ELECTROCARDIOGRAPHY

Institute of Pathophysiology

Faculty of Medicine in Pilsen, Charles University

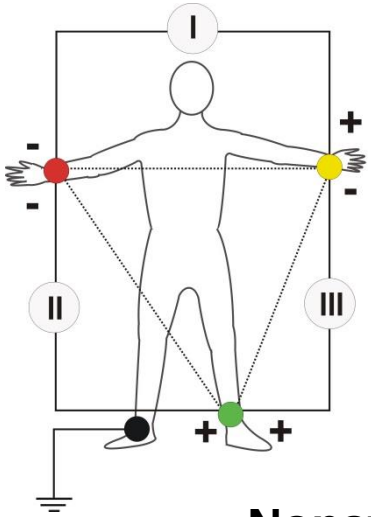
INTRODUCTION,
ECG RECORD DESCRIPTION

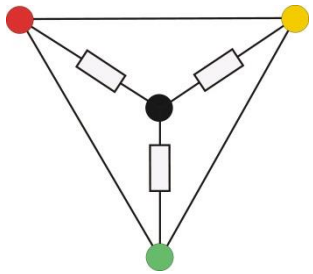
STANDARD ECG LEADS

3 bipolar extremity leads- I, II, III

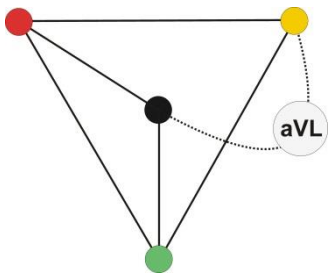
3 (pseudo)unipolar augmented extremity leads- aVR, aVL, aVF

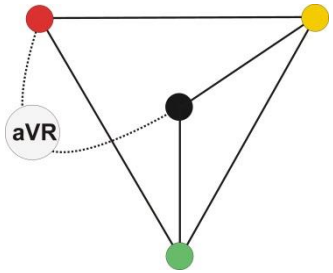
6 unipolar chest leads- V1 - V6

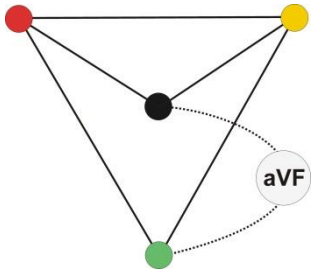




Wilson‘s central clamp







Goldberg‘s modification

Nonstandard ECG LEADS

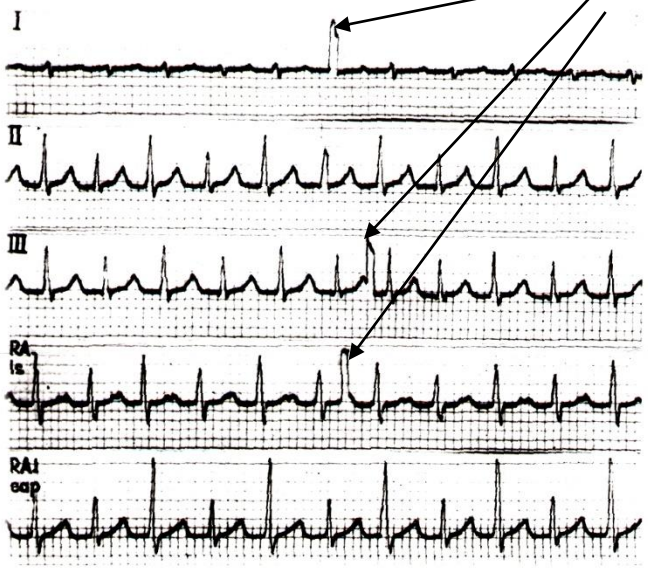
- right sided precordial leads (right ventricle, dextrocardia)
- chest leads V7... (posterior myocardial infarction)
- étage leads
- esophageal
- bronchial
- intracardial

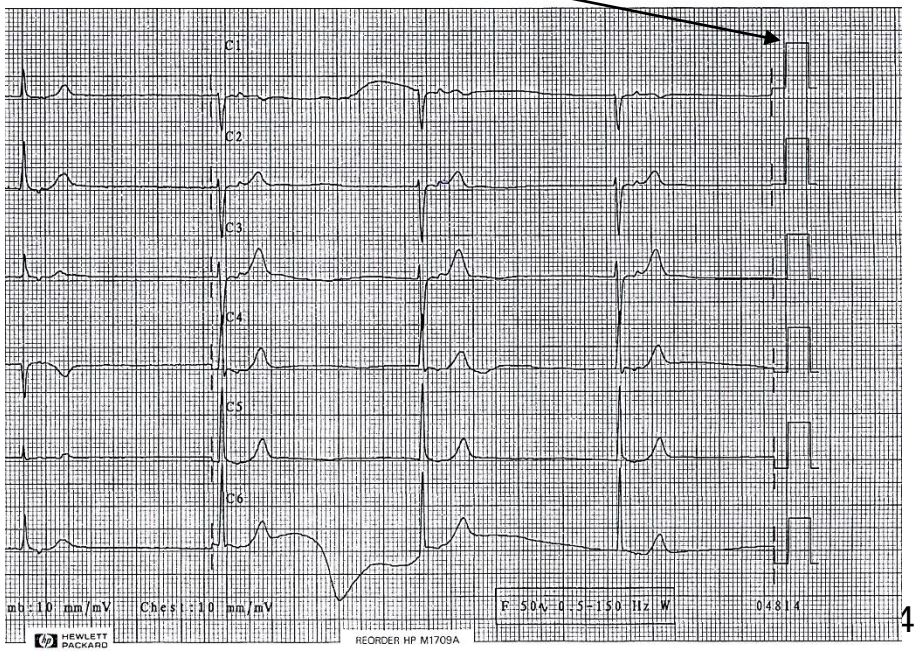
PC vectocardiography

REQUIREMENTS OF ECG EXAMINATION

- patients data
- date of examination
- correct inclusion of electrodes
- calibration (usually 0.1 mV/mm)
- speed of paper transmission (usually 25 mm/s)

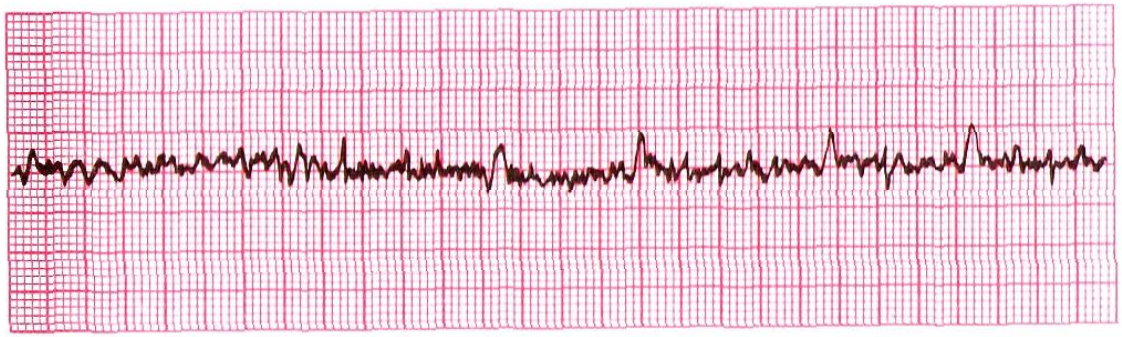
calibration (usually 10 mm = 1 mV)





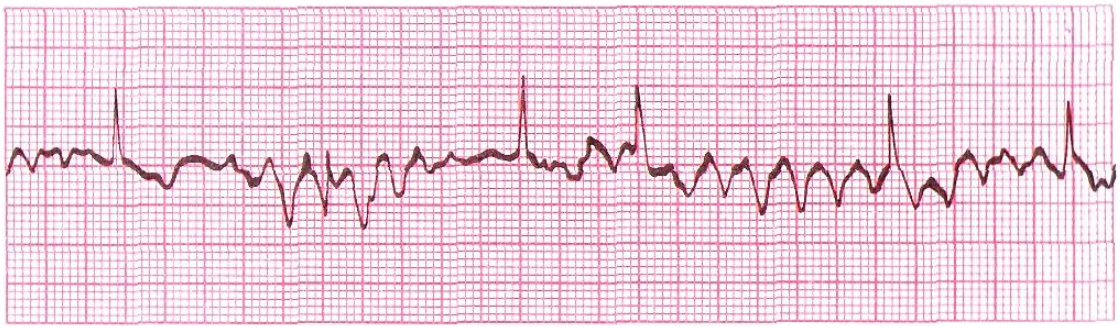
ARTEFACTS

Hypothermia



Muscular tremor – EMG overloads ECG

Parkinsonism



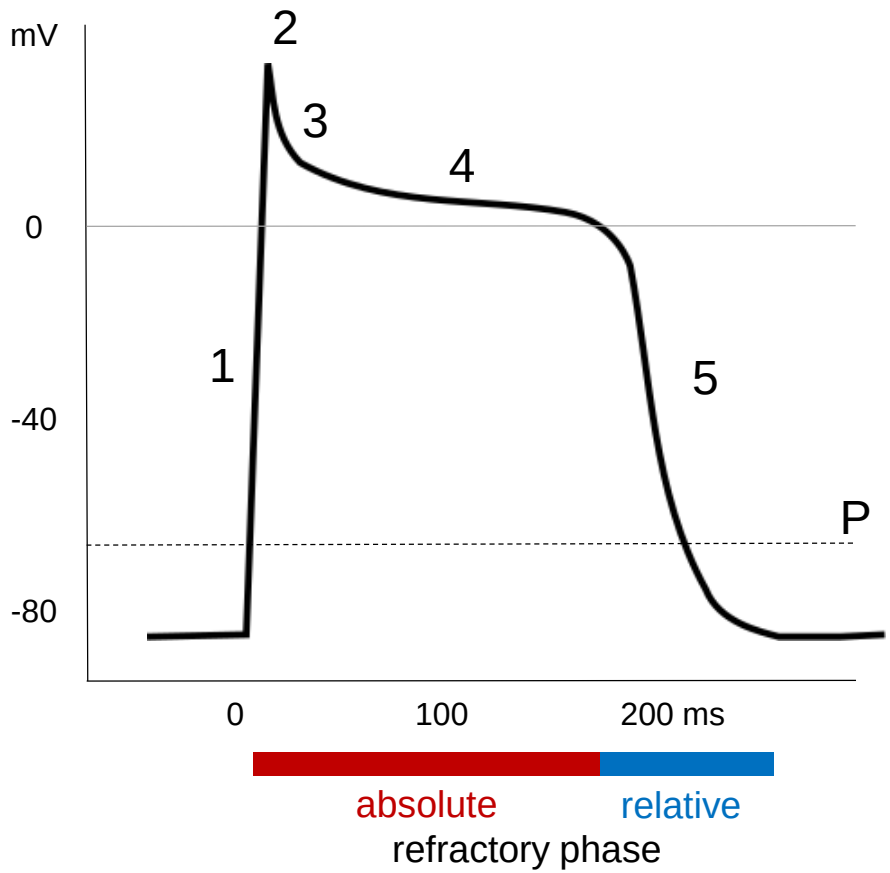
Muscular tremor and bad contact of electrodes (irregular appearance of QRS)

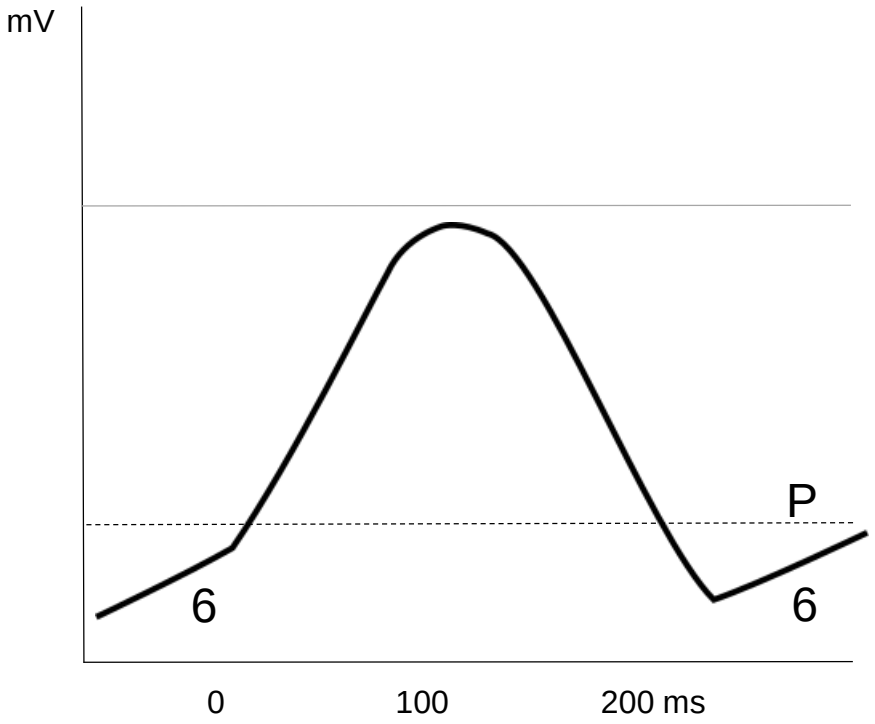
Myocardial action potential

1...depolarization
2...transpolarization
3...initial fast repolarization
4...plateau
5...fast repolarization
6...slow diastolic depolarization
P...action potential threshold

Working fiber
- able to maintain stable membrane potential

Conductive (automacy) system fiber
- slow diastolic depolarization
→ inability to maintain stable resting potential



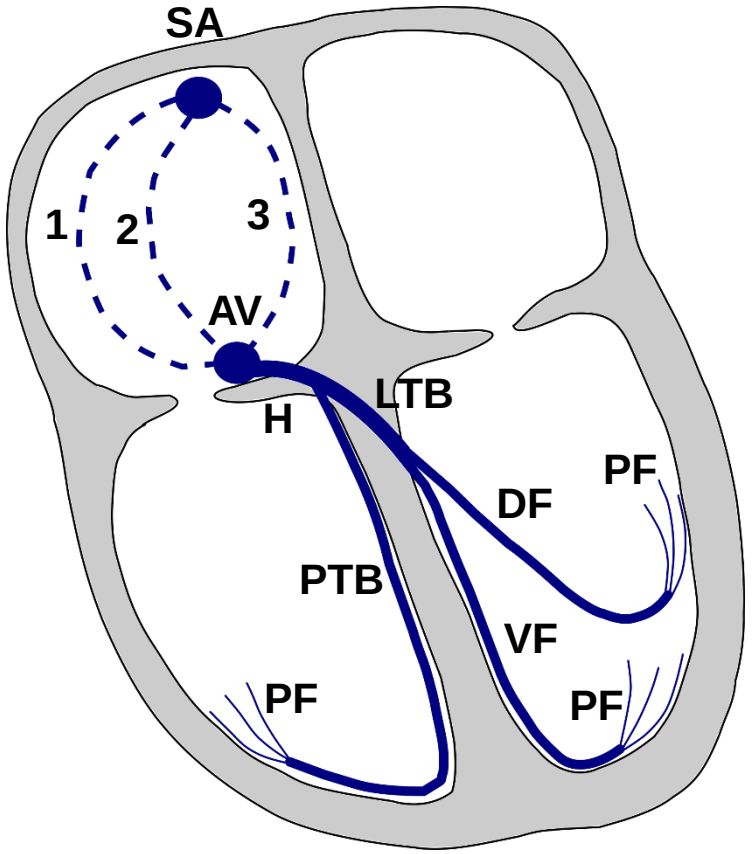


CELLS OF AUTOMACY

- part of the transfer system
- ability of spontaneous diastolic depolarization (action potential production)
- unable to maintain resting membrane potential (-50 up to -60 mV)
- threshold starting potential of the action potential - 45 mV

TRANSFER SYSTEM

- Sinoatrial node **(SA)**
- Preferential atrial pathways **(1, 2, 3)**
- Atrioventricular node **(AV)**
- His bundle **(H)**
- Tawara branch
  - right Tawara branch **(RTB)**
  - left Tawara branch **(LTB)**
    - dorsal fasciculus **(DF)**
    - ventral fasciculus **(VF)**
- Purkinje fibers **(PF)**

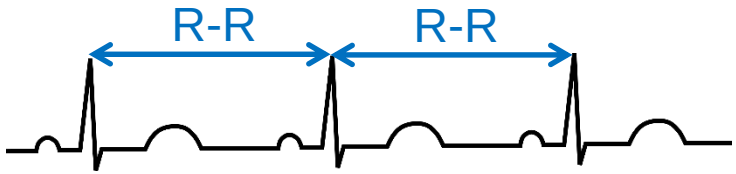


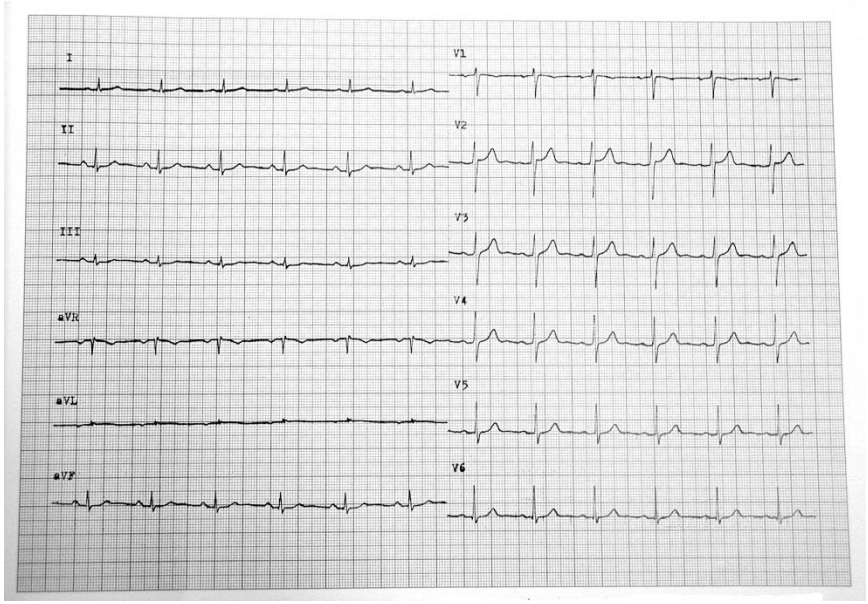
DESCRIPTION OF ECG CURVE

1. ACTION
2. FREQUENCY
3. RHYTHM
4. DECLINATION OF HEART ELECTRICAL AXIS
5. ANALYZIS OF INDIVIDUAL WAVES, OSCILLATIONS AND INTERVALS

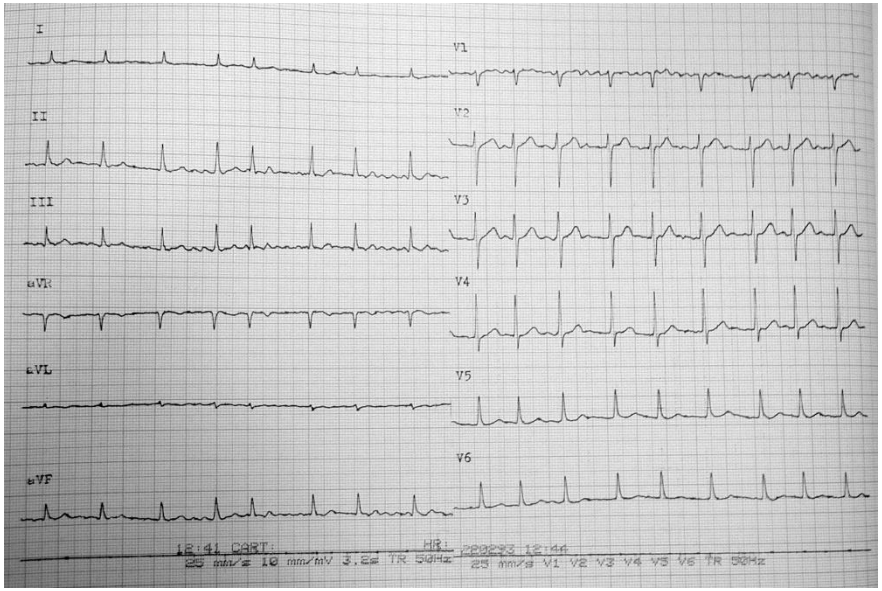
1. ACTION

- regular R – R = R – R
- irregular R – R ≠ R – R





regular action (physiological ECG)



irregular action (atrial fibrillation)

2. FREQENCY

- normal at rest 60 – 90 BPM
- tachycardia > 90 BPM
- bradycardia < 60 BPM

Calculation:

a) raster - small square … 0.04 s
- large square …0.20 s (shift of the paper – 25 mm/s)

example: R–R distance = 4 large sq. → 4 x 0.2=0.8; 60 : 0.8=75

Can be used only with regular action!

b) number of QRS complexes in the section, which is multiple of 25 mm (i.e. 1 s)

75 mm … 3 s (3 s=1/20 min)
150 mm … 6 (6 s=1/10 min)

example 1: in 75 mm ... 4 QRS 4 x 20 = 80 BPM

example 2: in 150 mm … 8 QRS 8 x 10 = 80 BPM

Can be used only with regular action!

c) 60 x number of QRS complexes in the whole register/length of the whole register (in s) = pulse frequency (BPM)

Can be used also with irregular action.

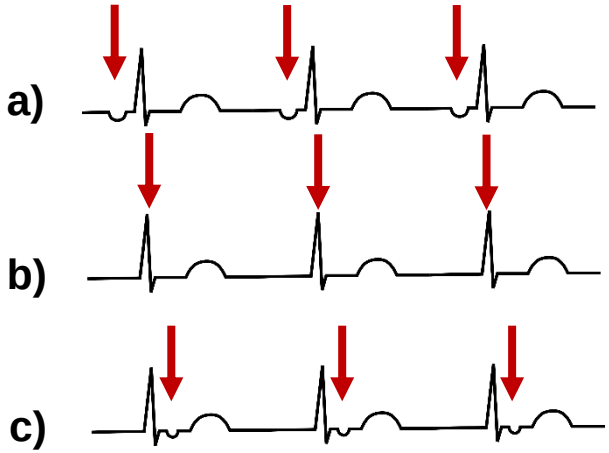
3. RHYTHM

- given by the centre of automacy
- primary pacemaker - SA node
- compensatory centres of automacy - AV junction, conductive system of the ventricles
- ectopic foci

1) **Sinusal rhythm** - P present, positive, in front of slim QRS, frequency at rest 60-90 BPM

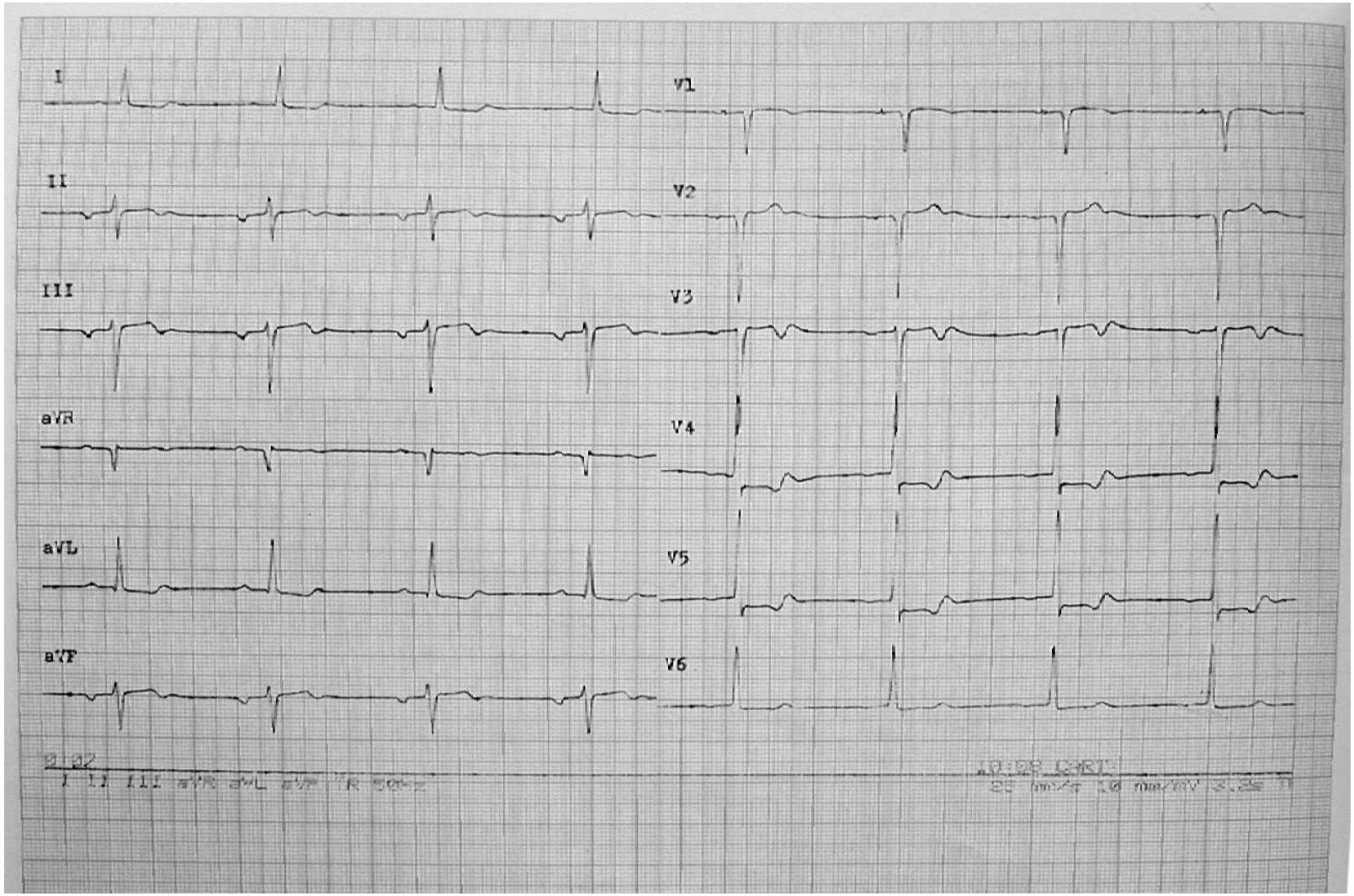
2) **Nodal rhythm**

a) upper - P negative, in front of QRS
b) middle - P hidden in QRS
c) lower - P negative, after QRS
A) passive - frequency lower than in SA (< 60)
B) active – frequency higher than in SA (> 60)

