Pázmány Péter Catholic University
Faculty of Information Technology and Bionics

Biomedical Signal Processing
2018-2019 Autumn

Signal Genesis - Physiology

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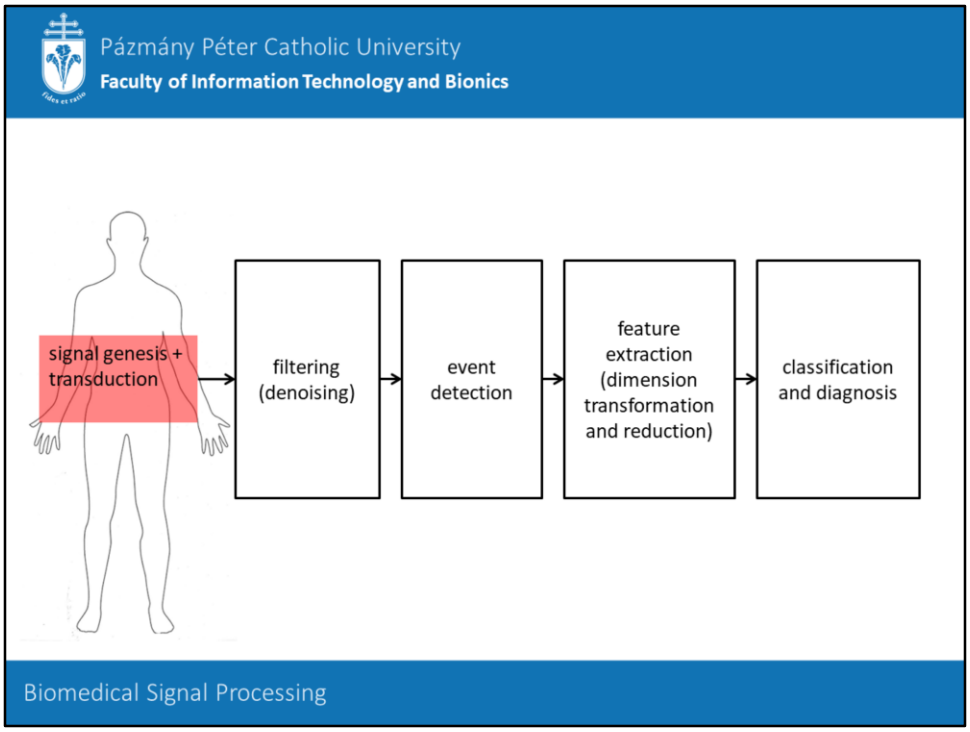
Responsible lecturer: dr. Miklós Gyöngy

Today's goal

Understanding the physical background of the following signals:

1. **Cardiovascular mechanical and optical signals**
   - Phonocardiography (PCG)
   - Blood pressure (BP)
   - Pulse oximetry (PO)
   - Photoplethysmography (PPG)
2. **Bioelectric signals**
   - ECG
   - EMG
   - EEG



Begin by discussing the origin of biomedical signals.
In this first lecture, we will look at some of the physiological processes that give rise to biomedical signals.
The next lecture will consider how we can model these processes using mathematical equations.

