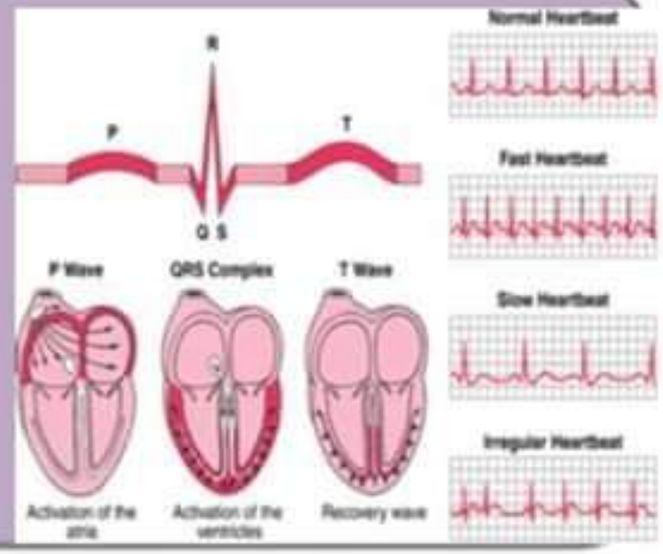
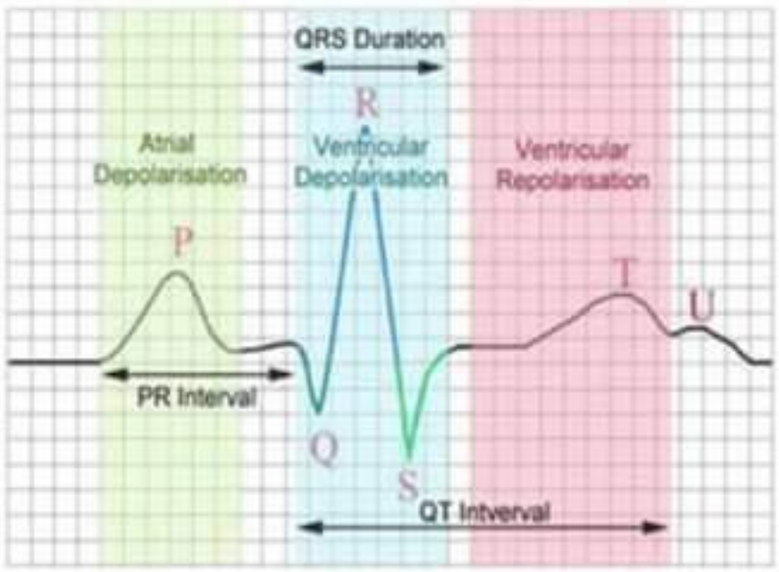
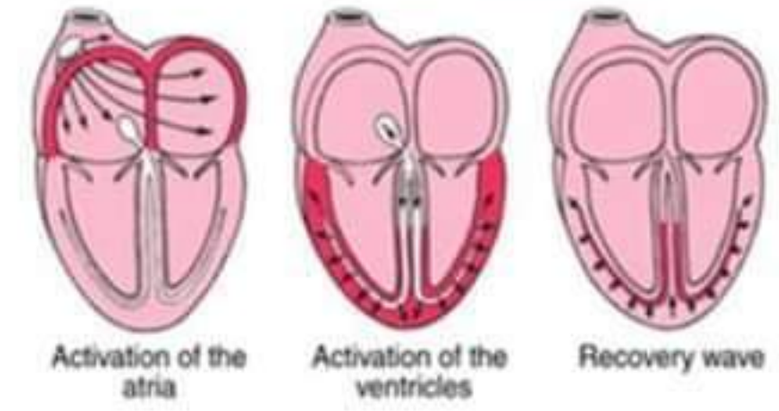
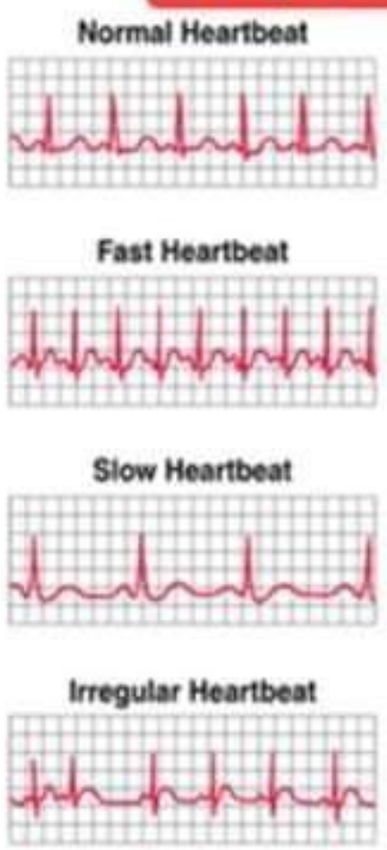
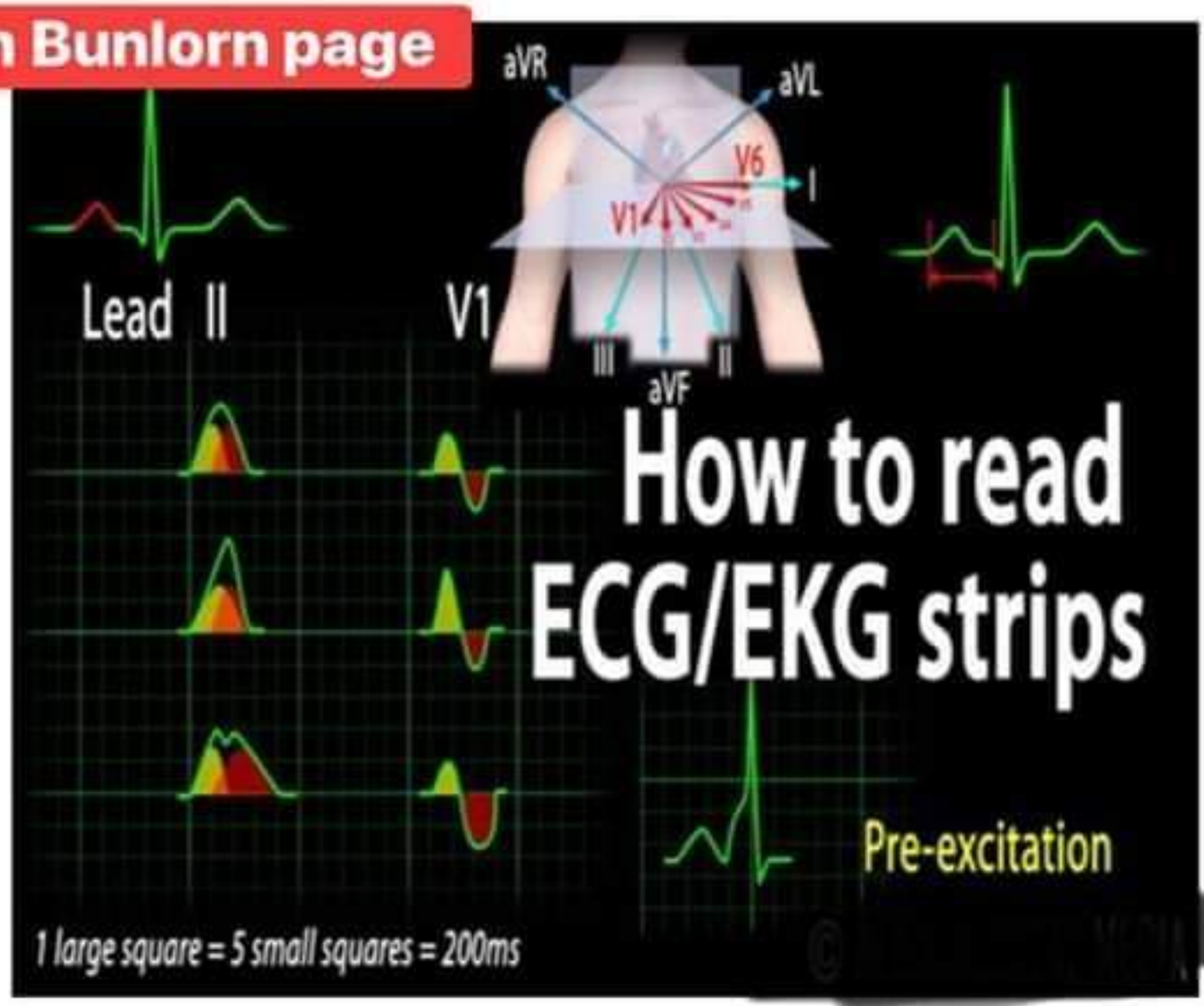
HOW TO READ AN ECG