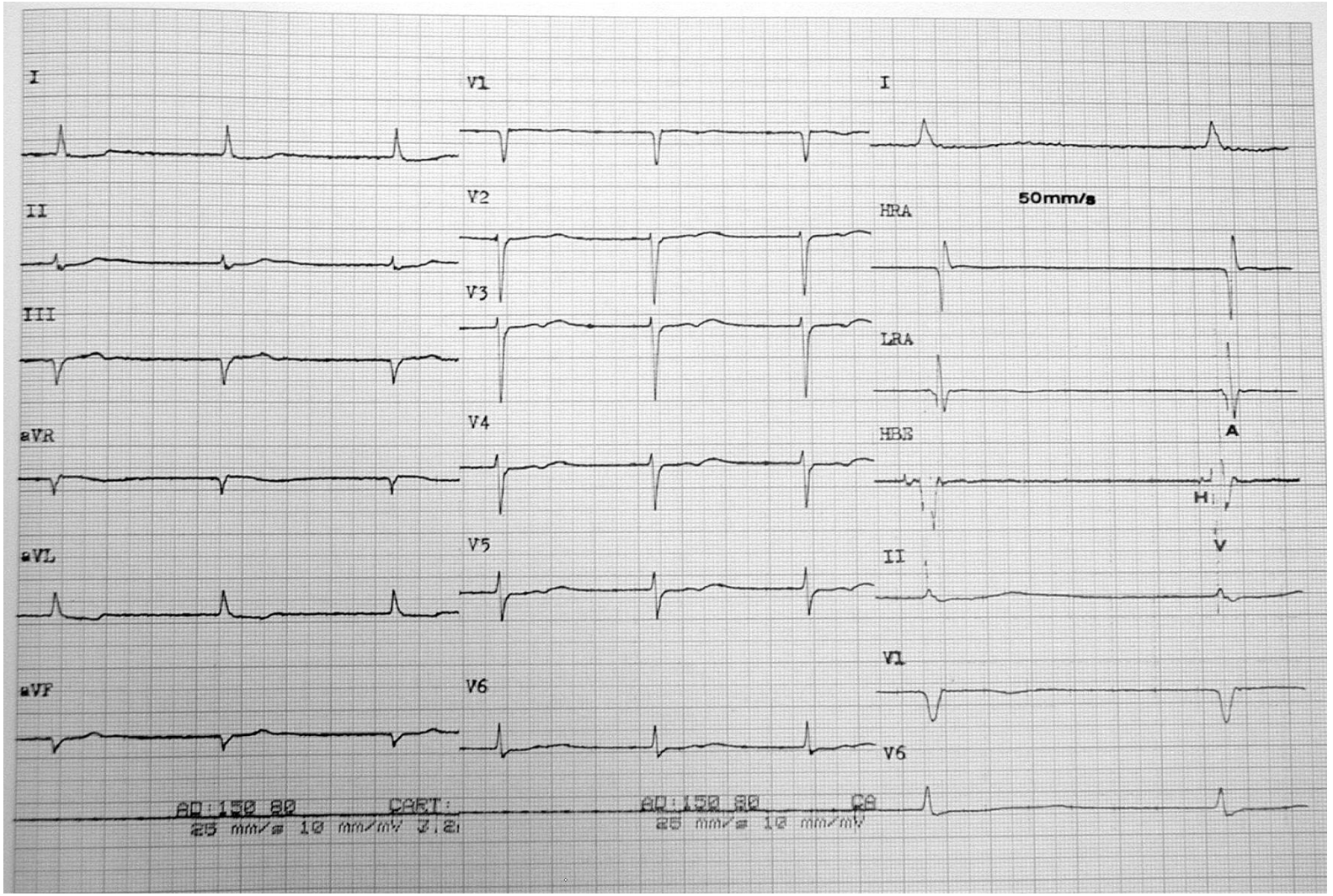


3) **Idioventricular rhythm**

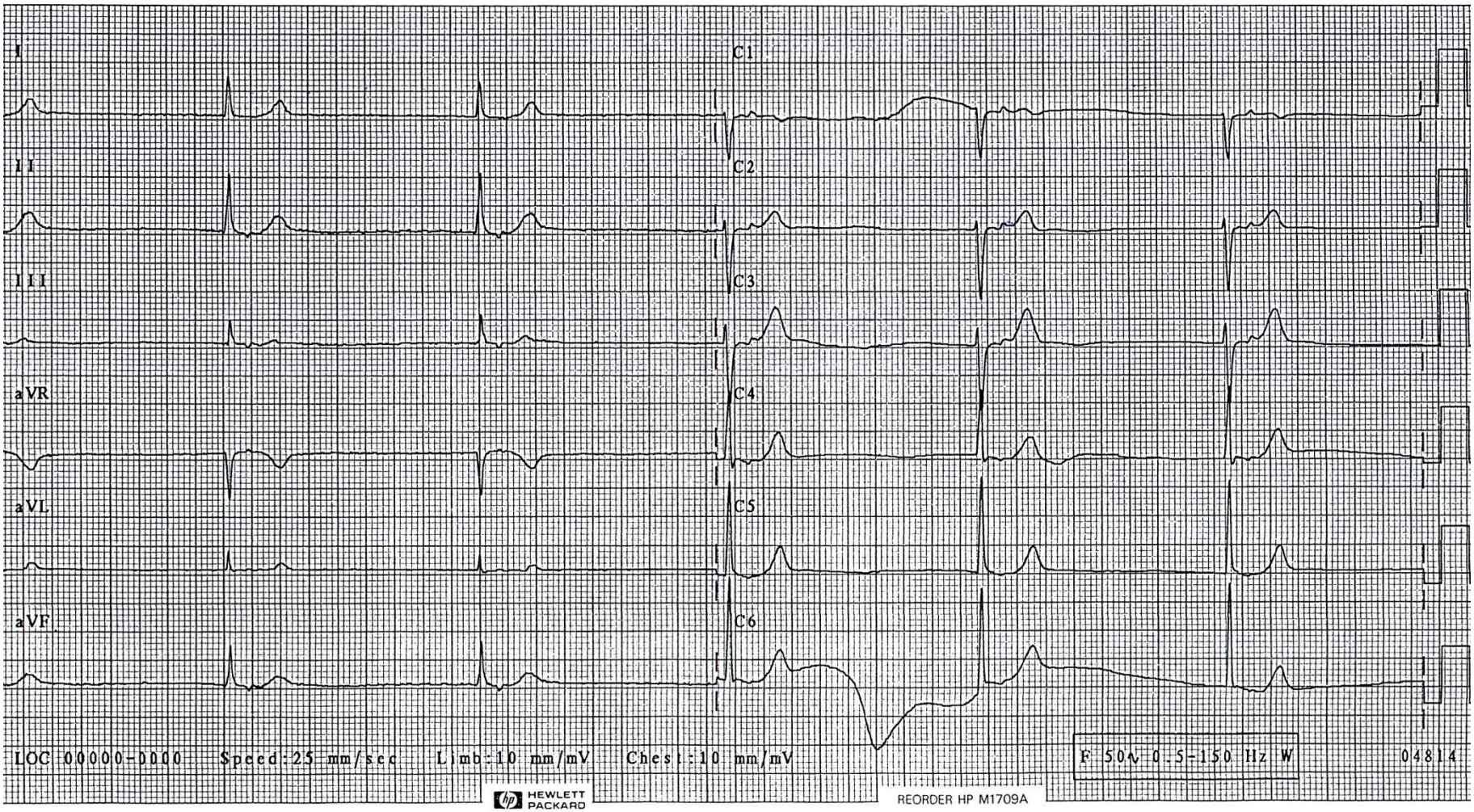
- P absent (P have no connection with QRS, see AV block IIIrd deg.)
- QRS aberrant
- frequency around 40 BPM



Upper nodal rhythm



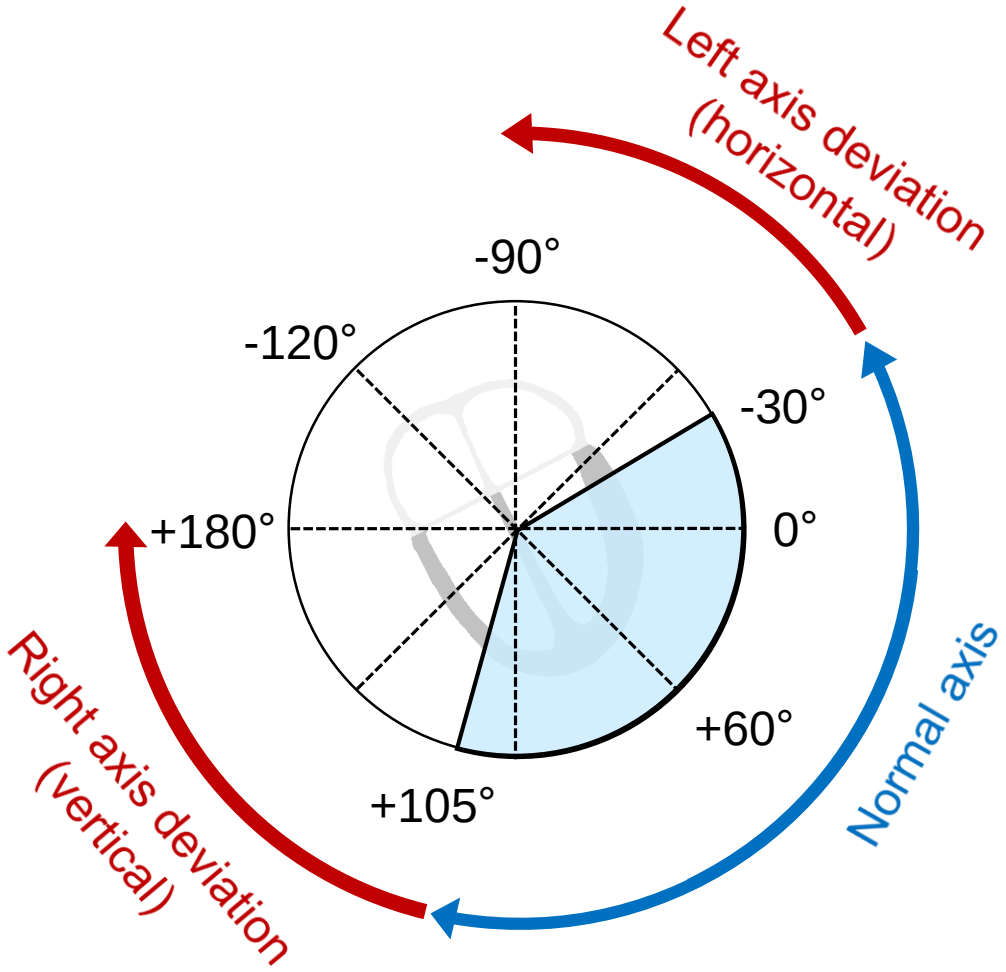
Middle nodal rhythm



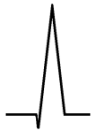
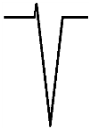
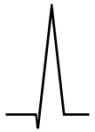
Lower nodal rhythm

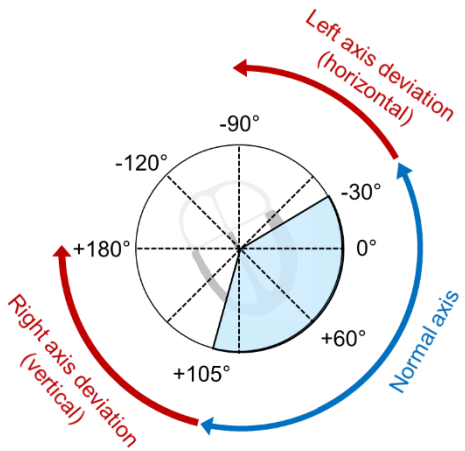
HEART AXIS

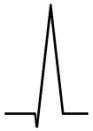
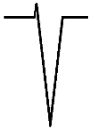
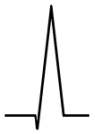
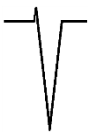
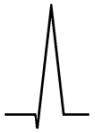
Normal range: from -30° to +105°



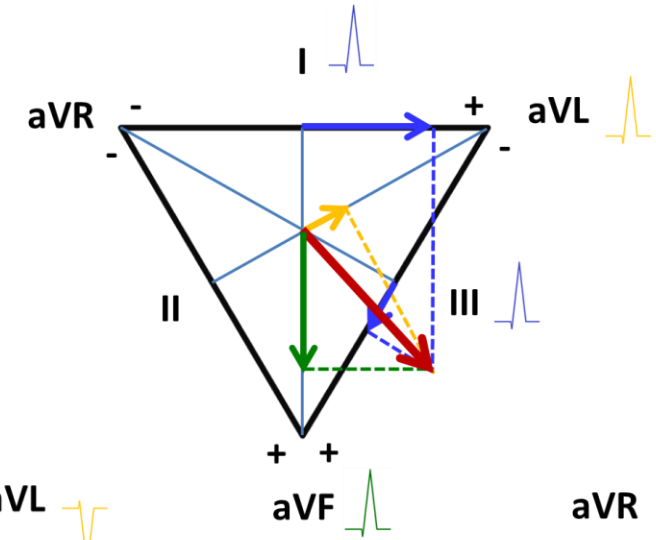
HEART AXIS DEVIATION

Deviation	I	III
Left		
Normal		
Right		

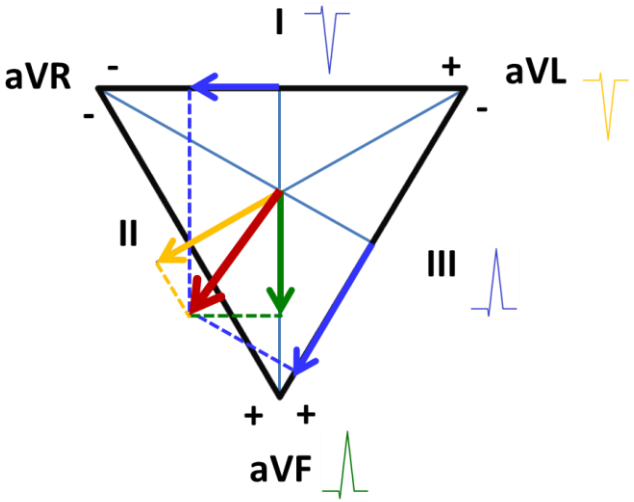


Deviation	aVL	aVF
Horizontal		
Semi-horizontal		
Intermediary		
Semi-vertical		
Vertical		

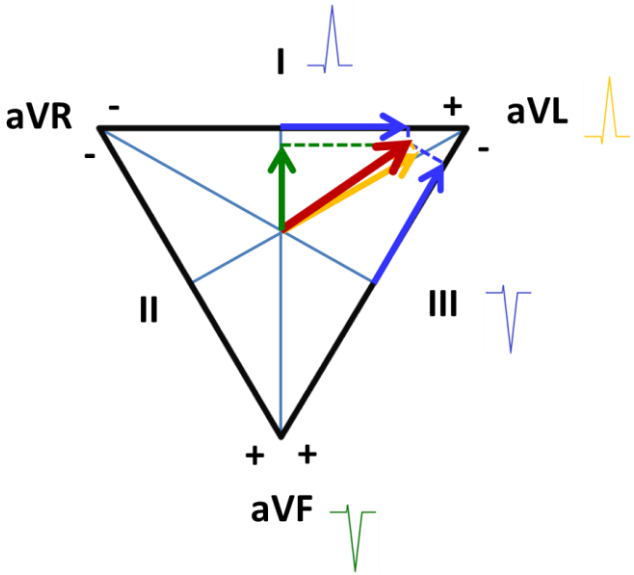
HEART AXIS DEVIATION



Normal axis
(intermediary)

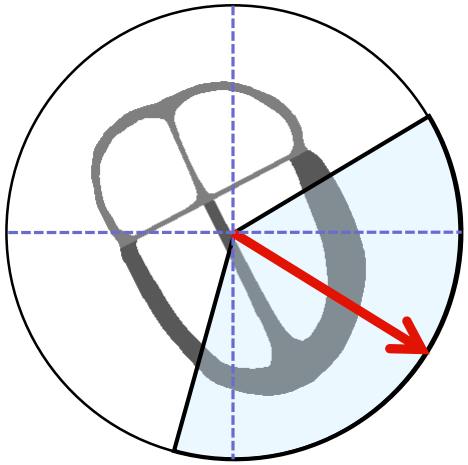


Right axis deviation
(vertical)

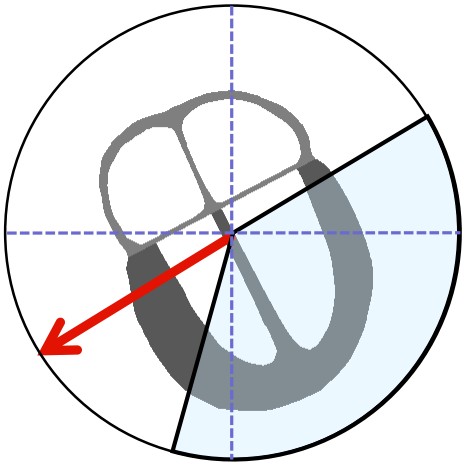


Left axis deviation
(horizontal)

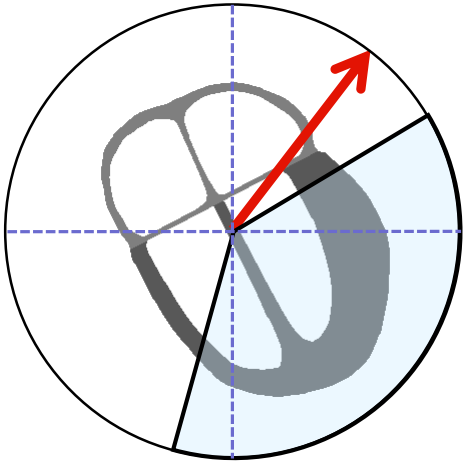
HEART AXIS DEVIATION - hypertrophy



Normal axis
(intermediary)

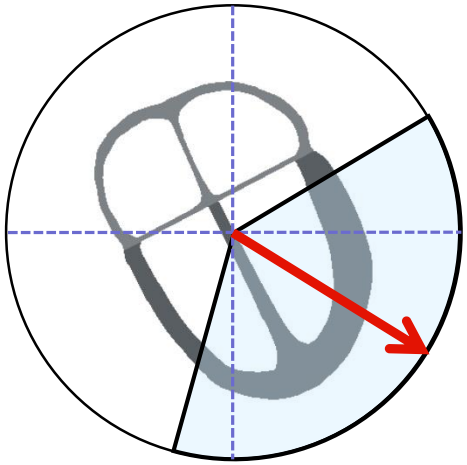


Right axis deviation
- right ventricle hypertrophy

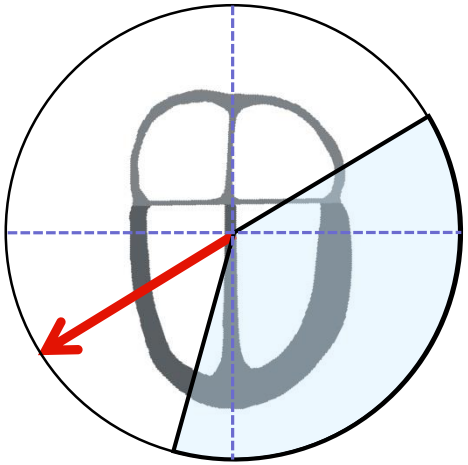


Left axis deviation
- Left ventricle hypertrophy

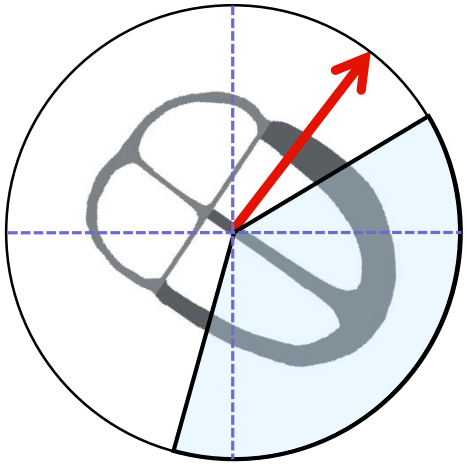
HEART AXIS DEVIATION – heart position



Normal axis
(intermediary)



Right axis deviation
- vertical heart position

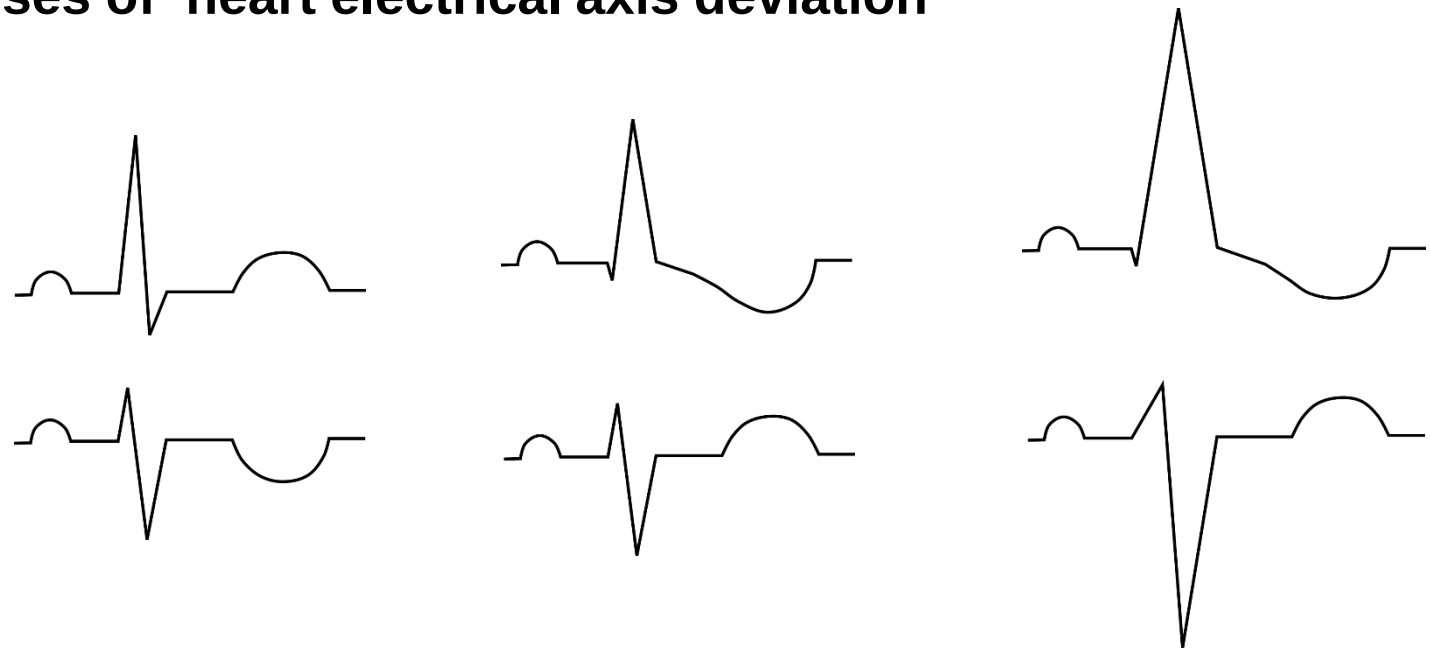


Left axis deviation
- horizontal heart position

Causes of heart electrical axis deviation

I (aVL)

III (aVF)



Heart position (horizontal)**:**
- left axis deviation
- concordant T

Heart (left ventricle) **overload:**
- left axis deviation
- discordant T
- descendant ST depression