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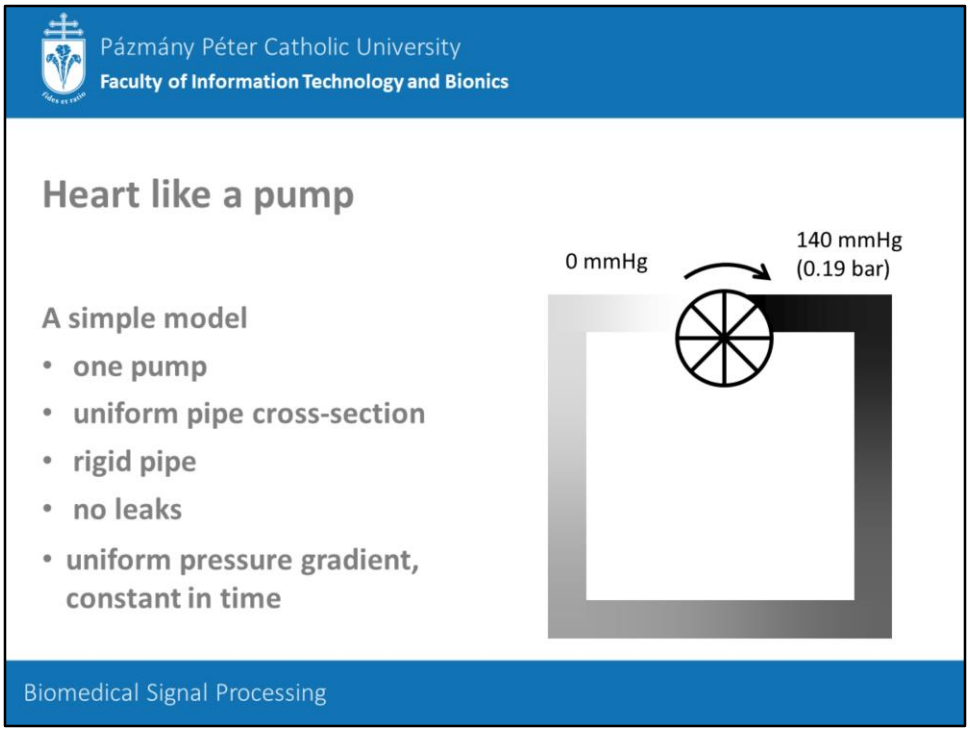
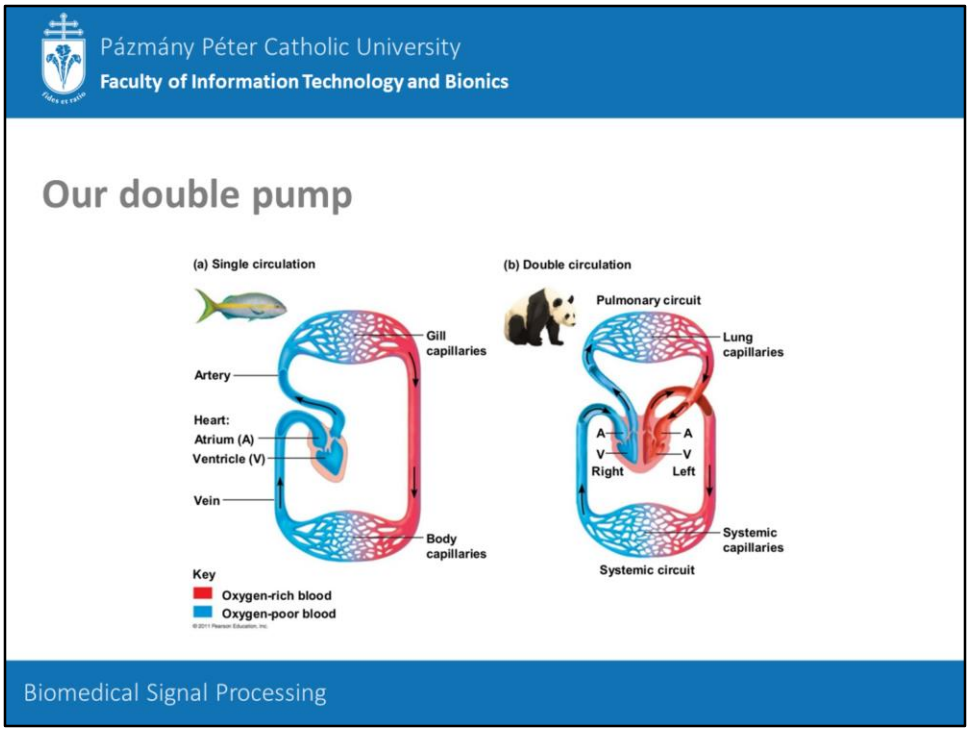
Part I – Mechanical and Optical Signals from the Cardiovascular System



In contrast:
The heart

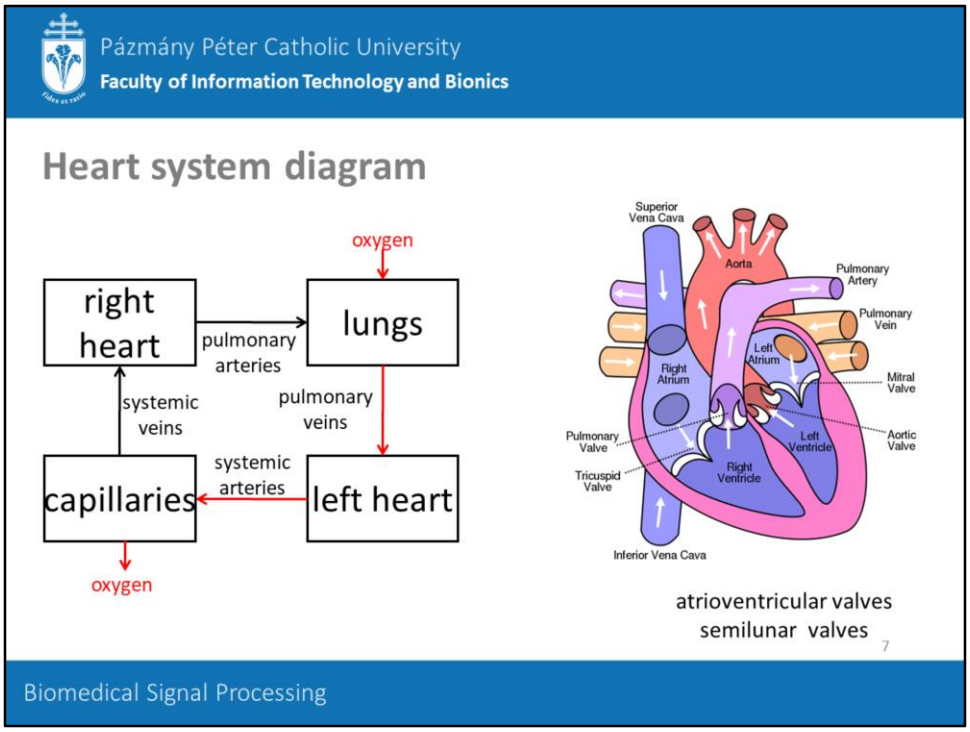
- two pumps
- pulsating pressure, non-uniform pressure gradient
- non-uniform pipe cross-section
- compliant pipe
- „leaky" vessels

