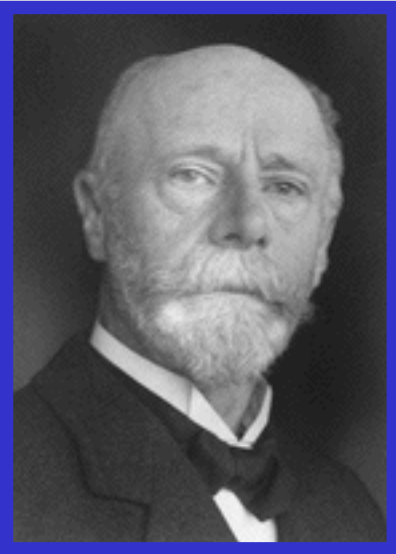


**ELECTROCARDIOGRAPHY** = methods enabling to register electrical changes caused by heart activity from body surface.



Willem Einthoven

1860 - 1927

1893 Einthoven introduces the term 'electrocardiogram'

1895 Einthoven distinguishes five deflections - P, Q, R, S and T

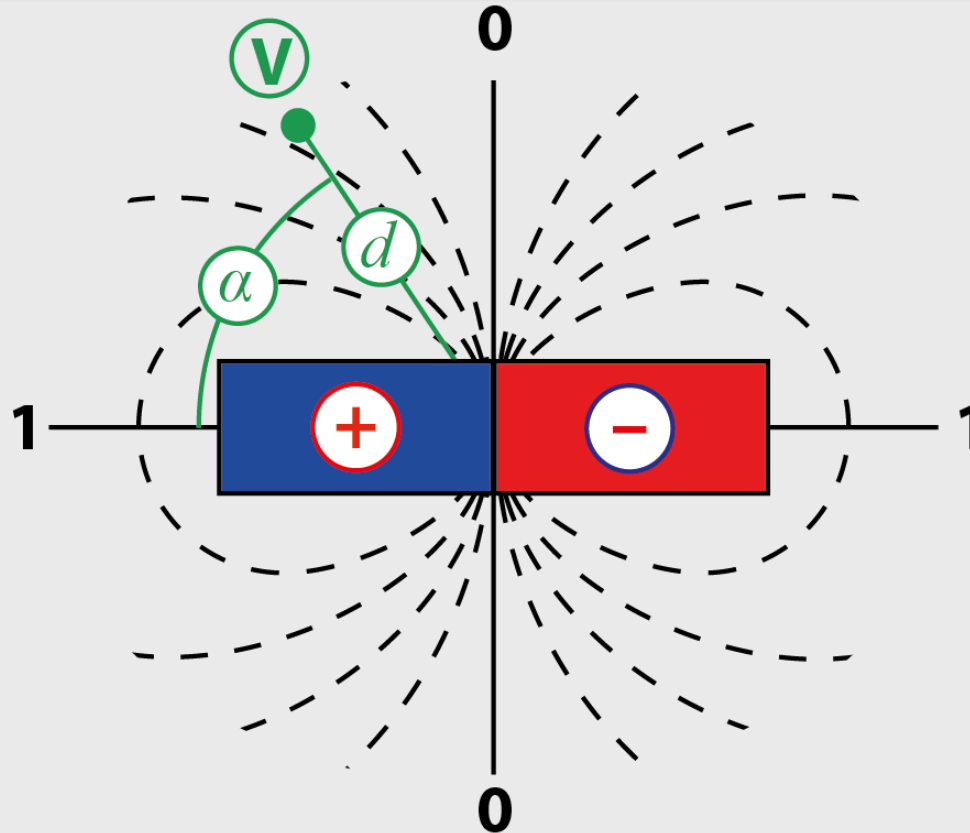
1902 Einthoven publishes the first electrocardiogram

1905 Einthoven starts transmitting electrocardiograms from the hospital to his laboratory 1.5 km away via telephone cable

1924 the Nobel prize

# ELECTRICAL DIPOLE

stationary in homogenously conducting environment



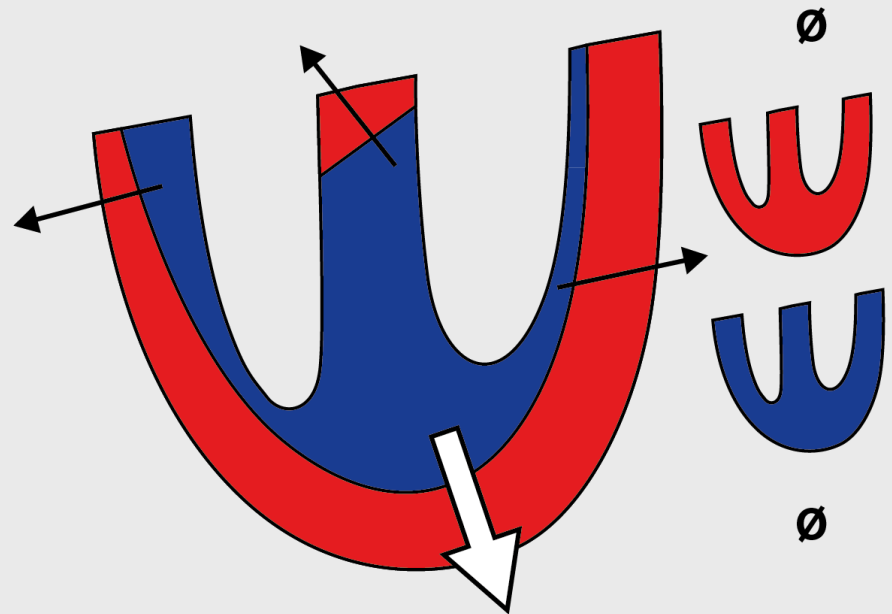
## Local currents

- Maximal in dipole axis (1)
- Zero in the place of the centre (0)

# SPREADING OF DEPOLARIZATION FRONT

## ELECTRICAL FIELD OF THE HEART (vector)

- Consists of sum of momentary dipoles on the depolarization front
- **Its size** is a function of number of dipoles and steepness of boundary line
- **Direction from** depolarized (-) to (re)polarized (+) area

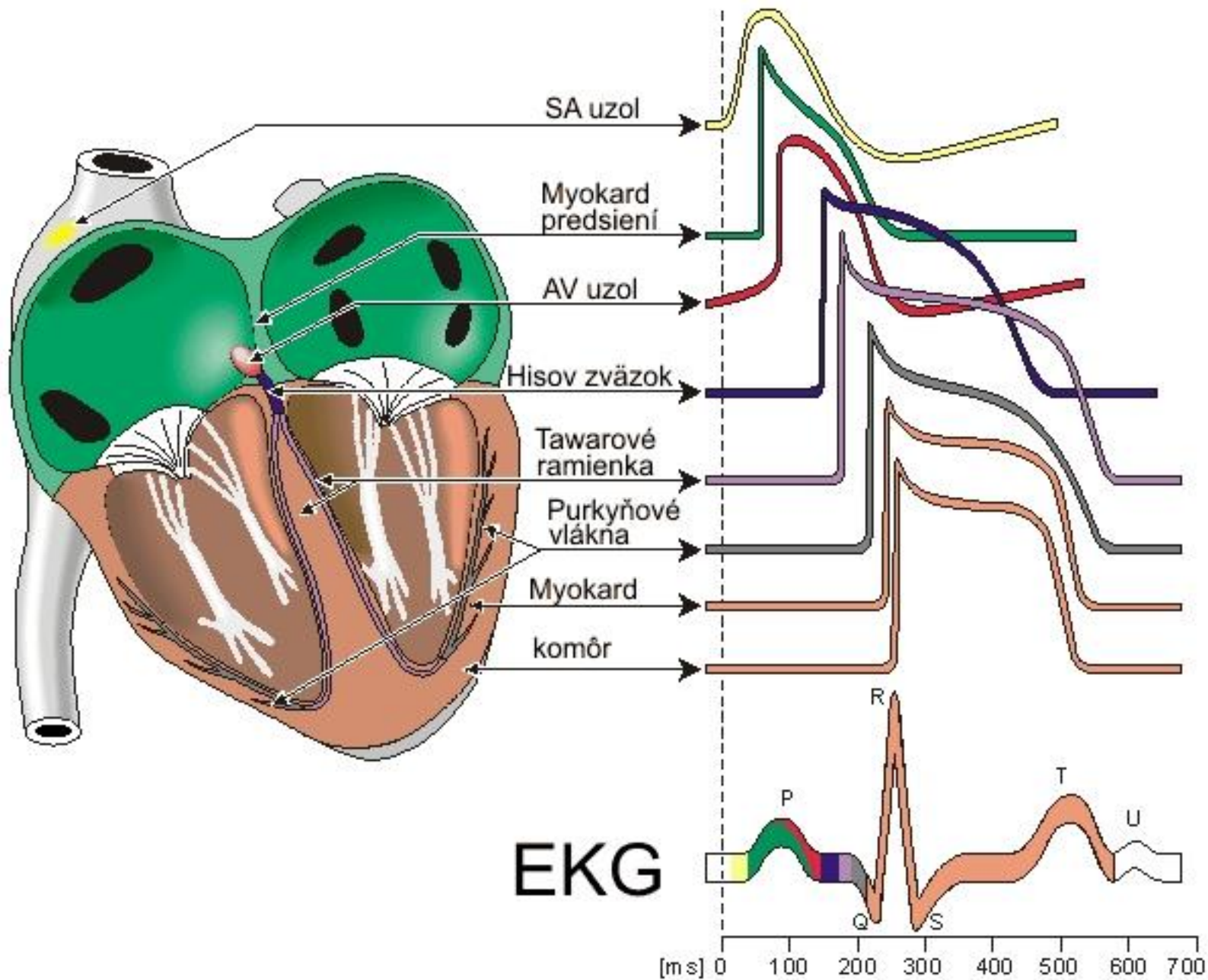


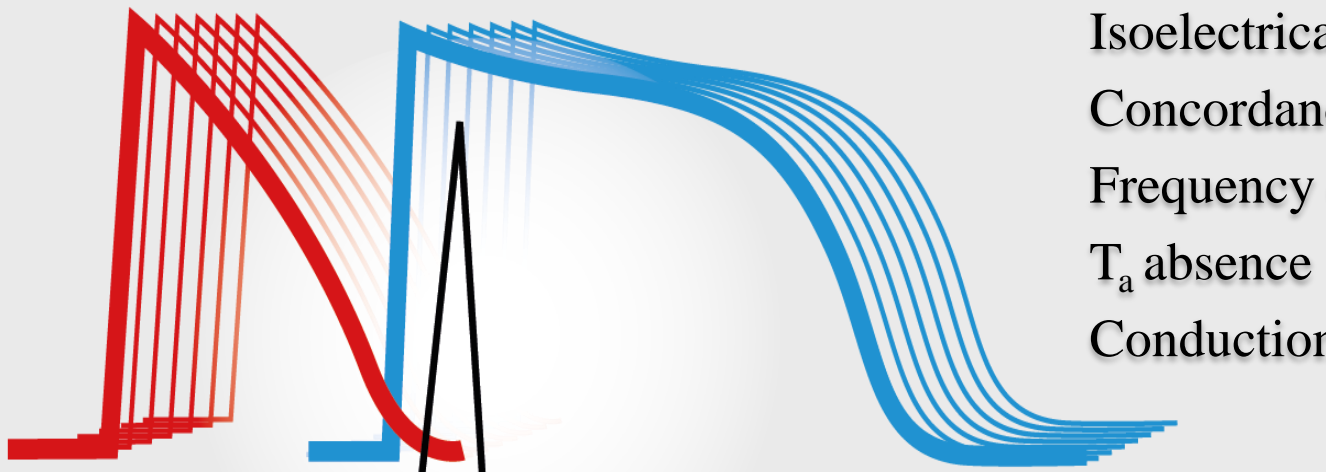
## REGIONAL VECTORS

## INTEGRAL VECTOR

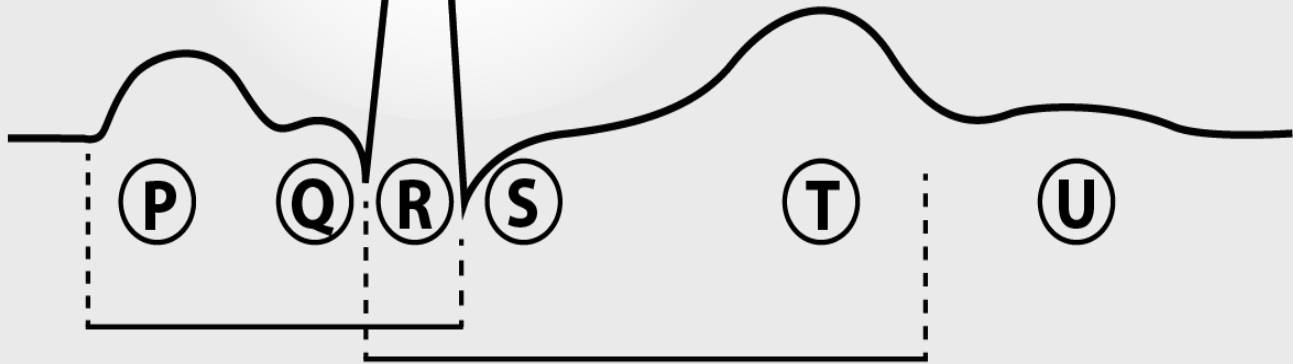
during excitation is changing:

- Size of momentary dipoles
- Their direction
- They are spreading to body surface – **ELECTROCARDIOGRAPHY**





Isoelectrical segments  
 Concordance of T wave  
 Frequency dependence  
 $T_a$  absence  
 Conduction system



PQ interv.  
 0,16

QRS  
 0,1

QT  
 0,3

**HR – dependent**

Atrial depol.

Ventricular complex

(depol.)

(repol.)

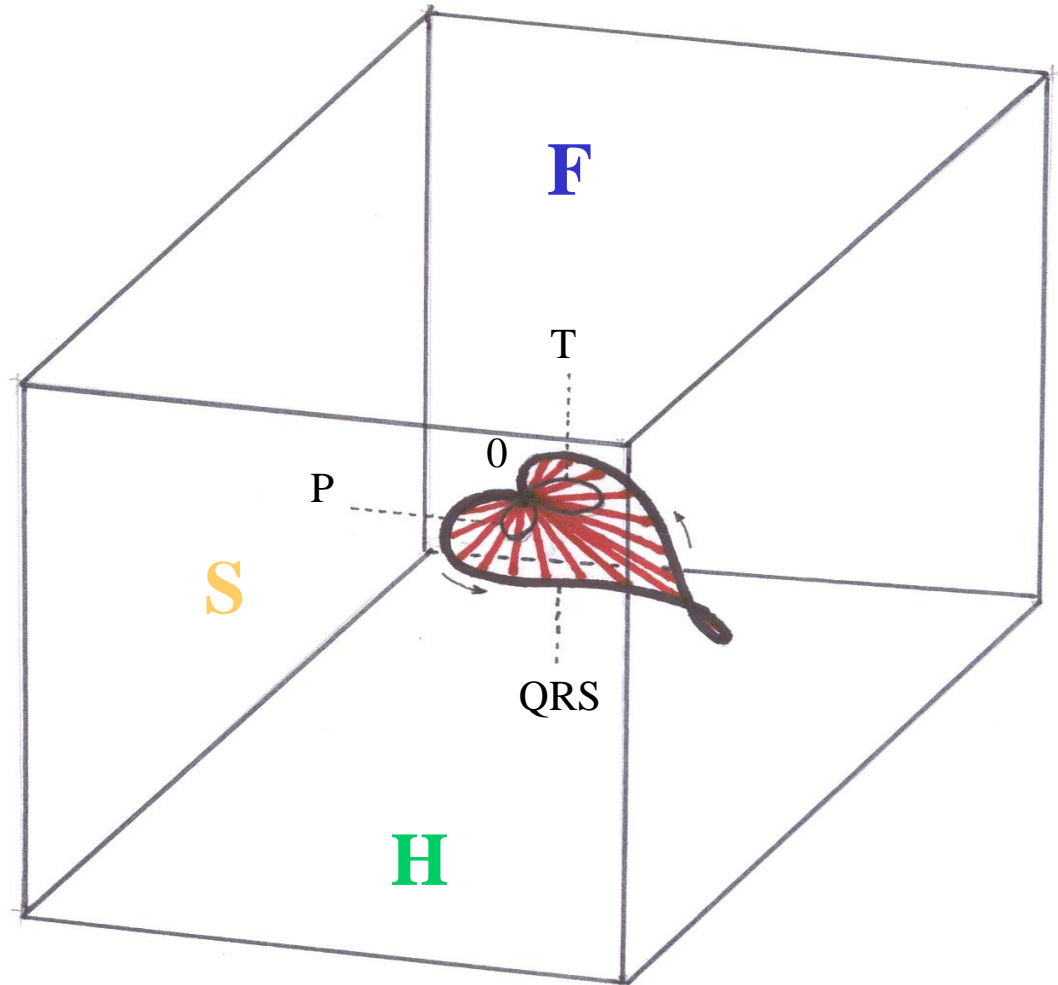
## ECG gives information about:

1. **Frequency** (changes of HR in SA node or arrhythmias, sick sinus syndrome)
2. **Conduction** (blocks – SA, AV)
3. **Rhythm** (ES – supraventricular, ventricular)
4. **Ventricular gradient** (relationship between depolarization and repolarization: origin – metabolic, hemodynamic, anatomic, physical...ischemia, hypertrophy, dilatation, cardiomyopathy, inflammations, changes in electrolytes, drugs...)

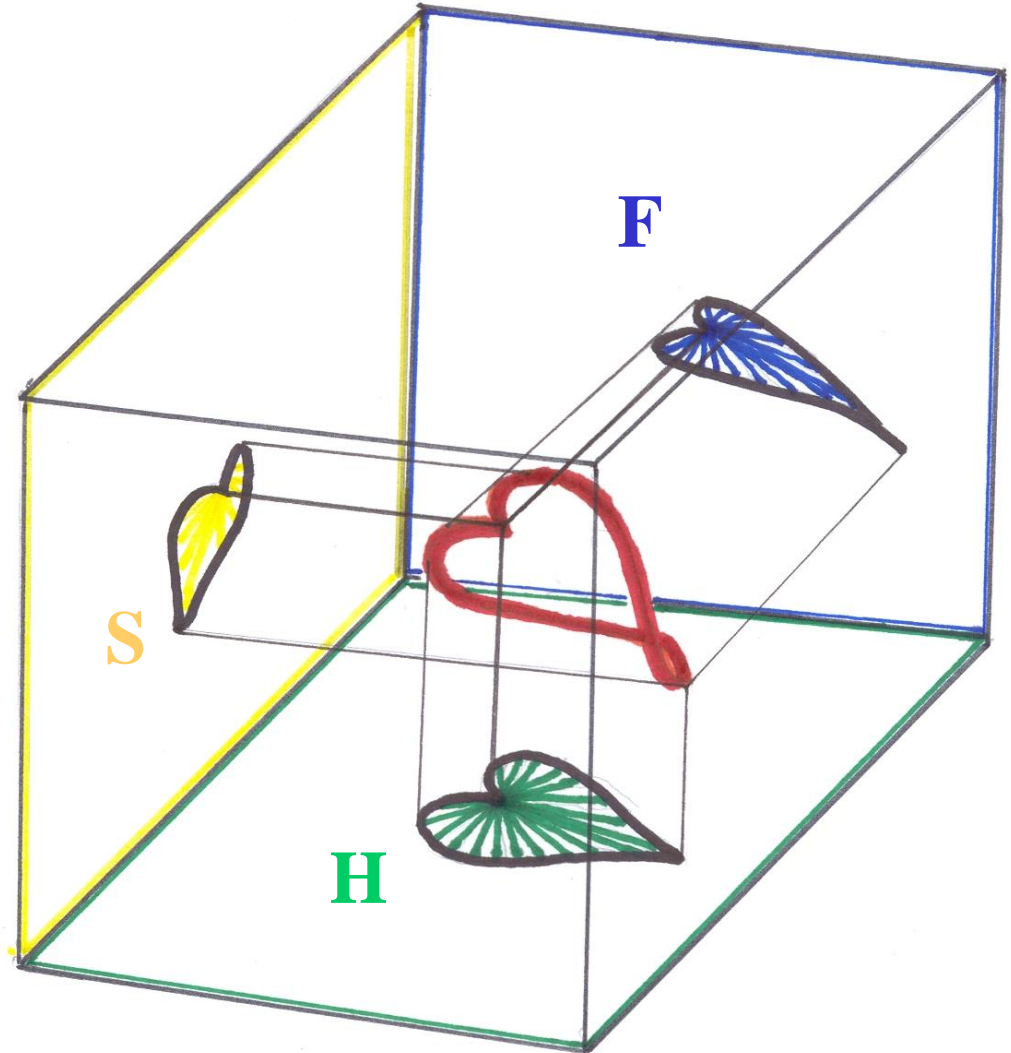
# 3D LOOPS OF ELECTRICAL AXIS

**F** – frontal plane  
**S** – sagittal plane  
**H** – horizontal plane

0 – elektrický střed srdce  
P – síňová depolarizace  
QRS – komorová depolarizace  
T – komorová repolarizace