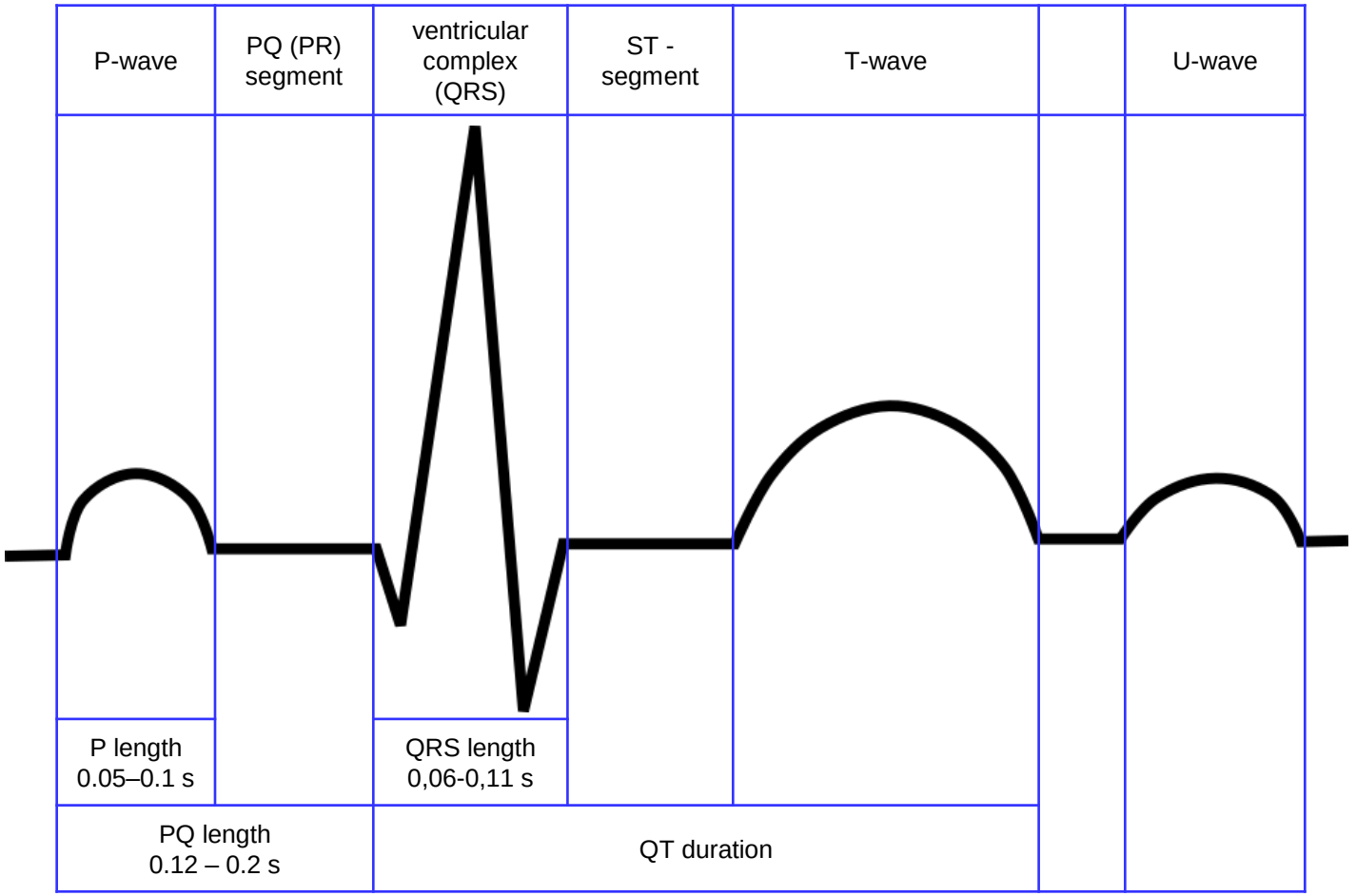
Heart (left ventricle) **hypertrophy:**
- left axis deviation
- high voltage of the QRS complex
- slightly wider QRS complex
- signs of overload (descendant ST depression, discordant T)

5. ANALYSIS OF WAVES

P - atrial depolarization
PQ - transmission to the ventricles
QRS – ventricular depolarization
ST + T – ventricular repolarization
U – uncertain origin

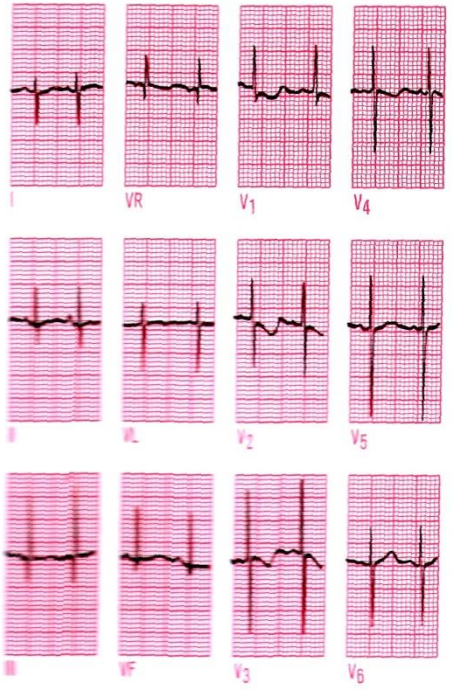
P height: < 0.25 mV

QTc: interval QT length corrected for frequency



ECG AND AGE

- newborn: - frequency 160/min, irregular action
  - right axis declination
  - negative T in V1 - 4
- 2 years: - frequency falls (150/min)
  - normal axis declination
  - negative T in V1 – 2
- 10 years: adult ECG



Physiological ECG record in a newborn

EKG AND SPORTSMAN

- current sinus arrhythmia, bradycardia
- wandering pacemaker
- AV block I. degree, II. degree Mobitz 1
- high P, R, deep S
- ST elevation
- T changes – high, inversion in lat. leads, biphasic
- U wave

PATOLOGIC STATES MANIPHESTED BY ECG CHANGES

1. **Arrhythmias**
   - A) Impulse origin disturbances:
     1. Disturbances of the sinus rhythm
     2. Supraventricular arrhythmias
     3. Ventricular arrhythmias
   - B) Impulse transmission disturbances:
     1. Sinoatrial (SA) block
     2. Atrioventricular (AV) block
     3. Bundle branch blocks
     4. Arborization bock
     5. Preexcitation syndrome (WPW, CLC)
2. **Ischemic heart disease (IHD)**
   - A) Myocardial infarction
   - B) Angina pectoris
   - B) Arrhythmic form of IHD
3. **Heart inflammations**
   - A) Myocarditis
   - B) Pericarditis
4. **Hypertrophy, overload (acute, chronic)**
5. **Other diseases and pathologic states of the heart**
6. **Ion disbalances**
7. **Effects of medicaments**
8. **Systemic diseases**

ARRHYTHMIAS

Classification of arrhythmias

Disturbances of impulse transmission

- Blocks
- Preexcitation

Disturbances of impulse origin

= changes of frequency or regularity of impulse generation in the primary pacemaker, generation of impulses in one of the alternative centers of automacy, generation of impulses in an ectopic focus in the working myocardium

According to the place of origin
According to frequency

ARRHYTHMIAS – classification according to frequency

Localization of the disorder (impulse generation)	Bradyarrhythmias	Tachyarrhythmias
SA node	Sinus bradycardia Sinus arrest	Sinus tachycardia
Atria		Atrial extrasystole Supraventricular tachycardia Atrial flutter Atrial fibrillation
AV node	AV block degree II and III	Junctional extrasystole Active junction rhythm
Ventricles		Ventricular extrasystole Ventricular tachycardia Ventricular flutter Ventricular fibrillation

ARRHYTHMIAS: DISTURBANCES OF IMPULSE ORIGIN

DISTURBANCES OF IMPULSE ORIGIN

1. Disturbances of sinus rhythm

- sinus tachycardia
- sinus bradycardia
- respiratory arrhythmia
- non-respiratory sinus arrhythmia
- sinus arrest
- sick sinus syndrome

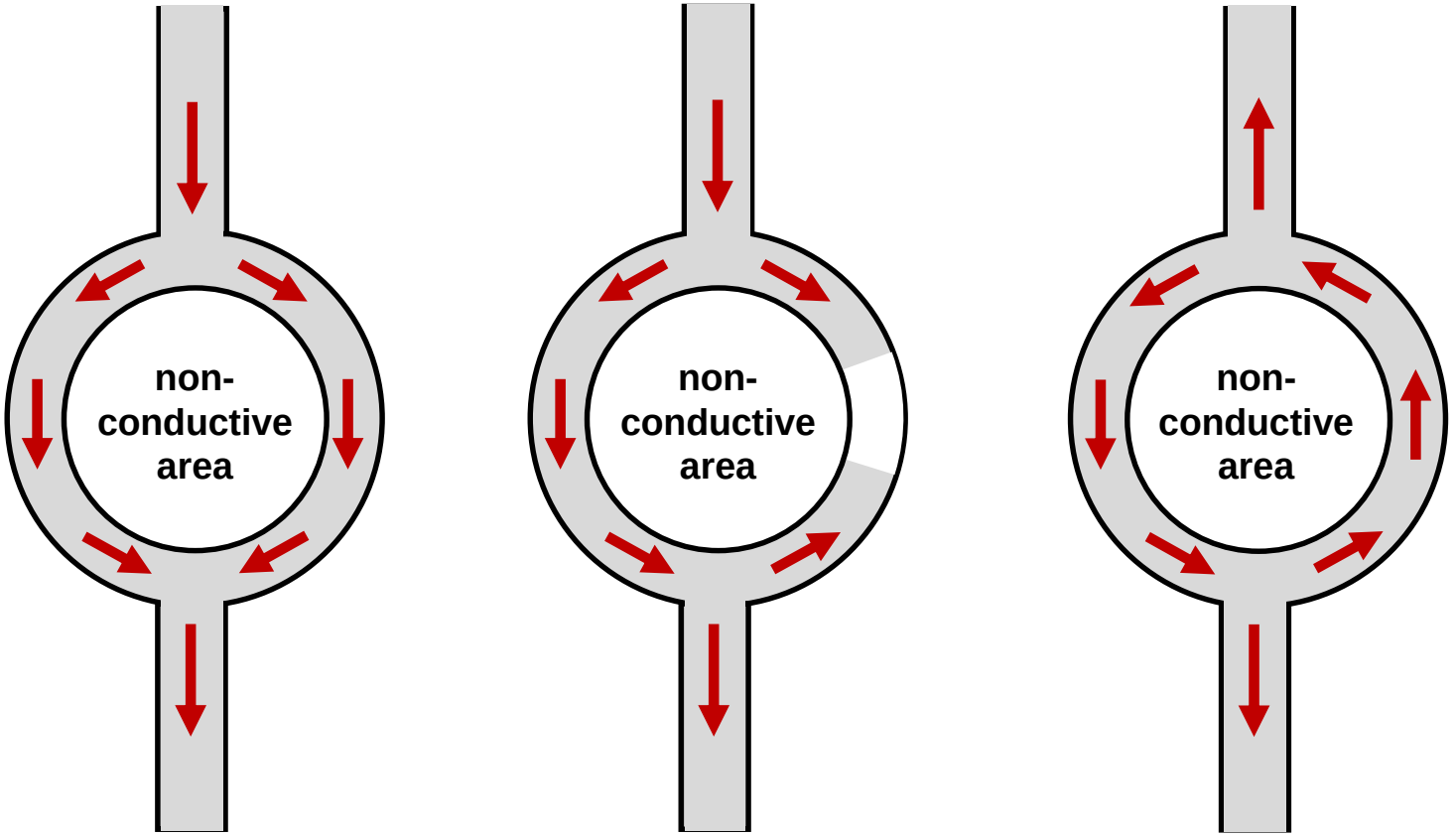
2. Supraventricular arrhythmias

- atrial extrasystoles
- supraventricular paroxysmal tachycardia
- atrial flutter
- atrial fibrillation
- wandering pacemaker

3. Ventricular arrhythmias

- ventricular extrasystoles
- ventricular paroxysmal tachycardia
- ventricular flutter
- ventricular fibrillation
- ventricular arrest

Reentry mechanism



normal conduction

delayed (blocked) conduction

reentry

Non-conductive area: e.g. fibrotized tissue
- Microskopic dimensions → microreentry
- Macroskopic dimensions → macroreentry

EXTRASYSTOLSES = premature beats

Origin: - mostly from an ectopic focus

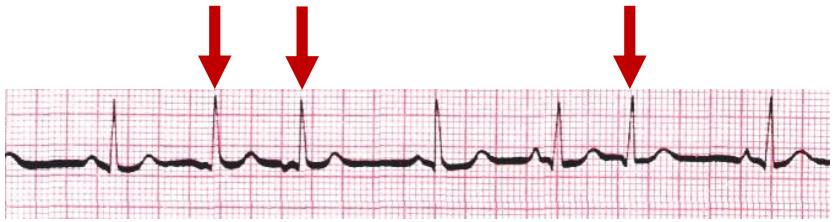
Appearance:
- solitary
- piled
- bound

Supraventricular ES:
incomplete compensatory pause (< 2 x R-R), usually normal QRS, P wave

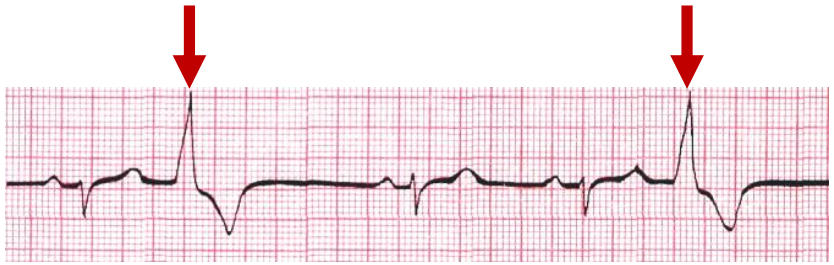
Ventricular ES:
complete compensatory pause (= 2 x R-R), aberrant QRS, no P wave

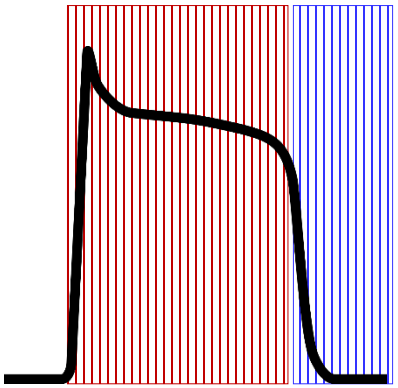
Interpoled ES:
without compensatory pause

supraventricular ES



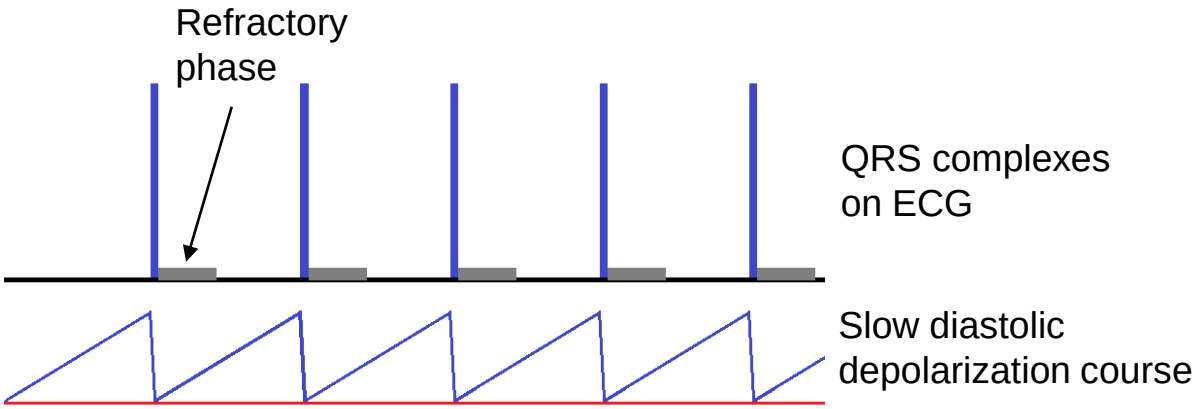
ventricular ES



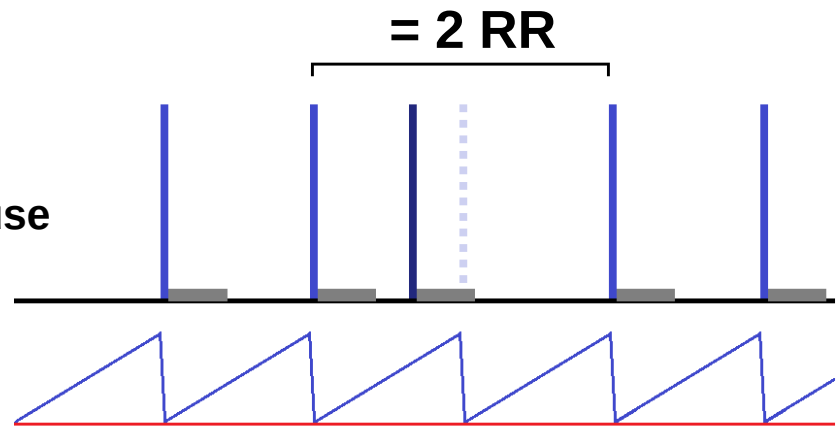


absolute / relative
refractory phase

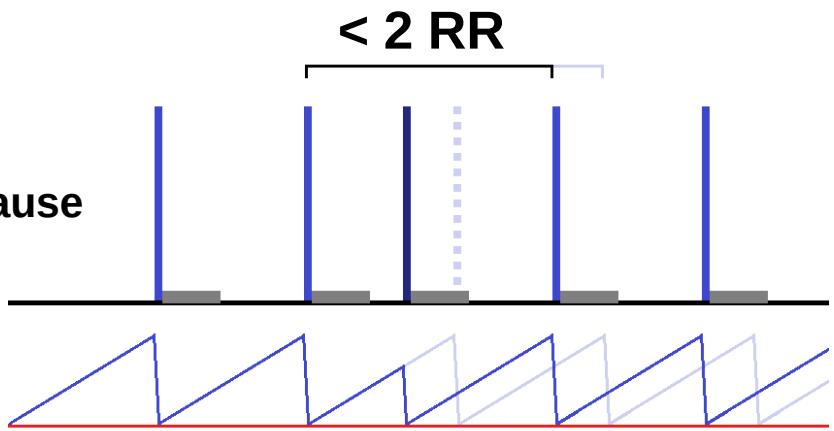
Normal state



Ventricular extrasystole
- complete compensatory pause

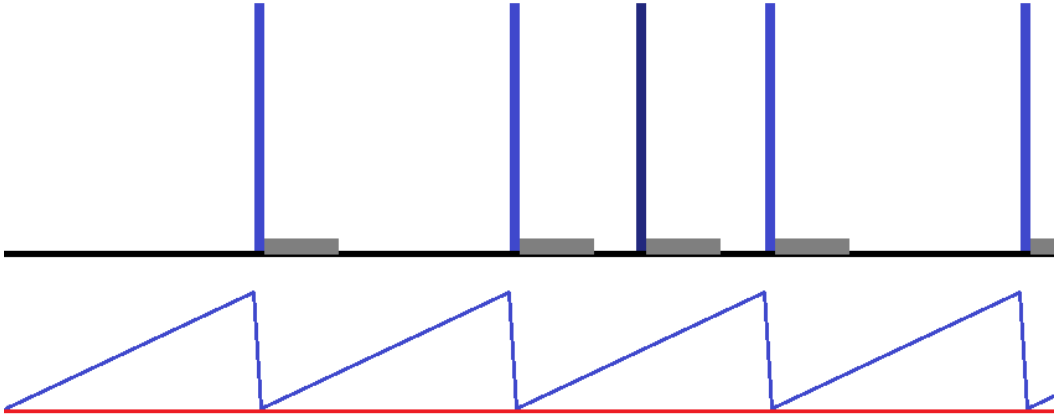


Supraventricular extrasystole
- Incomplete compensatory pause

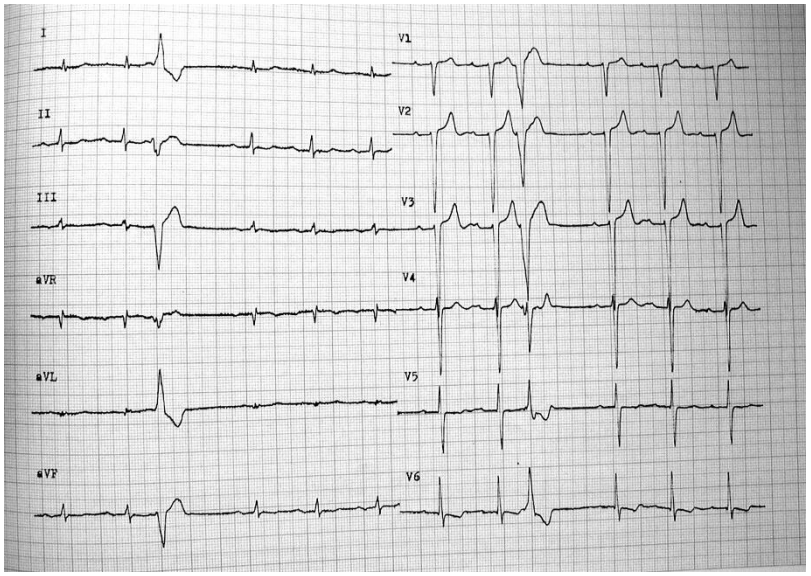


Interpolated extrasystole

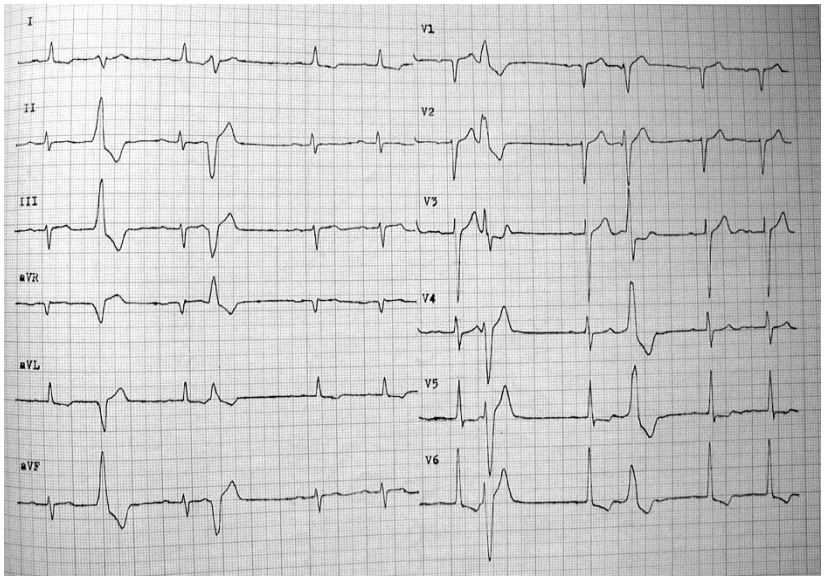
- no compensatory pause
- always ventricular
- can appear at bradycardia which enables termination of the refractory pause before the onset of the next QRS of the sinus origin



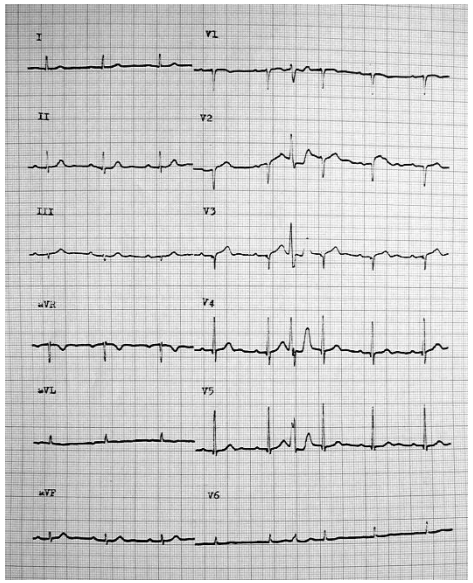
VENTRICULAR EXTRASYSTOLS



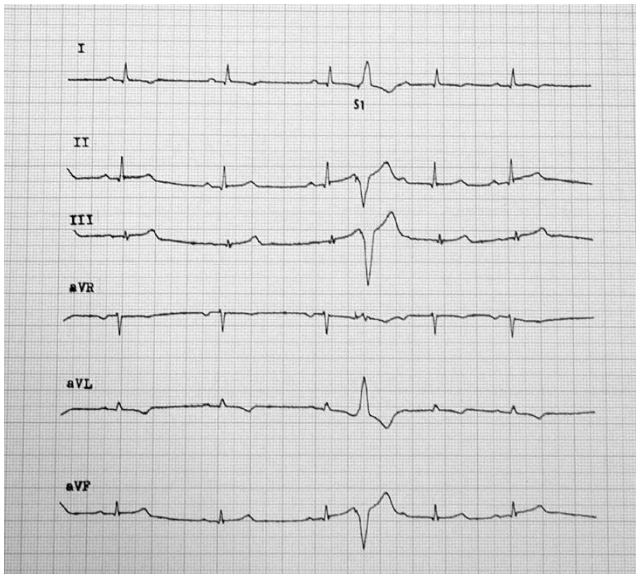
ventricular extrasystole



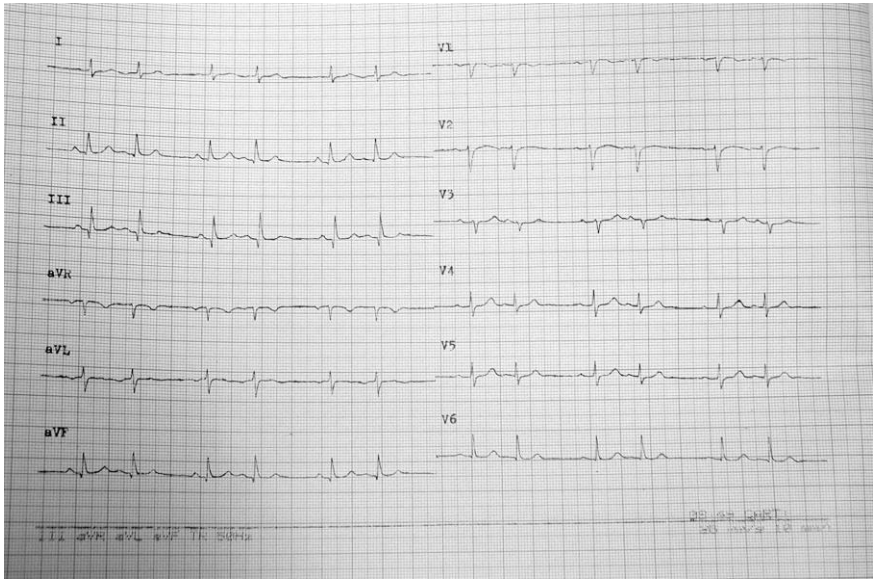
polytopic (polymorphic) ventricular extrasystols



