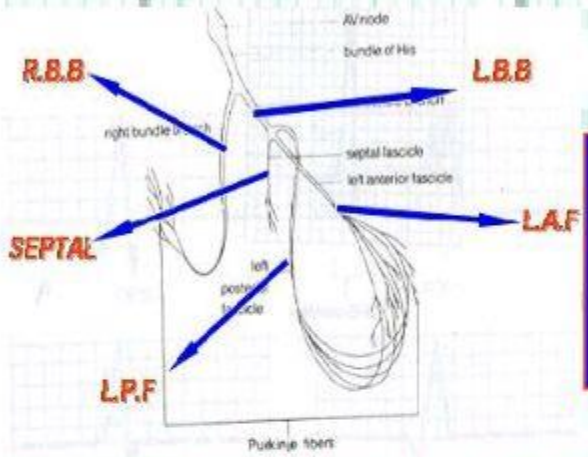
Third-degree SA block is caused by a failure to conduct impulses; sinus arrest results from failure to form impulses. Failure in each case causes atrial activity to stop.

In sinus arrest, the pause often ends with a junctional escape beat. In third-degree block, the pause lasts for an indefinite period and ends with a sinus beat.




1st degree AV Block
2nd degree AV Block
Wenkebach/Mobitz I
680
640
1180
680
2nd degree AV Block
Mobitz II
3rd degree AV Block

BUNDLE BRUNCH BLOCK



RBBB-1
LAHB-2
LPHB-3

RBBB+LAHB - 1
RBBB+LPHB - 2
RBBB+FDAVB - 3
LBBB - 4

RBBB+LAHB+FDAVB-1
RBBB+LPHB+FDAVB-2
LBBB+FDAVB-3

1- یک شاخه
Monofascicular

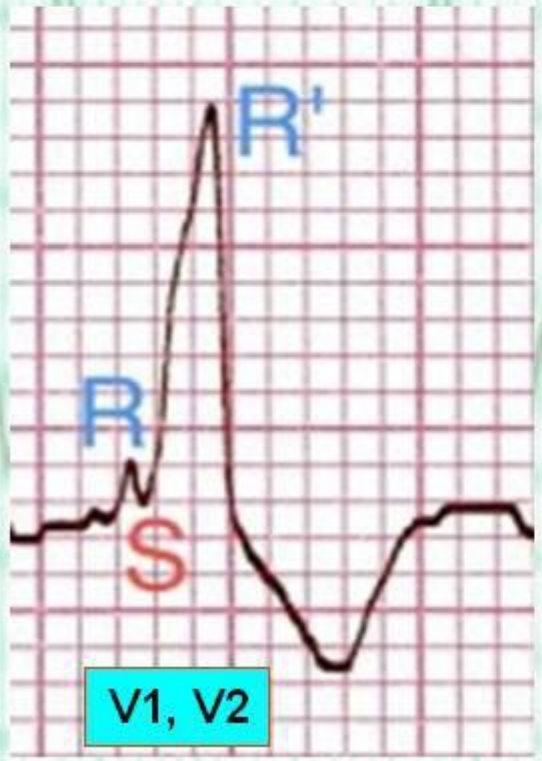
2- دو شاخه
Bifascicular

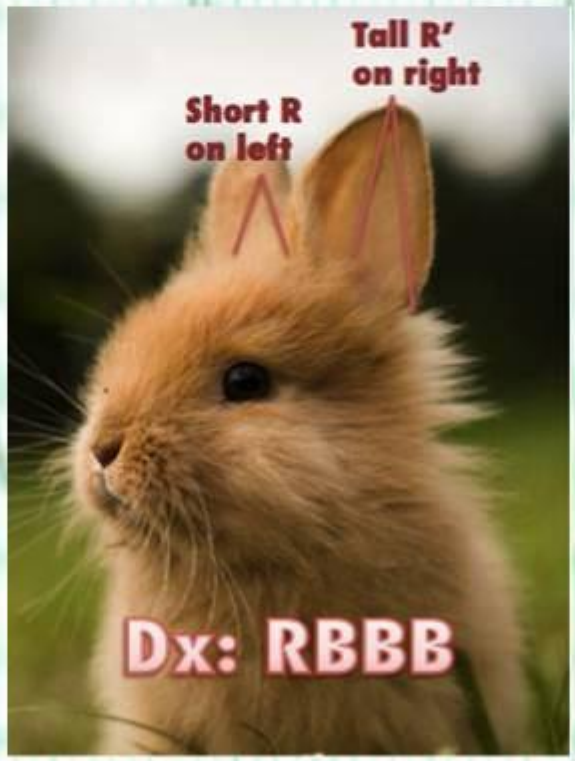
3- سه شاخه
Trifascicular

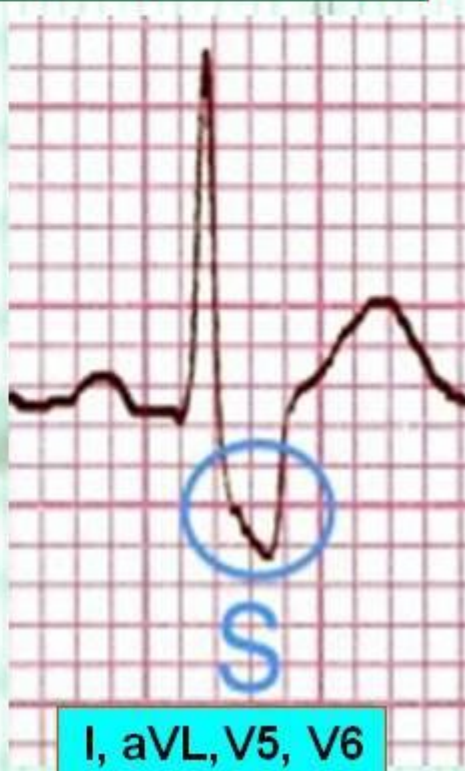
انواع B.B.B

RBBB

- Broad QRS > 120 ms
- RSR' pattern in V1, V2 ('M-shaped' QRS)
- Wide, slurred S wave in (I, aVL,V5, V6)
- ST depression and T wave inversion in (V1, V2, V3)







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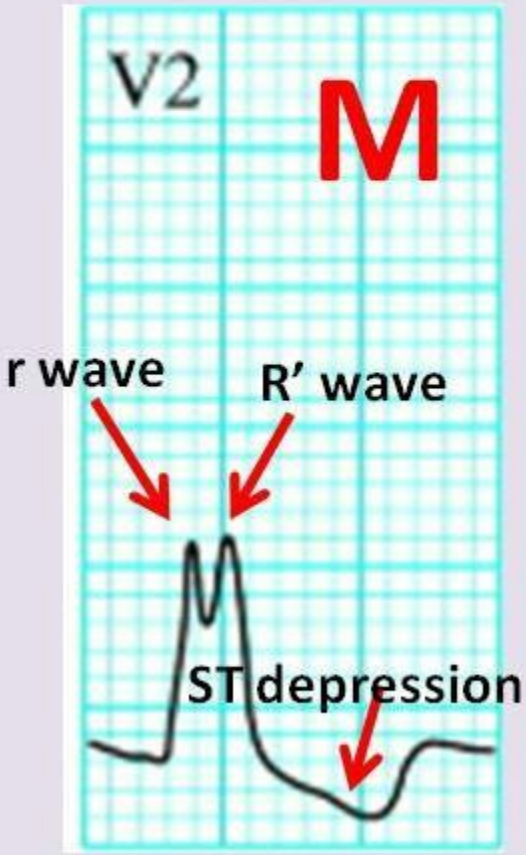
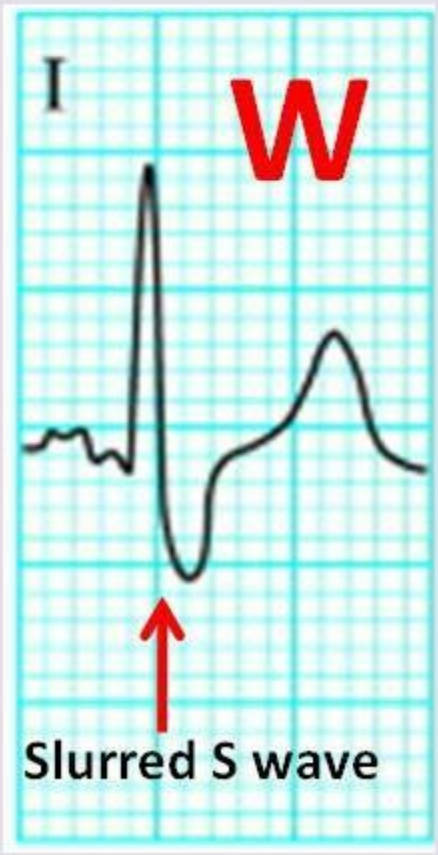
Right Bundle Branch Block (MARROW)

Lead V2: Shows the characteristic secondary R' wave in a complex known as **r S R'** . The R' is late right ventricular depolarisation. Note the M shape of Marrow

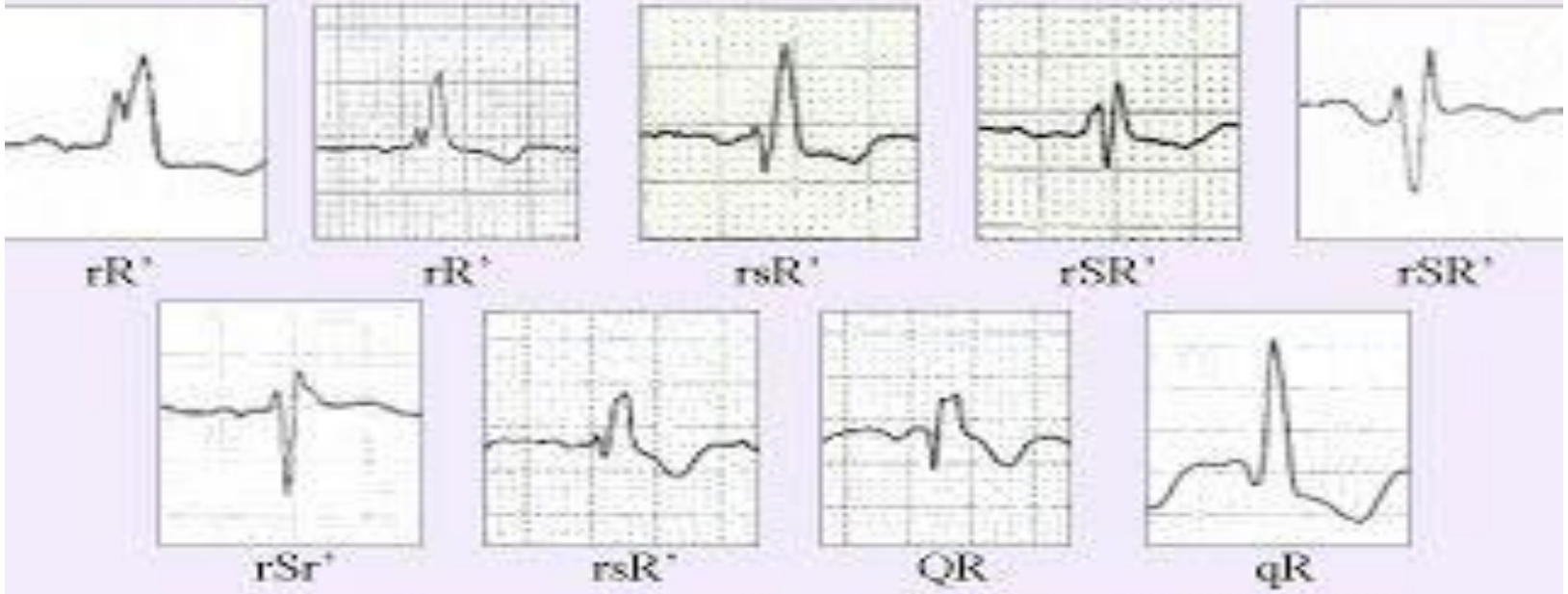


R



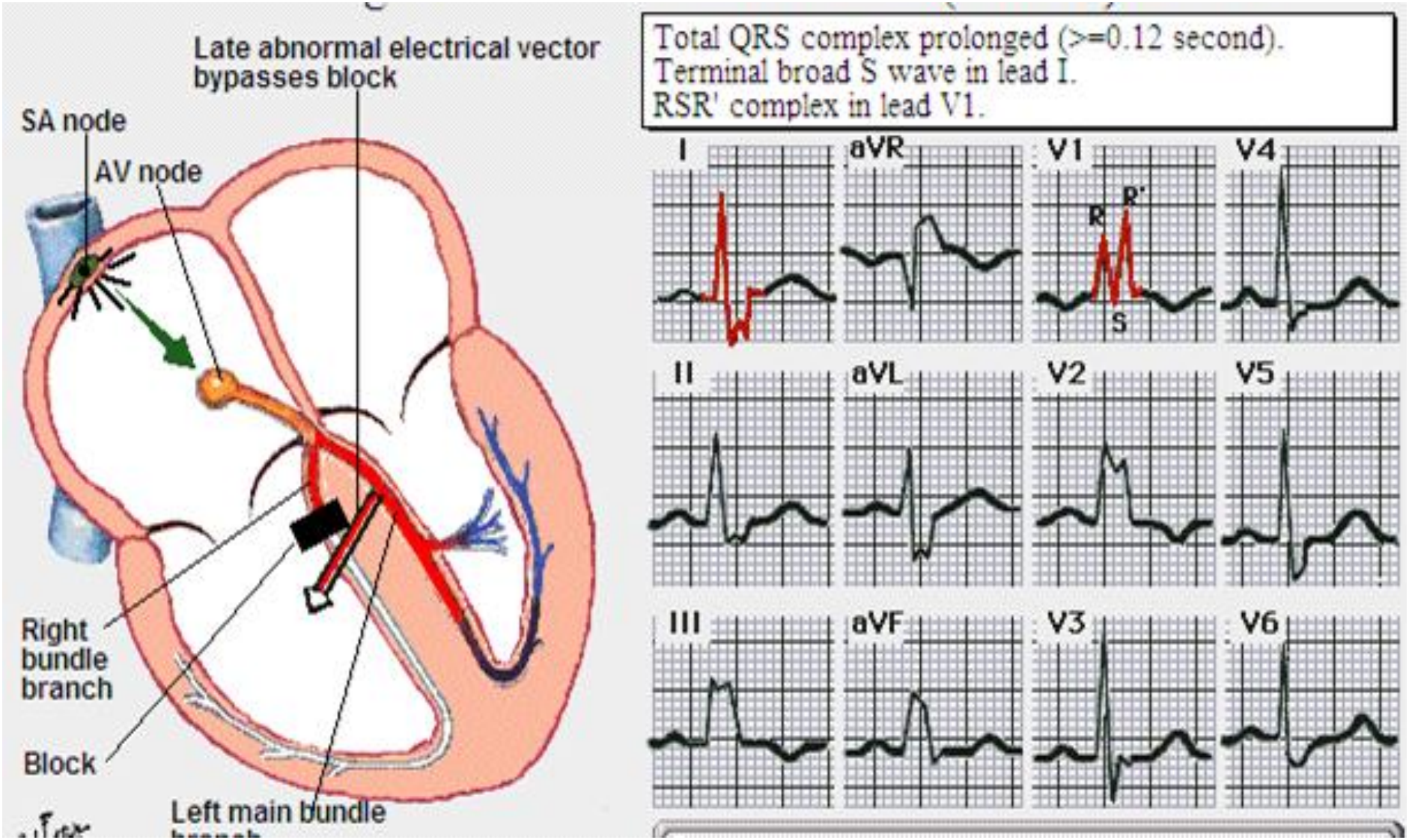
Lead I: Shows the characteristic slurred S wave which is how delayed right ventriclular depolarisation manifests in the lateral leads. Note the W shape of marroW.

Various RBBB Morphologies Lead V1

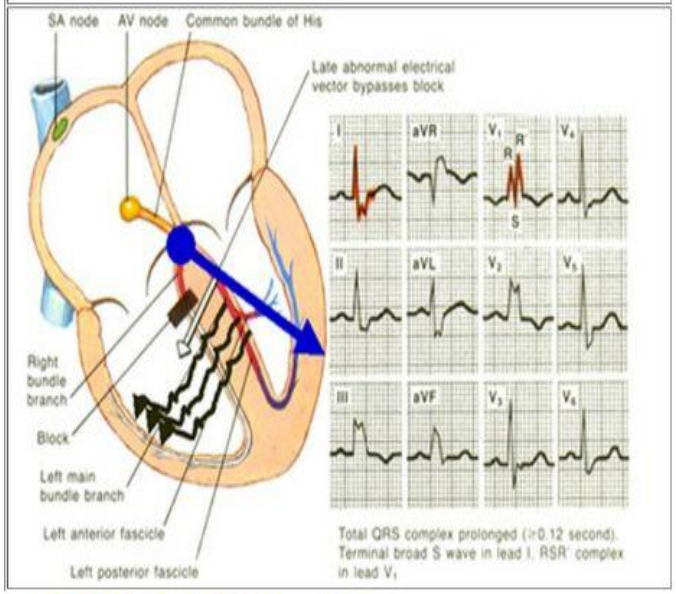


ems12lead.blogspot.com

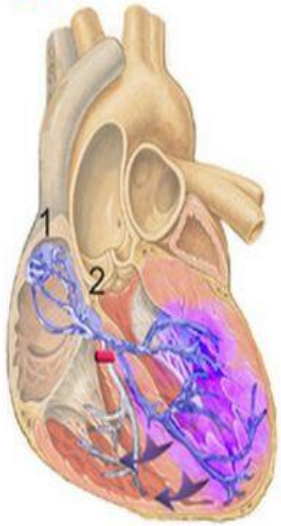
Complete RBBB



Right Bundle Branch Block



Right Bundle Branch Block (RBBB)



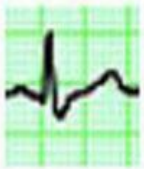
Dr.Nabil Paktin , MD.,Cardiology Case series

www.afghanheart.wordpress.com

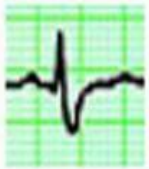
RBBB Criteria (Check QRS 1st)

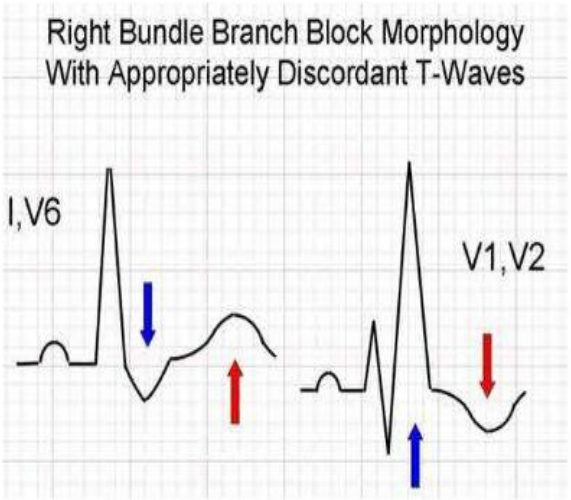
- **Look in V_1 & V_2**
  - R, R^1 wave!
- **Look in V_5, V_6, & Lead I**
  - "slurred S wave"