# 2D PROJECTION OF HEART AXIS



**F** – frontal plane

**S** – sagittal plane

**H** – horizontal plane



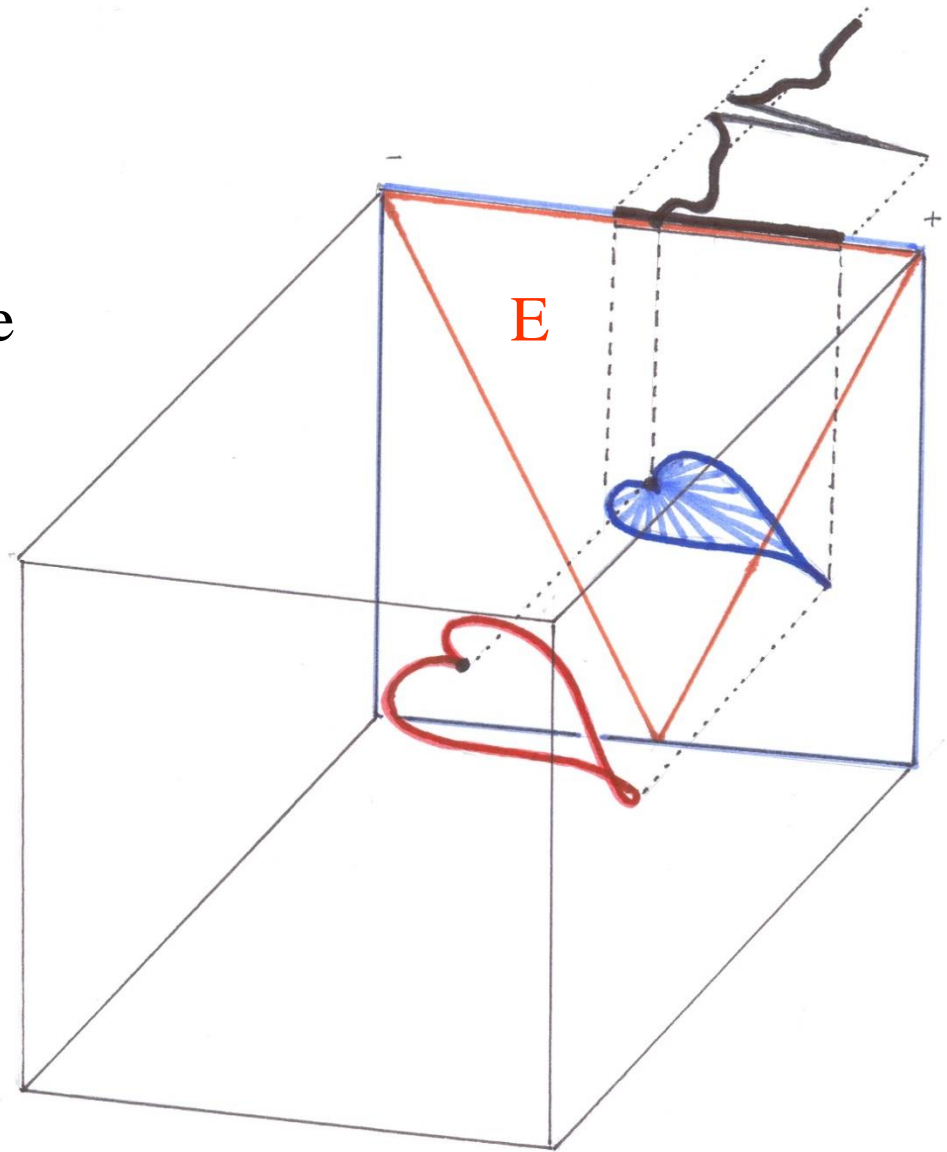
# 1D PROJECTION OF HEART AXIS

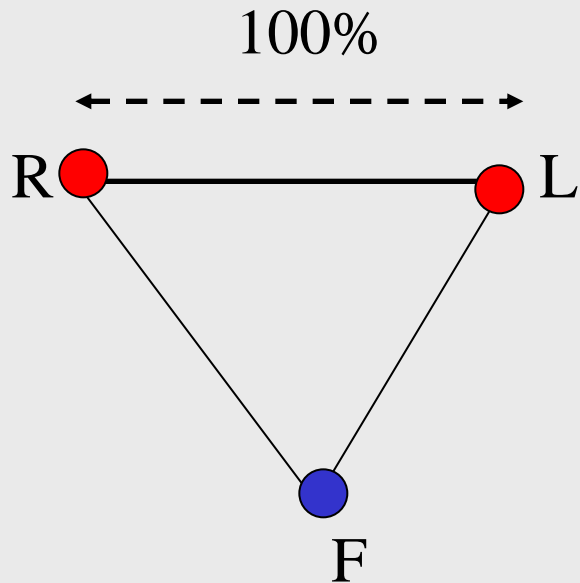
Projection on the chest surface  
into frontal plane (2D)