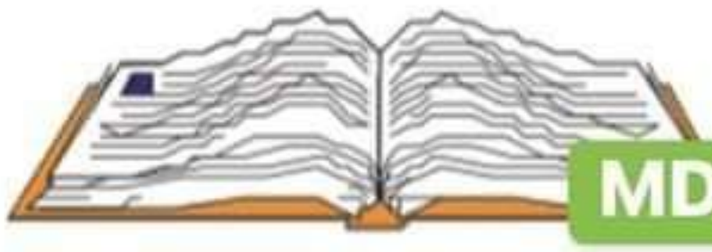






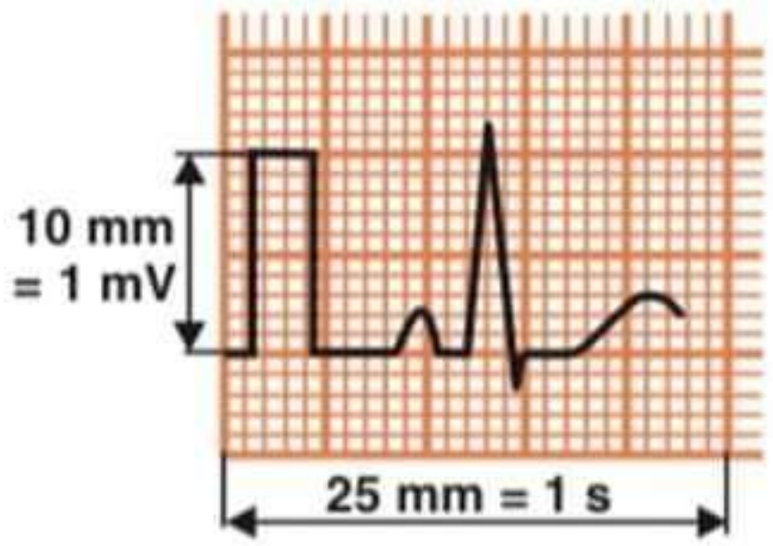
HOW TO READ AN ECG 1

STEP 1: Clinical history



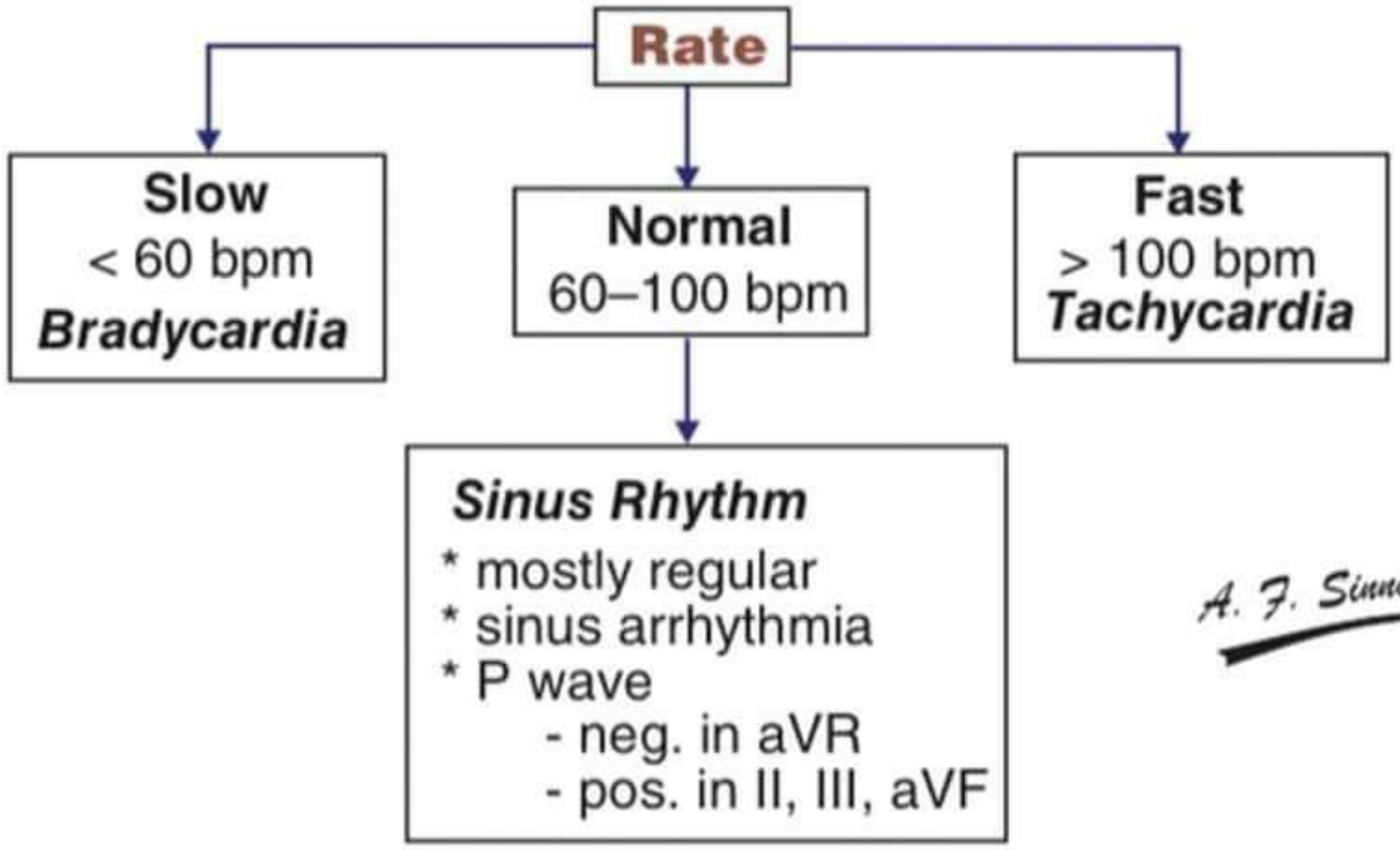
* Verify brief history
* Compare tracing with old records (if available)
* First look: "lay of the land"

STEP 2: Technical quality and calibration



* Check technical quality
* Check paper speed (25 mm/s ?)
* Check calibration (10 mm/mV ?)

STEP 3: Rate

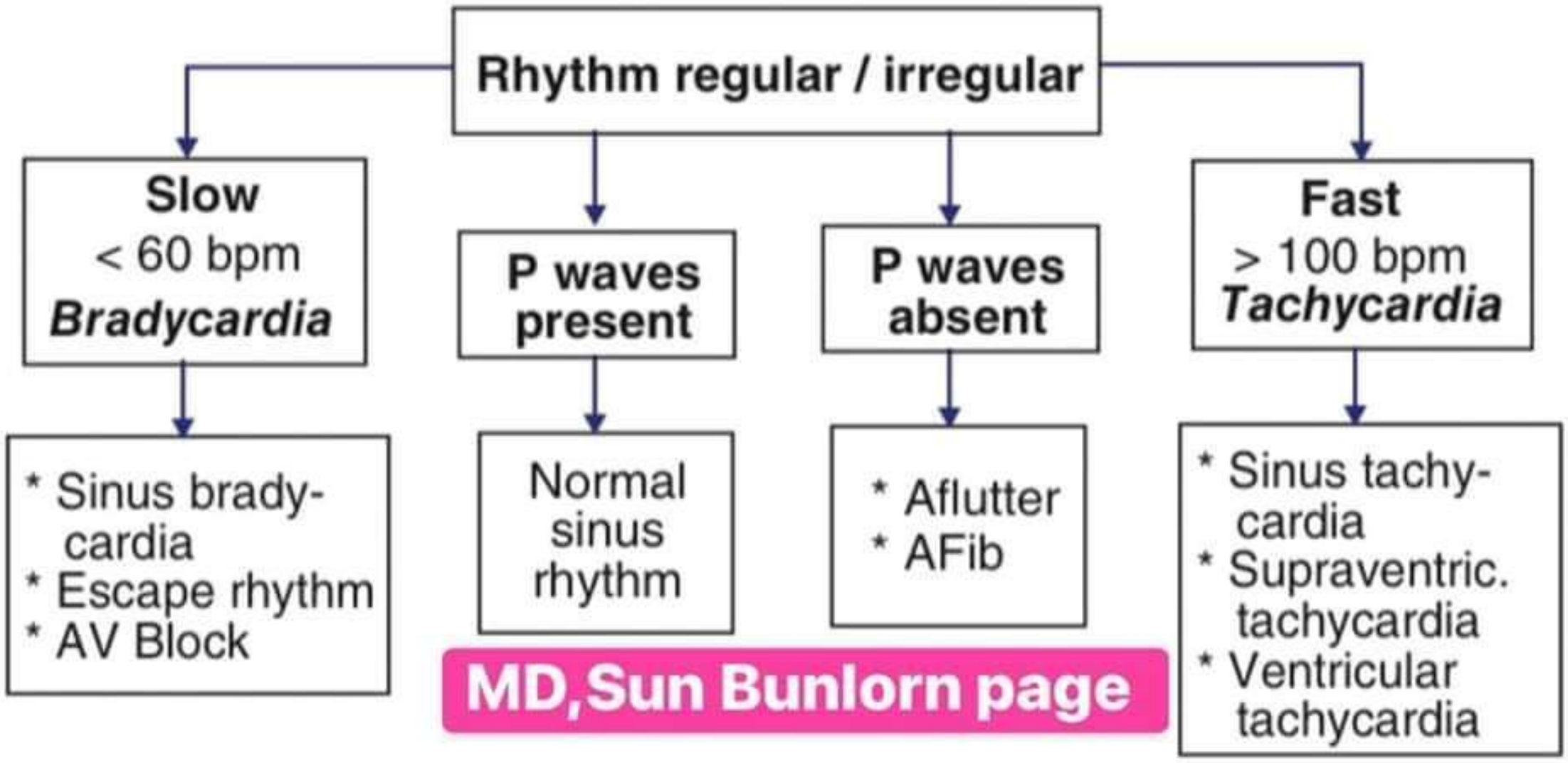


A. F. Sinnacve

if P wave is not upright in lead II consider
* arm lead reversal (P, QRS & T wave neg. in lead I)
* dextrocardia

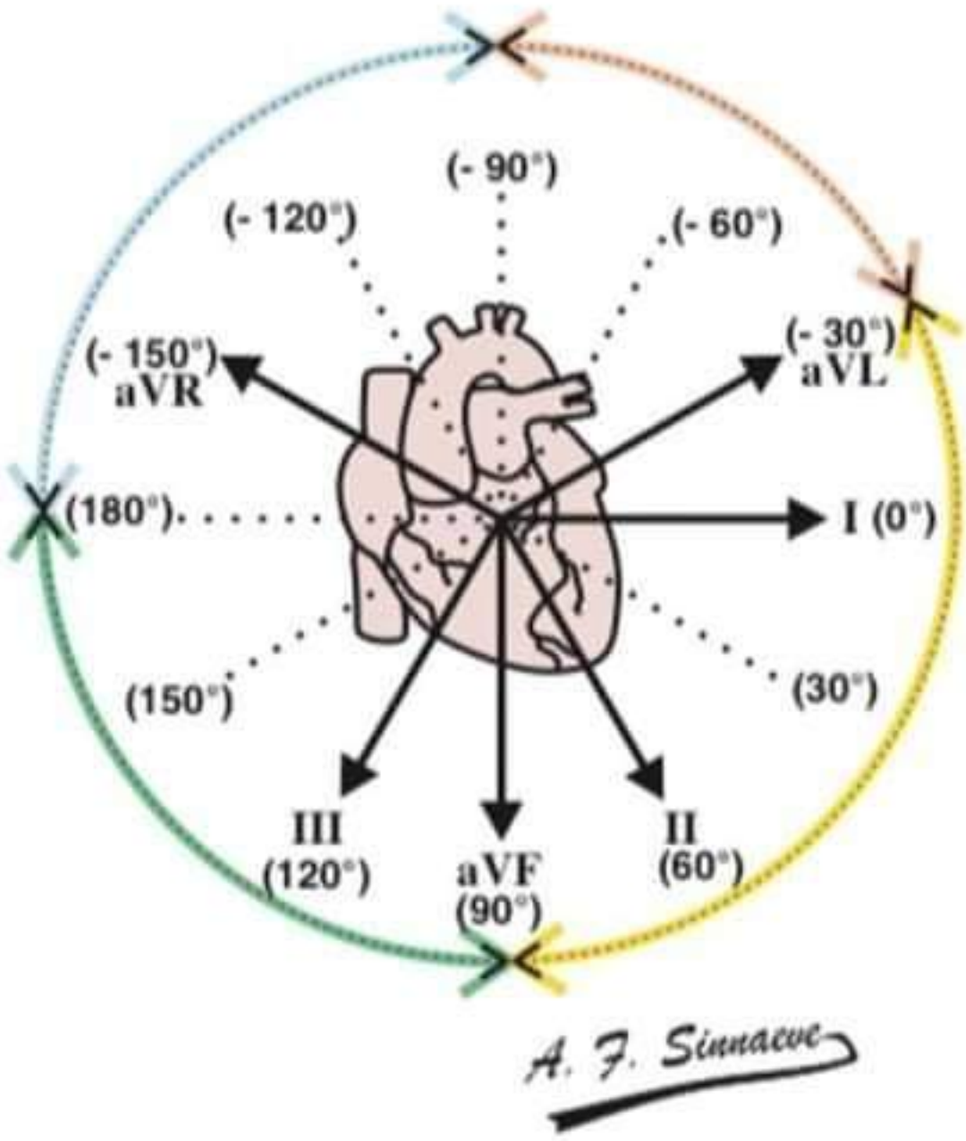
STEP 4: **Rhythm diagnosis**

* Rhythm regular or irregular? AFib (irregular), AFl (occasionally irregular), AV block (irregular with partial AV block)
* P waves present or absent?
* QRS narrow or wide?
* rate P: rate QRS relation? (rate P = rate QRS, rate P > rate QRS, rate QRS > rate P)



STEP 5: **Determine QRS axis in frontal plane**

Lead I	Lead III (or aVF)	*Quick and easy way*	
pos.	pos.	normal axis (–30° to +90°)	
pos.	neg.	**Look at lead II**	
		lead II neg.	left axis deviation
		lead II equiphasic (R = S)	axis –30°
		lead II pos.	normal axis
neg.	pos.	right axis deviation (+90° to +180°)	
neg.	neg.	right superior "no mans land" (–90° to –180°)	

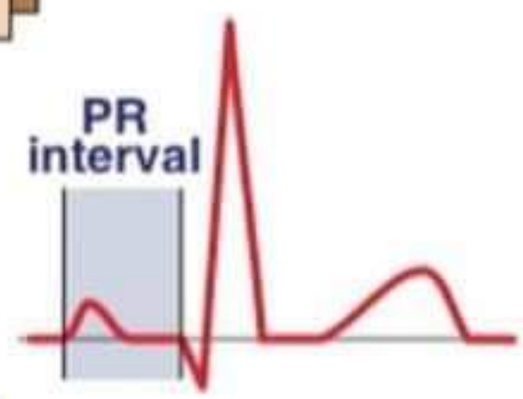


LAH : rS in Lead II and aVF and Lead III
LPH : rS in Lead I and aVL

Abbreviations: AFib: atrial fibrillation; AFl: atrial flutter; pos: positive; neg: negative; LAH: left anterior hemiblock; LPH: left posterior hemiblock