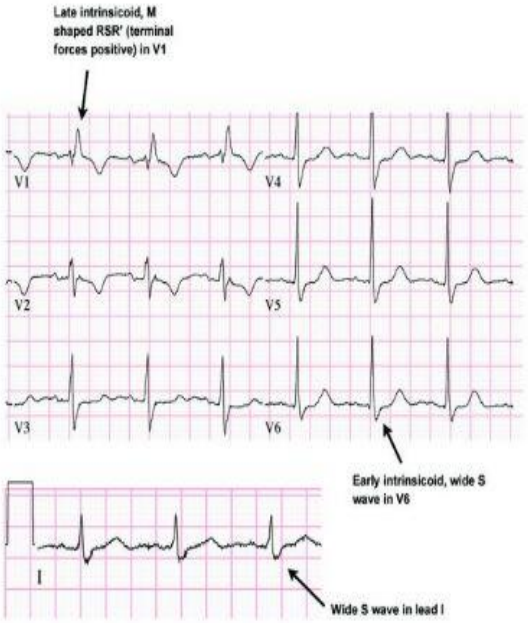
V_6



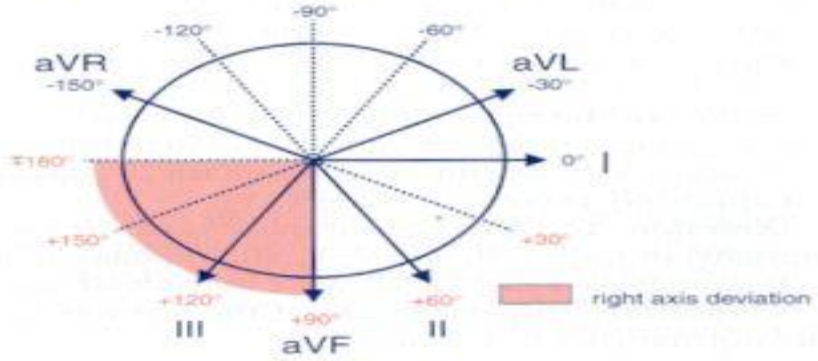
Lead I



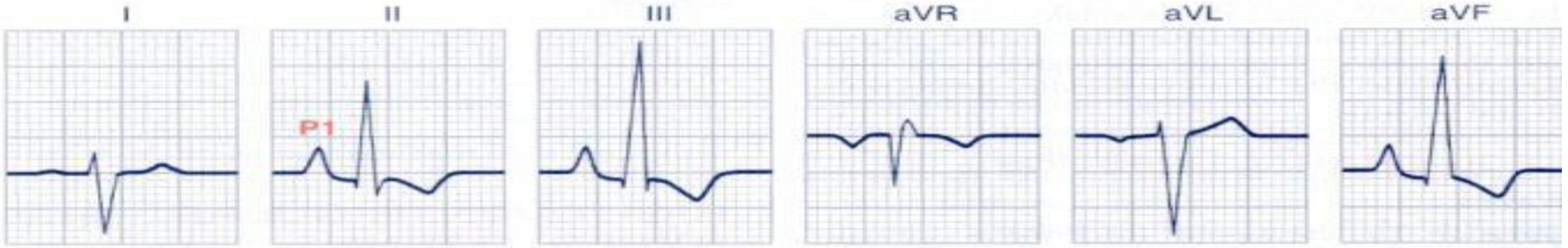


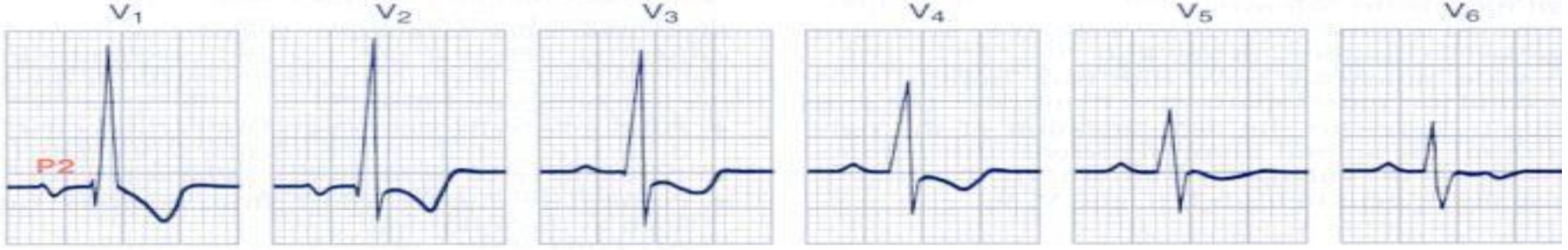


Right Ventricular Hypertrophy

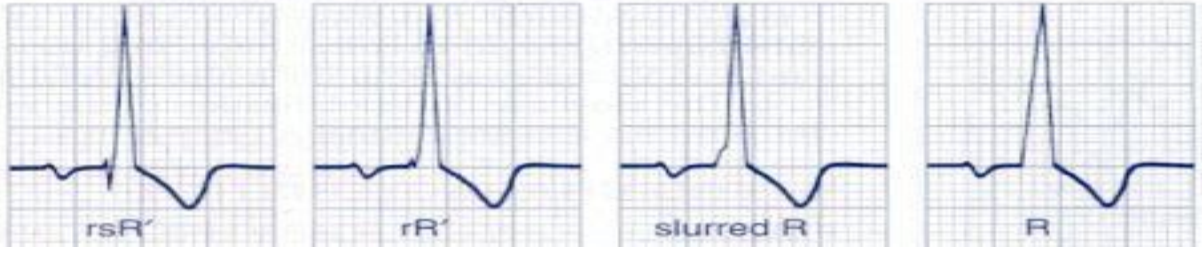


P1: P pulmonale
P2: biphasic P wave



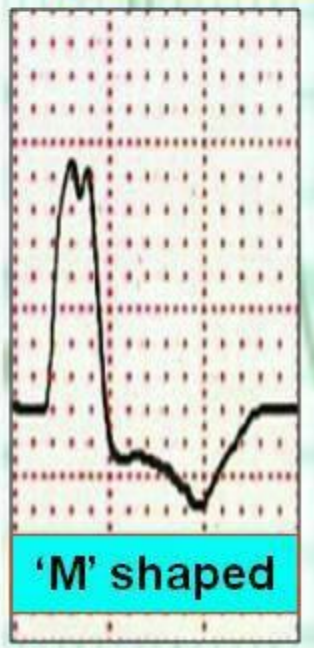


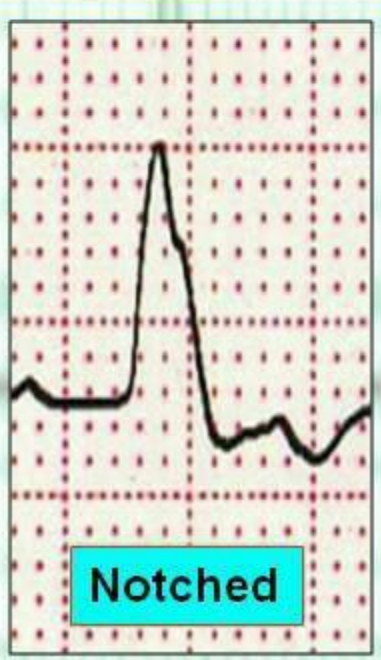
variations in the initial portion of the QRS complex in lead V_1

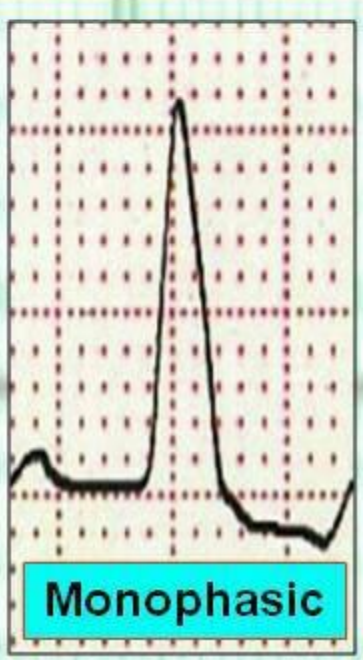


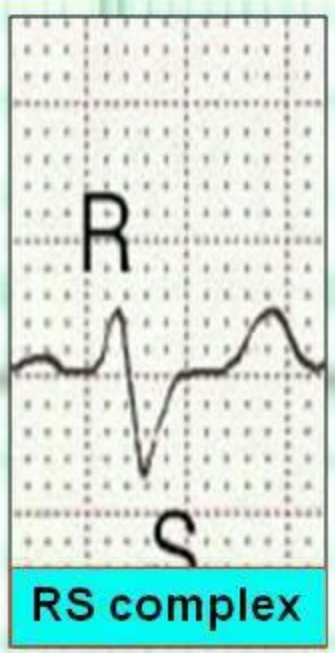
LBBB

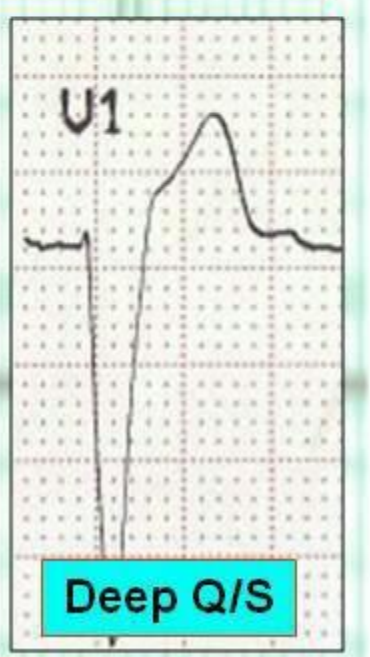
- QRS duration of 120 ms
- Deep Q/S wave with no preceding R wave(V1,V2,V3,V4)
- Wide monophasic R wave in lateral leads (I, aVL, V5-V6)
- QRS Morphology in I,aVL,V5,V6('M' shaped, Notched, Monophasic, RS complex)
- Left axis deviation











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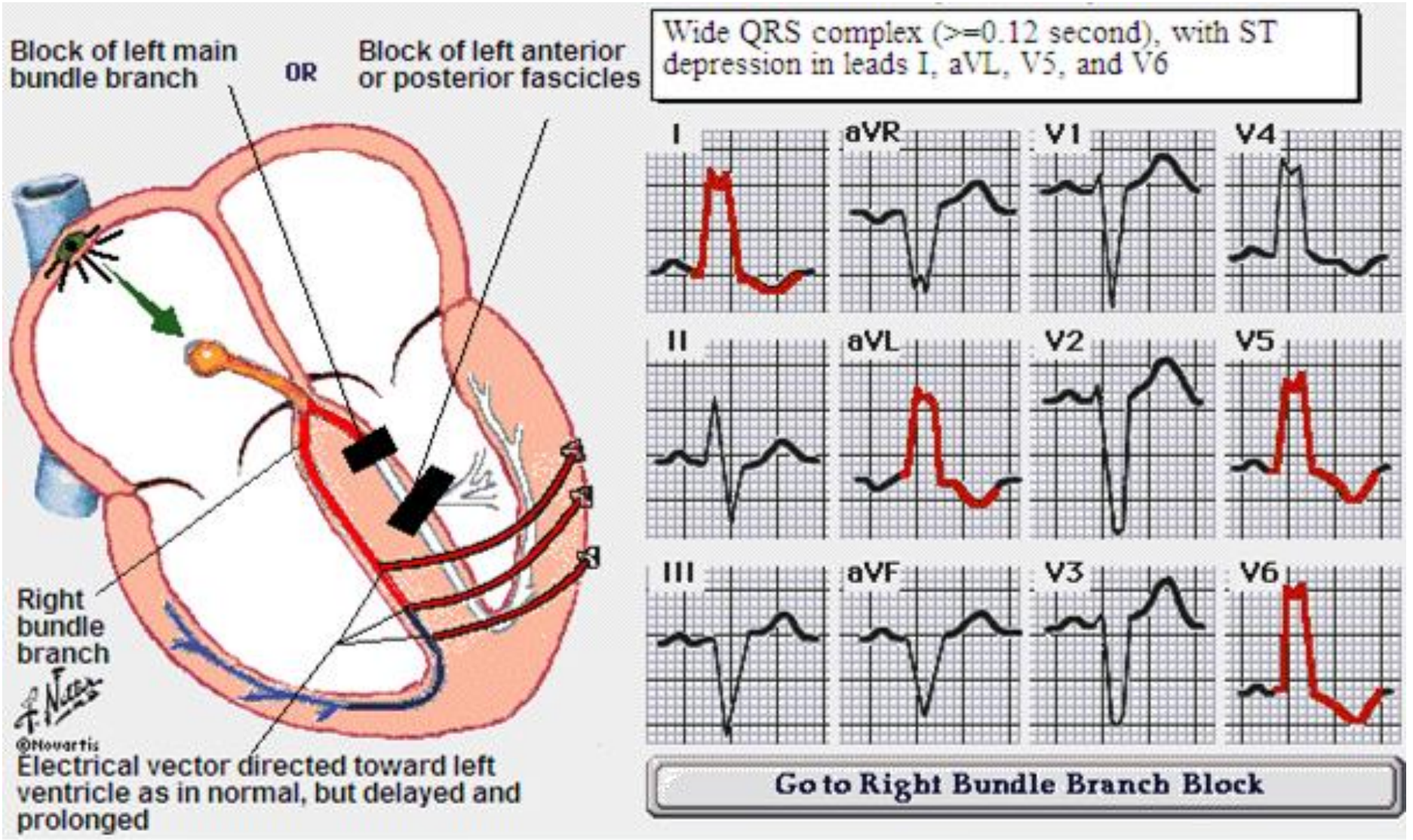
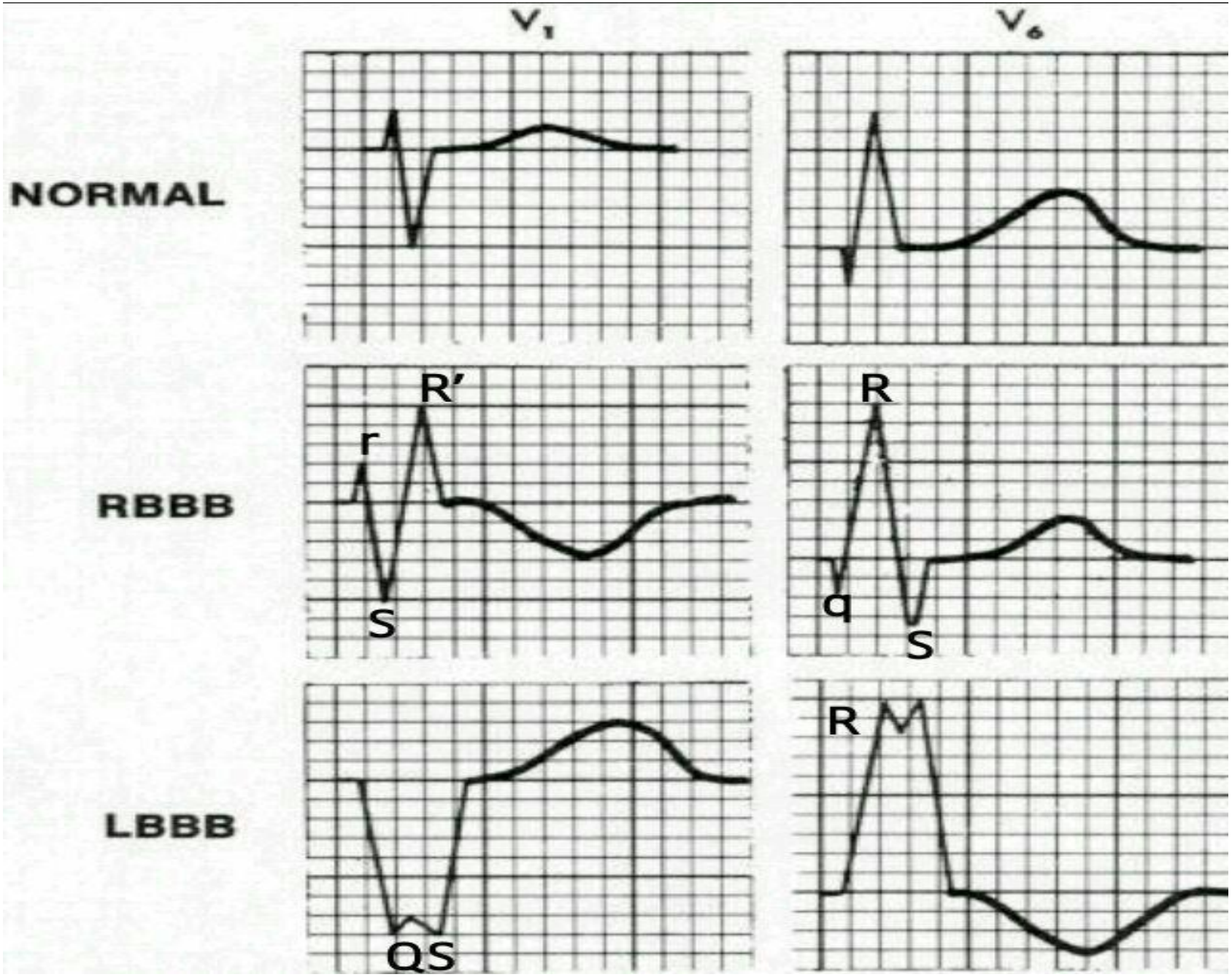
Complete LBBB

- Complete LBBB has a QRS duration > 0.12 sec
- Prominent S waves in lead V1, R in L I, aVL, V6
- Usually broad, Bizarre R waves are seen, M pattern
- Poor R progression from V1 to V3 is common.
- The "normal" ST-T waves in LBBB should be oriented opposite to the direction of the QRS
- Incomplete LBBB looks like LBBB but QRS duration is 0.10 to 0.12 sec, with less ST-T change.
- This is often a progression of LVH changes.