And its projection to line  
(1D), axis of the I. ECG lead

**in time**

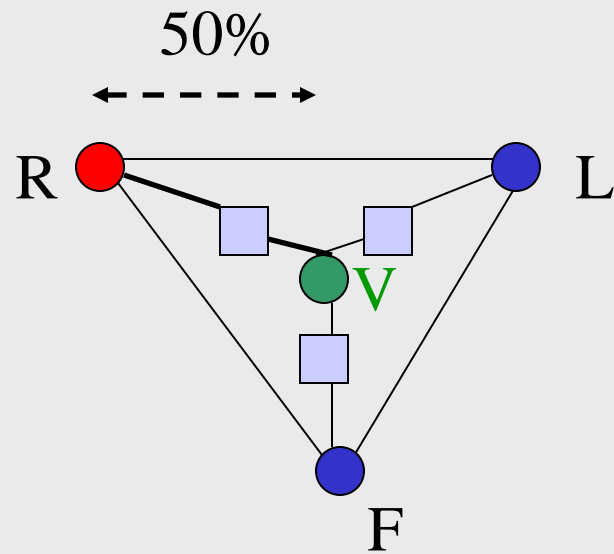
E – Einthoven triangle



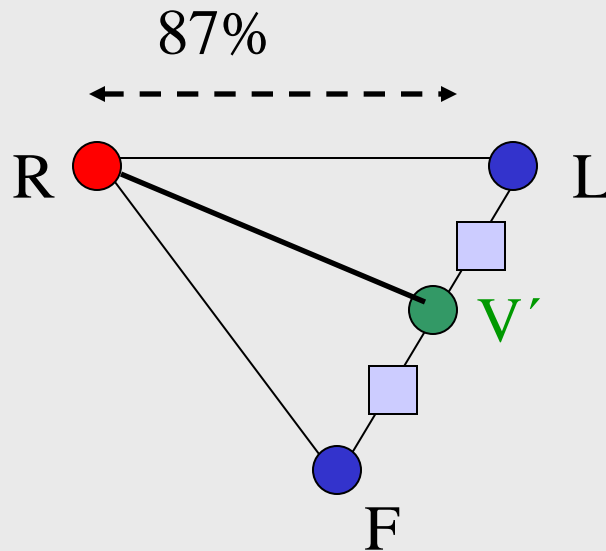


Einthoven, 1913

I, II, III

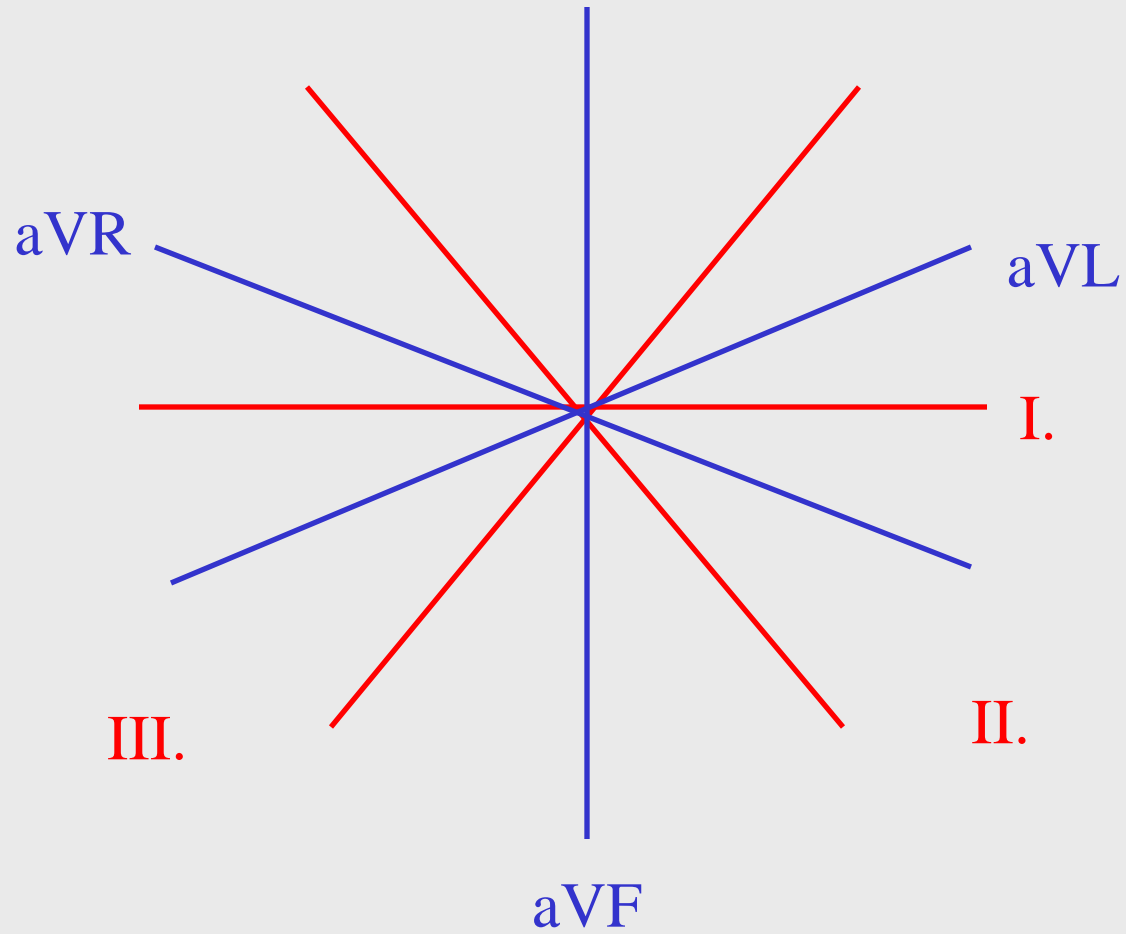


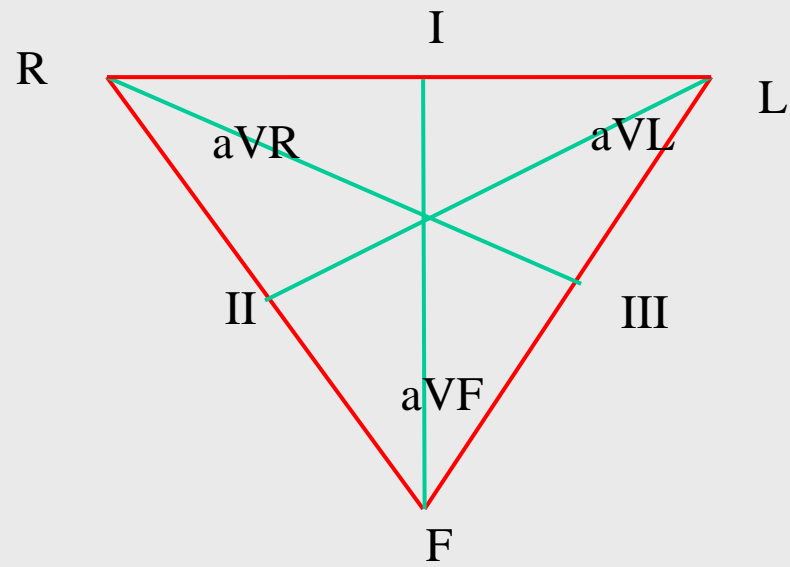
Wilson, 1934, VR, VL, VF



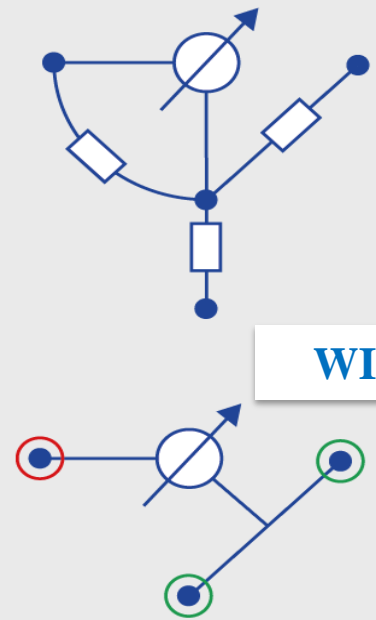
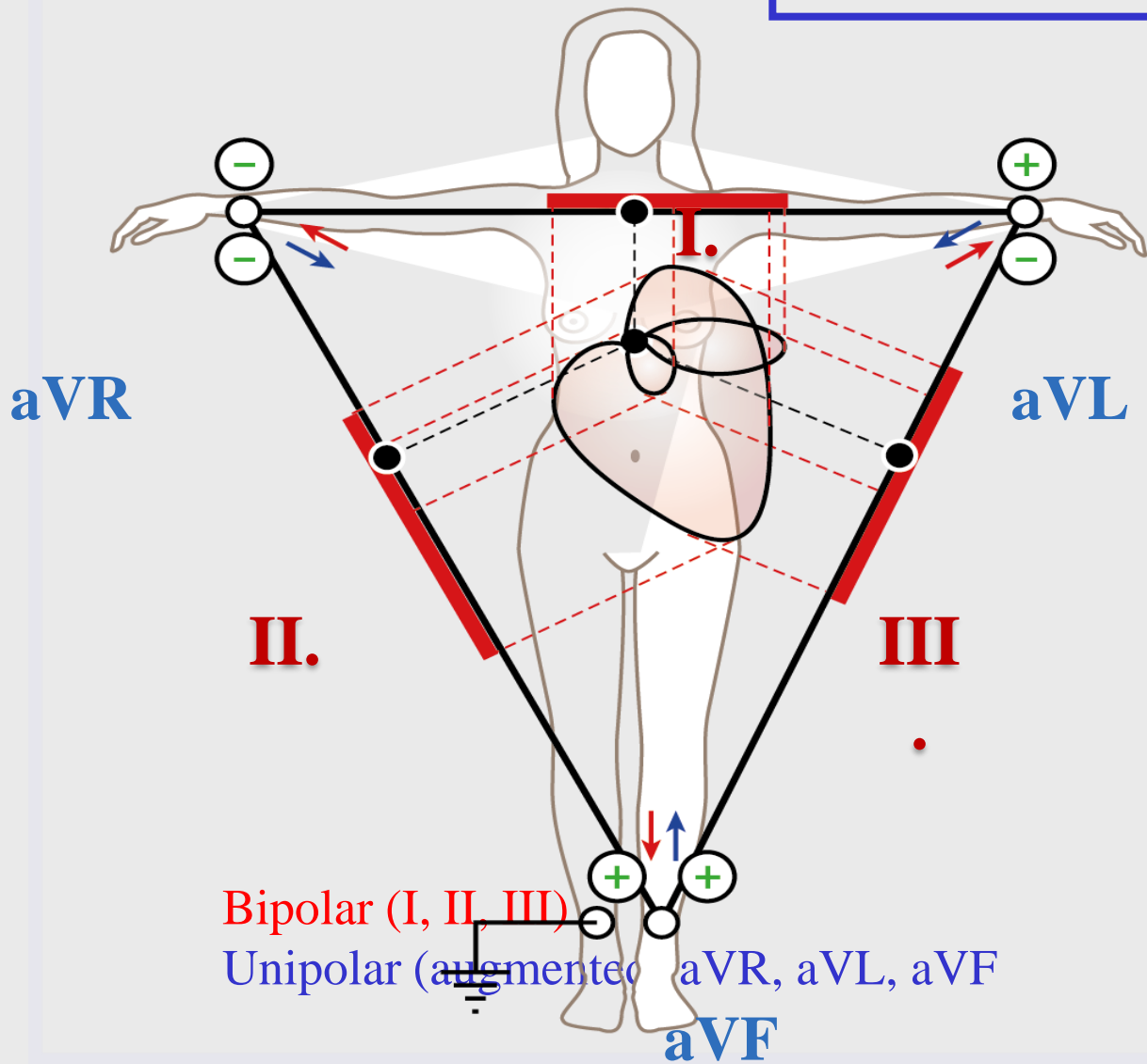
Goldberger, 1947, aVR, aVL, aVF

# HEXAAXIAL SYSTEM





**LIMB LEADS**

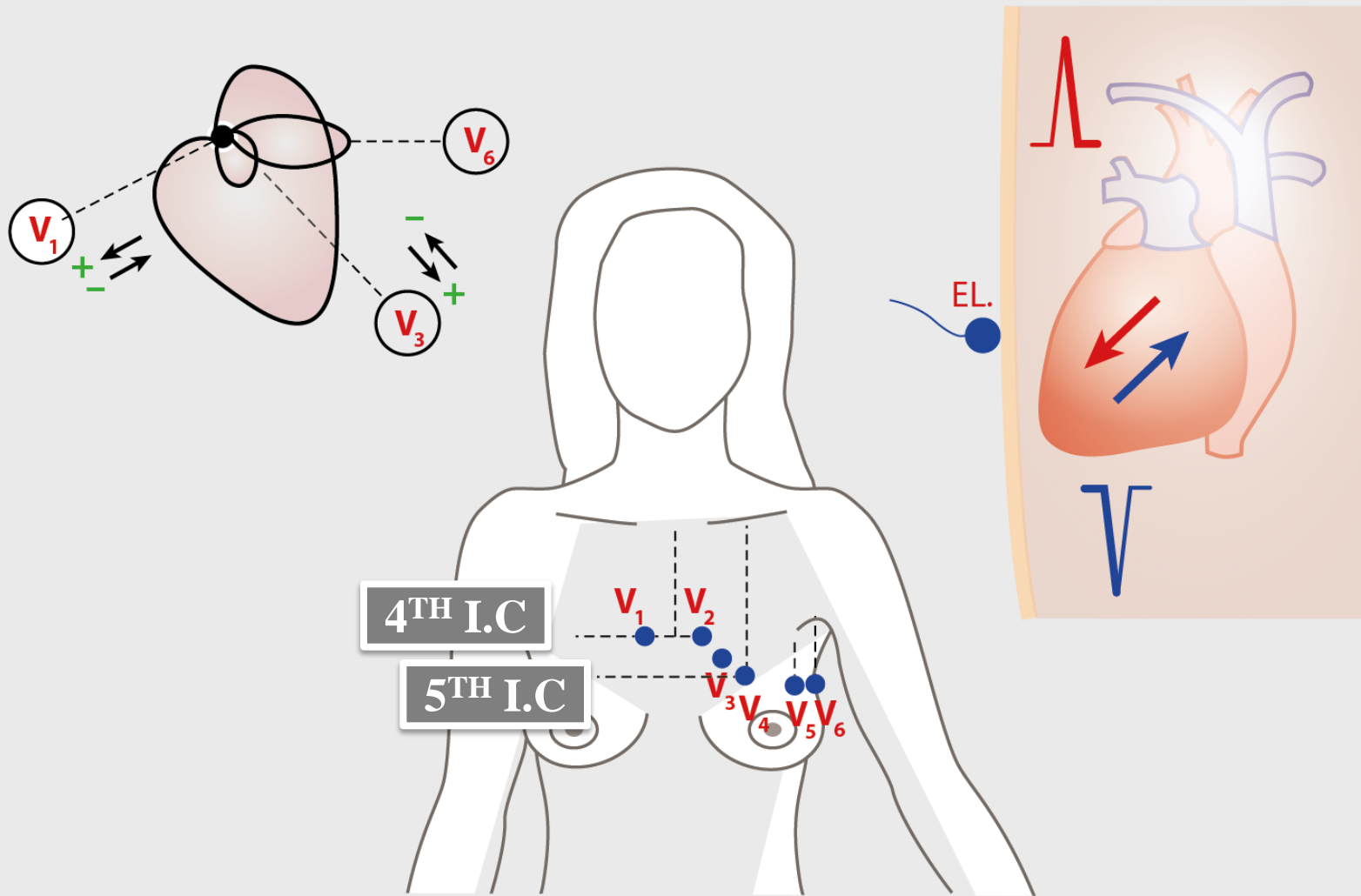


**WILSON**

**GOLDBERG**  
augmented

Frontal projection of vector!

# CHEST LEADS



Horizontal projection of vector!