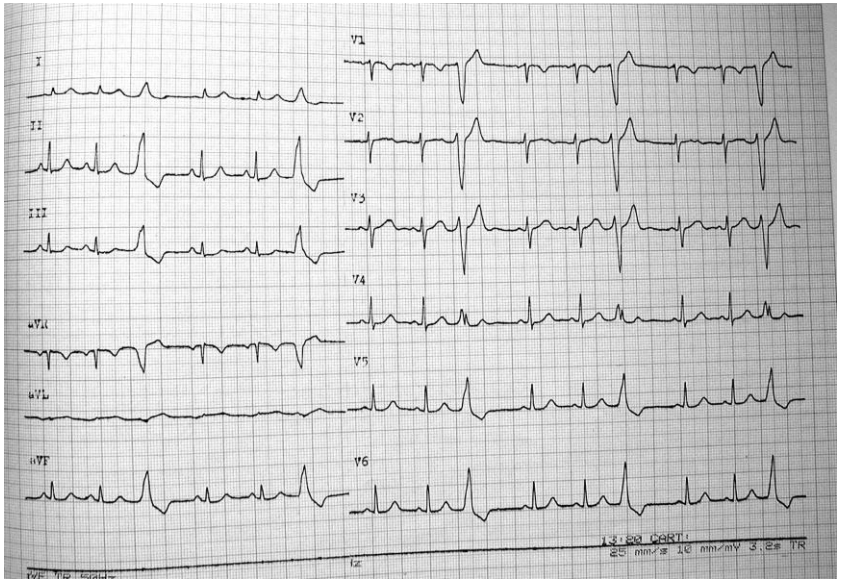
interpoled extrasystole



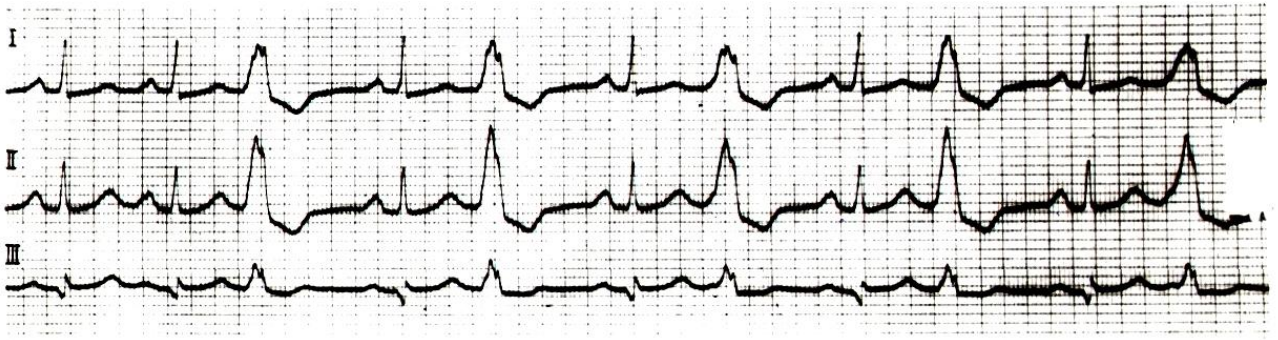
Bound extrasystols



supraventricular bigeminia



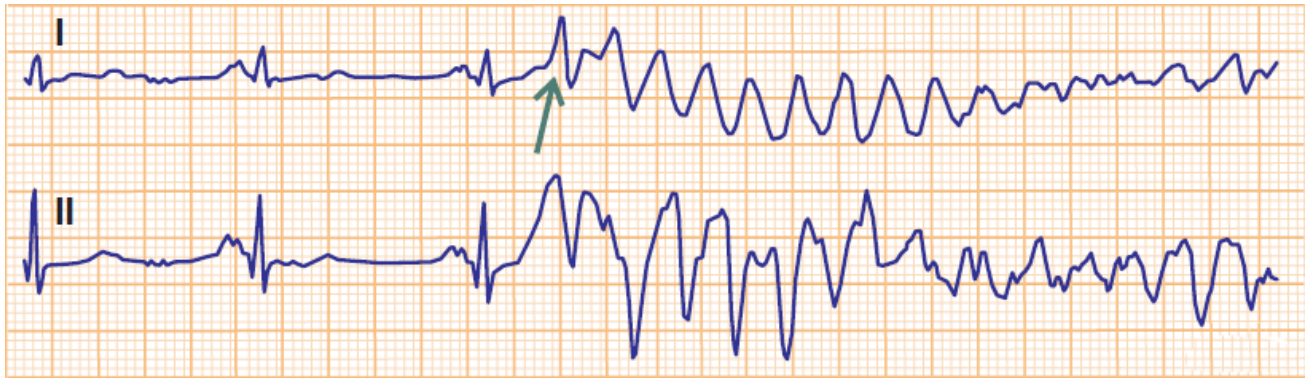
ventricular trigeminia

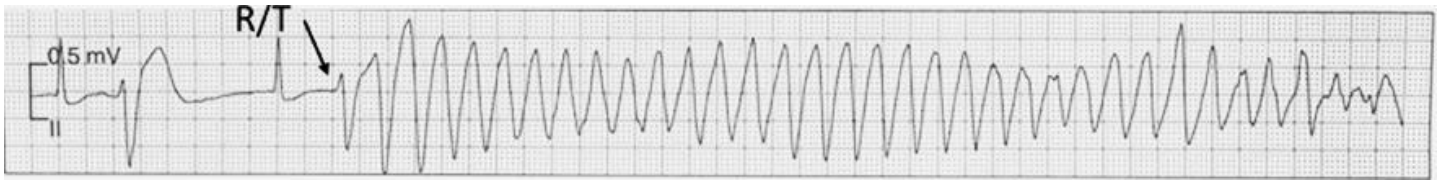


ventricular bigeminia

"R-on-T" phenomenon

- ventricular extrasystole stands on descendent part of T vawe
- can trigger an attack of ventricular tachycardia (mostly torsade de pointes) or ventricular fibrillation





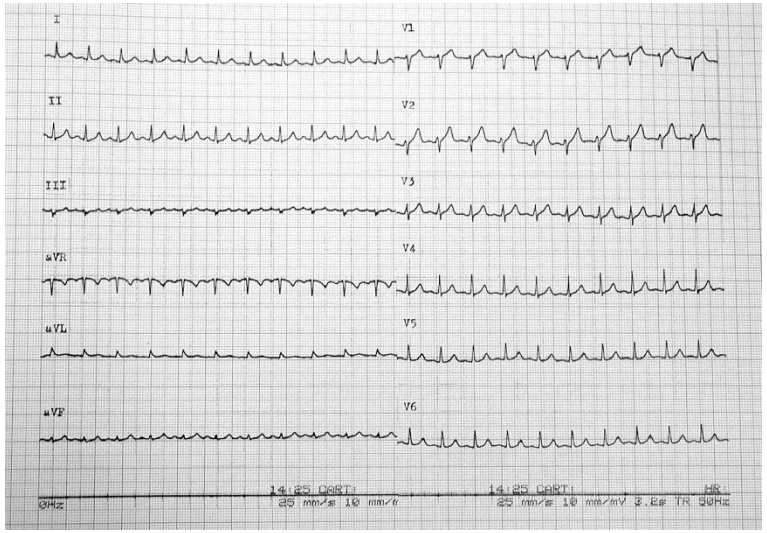
https://www.techmed.sk/fenomen-r-na-t/

Lown's grading of ventricular premature beats

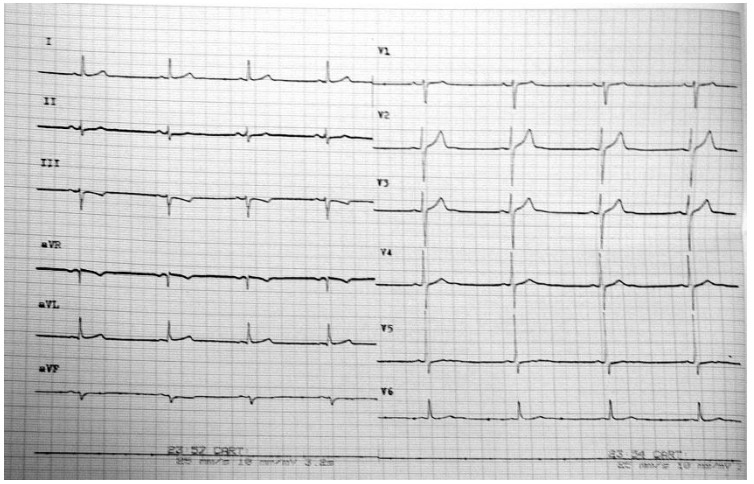
Grade	Character of ventricular premature beats (VPBs)
0	No VPBs
I	Unifocal and infrequent VPBs; <30 VPBs per hour
II	Unifocal and frequent VPBs; ≥30 VPBs per hour
III	Multifocal VPBs
IV A	Two consecutive VPBs (couplets)
IV B	Three or more consecutive VPBs (salves/non-sustained ventricular tachycardia)
V	"R-on-T" phenomenon

Grades III – V = complex forms of VPB

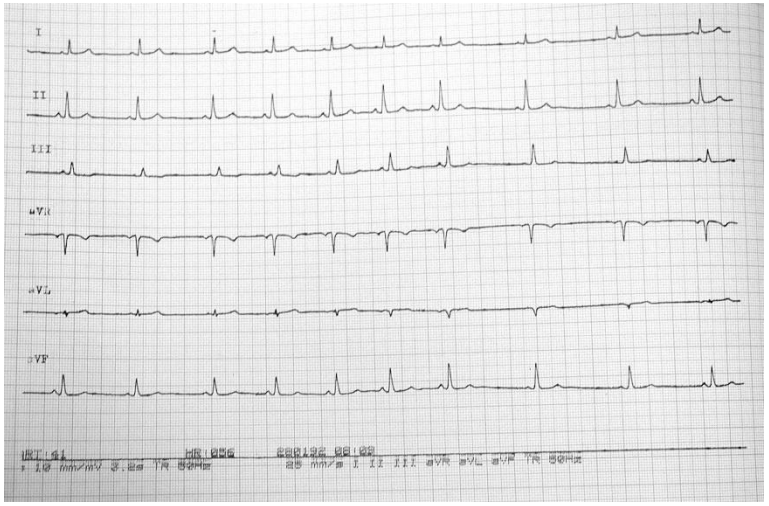
SINUSAL ARRHYTHMIAS



sinus tachycardia (f > 90)
(limit for a healthy person: 220 – age (years)

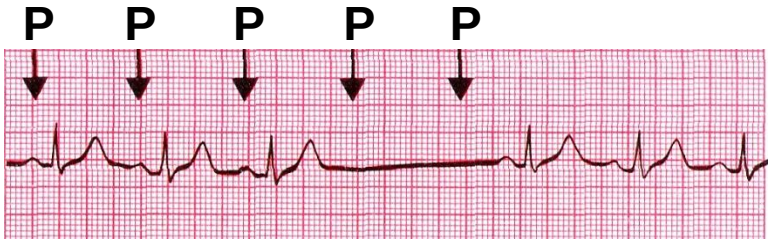


sinus bradycardia (f < 60)

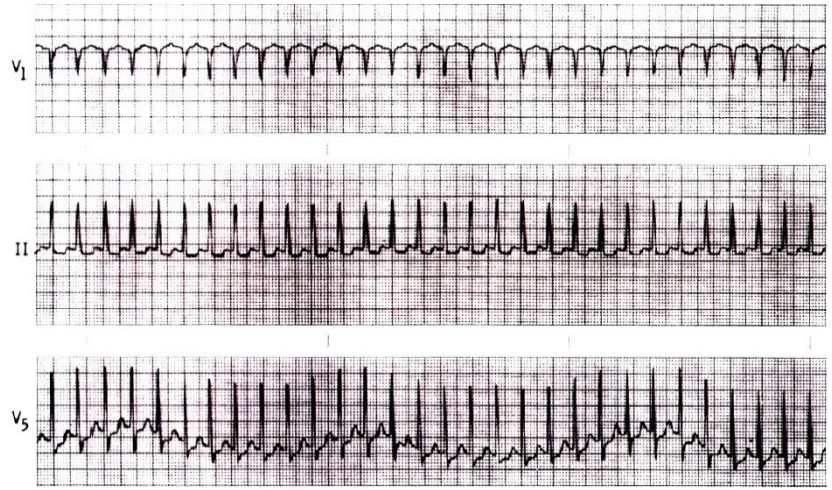


respiratory arrhythmia (Bainbridge‘s reflex)

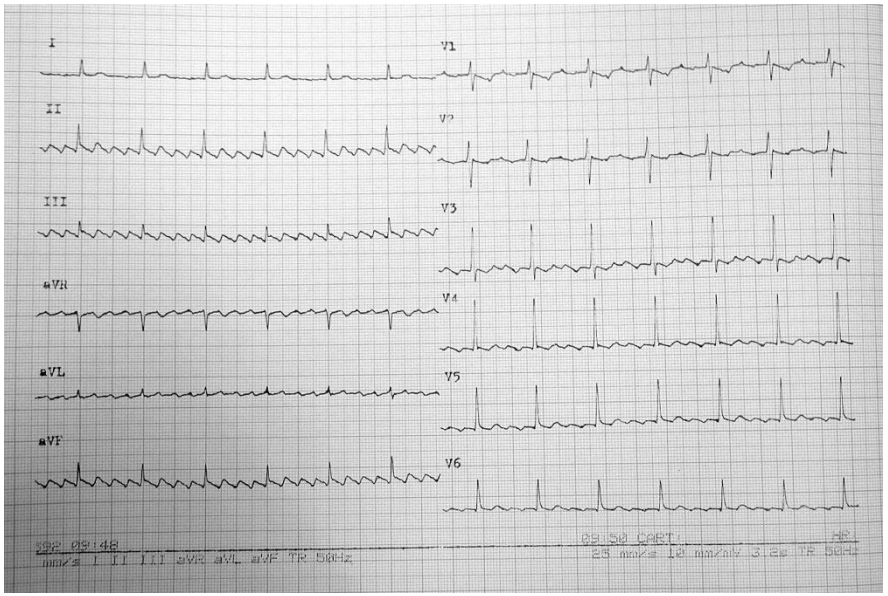
sinus arrest



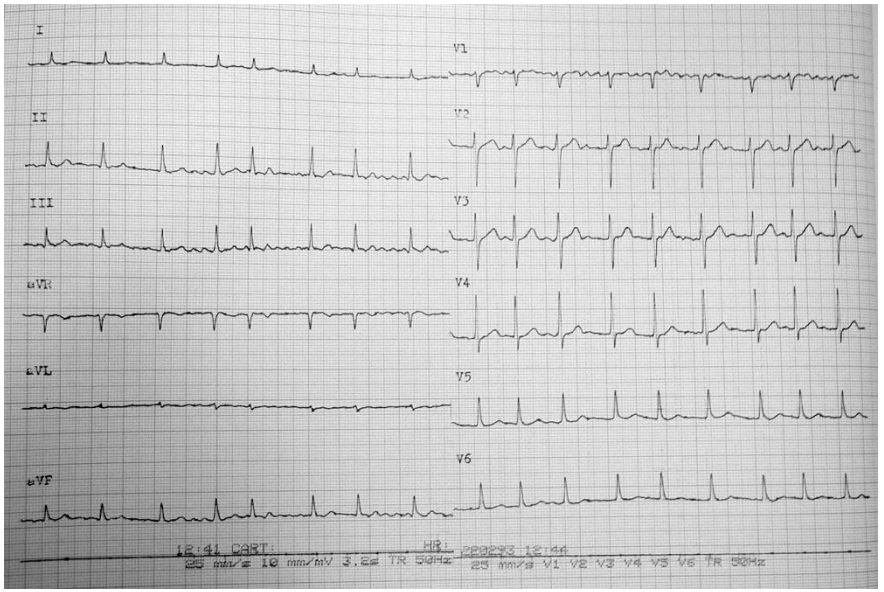
SUPRAVENTRICULAR ARRHYTHMIAS



supraventricular tachycardia



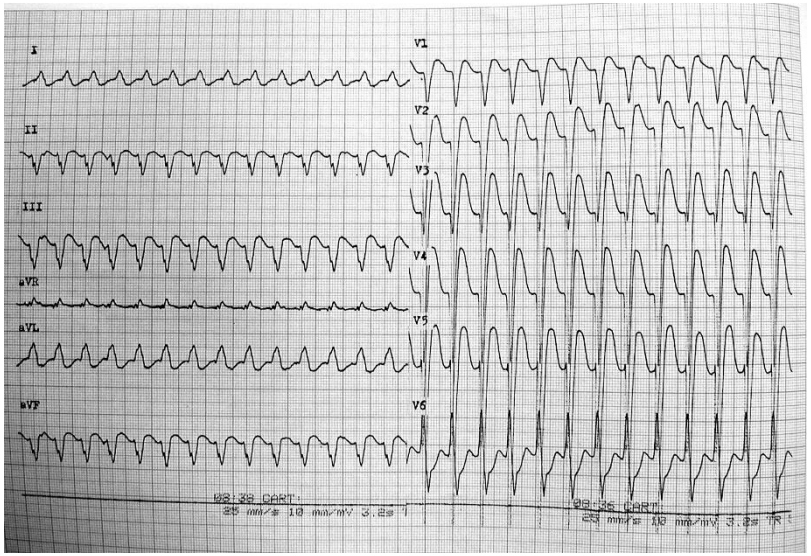
atrial flutter

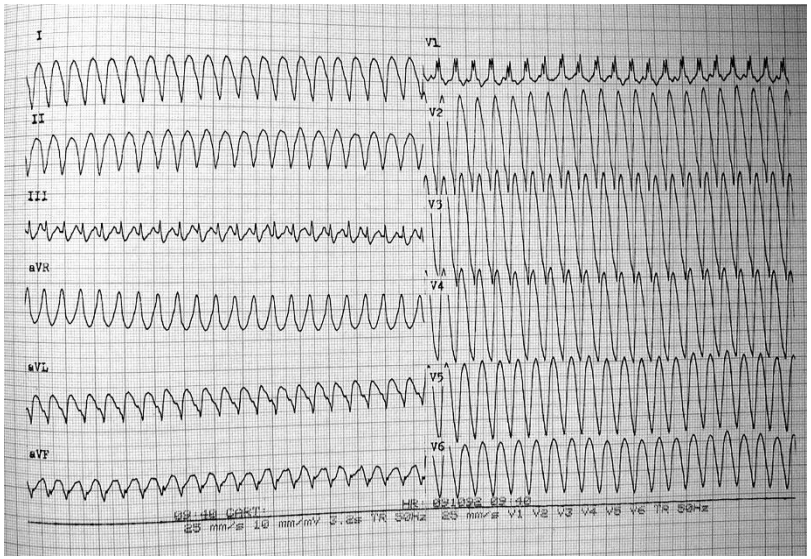


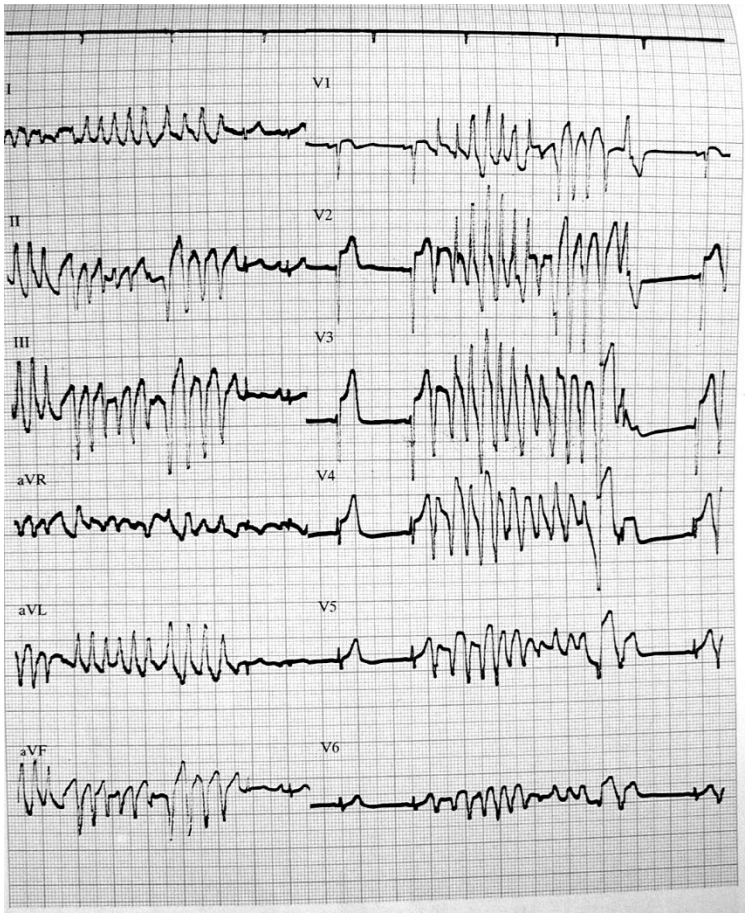
atrial fibrillation

VENTRICULAR PAROXYSMAL TACHYCARDIAS

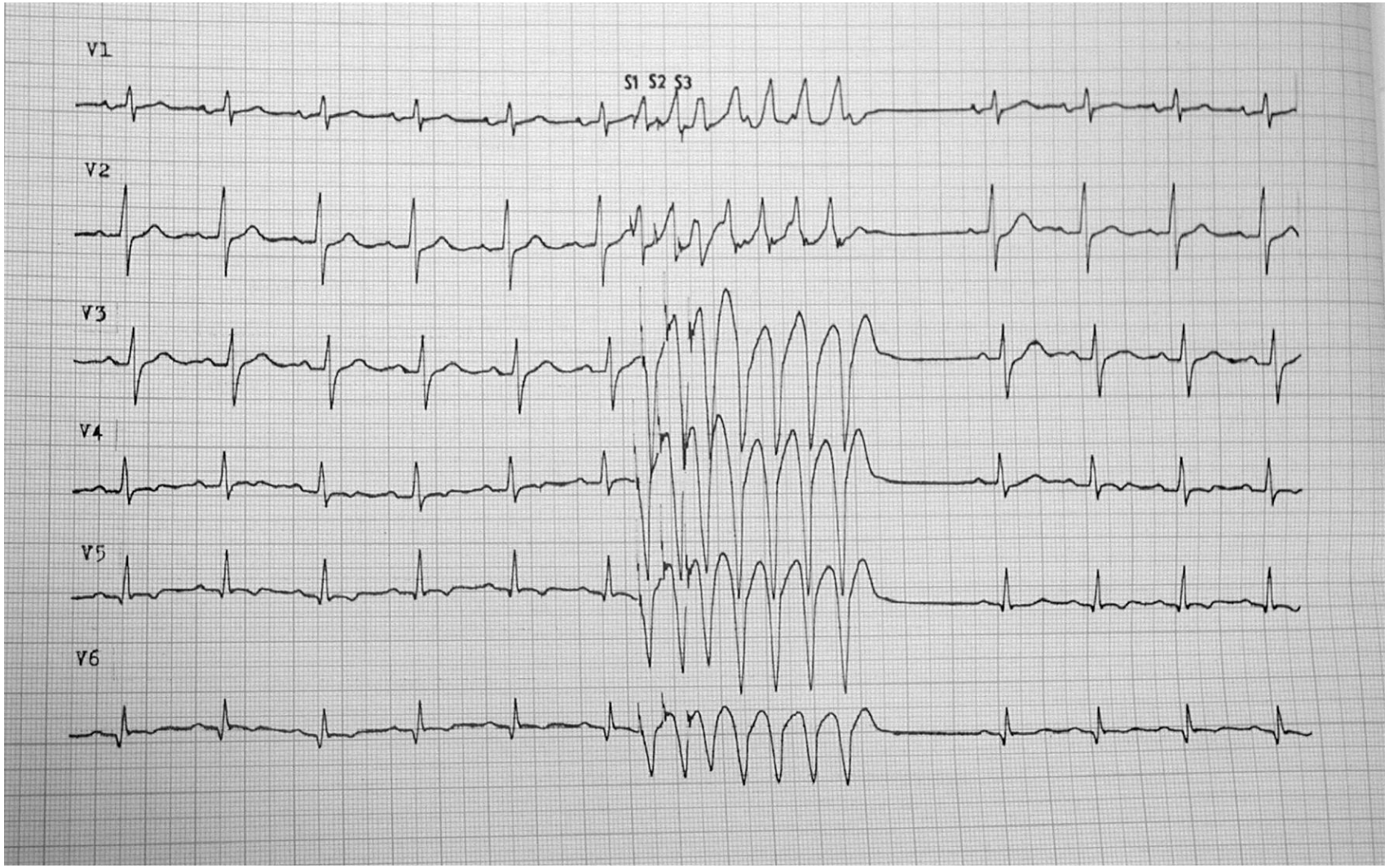
2 examples of ventricular paroxysmal tachycardias