HOW TO READ AN ECG 2

STEP 6A: Measurement of PR interval



PR interval

- **Short PR** < 0.12 s
  - * WPW
  - * Low atrial rhythm
  - * Upper AV junctional rhythm
  - * Normal variant
- **Normal** 0.12–0.20 s
- **Prolonged PR** with preserved AV conduction > 0.20 s
  - * No dropped beats: AVB I
  - * Dropped beats
  - * No AV conduction

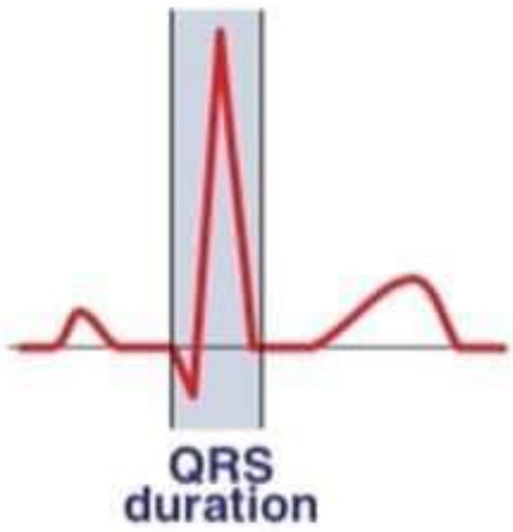
MD,Sun Bunlorn page

A very long PR may push the P wave into the T wave where it may not be identified

STEP 6B: Measurement of QRS complex: duration

QRS duration (QRSd)

- **QRSd Normal** < 0.10 s
- **QRSd** ≥ 0.10 s and < 0.12 s
  - * Consider incomplete BBB
  - * Intraventricular conduction disorder
- **QRSd wide** > 0.12 s
  - * Sinus rhythm: RBBB or LBBB
  - * No sinus rhythm: ventricular rhythm or VT



If sinus rhythm and wide QRS, look at three leads V1, V6 and I

STEP 6C: Measurement of QRS complex: voltage

QRS voltage

High QRS voltage

SV1 + RV6 > 35 mm
R in I > 15 mm
R > aVL > 11 mm

↓

LVH

↓

Exclude:
* Ventricular pacing (look for pacemaker stimuli)
* Hyperkalemia (if prolonged QRS)
* Look for delta wave (WPW)

↓

The absence of LVH on ECG means nothing, if present LVH is likely.

Low QRS voltage

Amplitude of all QRS complexes in limb leads < 5 mm or < 10 mm in the precordial leads

↓

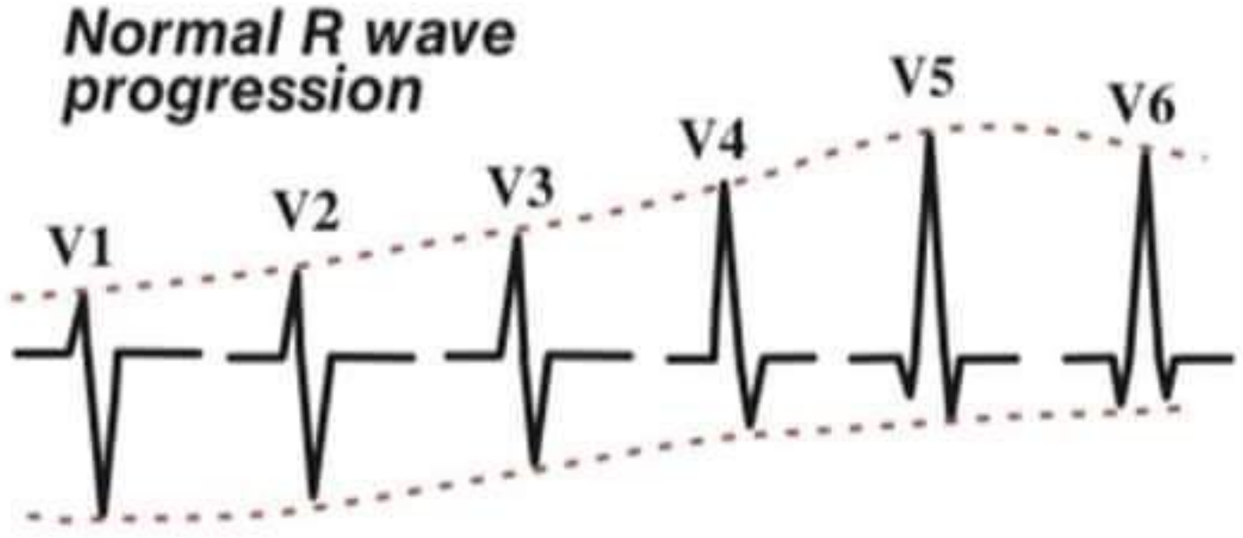
* Pericardial effusion (triad of low voltage, tachycardia, electrical alternans)
* Pleural effusion
* Emphysema (COPD)
* Pneumothorax
* Marked obesity
* Previous massive MI
* End-stage dilated cardiomyopathy
* Hypothyroidism
* Infiltrative myocardial disease

COPD: chronic obstructive pulmonary disease; MI: myocardial infarction; LVH: left ventricular hypertrophy

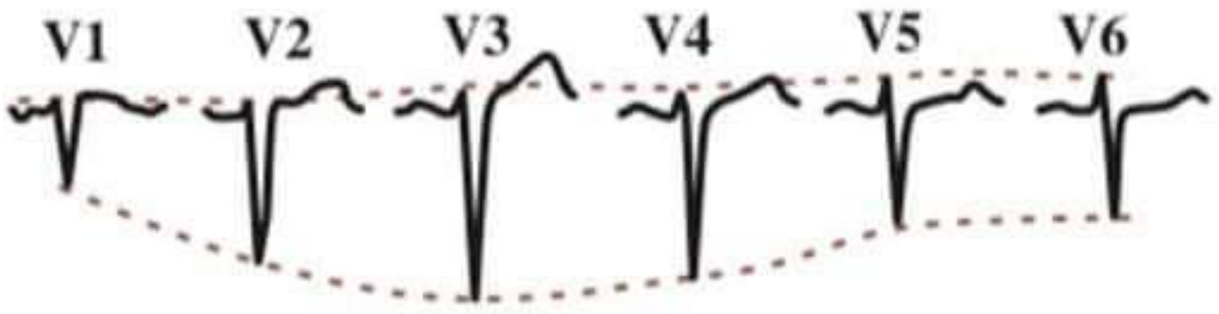
Thin people, athletes, and young adults frequently show tall QRS voltage without LVH

STEP 7A: Look at R wave progression in precordial leads

Normal R wave progression



Slow R wave progression



Causes of slow R wave progression:

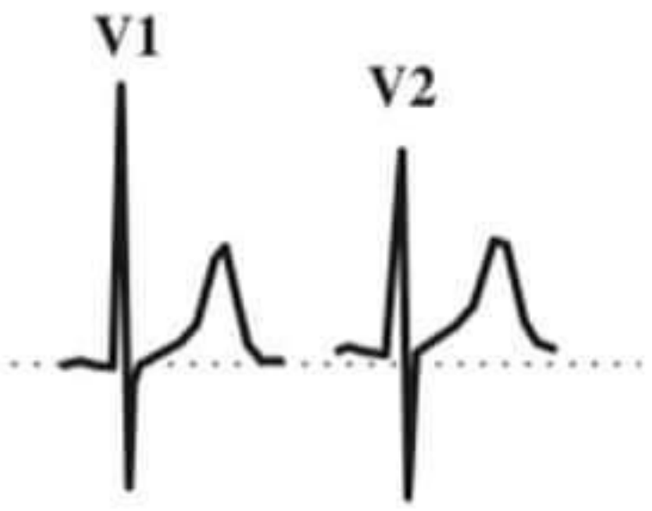
* Anterior myocardial infarction
* Incorrect lead placement (in obese women)
* LVH, * LBBB, * WPW
* Dextrocardia
* Tension pneumothorax with mediastinal shift
* Congenital heart disease

LVH: left ventricular hypertrophy; LBBB: left bundle branch block; WPW: Wolff-Parkinson-White

HOW TO READ AN ECG 3

STEP 7B: Look at R wave progression in precordial leads

Reversed R wave progression



This describes abnormal R waves in lead V1 that progressively decrease in amplitude if the QRS is narrow. This pattern may occur with a number of conditions, including RVH, posterior (or posterolateral) MI, dextrocardia (in concert with a limb lead reversal pattern), misplaced leads and rarely as a normal variant. If the QRS is wide, a dominant R wave in V1 may be caused by RBBB or WPW.

MI: myocardial infarction; RBBB: right bundle branch block; WPW: Wolff-Parkinson-White syndrome; RVH: right ventricular hypertrophy;

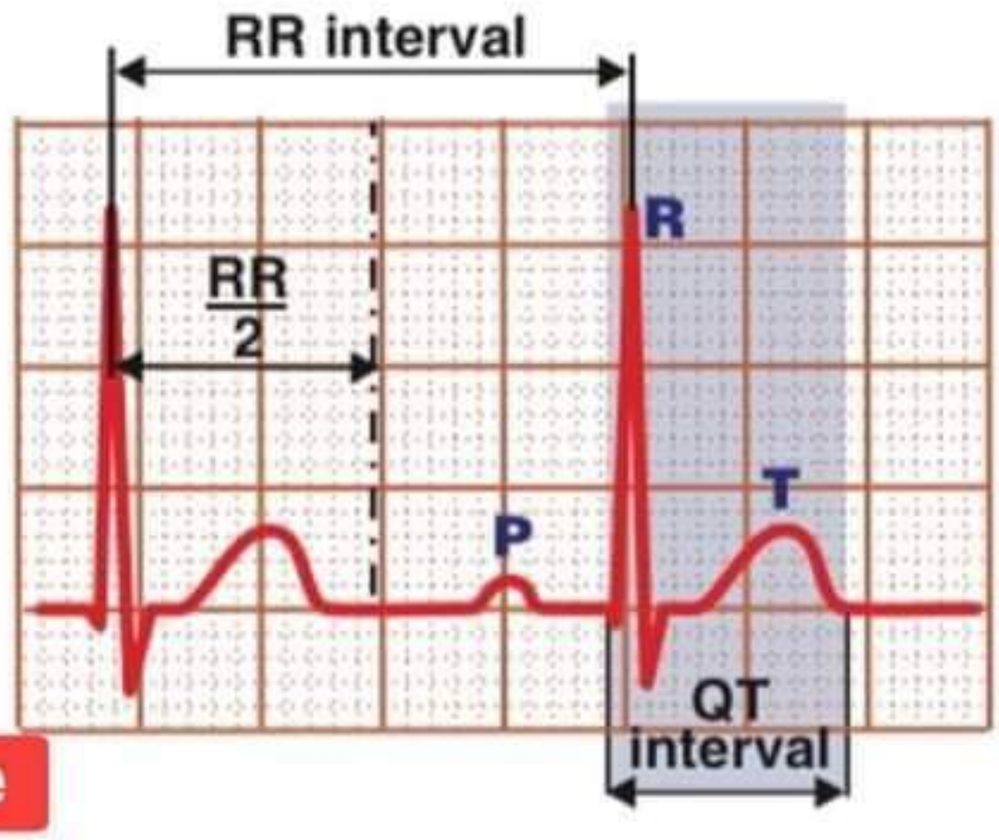
STEP 8: QT Interval

QT**c** = **c**orrected QT interval

$$QTc\ (s) = \frac{QT\ interval\ (s)}{\sqrt{RR\ interval\ (s)}}$$

Normal QTc ≤ 440 ms

slightly larger values are acceptable in women



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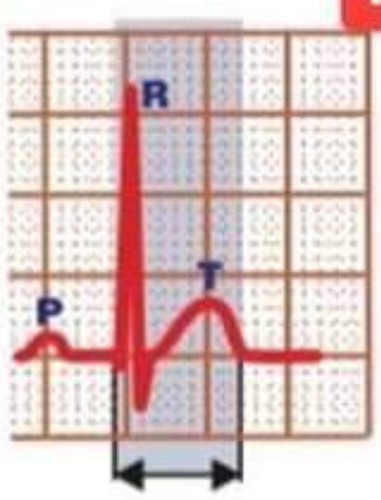
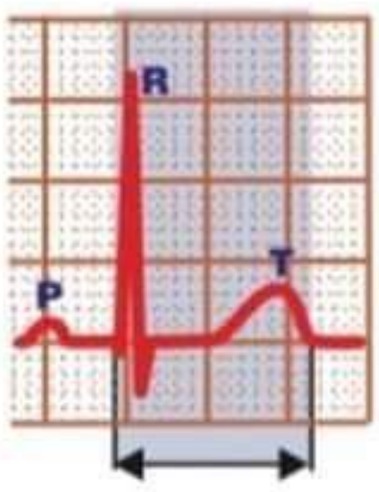


Short QTc ≤ 330 ms
Increased risk of AF & VF

Causes
* Congenital (requires family history)
* Acquired
  - drug toxicity
  - electrolyte imbalance



Long QTc ≥ 460 ms

If QTc ≥ 500 ms : high risk for EAD & TdP (after short-long-short sequence)

Abbreviations
EAD = early afterdepolarization
TdP = torsades de pointes
AF = atrial fibrillation
VF = ventricular fibrillation

The QT interval is a measure of ventricular action potential duration. It decreases when the heart rate increases.