# PROJECTION PLANES OF CARDIAC VECTOR AND ECG LEADS

## Frontal plane

limb leads

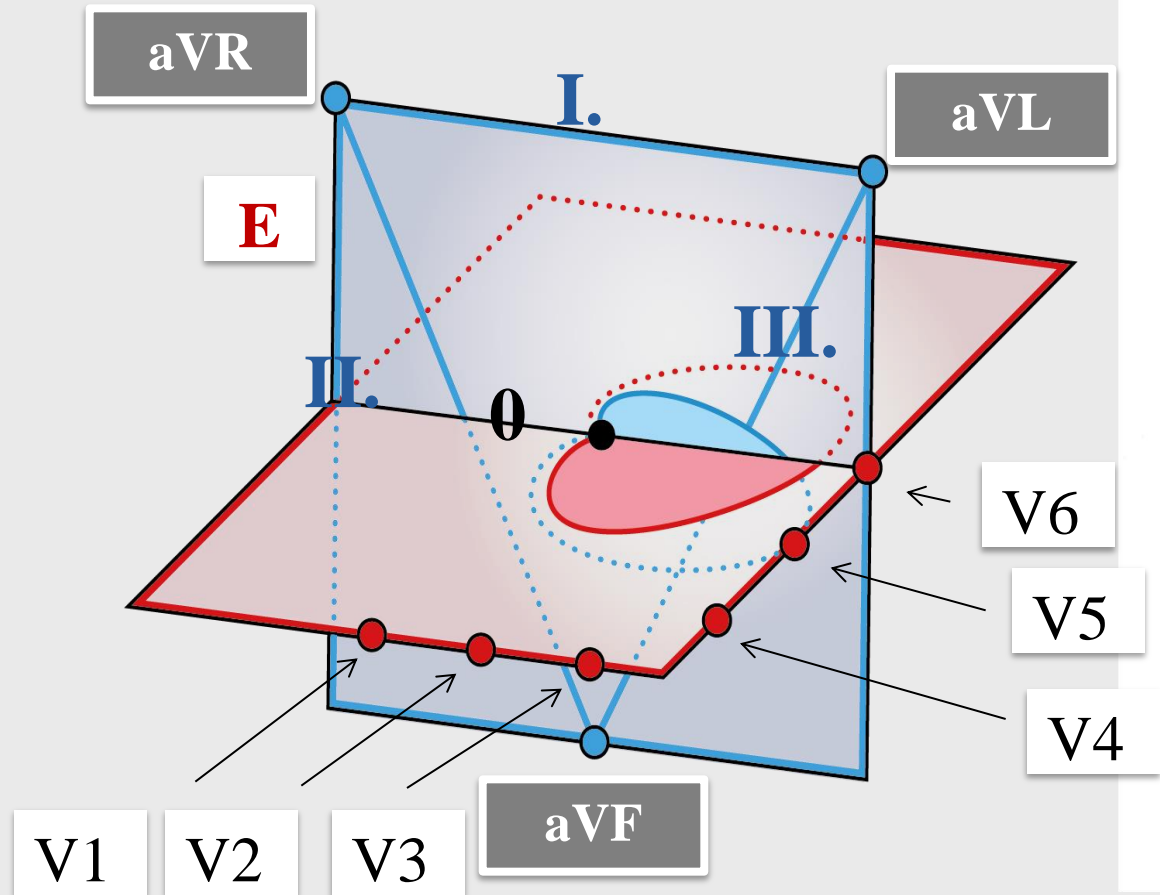
I., II., III., aVR, aVL, aVF

## Horizontal plane

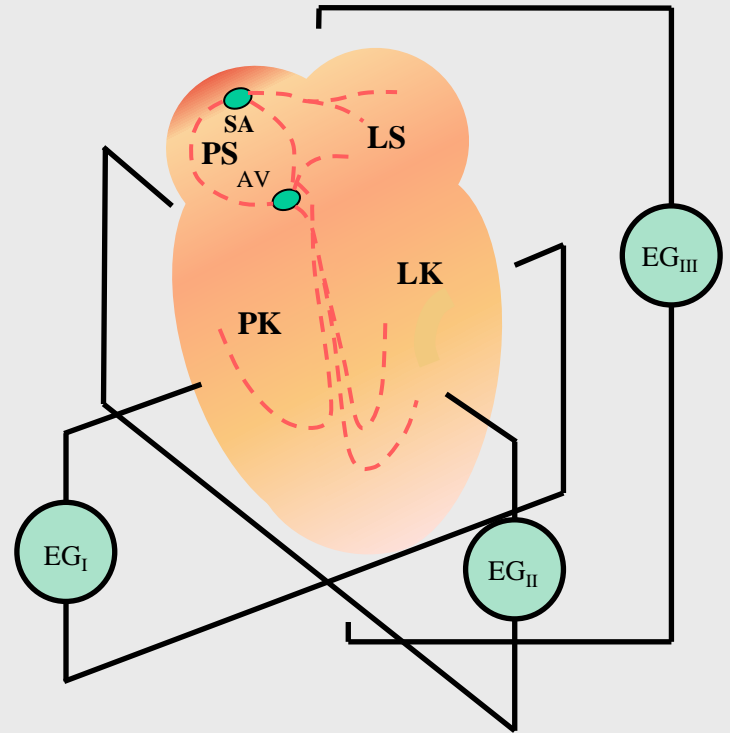
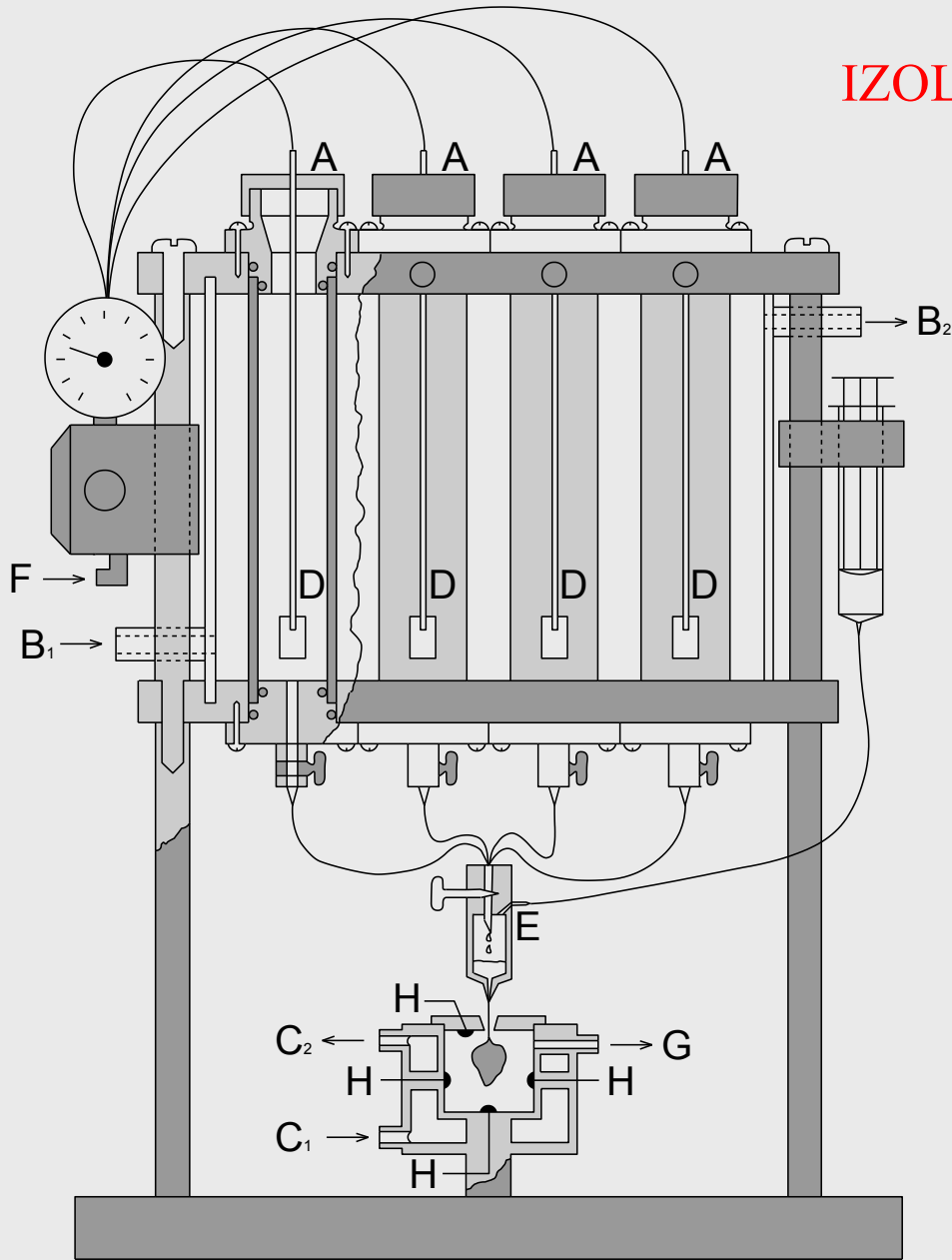
V1 – V6

Both planes are shifted into the level of electrical centre of the heart (0)

E – Einthoven triangle



# IZOLOVANÉ SRDCE dle Langendorffa



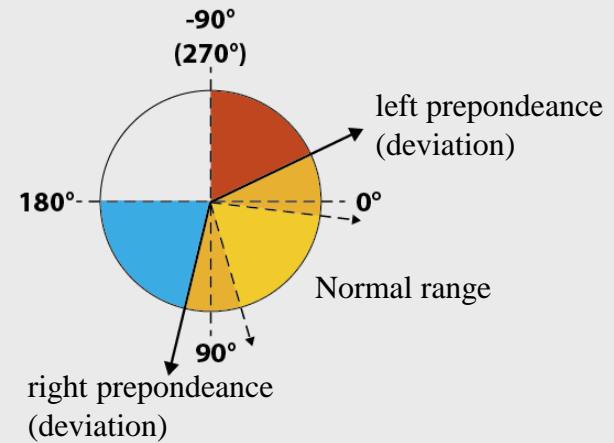
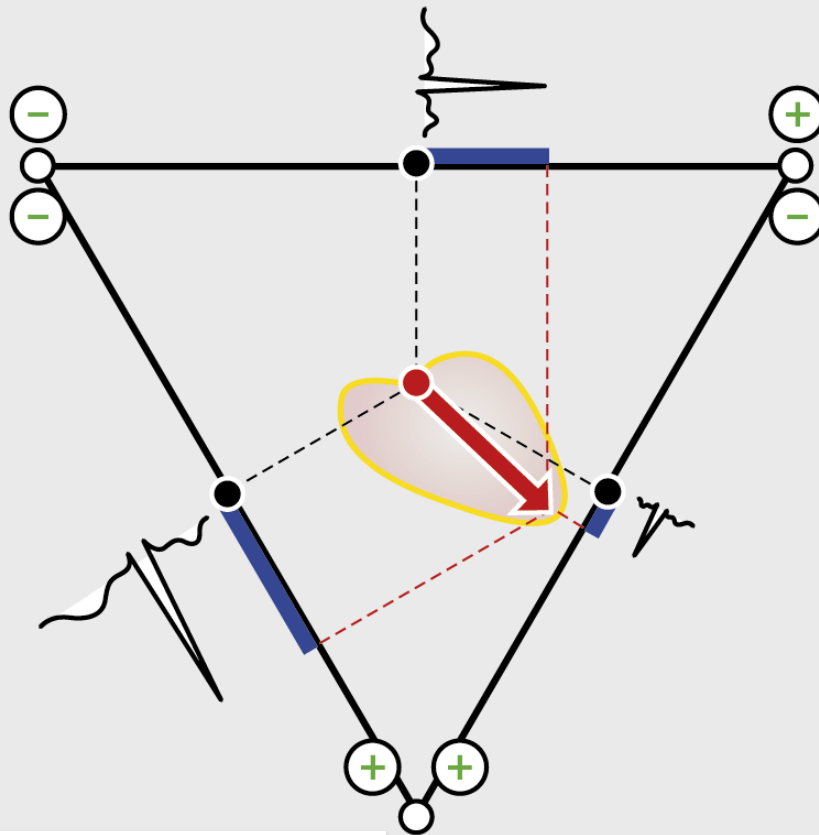


## ELECTRICAL AXIS OF THE HEART

Summary of all momentary vectors, which form ventricular depolarisation loop. Expresses the direction of ventricular activation. Reflects asymmetry in ventricular wall thickness and the position of the heart in the chest.

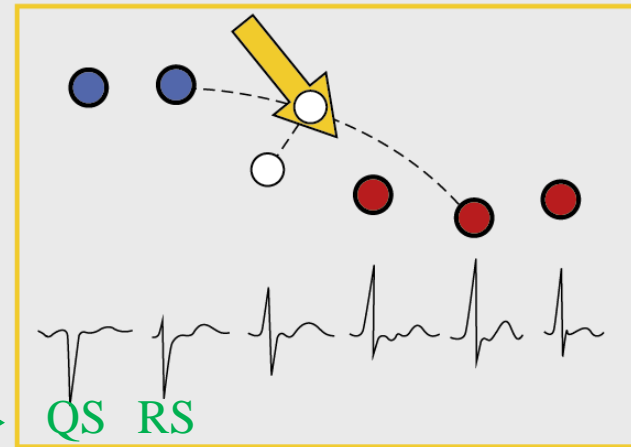
# ELECTRICAL AXIS – in the frontal plane

(R–Q–S) in lead I, II, III.

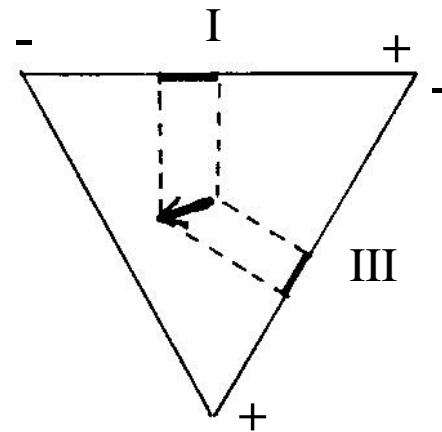
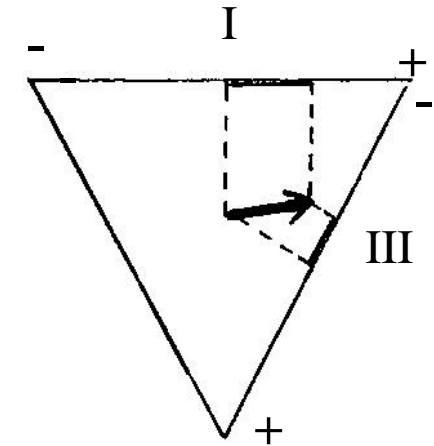
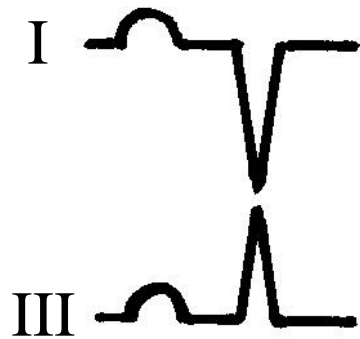
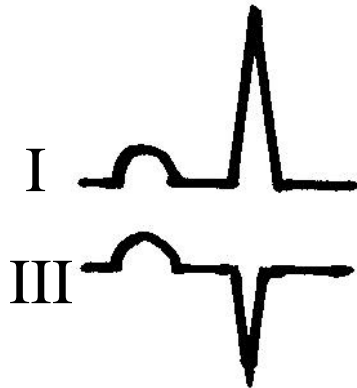