For the simple model the Hagen–Poiseuille equation would ive the pressure (wikipedia), where $\Delta P = ZQ, Z = \frac{8\mu}{\pi R^4} L$ (in order or appearance, pressure difference between two ends, impedance, volumetric flow rate, dynamic viscosity, pipe radius, length between two ends). Dynamic viscosity is shear stress / shear rate). Dynamic viscosity of blood is 2.78 mPa.s according to http://www.viscopedia.com/viscosity-tables/substances/whole-blood/)



The heart is actually a **double pump**, (but not in all animals ☺). One is responsible for transporting oxygenised blood to the body, the other pumps the deoxygenised blood to the lung.

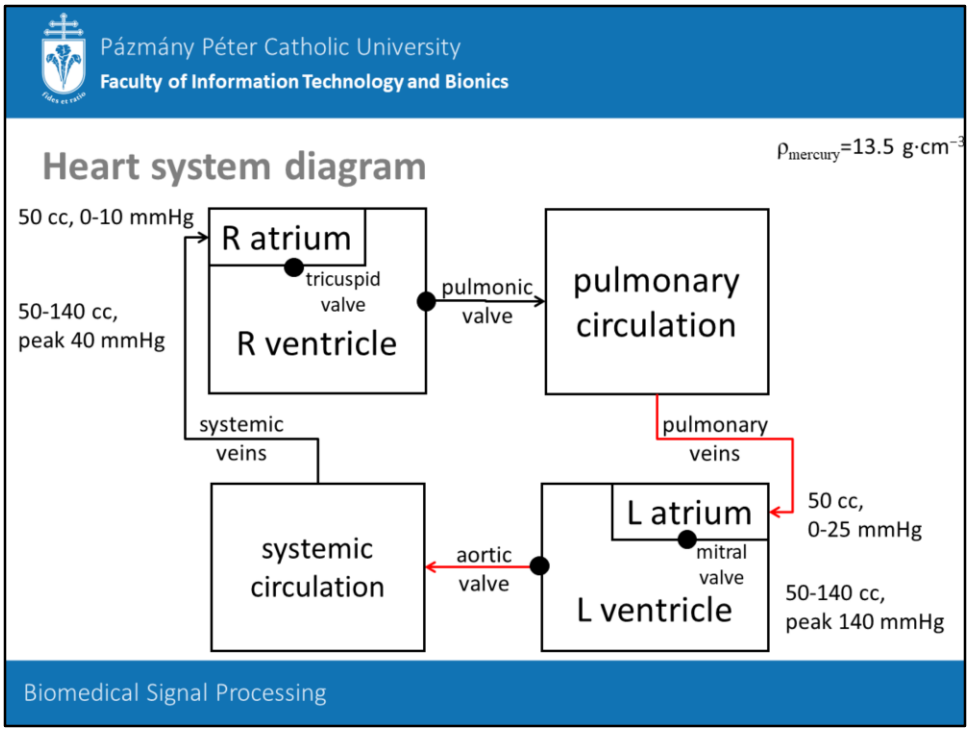
https://www.slideshare.net/Noornoorsd/biology-b-ch42



More like it! Two compartments of the heart, left and right.