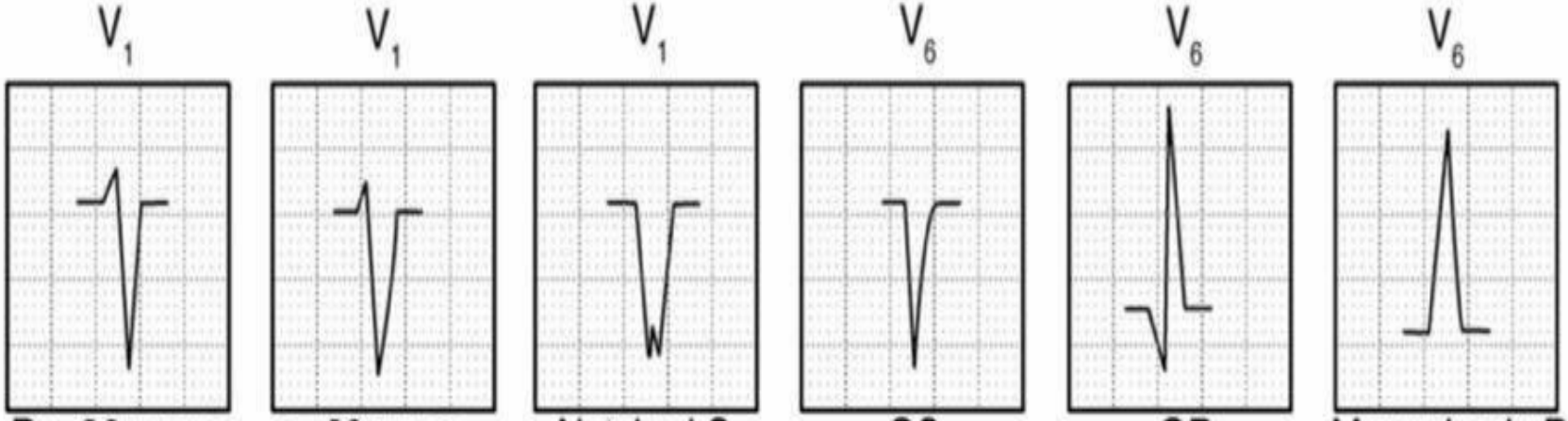
Complete LBBB



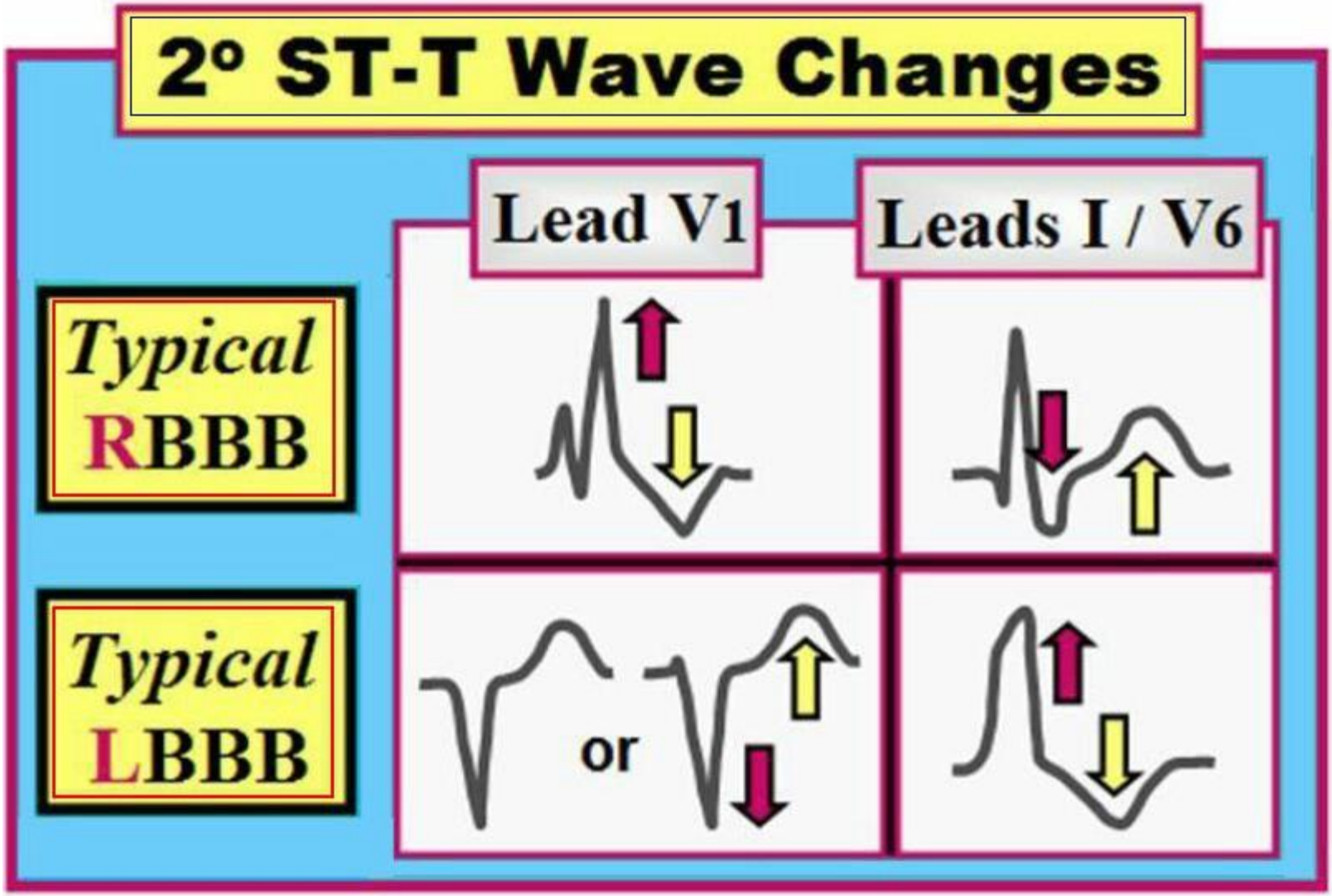
Causes of RBBB	Causes of LAFB
•Normal variant. •Cor pulmunale. •Pulmonary embolism. •MI, CMP`S, HHD,CHD •Mechanical damage. •Lev`s disease.	•Chronic hypertension •Aortic stenosis •Aortic root dilation •Dilated cardiomyopathy •Impairment of the cardiac electrical conduction system •Acute myocardial infarction •Lung diseases •Aging •Degenerative fibrotic disease

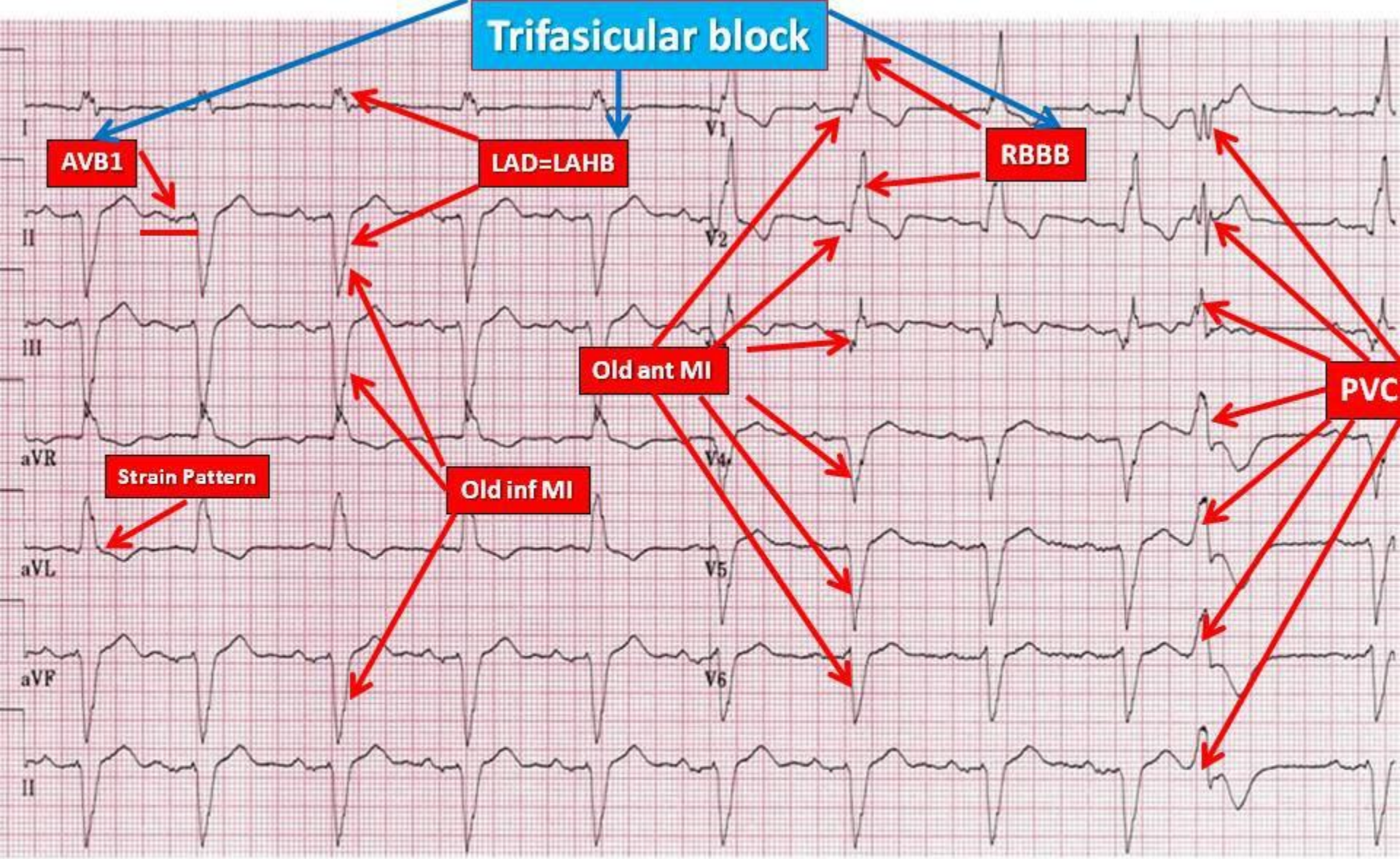

V1
V6
NORMAL
RBBB
R'
r
S
R
q
S
LBBB
QS
R

Examples of Morphology Criteria with LBBB-like QRS complexes



2º ST-T Wave Changes





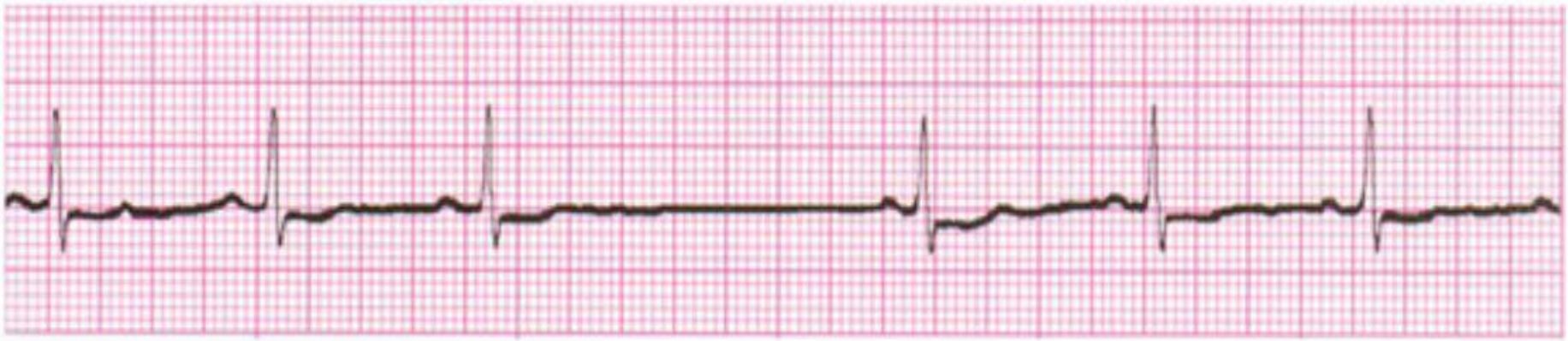
rtesy of B. Topfer, RN, Hilversum, The Netherlands

ECG PEDIA.ORG

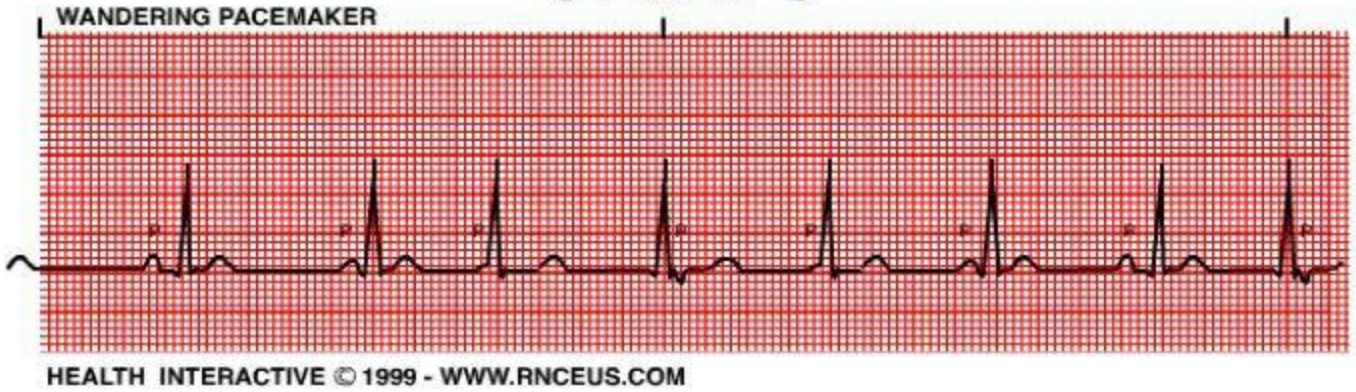
علائم الکتروکاردیوگرافیک

- یک یا چند کمپلکس **P-QRS-T** حذف و به جای آن خط **ایزوالکتریک** دیده می شود .
- فاصله بین **R** قبل و بعد از این خط **ایزوالکتریک** ، مضرب کاملا **صحیحی** از فاصله **R-R** دو کمپلکس متوالی است .

Sinus Block



Wandering Atrial Pacemaker (WAP)



Rhythm: *Slightly Irregular*

Rate: *Usually 60 – 100 bpm; sometimes slower*

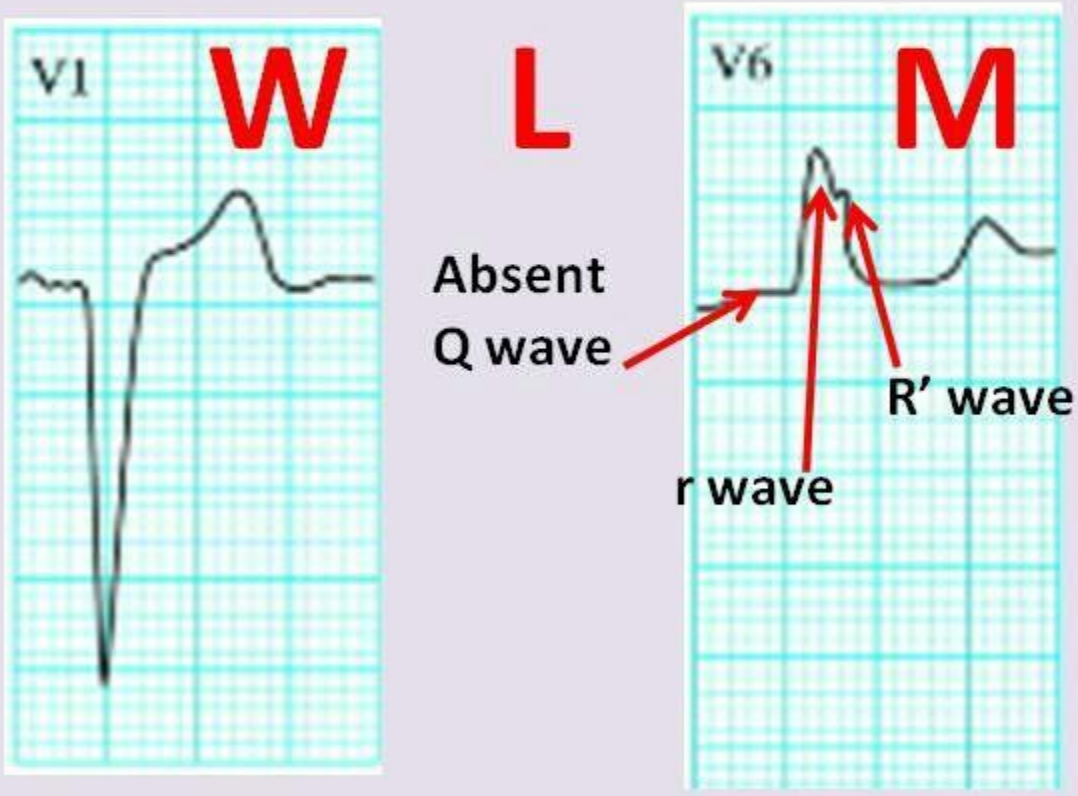
P waves: *Morphology of each P wave differs*

PRI: *0.12 – 0.20 sec; inconsistent*

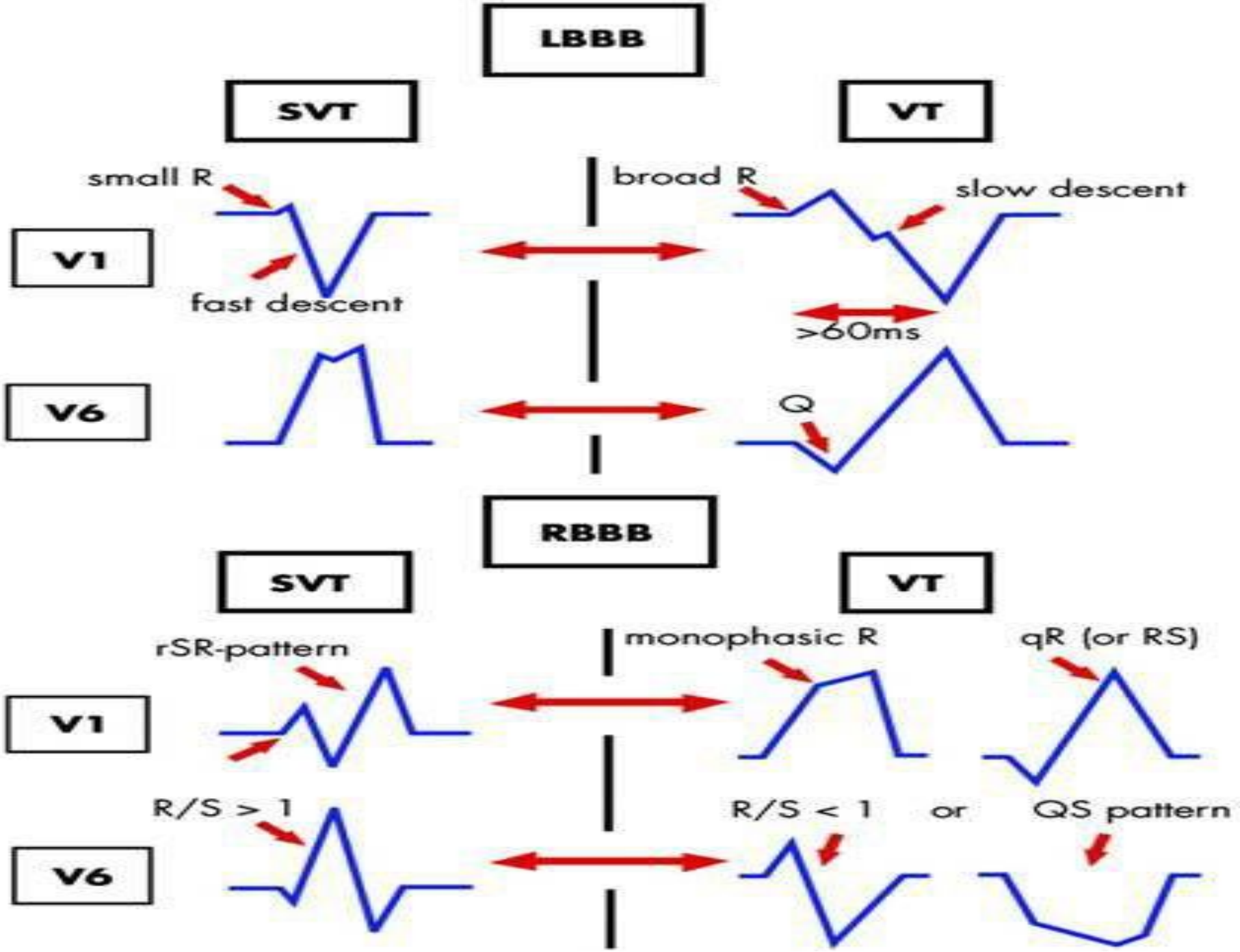
QRS: *Narrow (< 0.12 sec); sometimes wide*

