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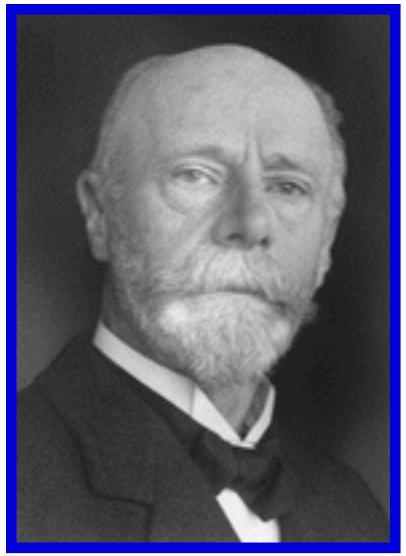
MED

ELECTROCARDIOGRAPHY. ARRHYTHMIAS.

1 Marie Nováková, Department of Physiology, Faculty of Medicine, Masaryk University

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



1893 Einthoven introduces the term 'electrocardiogram'

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

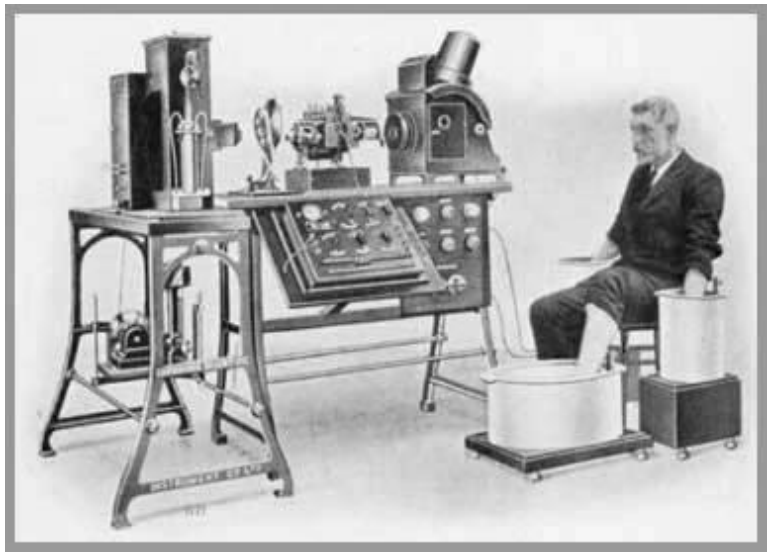
1902 Einthoven publishes the first electrocardiogram

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

1924 the Nobel prize

Willem Einthoven

1860 - 1927



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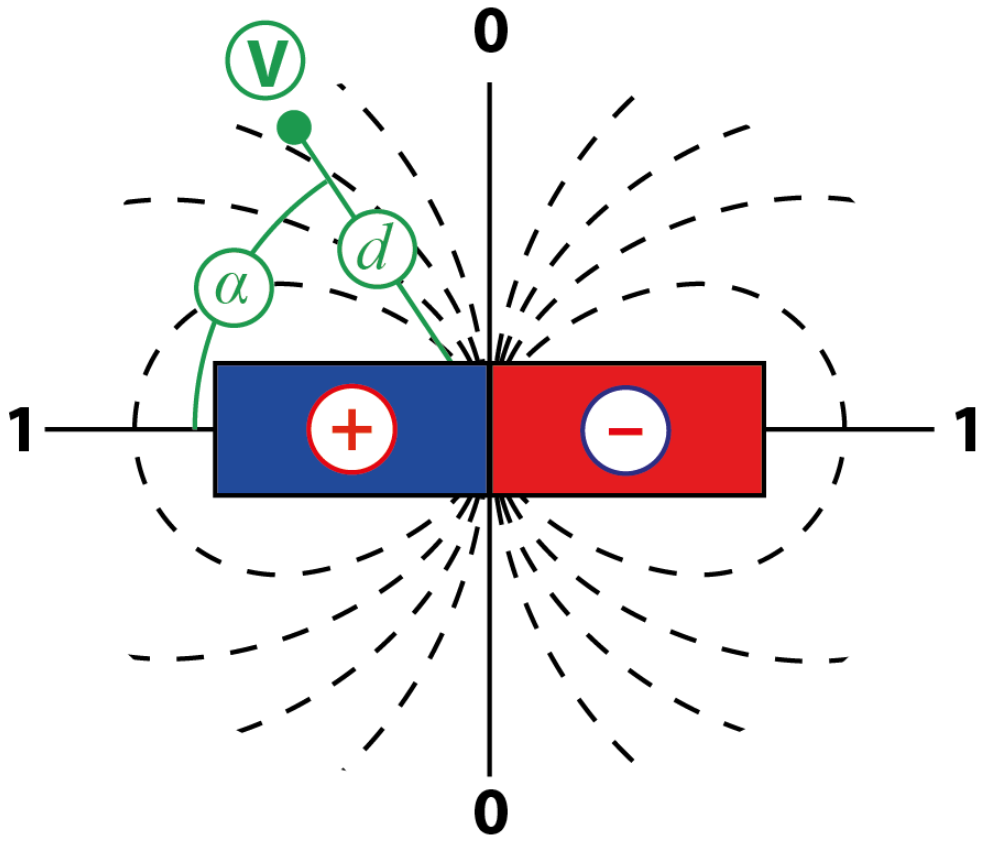
ELECTRICAL DIPOLE

stationary in homogenously conducting environment



Local currents
- Maximal in the dipole axis (1)
- Zero in the centre (0)

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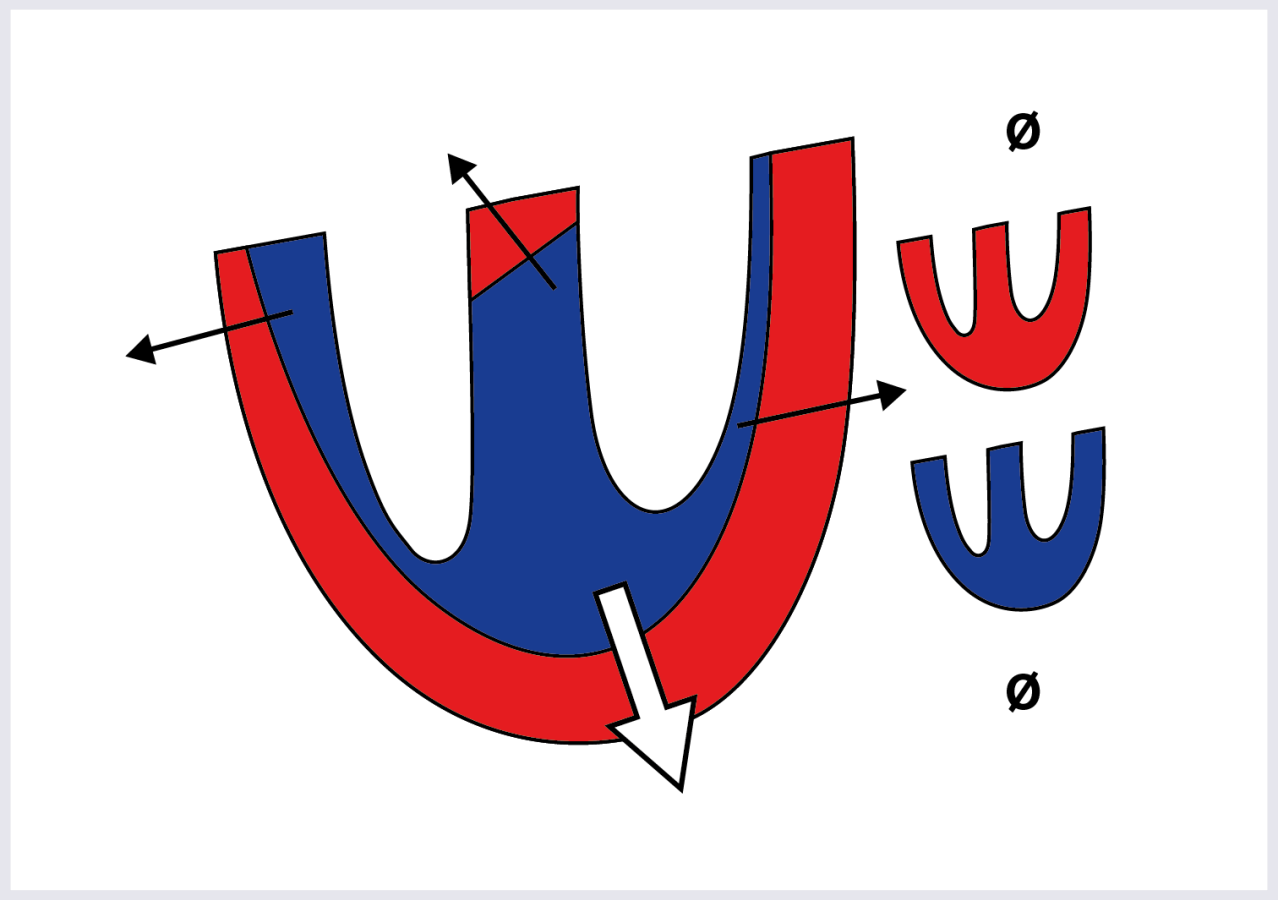
SPREADING OF DEPOLARIZATION FRONT

ELECTRICAL FIELD OF THE HEART
(can be described by a 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**