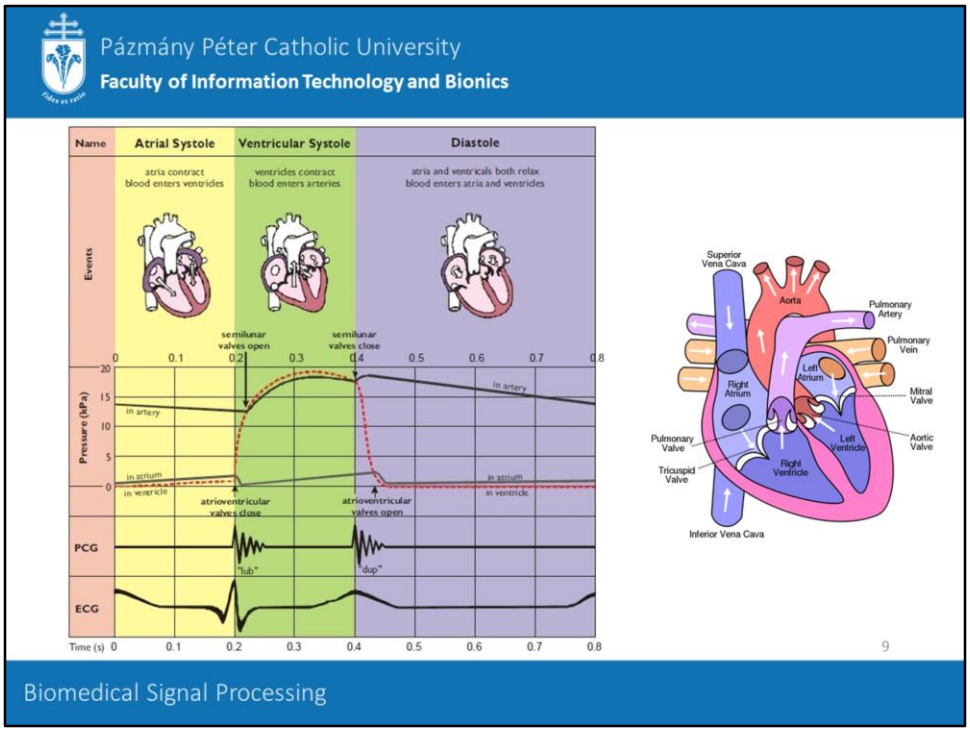
From the lung's pulmonary veins the oxygenised blood arrives to the **left atrium**, where through the open **atriovetricular bicupsid valve** (mitral valve as it looks like the cap, mitre of a bishop) it is pumped to the **left ventricle**. The left ventricle pumps the blood through **semilunar valves** to the **systemic arteries**. The arteries branch into smaller and smaller **capillaries**, until they are able to give the oxygenised blood to the tissue. From the tissue the deoxygenised blood returns to small capillaries and they are transporting it back towards the heart in **veins**. The deoxygenised blood enters the heart's **right atrium** through the systemic vena cavas. From the atrium the blood is pumped to the **right ventricle** through **the atrioventricular tricupsid valve**, where the contraction of the muscle pushes the blood through the **semilunar valves** to the **pulmonary arteries**, branching into the small capilleries of the lung.

http://commons.wikimedia.org/wiki/File:Diagram_of_the_human_heart_%28cropped%29.svg



credit question:
If a motorbiker suffers an accident punching his systemic artery close to the heart, how high can the blood flow?
(pressure * (rho_Hg / rho_blood))

From the diagram it can be seen, that the bloodpressure changes constantly through the cardiac system. While the blood exiting the left vetricle will have a pressure of 140 mmHg, when it arrives back to the right atrium, this pressure will be only 10 mmHg. It is good to remember, as we can see, that a blood measurement on the wrist will have different value than a catheter in the heart.

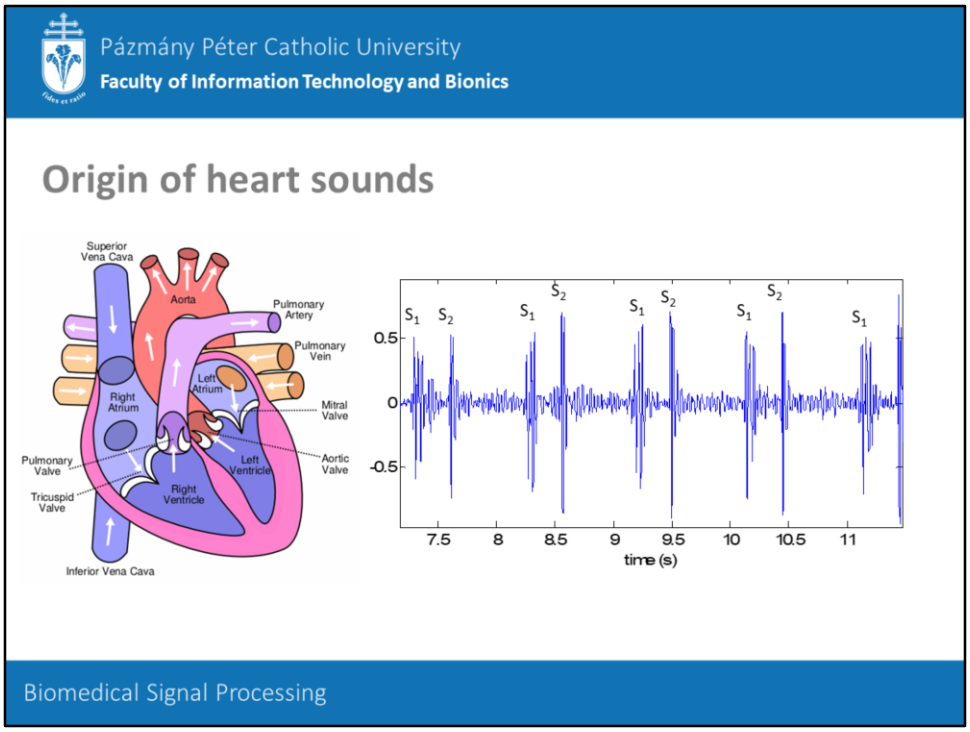


Remember simplest pump model? We had constant pressures at input and output, the pump's wheel was working smoothly.