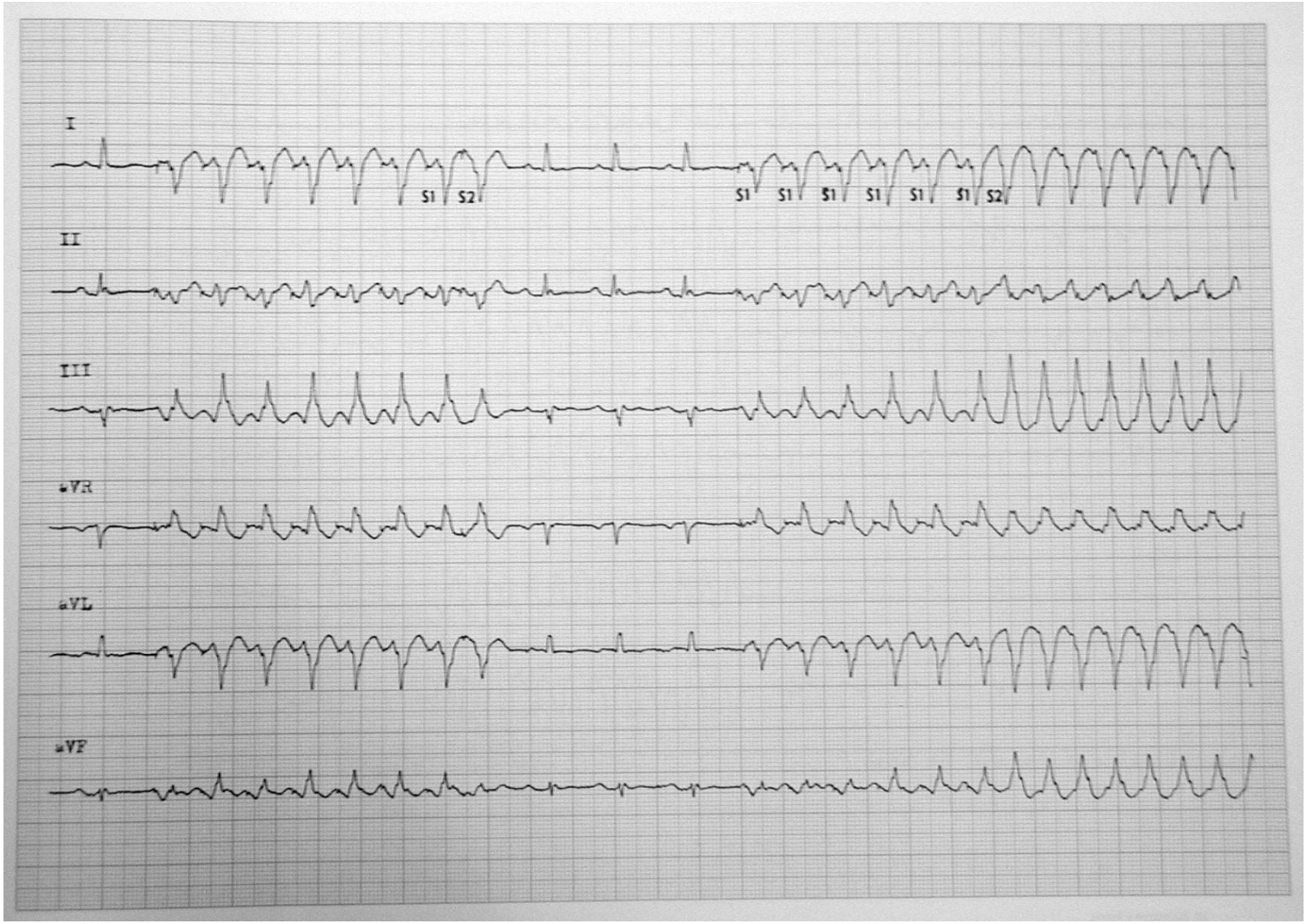




torsade de pointes

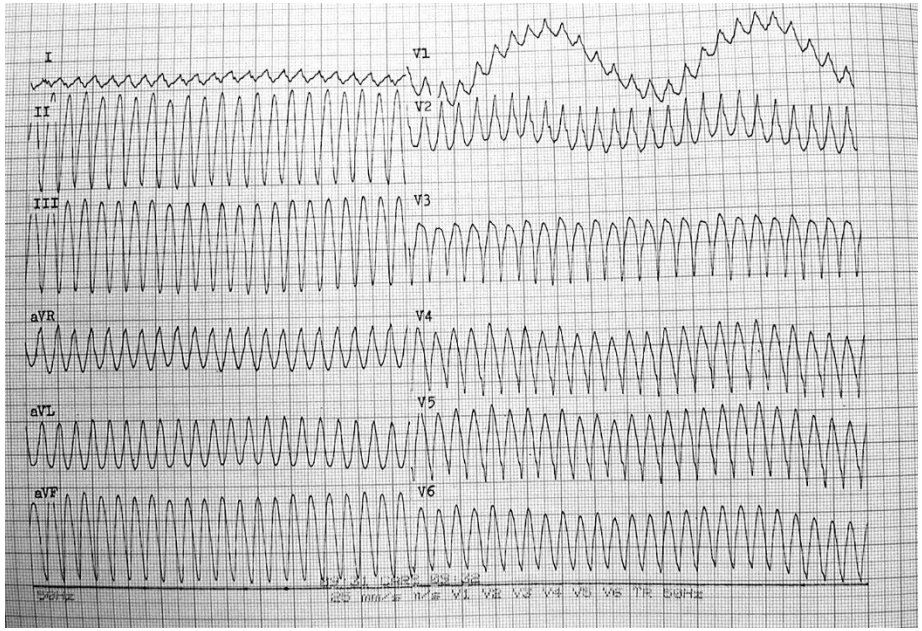


Short paroxysm of ventricular tachycardia

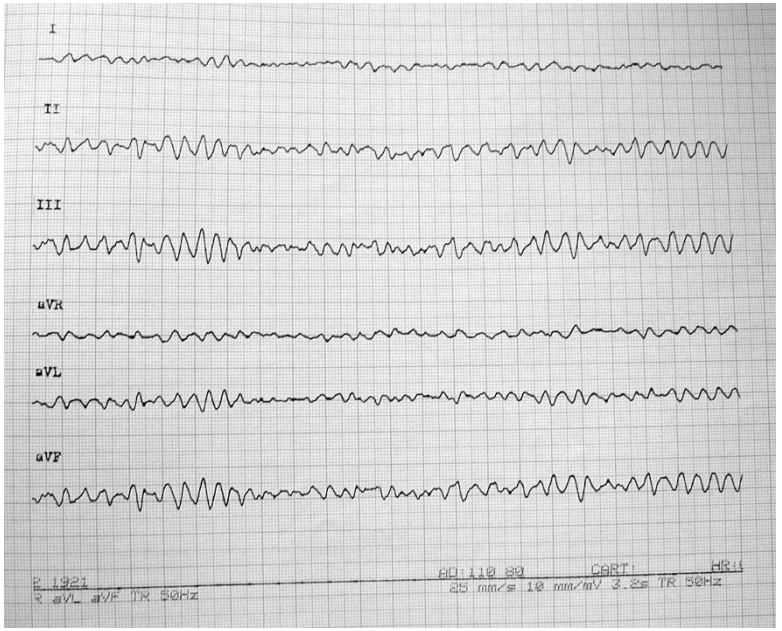


Repeated paroxysms of vetricular tachycardia

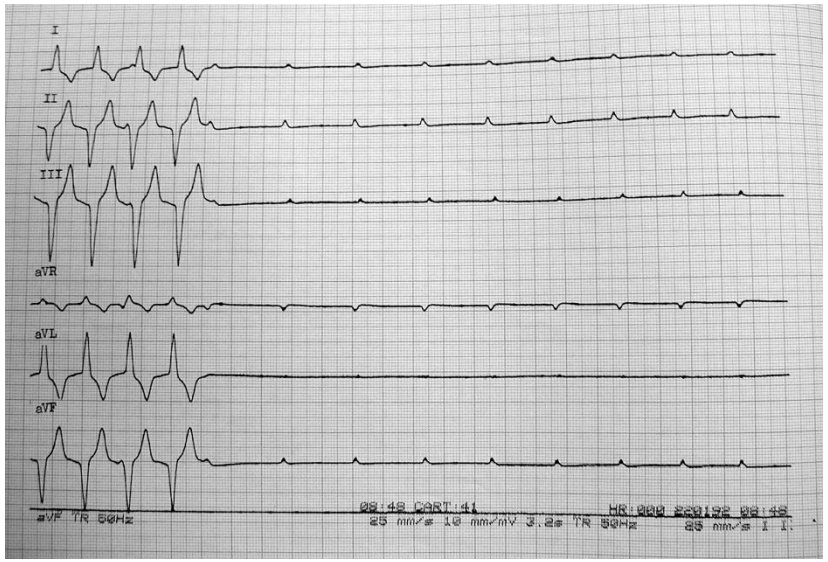
VENTRICULAR TACHYARRHYTHMIAS



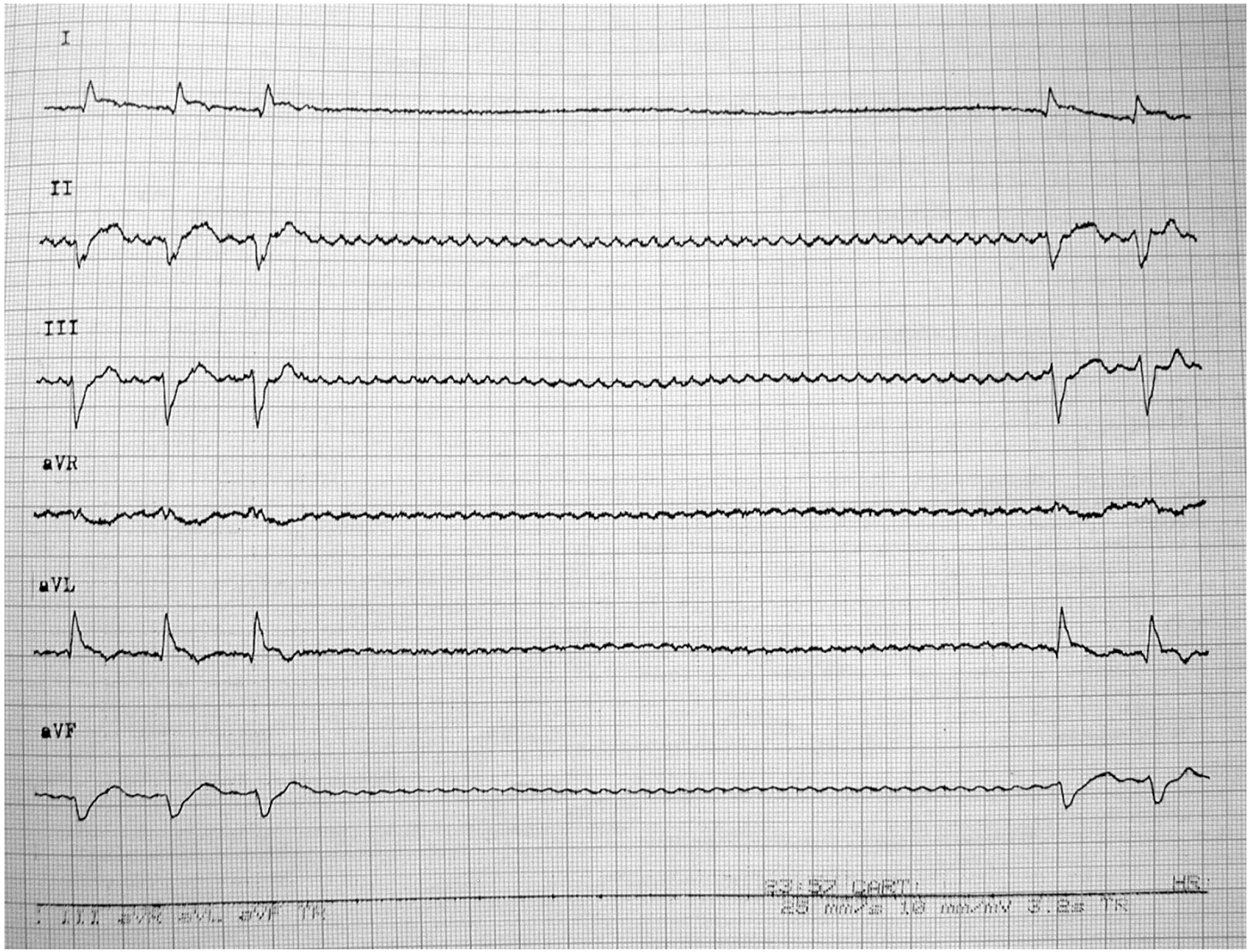
ventricular flutter



ventricular fibrillation



ventricular arrest



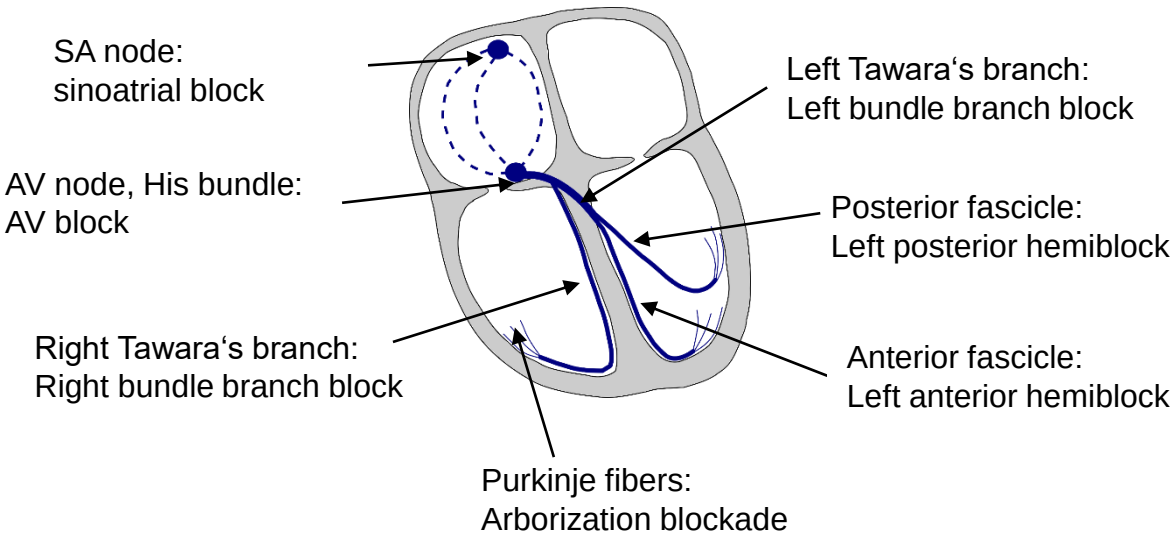
Ventricular arest at atrial flutter

ARRHYTHMIAS: DISTURBANCES OF IMPULSE TRANSMISSION

Speed of transmission and ECG

The transmission of depolarization can be slowed down or stopped anywhere from SA node to the muscles of ventricles. The disturbance of transmission can be detected with surface ECG.

POSSIBLE PLACES OF BLOCKS OF IMPULSE TRANSMISSION



B. DISTURBANCES OF IMPULSE TRANSMISSION

1. SA block - Ist degree
- IInd degree
- IIIrd degree (complete SA block)

2. AV block - Ist degree
- IInd degree - Mobitz 1 (Wenckebach periods)
  - Mobitz 2
- IIIrd degree (complete AV block, atrioventricular dissociation, double command)

3. Bundle blocks - RBBB
- LBBB
- hemiblocks, fascicular blocks (bifascicular, trifascicular blocks)

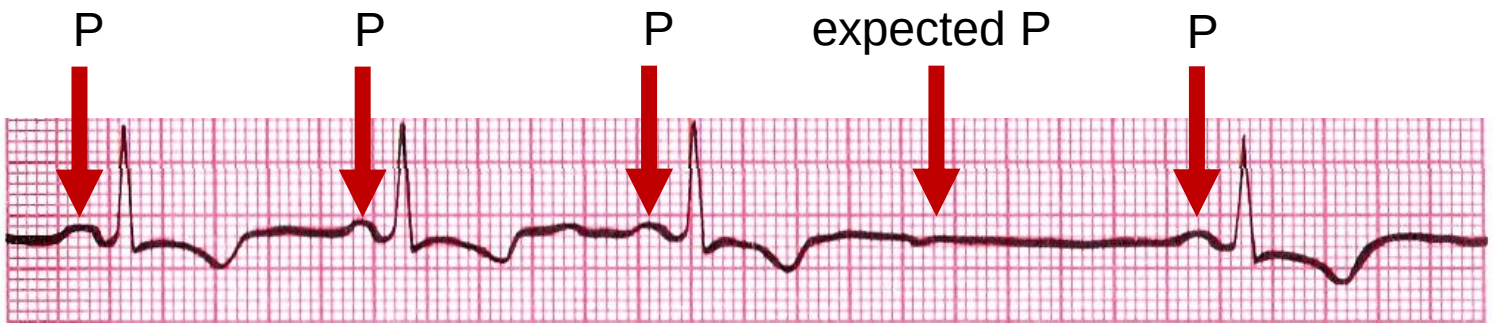
4. Block of arborisation

4. Preexcitation syndrome - WPW
- CLC (LGL)

1. SA block

- Ist degree – no ECG changes
- IInd degree - absence of some P and QRS
- IIIrd degree – alternative rhythm

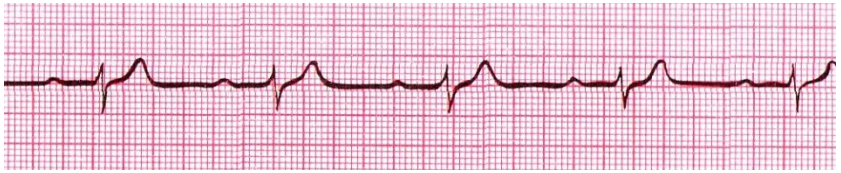
Sinoatrial block, IInd degree



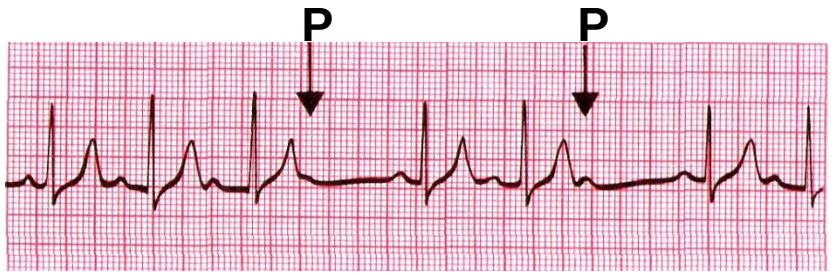
2. AV block

- Ist degree - PQ > 0.2 s
- IInd degree - Mobitz 1 (Wenckebach periods)
  - Mobitz 2
- IIIrd degree - double commande

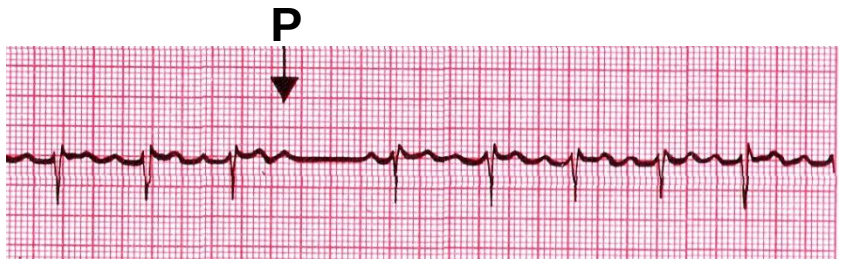
AV block Ist degree



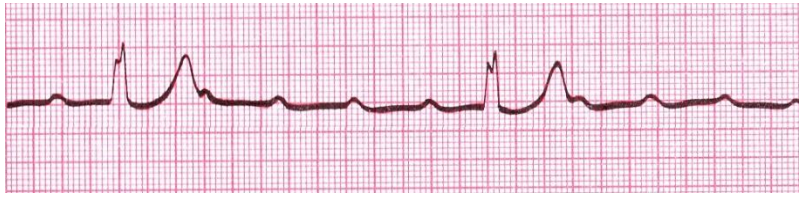
AV block IInd degree (Wenckebach type)



AV block IInd degree (Mobitz 2)



IIIrd degree = complete atrioventricular block

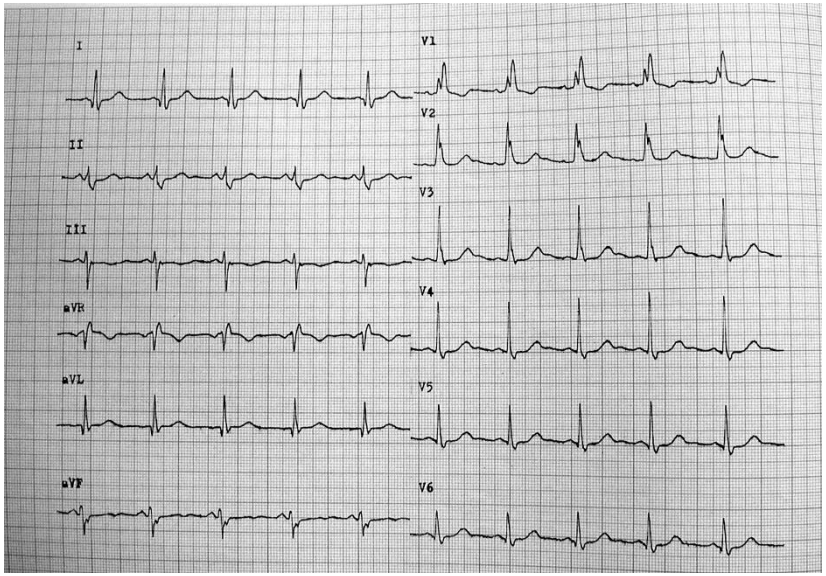


3. Bundle blocks

complete: QRS more than 0.12 s

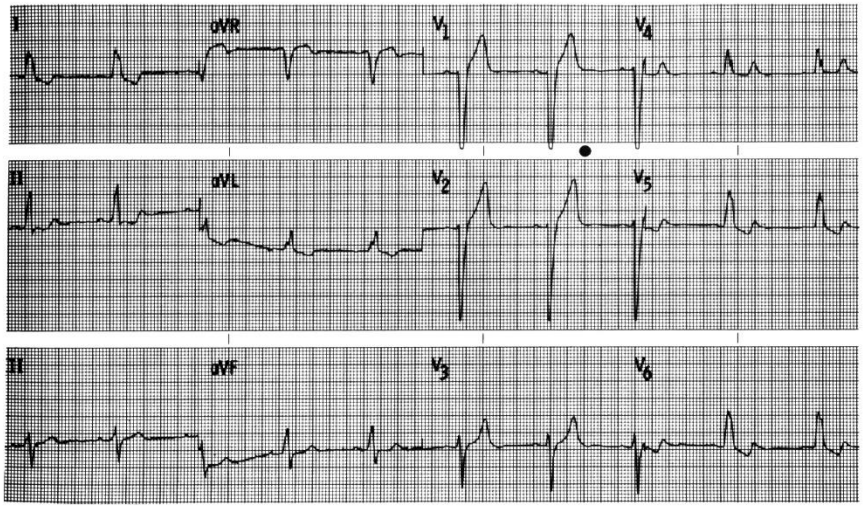
incomplete: QRS up to 0.11 s

- RBBB (right bundle branch block)
- LBBB (left bundle branch block)
- hemiblocks, fascicular blocks



RBBB

- activation of the right ventricle via the left bundle



LBBB

- abnormal right to left activation of the septum
- activation of the right ventricle via the right bundle

Fascicular blocks

Left anterior hemiblock
- left axis deviation
- S to V6
- R <S in the lead II
- lead I: R
- lead III and aVF: S

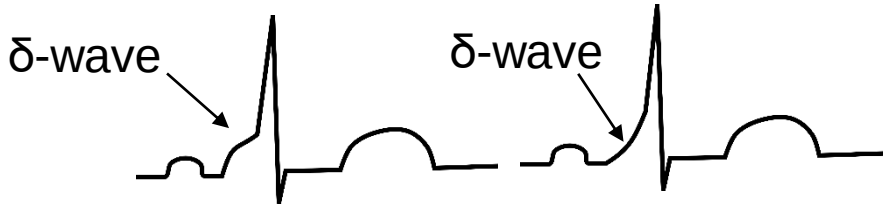
Left posterior hemiblock
- right axis deviation
- lead I: S
- lead II a III: R
- lead aVL: rS
- lead aVF: qR

RBBB + LAH: RBBB + left axis deviation
RBBB + LPH: RBBB + right axis deviation
Trifascicular block: AV block deg. III with aberrant QRS

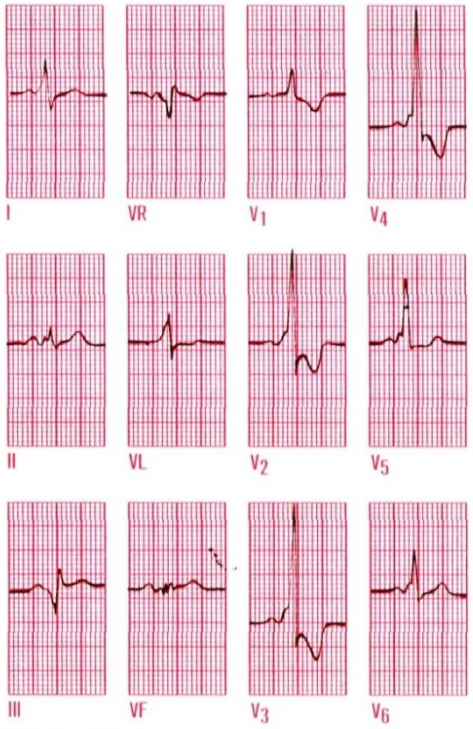
4. Preexcitation syndrome

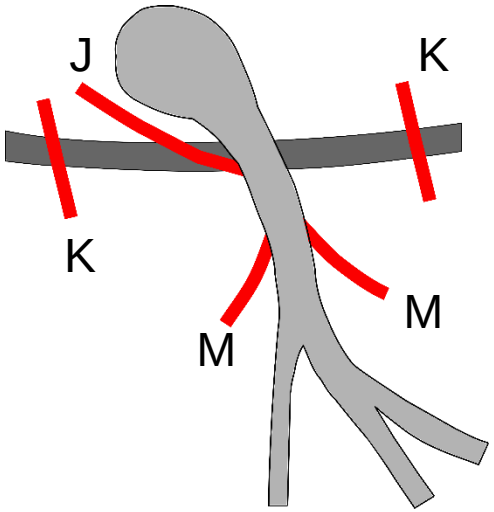
- developmental (or rarely acquired – e.g. inflammation) anomaly
- muscular conductive fibers connecting the atria and ventricles