STEP 9: U wave

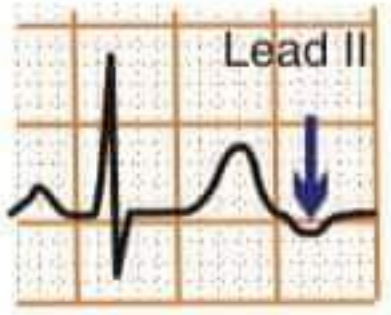
* small deflection (0.5 mm), immediately following the T wave
* usually same polarity as T wave
* possibly originating from Purkinje network
* best seen in lead V2 and V3 and during slow rate

Normal

Prominent U wave

Hypokalemia

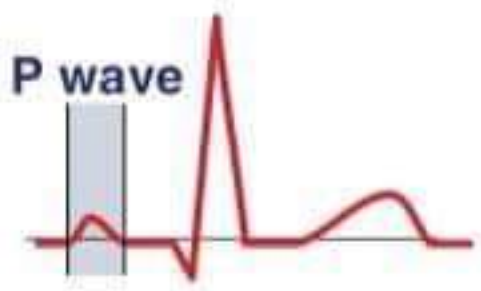
Inverted U wave



* Myocardial ischemia
* LV volume overload

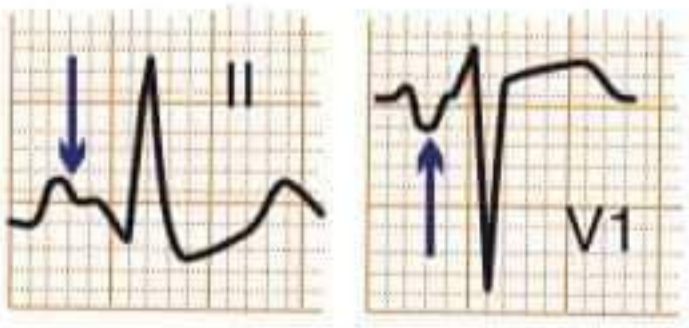
STEP 10: P wave

P waves are best seen in lead II and V1



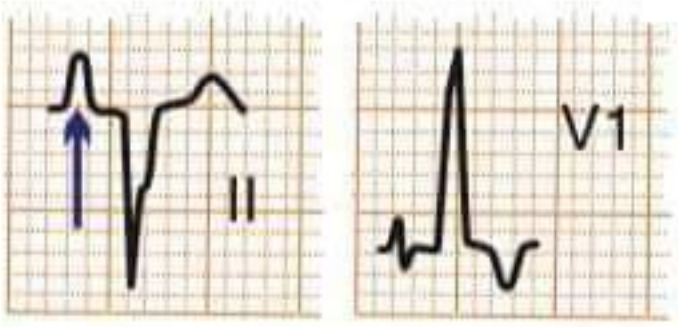
Normal P wave * lead II ≤ 0.12 s and < 2.5 mm
* lead V1 biphasic

P wave too wide
Lead II ≥ 0.12 s & notched
Prominent negative terminal forces in lead V1



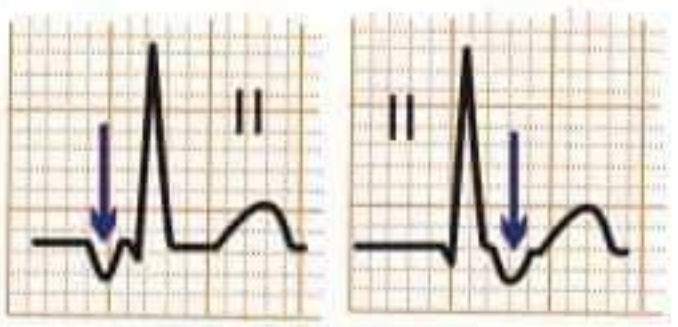
LA abnormality

P wave too tall
P peaked in lead II
P > 2.5 mm in inferior leads
P > 1.5 mm in lead V1



RA abnormality

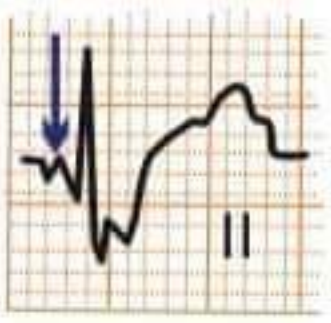
P wave negative
in inferior leads
(II, III, aVF)



Retrograde P wave ?
Ectopic beat ?

A. F. Simancas

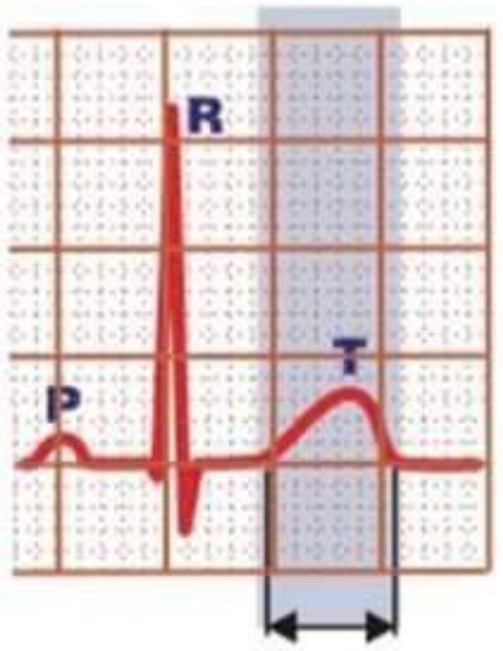
P wave funny



Ectopic beat ?

HOW TO READ AN ECG 4

STEP 11: T wave



Normal T wave

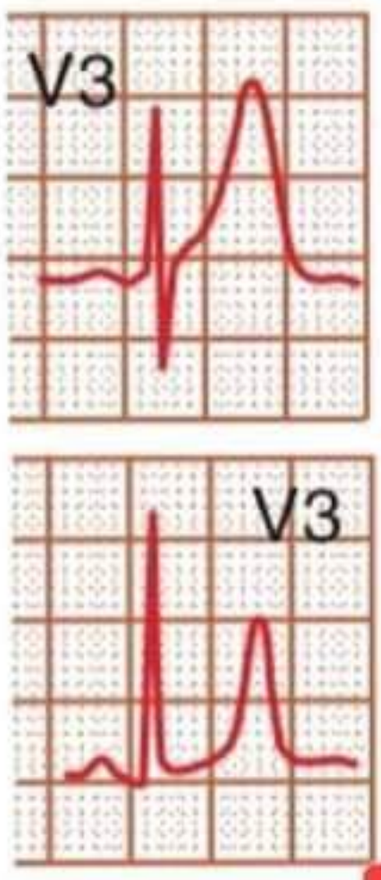
* T wave same polarity as main QRS deflection
* T wave is upright in I, II, V3 to V6
* T wave always inverted in aVR

T wave inversion in V1 to V3

* Common finding in children and adolescents
* Infrequently found in healthy adults

Is not associated with adverse outcome if T waves are normal in other leads

High amplitude T waves

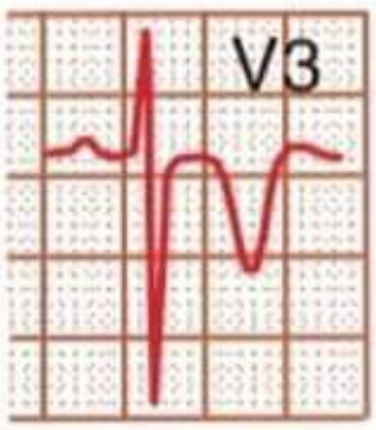


* Hyperacute T wave
  - *may be seen 5–30 min after onset of MI*
  - *broad-based T wave*
  - *round summit*

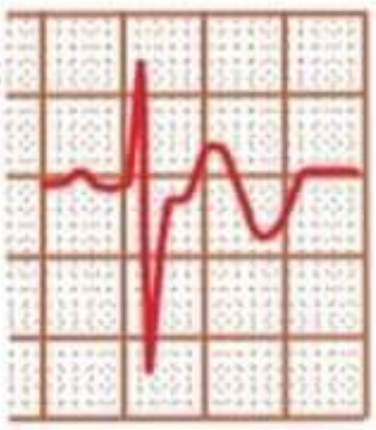
* Hyperkalemia (best seen in precordial leads)
  - *narrow-based T wave*
  - *tenting of T wave*