Here let's look at arterial (output) and atrial pressures (returning low pressure), ‚sort of constants', no big changes.
Look at **ventricular pressure** in between.
0.2 s: ventricle compresses, causing a sudden increase. As the pressure reaches higher values the arterial (semilunar) valves open allowing outflow, making the BP increase to slow down, even fall back after 0.3s
0.4 s: As the ventricle empties the semilunar valves close, the muscles relax, causing a sudden decrease of BP. As AV valves open, blood will start to flow to the atrium and ventricle.

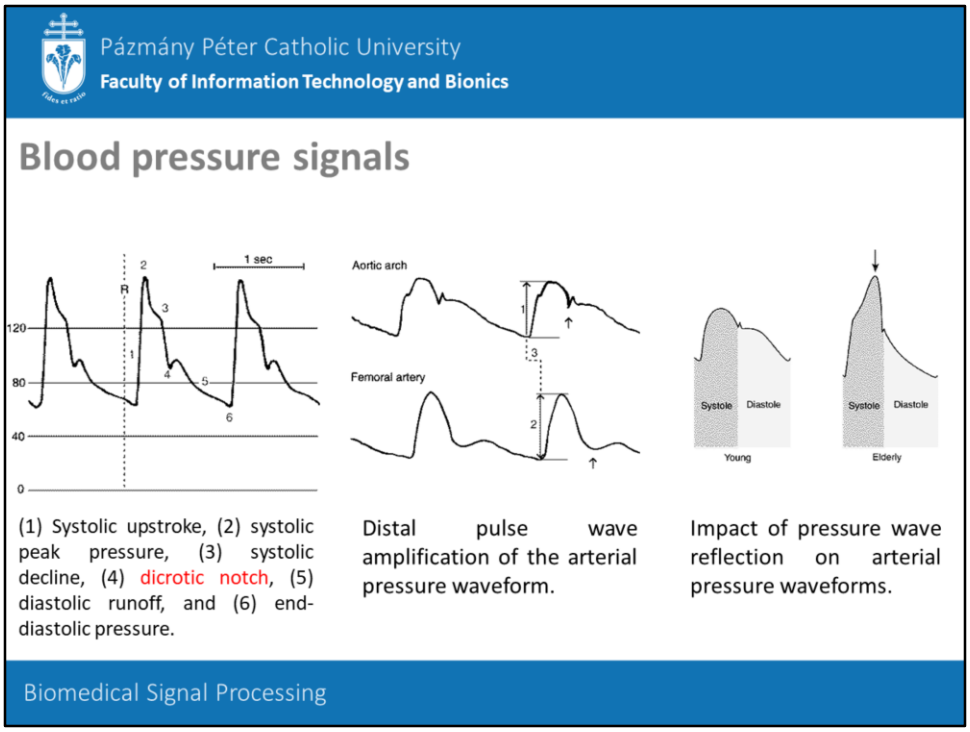
Closure of AV valves and then semilunar valves cause **S1, S2 sounds** respectively
ECG **QRS complex** sign of **ventricular contraction** signal

https://sites.google.com/a/ncea.org.uk/biology/the-cardiac-cycle
http://en.wikipedia.org/wiki/File:Cardiac_Cycle_Left_Ventricle.PNG



S_1: bicuspid + tricuspid AV (normally $t_b<t_t$ very slightly, large $|t_b-t_t|$→left/right bundle branch block; *splitting*)
S_2: aortic + pulmonary valves (normally $t_a<t_p$, split increasing up to 80 ms during inspiration; *physiological split*)
S_3,S_4 rarer (latter definitely pathologic)

http://commons.wikimedia.org/wiki/File:Heart_labelled_large.png,
http://en.wikipedia.org/wiki/File:Reizleitungssystem_RSB.png
Ádám Balogh: Analysis of the Heart Sounds and Murmur of Fetuses and Preterm Infants, p. 14