- Kent fascicle (K)
- James fascicle (J)
- Mahaim fascicle (M)

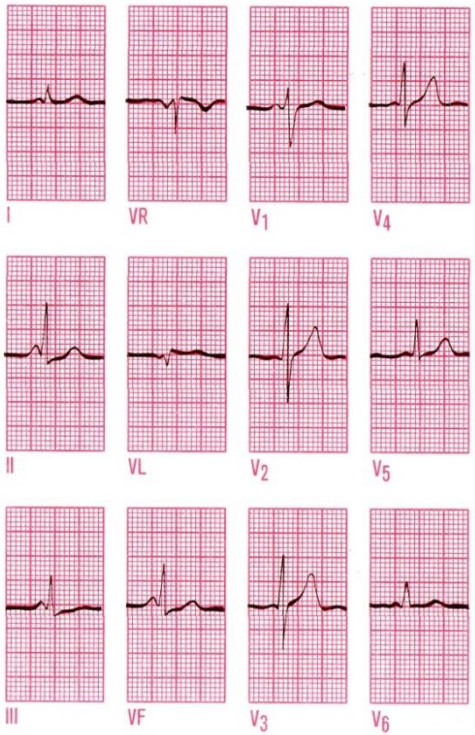


Wolf-Parkinson-White syndrome (WPW):
- short PR (PQ) interval, δ-wave





Lown-Ganong-Levin (LGL), or Clerc-Levy-Christesco (CLC) syndrome:
- short PR (PQ) interval



ICHEMIC HEART DISEASE (IHD)

Classification of ischemic heart disease

1) According to ECG changes:

Angina pectoris - stable
- instable
- Prinzmetal

Myocardial infarction - STEMI, Q IM
- non-STEMI, non-Q IM

Arrhythmic form of IHD

Sudden death

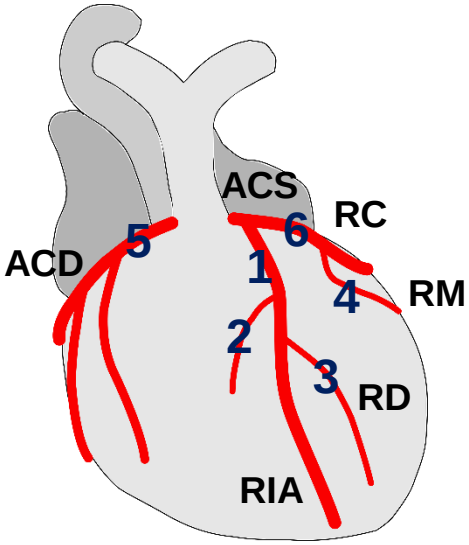
IHD without clinical symptoms

2) According to clinical symptoms:

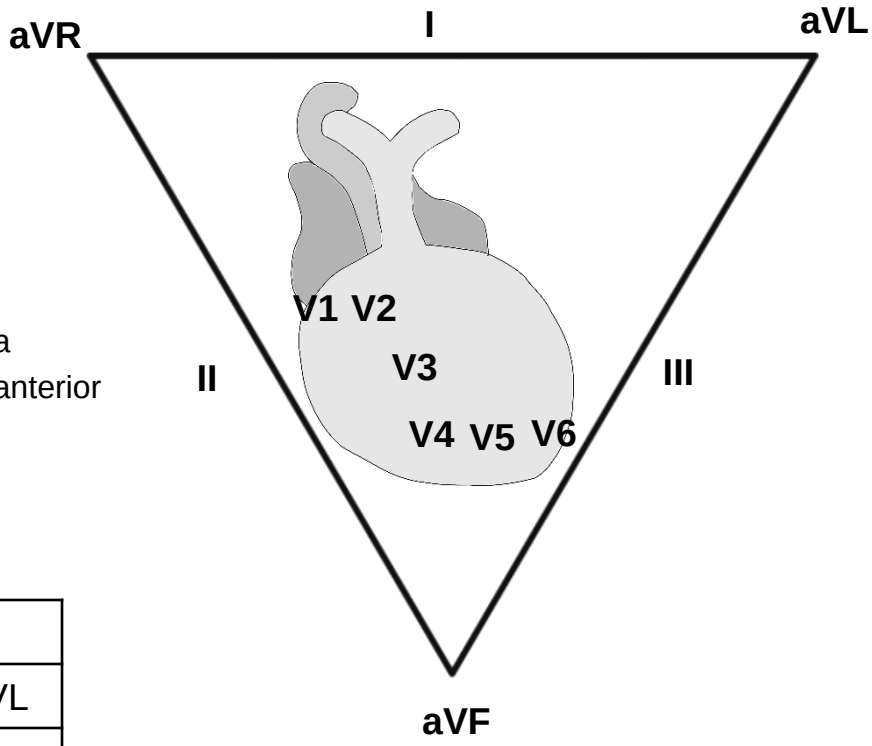
Acute: acute MI, instable AP, arrhythmic form, sudden death

Chronic: state after MI, stable AP, IHD without clinical symptoms

Localization of the ischemic focus



ACD – arteria coronaria dextra
ACS – arteria coronaria sinistra
RIA – ramus interventricularis anterior
RC – ramus circumflexus
RD – ramus diagonalis
RM – ramus marginalis

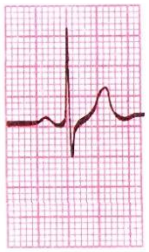


Occlusion	Ischemic focus	ECG leads
1	**anterior wall**	**V1 – V6**, I, aVL
2	anteroseptal	**V1-V3**, V4
3	anterolateral	**V4-V6**
4	high lateral	I, aVL
5	**diaphragmatic** (inferior)	**II, III, aVF**
6	diaphragmatic-lateral	**II, III, aVF, V5-6**
7	posterior	**V7, V8, V9...**
8	circular	**V1-6, II, III, aVF**

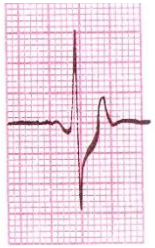
1. Angina pectoris (AP)

- stable – ST depression
- unstable – identical ECG as with stable AP
- Prinzmetal – ST elevation

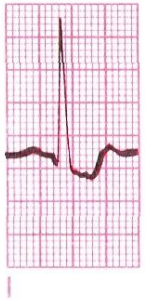
REST



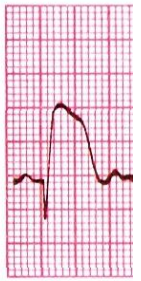
EXCERCISE



physiologic ascendant depression during exercise

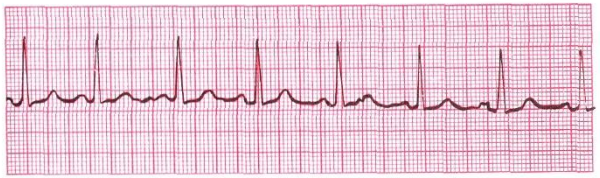


anginal depression during exercise

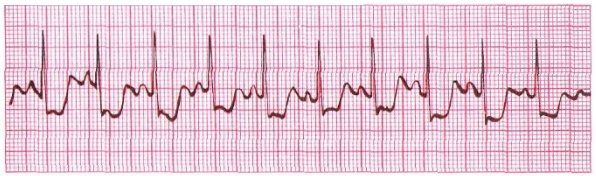


Prinzmetal AP – ST elevation during rest

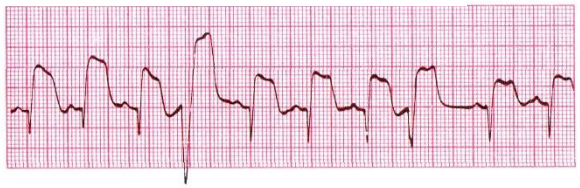
rest: without pain

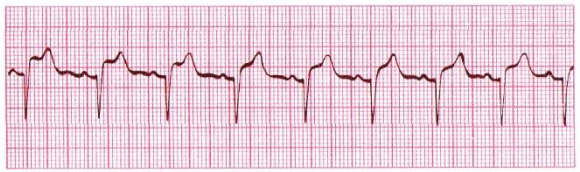


pain during rest



Prinzmetal AP

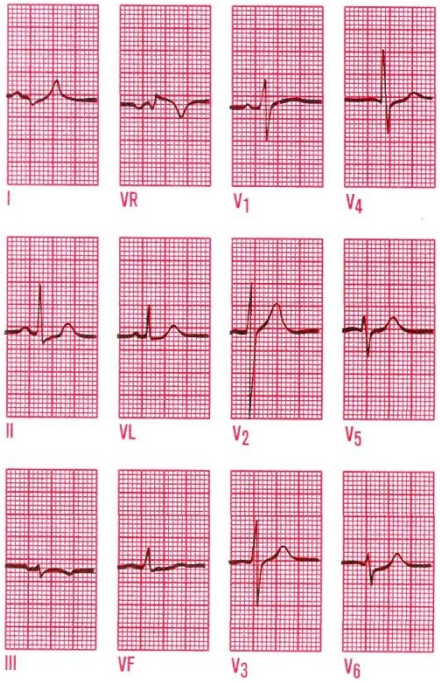




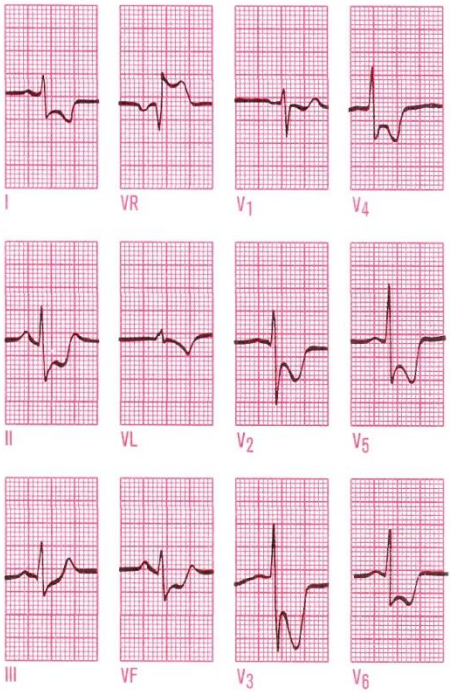
Evolution - rapid recession of the elevation

Angina pectoris

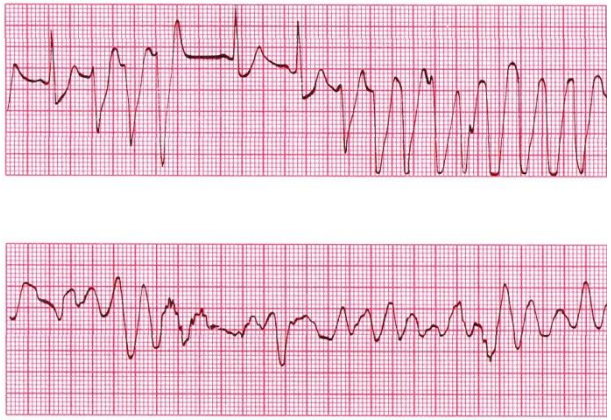
Rest



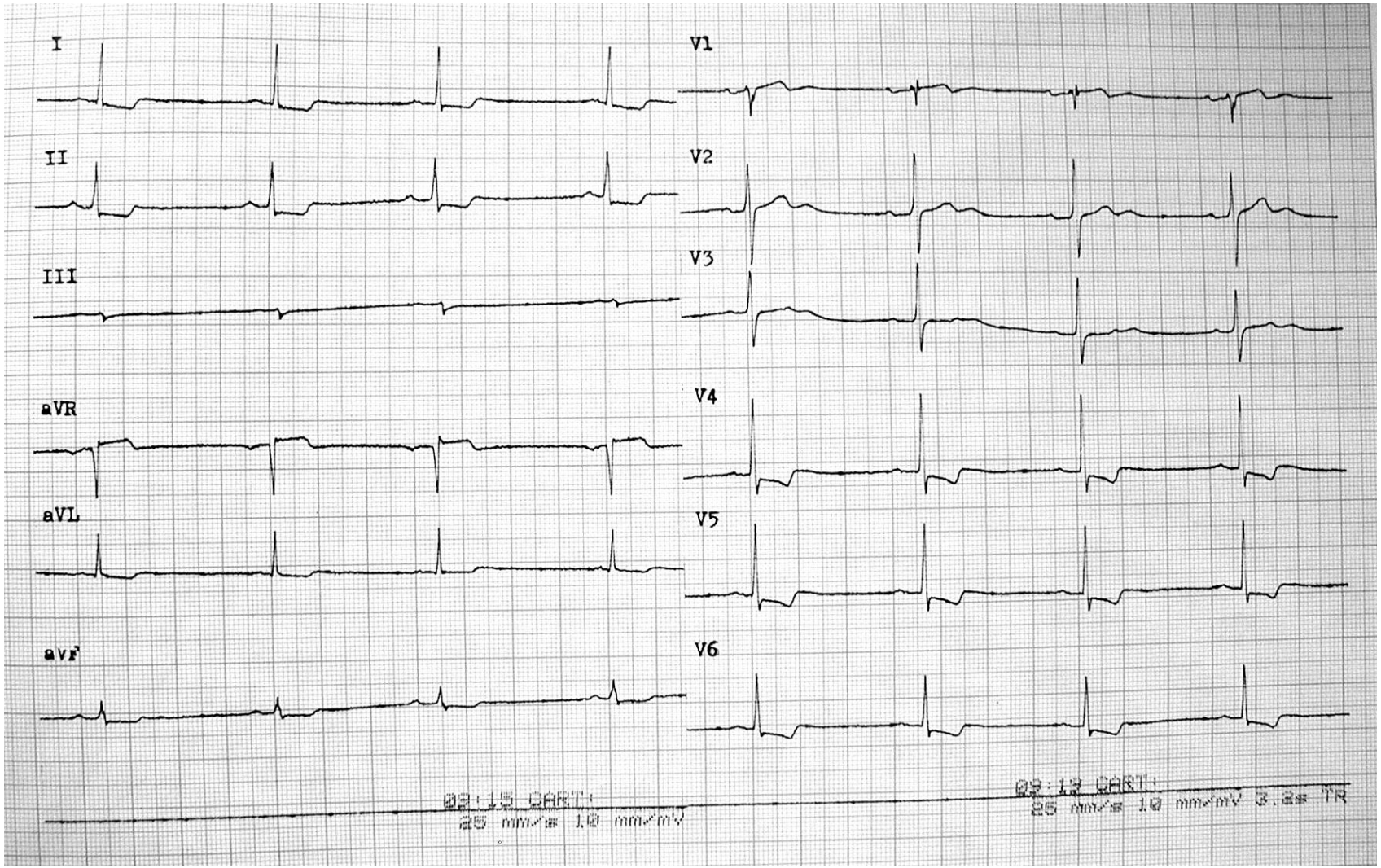
Exercise



1 min after exercise



short ventricular tachycardia, 2 sinus contractions
again ventricular tachycardia, crossing to fibrillation



Angina pectoris – anterior wall

2. Myocardial infarction (IM)

- **non-STEMI, non-Q MI** (often non-transmural – subendocardial)
  ST depression + negative T
- **STEMI, Q-MI** (often transmural)
  pathological Q, Pardee wave, later negative T

Pathologic Q = >0.04 s, deeper than 3 mm, larger than ¼ R

Myocardial lesion
- necrotic zone
- zone of injury
- ischemic zone

Stages
- peracute
- acute
- subacute
- chronic

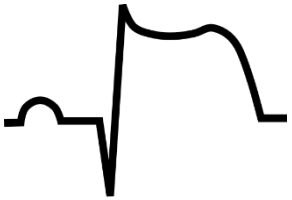
STEMI (ST-elevation myocardial infarction), Q myocardial infarction

normal ECG

acute MI (minutes – tens of hours)

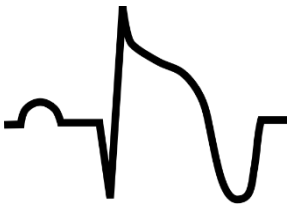
High sharp T-wave
= the firts sign of STEMI

High sharp T-wave
+ ST-elevation
= Pardee wave

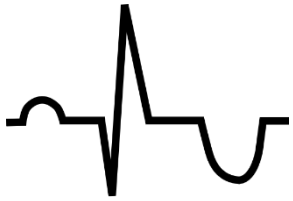


Pathologic Q develops.

subacute MI (days)

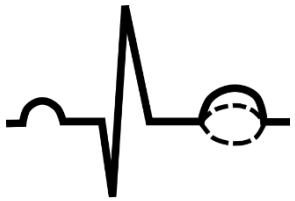


T-wave turns into negativity.



ST-elevation decreases gradually and disappears later, T-wave is negative.

state after MI (weeks - permanently)

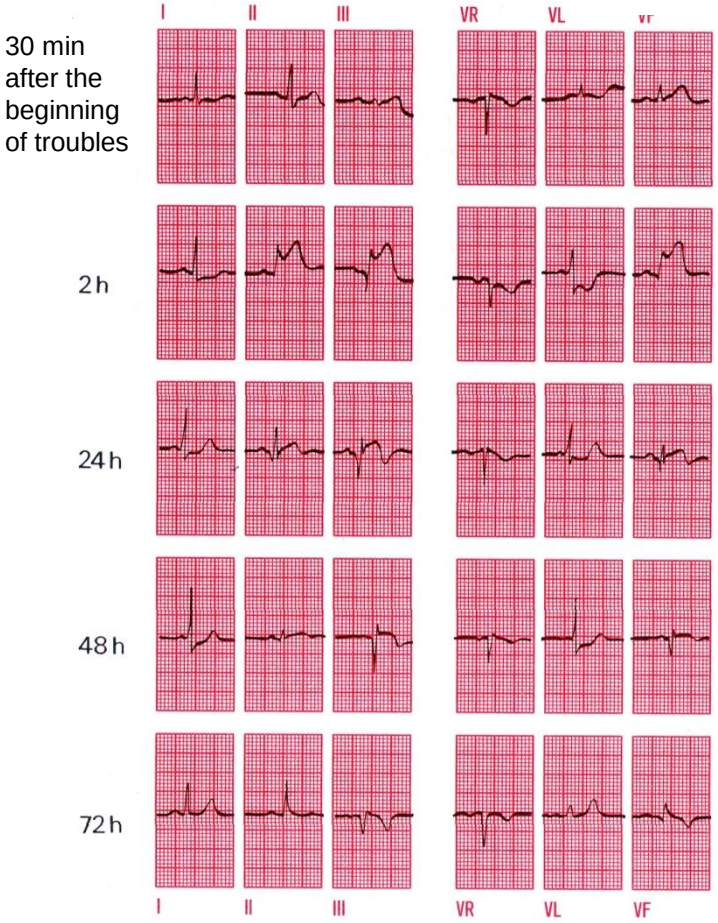


Pathologic Q persists, T-wave normalizes or remains flattened or slightly negative.

Note: Thanks to the timely treatment, pathologic Q does not develop in many cases.

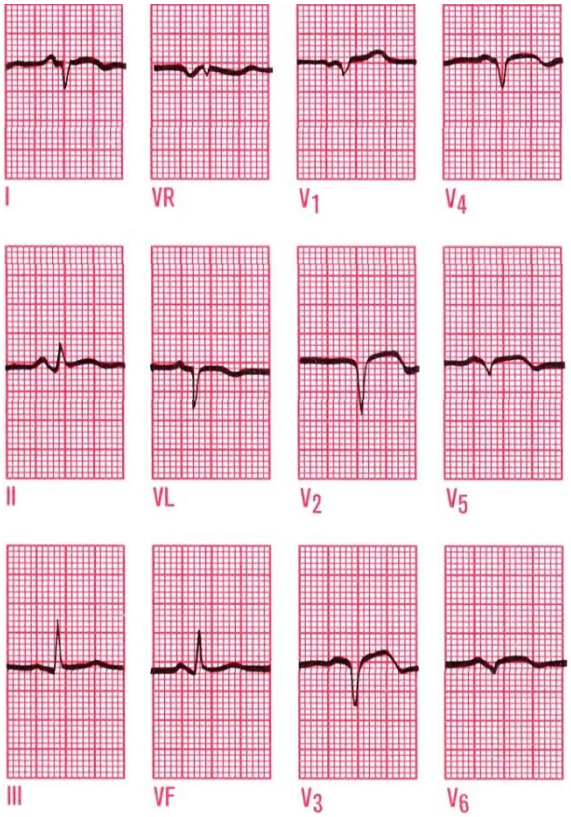
STEMI, Q myocardial infarction

Evolution of inferior STEMI



Old infarction of the anterior wall

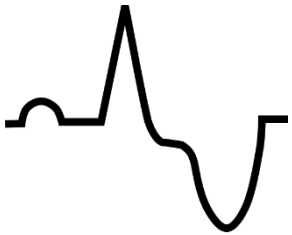
with left ventricle aneurysma



Non-STEMI (Non-ST-elevation myocardial infarction), non-Q myocardial infarction

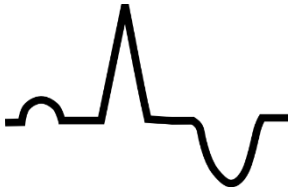
normal ECG

acute MI (minutes – tens of hours)



Negative sharp T = coronary T, horizontal ST-depression

subacute MI (days)

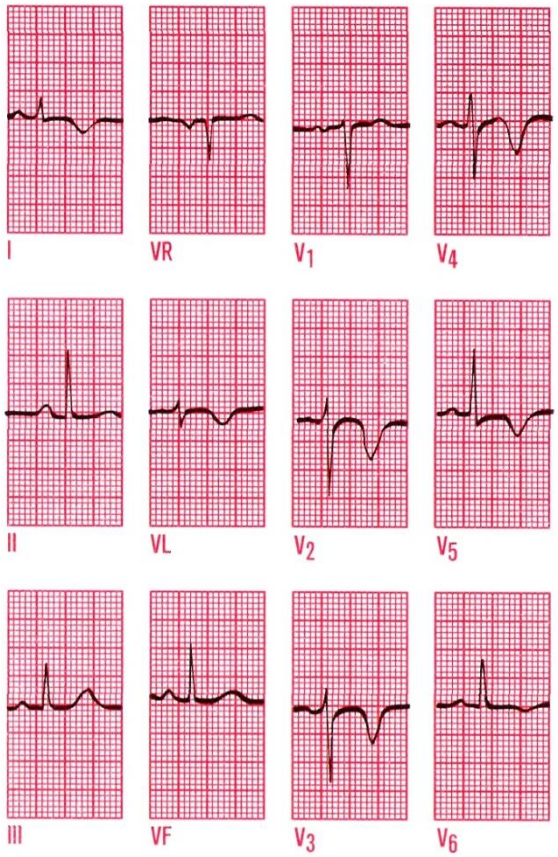


Negativity of the T-wave decreases, ST-depression disappears.

