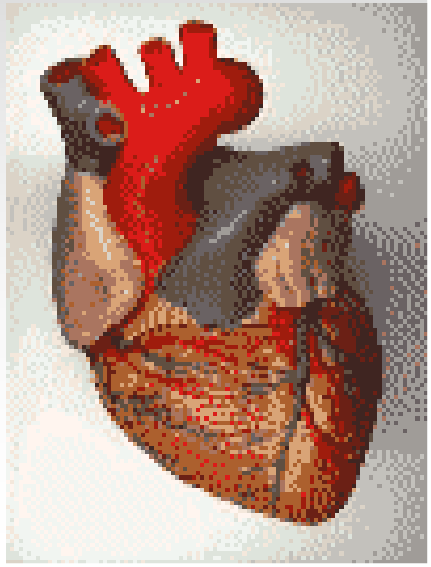
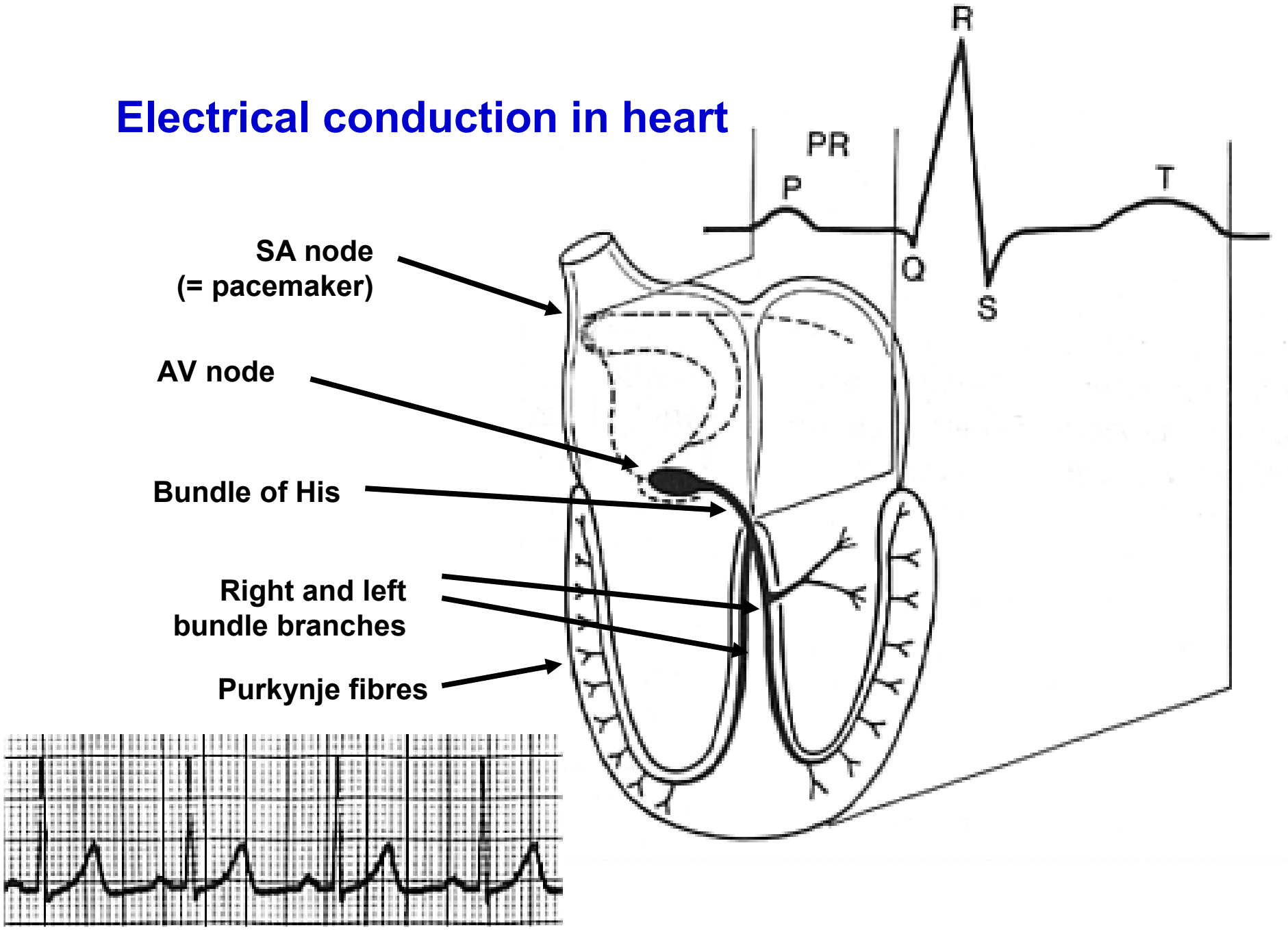
ECG EXAMINATION

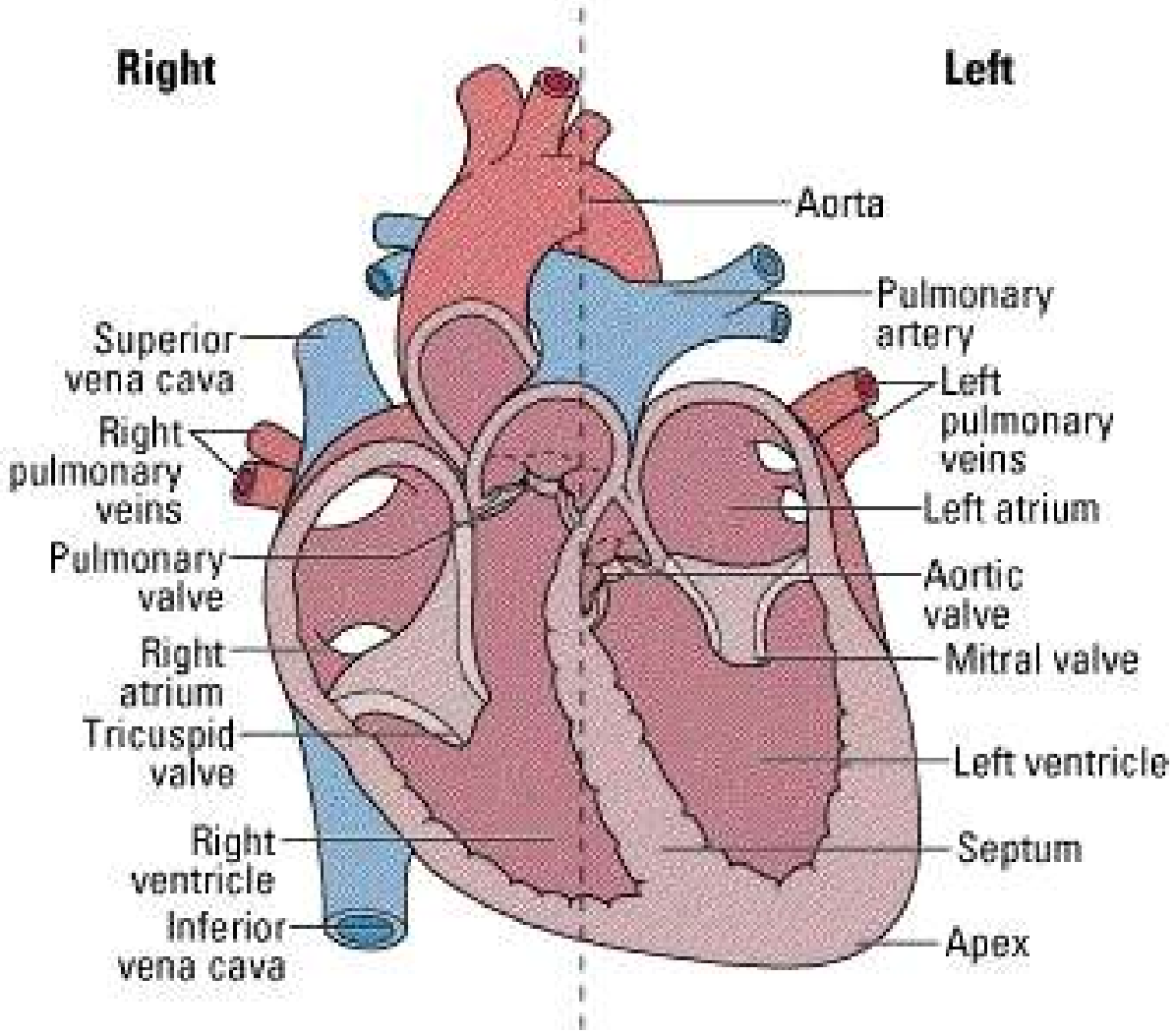
Seminar of pathophysiology

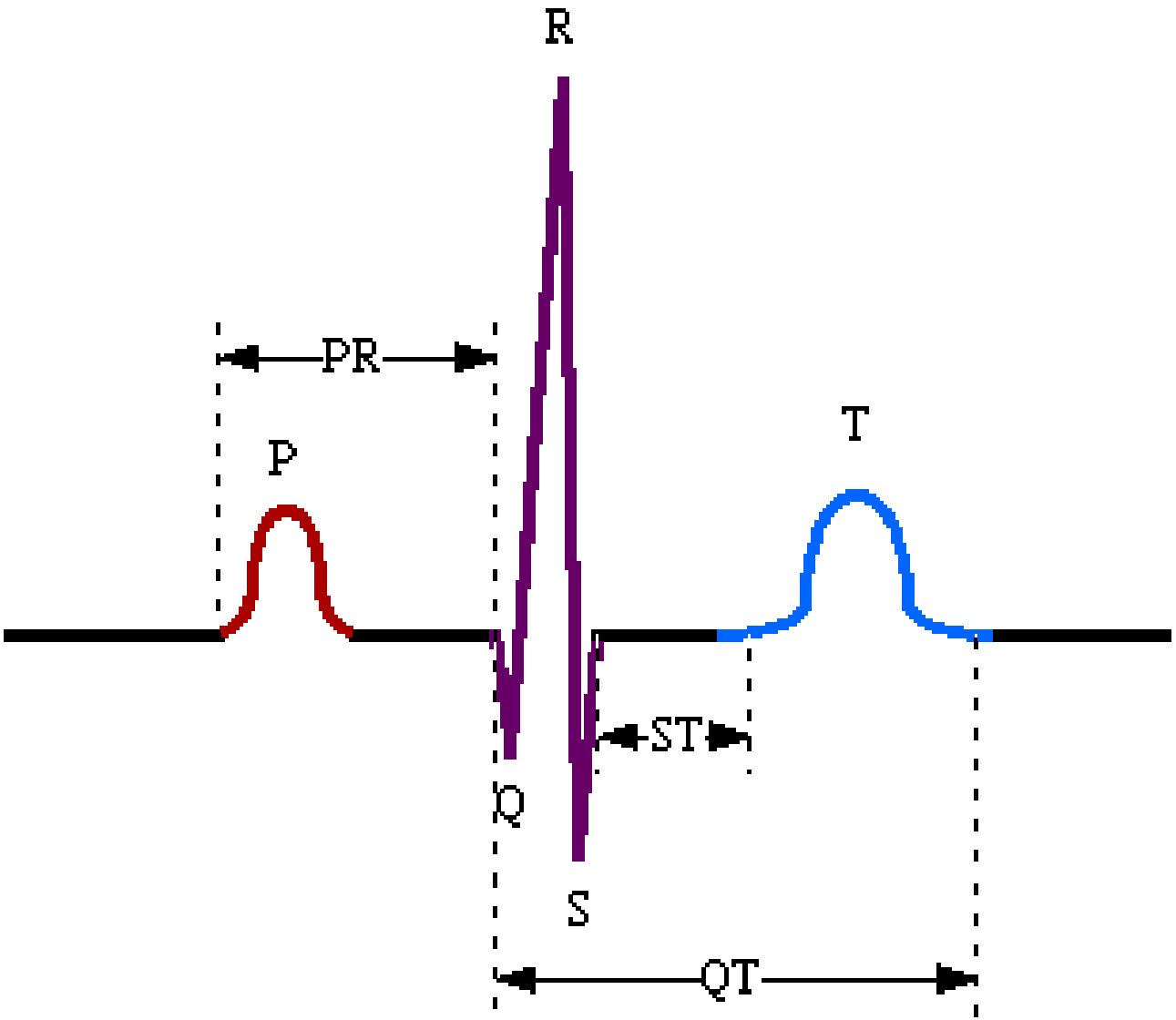
Pavel Maruna



Electrical conduction in heart

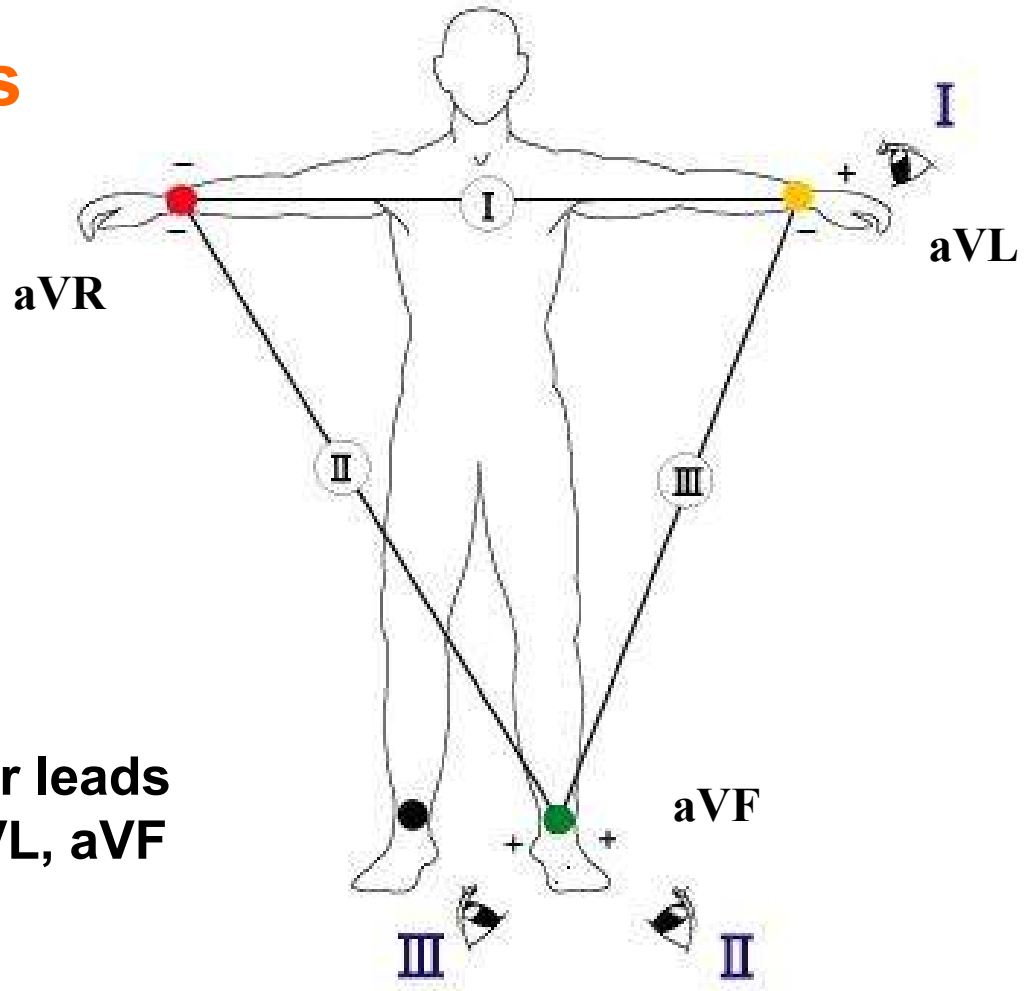



Right
Left
Aorta
Pulmonary artery
Superior vena cava
Left pulmonary veins
Right pulmonary veins
Left atrium
Pulmonary valve
Aortic valve
Right atrium
Mitral valve
Tricuspid valve
Left ventricle
Right ventricle
Septum
Inferior vena cava
Apex