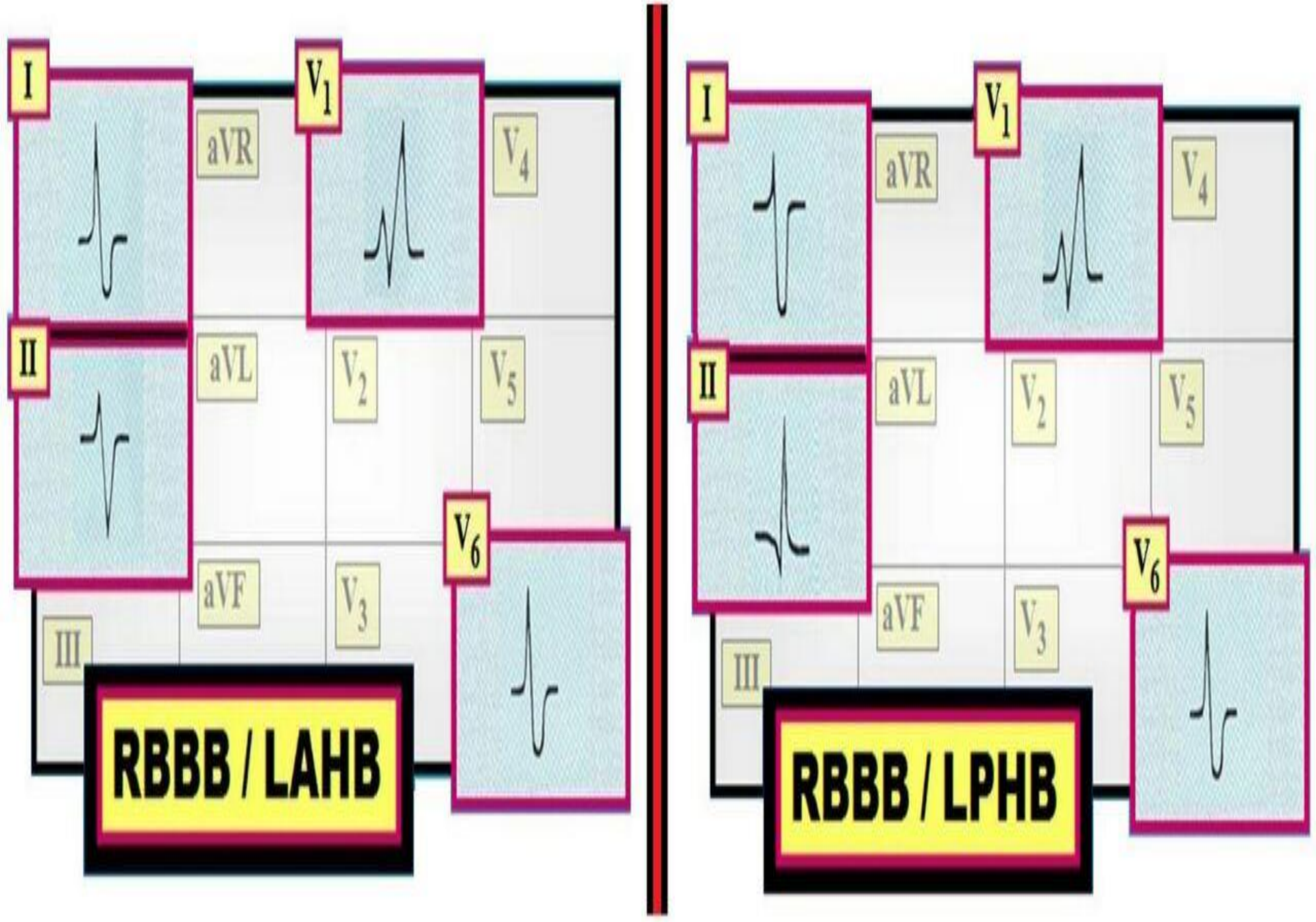
Left Bundle Branch Block (WILLIAM)

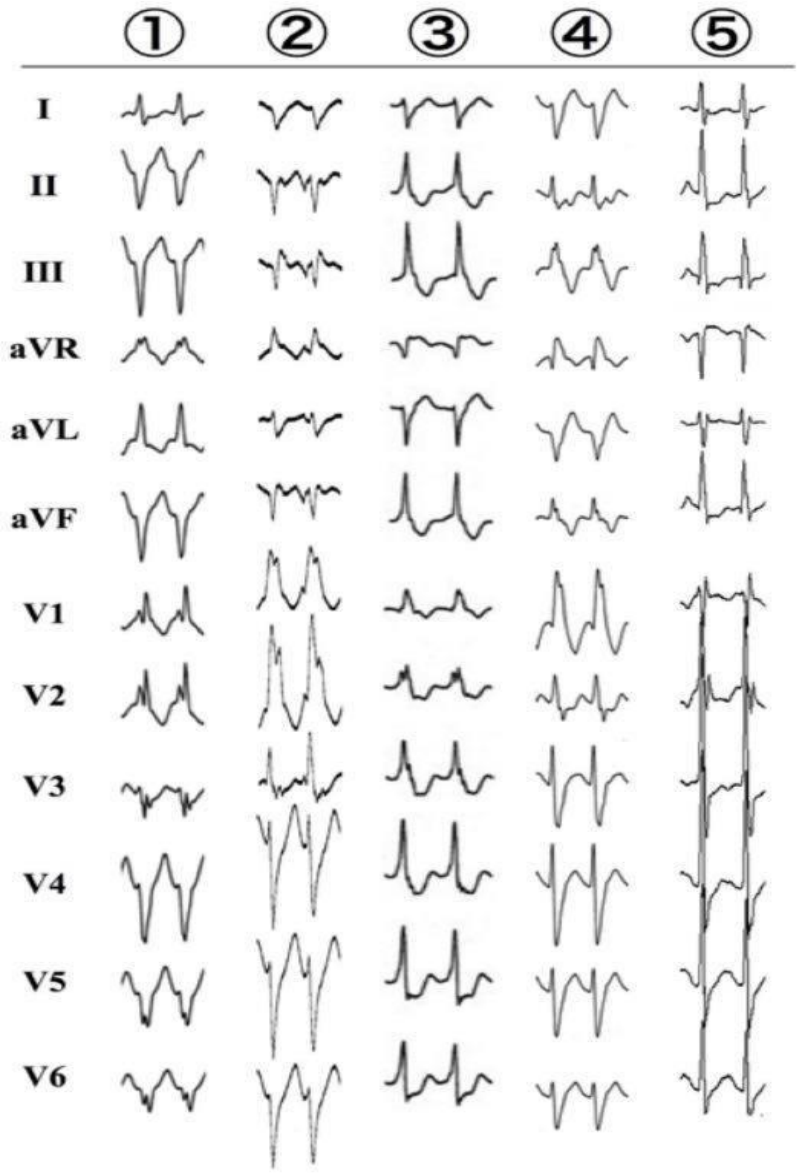
Lead V1: A widened abnormal QRS complex . Note the W shape of William

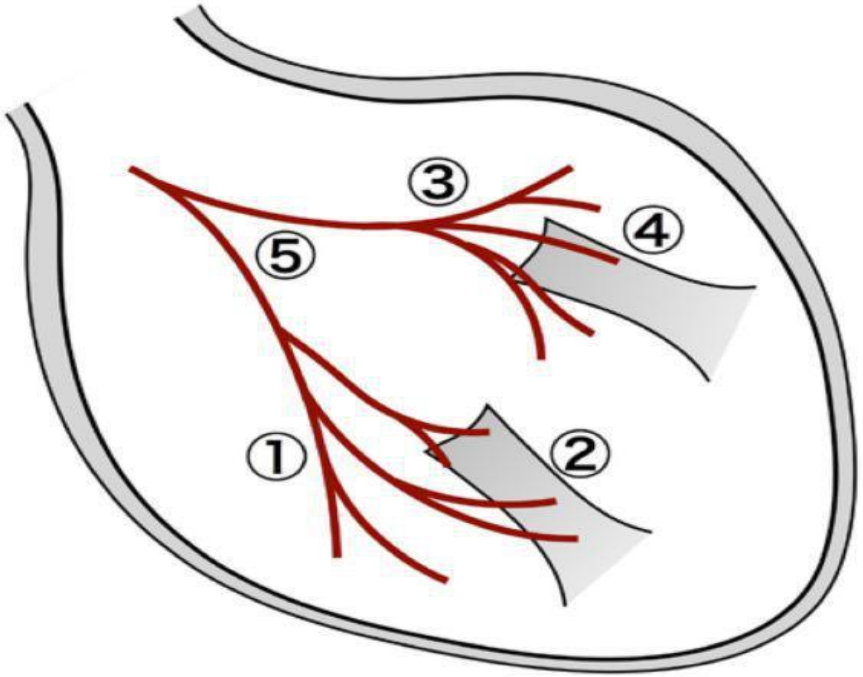


Lead V6: Shows the characteristic r S R' complex . Absent Q waves in lateral leads is singular to LBBB. Note the M shape of williaM.

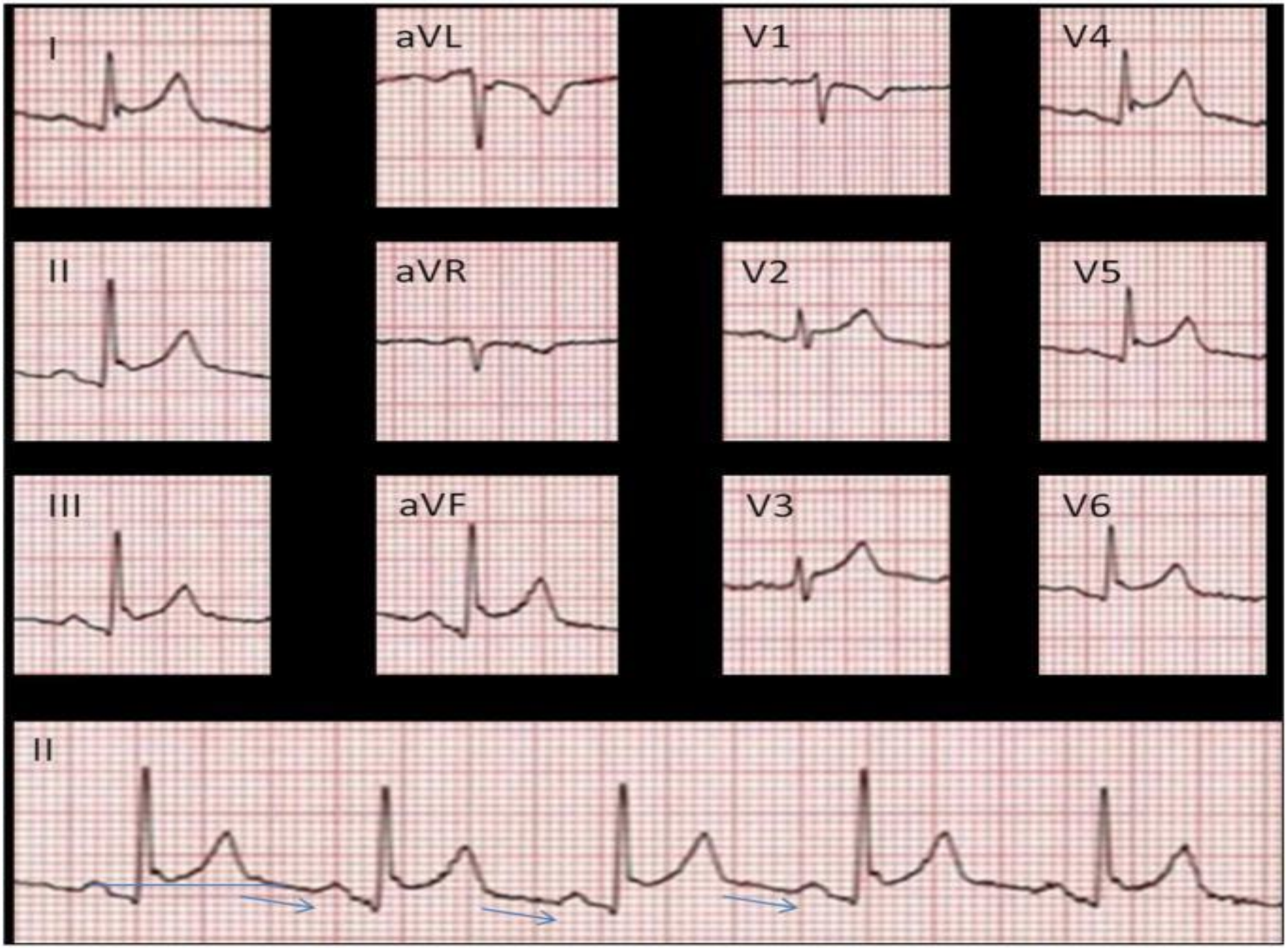

LBBB
SVT
VT
small R
broad R
slow descent
V1
fast descent
>60ms
V6
Q
RBBB
SVT
VT
rSR-pattern
monophasic R
qR (or RS)
V1
R/S > 1
R/S < 1
or
QS pattern
V6


I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
RBBB / LAHB
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
RBBB / LPHB

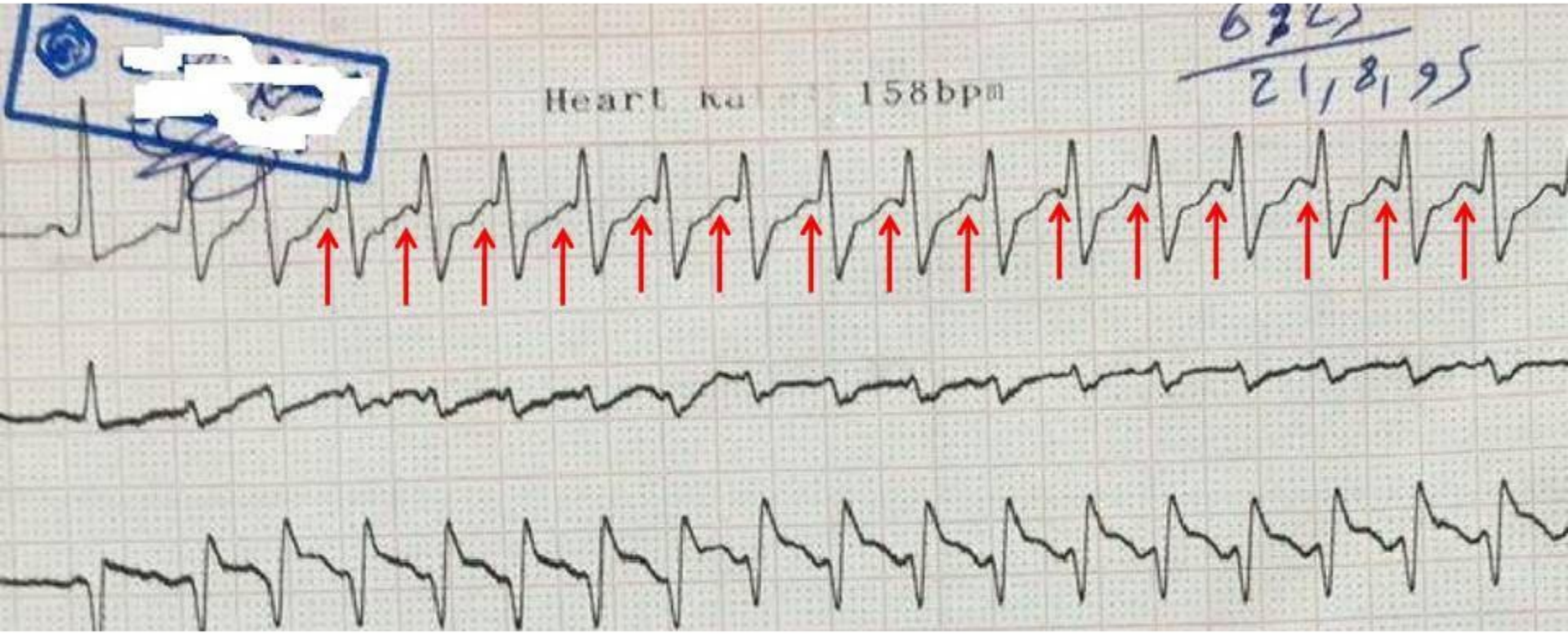


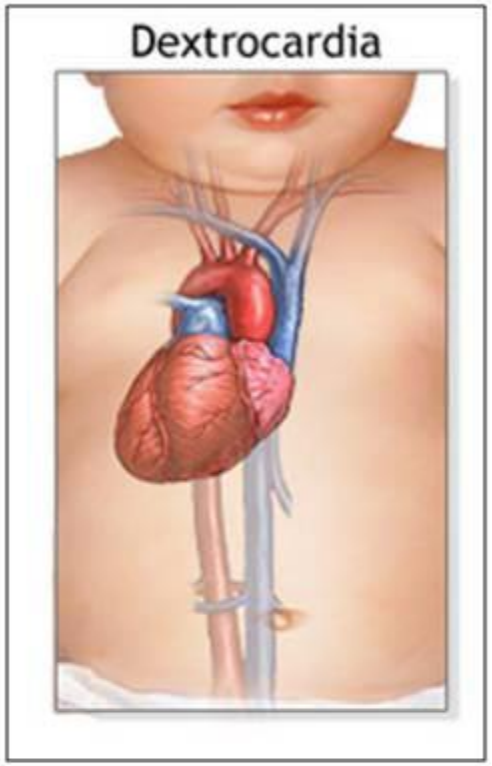


① Left posterior FVT
② PPM-FVT
③ Left anterior FVT
④ APM-FVT
⑤ Upper Septal FVT

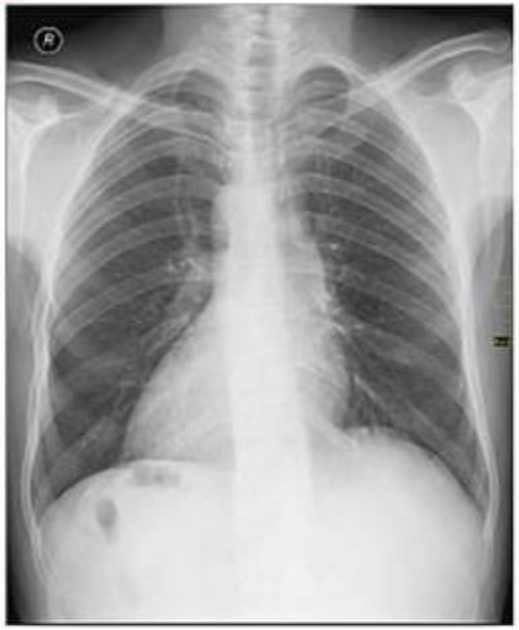

I
aVL
V1
V4
II
aVR
V2
V5
III
aVF
V3
V6
II

AV dissociation



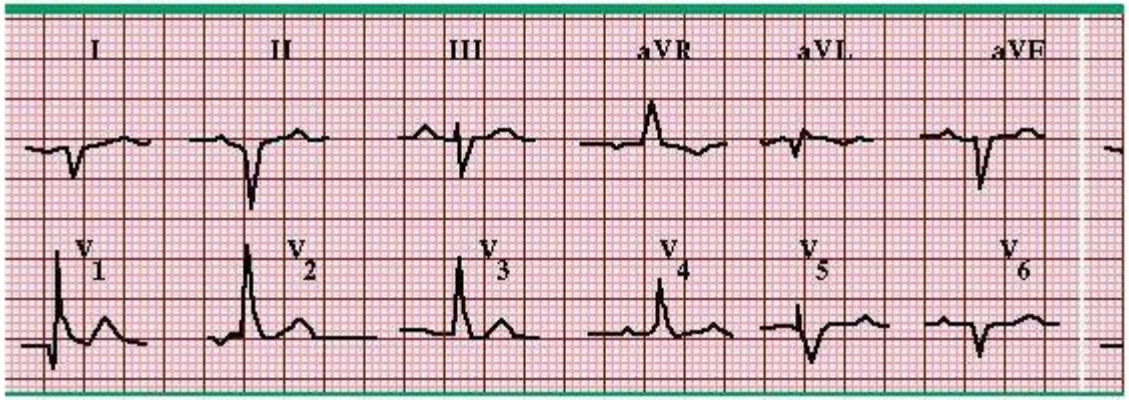


Dextrocardia



- Right axis deviation
- AVR : Positive QRS complexes with upright P and T waves
- Lead I: (inverted P wave, negative QRS, inverted T wave)
- Absent R-wave in the chest leads (dominant S waves)