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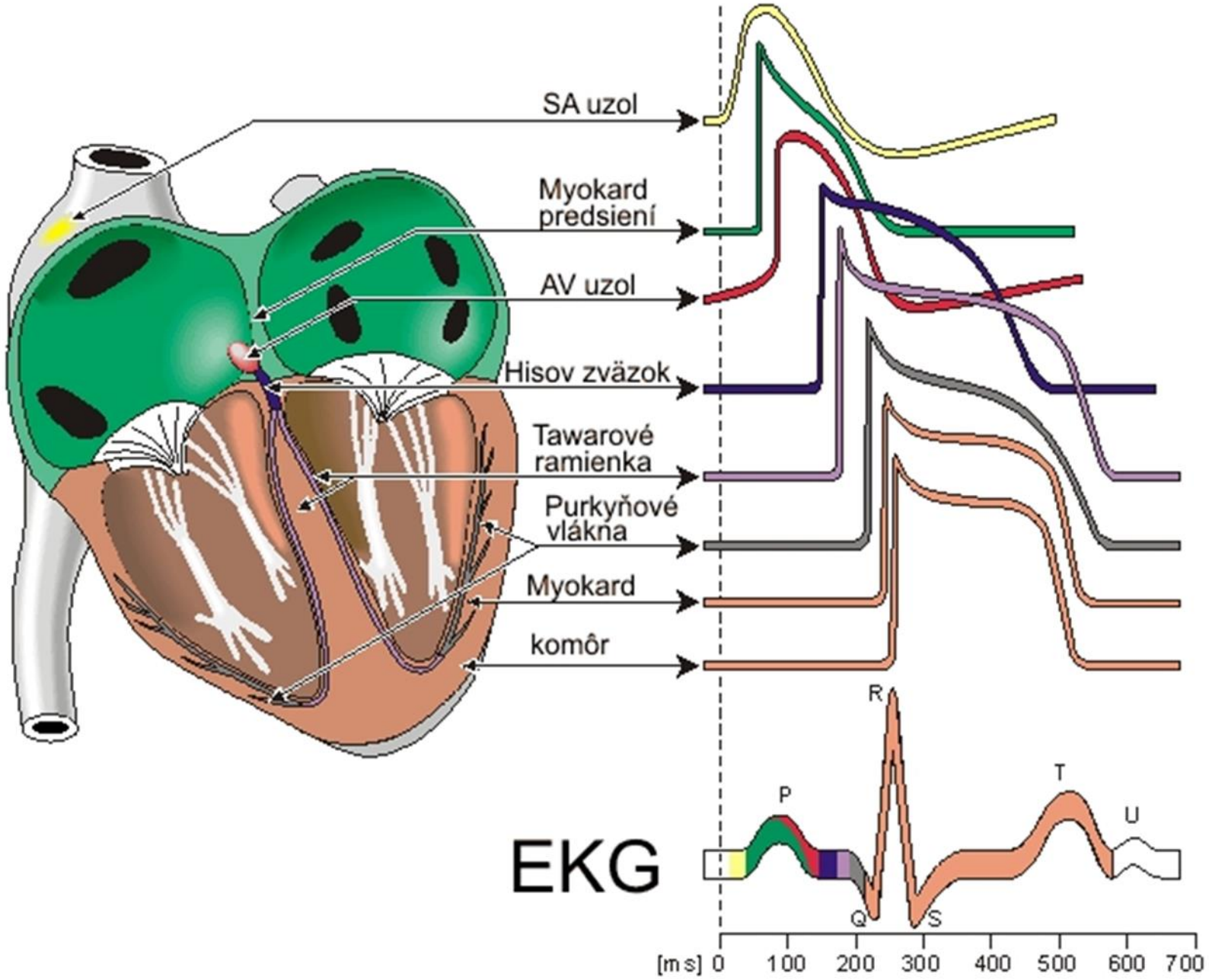
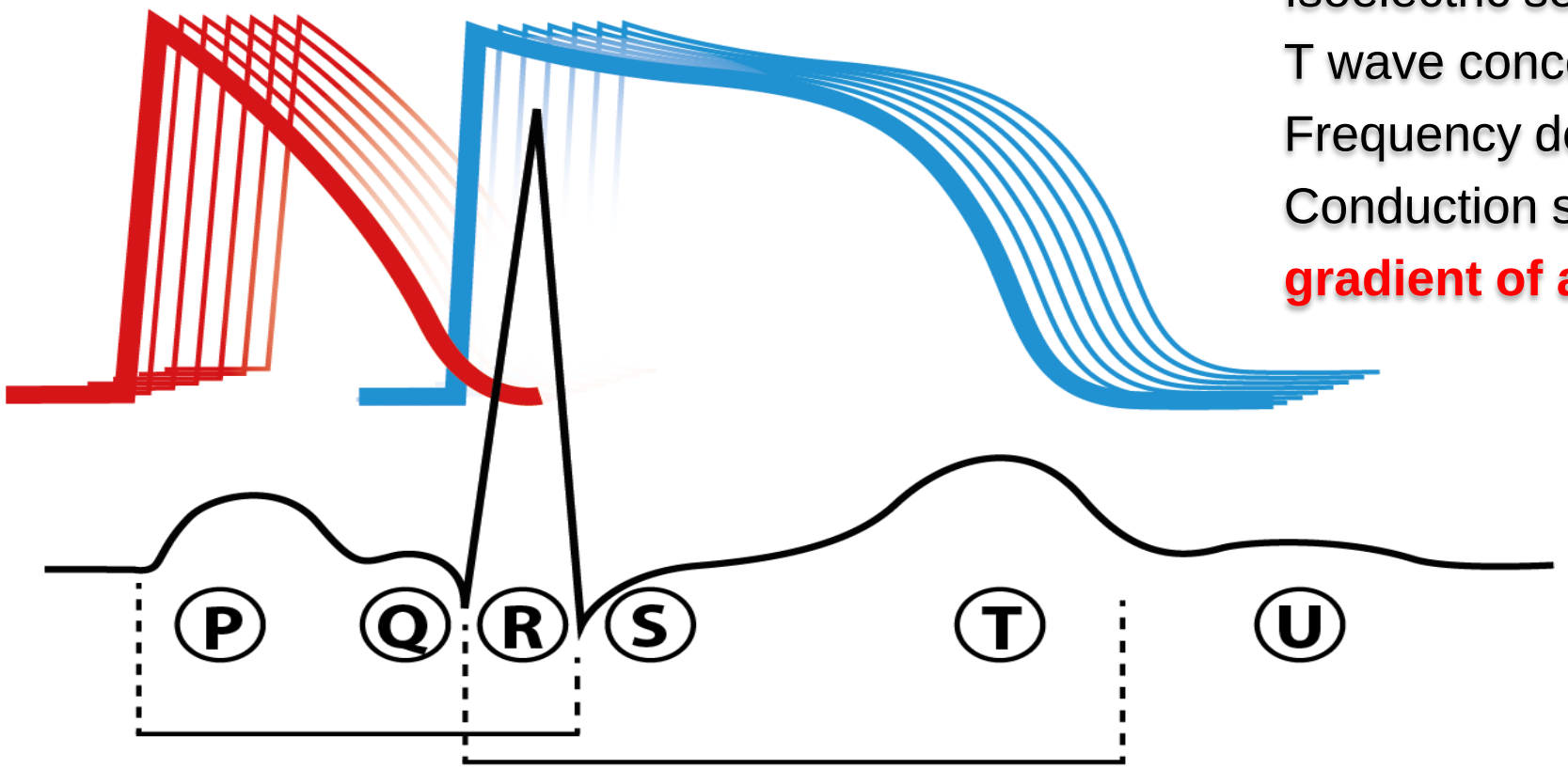

SA uzol
Myokard predsiení
AV uzol
Hisov zväzok
Tawarové ramienka
Purkyňové vlákna
Myokard komôr
EKG
R
P
T
U
Q
S
[ms] 0 100 200 300 400 500 600 700



Isoelectric segments

T wave concordance

Frequency dependence

Conduction system –

gradient of automaticity

HR – dependent

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ECG brings 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:

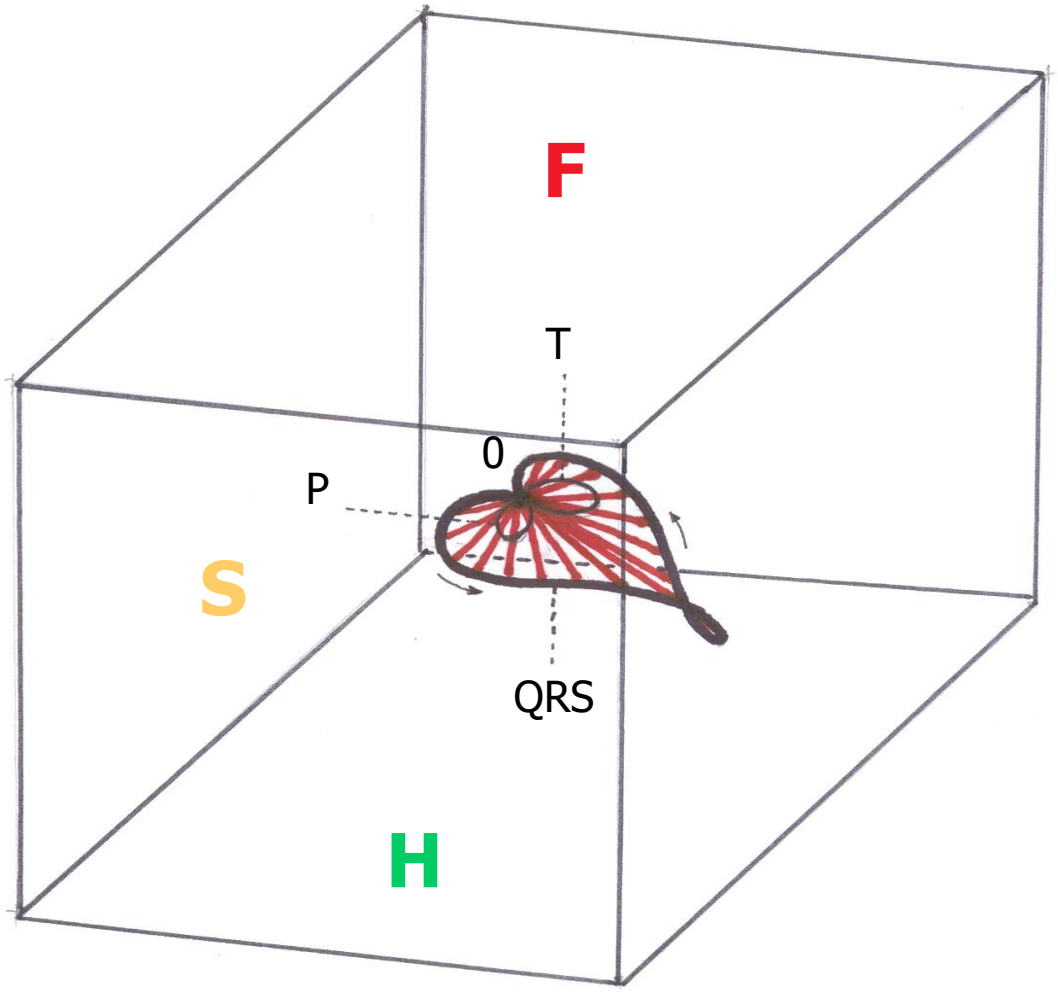
origin – metabolic, hemodynamic, anatomic, physical…

examples - ischemia, hypertrophy, dilatation, cardiomyopathy, inflammations, changes

in electrolytes, drugs…)

3D LOOPS OF ELECTRICAL AXIS

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



0 – electric center of the heart
P – atrial depolarization
QRS – ventricular depolarization
T – ventricular repolarization

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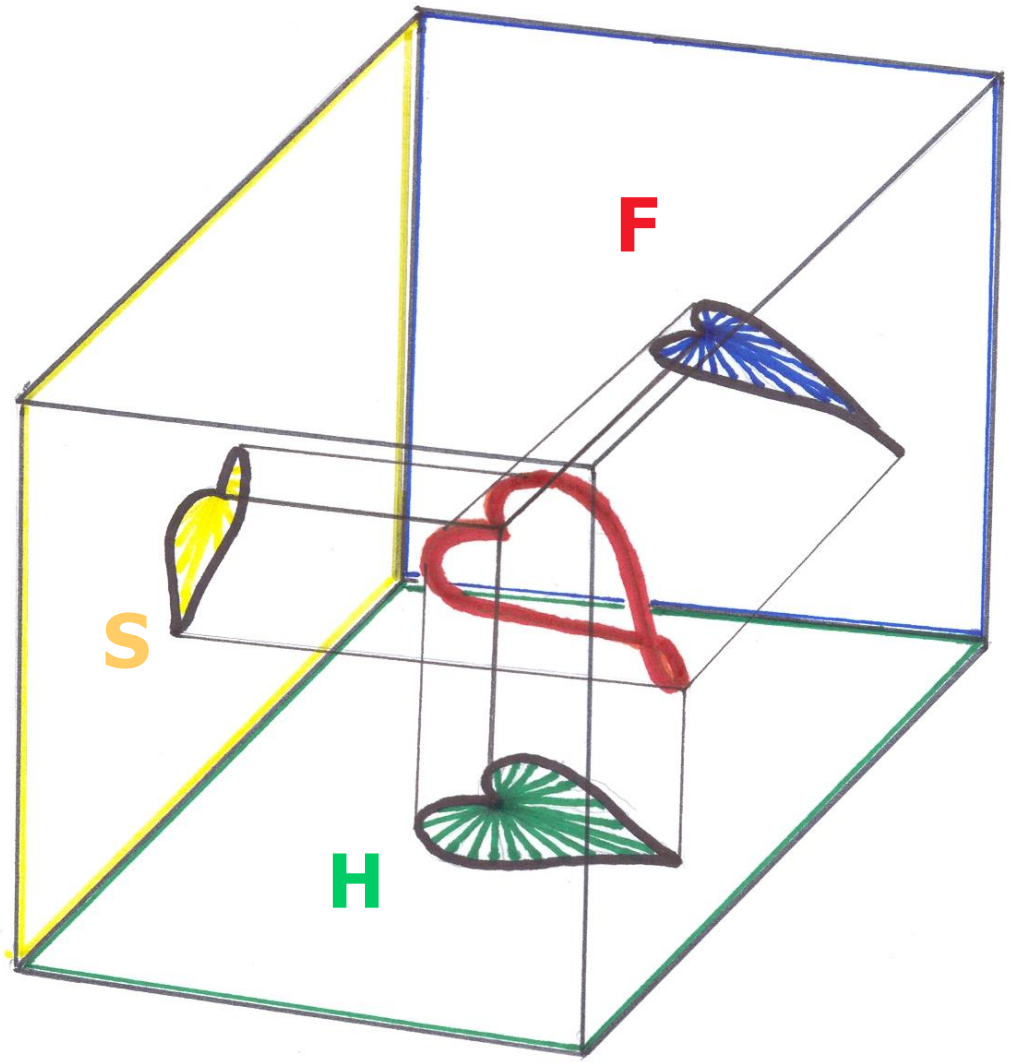
2D PROJECTION OF HEART AXIS