R
PR
P
T
ST
Q
S
QT

12-lead ECG examination

Limb leads



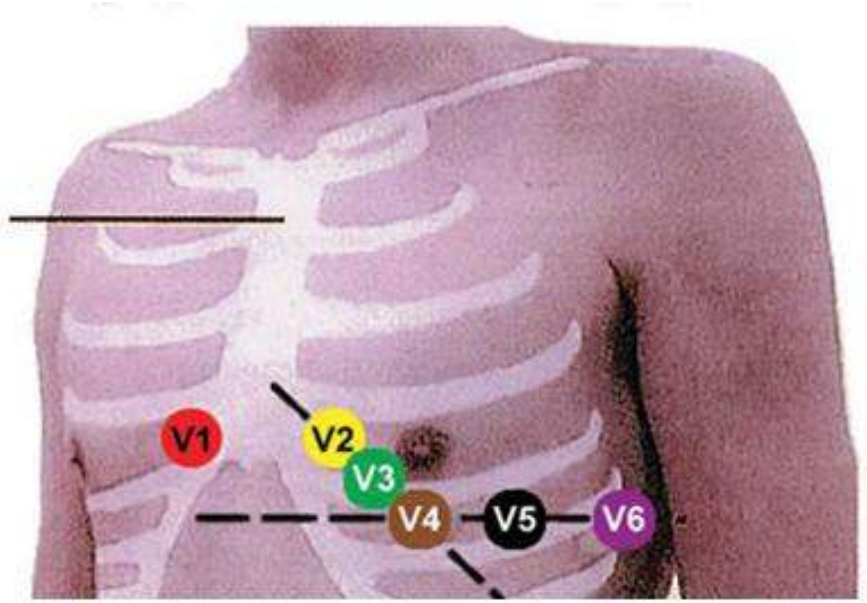
Bipolar leads
I, II, III

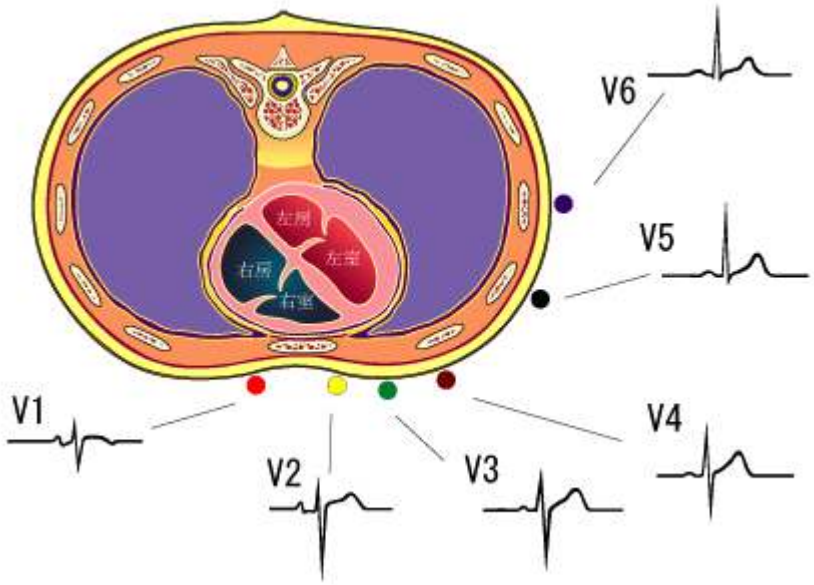
Pseudounipolar leads
aVR, aVL, aVF

12-lead ECG electrodes placement

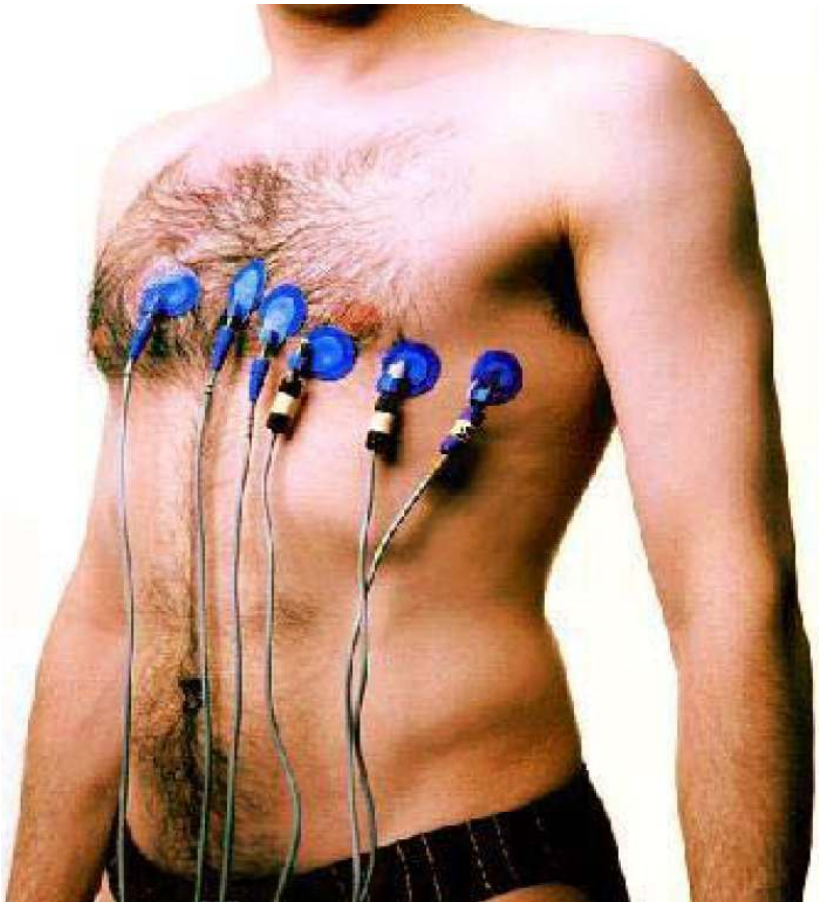
Chest leads

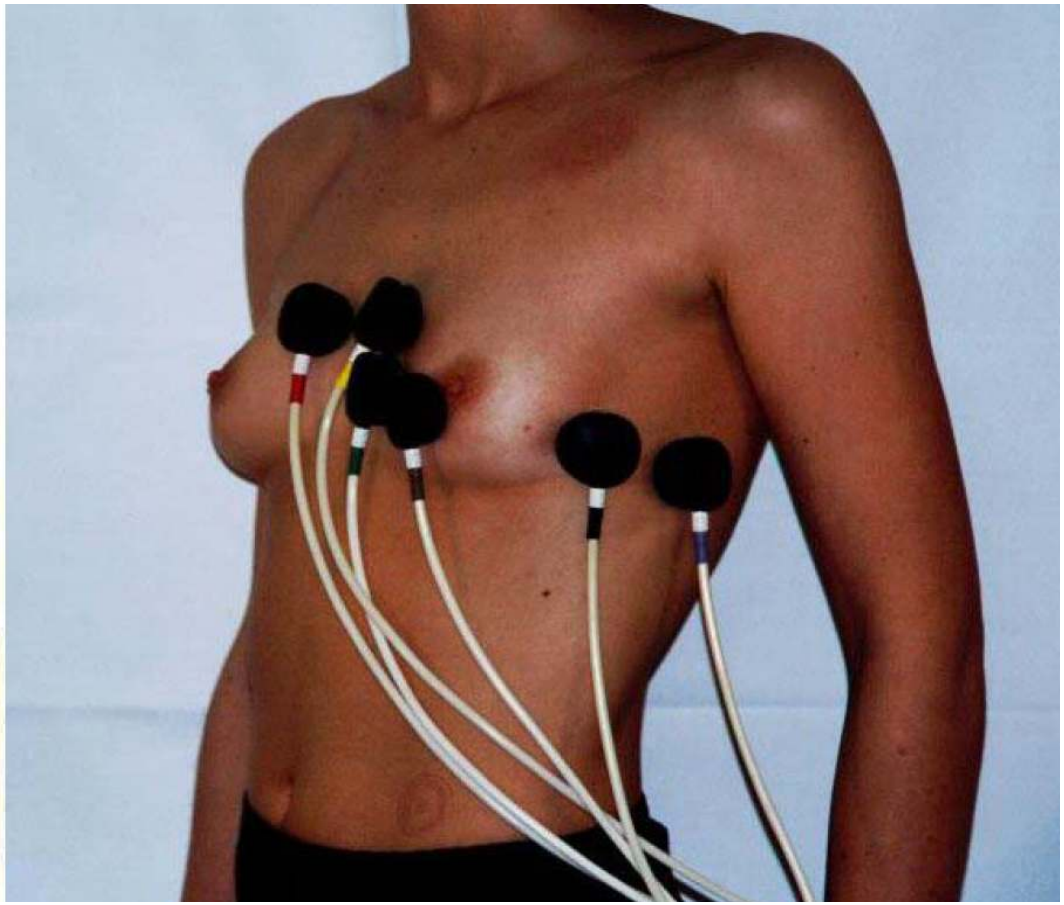
Unipolar leads



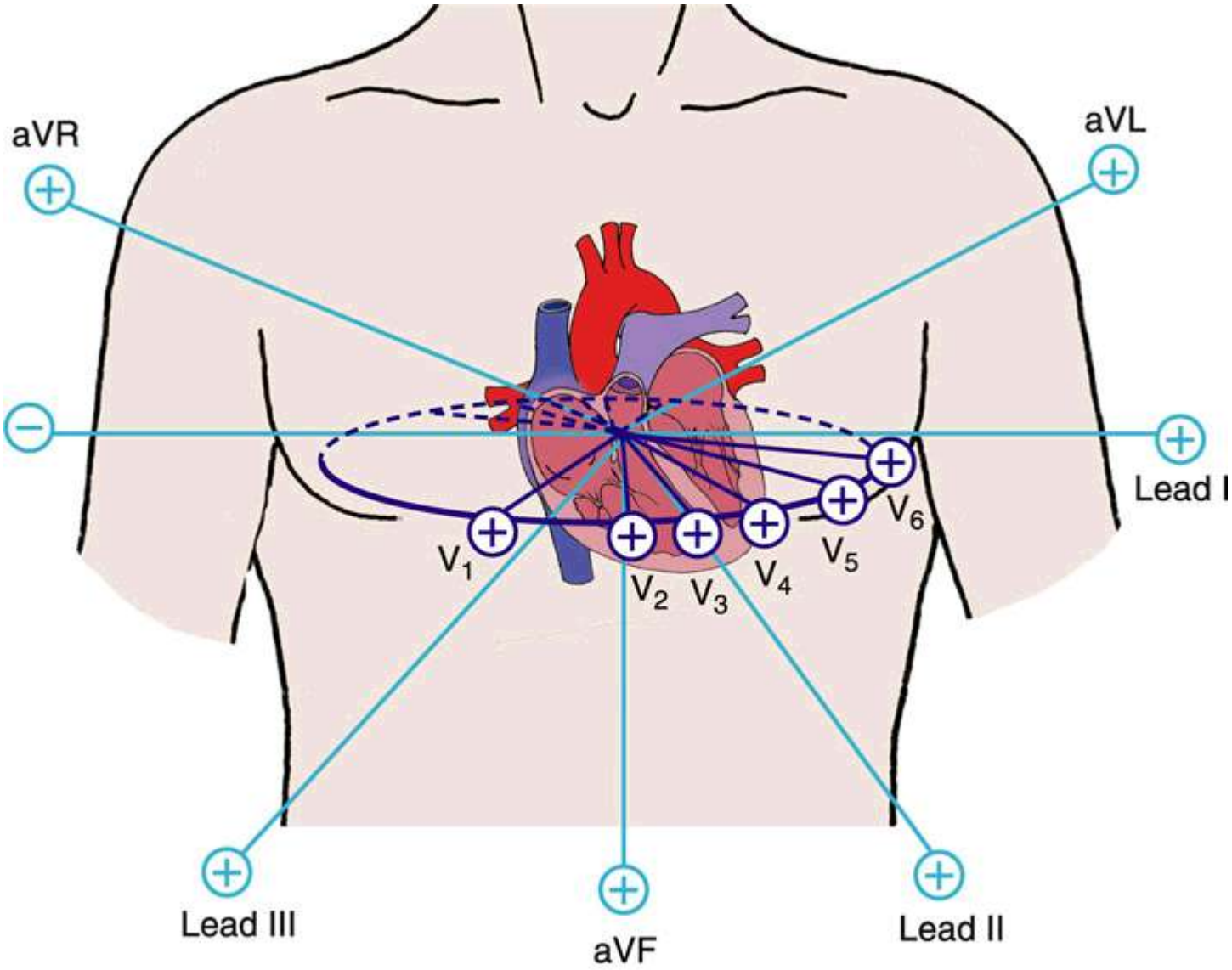


12-lead ECG electrodes placement



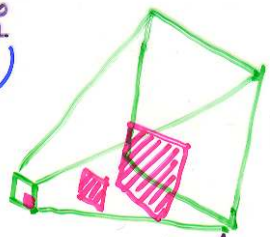


12-lead ECG examination

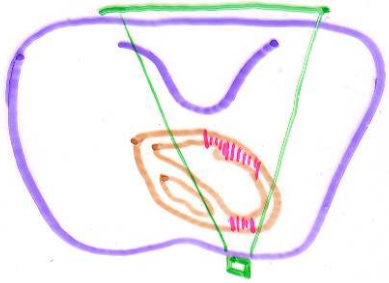


EKG PARADIGMATA

i) projekce svodů (lead projection)

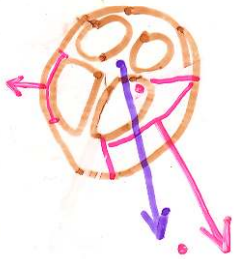


$S = k \cdot \frac{1}{r^2}$

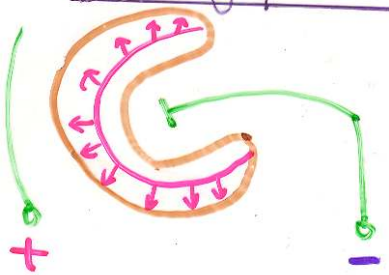


končetinové = globální, distanční (limb L. are global, distance)
hrudní = lokální, blízké (chest L. are local, near)

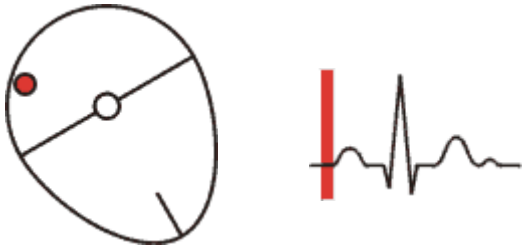
ii) převaha levé komory (left ventricle dominance)



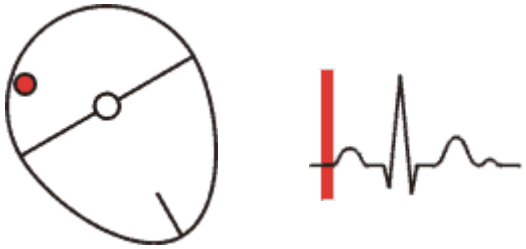
iii) dutinový potenciál (cavity potential)



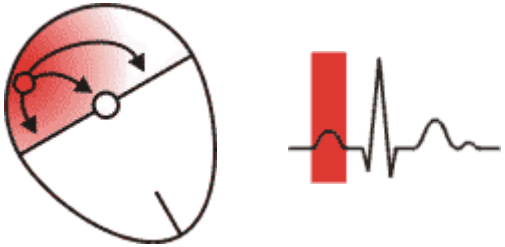
ECG paradigms



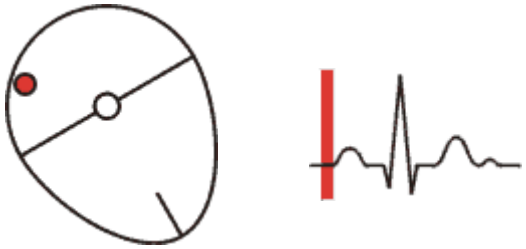
initiation of impulse in the SA node



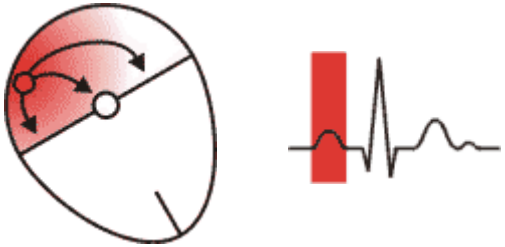
initiation of impulse in the SA node



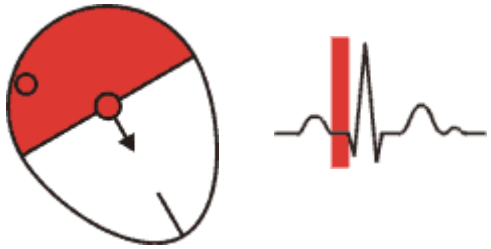
atrial depolarization



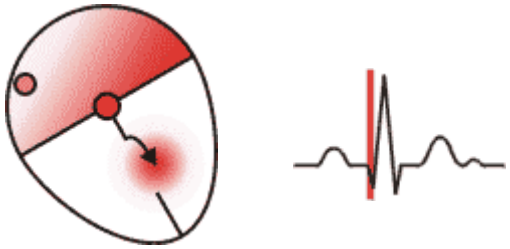
initiation of impulse in the SA node



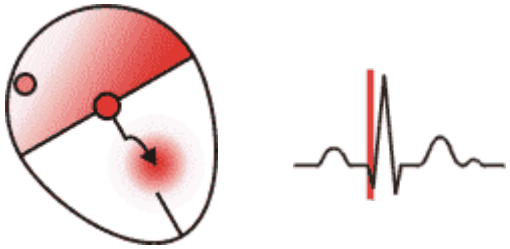
atrial depolarization



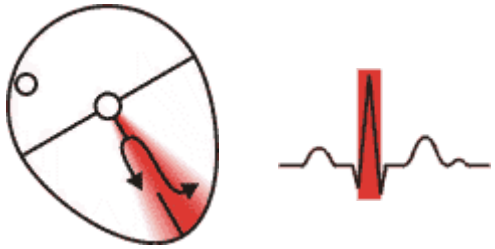
depolarization of AV nodus and bundle of His



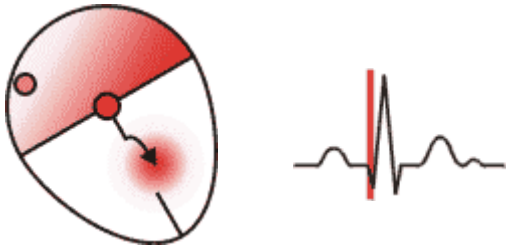
septal depolarization



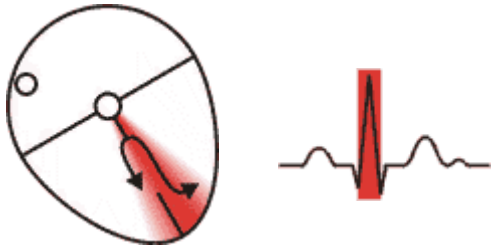
septal depolarization



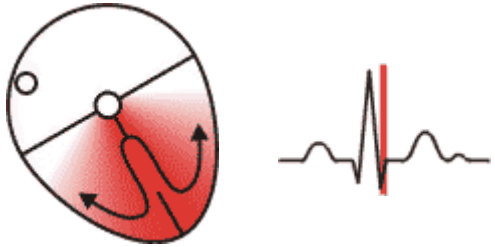
early ventricular depolarization



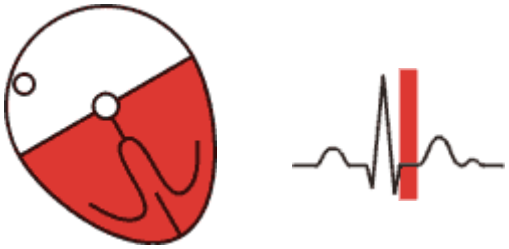
septal depolarization



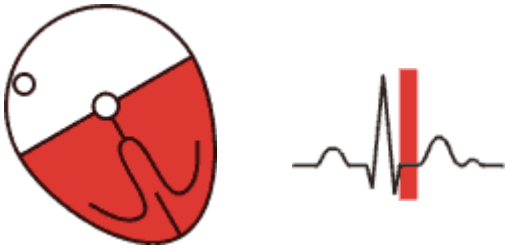
early ventricular depolarization



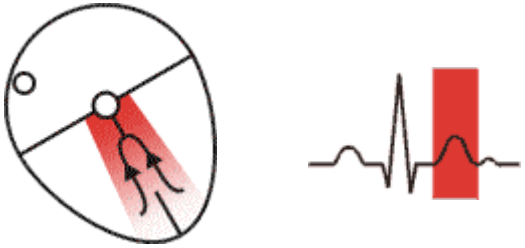
late ventricular depolarization



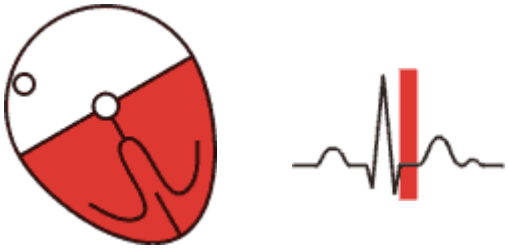
ventricular systole



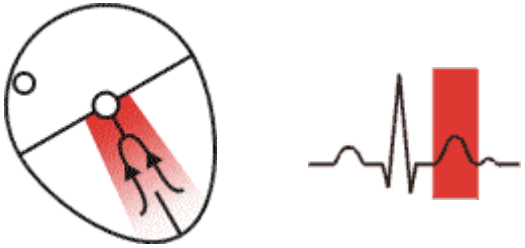
ventricular systole



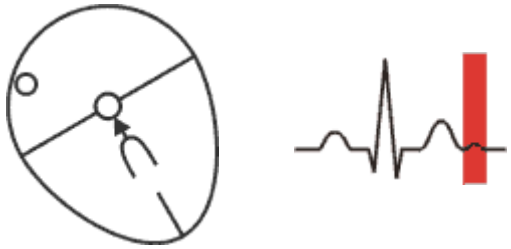
ventricular repolarization



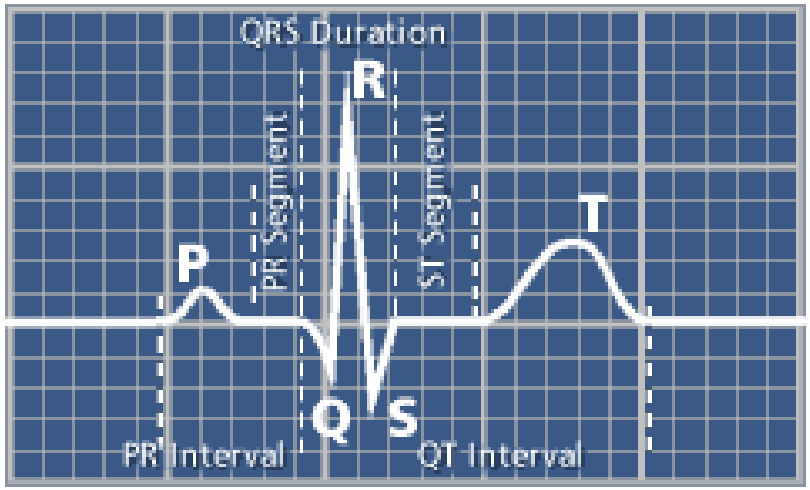
ventricular systole

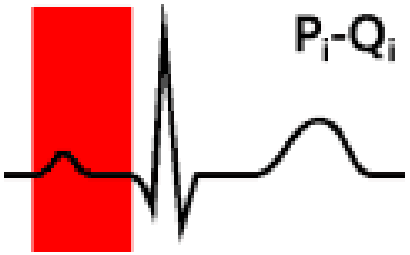


ventricular repolarization

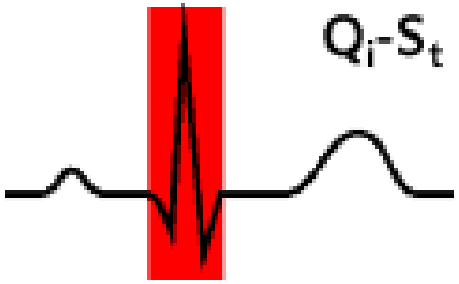


repolarization of bundle of His

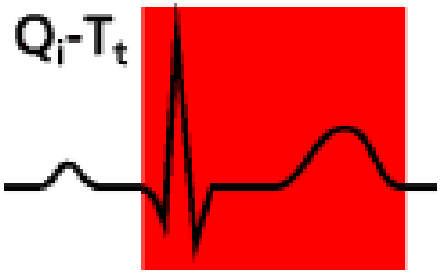




PR (PQ) interval
(0,12 - 0,20 s)



QRS complex
(0,06 - 0,10 s)

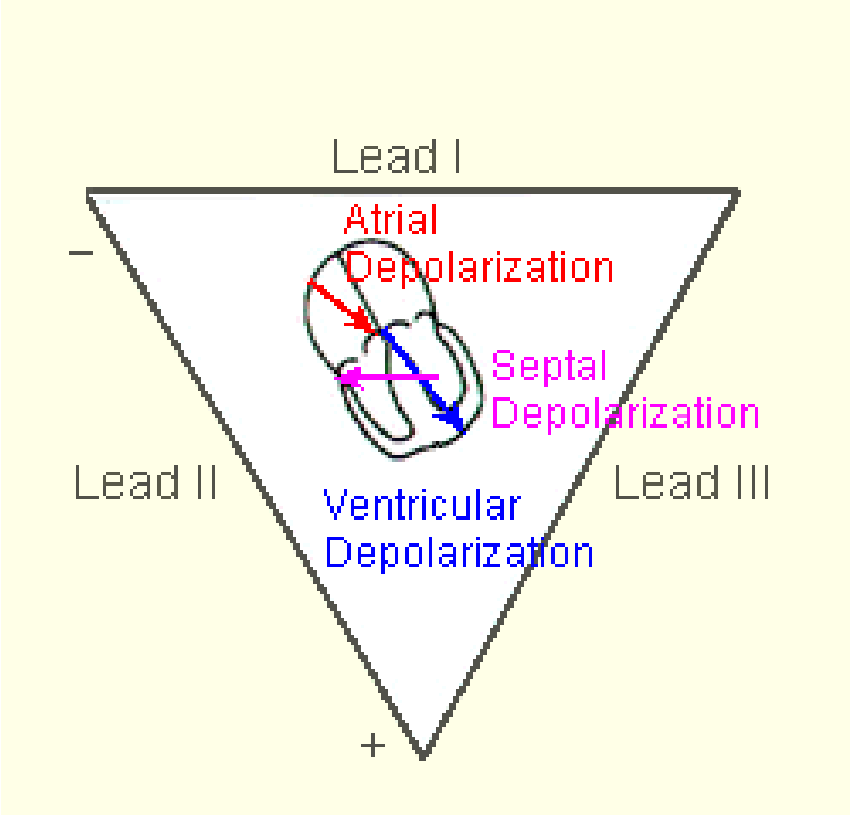


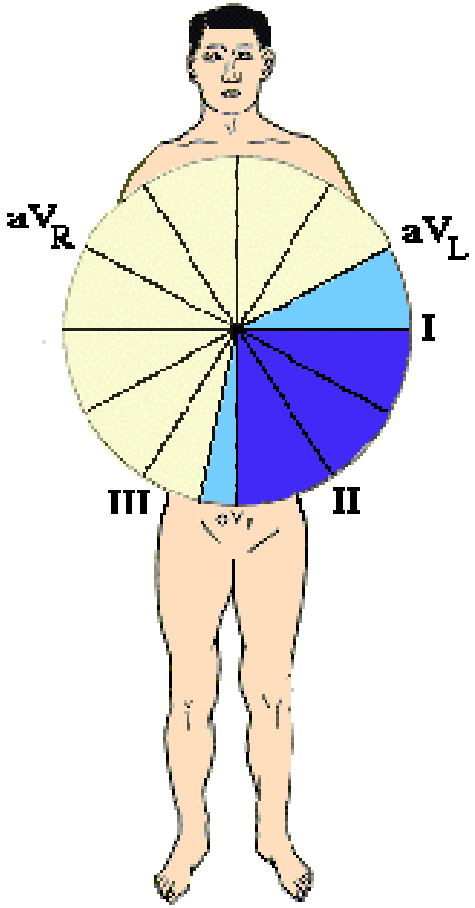
QTc interval
(to 0,44 s)