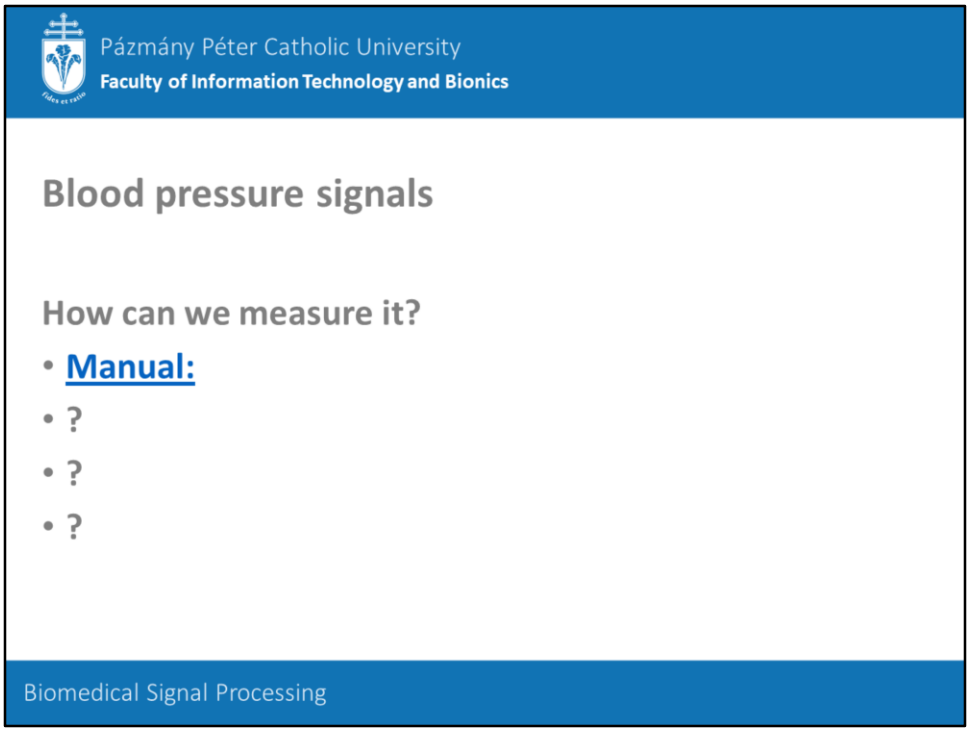


Left: **normal BP** signal from the artery. The **dicrotic notch** arises as the semilunar valves close, but can not necessarily be explained by the closure itself. Pressure waves in the circulation will be reflected back towards the heart from bifurcations, and arterioles where the radius is decreased, hence has a higher resistance against the blood flow.

Centre: Compared with pressure in the **aortic arch**, the more peripherally recorded **femoral artery** pressure waveform demonstrates a higher pulse pressure (compare 1 and 2), a delayed upstroke (3), a delayed, slurred dicrotic notch (compare arrows), and a more prominent diastolic wave.

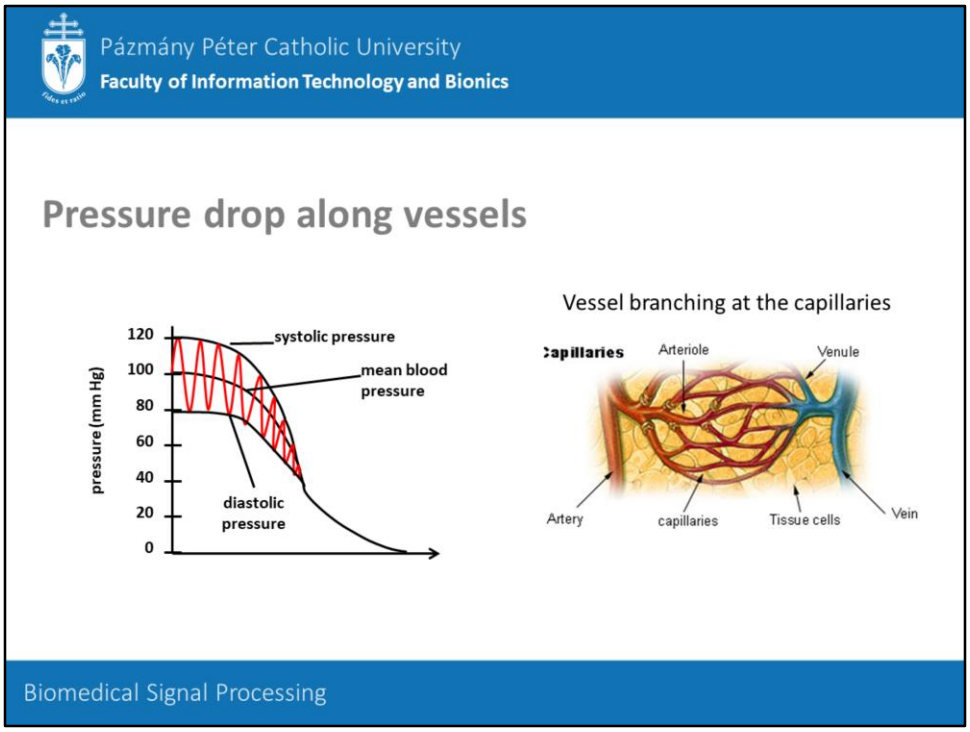
Right: In **elderly** individuals with reduced arterial elasticity, early return of reflected waves increases pulse pressure, produces a late systolic pressure peak (arrow), and attenuates the diastolic pressure wave.