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



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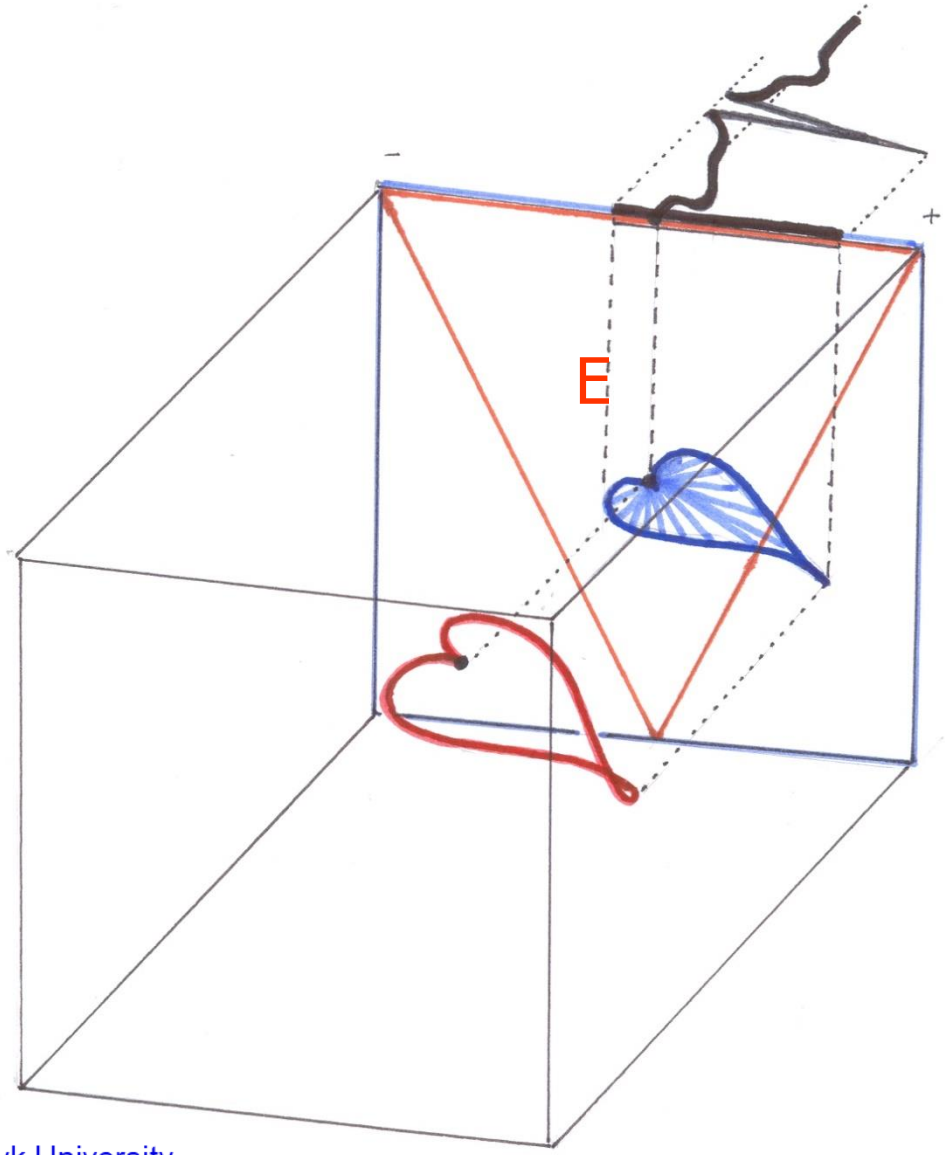
1D PROJECTION OF HEART AXIS

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



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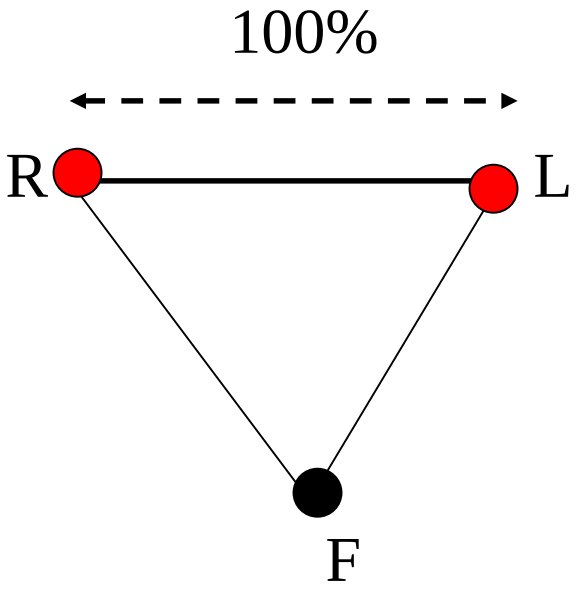
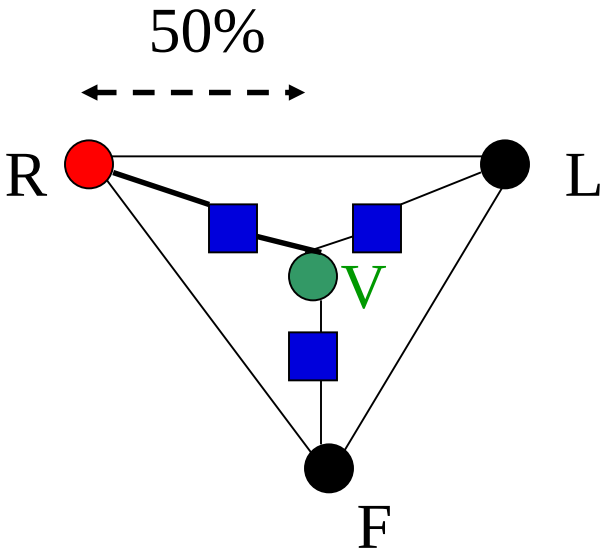
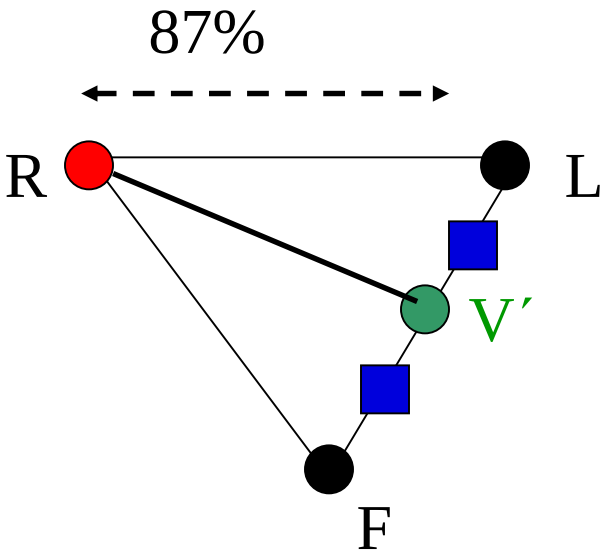