QRS axis

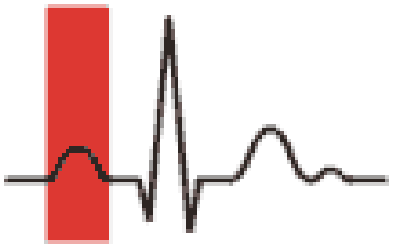
(Axis of QRS complex depolarization)

- 30° to + 105°





P wave



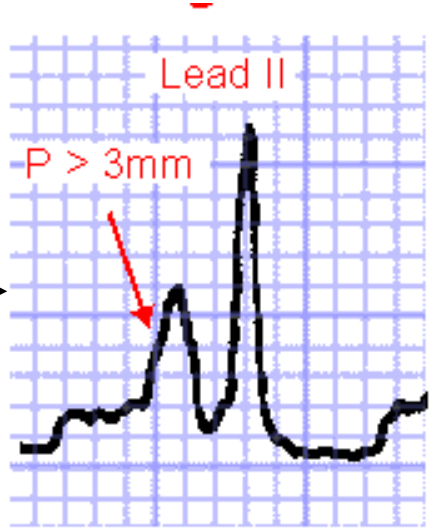
Electrical impulse originates in the SA node
Impulse triggers atrial depolarization

Physiology:

- **positive orientation (possible biphasic in I lea**
- **duration < 0,11 s, amplitude < 2,5 mm**

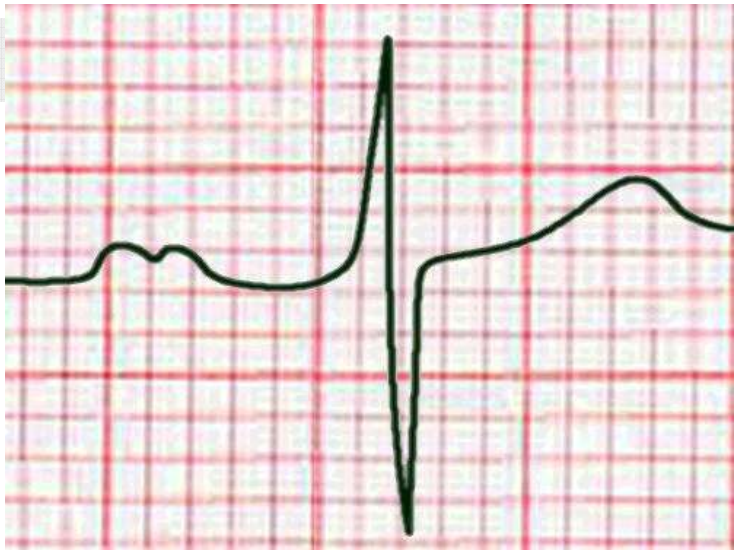
Pathology:

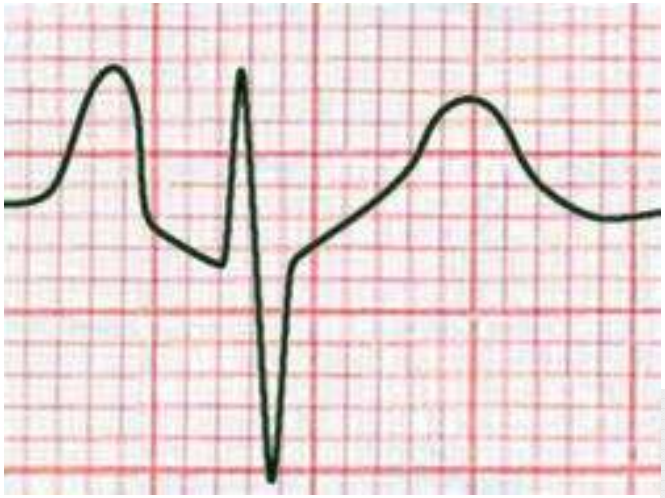
- **hypertrophy of left or right atrium**
- **abnormal conduction (SVES)**



P wave

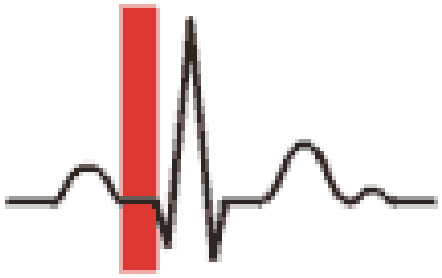
P mitrale





High P amplitude due to left atrial hypertrophy

PR (PQ) interval



depolarization of AV node and bundle of His

It represents the physiological delay in conduction from atrial depolarization to the beginning of ventricular depolarization. It is electrically neutral.

Limits: 0,12 - 0,20 s

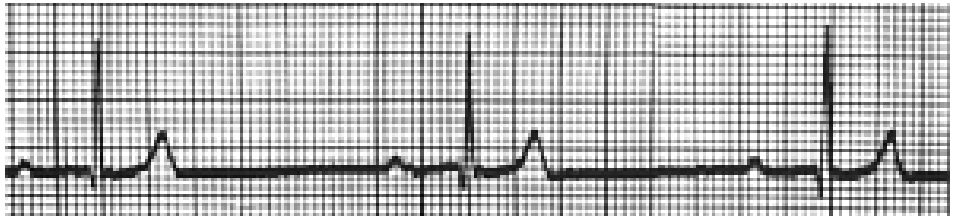
Physiological importance:

1. synchronization of both atrial and ventric. systoles
2. protection against the transmission of supraventricular tachyarrhythmia to vetricular tachycardia

PR (PQ) interval

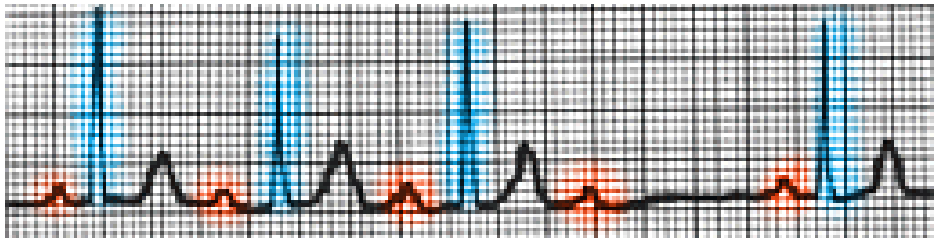
AV blockade

1st = prolongation of PR interval

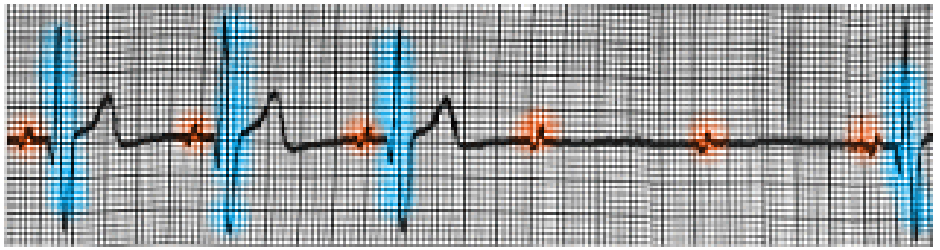


2nd = partial blockade (transmission of selected impulses)

- Wenckenbach



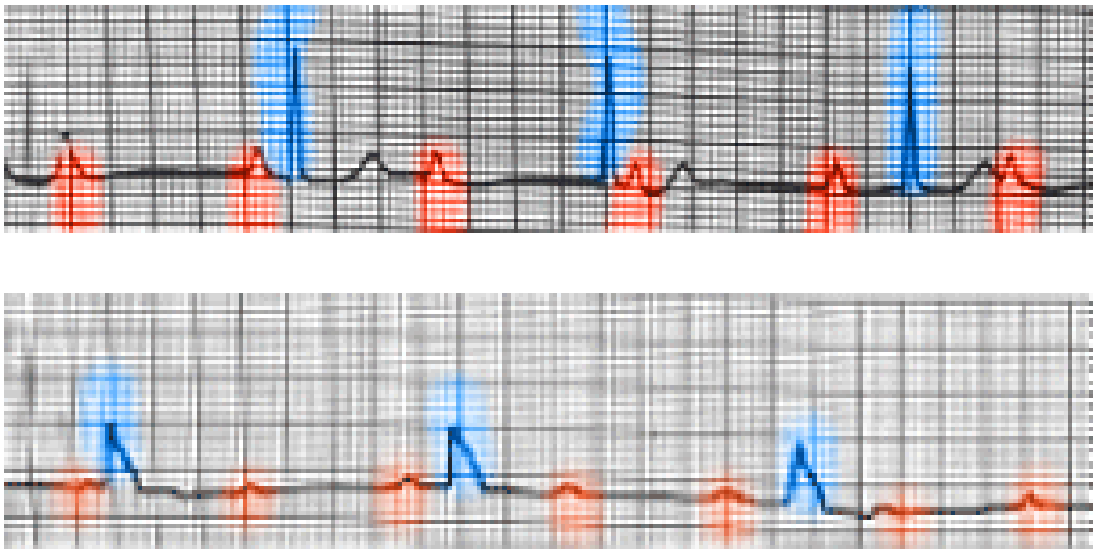
- Mobitz



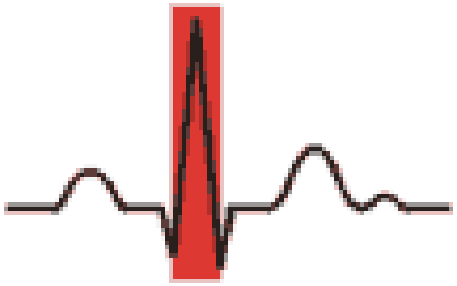
PR (PQ) interval

AV blockade

3rd = complete blockade



QRS complex

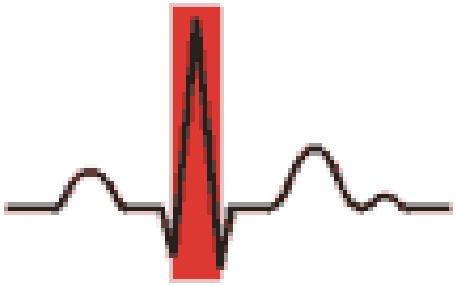


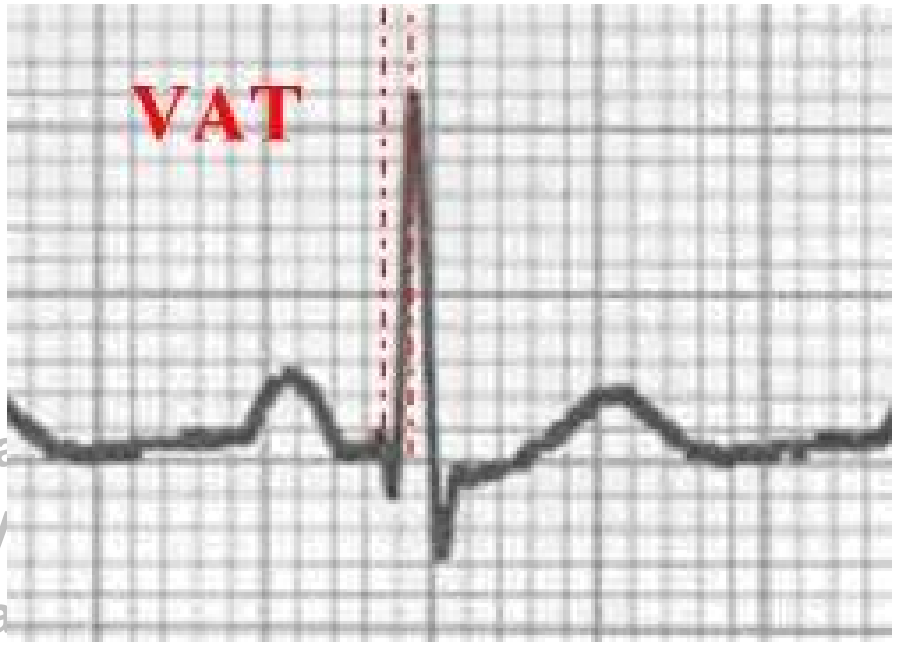
Depolarization of the ventricles

Physiology:

- **duration 0,06 - 0,10 s**
- **Q < 0,04 s, < 25 % of R wave**
- **Sokolow index (S in V2 + R in V5) < 35 mm (< 45 mm for young)**
- **axis of ventricular depolarization -30 to +105 °**

QRS complex





Physiology:

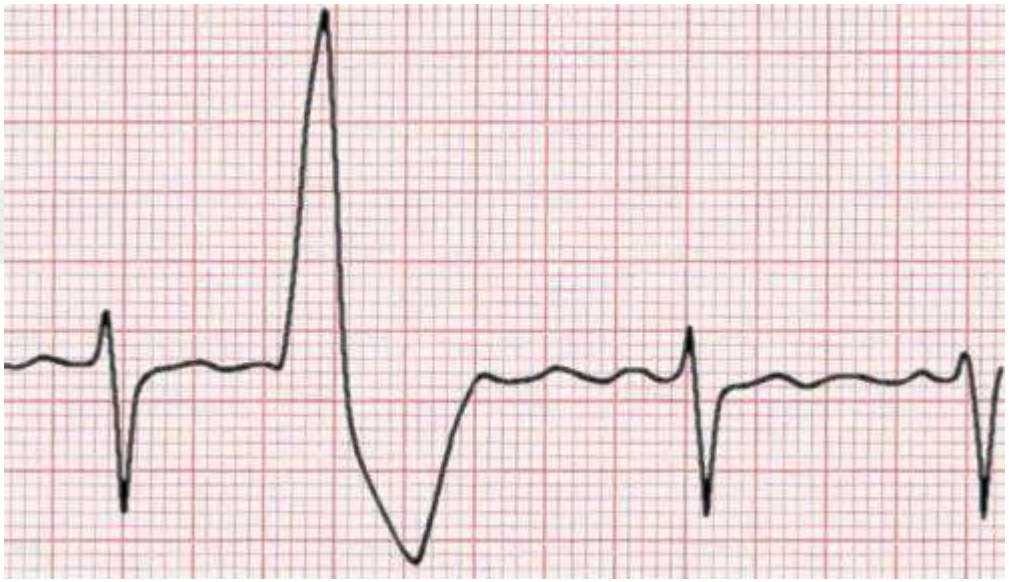
- duration 0,06 - 0,10 s
- Q < 0,04 s, < 25 % of R wa
- Sokolow index (SV2 + RV
- axis of ventricular depola
- **VAT (ventricular activation time) of LV < 0,04 s, RV < 0,03 s**

QRS complex

Both QRS complex duration and shape is depend on:

1. Physiology of His-Purkine´s system or aberrant signal passing

VES



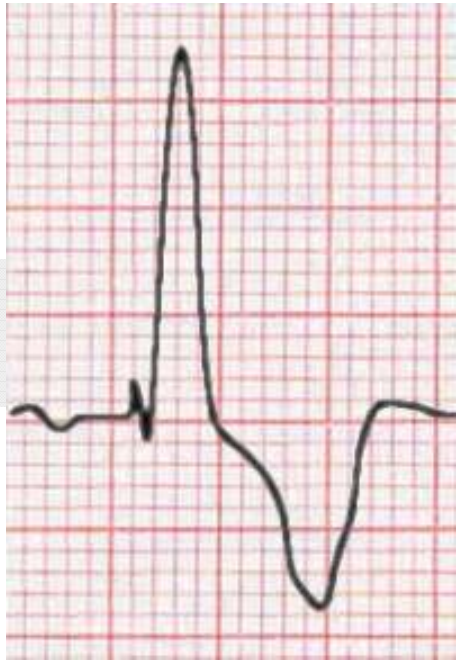
QRS complex

Both QRS complex duration and shape is depend on:

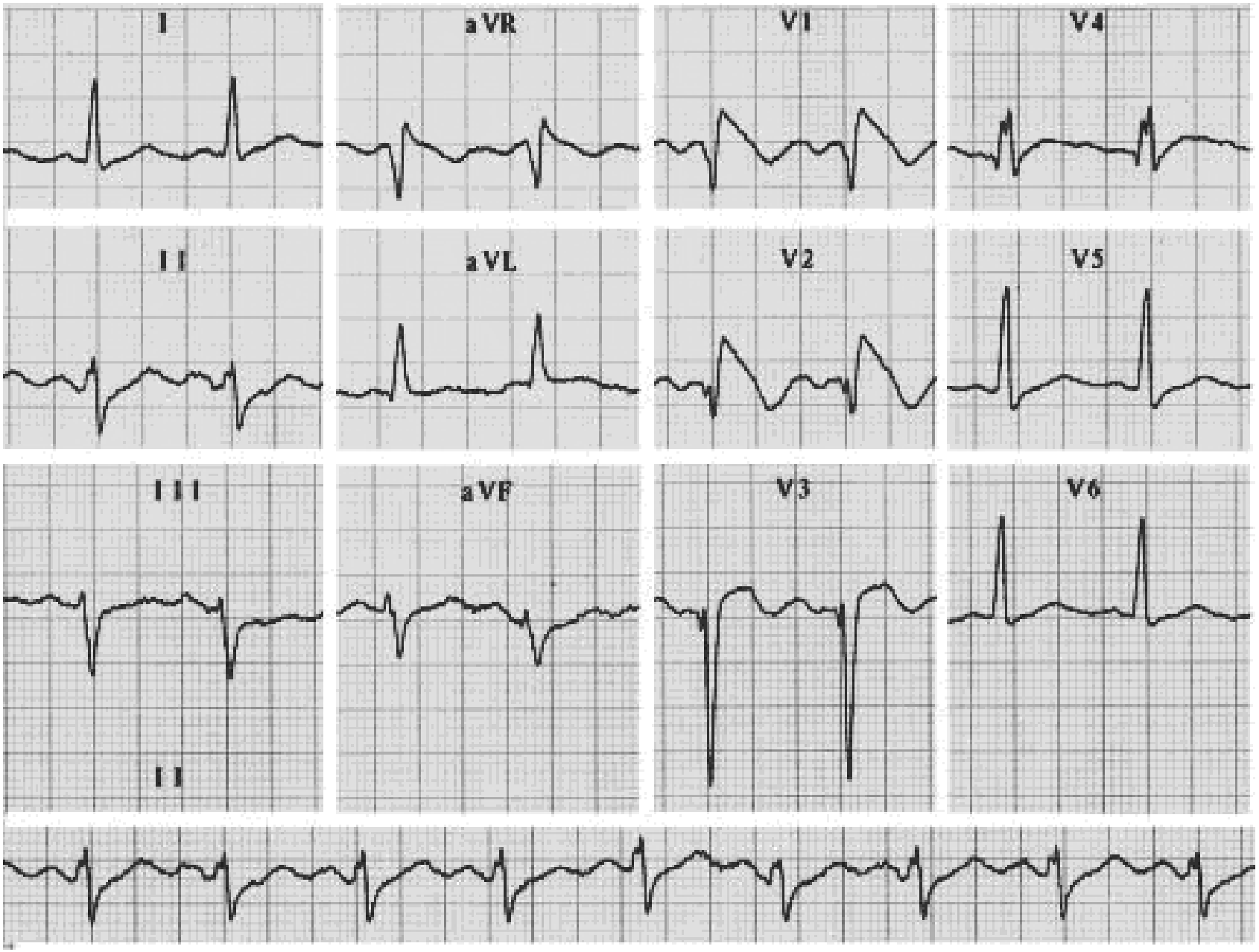
1. Physiology of His-Purkine´s system or aberrant signal passing

Intraventricular blockades

RBBB .....
(QRS prolongation, rSR´ in V1, negative T wave)



V1



RBBB

QRS complex

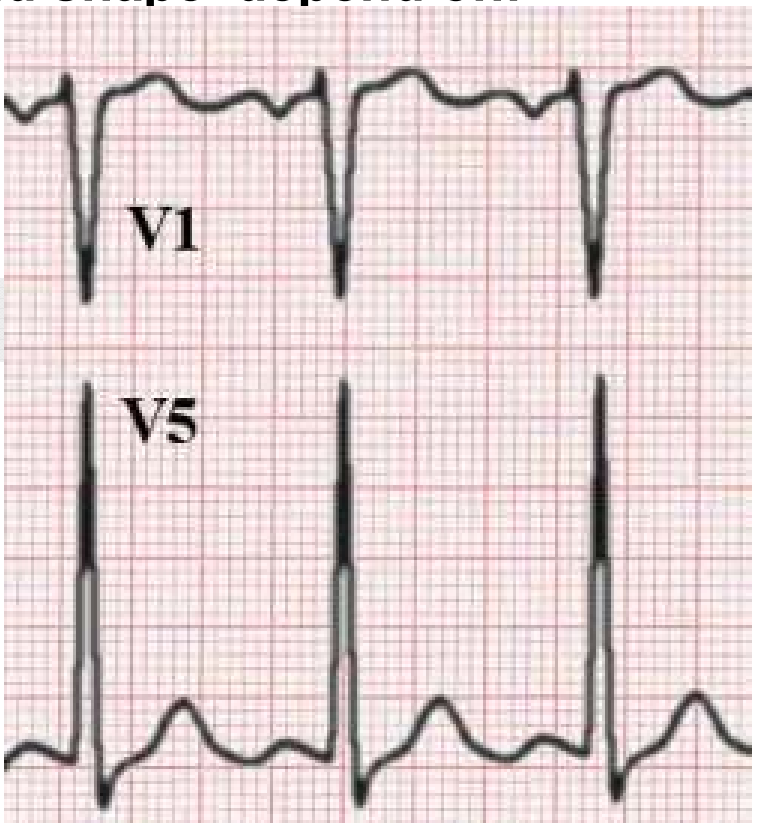
Both QRS complex duration and shape depend on:

2. <u>Myocardial mass</u>

LV hypertrophy

RV hypertrophy

cardiomyopathy

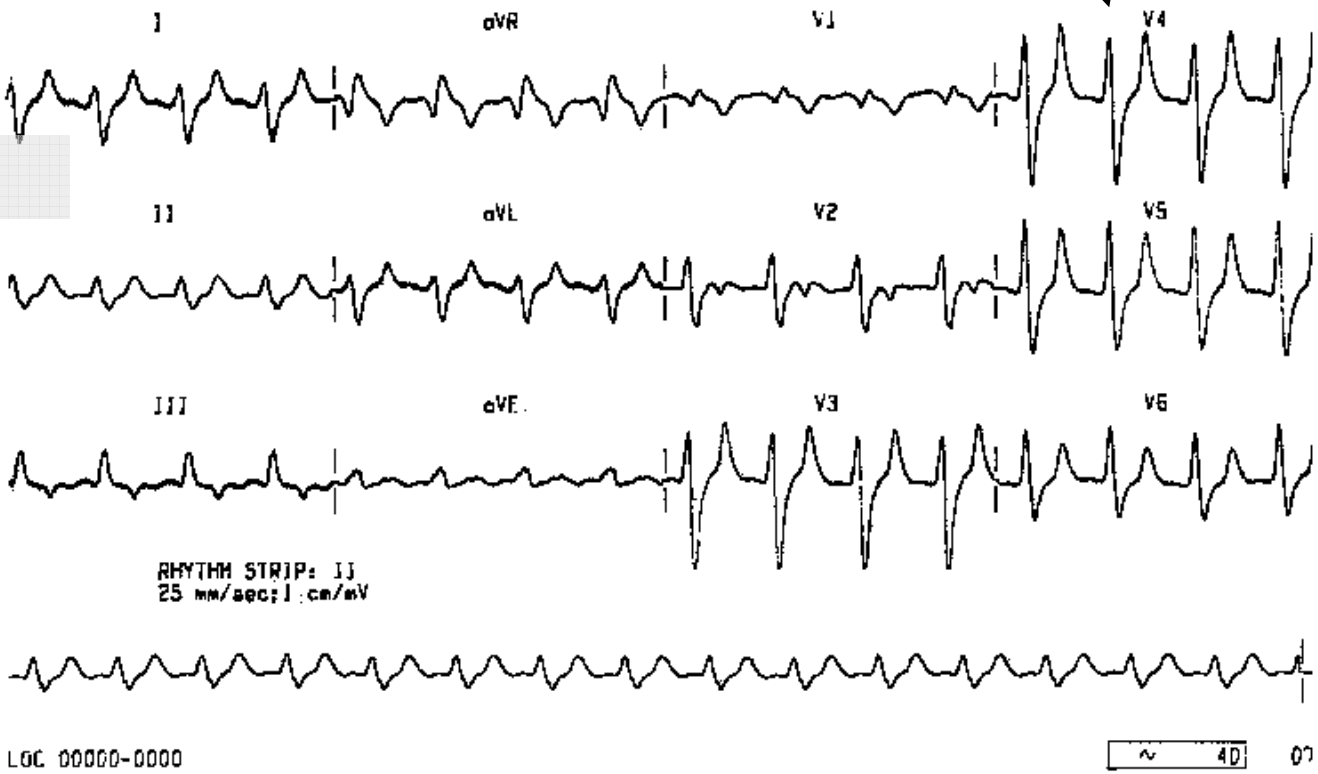


QRS complex

Both QRS complex duration and shape is depend on:

3. Factors affecting signal velocity - metabolic, endocrine, and pharmacological

hyperkalemia

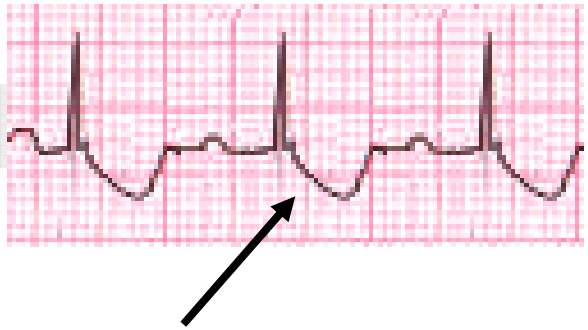


QRS complex

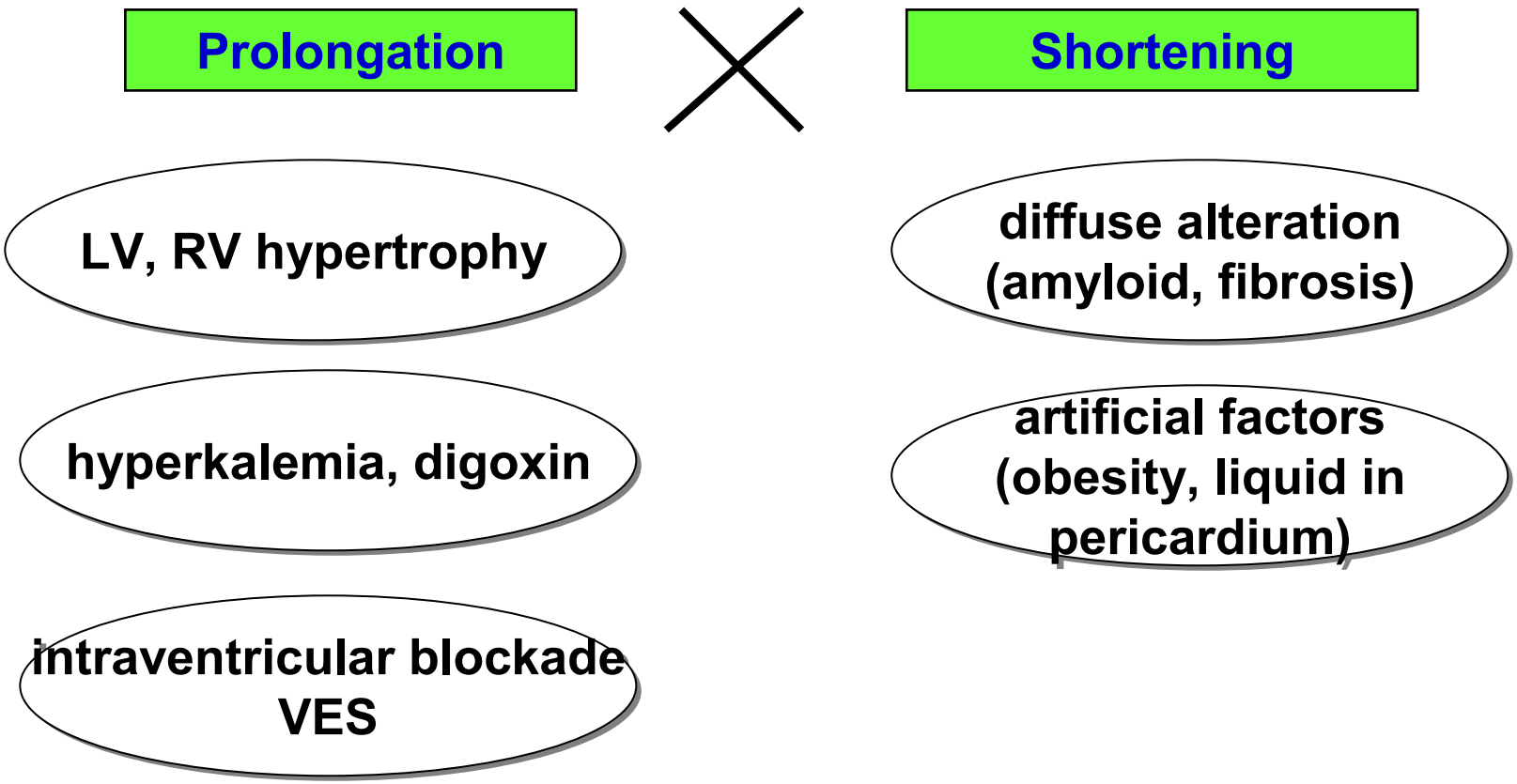
Both QRS complex duration and shape is depend on:

3. Factors affecting signal velocity - metabolic, endocrine, and pharmacological

digitalis



QRS complex


Prolongation
Shortening
LV, RV hypertrophy
hyperkalemia, digoxin
intraventricular blockade
VES
diffuse alteration
(amyloid, fibrosis)
artificial factors
(obesity, liquid in
pericardium)

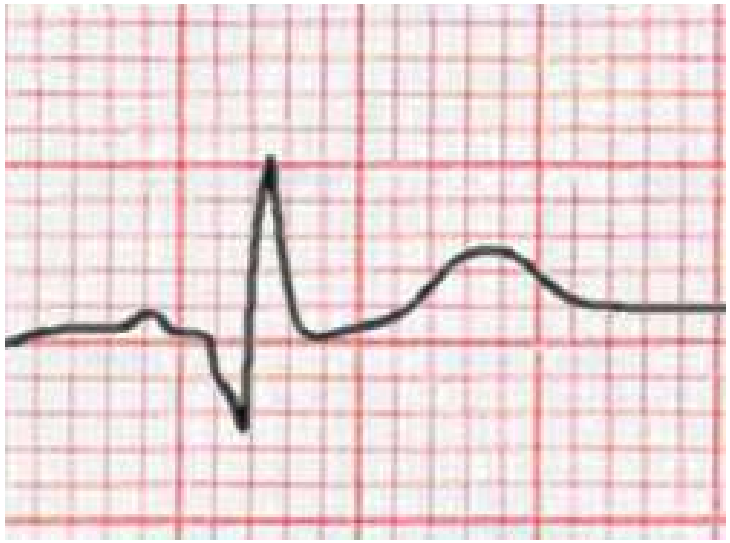
QRS complex

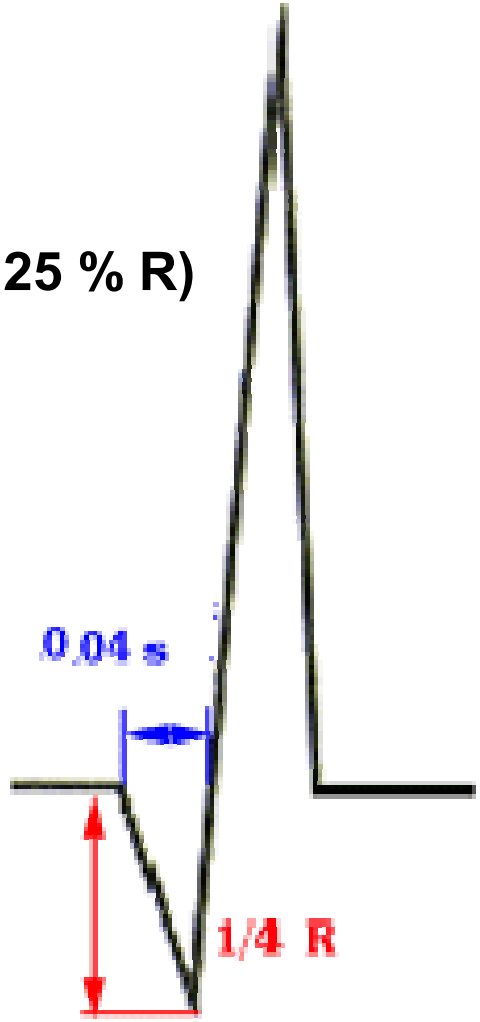
Pathological Q wave

Q wave prolongation (> 0,04 s) and depression (> 25 % R)

Manifestation of transmural myocardial necrosis

„Cavity potential“





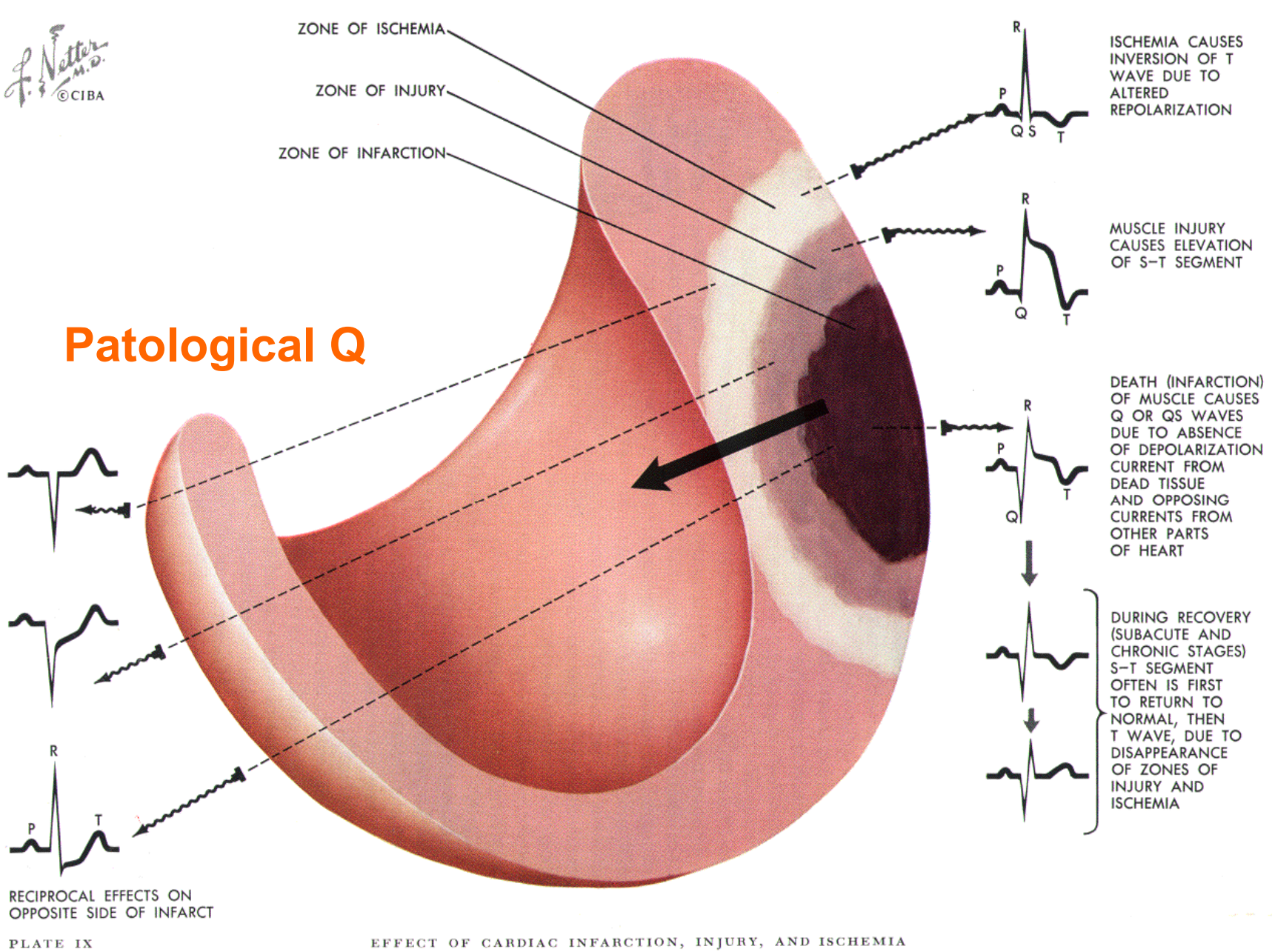
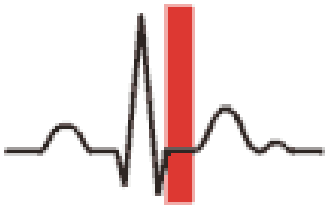


PLATE IX

EFFECT OF CARDIAC INFARCTION, INJURY, AND ISCHEMIA

ST segment



The length between the end of the S wave (end of ventricular depolarization) and the beginning of repolarization

- **From „J point“ on the end of QRS complex, to inclination of T wave**
- **Normally, all cells have the same potential = ST segment is electrically neutral (on isolectric line)**

ST segment

Physiological changes

sympathicus ... ST depression, „anchor-like“ curve
parasympathicus (vagus) ... ST elevation
syndrome of an early repolarization

Artificial changes

depend on lead localization, chest malformation etc.

Pathological changes

electric potential of destroyed myocardial area

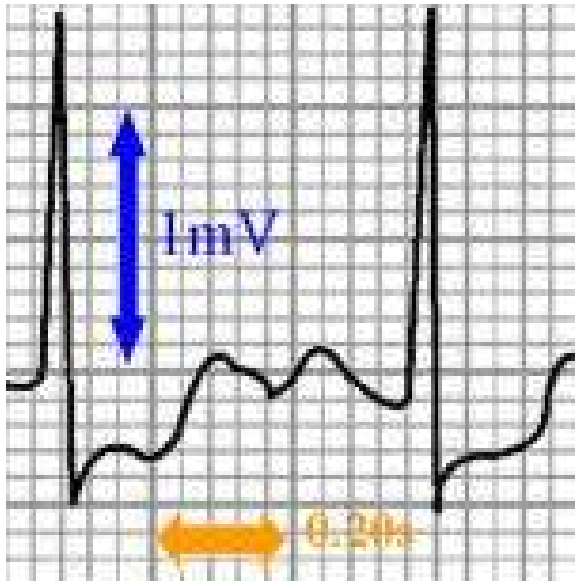
ST segment

Ischemic focus has a different electric potential
= electric vector leads to this area

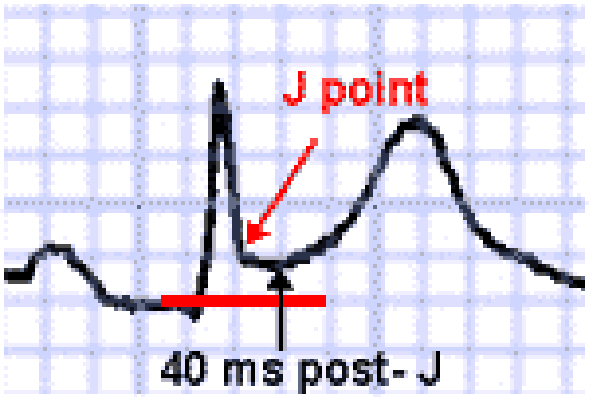
1. subendocardial ischemia

(non-Q MI, AP paroxysm)

... ST depresssion



ST segment

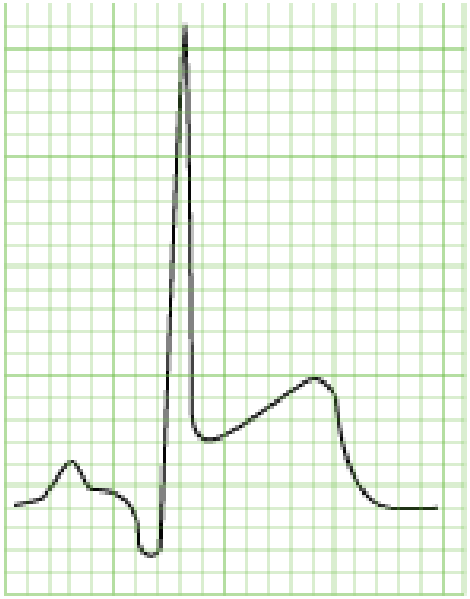


Ischemic focus has a different electric potential
= electric vector leads to this area

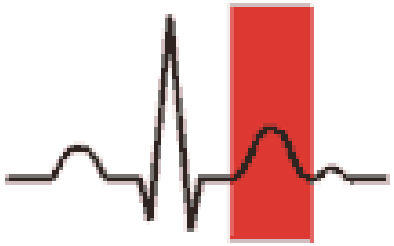
2. subepicardial ischemia

(Q-MI, spastic form of AP, aneurysma)

... elevation of ST segment



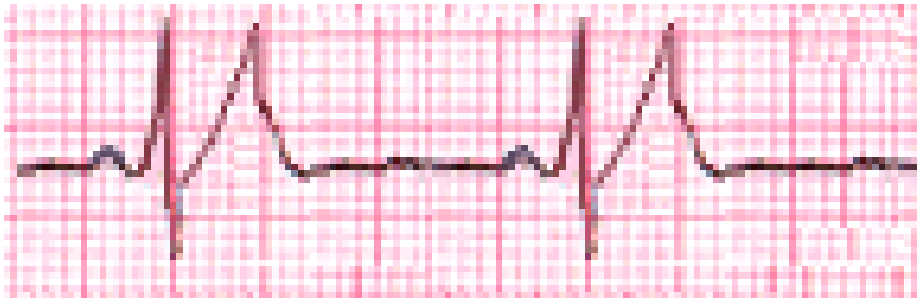
T wave

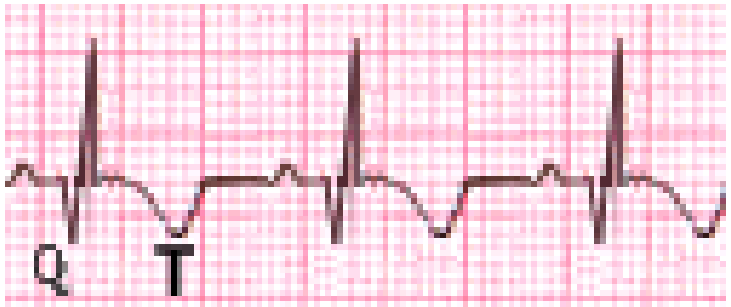


Ventricular repolarization

Normally: a repolarization directs from epicardium to endocardium = T wave is concordant with QRS complex

Ischemic area: a repolarization is delayed, an action potential is extended





Vector of repolarization is directed <u>from</u> ischemic area:
- subendocardial ischemia ... to epicardium ... T wave elevation
- subepicardial ischemia ... to endocardium ... T wave inversion