Abnormal T waves

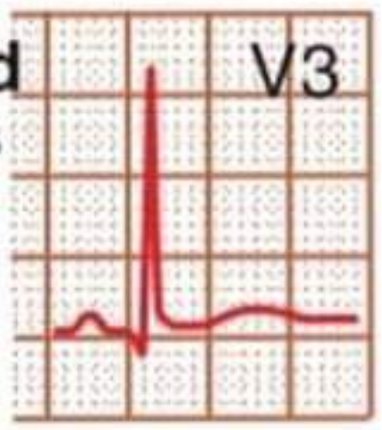
Inverted T waves



Biphasic T waves



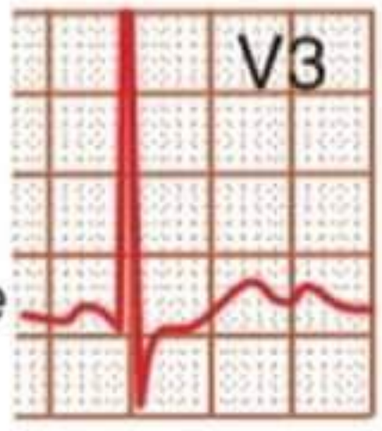
Flattened T waves

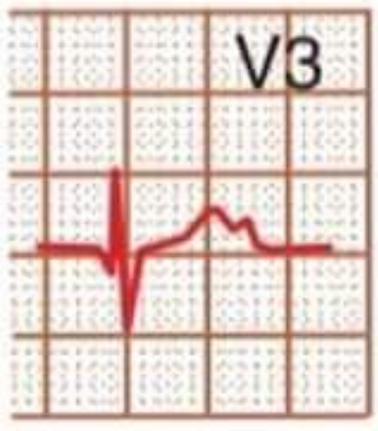


Camel hump T waves

Severe hypokalemia

prominent U wave fused to end of the T wave





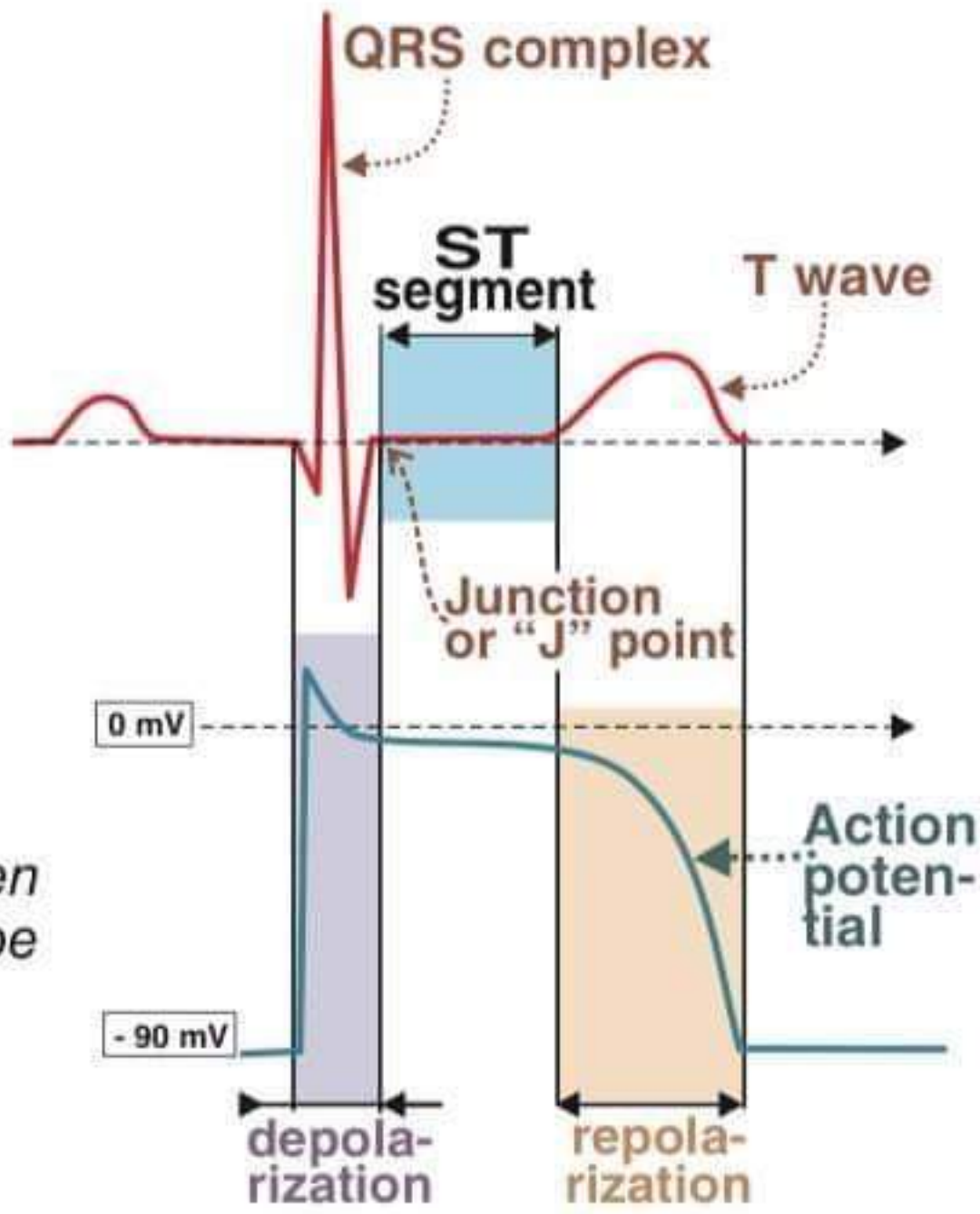
Hidden P waves

embedded in T wave

- *sinus tachycardia*
- *various types of heart block*

STEP 12: ST segment

* *The typical ST segment duration is usually around 0.08 s (80 ms).*
* *Usually flat and isoelectric and should essentially level with PR and TP segments.*
* *The ventricles remain depolarized during the ST segment.*
* *It is usually difficult to determine exactly where the ST segment ends and the T wave begins. Therefore the relationship between ST segment and T wave should be evaluated together.*



The most important cause of ST segment abnormality (elevation or depression) is myocardial ischemia or infarction.

A. F. Sinnacve

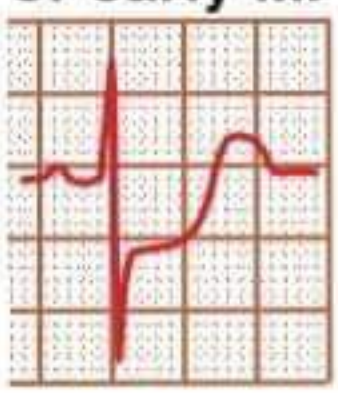
The diagnosis of AMI and severe ischemia depend on the careful assessment of the ST segment.

AMI: acute myocardial infarction

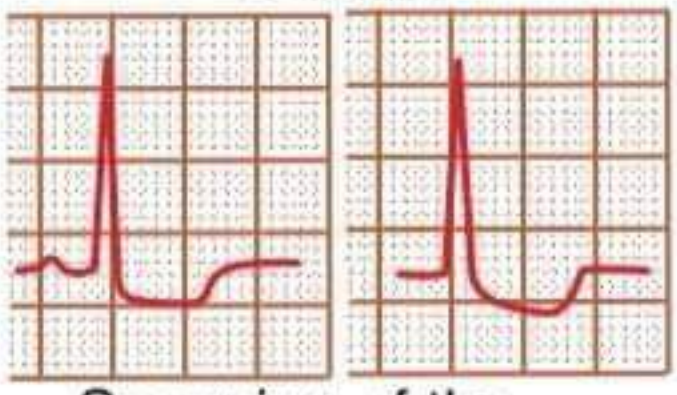
ST segment depression

* Ischemia (**most common**)
* Subendocardial infarction
* Reciprocal changes associated with AMI
* Drug effects

ischemia or early MI



digitalis effects

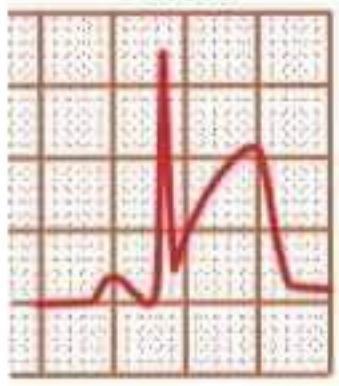


Scooping of the ST segment

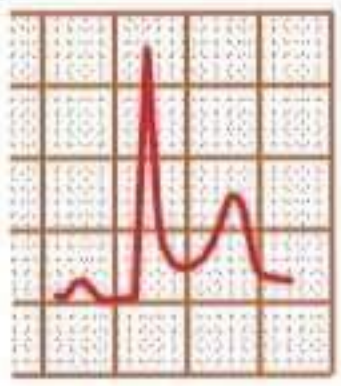
ST segment elevation

* AMI (*convex ST segment elevation*)
* Transmural ischemia
* Ventricular aneurysm *(ST elevation does not subside after AMI)*
* Pericarditis

AMI



pericarditis



Abbreviations

MI : myocardial infarction
AMI: acute myocardial infarction

HOW TO READ AN ECG 5

STEP 13: ST elevation MI (STEMI)

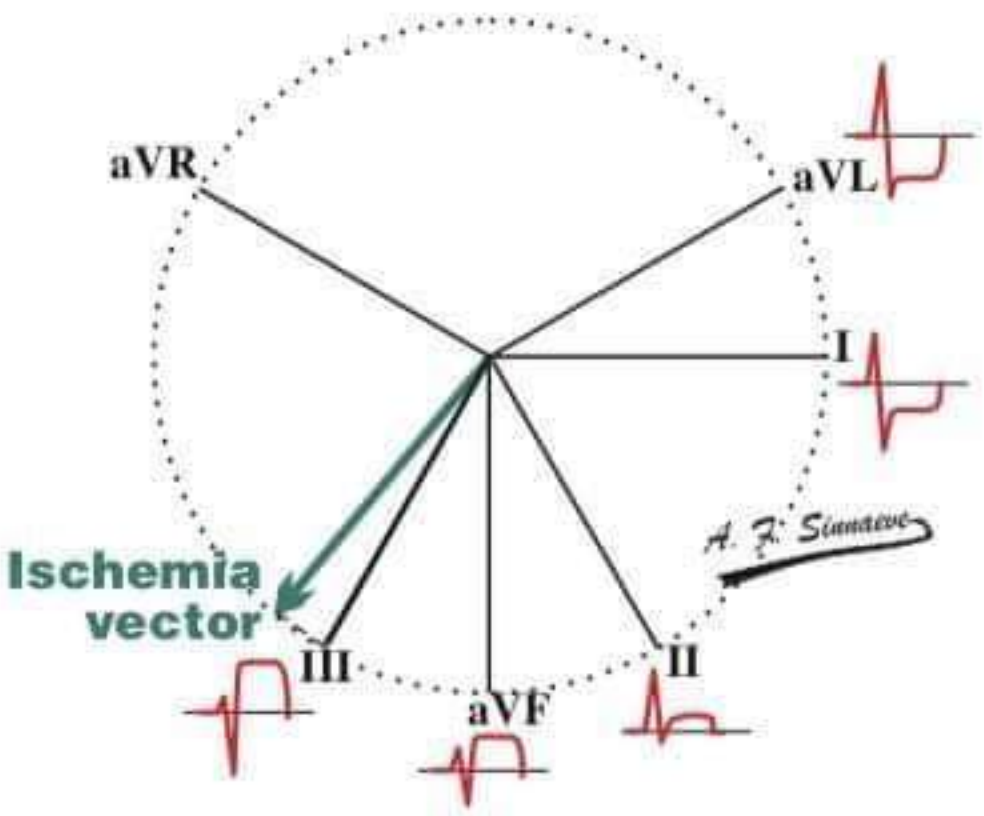
ECG diagnosis of STEMI

* Limb leads: ST segment elevation ≥ 1 mm (0.1 mV)
* Precordial leads: ST segment elevation ≥ 2 mm (0.2 mV)
* Elevations must be present in anatomically contiguous leads

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

* An approximation of the infarction size can be assessed from the extent of ST elevation.
* *Of note: Over 90% of healthy men have at least 1 mm (0.1 mV) of ST segment elevation in at least one precordial lead.*

Look for reciprocal changes of ST depression in leads opposite the area undergoing injury



For example in acute transmural inferior ischemia, the direction of the ST vector faces the injured area (resulting in a ST elevation in leads II, III, aVF) while the tail of the ST vector faces anatomically opposite sites where it causes ST segment depression (lead aVL and lead I).

The significance of these reciprocal changes or mirror-images is unclear but they are useful diagnostically by providing confirmatory evidence for the diagnosis of STEMI.

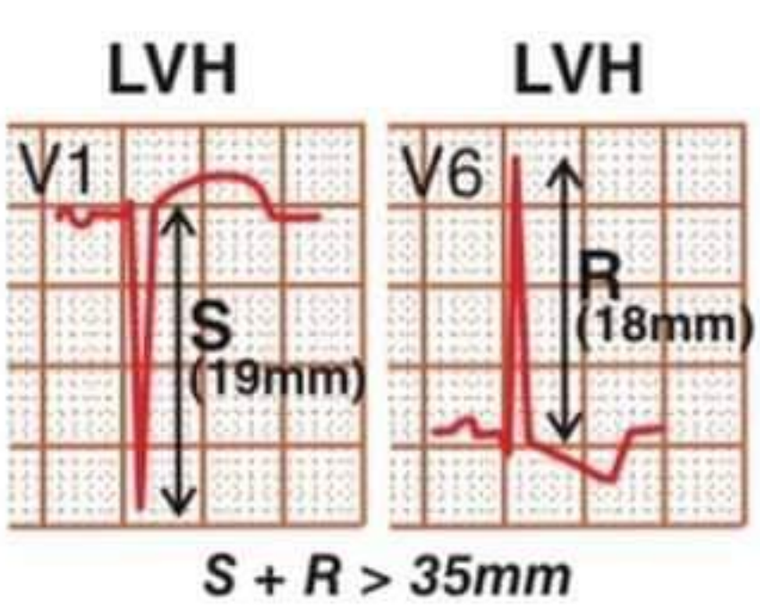
Mirror-image changes do not occur in pericarditis

An acute STEMI can present with upwardly concave ST elevation, so the mere fact that ST elevation is upwardly concave does NOT mean that a condition other than ischemia is present.