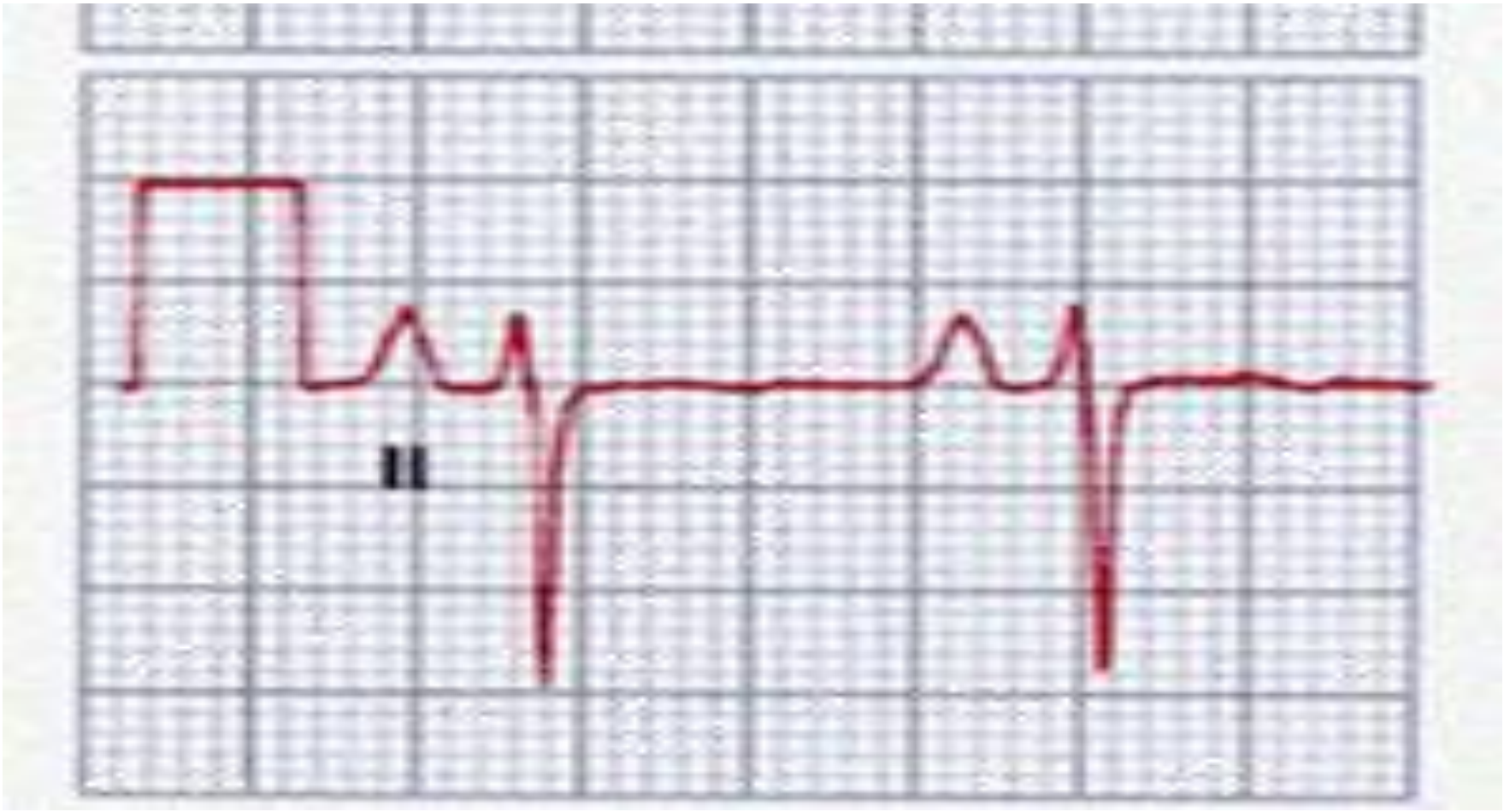
Dextrocardia



Characteristic changes of dextrocardia include a negative P wave and QRS complex in lead 1, since atrial and ventricular depolarization begin on the left and spread to the right. There is also reverse R wave progression across the precordium; the R wave is tallest in V1, and progressively decreases in amplitude in leads V2 to V6.

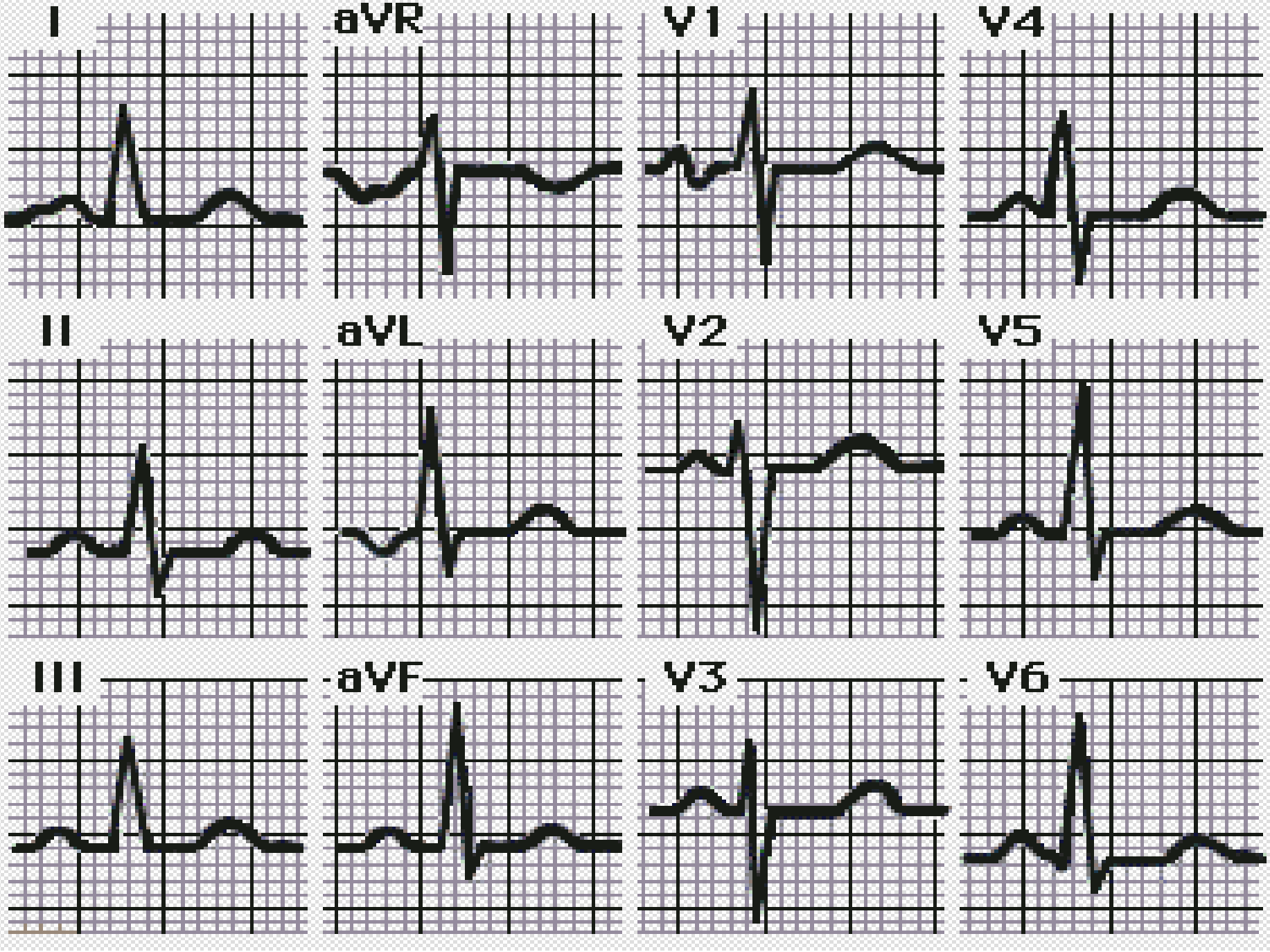
Right Atrial Enlargement (dilatation & hypertrophy)

- Increase pressure/volume results in overload in the RA.
- ECG Characteristics
  - Duration: P wave .10 or <
  - Shape: Tall/peaked in Leads II,III & aVF, biphasic in V1 & V2
  - Direction: Positive
  - Amplitude: 2.5mm or >
  - Causes PS,TS, TI, Pulmonary Hypertension


II

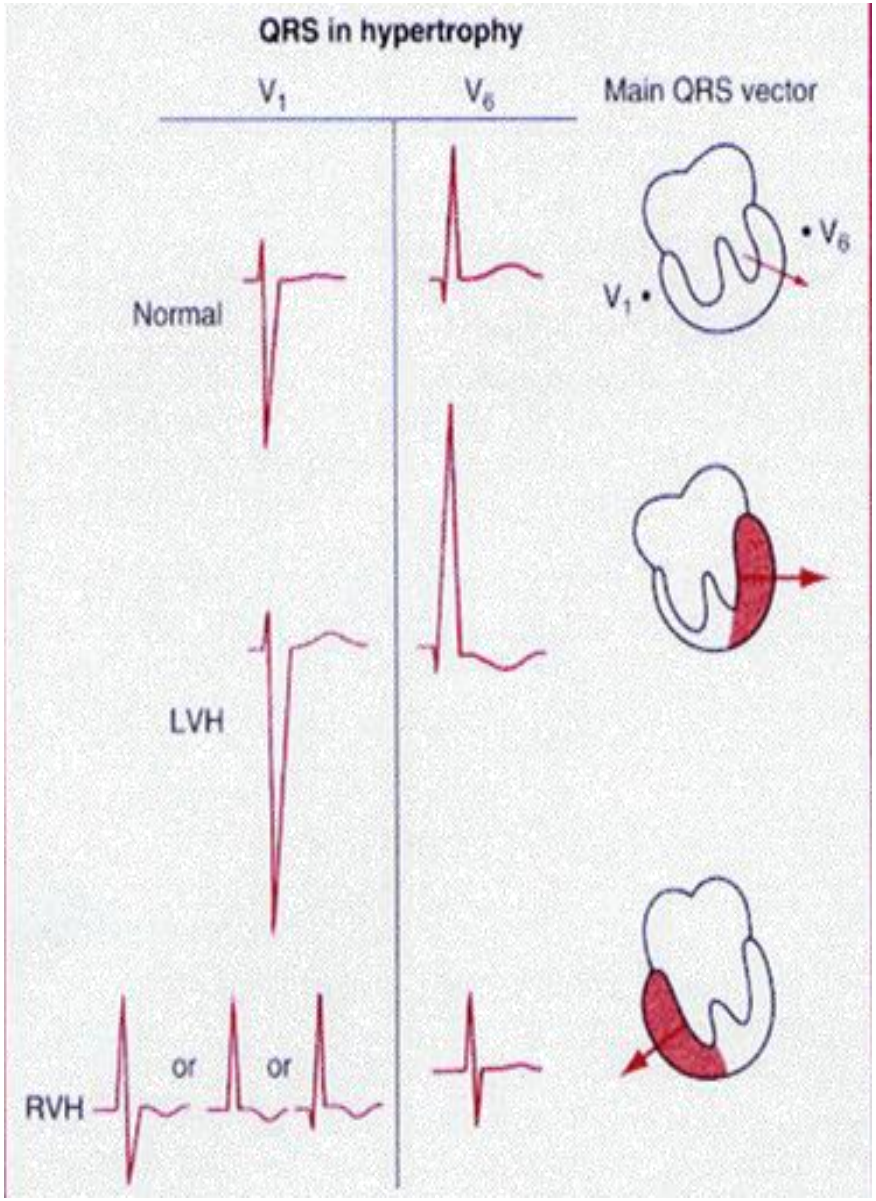
Left Atrial Enlargement (dilatation/hypertrophy)

- Increase pressure/volume results in overload in the LA.
- ECG Characteristics
  - Duration: P wave >.12
  - Shape: Broad positive upright, wide notched (two humps), biphasic
  - Direction: Positive Leads I,II & V4-V6
  - Amplitude: normal .5-2.5mm
  - Causes: MS, acute MI, LHF, AS or AI, HCOP


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

Ventricular Hypertrophy

- Ventricular Muscle Hypertrophy
- QRS voltages in V1 and V6, L 1 and aVL
- We may have to record to ½ standardization
- T wave changes opposite to QRS direction
- Associated Axis shifts
- Associated Atrial hypertrophy



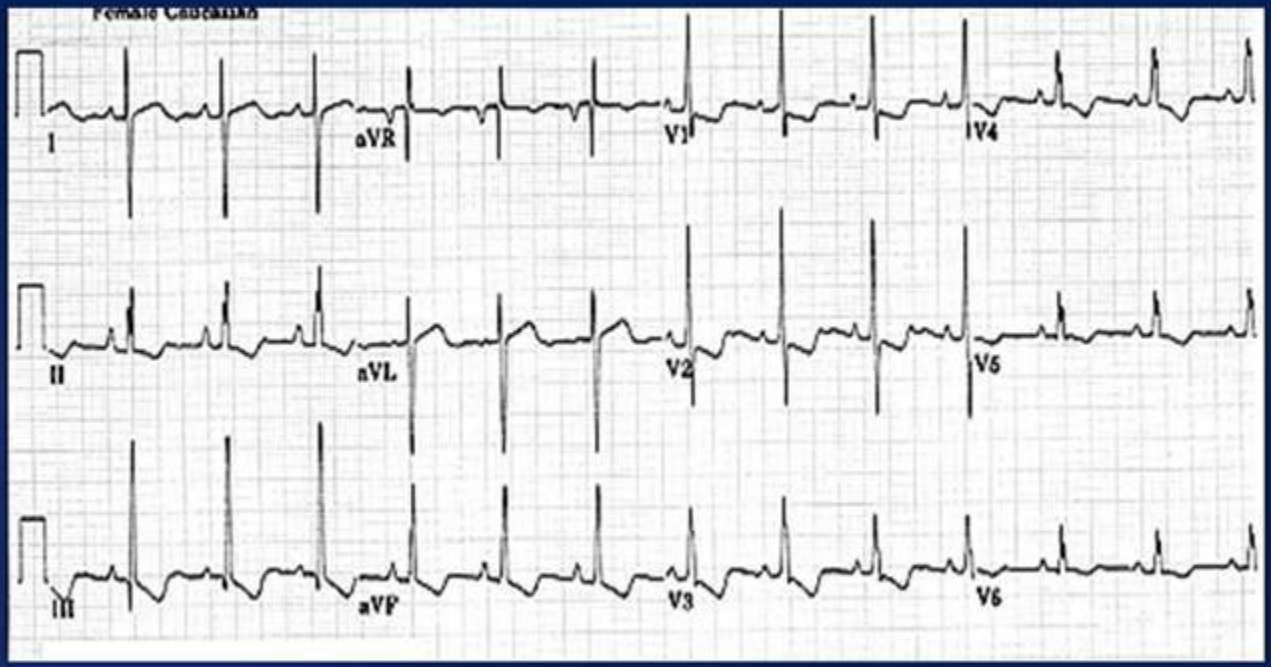
Right Ventricular Hypertrophy

- Increase pressure/volume results in overload in the RV.
- ECG Characteristics
  - Duration: QRS .10 or <
  - QRS Axis: RAD +90 ^{0} or <
  - VAT: prolonged = .035 sec in V1 & V2
  - QRS Pattern: q waves leads II,III & aVF
  - R waves: = or > S waves in Lead V1
  - ST segment: Downsloping of 1mm or >
  - Hockey stick: ST depression & T-wave inversion
  - T wave: inversion in II, III, aVF & V1,V2 & V3
  - Causes: MS, acute MI, COPD

Right ventricular hypertrophy

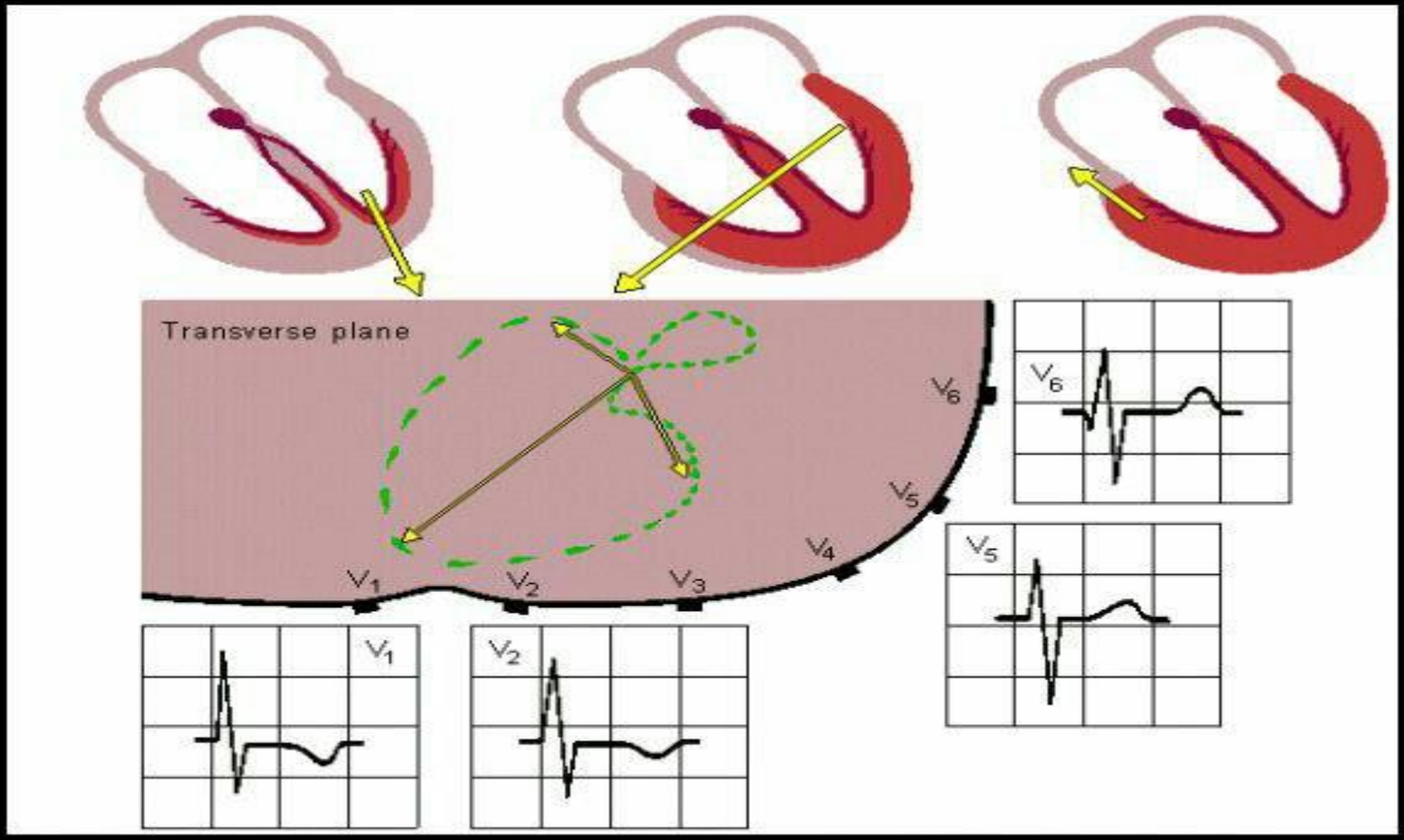
RVH

- Take a look at this ECG. What do you notice about the axis and QRS complexes over the right ventricle (V1, V2)?



There is right axis deviation (negative in I, positive in II) and there are tall R waves in V1, V2.

Right Ventricular Hypertrophy



Right Ventricular Hypertrophy Criteria

1. In V_1, R wave is greater than the S wave - or - R in V1 greater than 7 mm
    1. Right axis deviation
    2. In V_1, T wave inversion (reason unknown)
    3. S waves in V5 and V6

Left Ventricular Hypertrophy cont...