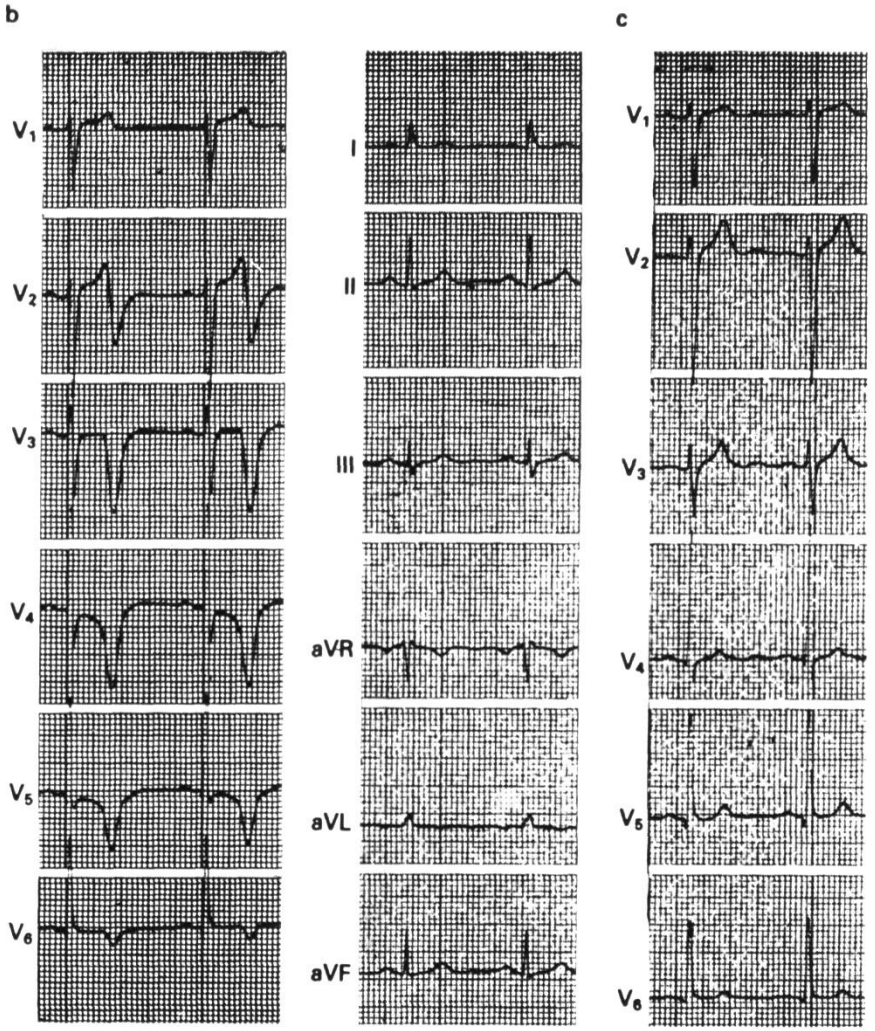
state after the MI (weeks - permanently)

ECG is completelly normalized. Minor chenges of the T-wave persis sometimes (flattened or slightlynegative).

subendocardial infarction

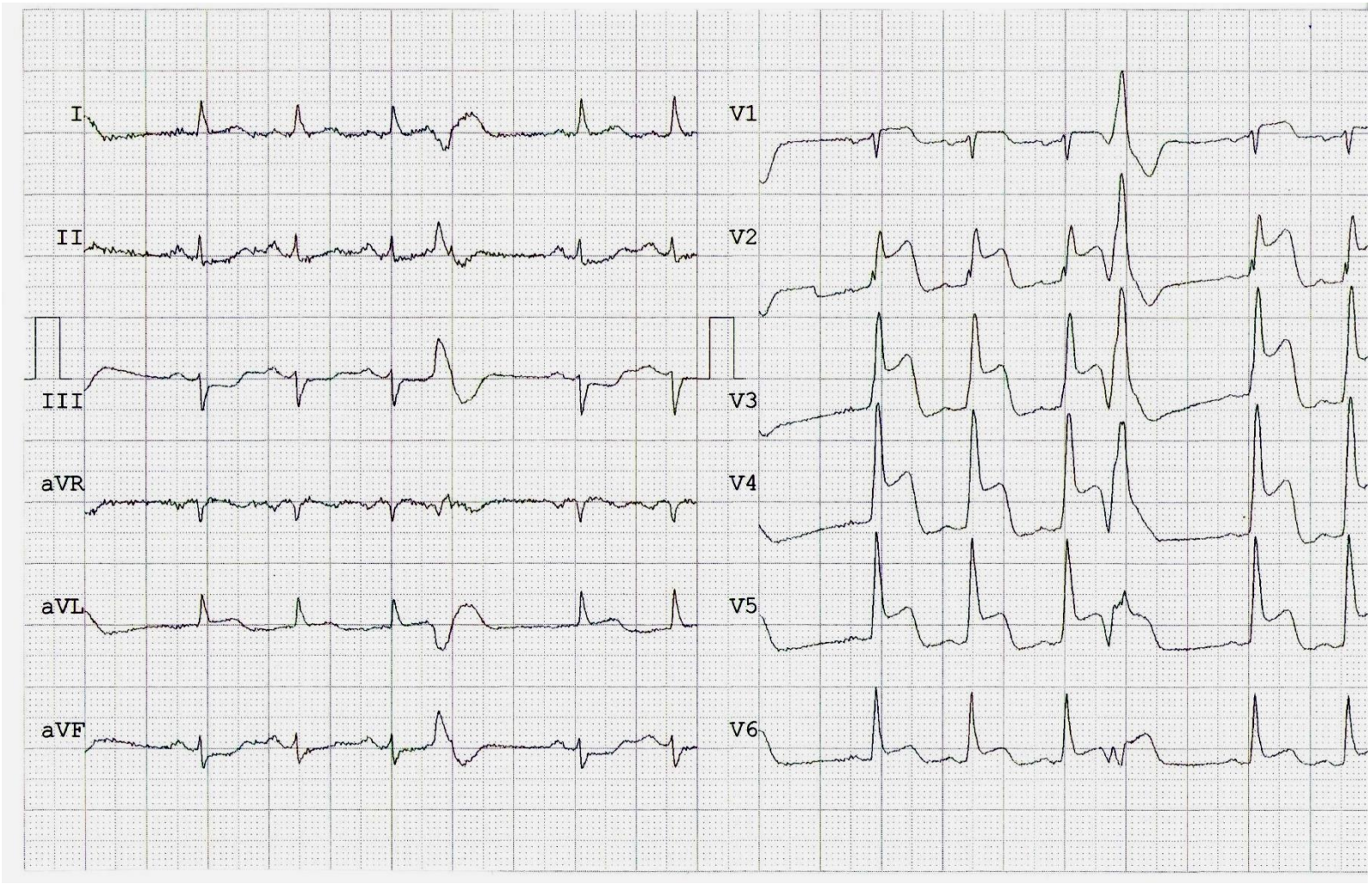


subendocardial IM (non Q) anterolateral

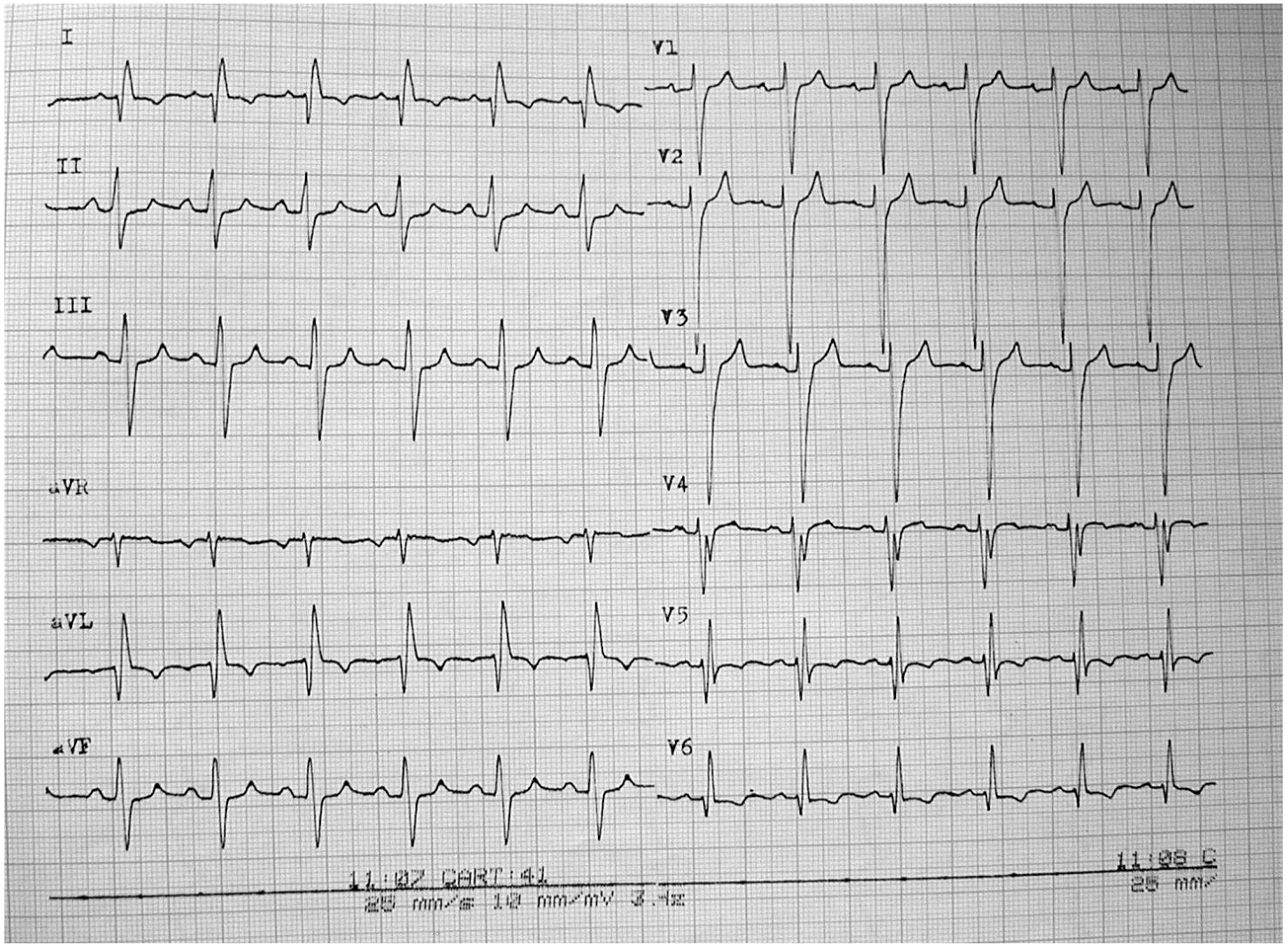


acute anterior MI (non Q)

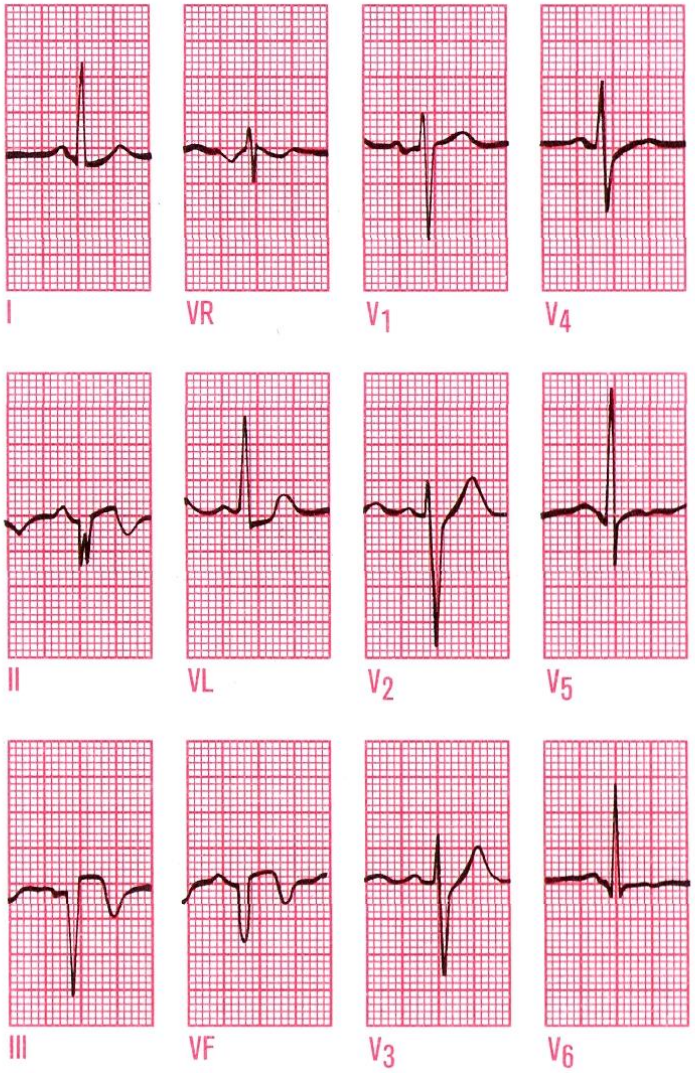
state after non Q MI, normalization of the T-wave



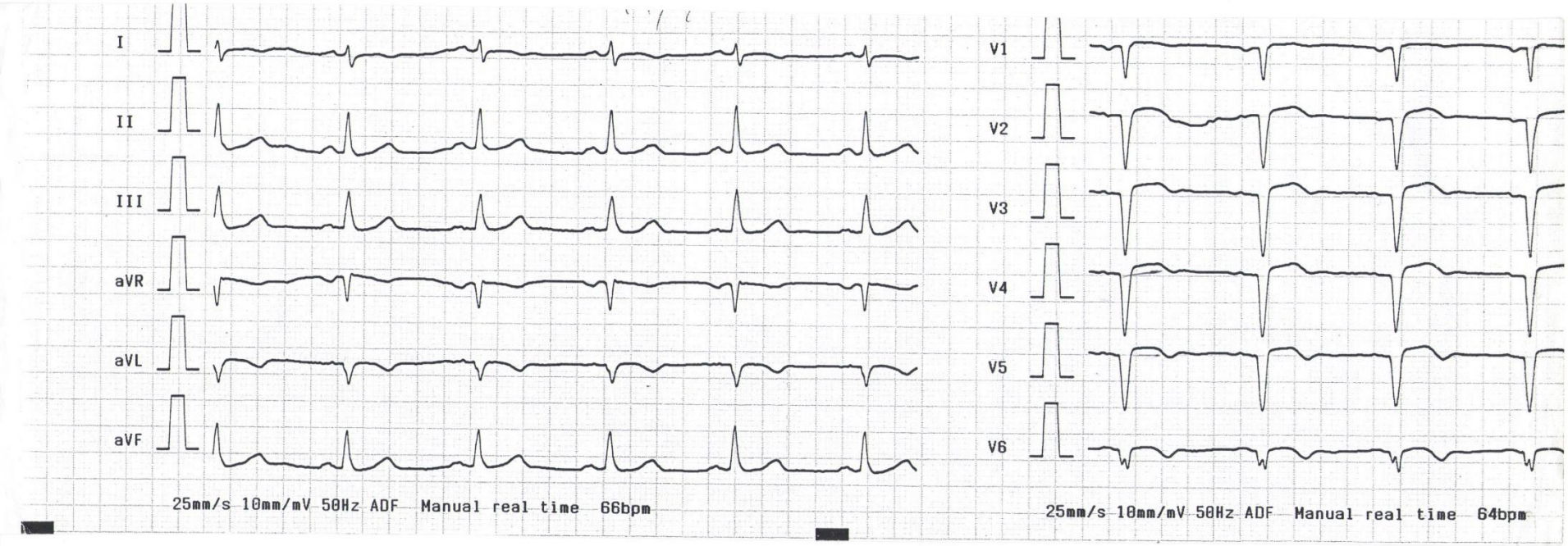
STEMI anterior wall, ventricular ES



Subacute Q-MI lateral



Subacute Q-MI posterior wall



Anterior wall aneurysm

OTHER PATHOLOGICAL STATES WITH ECG CHANGES

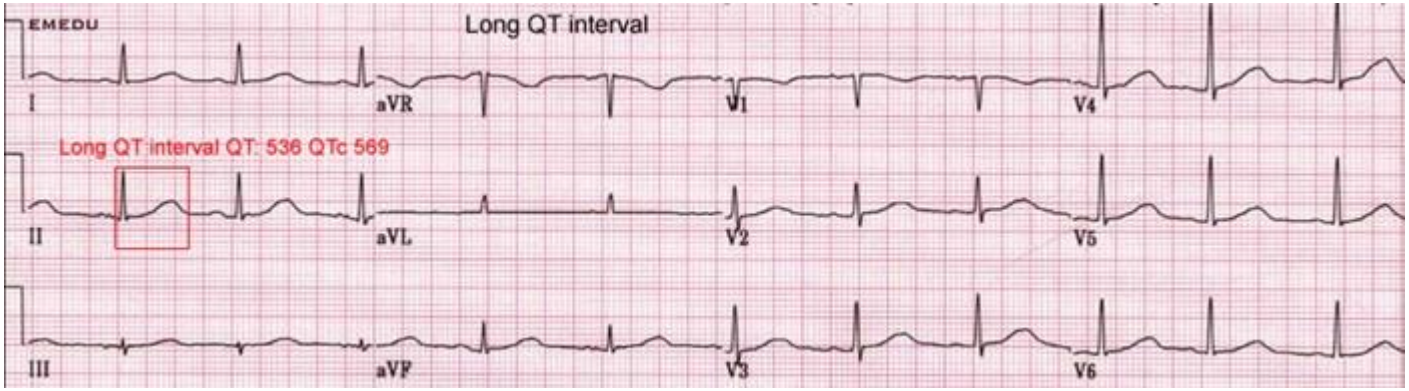
Pulseless **E**lectrical **A**ctivity: PEA

= electromechanical dissociation

- The electrocardiogram shows a heart rhythm that should produce a pulse, but does not!
- Causes: hypothermia, hypoxia, hypovolemia, hypoglycemia, cardiac tamponade, tension pneumothorax…
- Pulseless electrical activity leads to a loss of cardiac output, cardiopulmonary resuscitation should be initiated promptly!

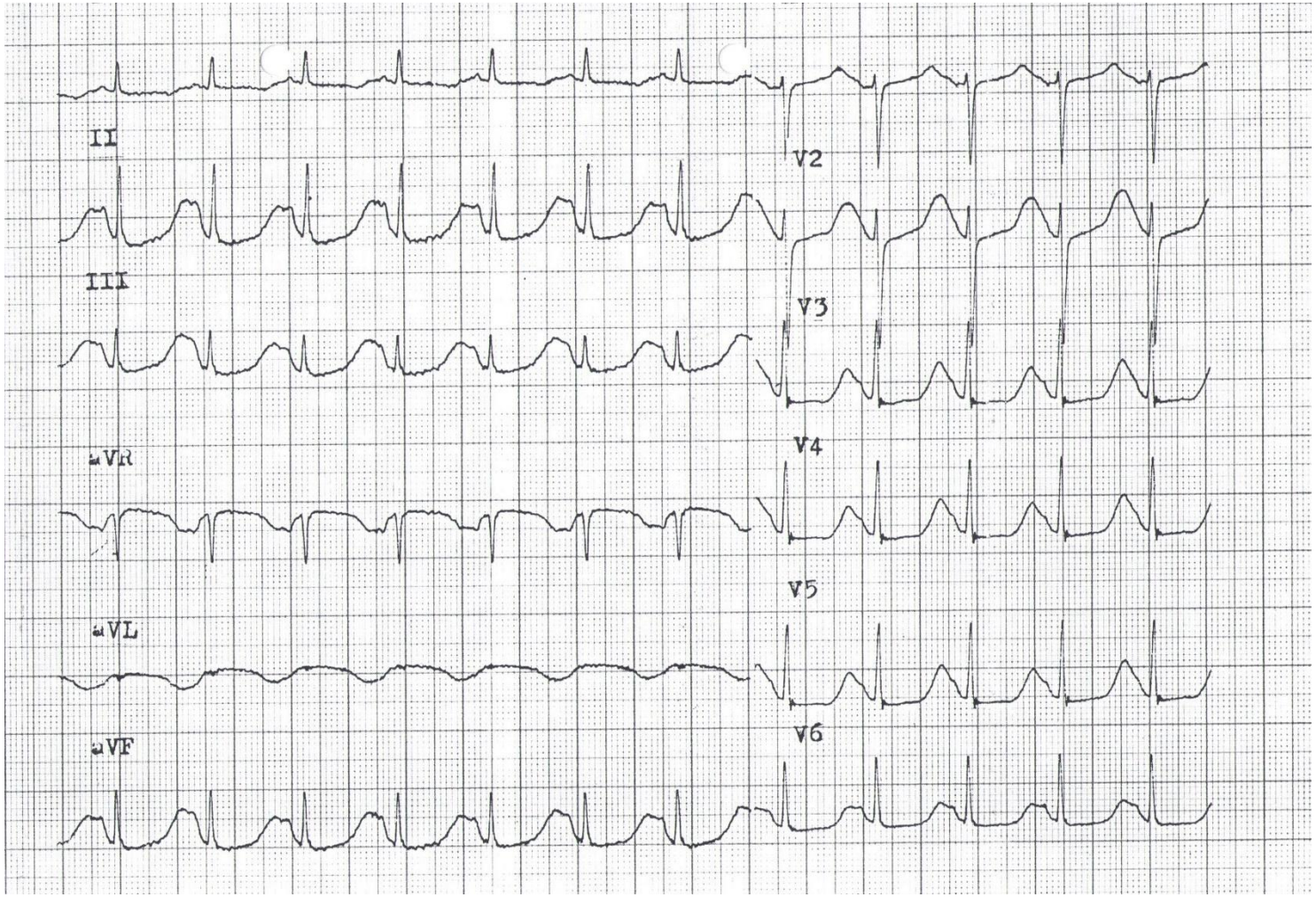
Prolonged QT interval

QT interval is evaluated as corrected for pulse rate: QTc
QTc=QT/√R-R
Prolonged QTc is longer than 450 ms.
Causes: hereditary canalopathies, organic heart diseases, some drug classes
Prolonged QT interval is a risk factor of ventricular tachycardia torsade de pointes and sudden death of the patient.

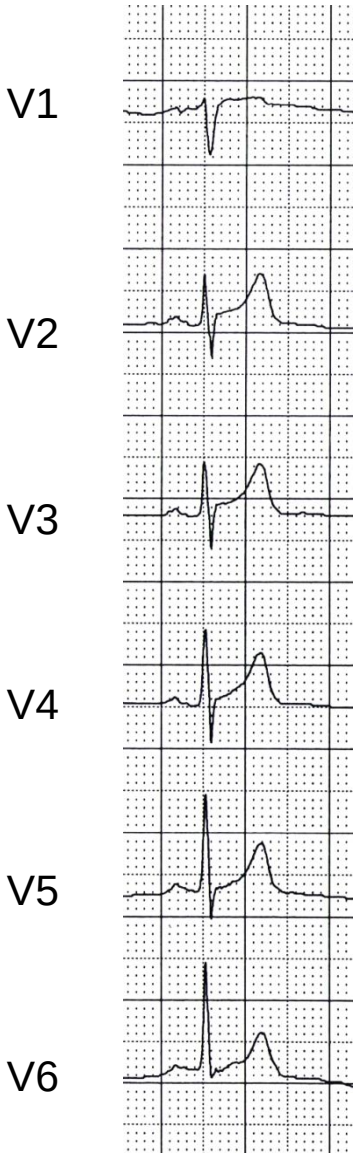


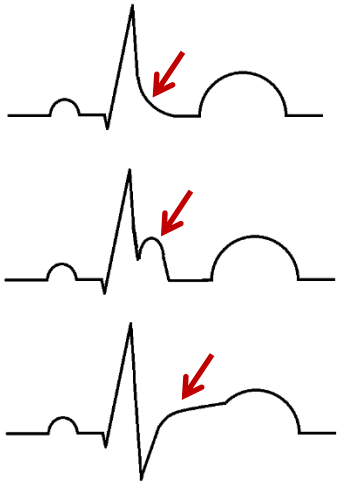
https://www.google.com/search?q=dlouh%C3%BD+qt+interval&source=lnms&tbm=isch&sa=X&ved=0ahUKEwj9t66_-9PeAhXSC-wKHaP-CDoQ_AUIDigB&biw=1600&bih=884#imgrc=TyRe95nDkeUY9M:&spf=1542201289849

PROLONGED QT



SYNDROME OF PREMATURE REPOLARIZATION



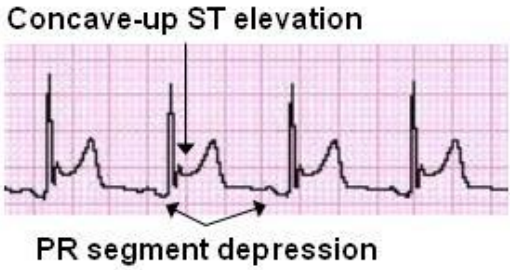


The arrows indicate the so-called point J: the place where the QRS complex passes into the ST segment.
If this place is increased of more than 1mm, it is a wave J that deforms the terminal phase of the QRS complex. Pathophysiological background of wave J is a change in potassium ion eflux during repolarization of the cell. There is a relationship between the finding of premature repolarization (J wave) and the risk of sudden arrhythmic death.