Einthoven, 1913

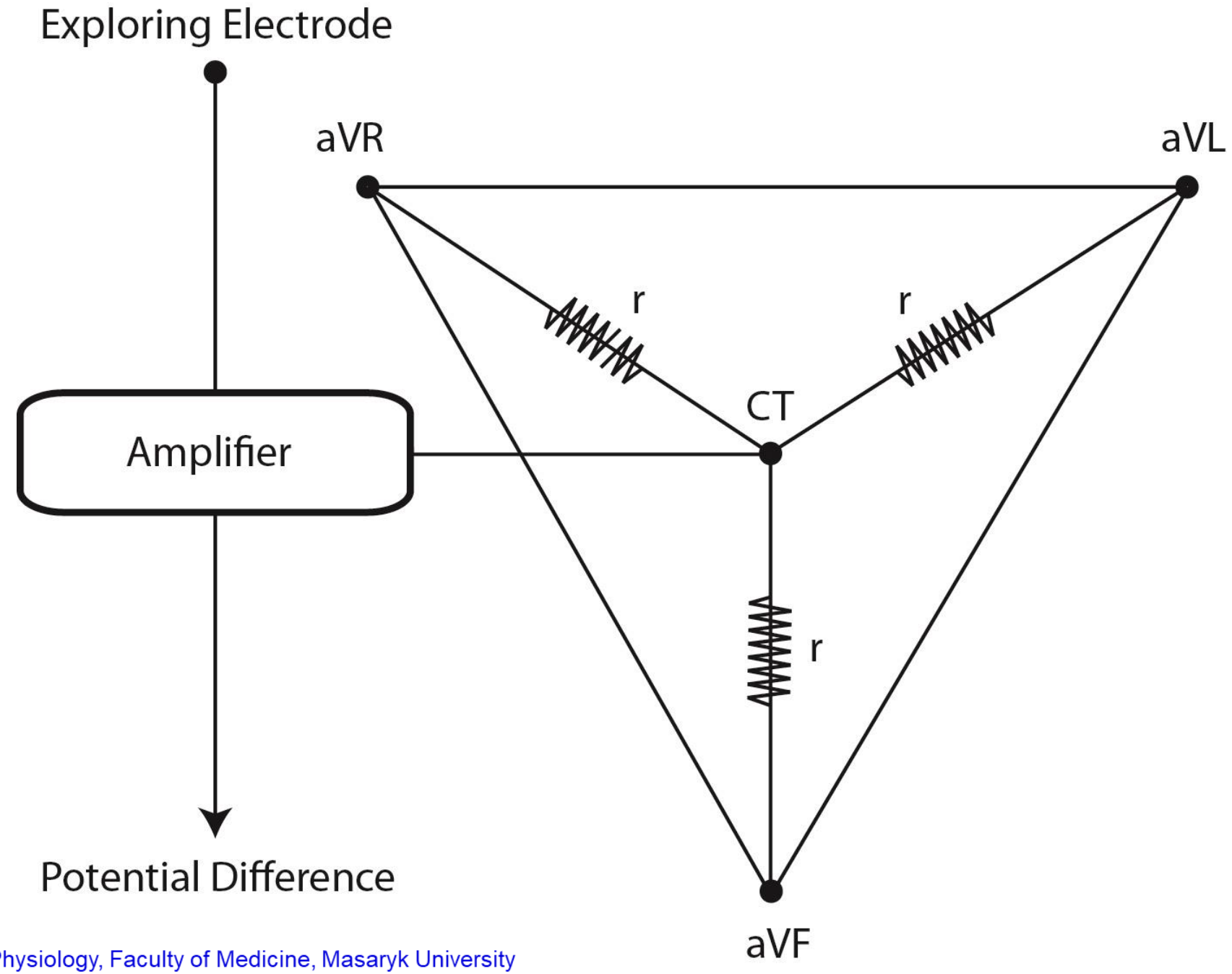
I, II, III



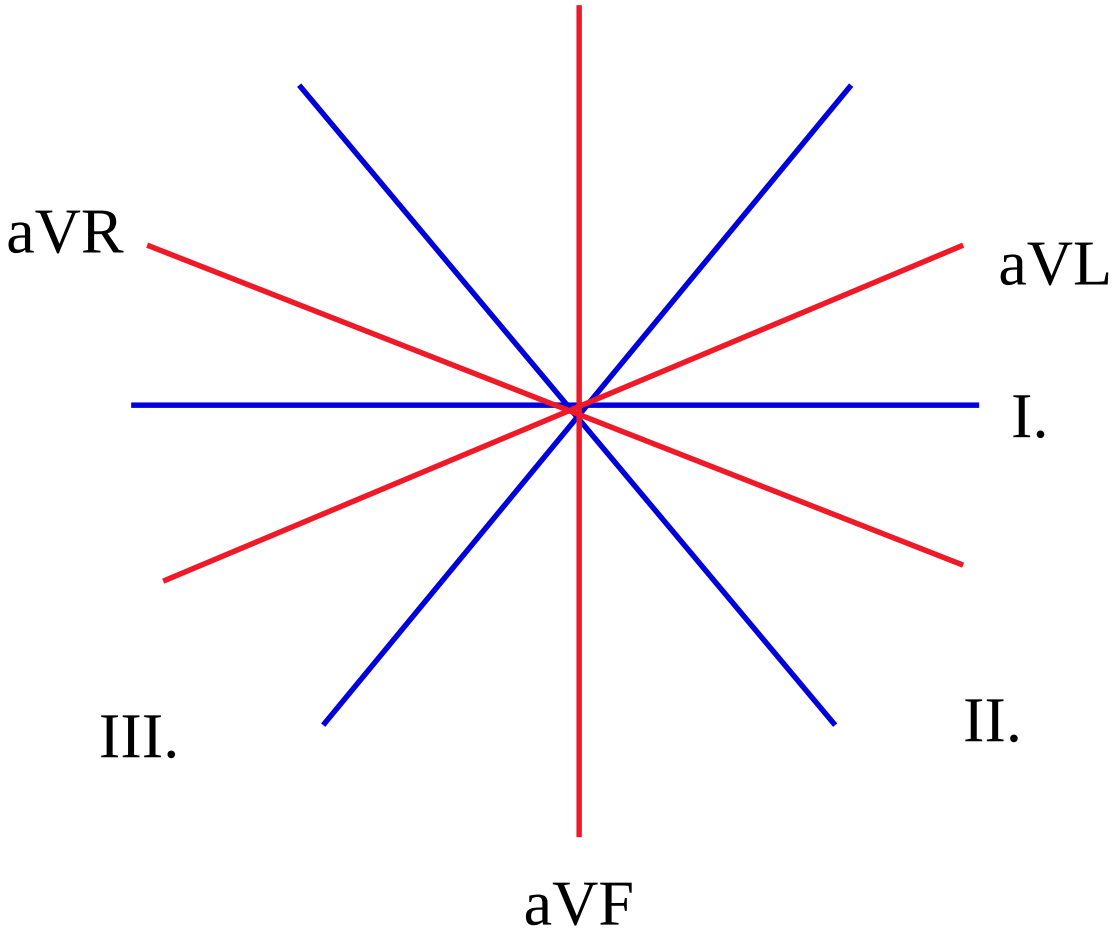
Wilson, 1934, VR, VL, VF

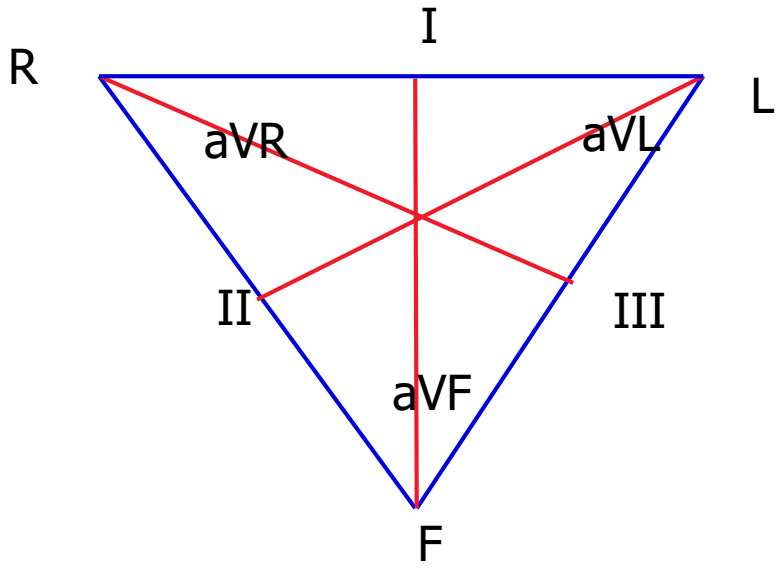


Goldberger, 1947, aVR, aVL, aVF


Exploring Electrode
aVR
aVL
r
r
CT
Amplifier
r
Potential Difference
aVF

HEXAAXIAL SYSTEM





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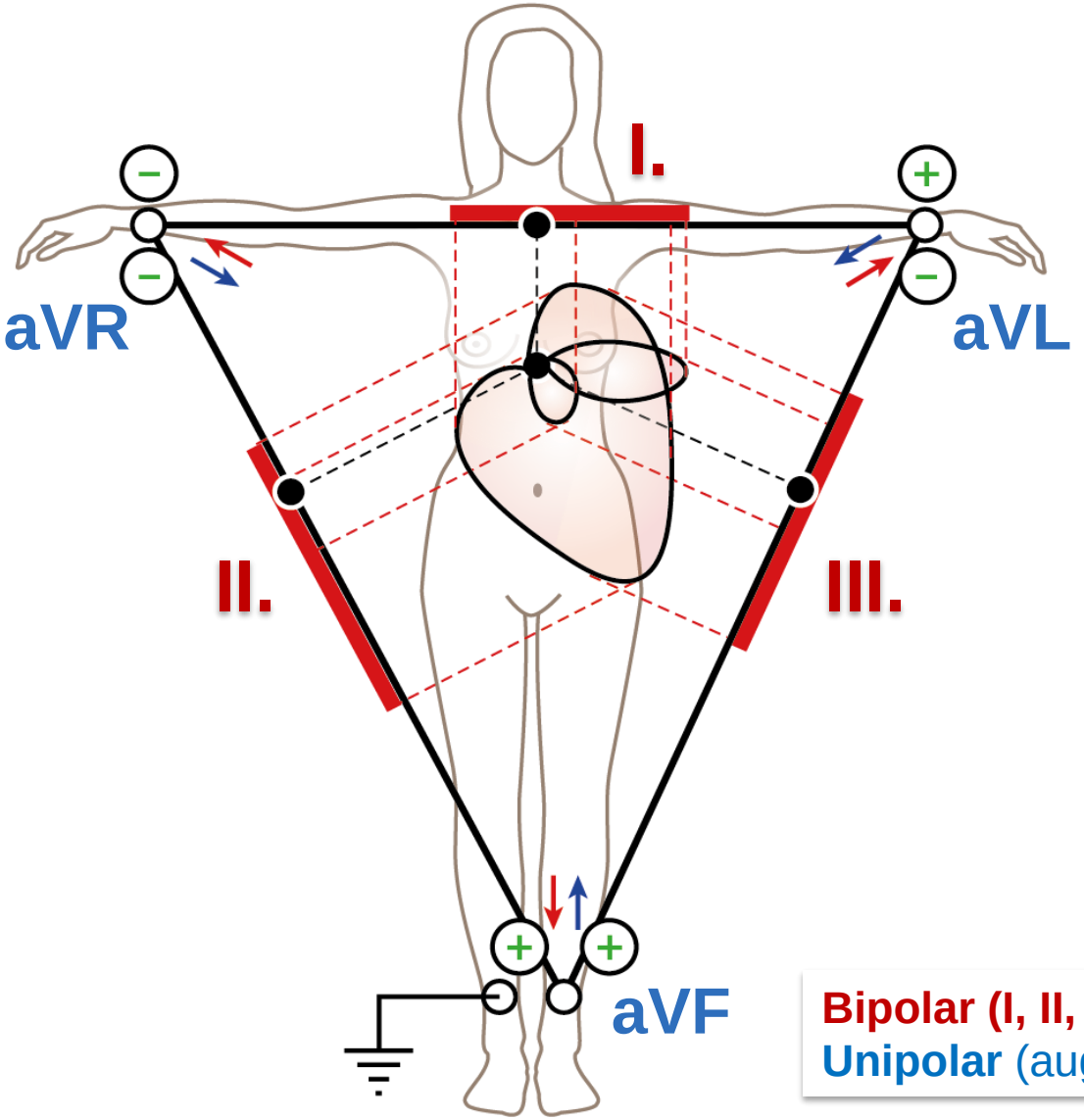
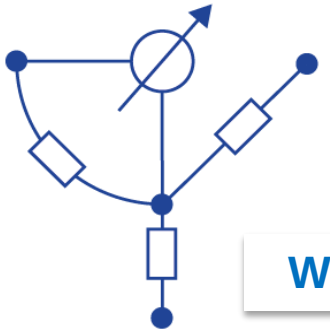
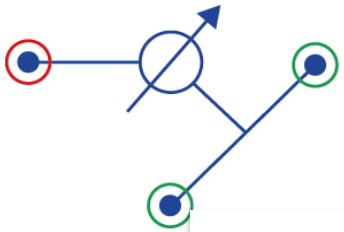
LIMB LEADS

Frontal projection of vector!





WILSON



GOLDBERGER
augmented

Bipolar (I, II, III) - standard
Unipolar (augmented) **aVR, aVL, aVF**

Marie Nováková, Department of Physiology, Faculty of Medicine, Masaryk University