- ECG Characteristics cont..
  - Sum of R/S: Any R wave and S wave > 20 mm & Sum of S wave in V1 or V2 and R wave in Lead V5 or V6 is 35 mm or> .
  - ST segment: Downsloping ST depression of 1m or > in Leads I, aVL & V5, V6
  - T wave: Inversion in I, aVL & V5, V6

TABLE 12-4 Common Diagnostic Criteria for Left Ventricular Hypertrophy

MEASUREMENT	CRITERIA
Sokolow-Lyon voltages	$SV_1 + RV_5 > 3.5$ mV RaVL >1.1 mV
Romhilt-Estes point score system*	Any limb lead R wave or S wave > 2.0 mV (3 points) or SV_1 or $SV_2 \geq 3.0$ mV (3 points) or RV_5 to $RV_6 \geq 3.0$ mV (3 points) ST-T wave abnormality, no digitalis therapy (3 points) ST-T wave abnormality, digitalis therapy (1 point) Left atrial abnormality (3 points) Left axis deviation ≥–30 degrees (2 points) QRS duration ≥90 msec (1 point) Intrinsicoid deflection in V_5 or V_6 ≥50 msec (1 point)
Cornell voltage criteria	SV_3 + RaVL ≥2.8 mV (for men) SV_3 + RaVL >2.0 mV (for women)
Cornell regression equation	Risk of LVH = $1/(1+ e^{-exp})^{\dagger}$
Cornell voltage duration measurement	QRS duration × Cornell voltage >2436 mm-sec‡ QRS duration × sum of voltages in all leads >1742 mm-sec

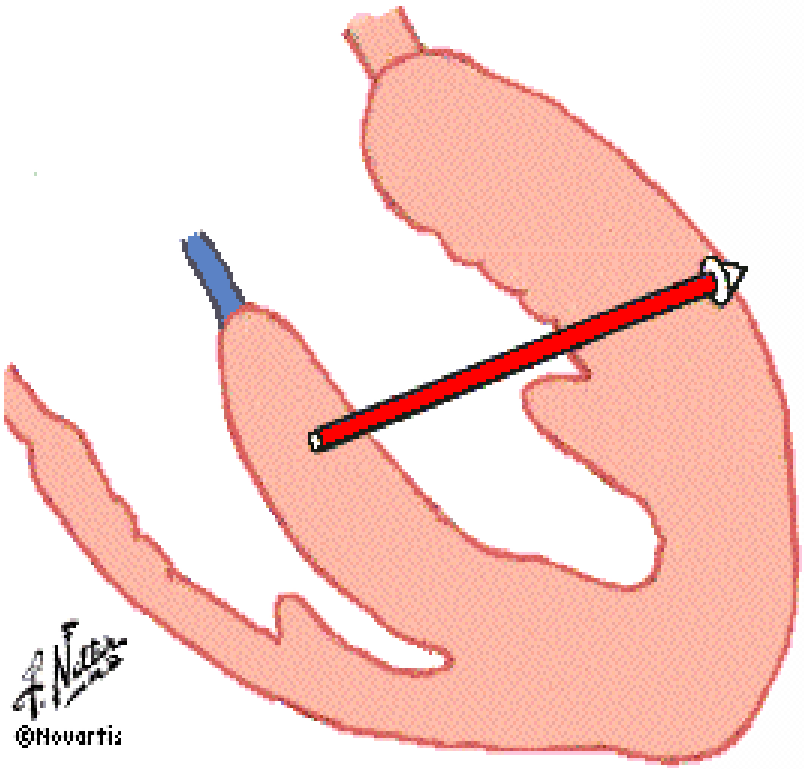
PTF = P terminal force; $PTFV_1$ = P terminal force in lead V_1.
*Probable LVH is diagnosed with totals of 4 points, and definite LVH is diagnosed with totals of 5 or more points.
†For subjects in sinus rhythm, exp = 4.558 − 0.092 (SV_3 + RaVL) − 0.306 TV_1 − 0.212 QRS − 0.278 $PTFV_1$ − 0.559 (sex). Voltages are in mV, QRS is QRS duration in milliseconds, PTF is the area under the P terminal force in lead V_1 (in mm-sec), and sex = 1 for men and 2 for women. LVH is diagnosed as present if exp < −1.55.
‡For women, add 8 mm.

Left Ventricular Hypertrophy

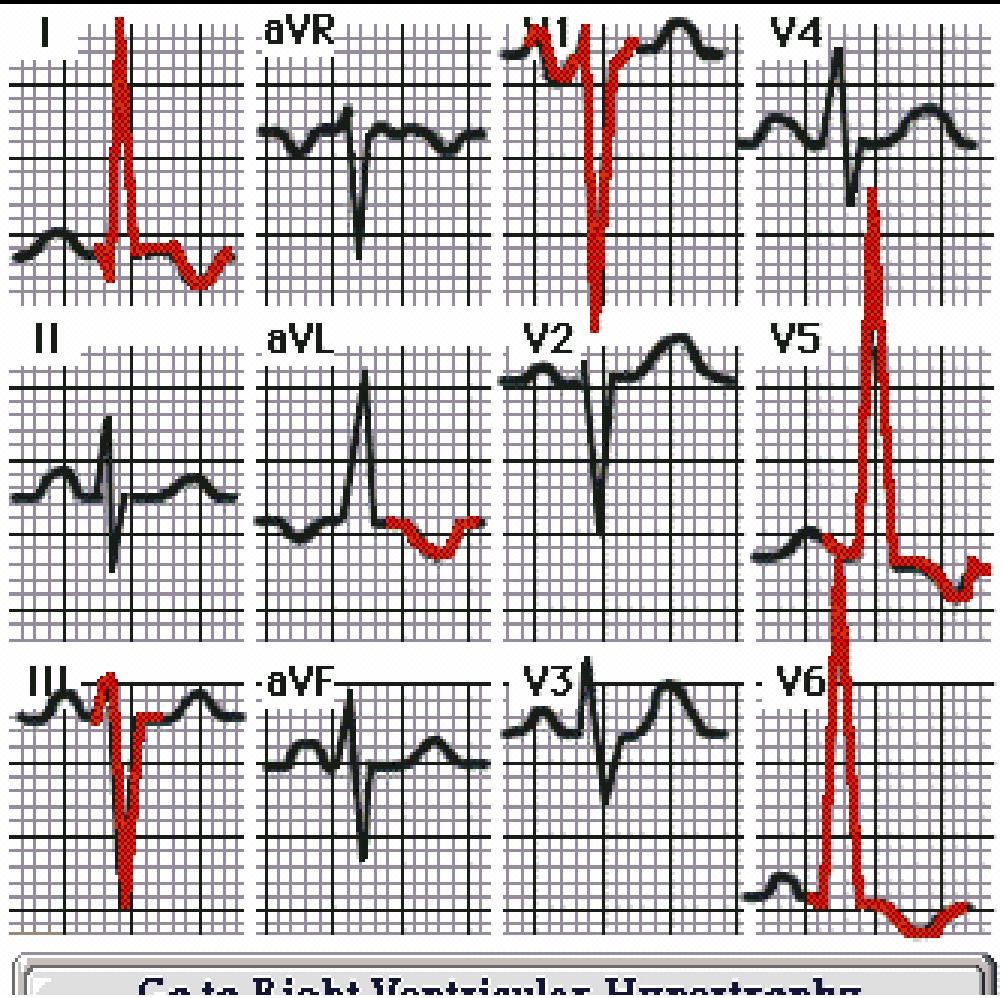
- High QRS voltages in limb leads
- R in Lead I + S in Lead III > 25 mm
- S in V1 + R in V5 > 35 mm
- R in aVL > 11 mm or S V3 + R aVL > 24 ♂, > 20 ♀
- Deep symmetric T inversion in V4, V5 & V6
- QRS duration > 0.09 sec
- Associated Left Axis Deviation, LAE
- Cornell Voltage criteria, Estes point scoring

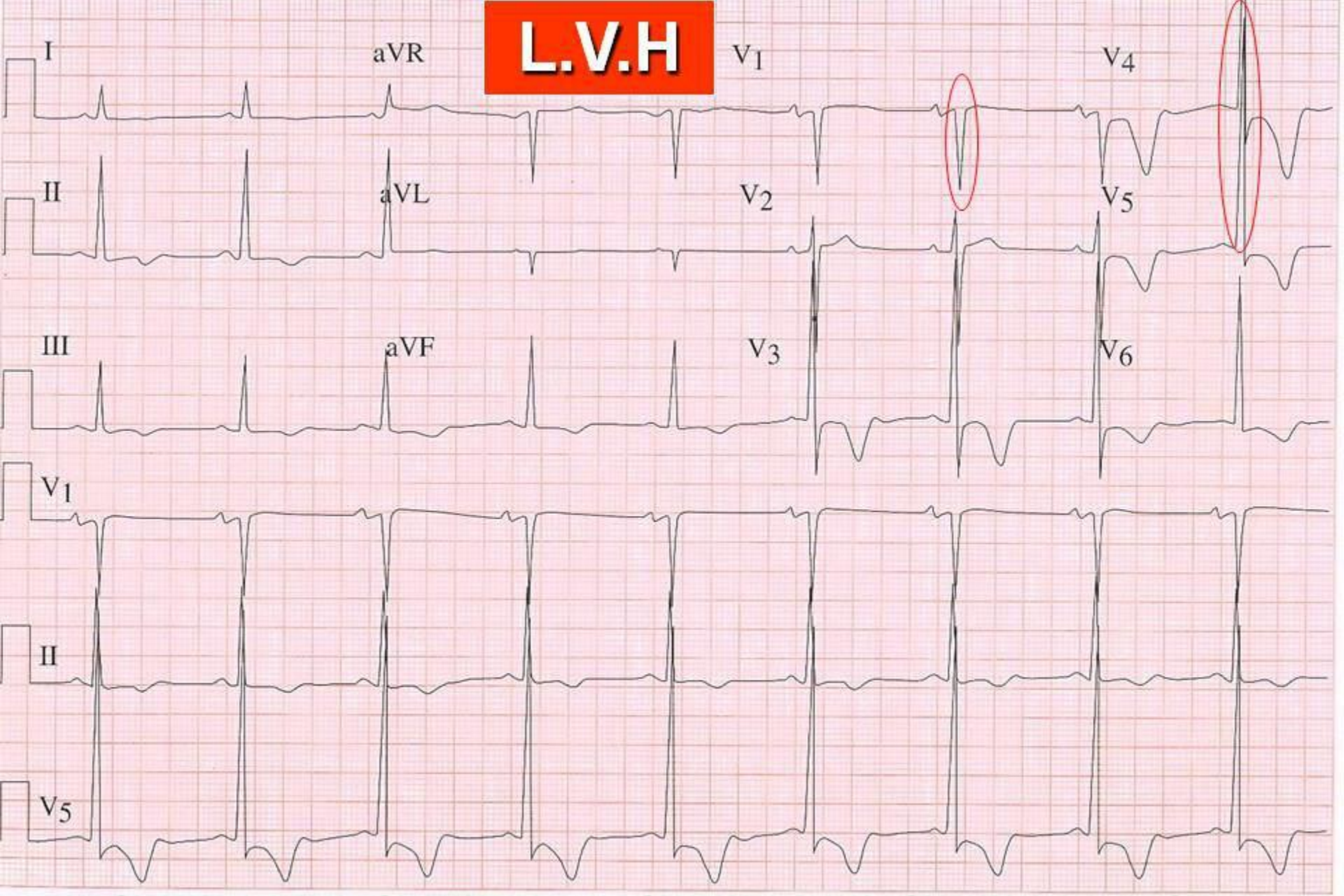
Left Ventricular Hypertrophy

High voltage in limb leads: (R I + S III >25 mm)
Or precordial leads: (S V1 + R V5, or S V1 + R V6, >=35 mm)
Often, left atrial enlargement, ST-T abnormalities



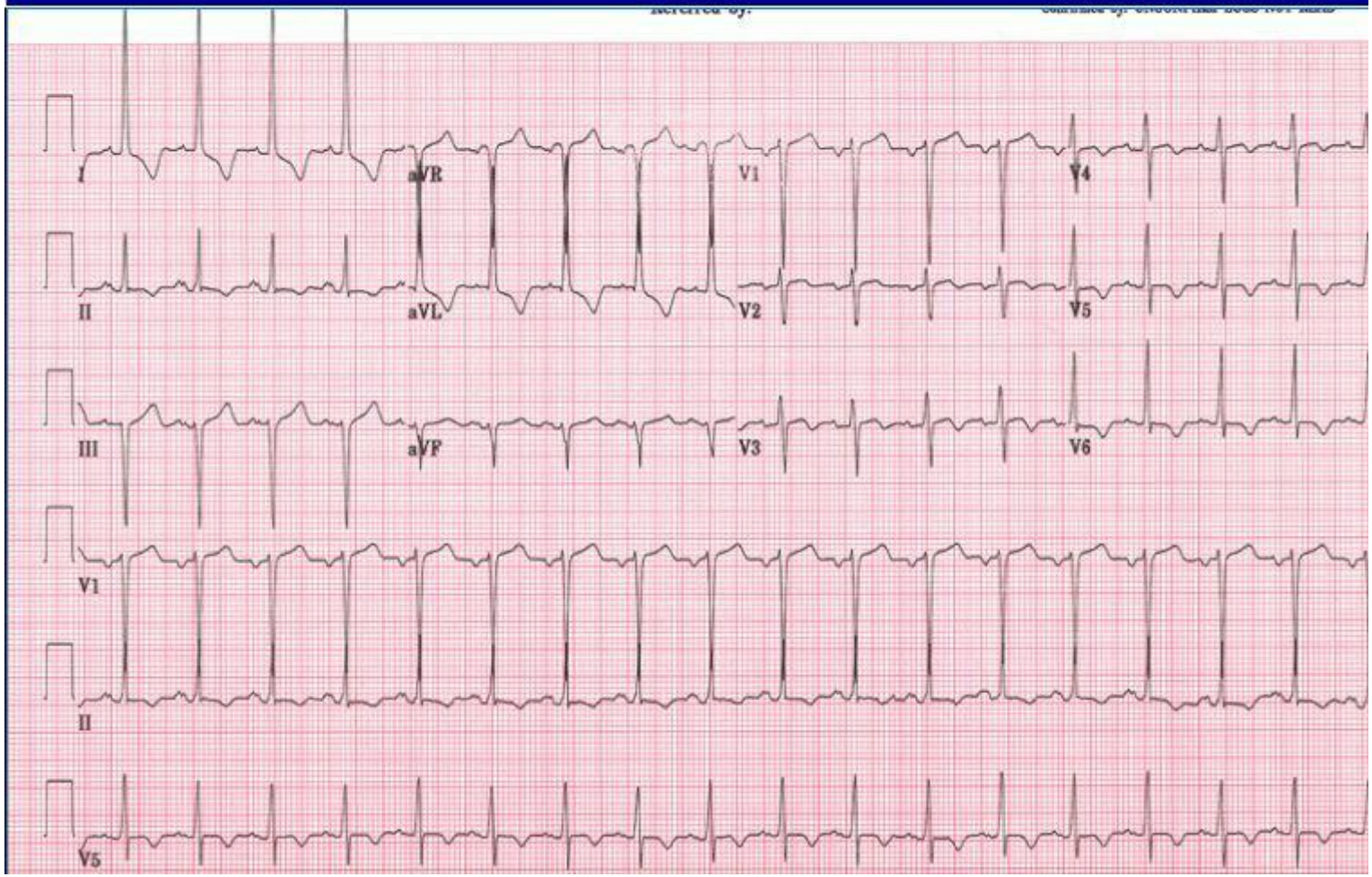
Arrow indicates major electrical vector of ventricular depolarization



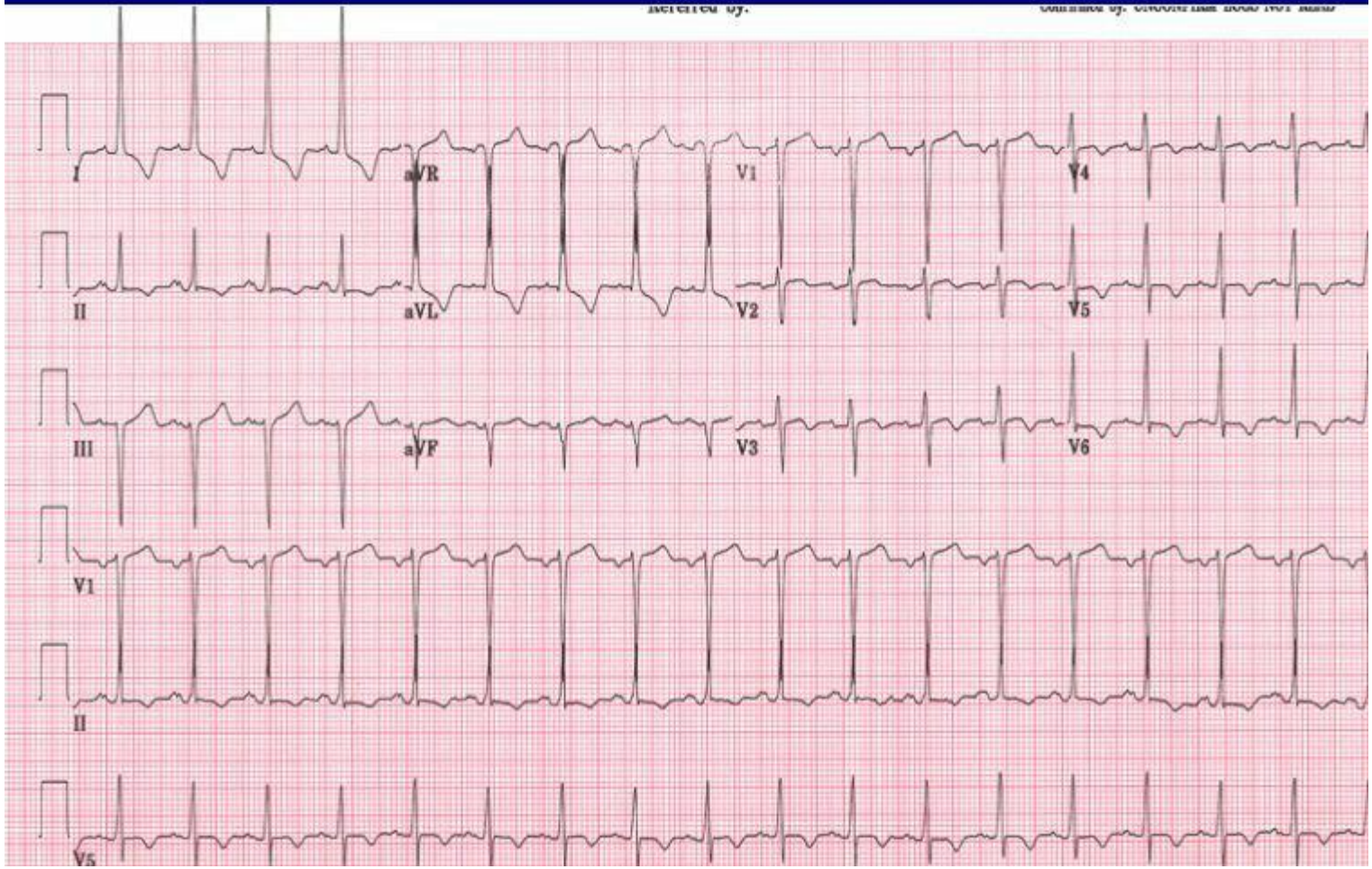


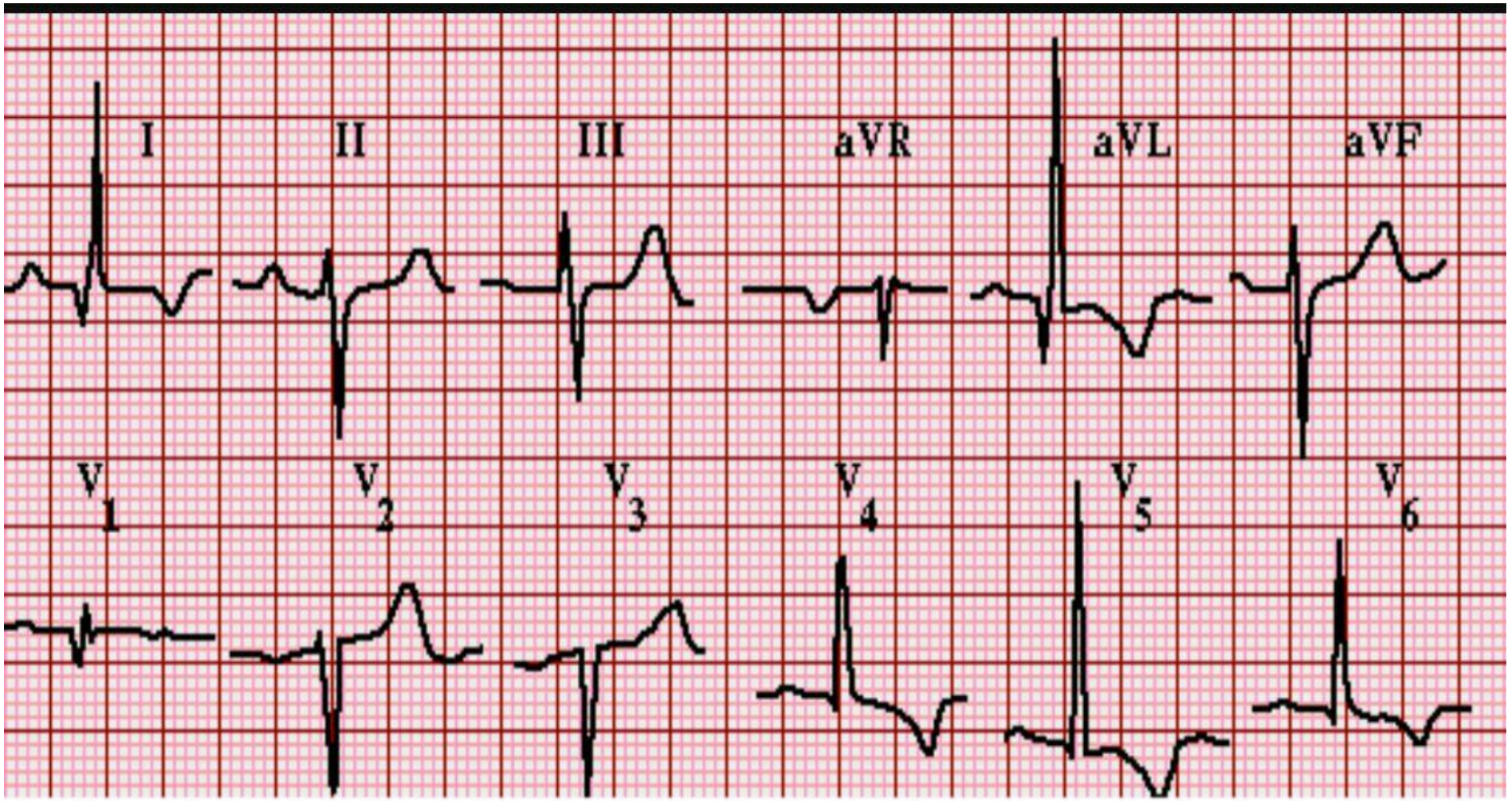
MSSHERMEH@YAHOO.COM

LVH By Cornell Product (QRS=80ms)