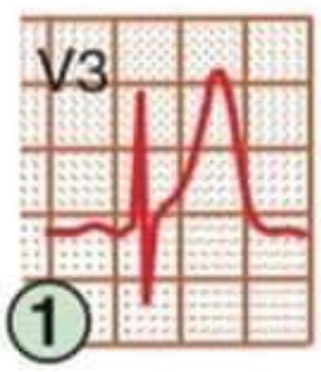
ST elevation due to ischemia or infarction: Focus on contiguous leads and those showing reciprocal changes!

One must be well versed in recognizing the so-called ECG mimics of acute myocardial infarction. The development of reciprocal changes during STEMI helps the differentiation from the listed conditions:

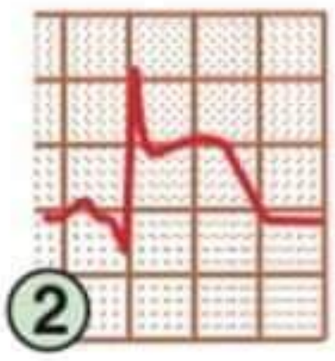
ECG mimics of AMI:
* Left ventricular hypertrophy (LVH)
* Left bundle branch block (LBBB)
* Paced rhythm
* Early repolarization
* Pericarditis
* Hyperkalemia
* Ventricular aneurysm



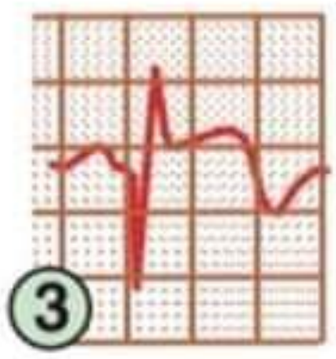
Evolution of STEMI



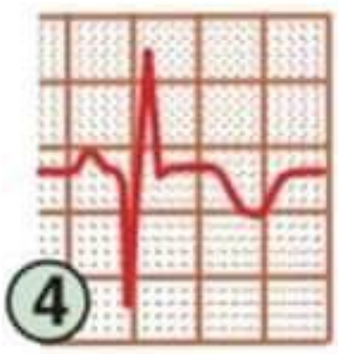
Sometimes the earliest presentation of AMI is the hyperacute T wave, which is treated the same as ST elevation. In practice this is rarely seen



In the first few hours the ST segments usually begin to rise

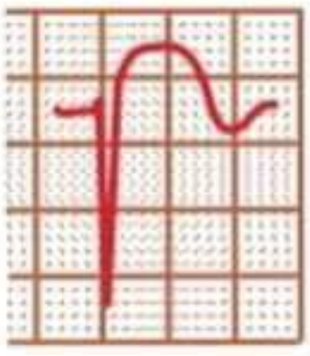


Pathologic Q waves may appear within hours or may take more than 24 hr. The T wave will generally become inverted in the first 24 hr, as ST elevation begins to resolve



Long-term changes of ECG include persistent Q waves (in 90% of cases) and persistent inverted T waves

Note :



Persistent ST elevation is rare except in the presence of a ventricular aneurysm

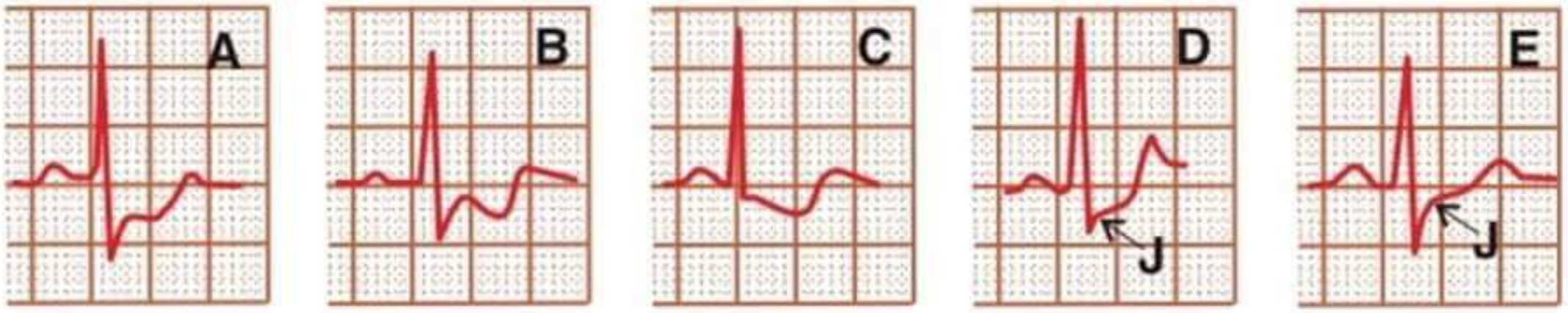
A. F. Sinnaeve

Abbreviation: *AMI: acute myocardial infarction*

HOW TO READ AN ECG 6

STEP 14: Non-ST elevation MI (NSTEMI)

* The ECG sign of non-STEMI is ST segment depression
* ST segment depression seen in subendocardial ischemia or infarction can take on different patterns. The most typical is a horizontal or downsloping depression (A, B, C). Upsloping ST depression (D) is less specific.



* Upsloping depression of less than 1 mm at 80 ms beyond the J point (E) is simply J point depression and not ST segment depression.

The label of nonspecific ST-T wave abnormalities is somewhat vague but it does not mean it's not important.

* Depression is reversible if ischemia is only transient but depression persists if ischemia is severe enough to produce infarction.
* T wave inversion with or without ST segment depression is sometimes seen but not ST segment elevation or new Q waves.
* The nonspecific ST-T wave changes should be evaluated with old ECGs because myocardial ischemia is not a static process.

GUIDELINES

MD,Sun Bunlorn page

ECG manifestations of acute myocardial ischemia (in absence of LVH and LBBB) according to ESC/ACC/AHA (2012 definitions)

1. ST Elevation

New ST elevation at the J point in 2 contiguous leads with the following cut-points:

Age and gender specific !

* ≥ 0.1 mV in all leads except leads V2-V3 in men and women
* in leads V2-V3: ≥ 0.2 mV in men ≥ 40 years and
  ≥ 0.25 mV in men < 40 years
* in leads V2-V3: ≥ 0.15 mV in women

2. ST Depression and T wave changes

* New horizontal or down-sloping ST segment depression ≥ 0.05 mV in 2 contiguous leads
* and/or T wave inversion ≥ 0.1 mV in 2 contiguous leads with prominent R wave or R/S ratio > 1

STEP 15: Additional information

Early Q waves

* In the chronic phase of myocardial infarction, Q waves are regarded as a sign of irreversible necrosis.
    - *However, about 50% of patients presenting within 1 hour of onset of ST elevation acute coronary syndrome already have Q waves in the leads with ST elevation, especially in the anterior leads.*
    - *These Q waves may be transient and not necessarily represent irreversible damage.*
    - *They may represent transient loss of electrical activity in the region at risk ("myocardial concussion").*
* Thus, Q waves on presentation may reflect either irreversible damage and/or a large ischemic zone.

Do not overlook RV infarction

* Request right-sided leads for the diagnosis of right ventricular (RV) myocardial infarction (MI) if ECGs show acute inferior MI, anterio-lateral and posterior MI.
* The 12-lead ECG may suggest RV MI if the magnitude of ST elevation in V1 > the magnitude of ST elevation in V2.
* The combination of ST elevation in V1 and ST depression in V2 is highly specific for right ventricular MI.

Abnormal Q waves

* In the acute phase of myocardial infarction, ST elevation is the key to the diagnosis and therapy.
* The presence of Q waves is far less important for diagnosis and treatment. Indeed, the early diagnosis does not depend on Q waves.

Definition of significant q/Q wave in myocardial infarction (MI). ECG changes associated with prior MI according to ESC/ACC and AHA (2012 definitions)

* Any Q wave in leads V2-V3 ≥ 0.02 s (20 ms) or QS complex

or

* Q wave ≥ 0.03 s (30 ms) and ≥ 0.1 mV deep
  or QS complex in leads I, II, aVL, aVF or in V4-V6
  or in any 2 contiguous lead grouping (I, aVL, V1-V6, II, III, aVF)

or

* R wave ≥ 0.04 s (40ms) in V1-V2 and R/S ≥ 1 with concordant positive T wave (in absence of conduction defect)

Abbreviations: *LVH: left ventricular hypertrophy; LBBB: left bundle branch block MI: myocardial infarction*

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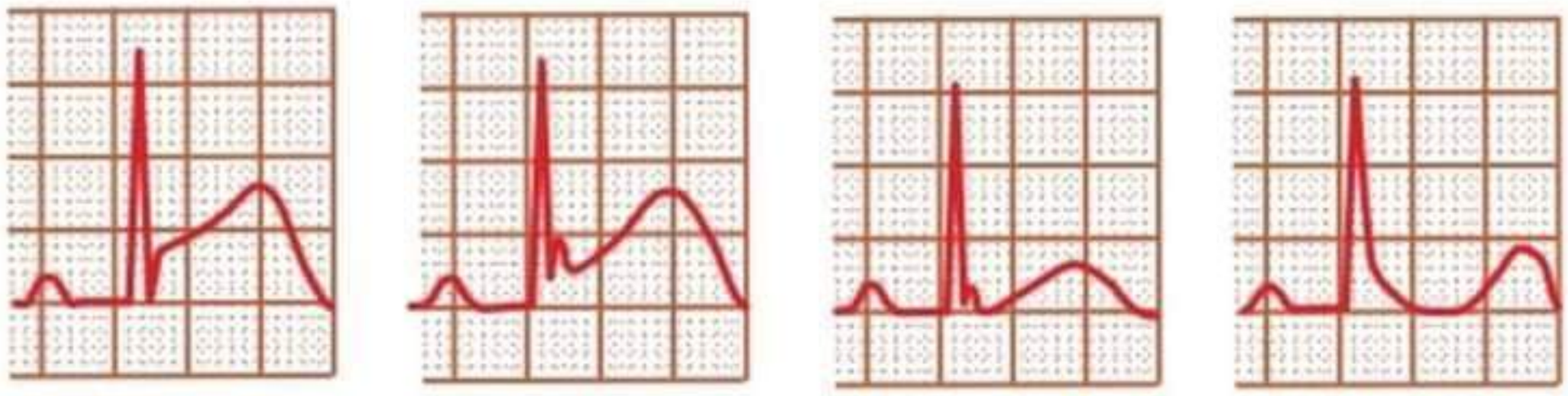
STEP 16: Early repolarization

Early repolarization (ER) is defined as J point elevation with the terminal QRS showing either:

* notching (a positive deflection on terminal QRS complex) or
* slurring (on the downslope portion of the QRS complex)



Various patterns of early repolarization

The changes tend to disappear with tachycardia.