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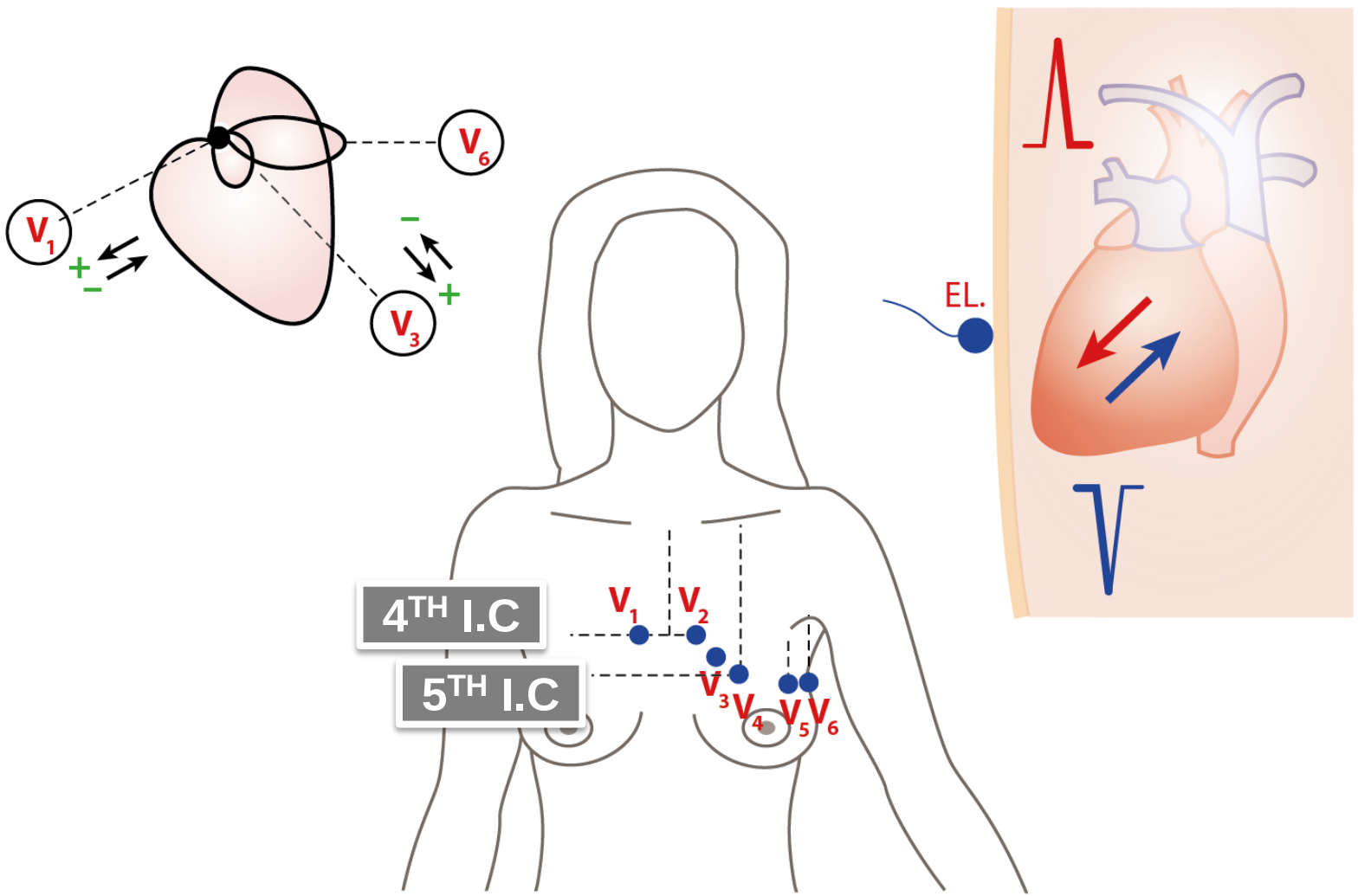
CHEST LEADS

Horizontal projection of vector!



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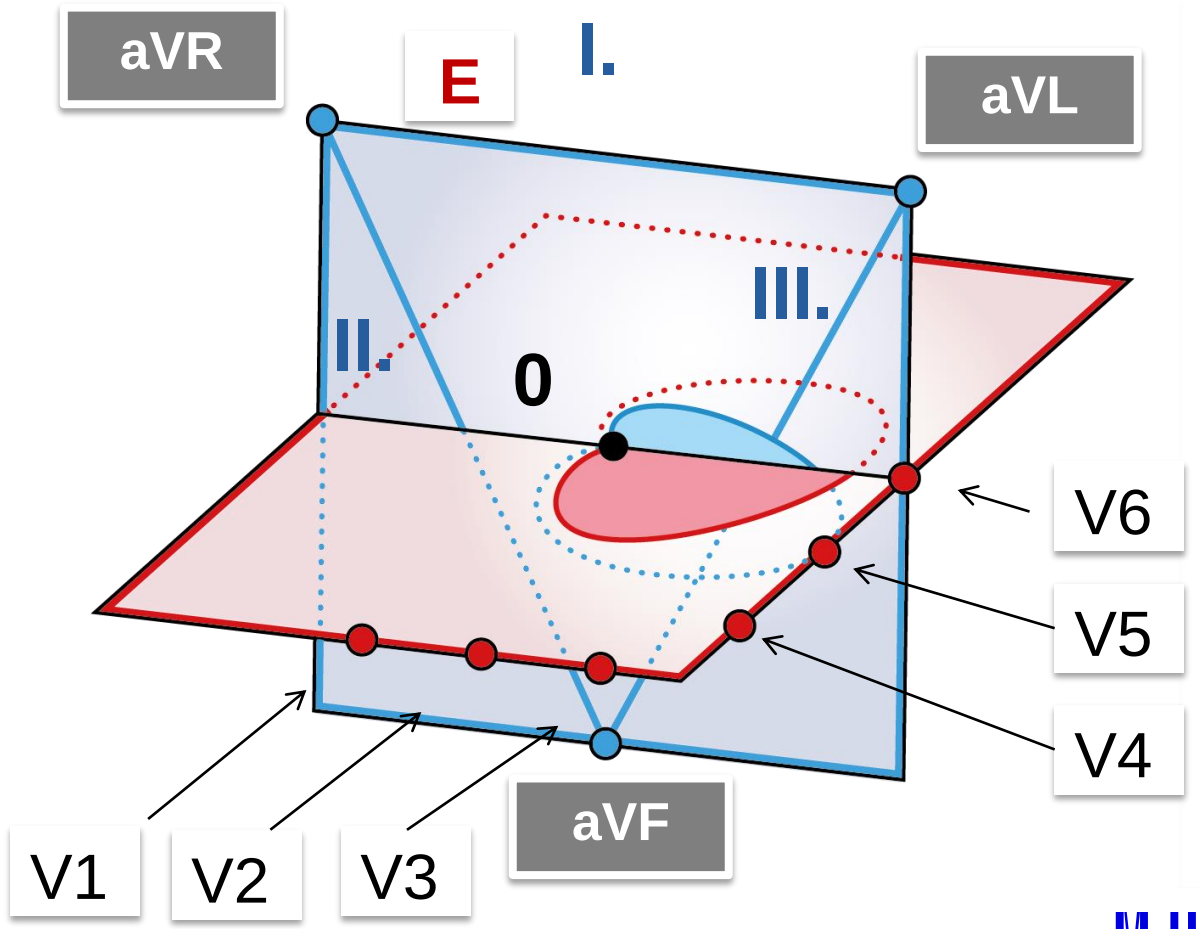
PROJECTION PLANES OF CARDIAC VECTOR
and ECG LEADS

Frontal plane
Limb leads
I., II., III., aVR, aVL, aVF

Horizontal plane
V1 – V6

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

E – Einthoven triangle



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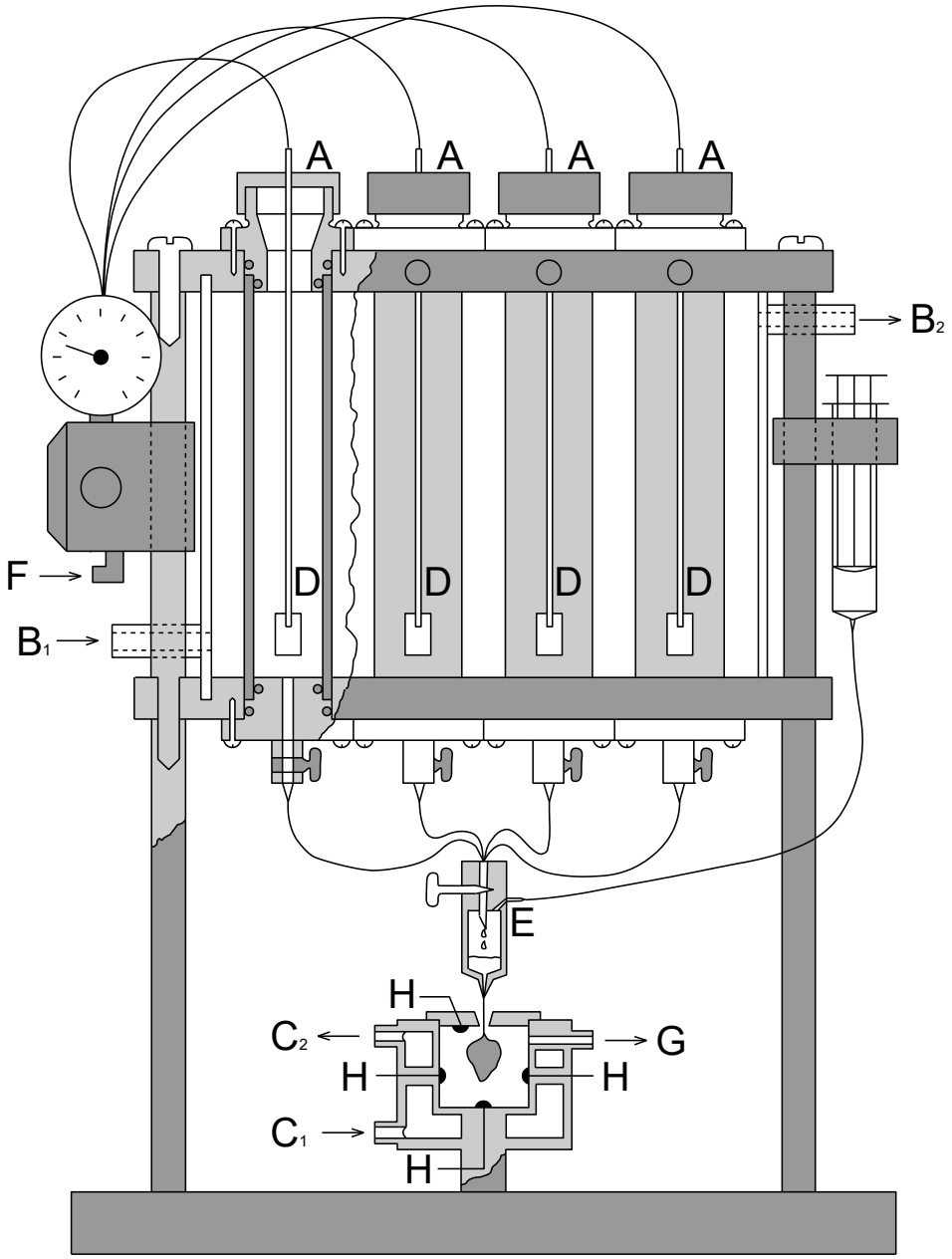
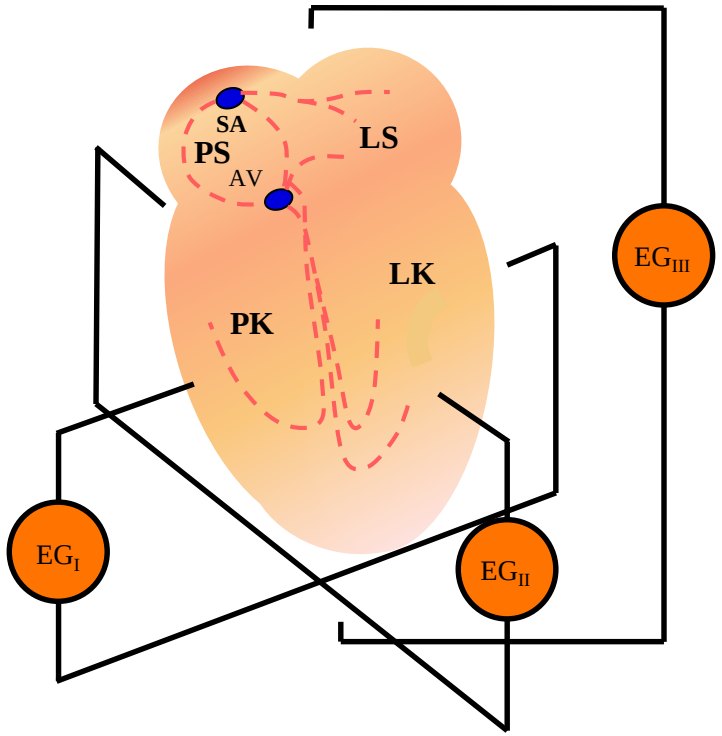
ISOLATED HEART
perfused according to Langendorff