• **Eqilateral**  
Einthoven  
triangle

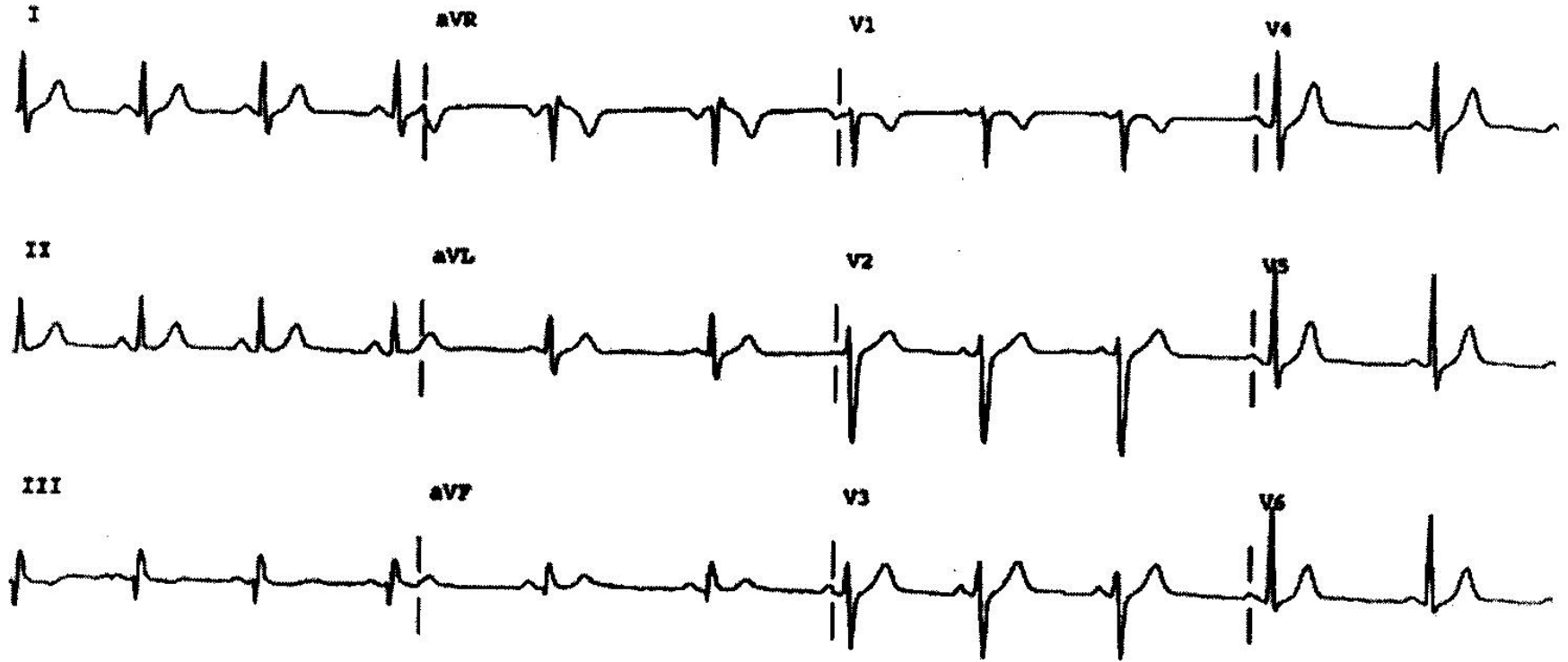
Terminology →



# LEFT DEVIATION, RIGHT DEVIATION

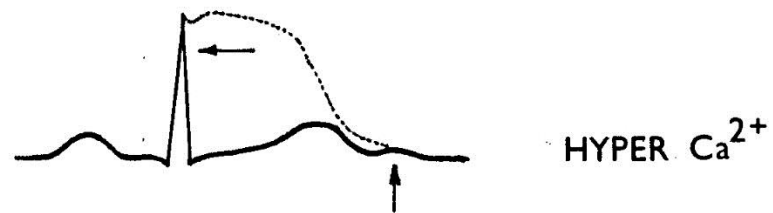
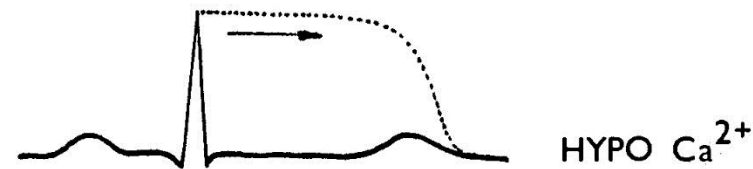
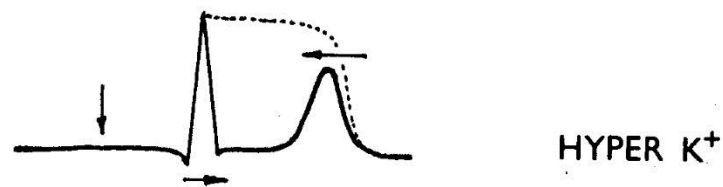
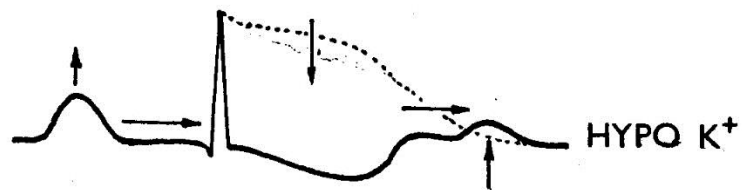
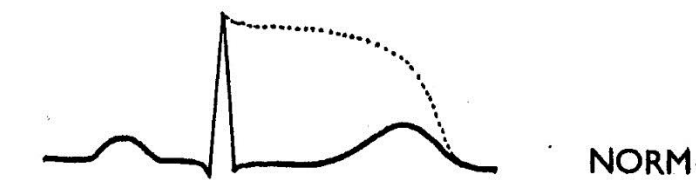
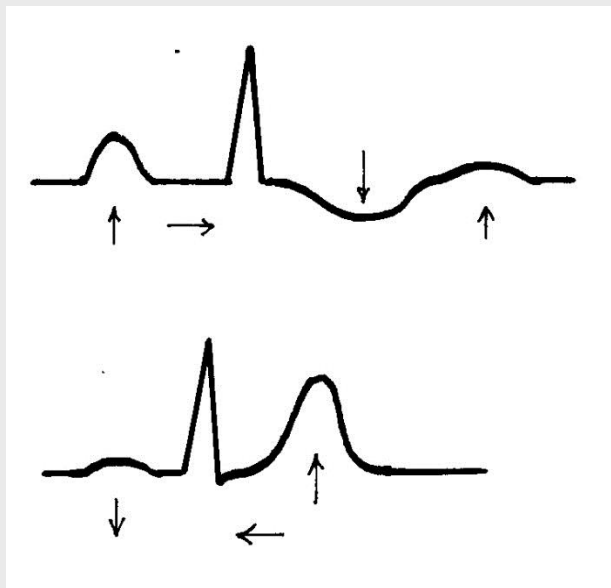


## Normal 12-lead electrocardiogram



## ECG – information about:

1. Magnitude and position of the heart (electrical axis)
2. Site of impulse origin (P, QRS)
3. Conduction path (P-Q, QRS)
4. Impulse regression (T)
5. Rhythm (P-P, R-R)
6. Action potential alterations (ST, T)
7. Effect of drugs, remedies, ion composition changes,...



# RESPIRATORY (SINUS) ARRHYTHMIA

1847, Ludwig, ECG and breathing of dog – respiratory sinus arrhythmia

Detectable already during prenatal life.

Present in numerous species in animal kingdom – in all vertebrates.

Physiological meaning ???? STABILISATION OF MEAN BP (protection against mechanical effect of intrathoracic pressure on arterial BP)

Key effect of parasympathetic NS (decrease of its tonus), sympathetic NS only modulates!!!

## MECHANISMS:

- 1) CENTRAL
- 2) REFLEXES FROM LUNGS
- 3) REFLEXES FROM BARORECEPTORS
- 4) REFLEXES FROM RECEPTORS IN THE RIGHT ATRIUM
- 5) LOCAL EFFECTS ON SA NODE
- 6) EFFECT OF OSCILLATIONS OF pH,  $paO_2$ ,  $paCO_2$

# Central mechanisms

- Central generator of RSA
- Respiratory neurons in medulla oblongata hyperpolarise preganglionic vagal neurons
- Vagal tonus decreases during inspiration – HR increases

## Reflexes from lungs – inflation reflexes

- Stimulation of vagal stretch-receptors during inspiration  
supresses inspiratory centre and also cardio-inhibitory  
centre in medulla oblongata



## **Reflexes from baroreceptors**

- Diverse opinions about the effect of arterial baroreceptors on RSA
- Fluctuation of sensitivity of baroreceptors during respiratory cycle

## **Reflexes from receptors in the right atrium**

- Bainbridge, 1915
- Reflex increase of HR during atria stretching
- Applicable in explanted (denervated) heart

## Local effects on SA node

- Stretching of SA node causes faster spontaneous depolarisation
- Effect of mechanosensitive chloride channels
- Changes of SA node perfusion (a. centralis) and possible compression of SA node by expanding lungs

## Effect of pH, $p_aO_2$ and $p_aCO_2$ oscillations

- Oscillatory activity of peripheral chemoreceptors contributes to formation of RSA and increases its amplitude

**ARRHYTHMIAS** = disturbance of impulse generation or conduction

## RHYTHM and FREQUENCY:

### Regular

- 1) Normal HR range: 70 – 220 bpm; effect of age)
- 2) Sinus tachycardia (60 - 100 bpm; exercise; aging)
- 3) Sinus bradycardia (below 60 bpm; athletes' heart)
- 4) Nodal rhythm - below 40 bpm, ventricular rhythm - below 20 bpm)

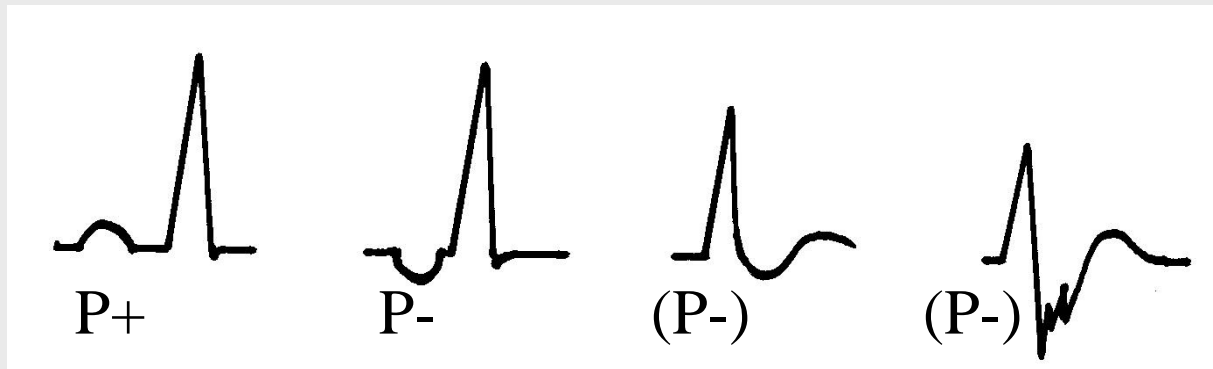
### Irregular

1. sinus respiratory arrhythmia (**physiological**)
2. Sick sinus syndrome
3. Extrasystoles (ES) single or coupled (bigeminy, trigeminy), according to site or origin - sinus, atrial, junction, ventricular