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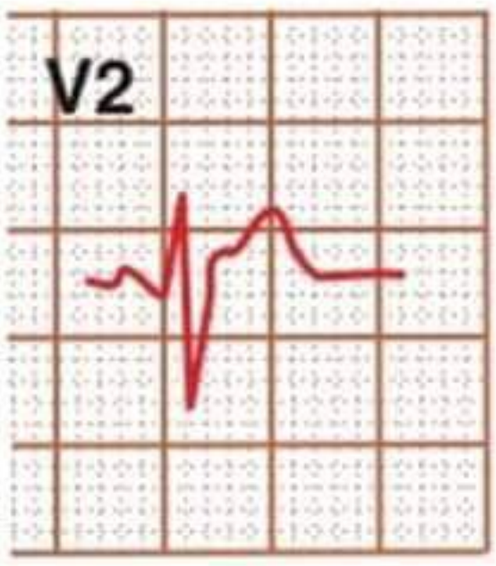
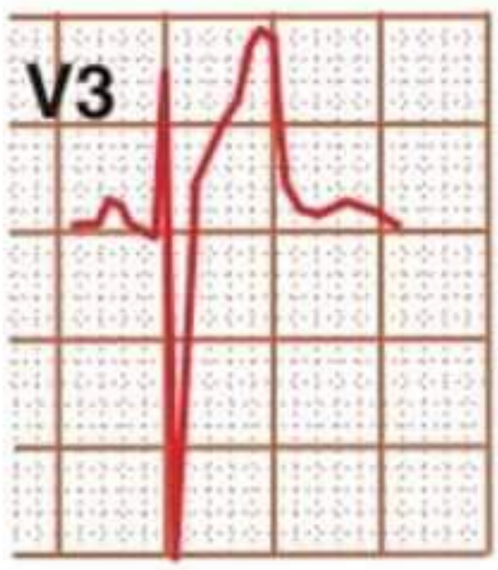




Early repolarization has recently been subject to much research because of the association of sudden death and malignant arrhythmias in patients with certain specific ECG features.
The common form of early repolarization with a high ST take-off in the right precordial leads is considered benign and common, especially in athletes.

* There is a typical concave upward ST segment elevation (1–4 mm), prominent symmetrical T waves and absence of reciprocal ST depression.

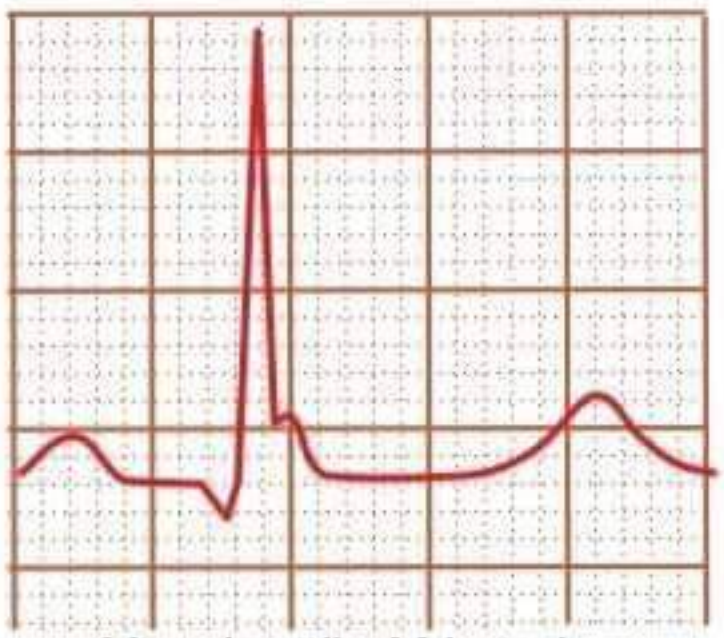
* These features are present in at least two conti-guous leads.

* It is generally a benign entity commonly seen in young men. The characteristics of ER may persist for many years. It is important to discern ER from ST segment elevation due to other causes such as ischemia.

Cardiac ischemia is a dynamic process with a changing ECG while the ECG of ER generally remains stable. A changing ECG favors ischemia.

Inferolateral Early Repolarization (ER)

Inferolateral ER is characterized by a deflection in the R wave descent (slurred pattern) or a positive deflection with a secondary "r" (notching pattern) in the terminal part of the QRS complex in at least two inferior leads (II, III, aVF), in two lateral leads (I, aVL, V4 to V6) or in both.

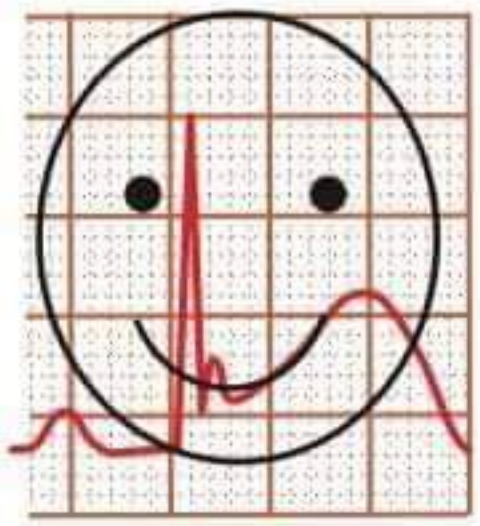


After Junttila MJ et al. European Heart Journal 2012; 33 : 2639

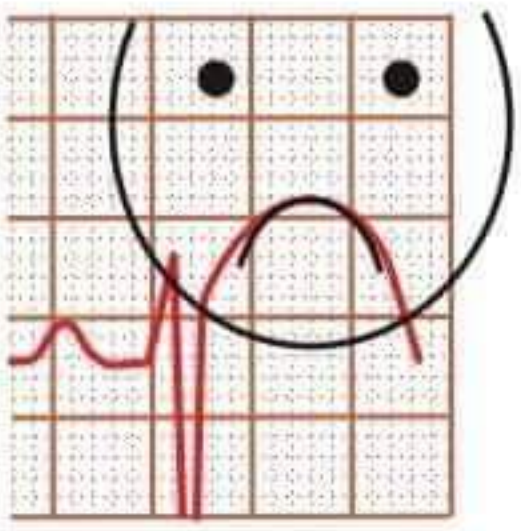
* A pattern of > 0.2 mV in two inferior (II, III, aVF) leads has been shown to impart a higher risk of malignant arrhythmia and sudden death.

* Early repolarization > 1 mV of horizontal or descending ST segment also carries a higher risk of sudden death.

* The management of asymptomatic patients with high risk ECG forms of early repolarization is unresolved.



A Smiley face with concave ST segment elevation is showing a happy face because a concave form may be benign as in early repolarization.



Convex ST elevation superimposed on a face as before, produces a frowny sad face because of the poor prognosis (because of acute myocardial infarction).

HOW TO READ AN ECG 8

STEP 17: Congestive heart failure

As congestive heart failure (CHF) is the outcome of many pathophysiologic disorders, the ECG may show a large variety of abnormalities. Occasionally the ECG is normal. However, CHF is unlikely if the ECG is entirely normal. In other words, a normal ECG does not rule out CHF.

The ECG abnormalities in CHF may be seen in many disorders. They consist of left ventricular hypertrophy, atrial and ventricular arrhythmias, atrioventricular and intraventricular conduction abnormalities, evidence of myocardial ischemia and infarction, right ventricular hypertrophy and atrial abnormalities.

No specific ECG feature is indicative of heart failure

Atrial fibrillation is present in 25% of patients with cardiomyopathy, especially elderly patients with severe heart failure. The prognosis is worse for patients with atrial fibrillation, ventricular tachycardia, or left bundle branch block. The presence of left bundle branch block with right axis deviation almost always indicates the presence of cardiomyopathy. Heart failure patients with implanted cardiac devices may show a paced rhythm with no diagnostic features of left ventricular function.

A prominent negative component of the P wave in lead V1 reflects elevated left ventricular end-diastolic pressure. The negativity may subside with the relatively early improvement of heart failure.

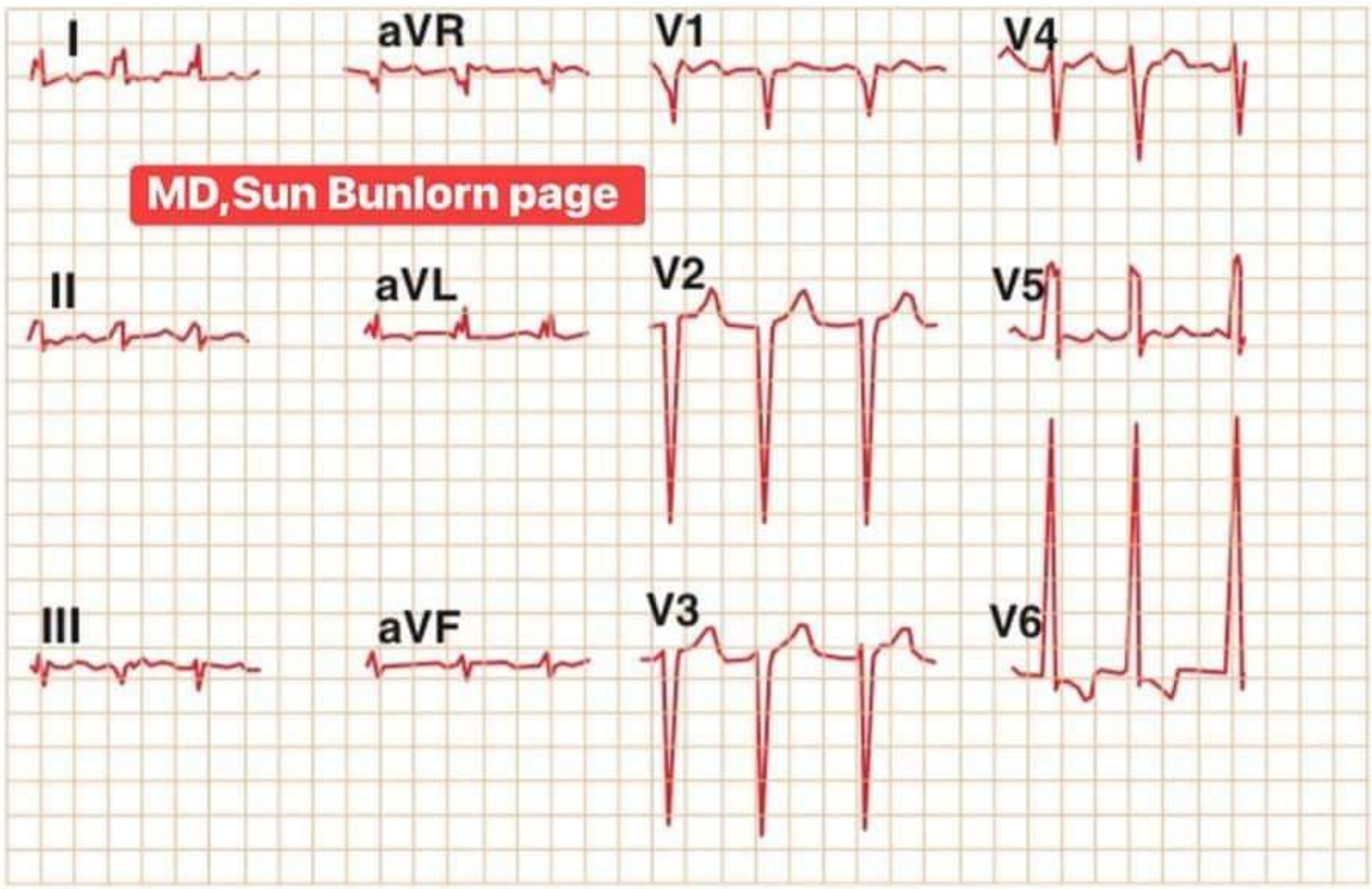
In CHF, peripheral edema may be associated with a decrease in amplitude (voltage) and duration of the QRS complex and the QT interval. These changes may hide important underlying abnormalities such as bundle branch block. The QRS and QT interval return to their baseline values when peripheral edema has subsided. The QRS abnormalities correlate with weight gain (peripheral edema). The mechanism of the attenuation of the ECG amplitude with peripheral edema is based on an increase in the electrical conductivity (i.e. decrease of resistivity) resulting in decrease of ECG voltage as per Ohm's law. Thus, QRS and even P wave changes (in V1) can be used in the follow-up of heart failure therapy.

During congestive heart failure with peripheral edema there is shortening of the QRS and QT interval.