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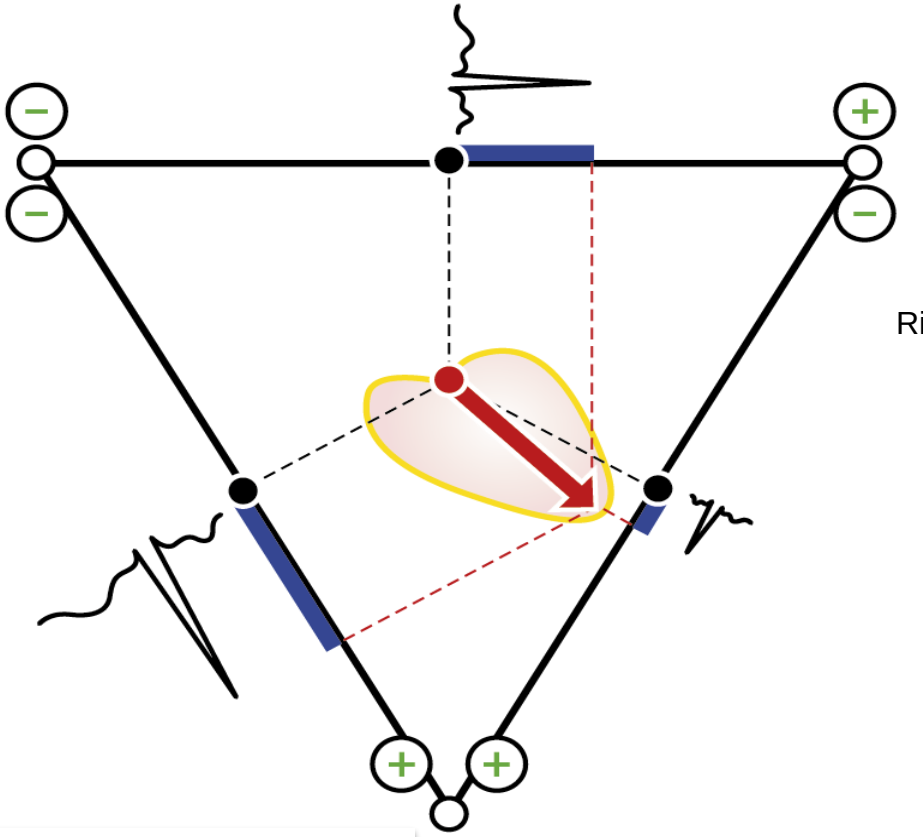
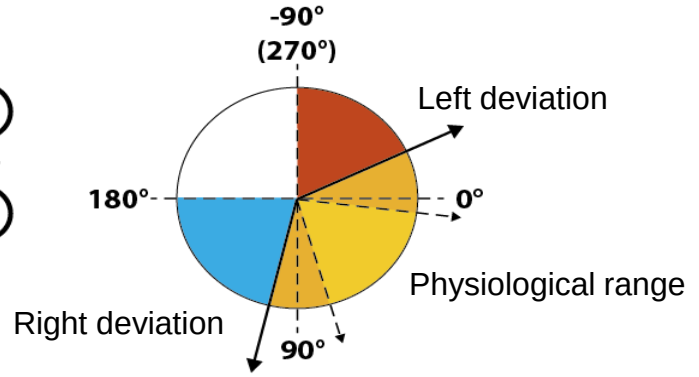
ELECTRICAL AXIS OF THE HEART

Summary of all momentary vectors, which form **ventricular depolarisation loop**. It expresses the direction of ventricular activation. It reflects the 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.

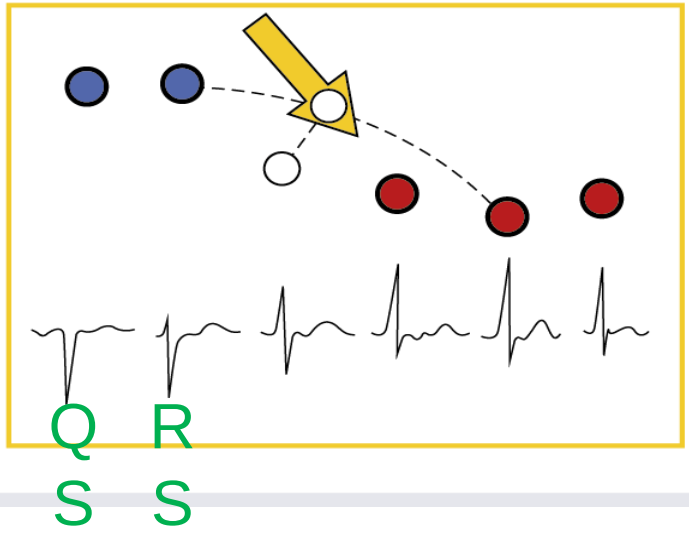




- **equilateral**

Einthoven triangle

Terminology →



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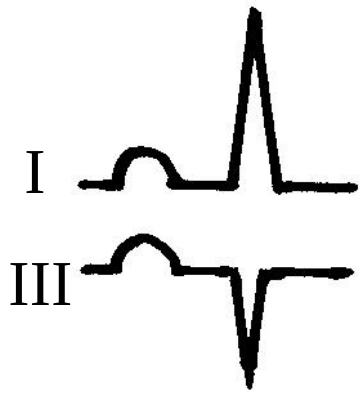
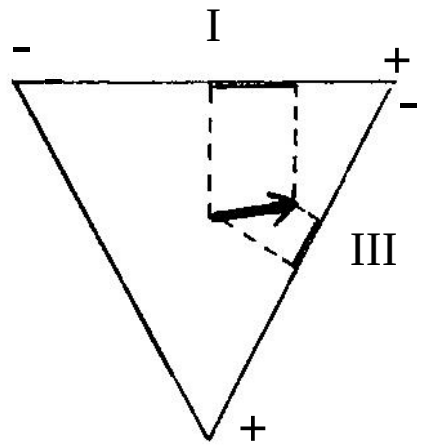
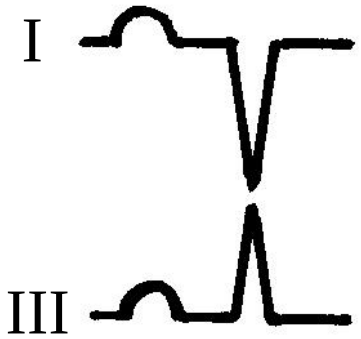
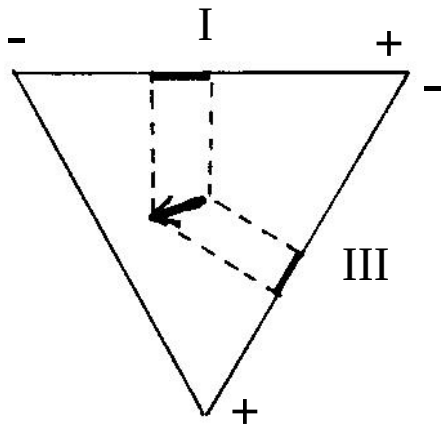
LEFT DEVIATION, RIGHT DEVIATION









Marie Nováková, Department of Physiology, Faculty of Medicine, Masaryk University

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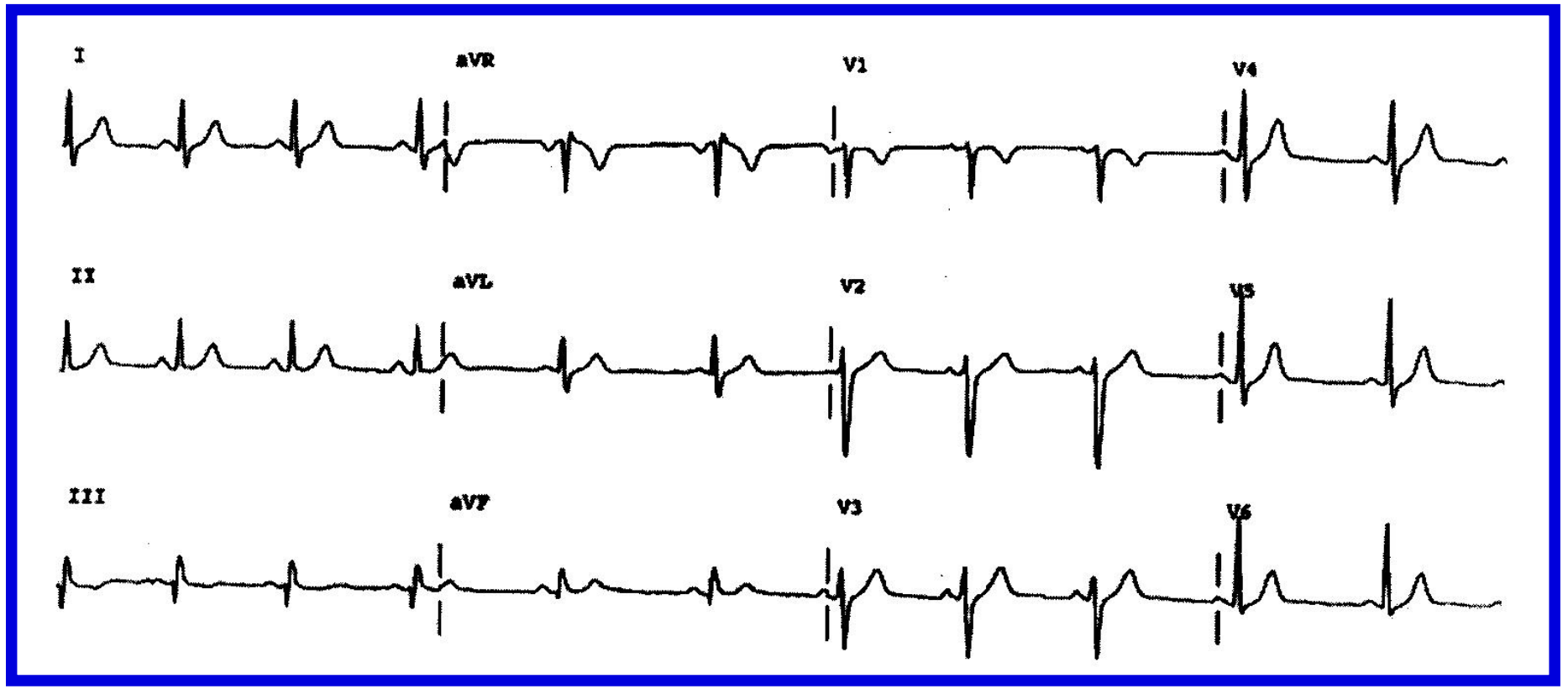
Physiological 12-lead electrocardiogram