http://web.squ.edu.om/med-Lib/MED_CD/E_CDs/anesthesia/site/content/v03/030267r00.HTM



http://www.knowyourph.org/diagnosis/index.php?q=19
You can see this page for ultrasonic measurement and to try how catheterization works

Ultrasound, invasive, wrist cuff, PPG, … see on lab ☺



vascular blood perfusion

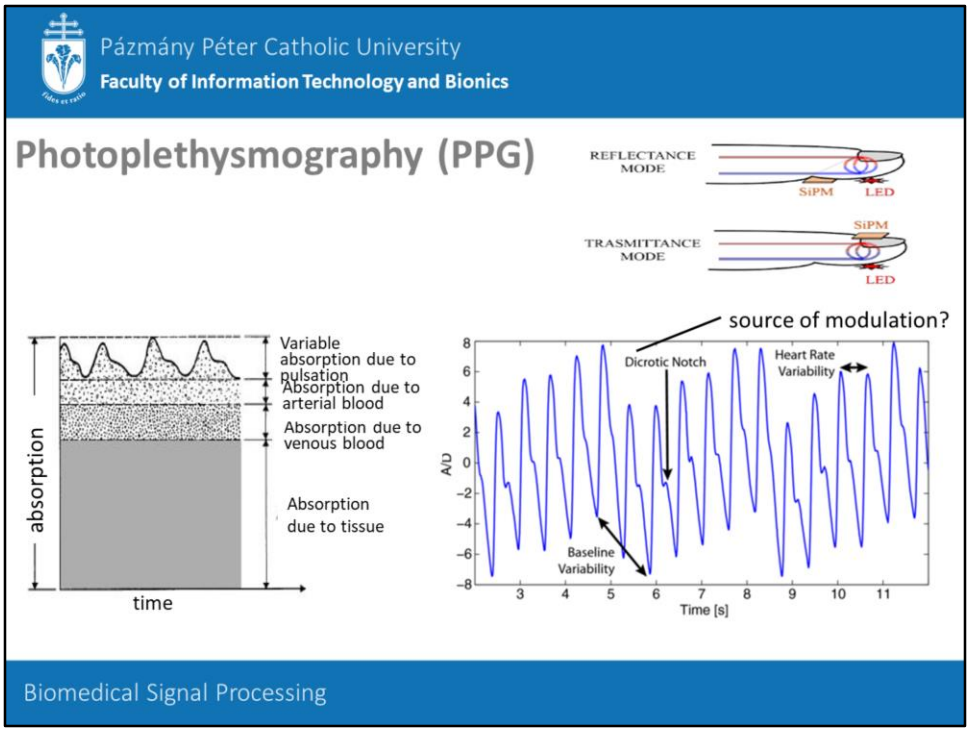
- flow due to pressure gradient
- mass conservation
  - -total flow rate constant
  - -90 % of blood returns via veins
  - -surface area ↑ velocity ↓

-surface area greatest at capillaries

- -40-50 cm/s at arteries
- -0.03 cm/s at capillaries
- -speed again rises towards veins but does not reach arterial blood velociy due to blood loss at capillary bed (collected by lymphatic vessels)

http://commons.wikimedia.org/wiki/File:Illu_capillary.jpg

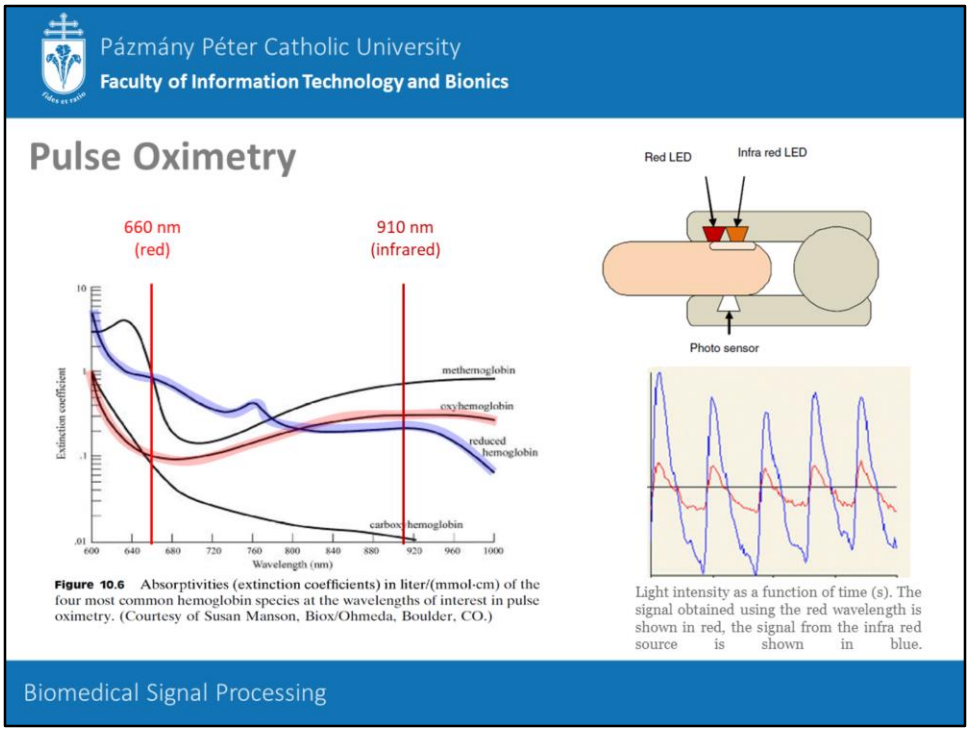
adapted from [Uzwiak 2010]