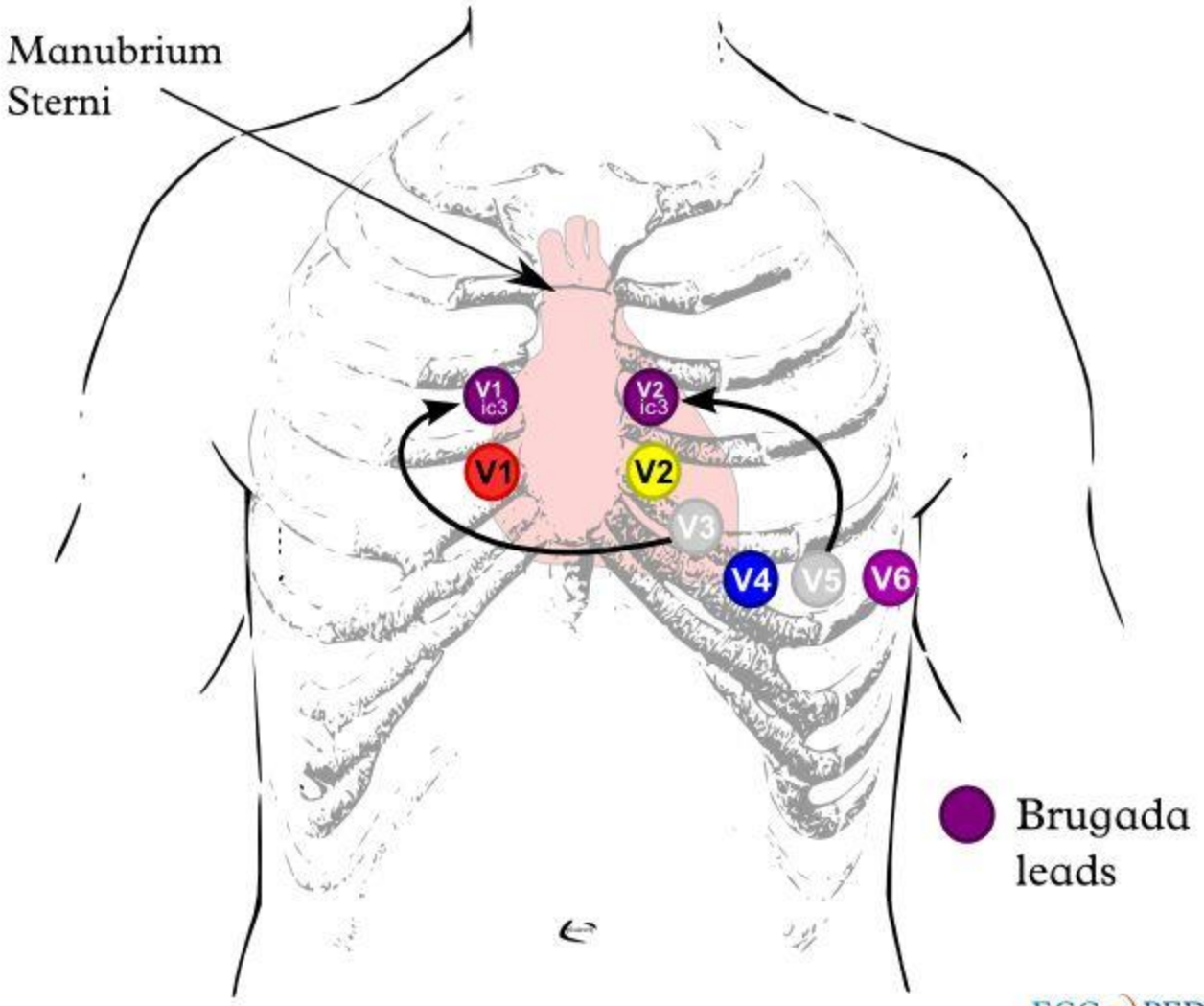


Sokolow-Lyon voltage criteria

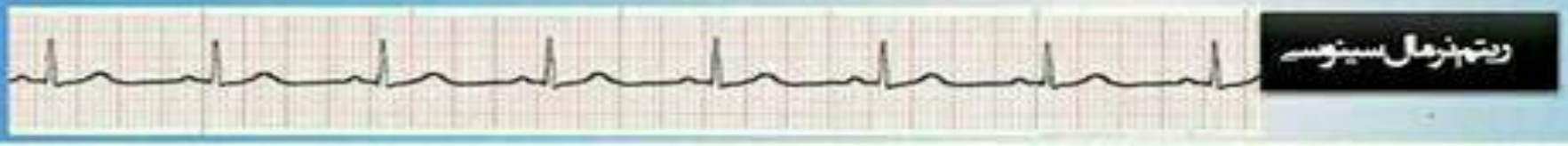




Left ventricular hypertrophy This electrocardiogram demonstrates several features of left ventricular hypertrophy: the QRS complex is slightly widened due to an intraventricular conduction delay; there is left axis deviation; there is ST depression and inverted T waves noted in several leads; several voltage criteria are met, including an R wave in aVL which is greater than 18 mm (in this case 20 mm).


Manubrium
Sterni
V1
ic3
V2
ic3
V1
V2
V3
V4
V5
V6
Brugada
leads
ECGPEDIA.ORG

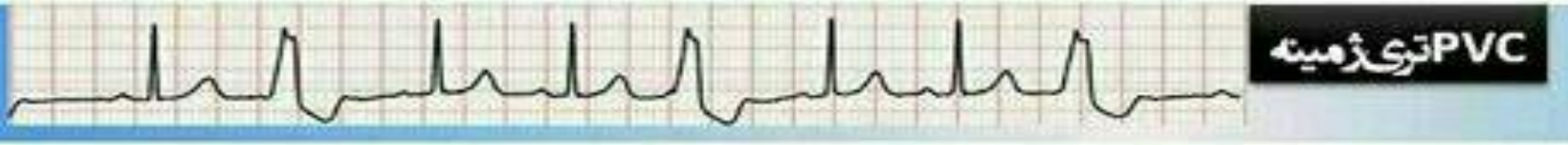
تشخیص خود PVC و انواع مختلف آن:



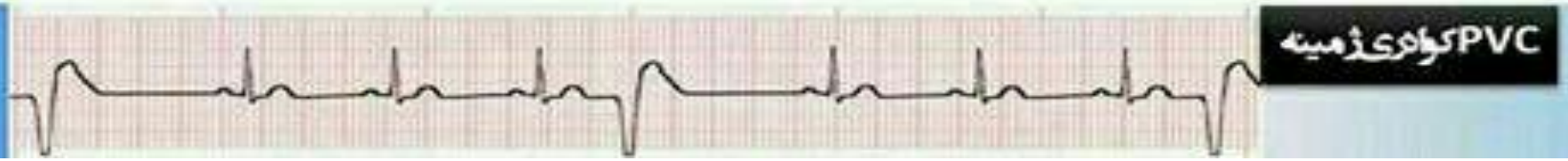
PVC یا ضربان زود رس بطنی، در EKG بصورت عکس شدن کمپلکس QRS و قطعه ST ظاهر می‌شود در واقع موج T و R عکس هم می شوند

PVC بای ژمینه، همان PVC است اما با این تفاوت که یک کمپلکس طبیعی داریم و یک PVCکه مرتب تکرار می شود

PVC تری ژمینه، همان PVC است اما با این تفاوت که دوکمپلکس طبیعی داریم و یک PVCکه مرتب تکرار می شود

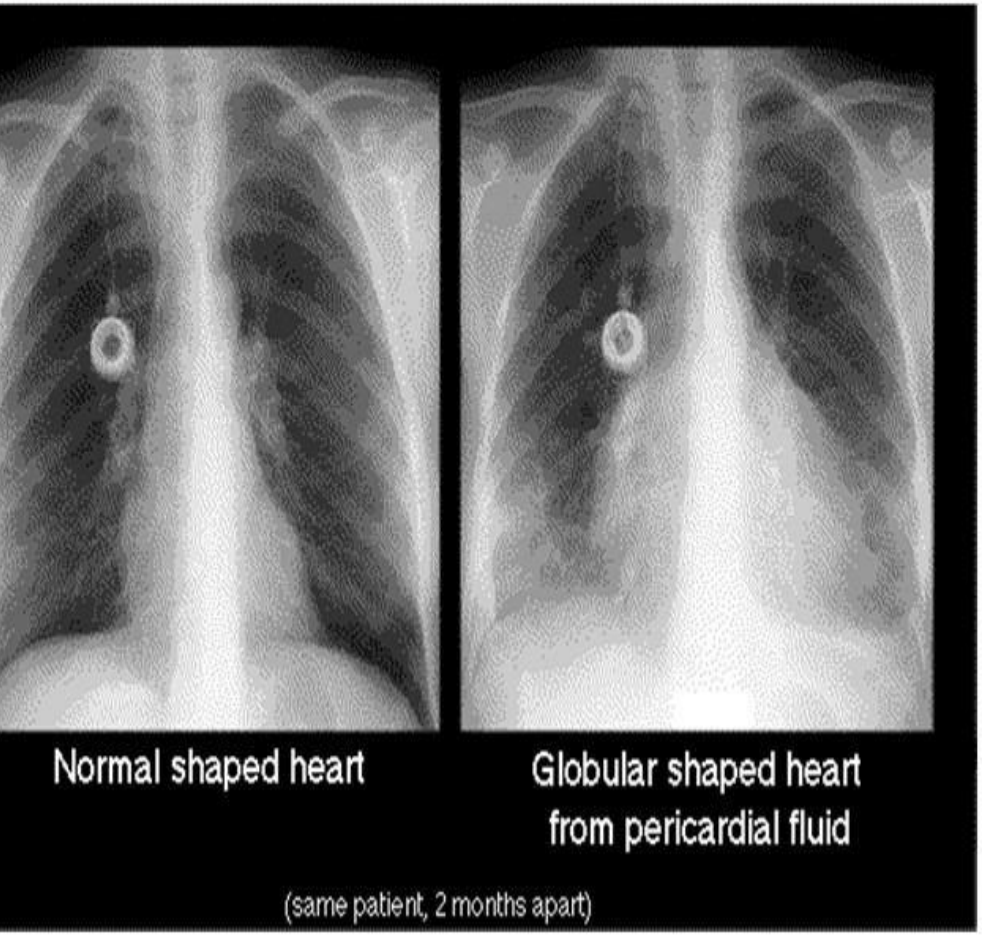


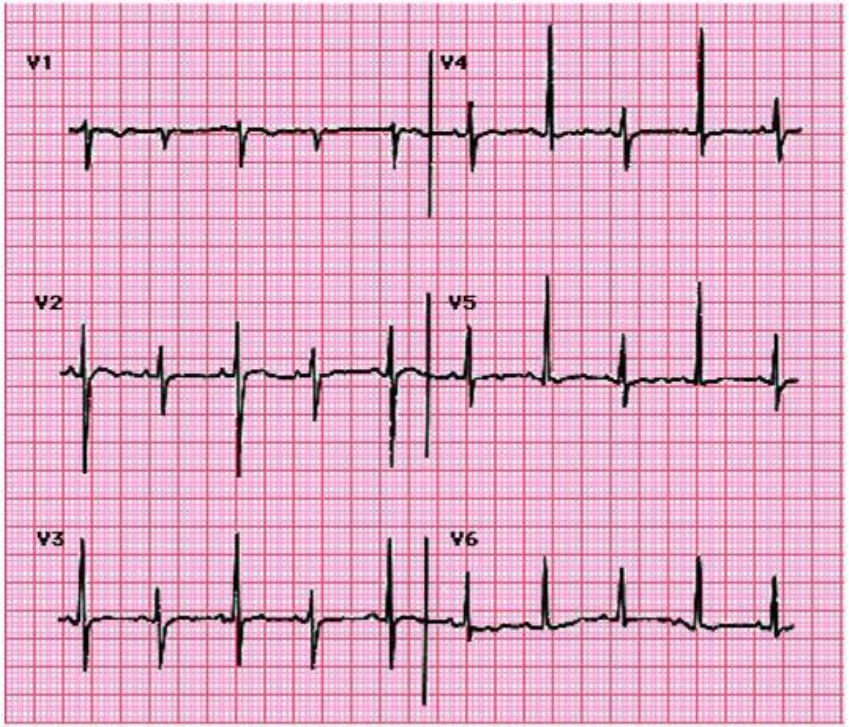
PVC کوادری ژمینه،همان PVC است اما با این تفاوت که سه کمپلکس طبیعی داریم و یک PVCکه مرتب تکرار می شود



تامپوناد قلبی (Cardiac tamponade)

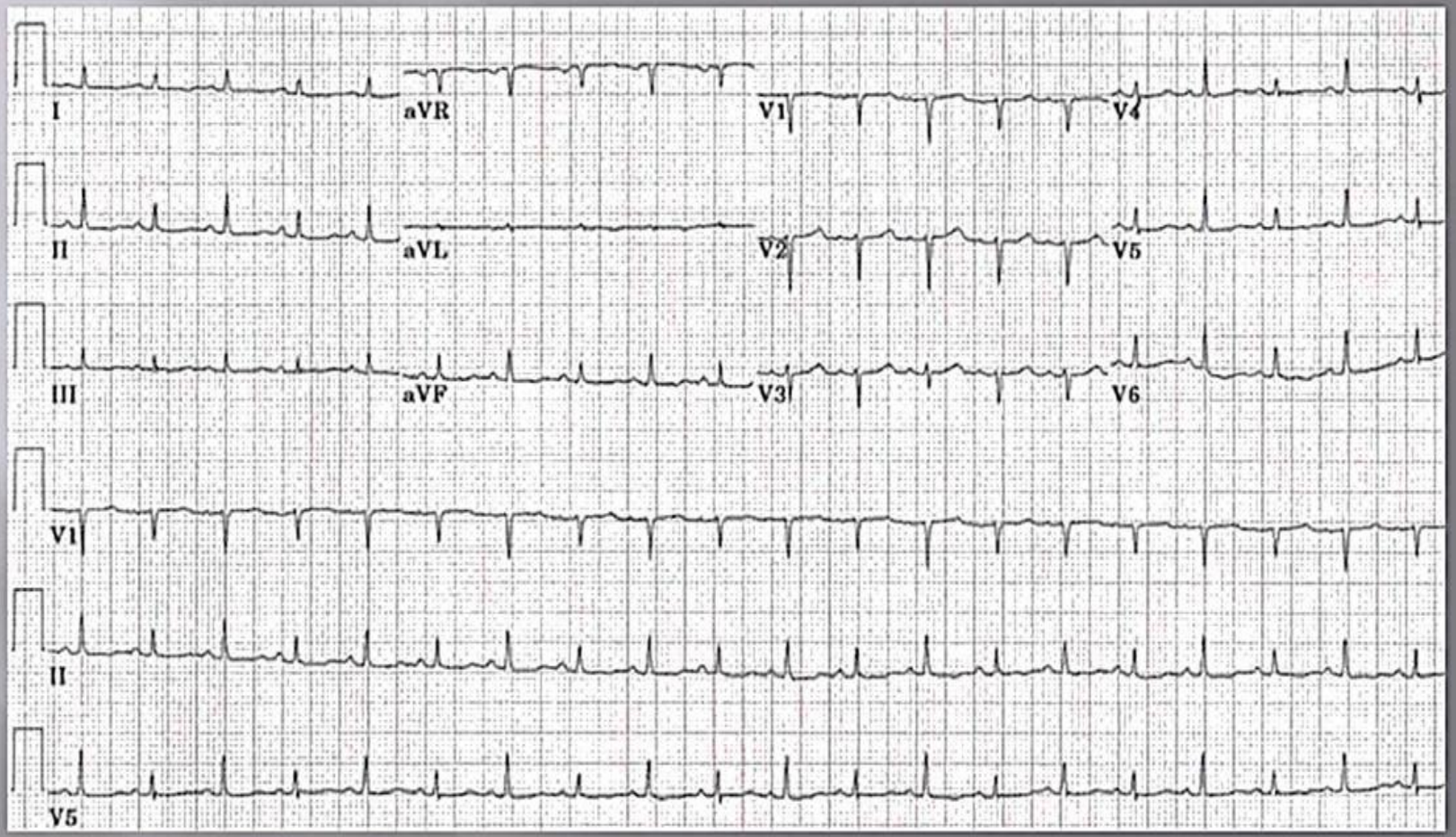
تامپوناد قلبی ناشی از تحت فشار قرار گرفتن قلب بصورت آهسته یا سریع توسط خون، چرک، گاز و مایع تجمع‌یافته در فضای پریکارد ایجاد می‌شود.این حالت باعث کاهش جریان ورودی به قلب، کاهش حجم ضربه‌ای و در کل باعث اختلال در پویایی خون (اختلال همودینامیک) در بدن می‌شود





Electrical alternans Sinus tachycardia with electrical alternans which is characterized by beat-to-beat alternation in the QRS appearance (best seen in leads V2 to V4). These findings are strongly suggestive of pericardial effusion, usually with tamponade. The alternating ECG pattern is related to back-and-forth swinging motion of the heart in the pericardial fluid. Courtesy of Ary Goldberger, MD.