T wave

- LV overload
- neurocirculatory asthenia
- sympathetic system
- hypokalemie
- hyperglycemia
- myxoedema
- pancreatitis
- pneumotorax

Nonspecific changes

Diffuse T changes,
T wave
asymmetric
or biphasic

T wave

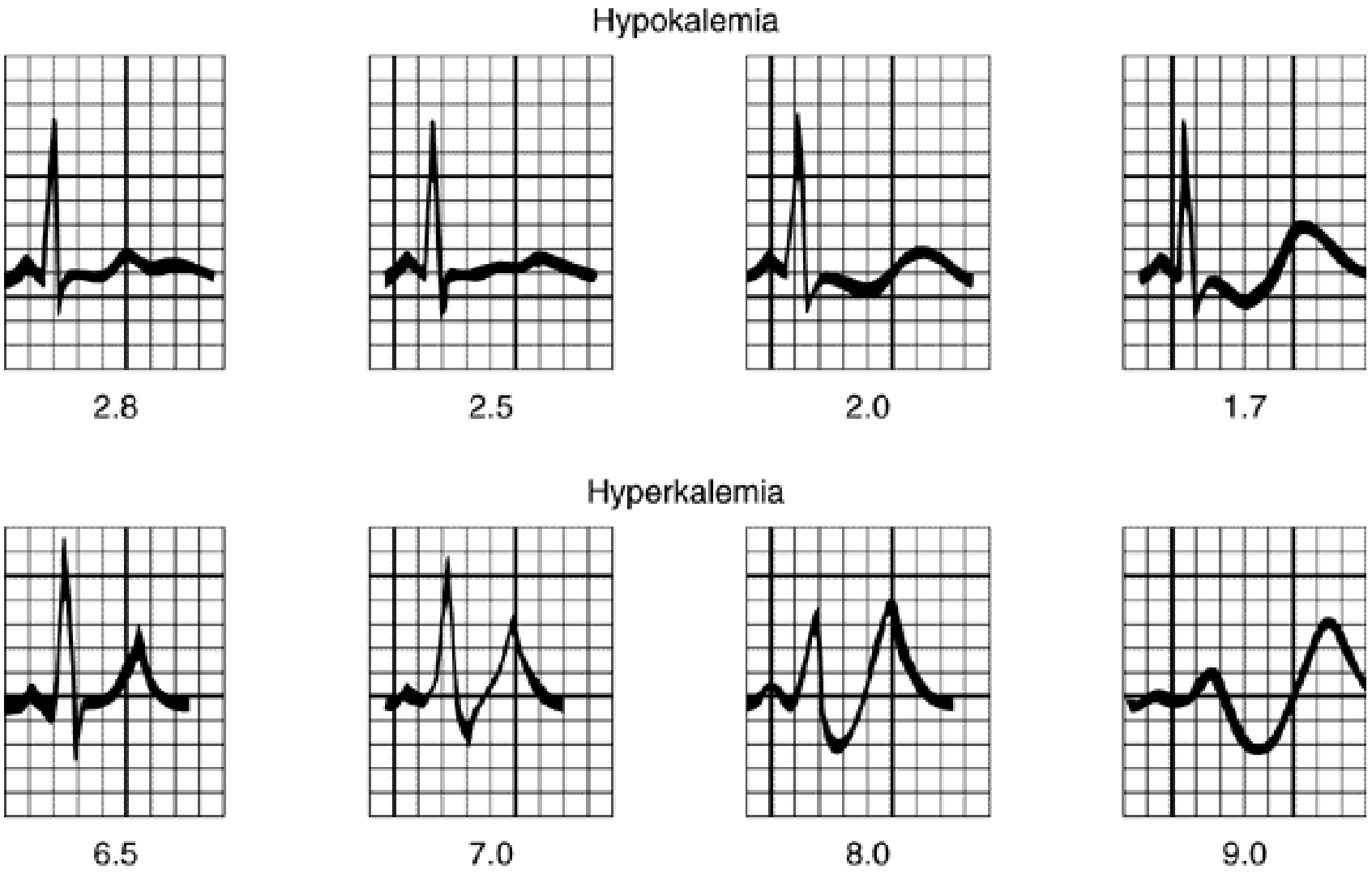
Ischemia

Localized T changes
T wave - symmetric negativity

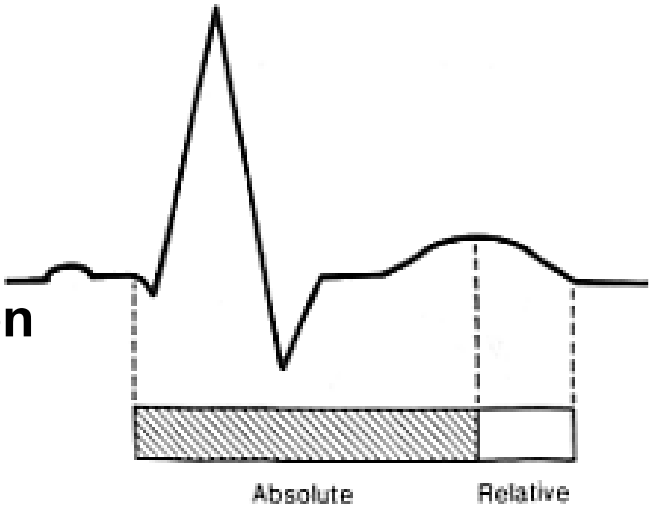
Nonspecific changes

Diffuse T changes,
T wave
asymmetric
or biphasic

K^+ influence on cardiac conductivity



Refractory period

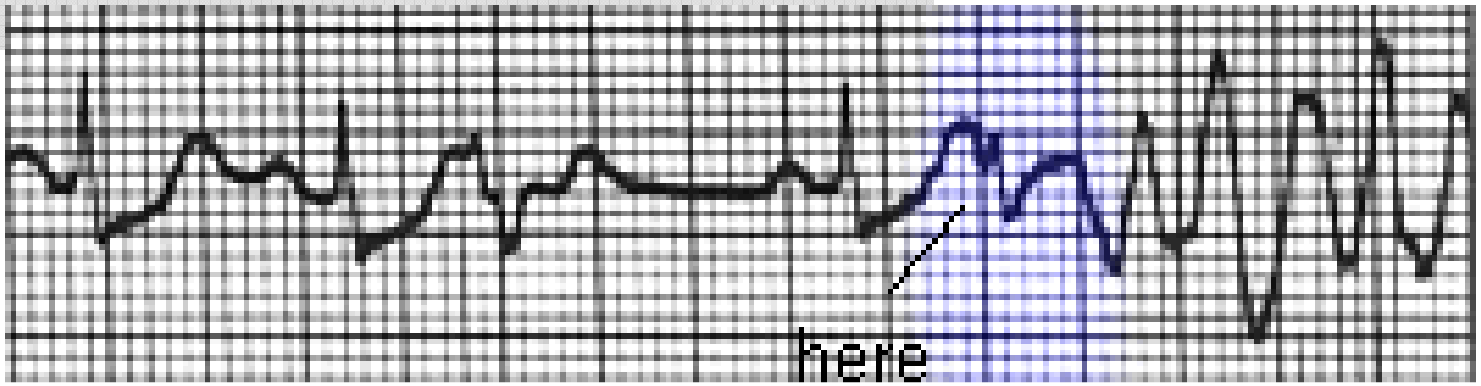


1. **Absolute** = Absolutely no stimulation can cause another action potential

2. **Relative** = It is possible to cause another action potential, but the intensity of the premature contraction will be relative to the time in this period.

„R on T“ phenomena

„Malignant VES“: R wave of the next beat falls in certain portions of the previous T-wave
... Serious and life-threatening arrhythmia



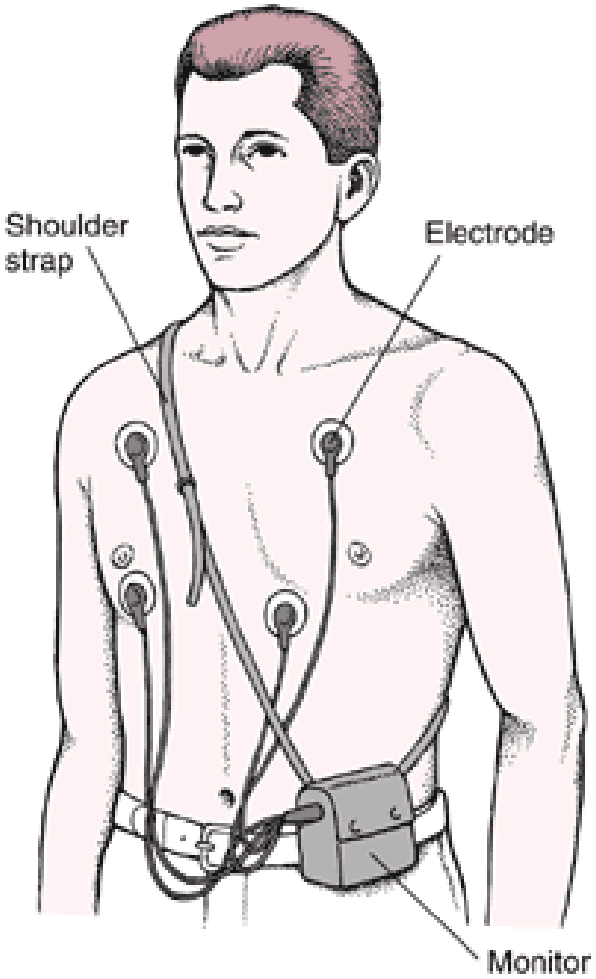
Holter monitoring

24-h ECG recording

Ambulatory ECG device

Analysis of mean, maximal, and minimal HI occurrence and frequency of major arrhyth

Confrontation of record and subjective difficulties (patient activity log)



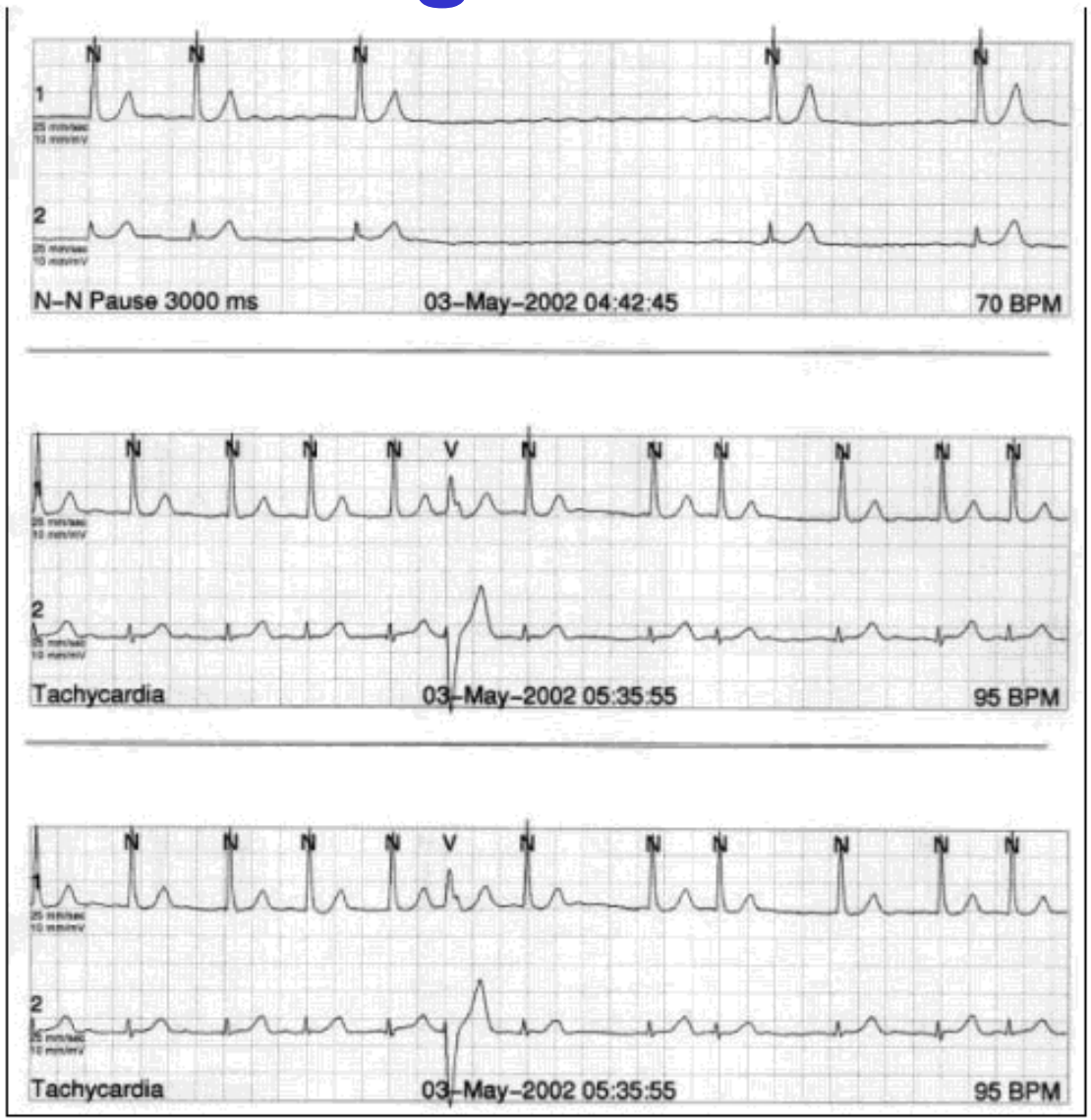
Indications:

1. **syncope or palpitation of unclear origin**
2. **an unveiling of latent ischemia**
3. **an antiarrythmic therapy control**
4. **a pacemaker control**

Holter monitoring

Patient No. 1

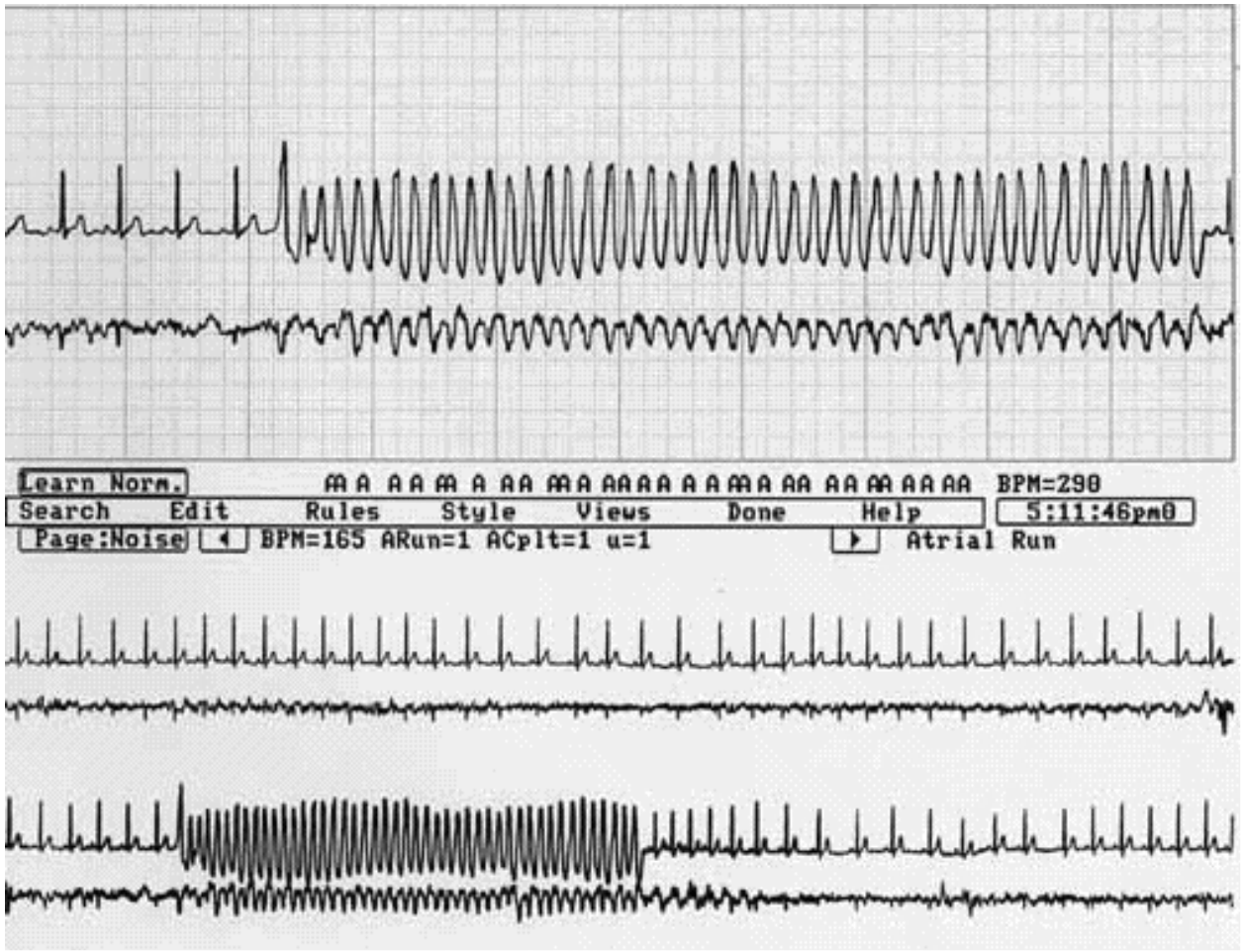
Finding of atrial fibrillation.
Pauses > 2 s
Rare ventricular ES



Holter monitoring

Patient No. 2

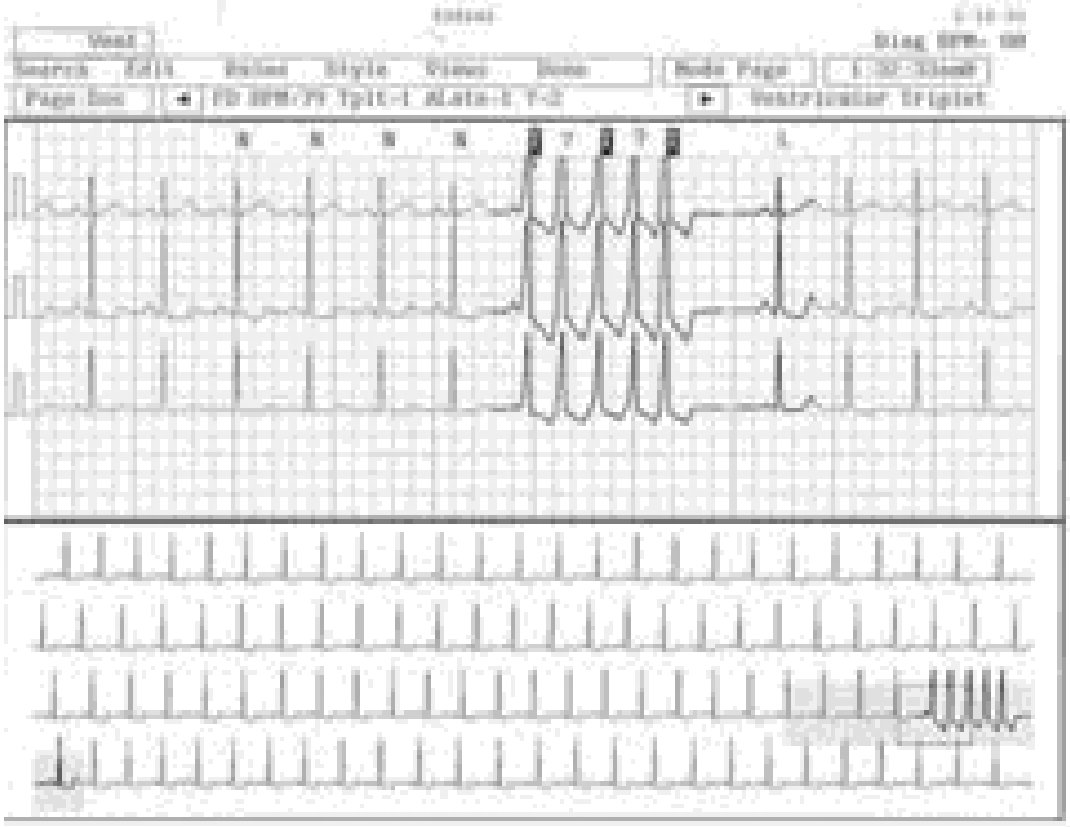
Ventricular fibrillation



Holter monitoring

Patient No. 3

Ventricular tachycardia

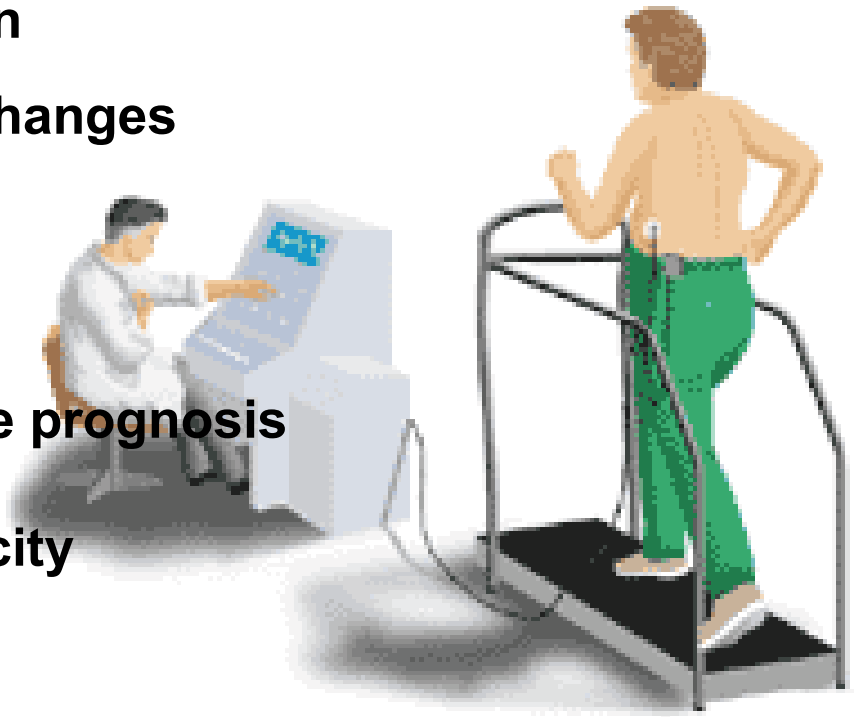


Ergometry, exercise ECG

Gradual load increase in 4-min. intervals, basic level 25 - 75 W

Stopping - submaximal load or complications (accelerated hypertension, polytopic VES, blockades, ST elevation ST, ST depression > 2 mm, T inversion