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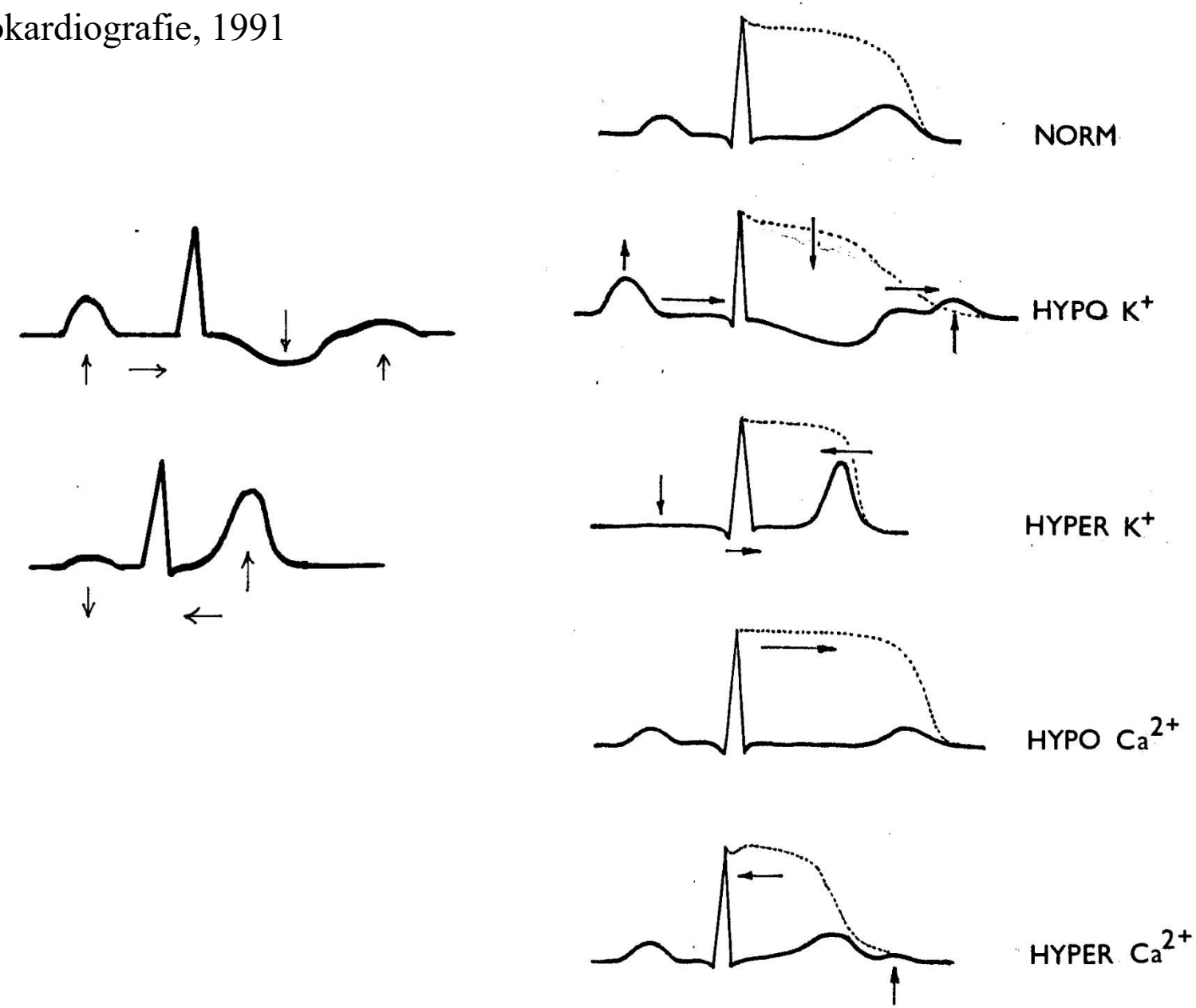
ECG – information about:

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 – repolarization (T)
5. Rhythm (P-P, R-R)
6. Action potential alterations (ST, T)
7. Effect of drugs, remedies, ion composition changes,…

Štejfa et al.: Základy elektrokardiografie, 1991



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ARRHYTHMIAS

disturbance of impulse generation

or

disturbance of impulse conduction

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

Lung reflexes – 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 (denerved) 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

Description of ECG curve: **RAFO**

RHYTHM, ACTION, FREQUENCY, „osa“ AXIS:

Rhythm – sinus or ectopic rhythms: nodal (below 40 bpm), ventricular (below 20 bpm)

Action regular *vs.* irregular :

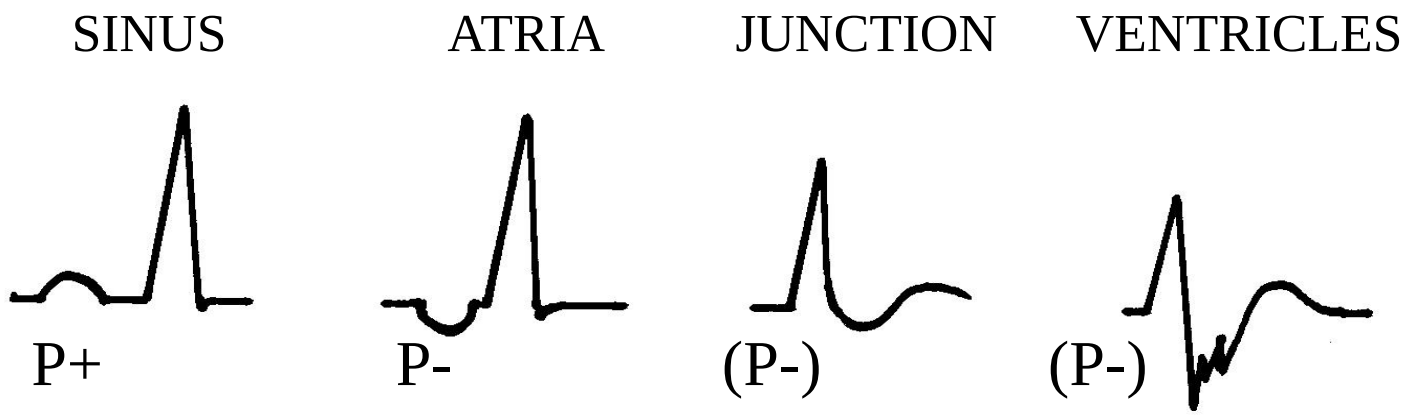
sinus respiratory arrhytmia (physiological)
sick sinus syndrom
extrasystoles (ES) single or coupled (bigeminia, trigeminia), according to site or origin - sinus, atrial, junction, ventricular

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)

RHYTHM

SITE OF ORIGIN



- P wave polarity
- PQ (QP) interval(physiological PQinterval : 0.12 – 0.2 s)

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CONDUCTION DISTURBANCES (**BLOCKS**)

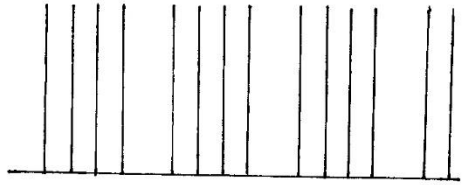
- Sick sinus syndrom
- AV blocks
- bundle branch block (BBB)
  – left, right

I°

PQ>0.2sec

LBBB

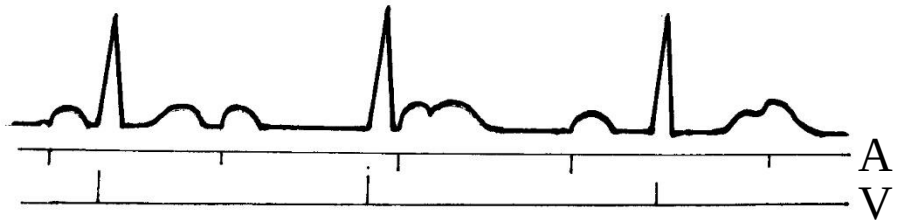
II°



RBBB

Wenckebach periodes

III°



A-V disociation

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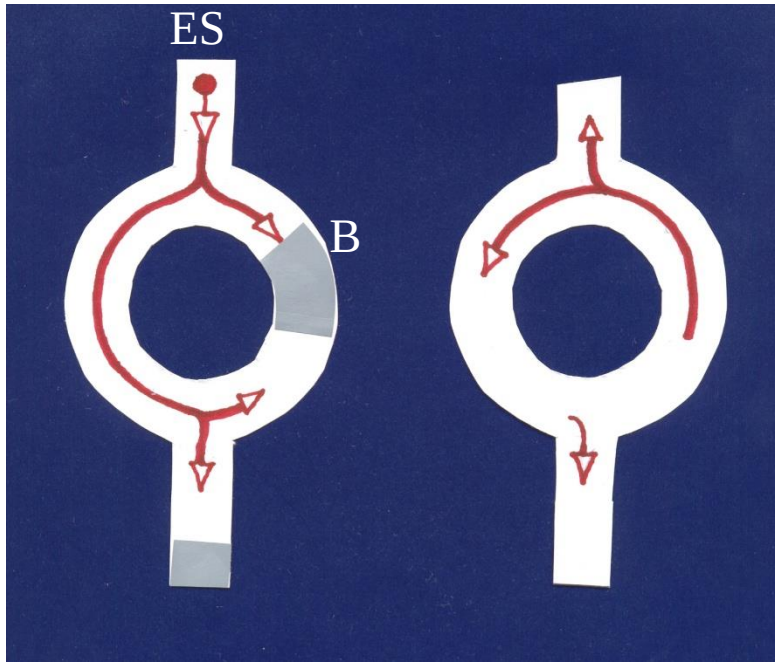
REENTRY

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

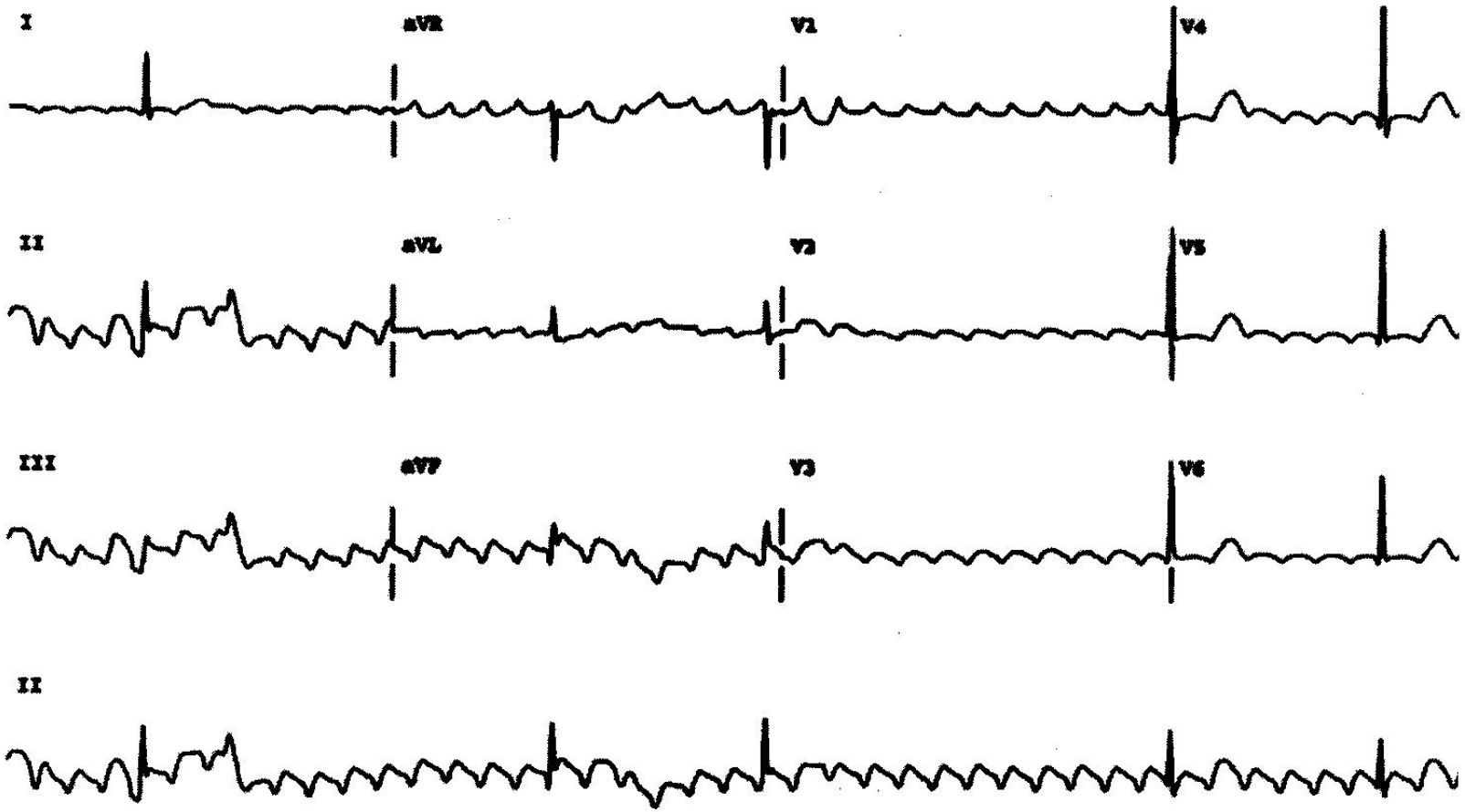
- **Loops most often at the level of AV junction**
- **Determinants of re-entry**:

1. Proper dimension of the loop
2. Proper timing of the trigger ES

TACHYARYTMIAS

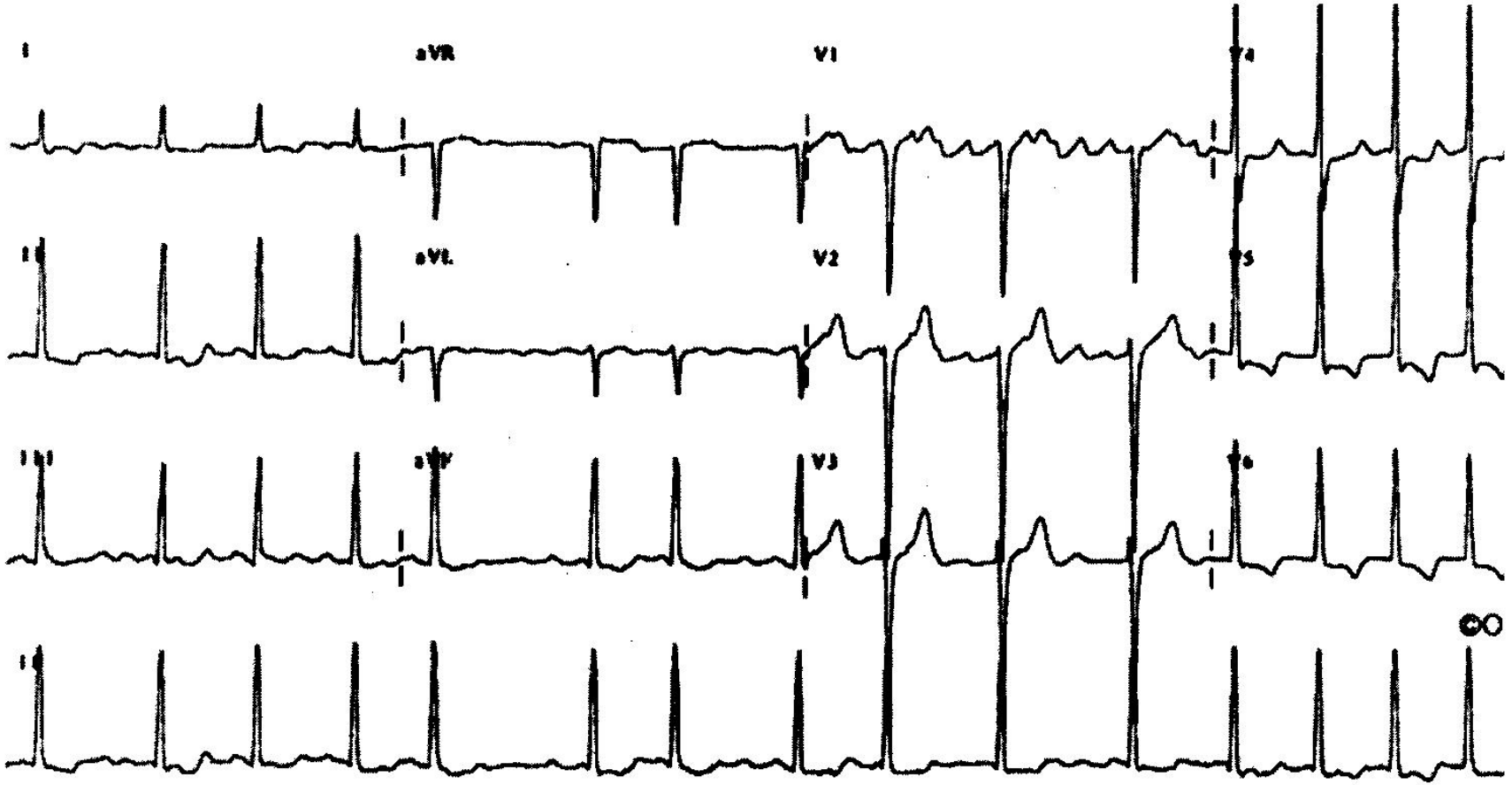
- **SINUS TACHYCARDIA**
- **PAROXYSMAL TACHYCARDIA** (supraventricular, ventricular)
- **FLUTTER** (>250/min; atrial)
- **FIBRILLATION** (>600/min; **atrial**, **ventricular**; breakdown of electrical homogeneity)

ATRIAL FLUTTER



Frequency 250 – 600/min
Atrioventricular block n:1

ATRIAL FIBRILLATION



Irregular ventricular rhythm

+ f-waves

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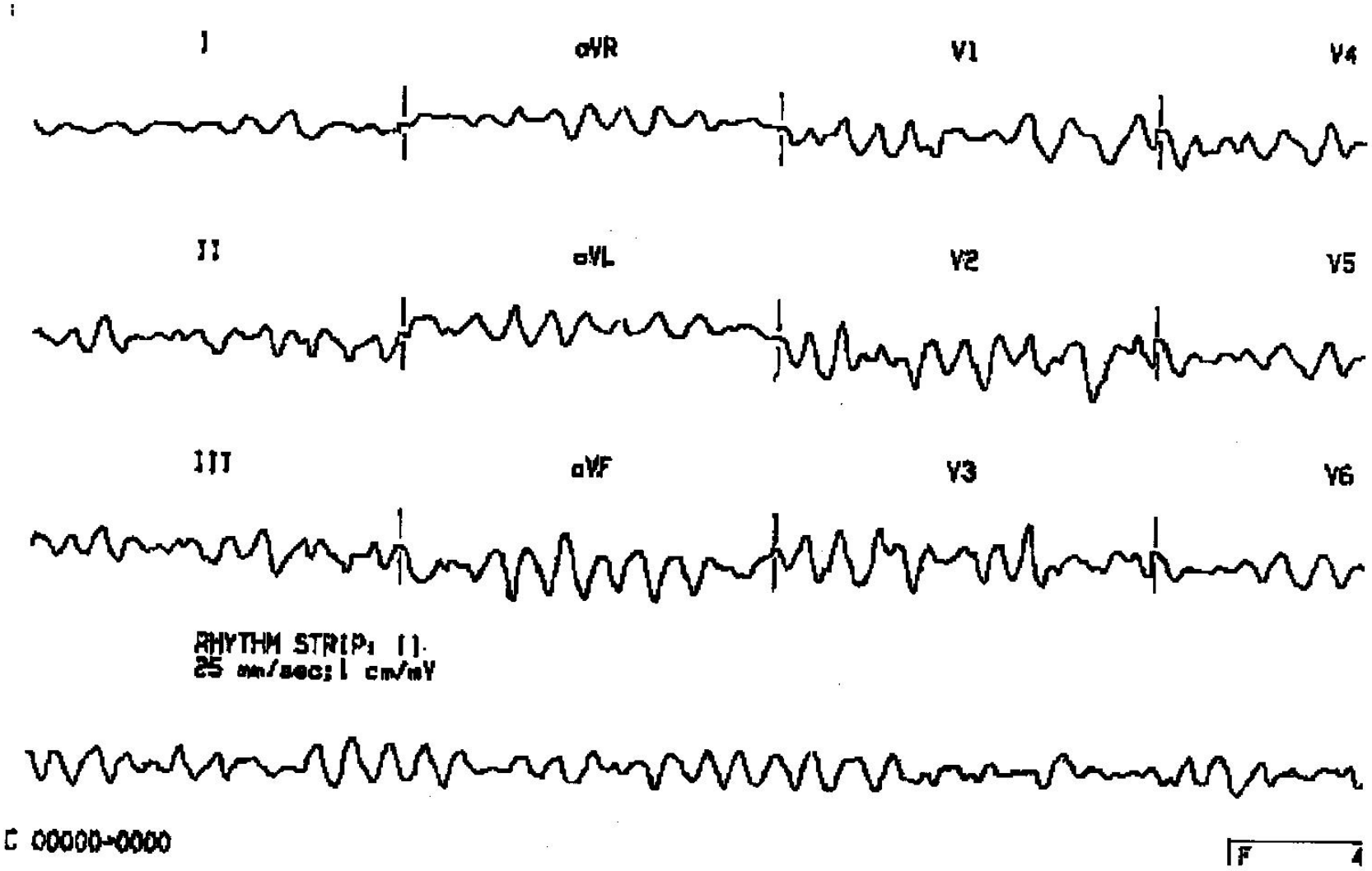
VENTRICULAR FIBRILLATION

Frequency above 600/min, **LETHAL**



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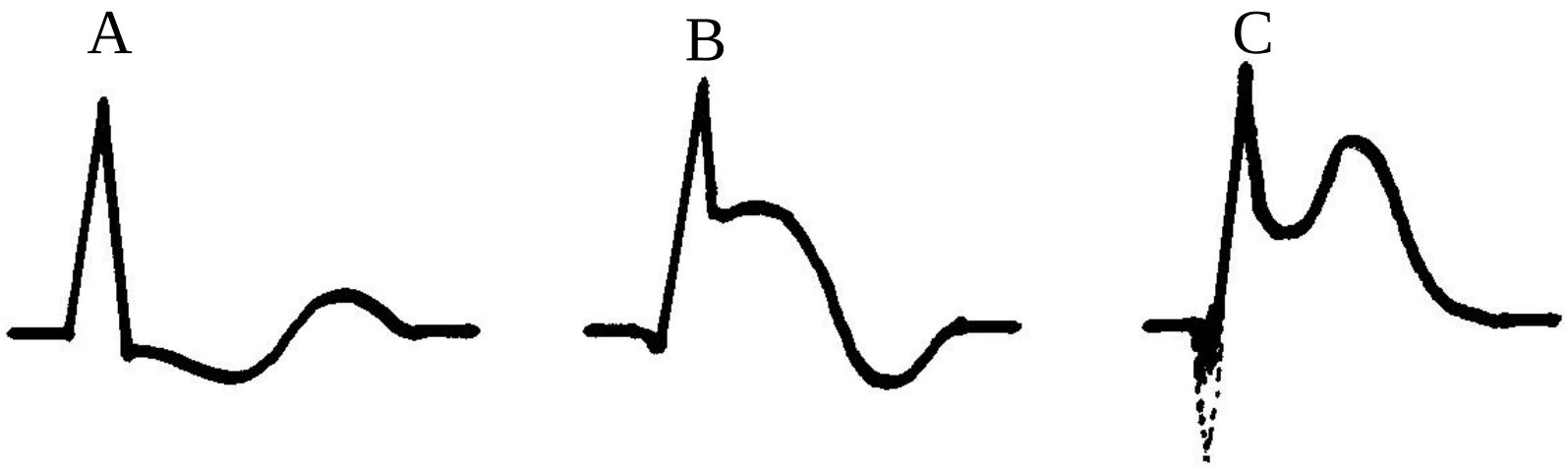
HEART ISCHEMIA



A: exercise angina pectoris
B: acute non-Q myocardial infarction
C: acute Q myocardial infarction

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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
- β-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

esf evropský sociální fond v ČR
EVROPSKÁ UNIE
MINISTERSTVO ŠKOLSTVÍ, MLÁDEŽE A TĚLOVÝCHOVY
OP Vzdělávání pro konkurenceschopnost
UNIVERSITAS MASARYKIANA BRUNENSIS

INVESTICE DO ROZVOJE VZDĚLÁVÁNÍ

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