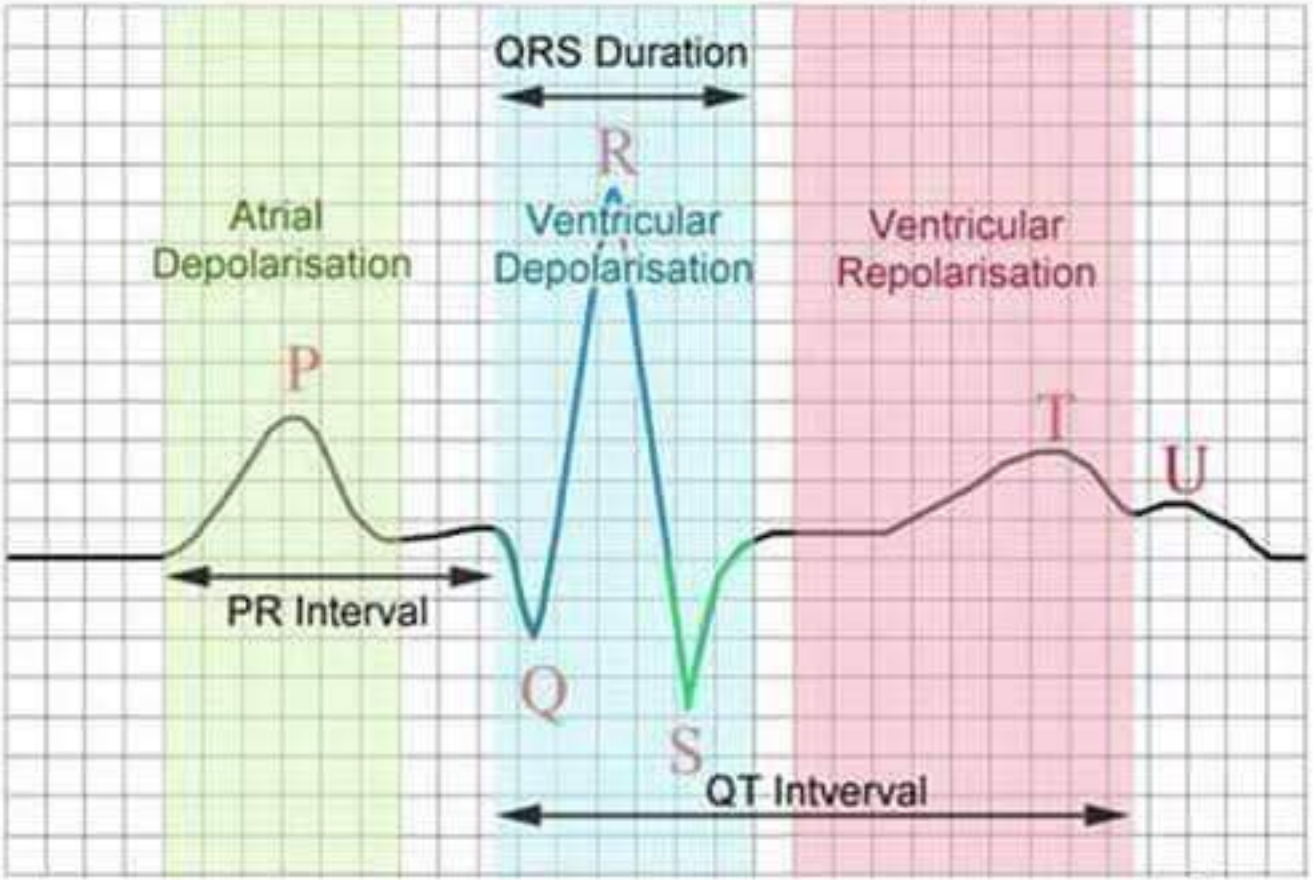
The ECG triad suggestive of CHF is characterized by low voltage in the limb leads, and high voltage in the precordial leads, and an R/S ratio < 1 in lead V4. There is a modest sensitivity and good specificity. The absence of the ECG triad does not exclude heart failure !

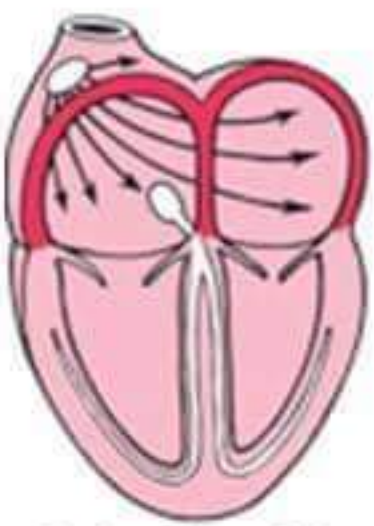
ECG triad of congestive heart fallure

* Relatively low QRS voltage in all six limb leads (≤ 0.8 mV)
* High QRS voltage in precordial leads (S in V1 or S in V2 and R in V5 or R in V6 > 3.5 mV)
* Poor R wave progression with R/S ratio < 1 in lead V4

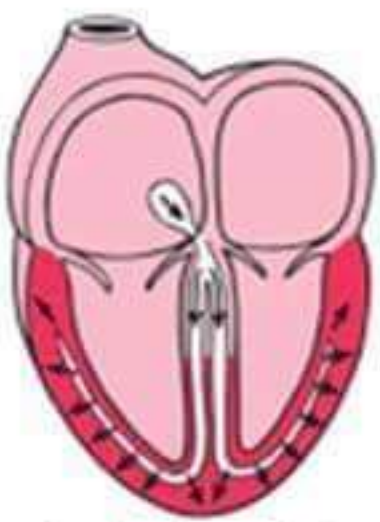


ECG showing atrial fibrillation and the typical features of the congestive heart failure triad.

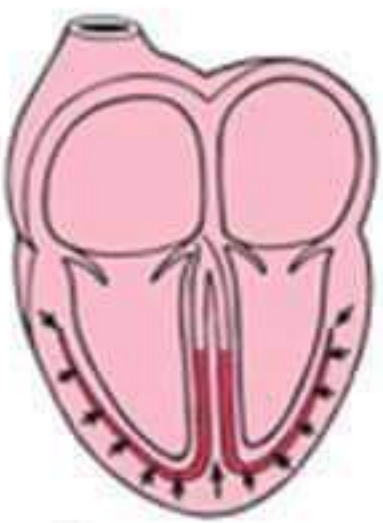




Activation of the atria

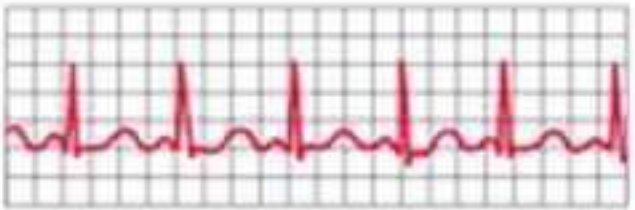


Activation of the ventricles

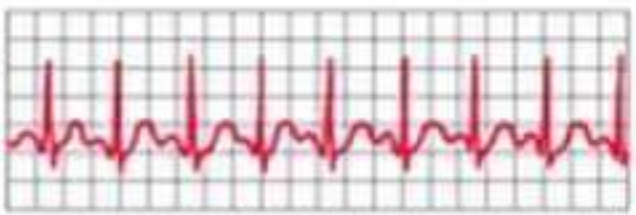


Recovery wave

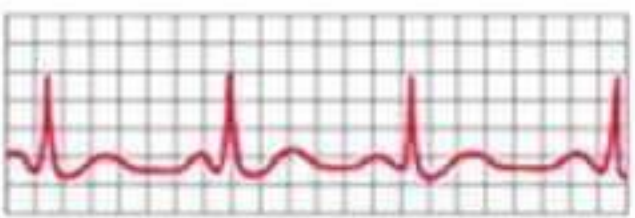
Normal Heartbeat



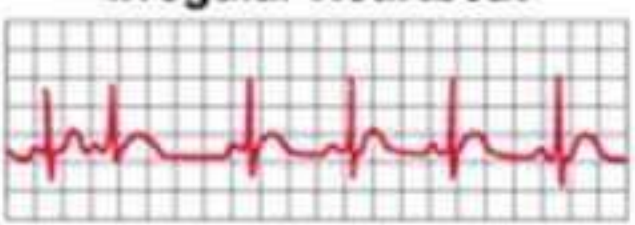
Fast Heartbeat

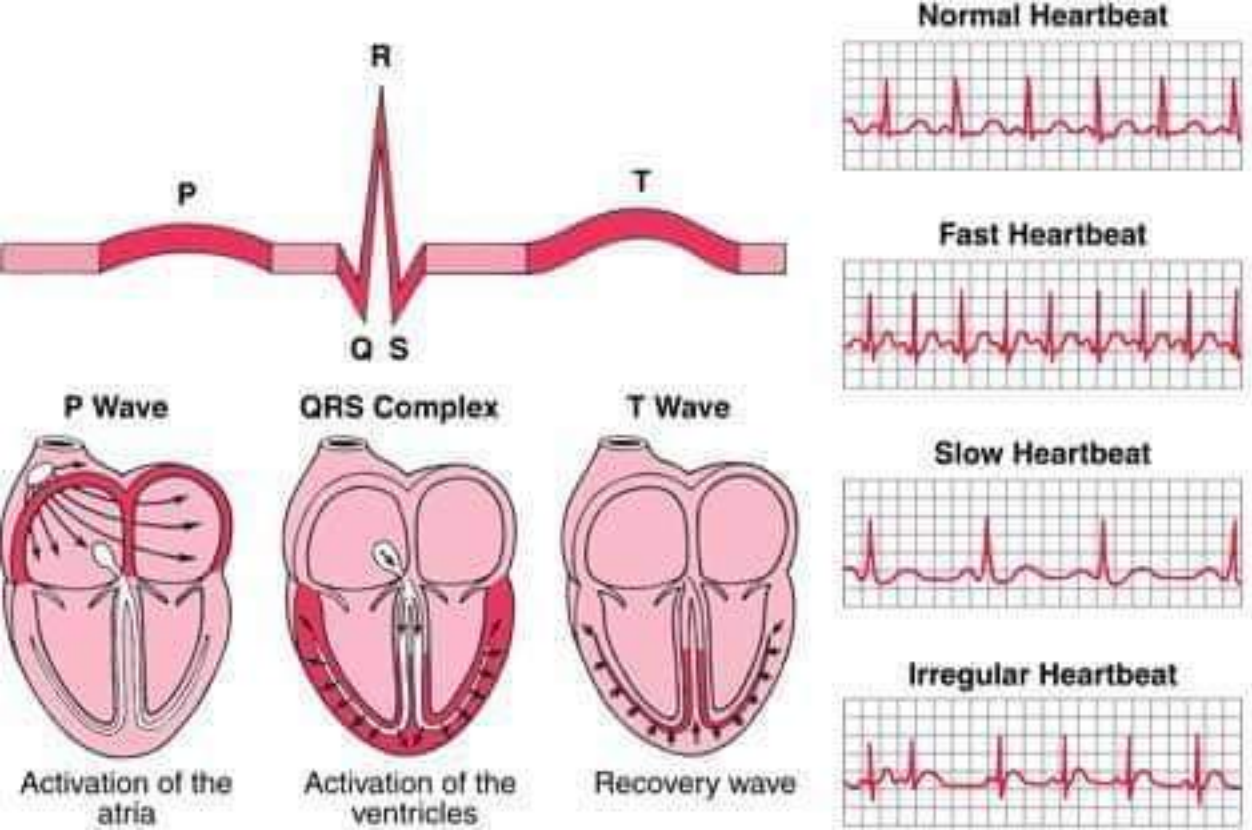


Slow Heartbeat



Irregular Heartbeat




R
P
T
Q S
P Wave
QRS Complex
T Wave
Activation of the atria
Activation of the ventricles
Recovery wave
Normal Heartbeat
Fast Heartbeat
Slow Heartbeat
Irregular Heartbeat


ECG
10:09
Hold your finger on
the crown.