Coincidence of chest pain + ST changes = confirmation of ischemia



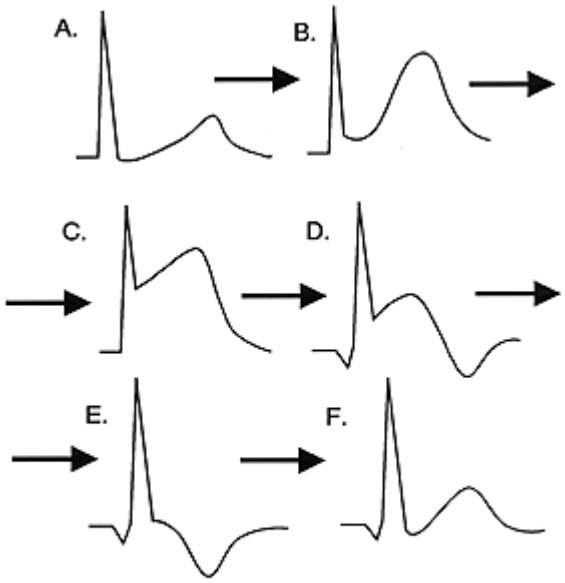
Indications:

1. specification of ischem. disease prognosis
2. suspicion on ischemic disease
3. examination of functional capacity

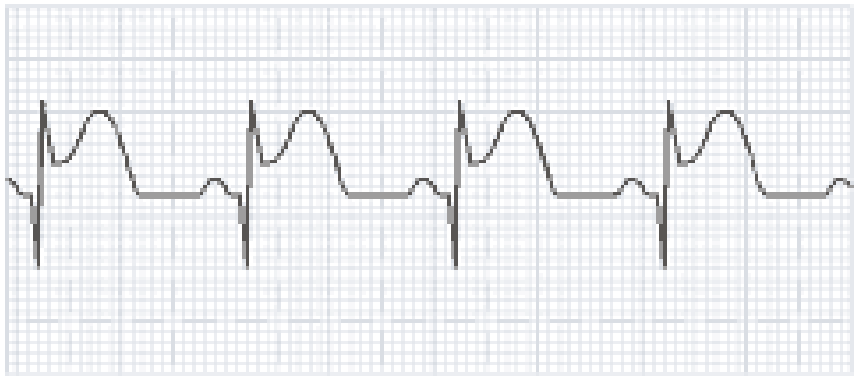
Q myocardial infarction

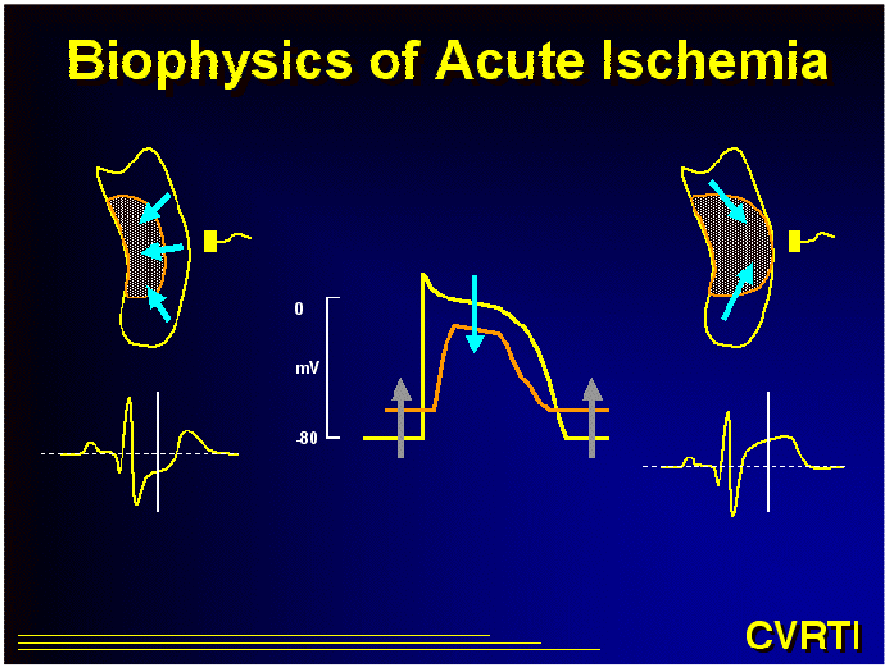
ECG changes

Martin Vokurka

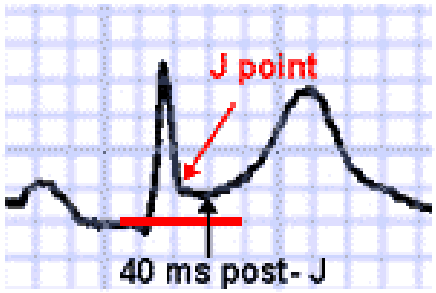


Evolution of Acute MI

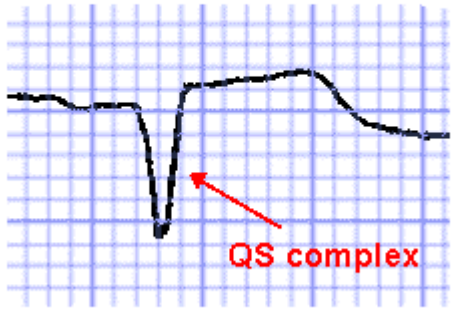




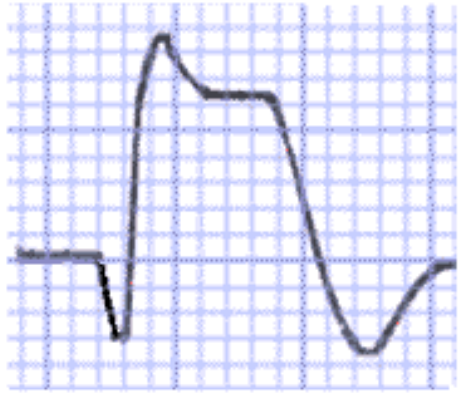
S-T Elevation



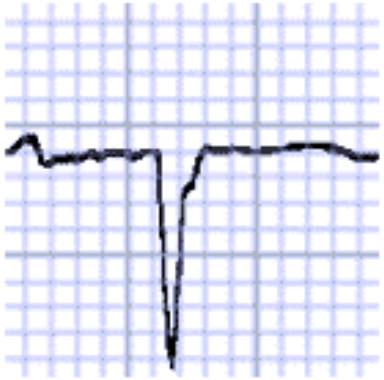
Pathological Q wave in V1



Inverted T wave



Persistent Q wave



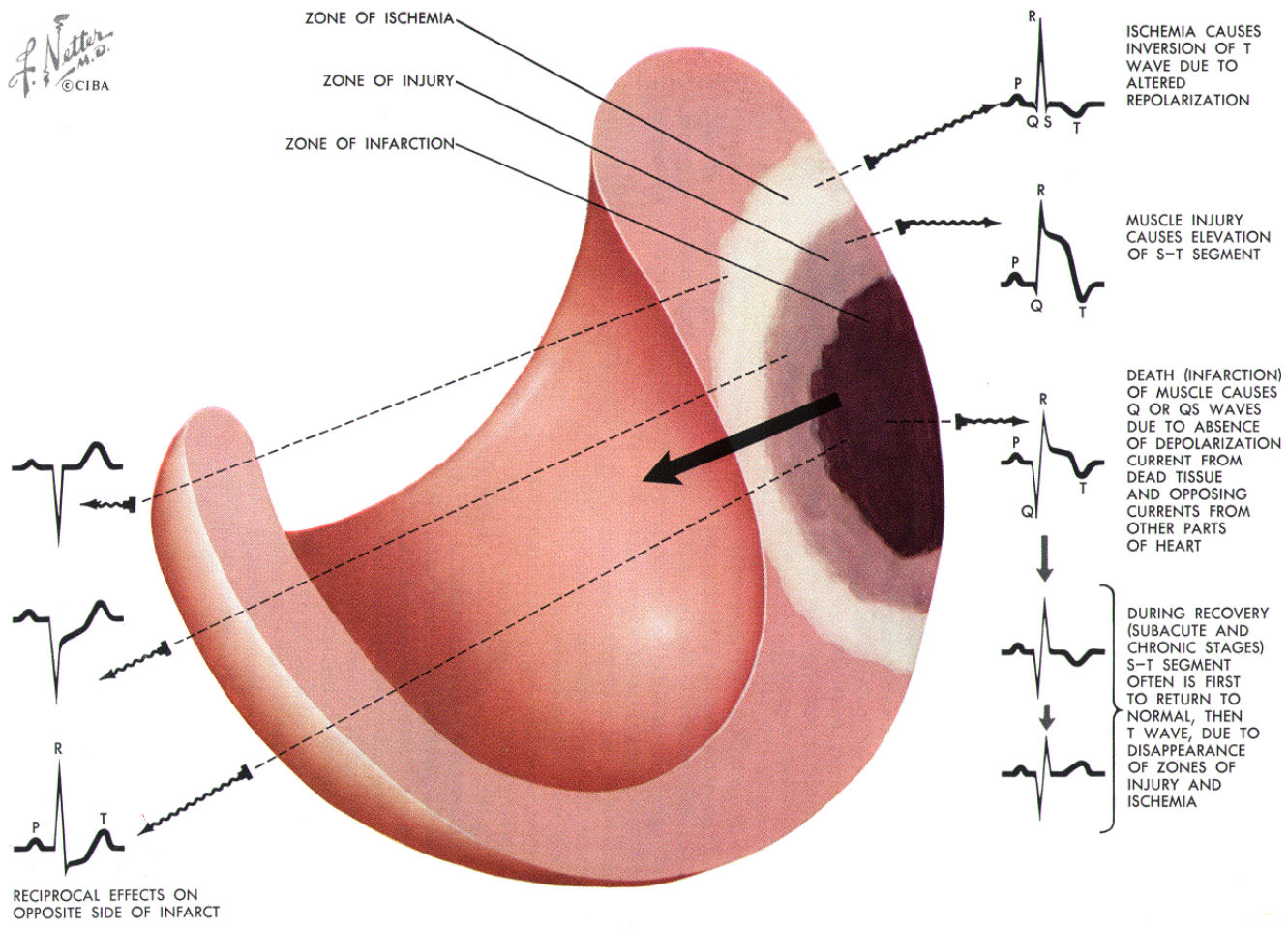
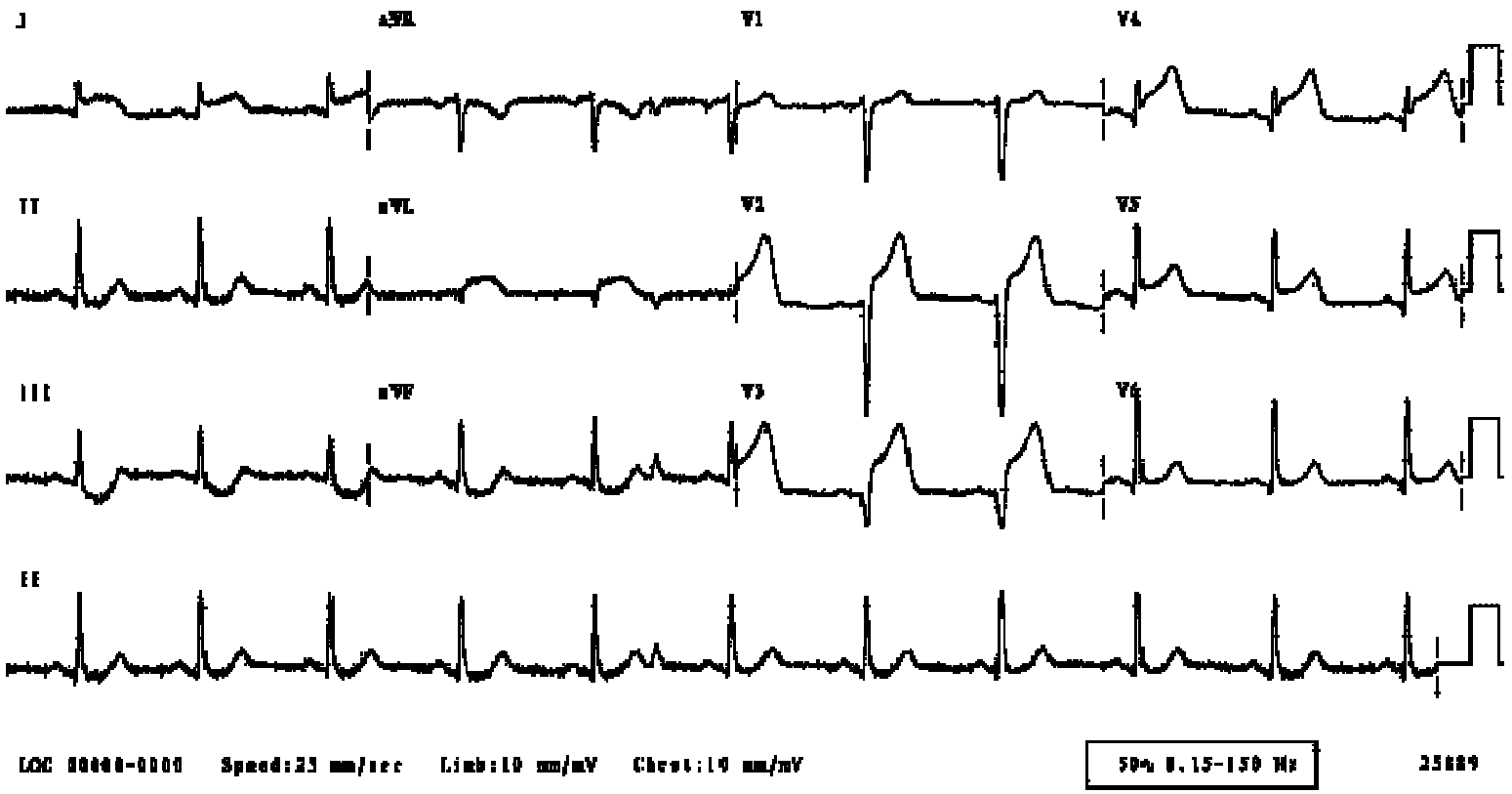