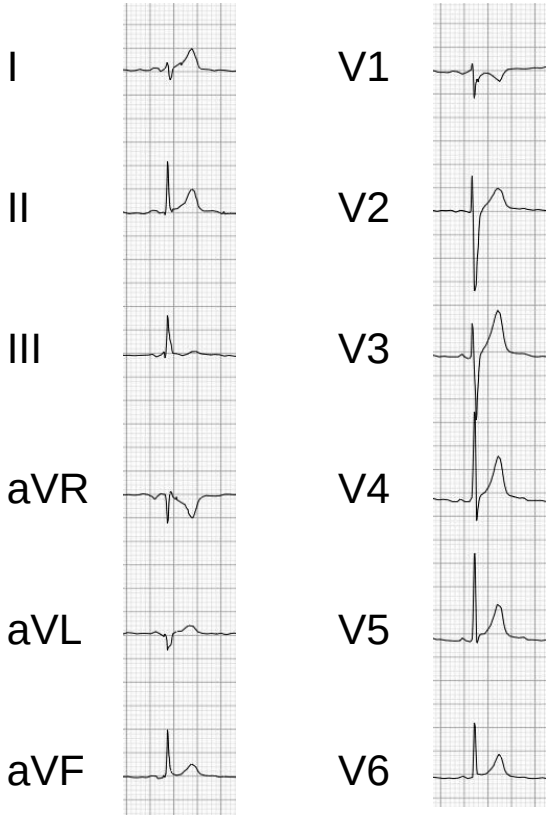
INFLAMMATORY HEART DISEASES

Pericarditis

- horizontal elevations – concave in contradistinction to IM in all leads and Q absent!
- with effusion lower voltage
- PQ (PR) depression



https://www.google.com/search?q=pr+segment+depression&source=lnms&tbm=isch&sa=X&ved=0ahUKEwii5v3cyNbeAhUEDOwKHRqrAWYQ_AUIDigB&biw=1600&bih=884#imgrc=8hCJKGMNlQqMPM:&spf=1542290741123

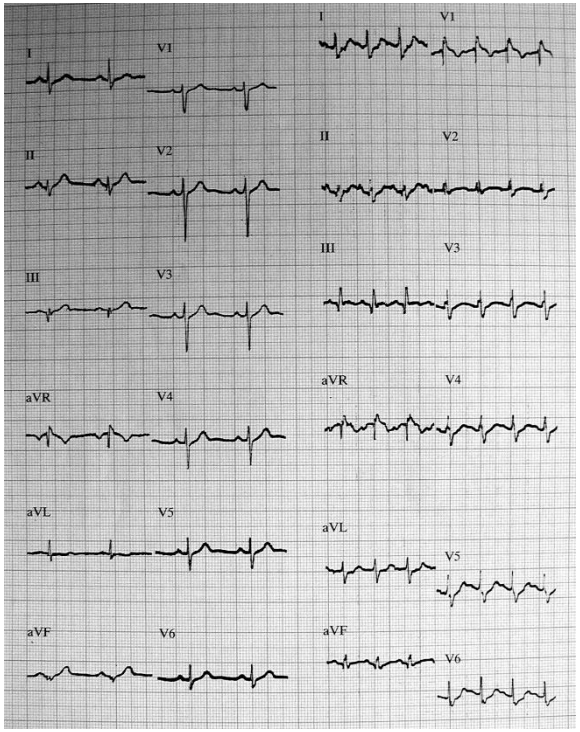


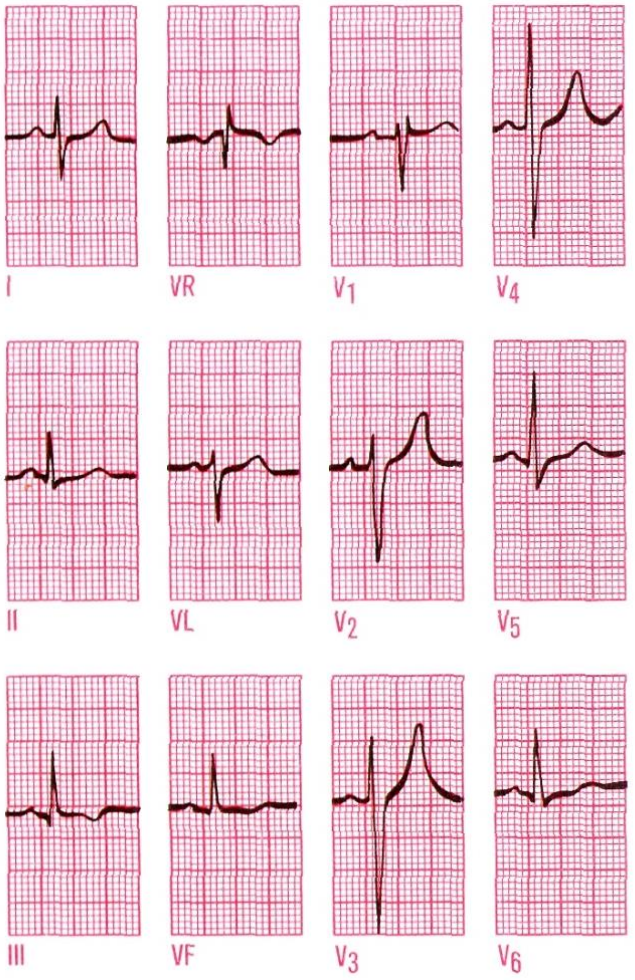
Myocarditis

- unspecific changes:
  - various arrhythmias
  - flat till inverted T wave
  - insignificant ST depressions
  - low voltage (with dilatation or effusion)

PULMONARY EMBOLISM

- often only sinusal tachycardia with chest pain
- right heart axis deviation
- pathological Q3
- negative T3
- deep S1

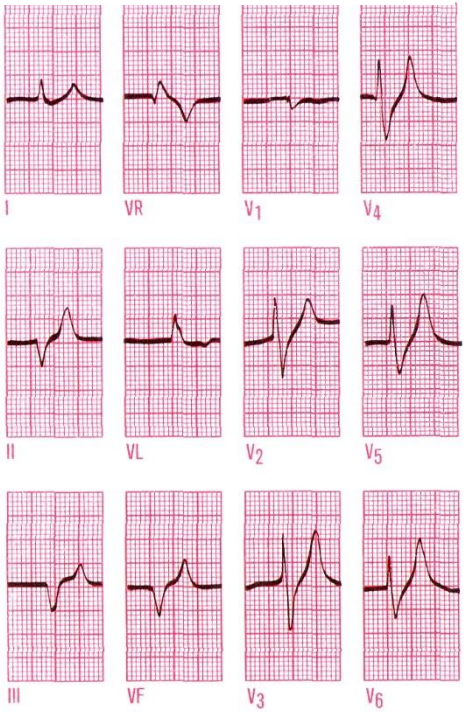




Pulmonary embolism - development

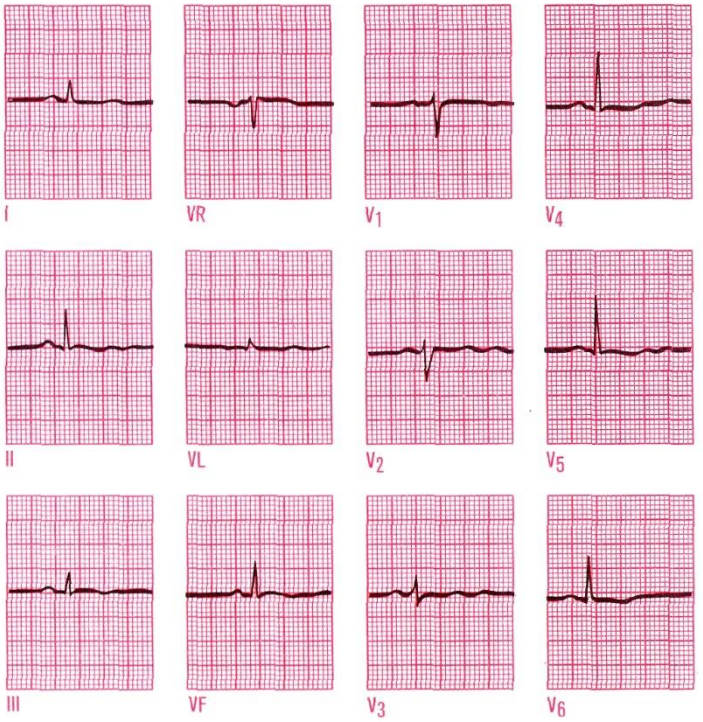
IONIC IMBALANCE - K, Ca

hyperkaliaemia



shortage of depolarisation,
high pointed T,
absence of P

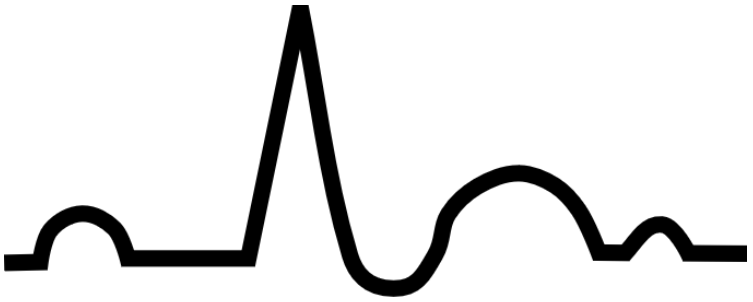
hypokaliaemia



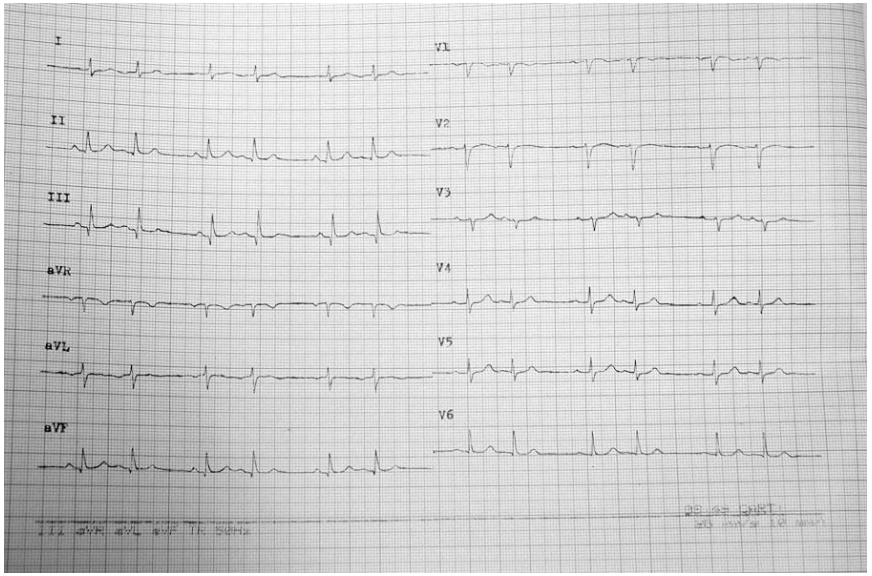
flat T, U wave

INFLUENCE OF PHARMACS - digitalis

Normal therapeutically levels:
- PQ elongation
- QTc shortage
- navicular ST segment
- flat up to negative T wave
- possibility of U wave appearance



Digitalis intoxication:
- various types of AV block
- bigeminia
- tachycardia
- atrial fibrillation



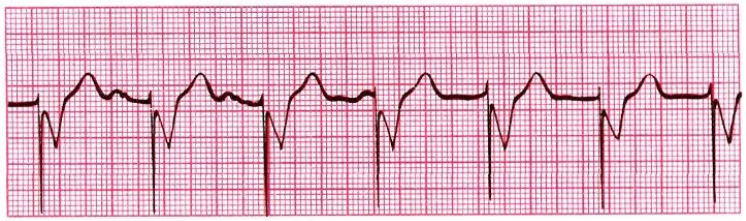
supraventricular bigeminia

Systemic diseases

- hyperthyreosis - tachycardia , high bifidic T, fibrillation , atrial flutter
- hypothyreosis - bradycardia , negative T, effusion – low voltage
- anaemia - ST depression, flat up to negative T
- malignities (with heart metastasis) - atrial fibrillation, AV block, possible effusion

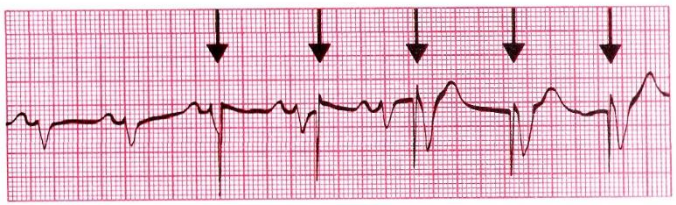
Brain haemorrhage - changes especially in the region ST - T

Stimulated rhythm



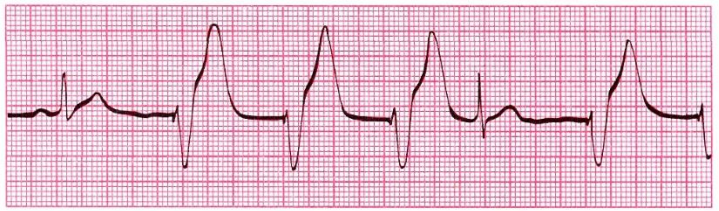
Stimulated rhythm

Cardiostimulation with firm frequency stimulator switched on

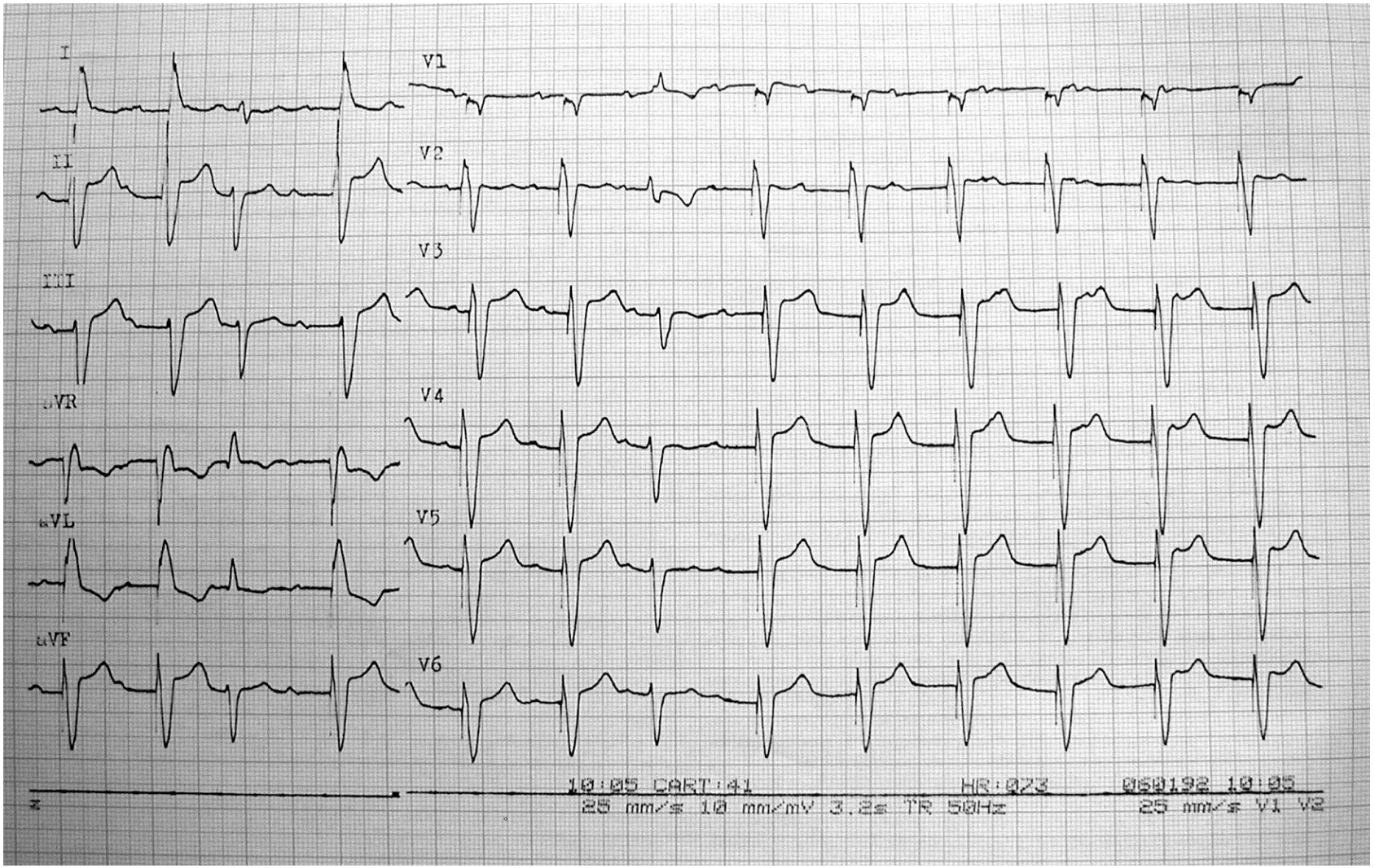


Beginning interferrence with sinusal QRS

Cardiostimulation „on demand“



activation of the stimulator after the drop out of sinus rhythm
inhibition by sinusal contraction, repeated activation

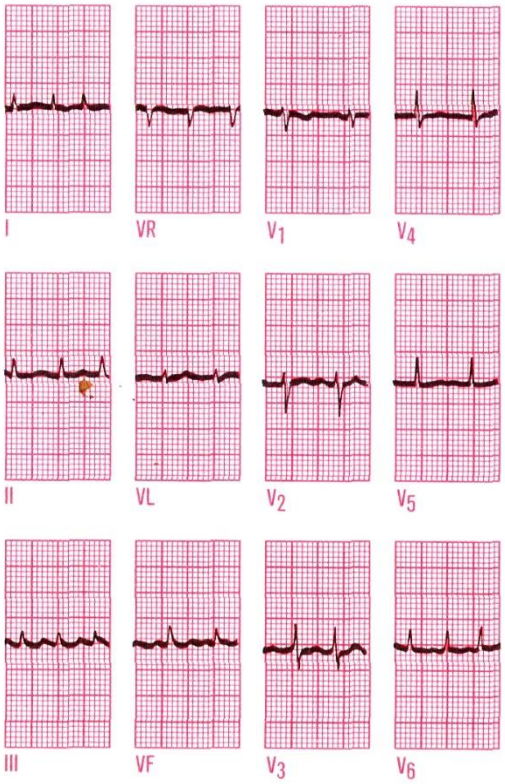


Stimulated rhythm

LOW VOLTAGE

- obezity
- pericardial, pleural effusion – pericarditis, neoplasm, TBC
- processes substituting the heart musculature – amyloidosis, sarcoidosis, multiple IM cicatrix

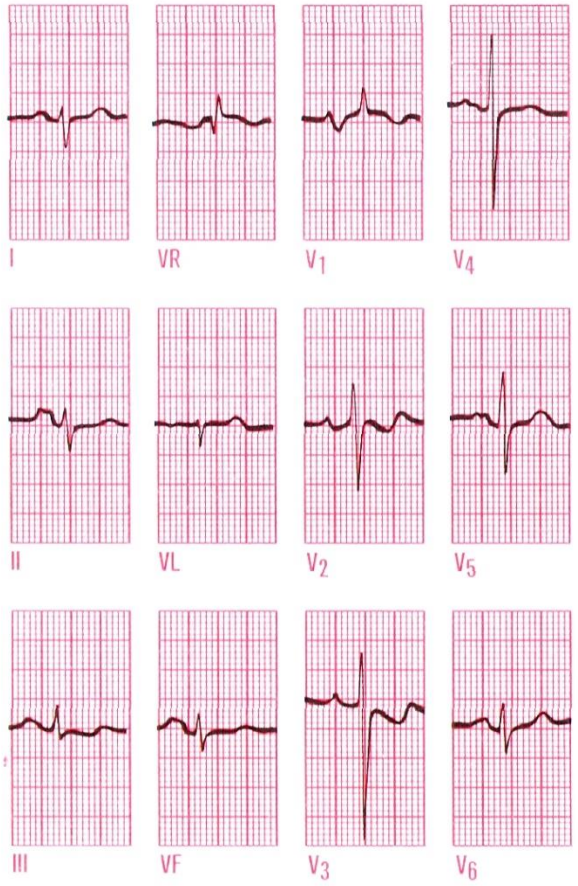
Pericardial effusion by neoplasm



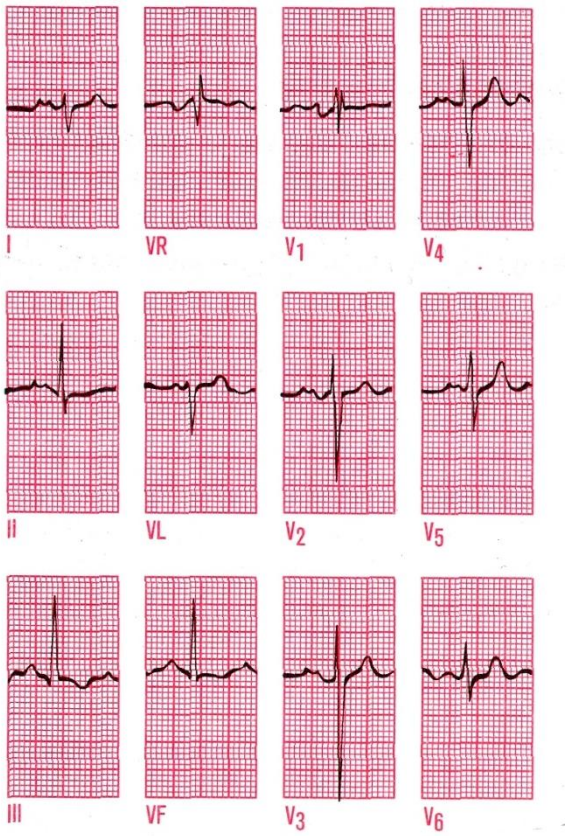
HIGH VOLTAGE

- in astenics
- hypertrophy of ventricles

Left atrium and right ventricle hypertrophy by mitral stenosis

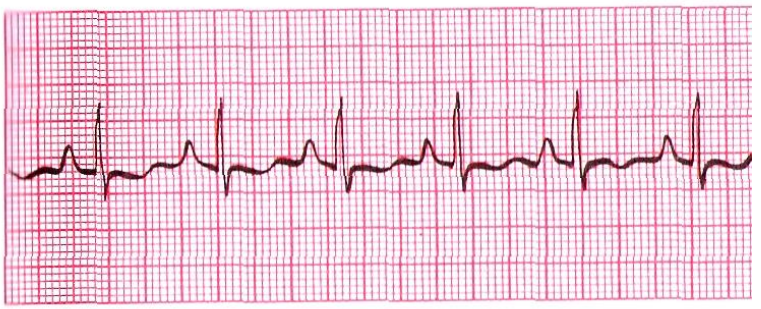


P mitrale - bifidic P - due to hypertrophy of the left atrium - mitral disorders

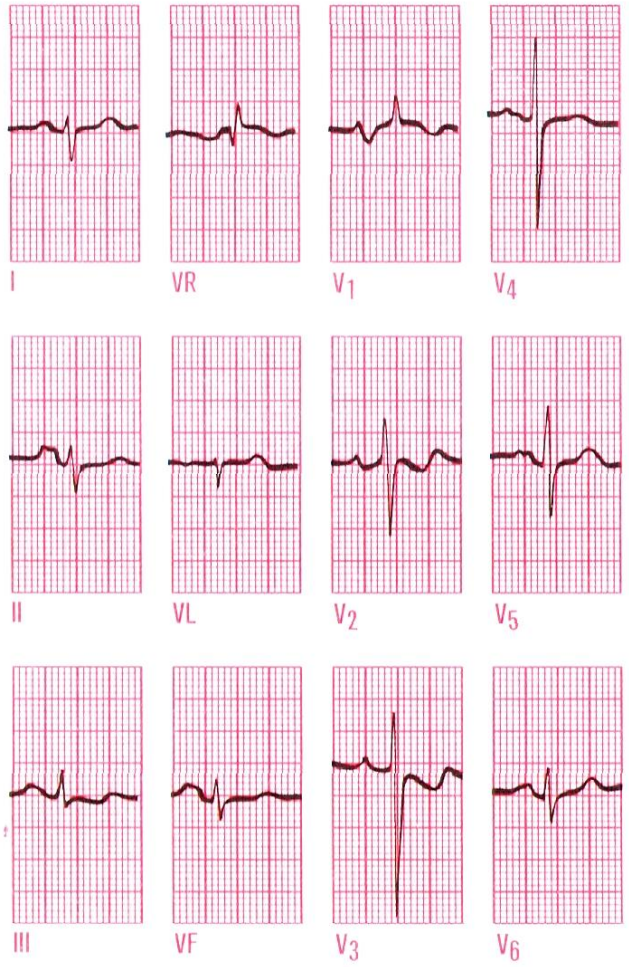


mitral stenosis

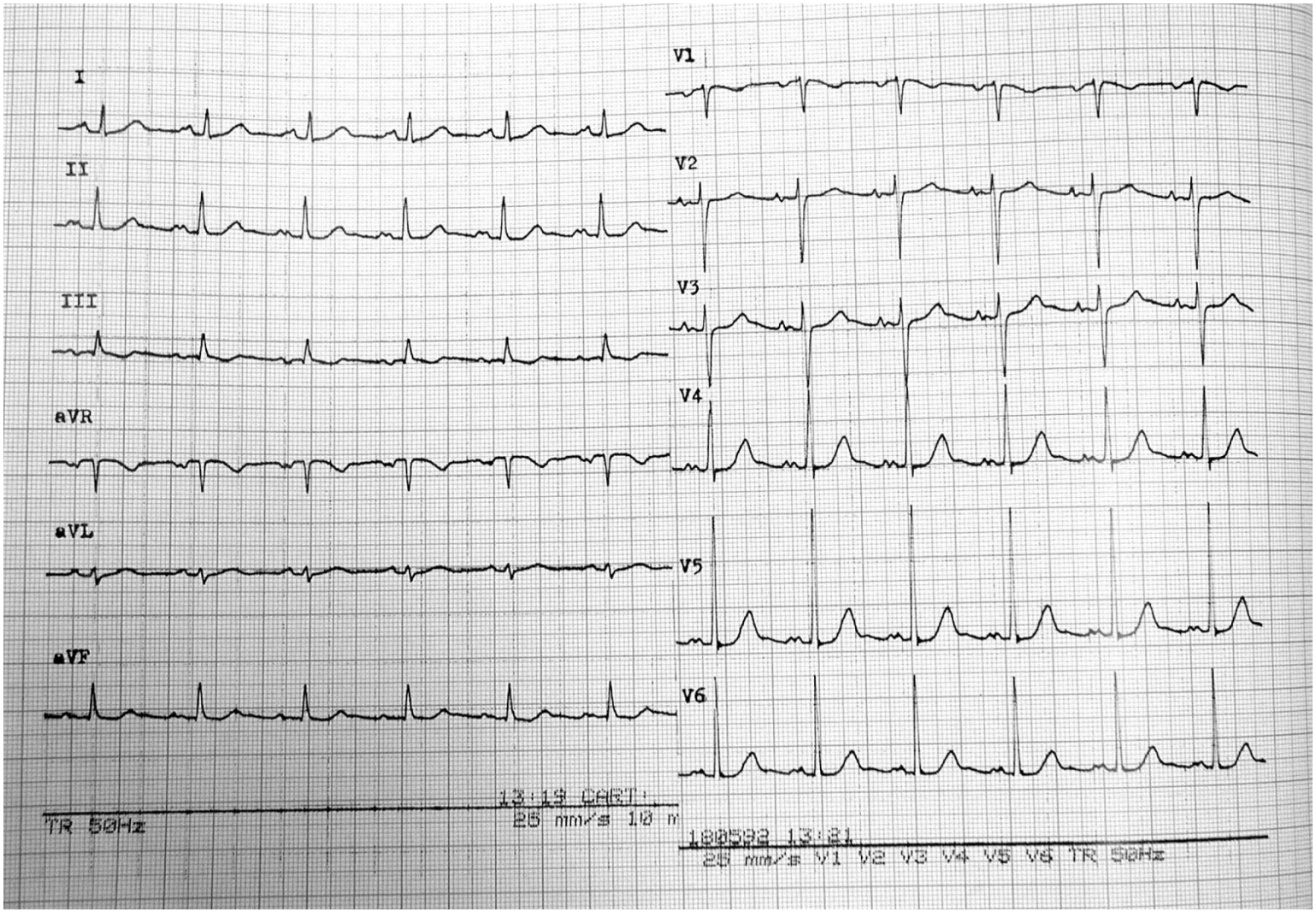
P pulmonale - high sharp P - due to overload or hypertrophy of the right heart



Stenosis of tricuspidal valve – P pulmonale



P mitrale, left axis deviation due to right ventricle hypertrophy, mitral stenosis


I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
TR 50Hz
25 mm/s 10 m
180592 13:21
25 mm/s V1 V2 V3 V4 V5 V6 TR 50Hz

P mitrale