# ARHYTHMIAS

## SITE OF ORIGIN

SINUS                  ATRIA                  JUNCTION                  VENTRICLES



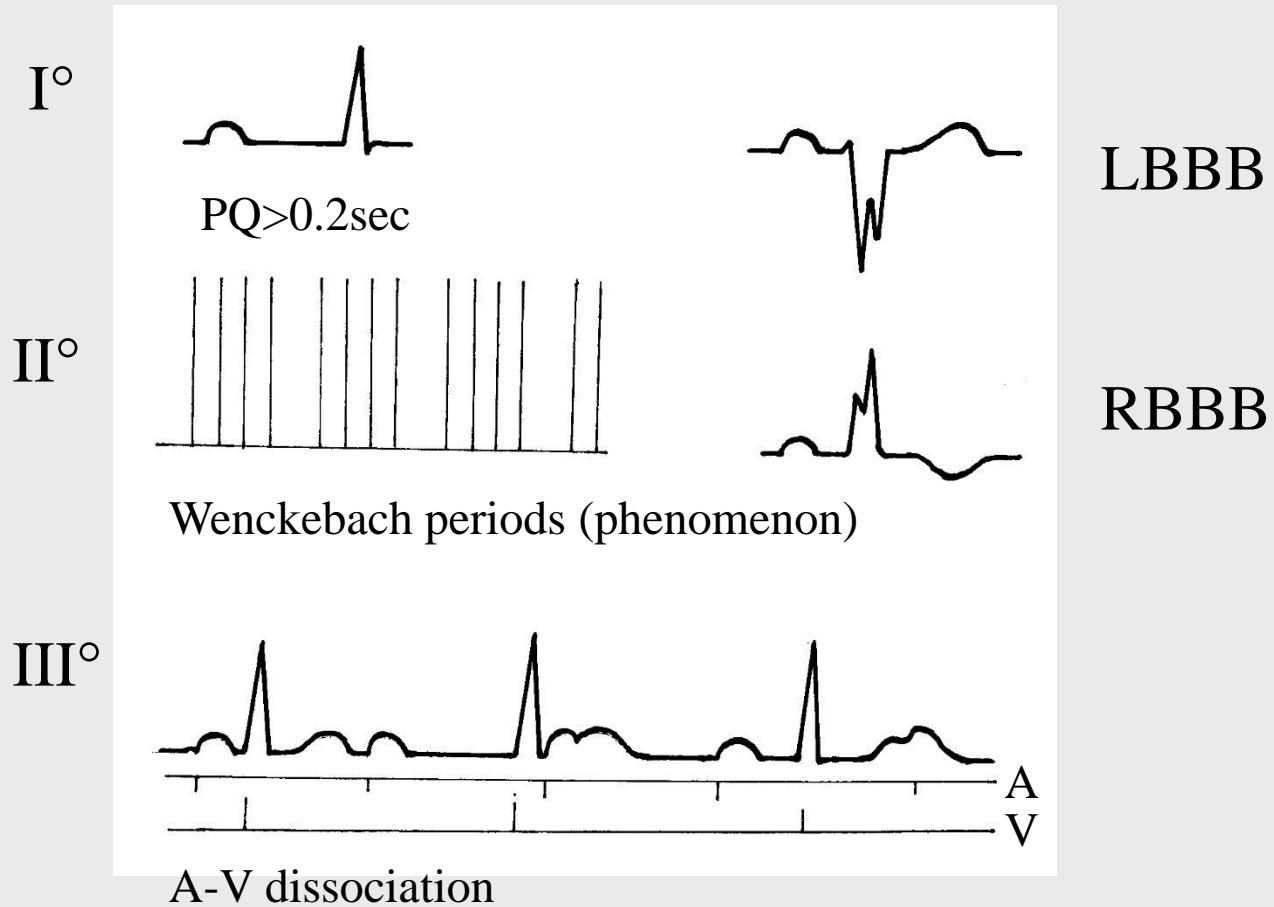
- Polarity of P wave

- PQ interval (QP)

(physiological PQ interval: 0.12 – 0.2 s)

# BLOCKS

- SICK SINUS SYNDROM
- AV BLOCKS

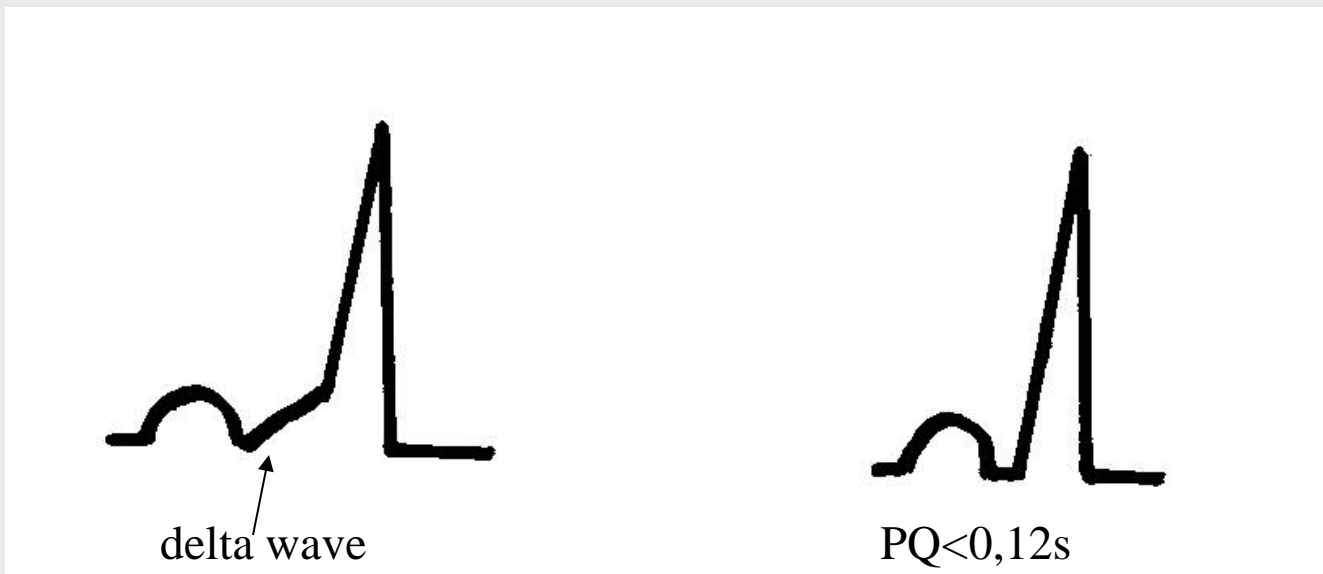


- BUNDLE BRANCH BLOCK (BBB) – LEFT, RIGHT

# PREEXCITATION

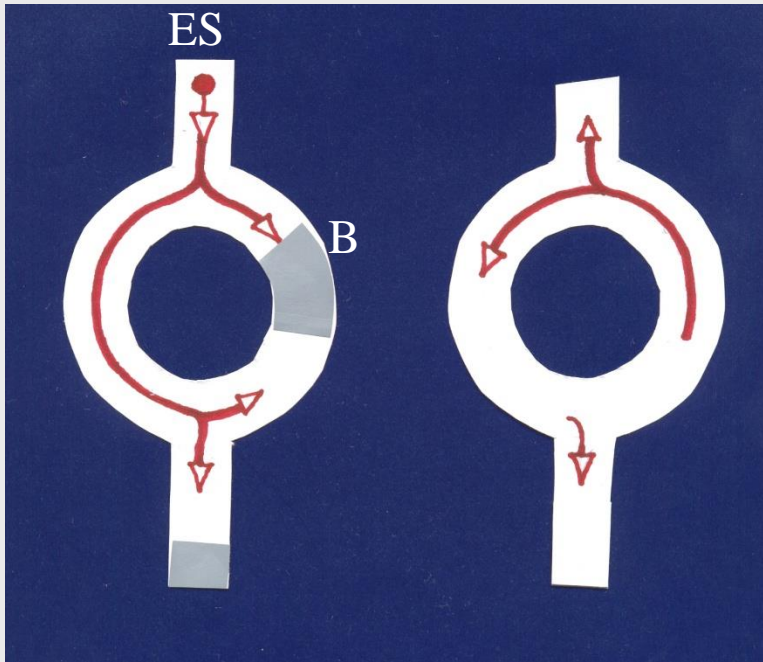
AV node is „by-passed“, fast conduction

- „short nodus“
- Wolf-Parkinson-White syndrome (WPW) – sensitive to paroxysmal tachycardia – see re-entry



# REENTRY

Common mechanism of (paroxysmal) tachycardias, extrasystoles, bigeminy, etc.



- Double pathway  
Diverging and converging of excitation pathways
- Unidirectional block
  1. Long refractory period
  2. Slowed conduction
  3. Reentry

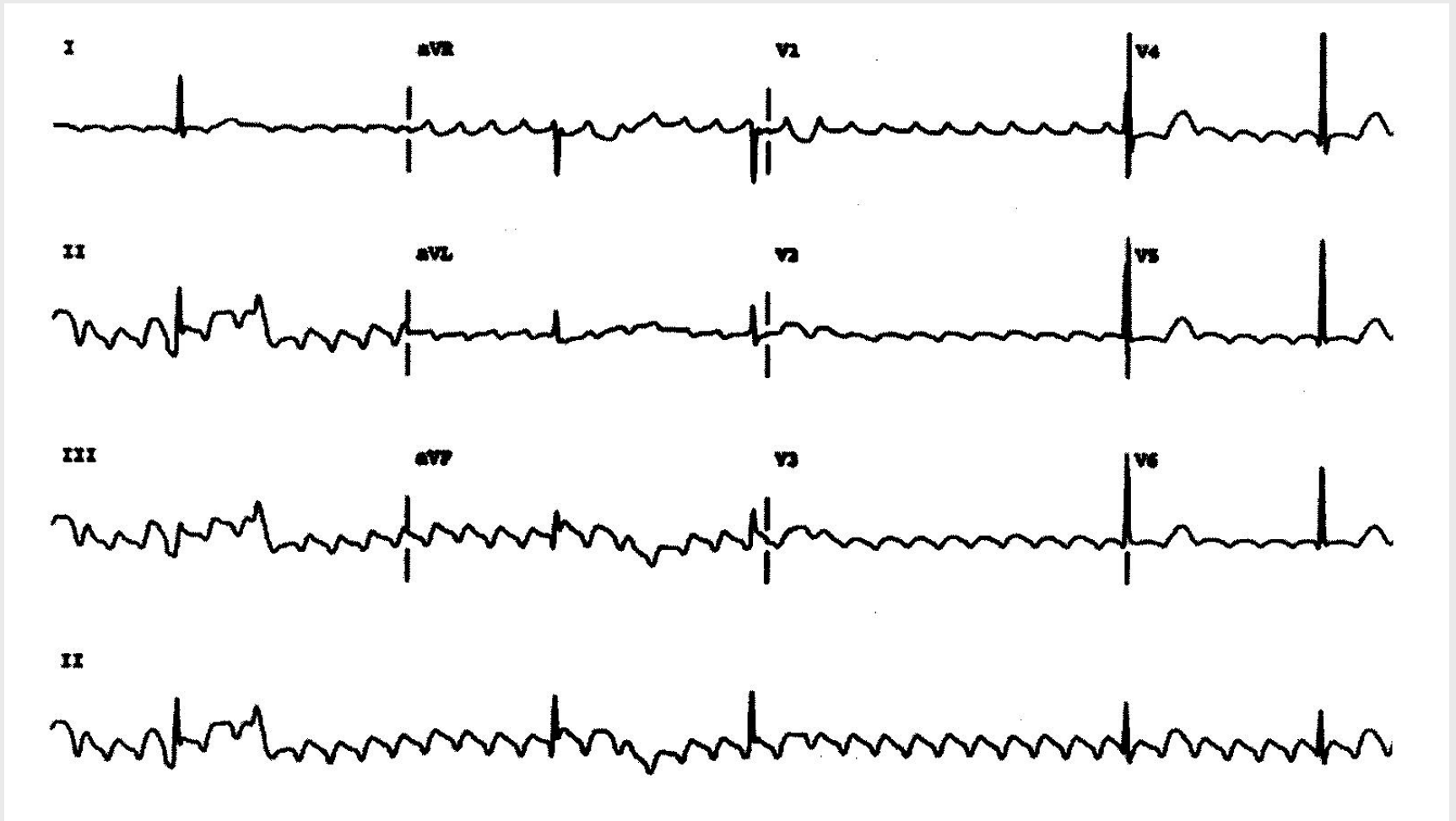
- **Loops most often at the level of AV junction**
- **Determinants of re-entry:**
  1. Proper dimension of the loop
  2. Proper timing of the trigger ES