PLATE IX

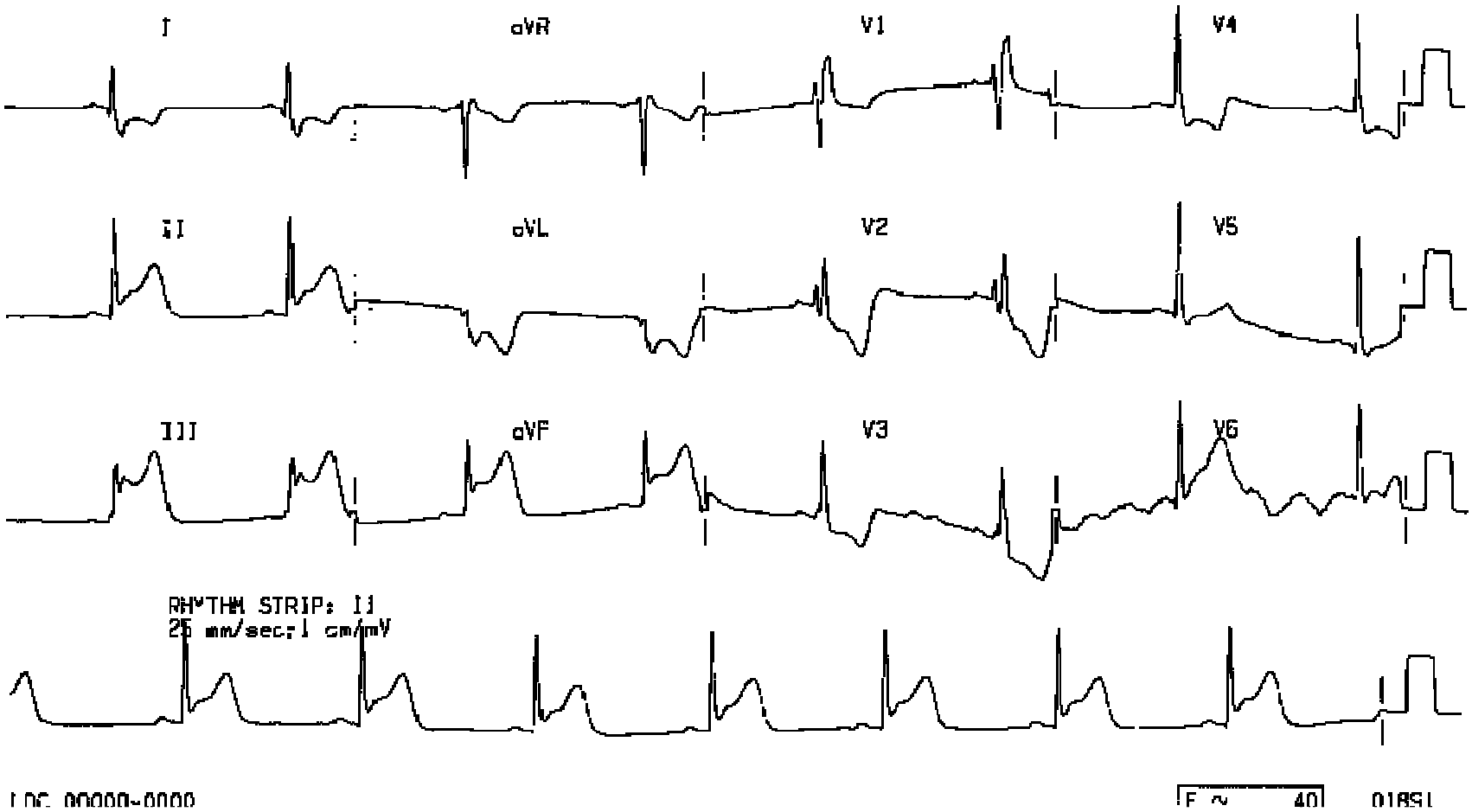
EFFECT OF CARDIAC INFARCTION, INJURY, AND ISCHEMIA



Acute anterior myocardial infarction

ST elevation in the anterior leads V1 - 6, I and aVL

reciprocal ST depression in the inferior leads

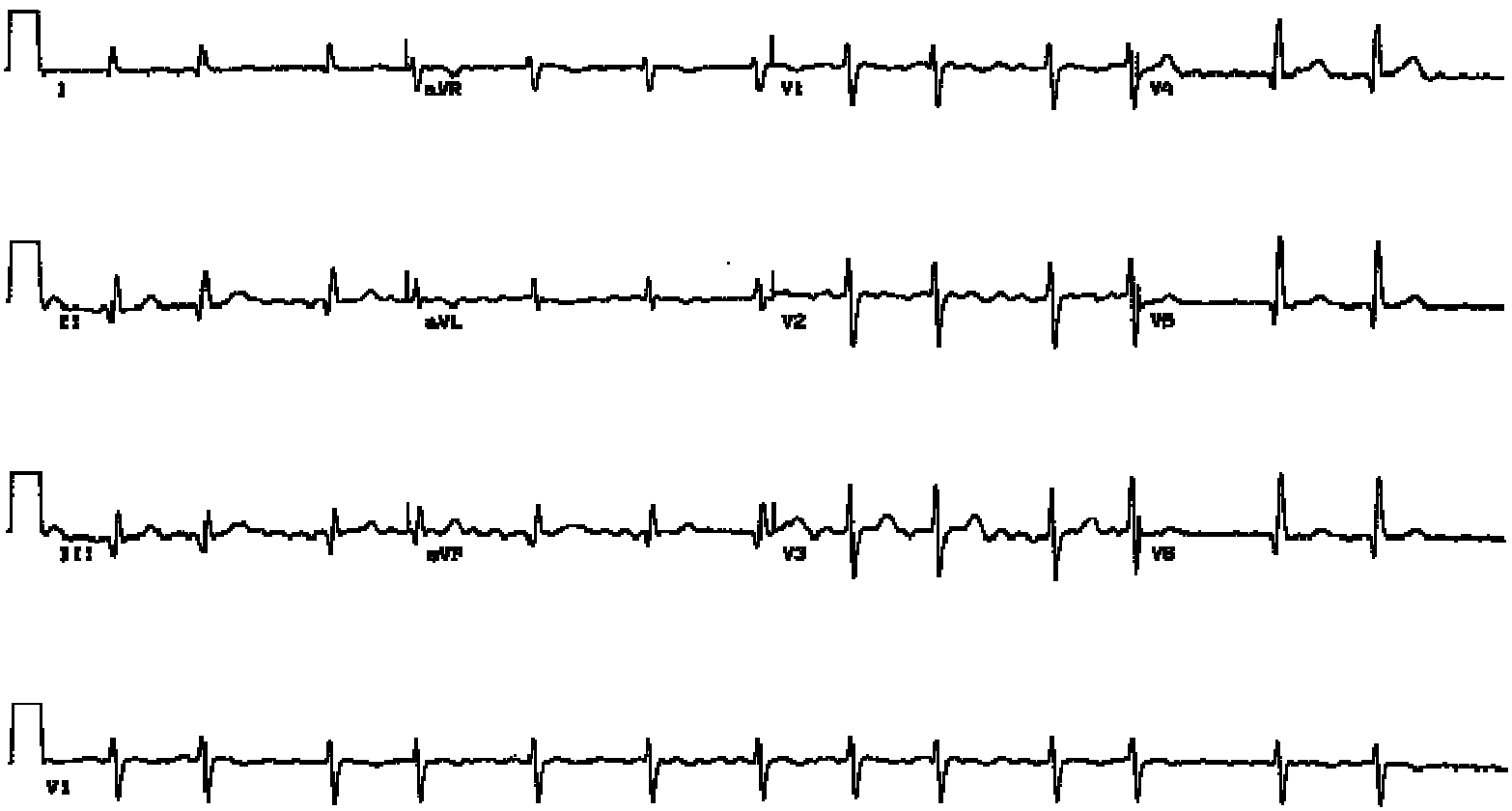


Acute inferior myocardial infarction

ST elevation in the inferior leads II, III and aVF

reciprocal ST depression in the anterior leads

RBBB and bradycardia are also present

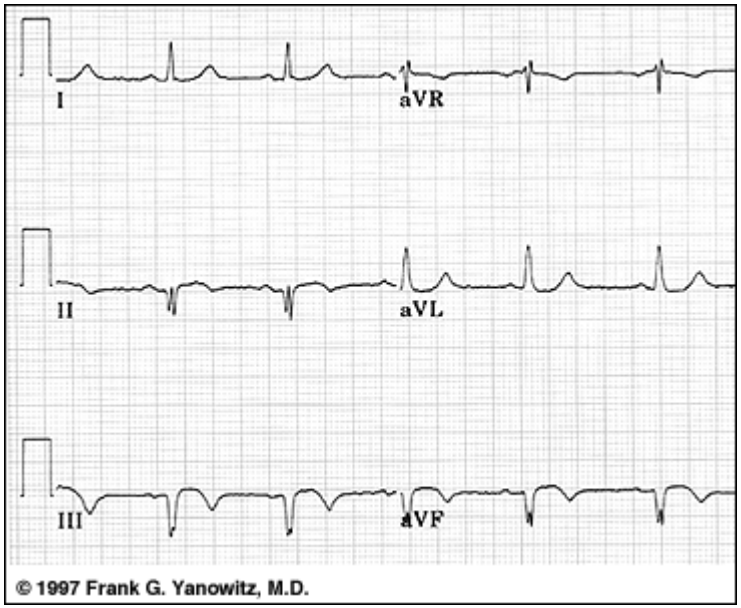


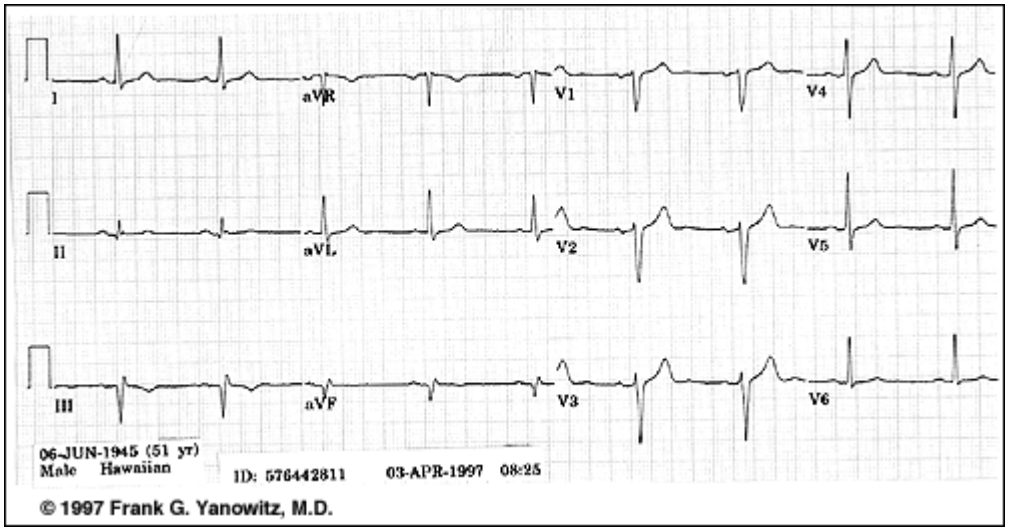
Old inferior myocardial infarction

Q wave in lead III wider than 1 mm (1 small square) and

Q wave in lead aVF wider than 0.5 mm and

Q wave of any size in lead II





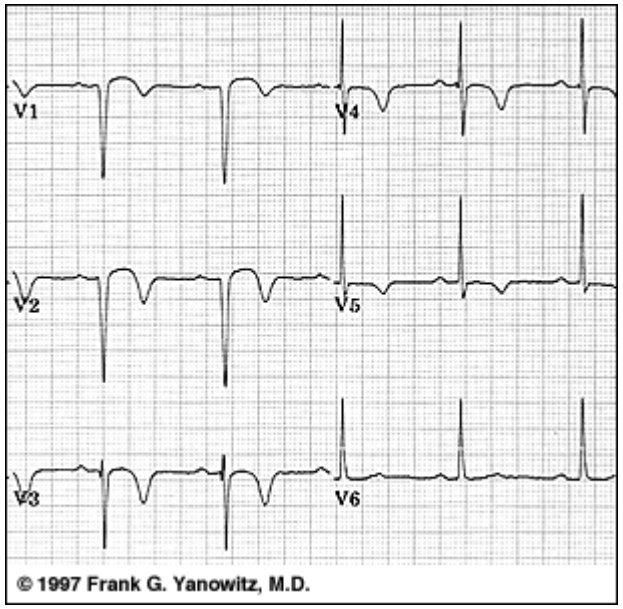
<u>Old inferior MI</u> (note largest Q in lead III, next largest in aVF, and smallest in lead II)

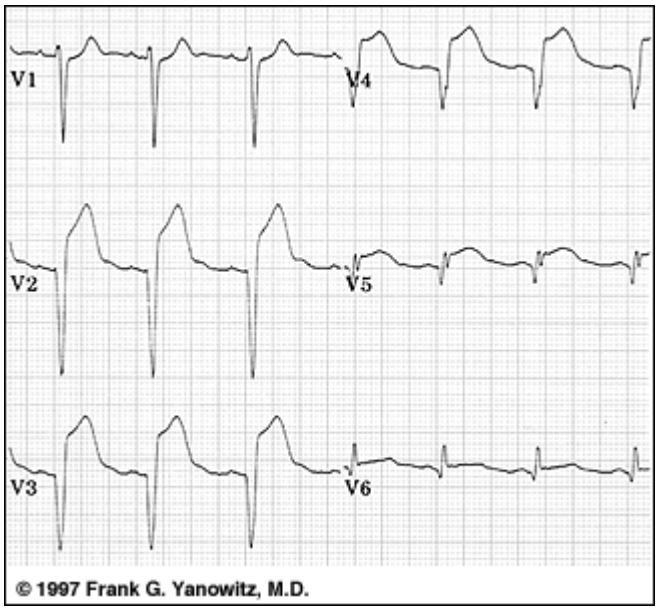
<u>Inferior MI</u>

Pathologic Q waves and evolving ST-T changes in leads II, III, aVF

Q waves usually largest in lead III, next largest in lead aVF, and smallest in lead II

Example: frontal plane leads with fully evolved inferior MI (note Q-waves, residual ST elevation, and T inversion in II, III, aVF)





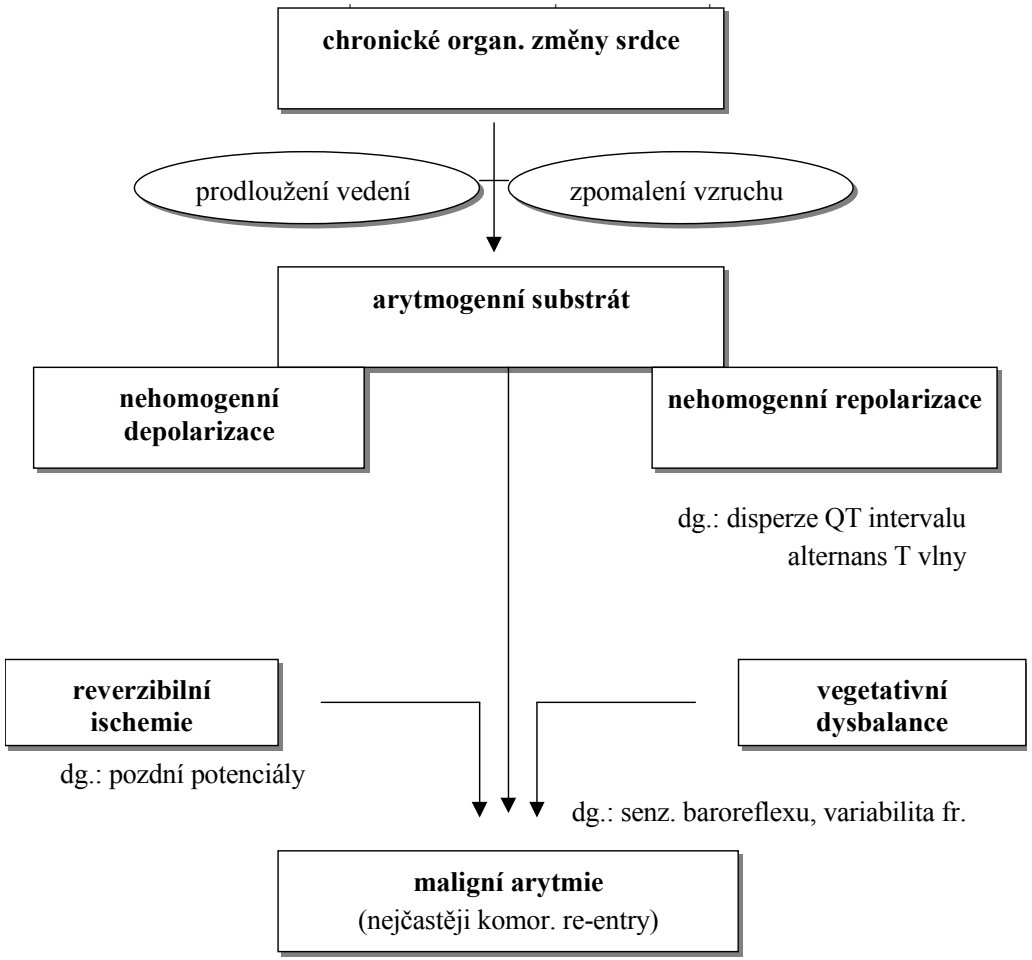
<u>Anteroseptal MI</u>

Q, QS, or qrS complexes in leads V1-V3 (V4)

Evolving ST-T changes

Example: Fully evolved anteroseptal MI
(note QS waves in V1-2, qrS complex in V3,
plus ST-T wave changes)

<u>Acute anterior or anterolateral MI</u> (note Q's V2-6 plus hyperacute ST-T changes)



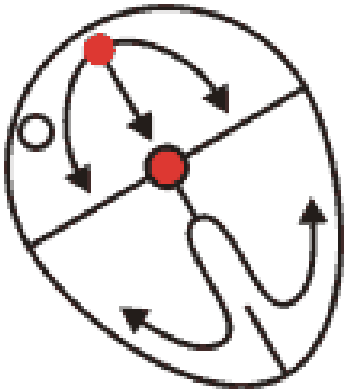
náhlá smrt

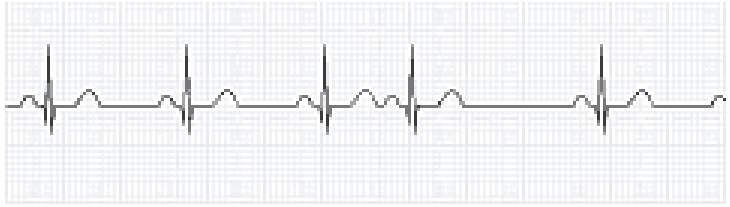
častější u mužů, etiol. kardiální 30% (ve stáří 80-90%), z kardiálních příčin: 80% tachyarytmie

Nové metody predikce kardiální náhlé smrti (predikce arytmogeneze):

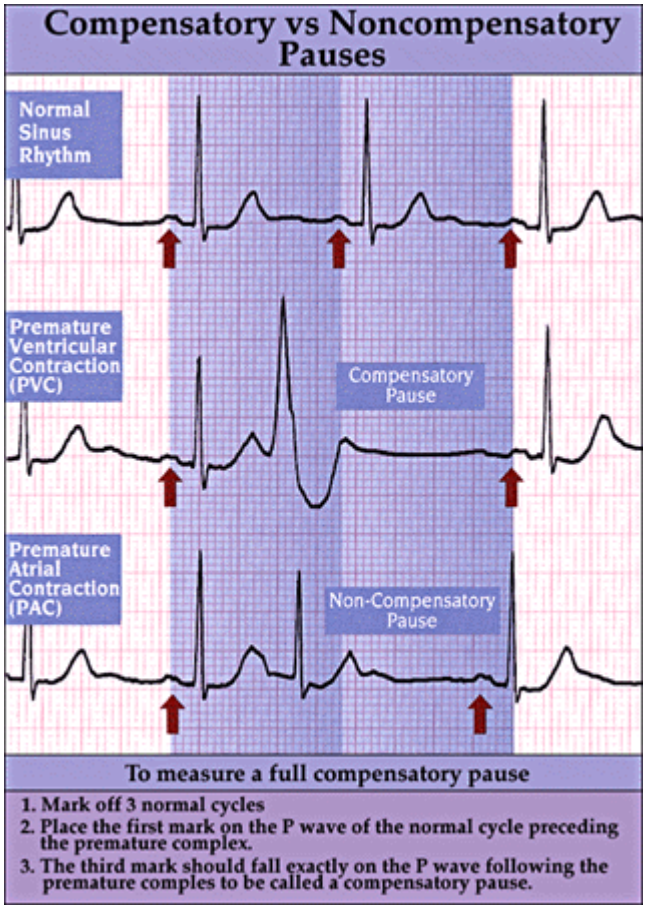
disperze QT intervalu	
alternans T vlny	spontánní variabilita voltáže T vlny
pozdní potenciály	v závěru QRS a během ST
senzibilita baroreflexu	index ↓f/↑TK po farmakol.stimulaci nebo spontánně
variabilita frekvence	periodicita respirační, baroreflexní, termoregulační atd., oplošťuje se s věkem, sympatikotomií (její snížení tedy odráží vyšší riziko maligních dysrytmií)

Atrial extrasystole

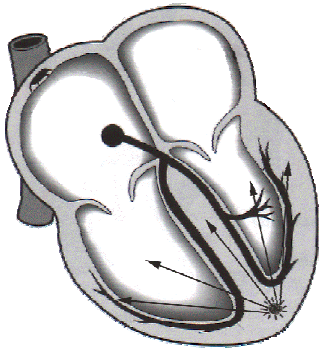


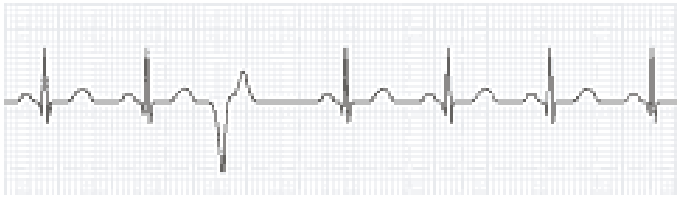


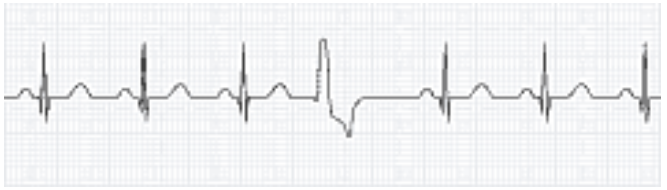
Compensatory pauses

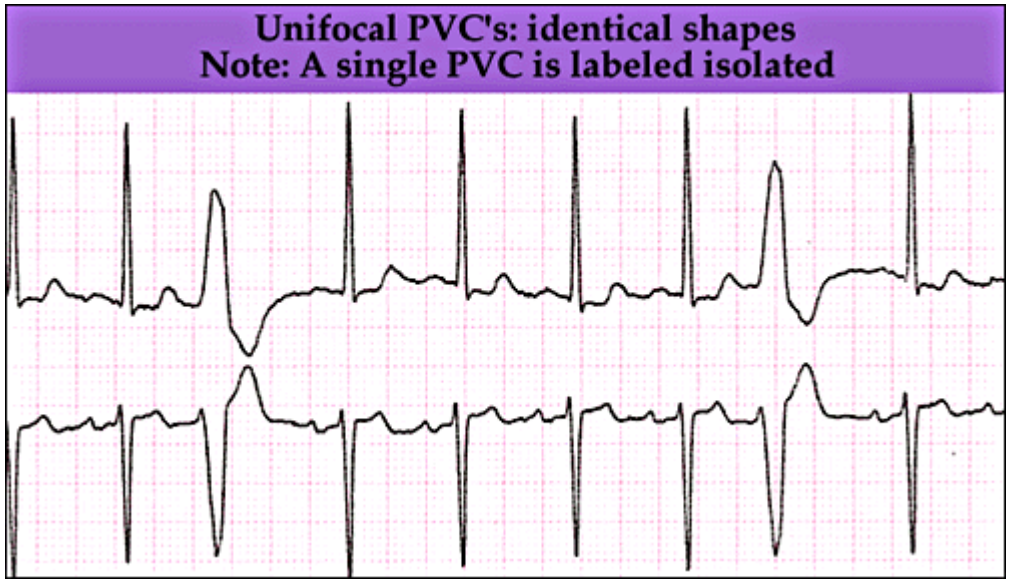


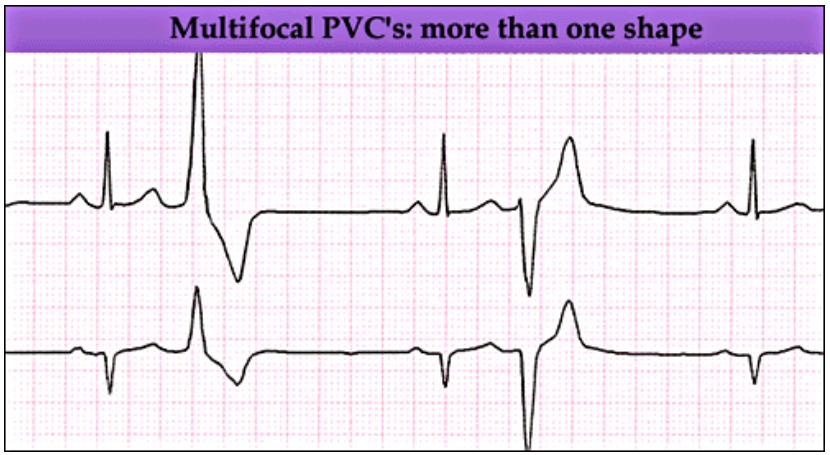
Ventricular extrasystole



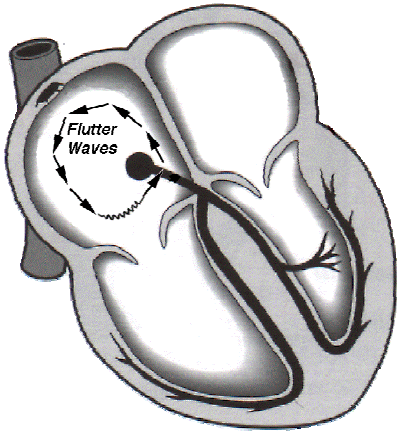




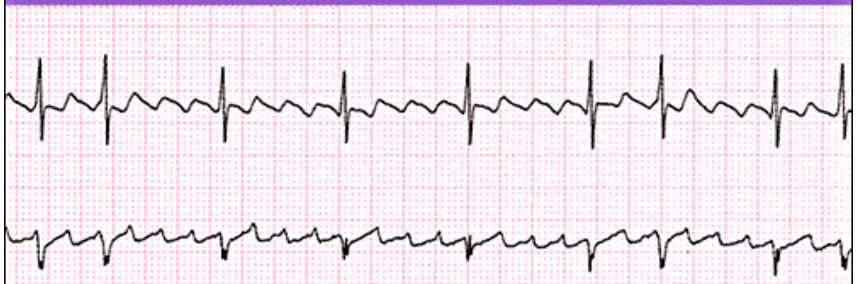




Atrial flutter

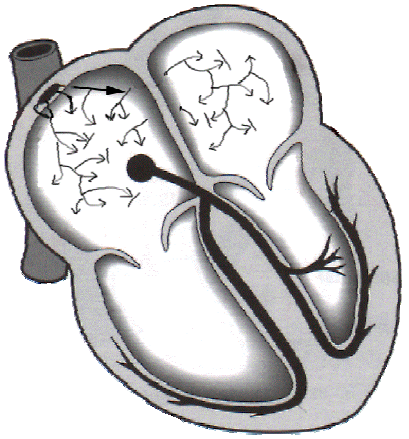


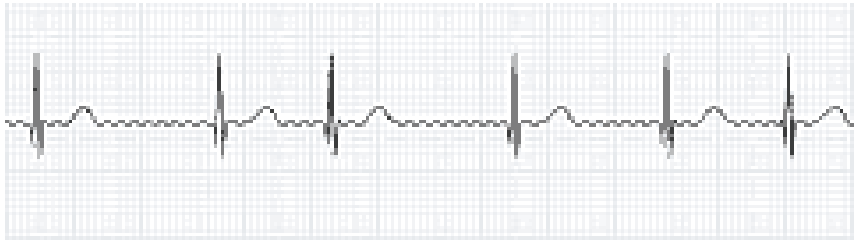
Atrial Flutter



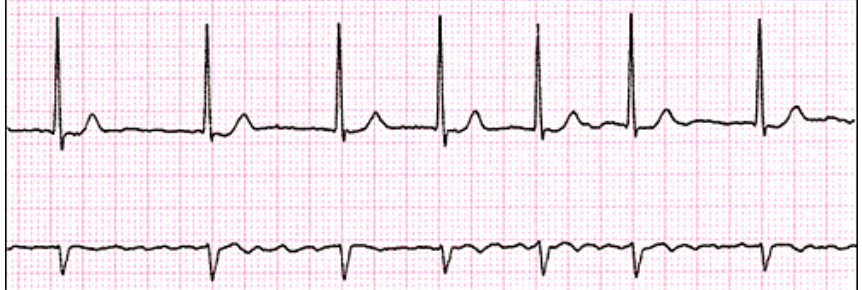
Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
A: 220-430 bpm V: <300 bpm	Regular or variable	Sawtoothed appearance	N/A	<.12