تامپوناد قلبی (Cardiac tamponade)

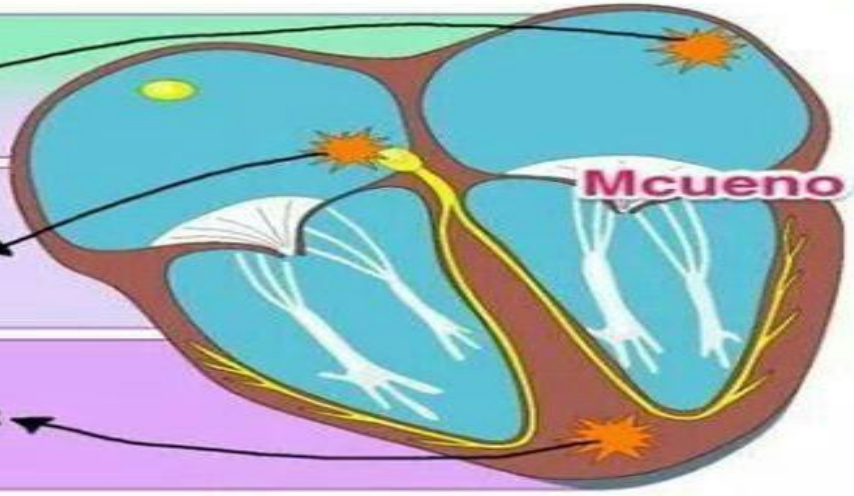


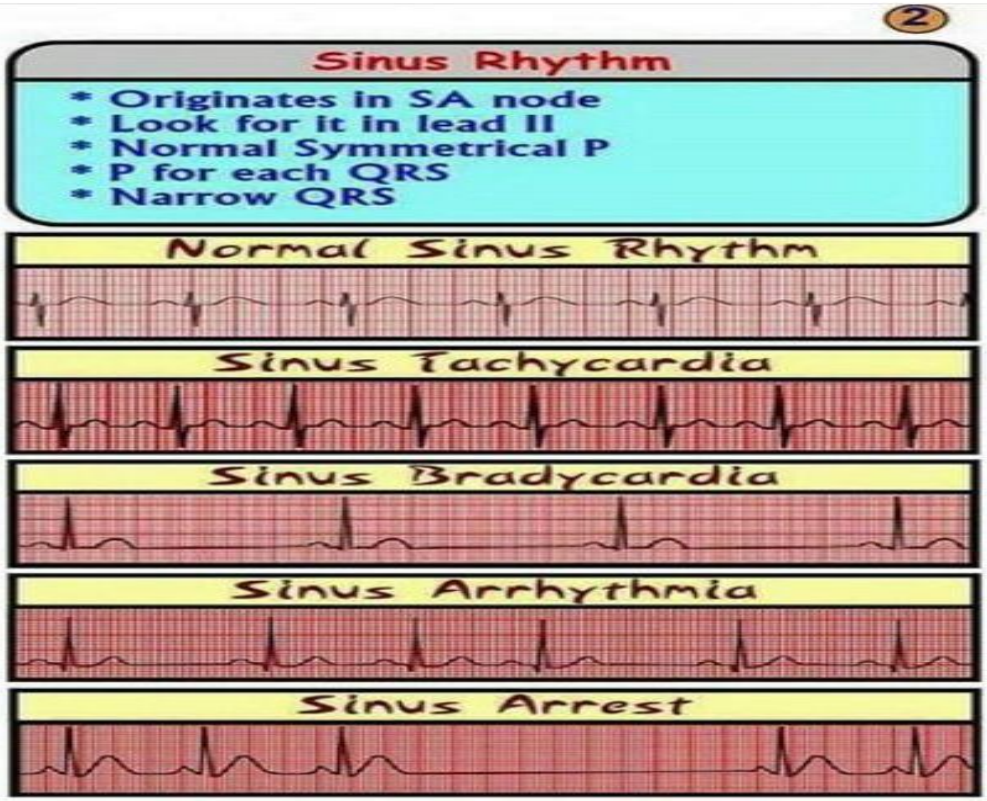
Pericardial effusion

350	FIBRILLATION	ATRIAL FIBRILLATION VENTRICULAR FIBRILLATION
	FLUTTER	ATRIAL FLUTTER VENTRICULAR FLUTTER
250	PAROXYSMAL TACHYARRHYTHMIA	ATRIAL TACHYCARDIA JUNCTIONAL TACHYCARDIA VENTRICULAR TACHYCARDIA
150	SIMPLE TACHYCARDIA	AVNRT (HR >140) AVRT (HR < 130) └ PRE-EXCITATION SYNDROMES SINUS TACHYCARDIA MULTIFOCAL ATRIAL TACH
100	NORMAL SINUS RHYTHM 1. HR 60-100 2. ORIGIN SHOULD BE SA NODE 3. IMPULSE PROPAGATE THROUGH NORMAL CONDUCTION PATHWAY 4. NORMAL VELOCITY	ATRIAL ESCAPE RHYTHM (HR= 60-80)
60	MILD BRADYARRHYTHMIA	SINUS BRADYCARDIA JUNCTIONAL ESCAPE RHYTHM (HR= 40-60)
40	MODERATE BRADYARRHYTHMIA	VENTRICULAR ESCAPE RHYTHM (HR= 20-40)
20	SEVERE BRADYARRHYTHMIA	
0		

SUPRAVENTRICULAR

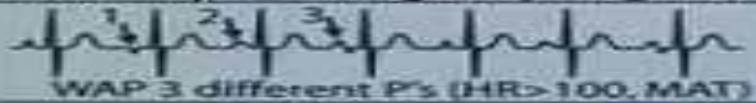
ESCAPES	PREMATURE BEATS	
ATRIAL ESCAPE BEAT	PREMATURE ATRIAL COMPLEX	ATRIAL
JUNCTIONAL ESCAPE BEAT	PREMATURE JUNCTIONAL COMPLEX	JUNCTIONAL (AV NODAL)
VENTRICULAR ESCAPE BEAT	PREMATURE VENTRICULAR CONTRACTION	VENTRICULAR






Sinus Rhythm (HR 60 - 99, regular)

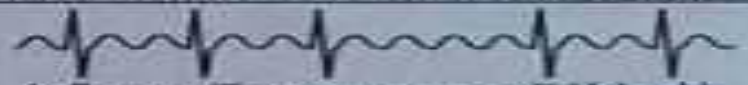

Sinus arhythmia (regularly irregular)



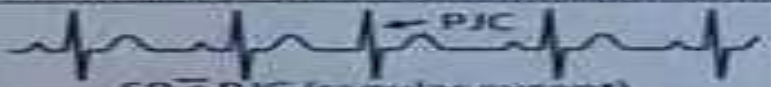
WAP 3 different P's (HR>100, MAT)

SR ⊂ PAC (regular except)


A-Fib (Wavy baseline irreg irregular)


A-flutter (F-waves, note AV block)


Junctional (Ps before, in or after QRS)

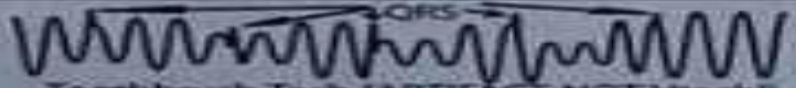


SR ⊂ PJC (regular except)

SR ⊂ PVC (PVC wide, bizarre and early)


Wandering Baseline (Resp. ARTIFACT)


60 Hz interference (Elec. ARTIFACT)



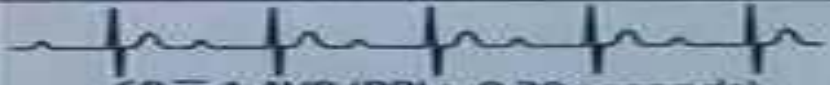
Toothbrush Tach (ARTIFACT, NOT Vtach!)

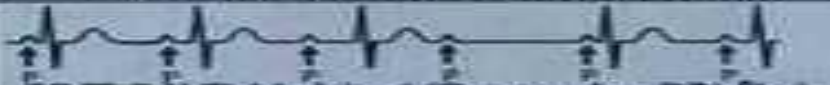

Coarse V-fib (No pulse) Fine V-fib


V-tach (Wide, T opposite, HR > 100)

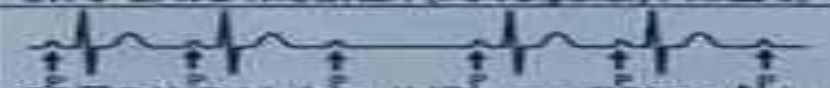

Torsades de Pointes (Twisting of Points)

Asystole (No pulse, check 2nd lead)


SR ⊂ 1 AVB (PRI > 0.20 seconds)



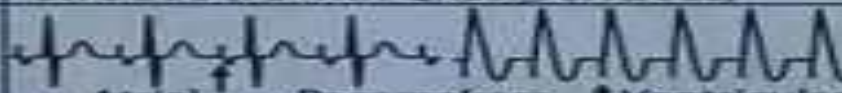
SR ⊂ 2AVB Mobitz I (Ps regular, PRI Δ s)



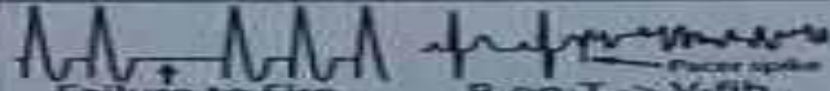
SR ⊂ 2AVB Mobitz II (P's reg, PRI no Δs)

SR ⊂ 3AVB (PRI Δ's, Ps and QRSs regular)


SR ⊂ BBB (Ps with QRSs > 0.12 sec)

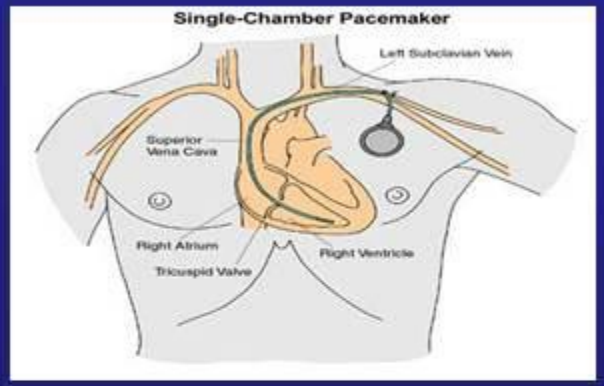

Atrial Pacemakers ↑ Ventricular


Failure to Capture Failure to Sense

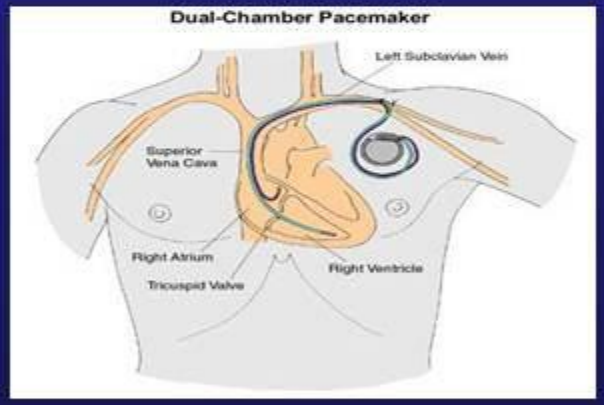


Failure to Fire R-on-T -> V-fib

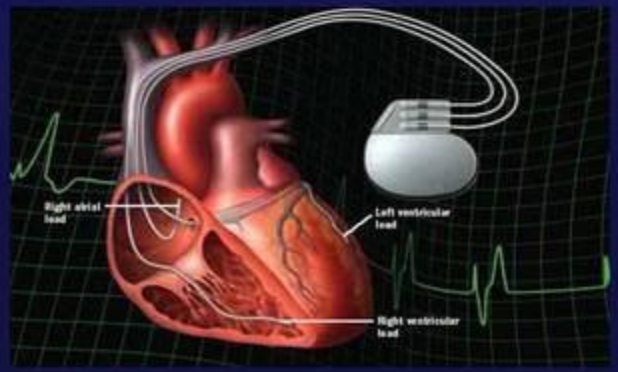
انواع پیس میکر از نظر حفرات درگیر



یک حفره ای SINGLE CHAMBER

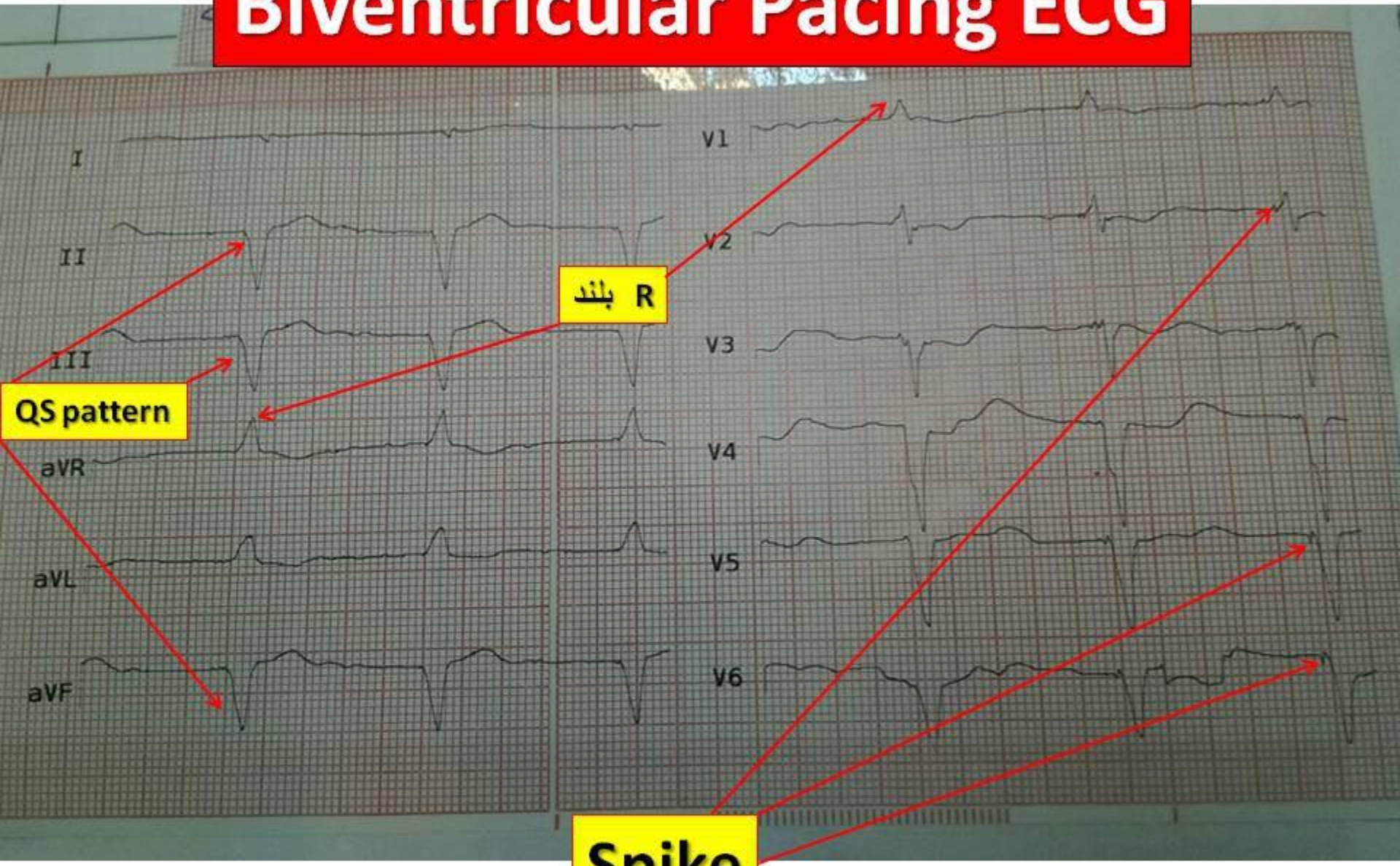


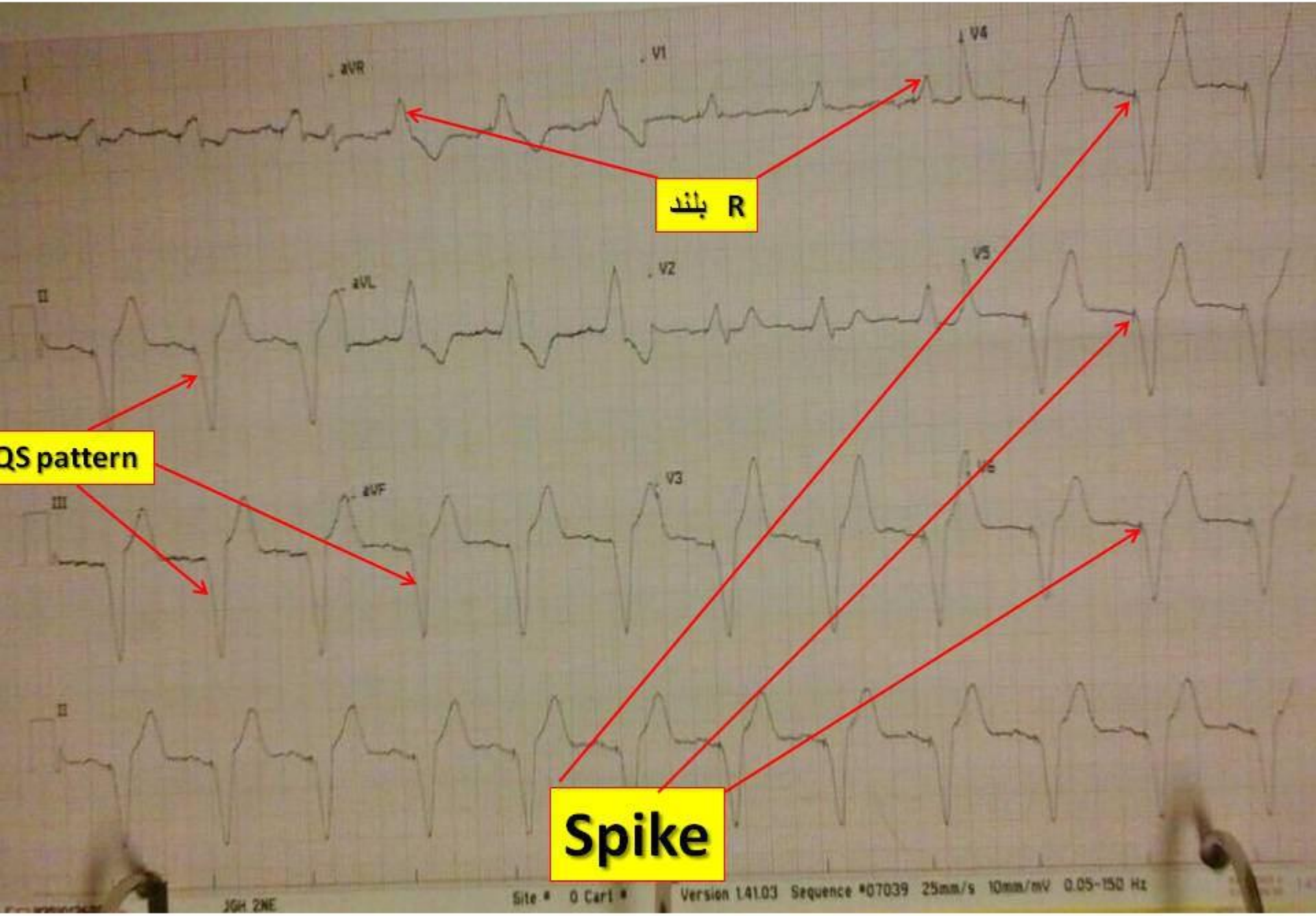
دو حفره ای DUAL CHAMBER



سه حفره ای BIVENTRICULAR PACING

Biventricular Pacing ECG




بلند R
QS pattern
Spike
Site # 0 Cart #
Version 1.41.03 Sequence #07039 25mm/s 10mm/mV 0.05-150 Hz

Disclaimer

The information and tools presented to you today are outstanding aids to your patient assessment and treatment, and ideally will enable you to provide better patient care.

The devices and information provided are designed to **AUGMENT** your patient assessment

NOT REPLACE IT!

PLEASE,

TREAT THE PATIENT, NOT THE MONITOR!


Thank you so much for your attention
Donna Sherwood