# TACHYARYTHMIA

- **SINUS TACHYCARDIA**
- **PAROXYSMAL TACHYCARDIA** (supraventricular, ventricular)
- **FLUTTER** (>250/min; atrial)
- **FIBRILLATION** (>600/bpm; **atrial, ventricular; breakdown of electrical homogeneity**)



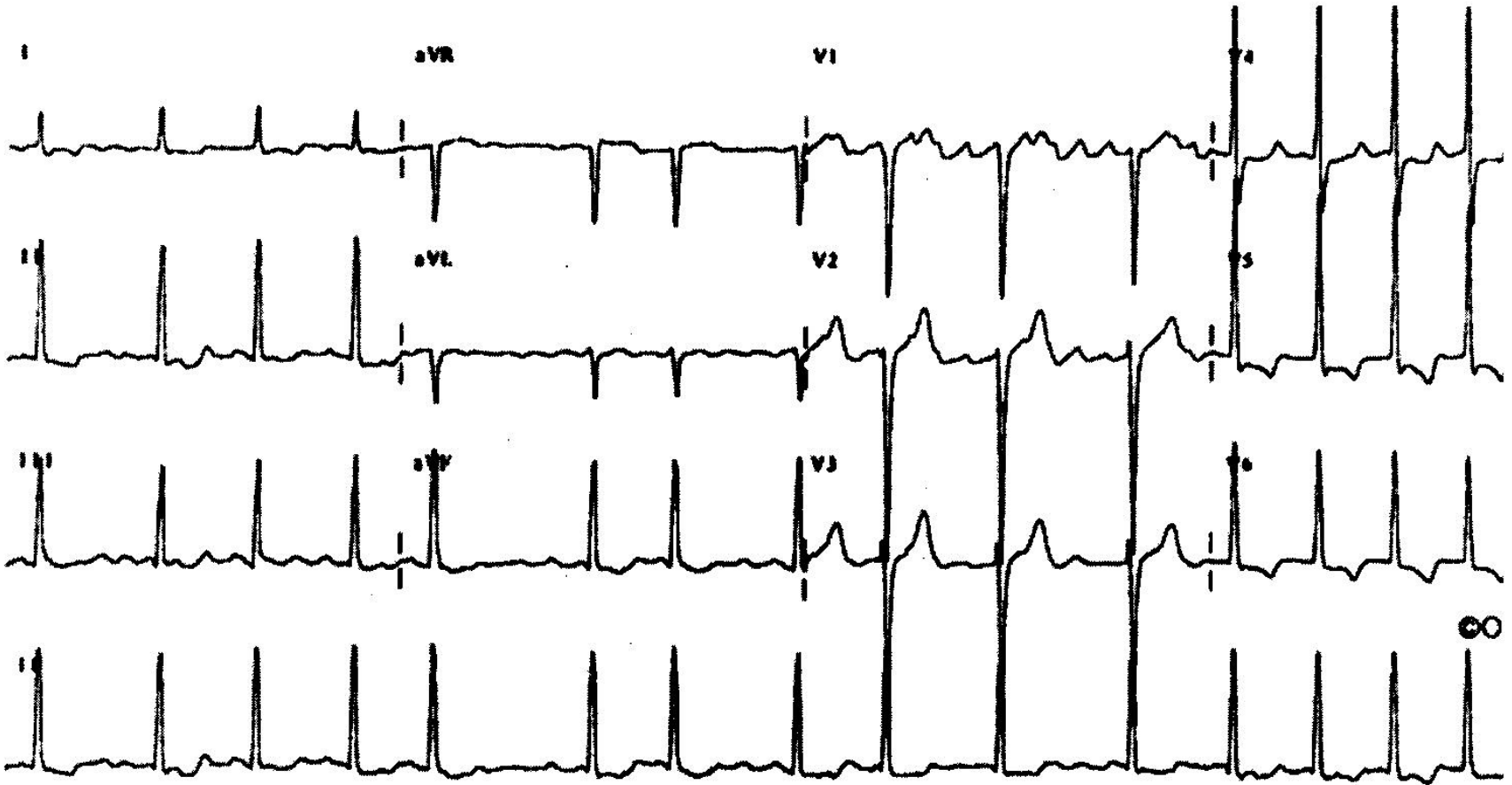
# ATRIAL FLUTTER



Frequency 250 – 600/bpm

Atrioventricular block n:1

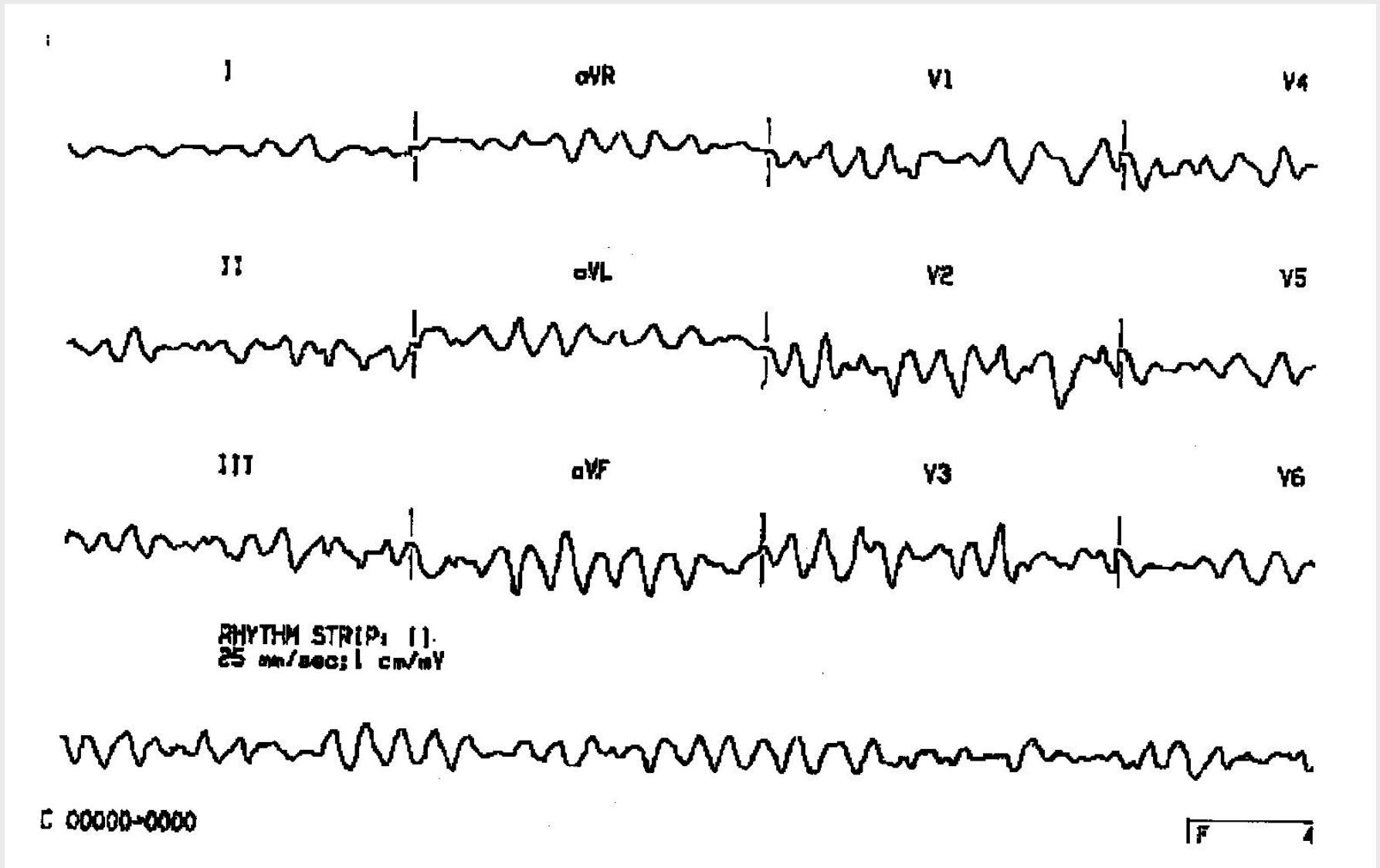
# ATRIAL FIBRILLATION



Irregular ventricular rhythm

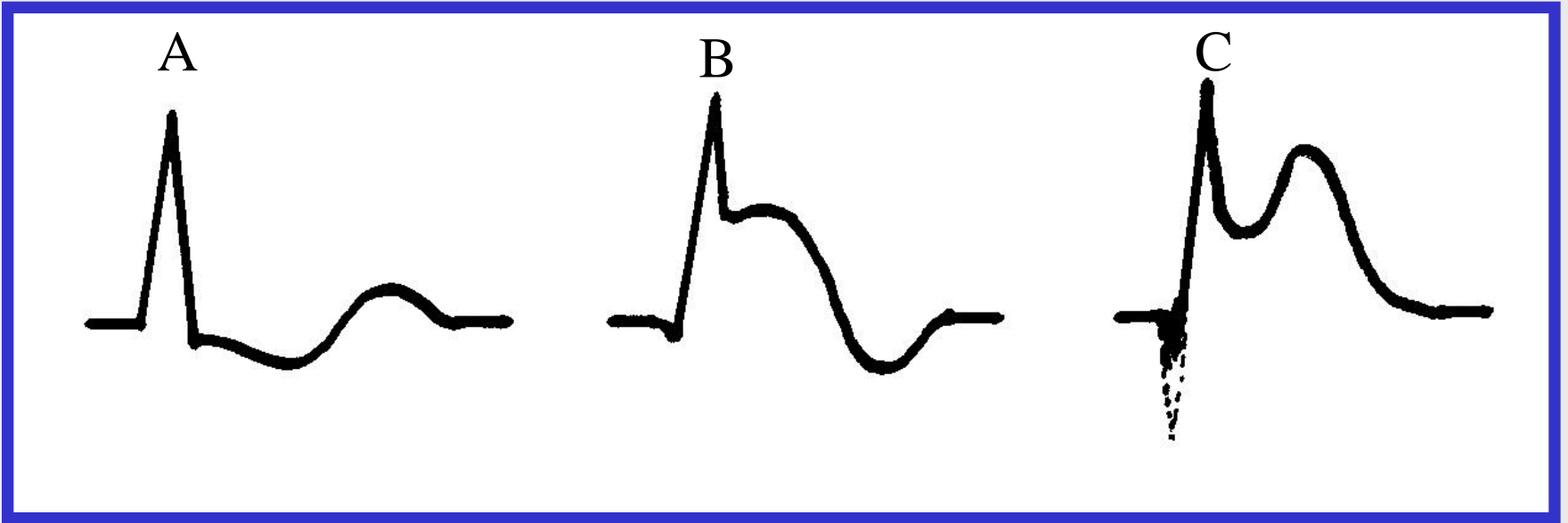
+ f-waves

# VENTRICULAR FIBRILLATION



Frequency above 600/bpm, **LETHAL**

## HEART ISCHEMIA



A: exercise angina pectoris

B: acute non-Q myocardial infarction

C: acute Q myocardial infarction

# ANTIARRHYTHMICS

- BLOCKERS OF Na CHANNEL – prolong inactivation of  $I_{Na}$ , e.g. refracterity, „blocking“ fast ways
- BLOCKERS OF Ca CHANNELS – „blocking“ fast ways
- BLOCKERS OF K CHANNEL – prolonging refractory period
- $\beta$ -SYMPATOLYTICS – slowing HR

Schémata a animace zpracovalo

**Servisní středisko pro e-learning na MU**

<http://is.muni.cz/stech/>

CZ.1.07/2.2.00/28.0041

Centrum interaktivních a multimediálních studijních opor pro inovaci výuky a efektivní učení



INVESTICE DO ROZVOJE VZDĚLÁVÁNÍ