Atrial fibrillation





Atrial Fibrillation



Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
A: 350-650 bpm V: Slow to rapid	Irregular	Fibrillatory (fine to course)	N/A	<.12

SV tachycardia

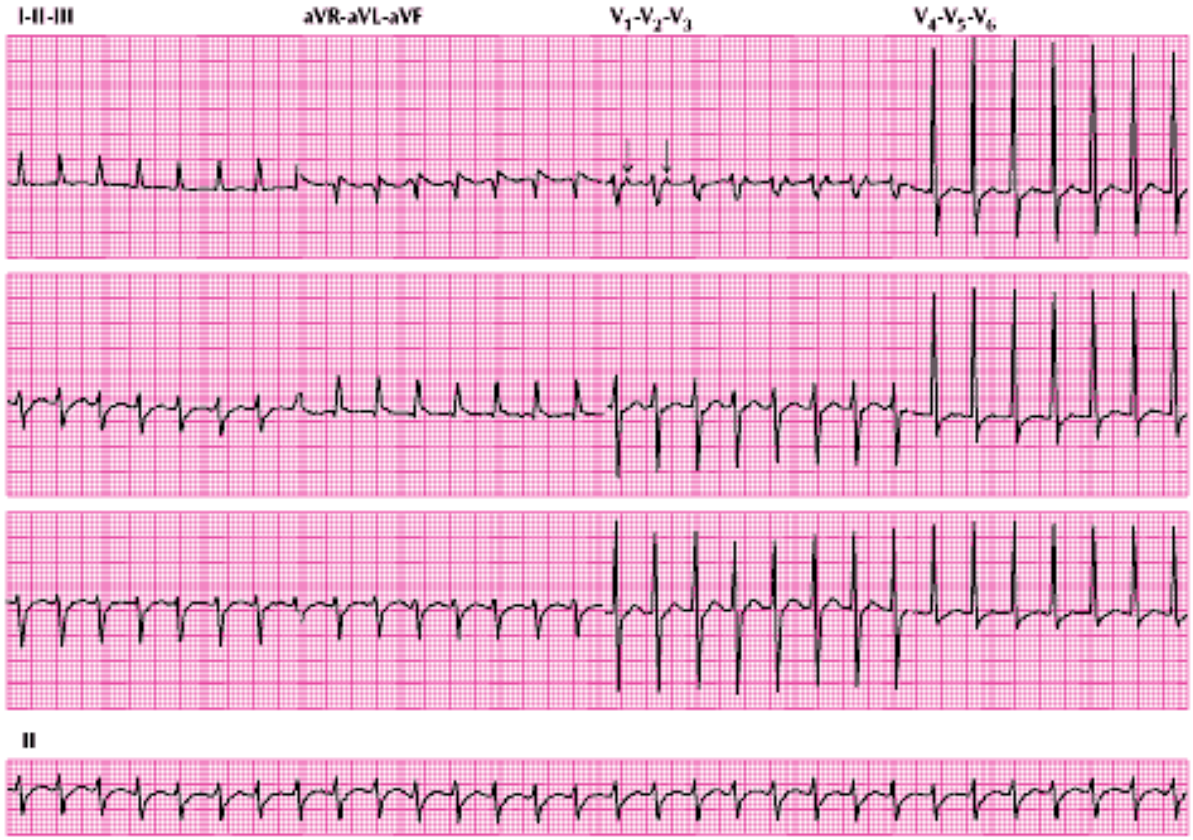
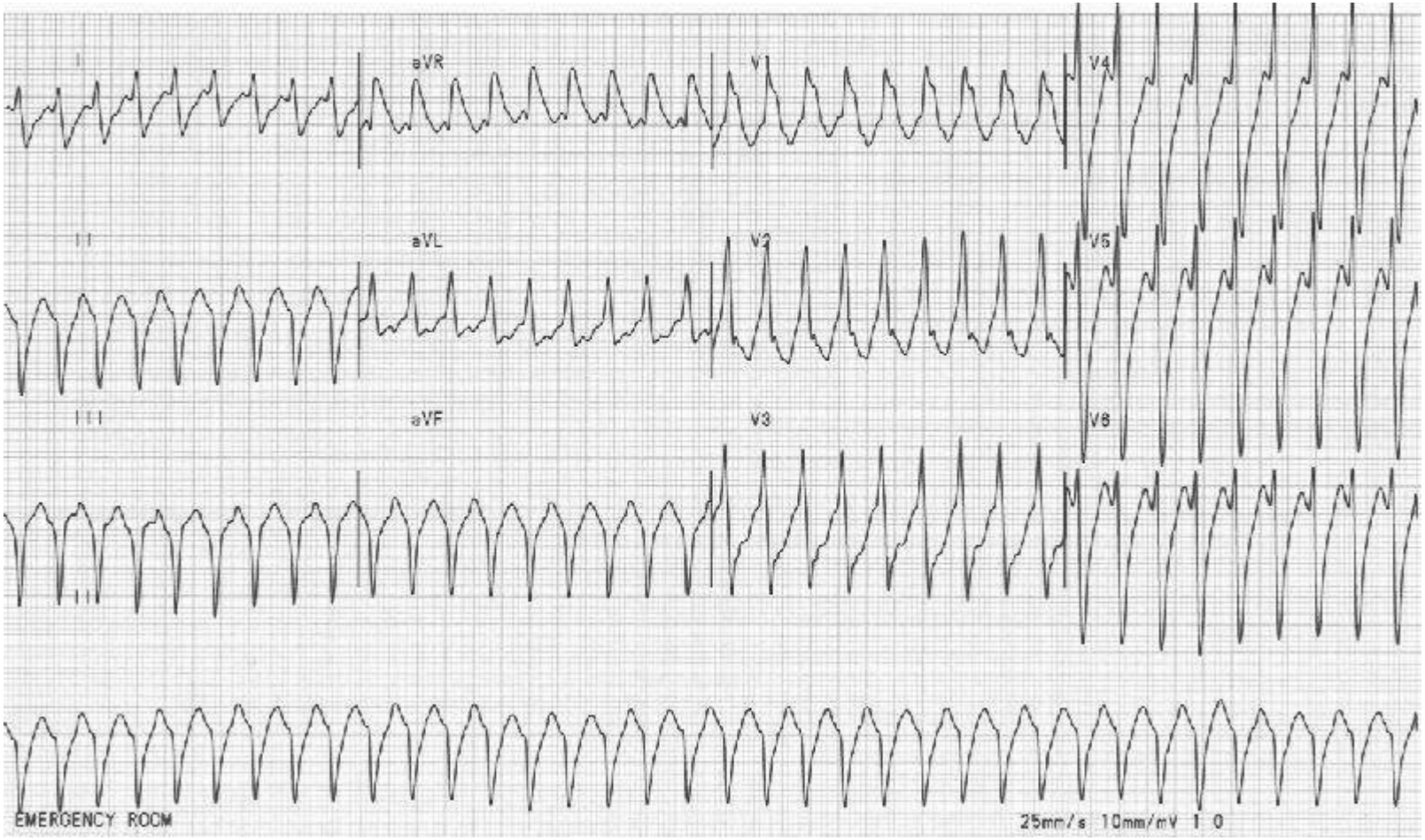


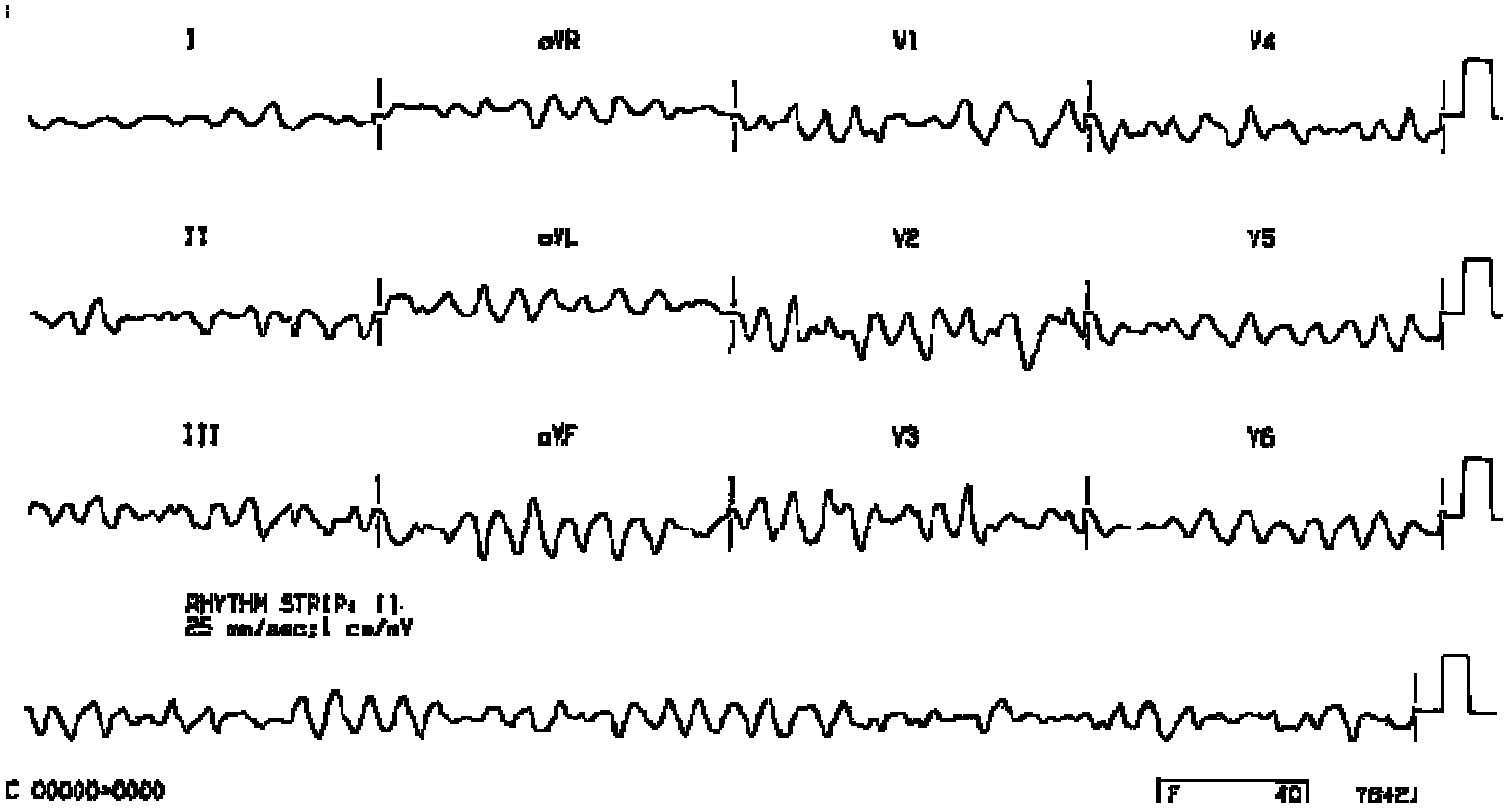
Figure 2. ECG shows supraventricular tachycardia in a 36-year-old woman with frequent episodes of sudden-onset, rapid, and regular heart rate. The ventricular rate is 183 bpm. Note the P waves at the end of the QRS complex (arrows in V_1). Symptoms persisted despite treatment with oral verapamil and metoprolol, and the patient was referred for radiofrequency ablation. AV-node reentry tachycardia was diagnosed on electrophysiologic testing. The patient underwent successful ablation of the "slow pathway" with resolution of symptoms.

Ventricular tachycardia

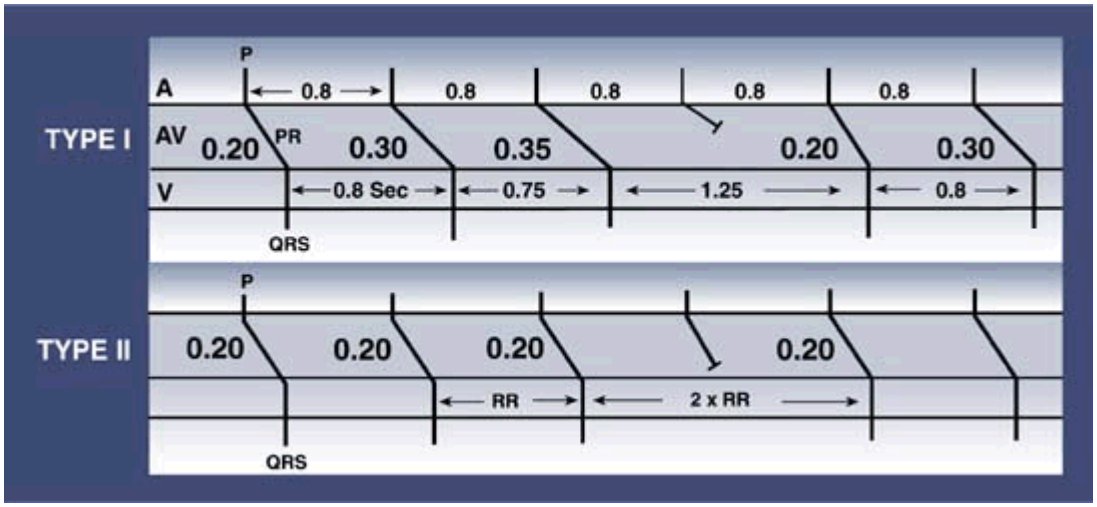


Ventricular fibrilation (or flutter)

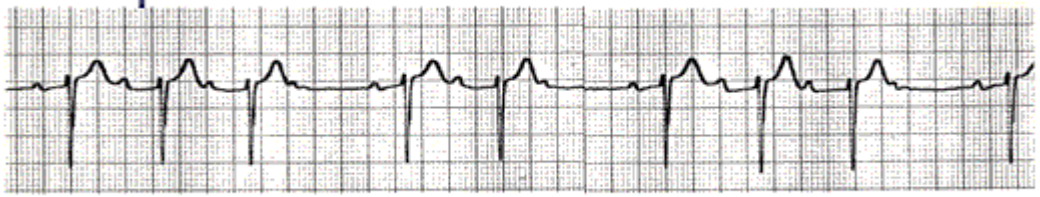
Acute situation, hemodynamic arrest –
0 cardiac output, 0 pulsation, coma,
resuscitation



AV block 2rd degree

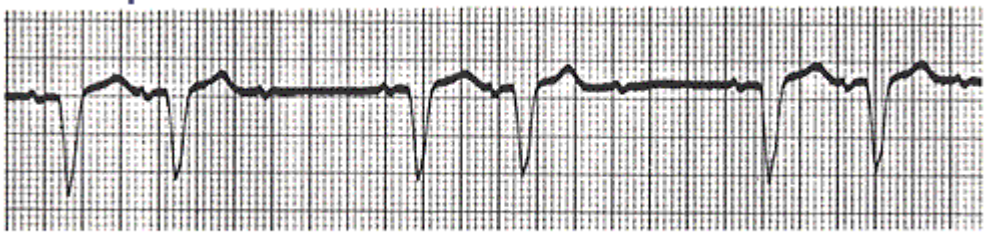


Lead V_1 "Classic Wenckebach"



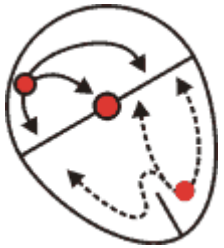
680 640 1180 680

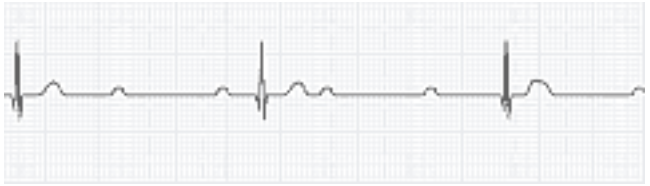
Lead V_1

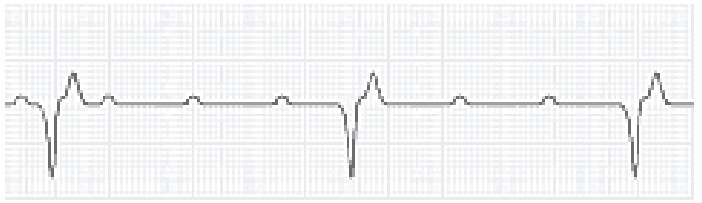


2nd degree AV block (type II) with LBBB

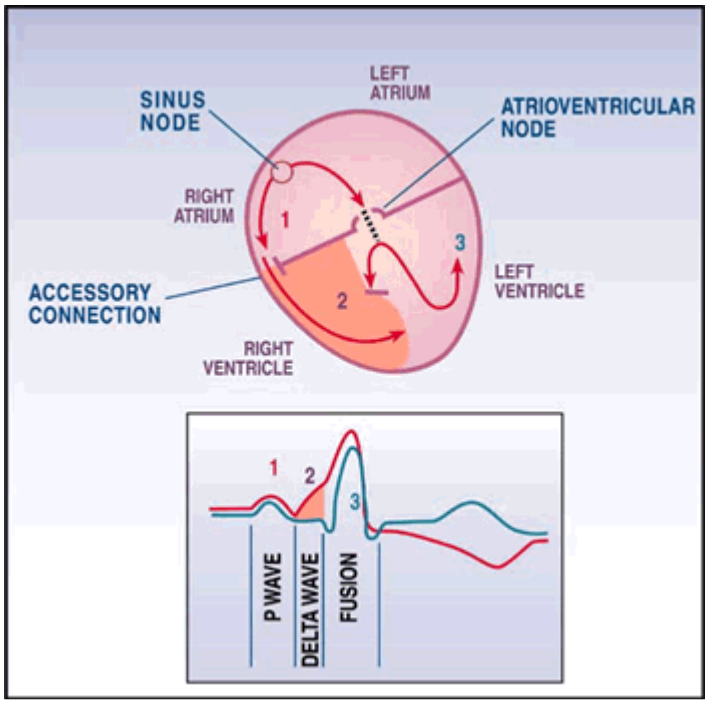
AV block 3rd degree





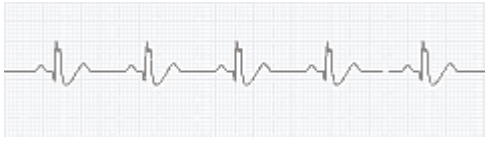


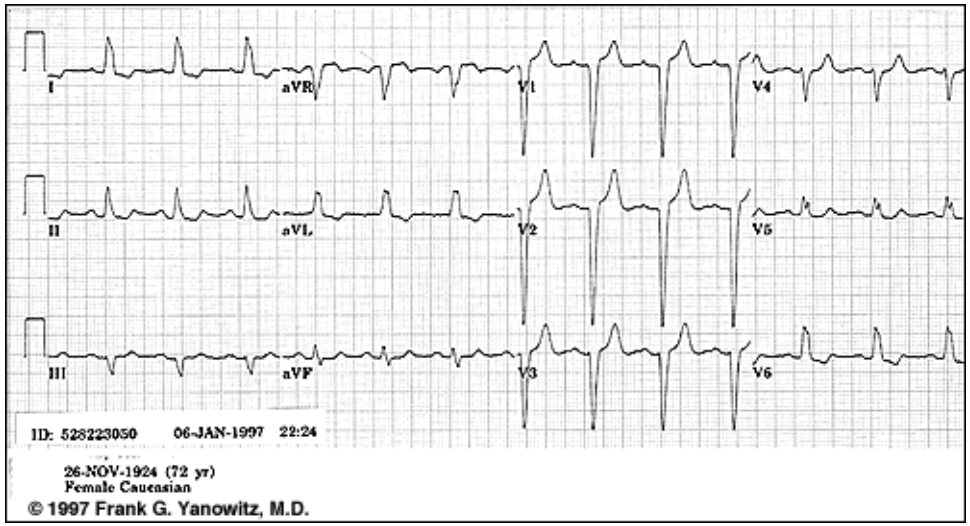
Preexcitation, WPW syndrome



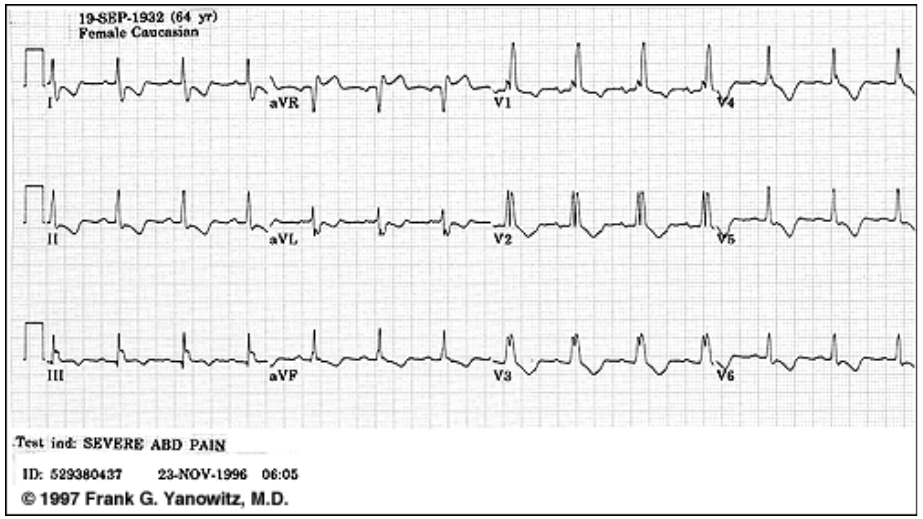
Bundle branch blocks

LBBB





RBBB



Left anterior fascicular block