Photoplethysmography (volume measurement)

The main idea is that because of pulsation the **volume of blood** changes through a cardiac cycle in the veins. The more blood is in the tissue, the more light will be absorbed, making possible the monitoring of the cycle.
The device measures either the reflected or transmitted amount of light.
On the left you can see, what parts of your finger how can influence the absorption.
The only varying component is the volume of the blood, the pulsation can be followed.

http://iopscience.iop.org/article/10.1088/0967-3334/33/10/1617;jsessionid=1092E0C890BDBF0ABE5951111E460C5D.c1
http://en.wikipedia.org/wiki/Photoplethysmogram
Allen (2007): Photoplethysmography and its application in clincal physiological measurement

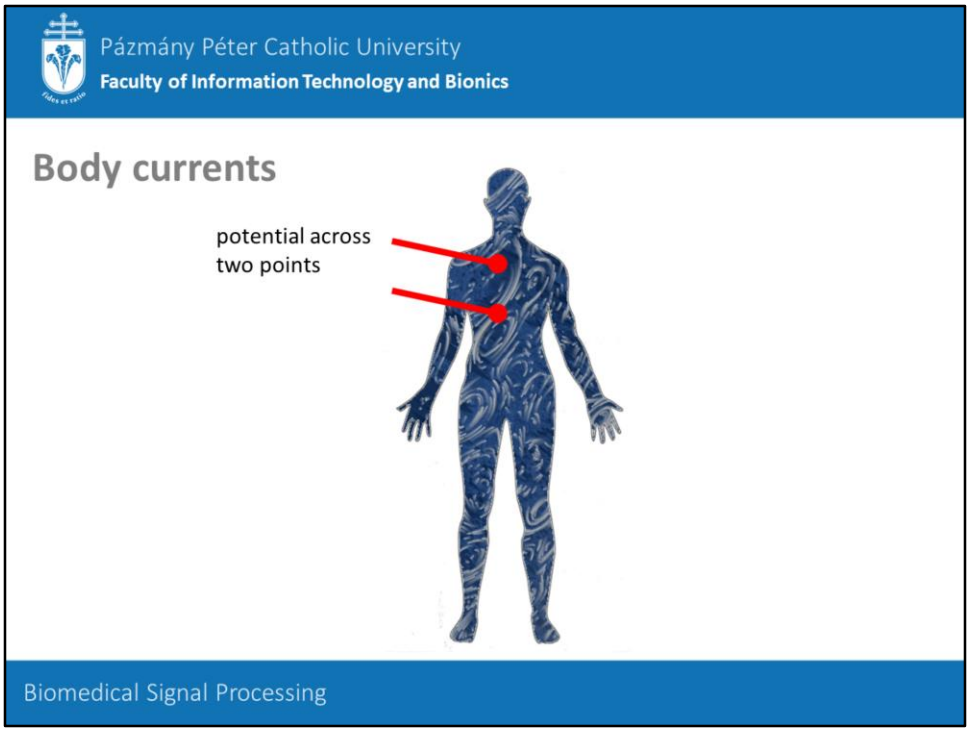


Pulse oximetry (oxygen saturation)

The oxygen saturation is the fraction of oxygen-saturated hemoglobin relative to total hemoglobin (unsaturated + saturated) in the blood.
The principle of this measurement is the **Beer-Lambert Law**, which establishes a relationship between absorbance and concentration of an absorbing liquid.
Oxygenised and deoxygenised blood has different absorbances under different wavelengths. If the absorption on two different wavelengths is measured, the oxygen saturation can be calculated.

Webster, ed. (1998): Medical Instrumentation: Application and Design, p. 452, 471
Tar Ákos: Pulse Oximeter, http://www.soveinc.com/graphics.htm
http://www.electro.co.uk/TCPOexp7.php

Part II – Bioelectric signals



Biocurrents arise in loops.
Between any two points, there will be a potential difference V = IR
If we zoom in, what do we see? How does biocurrent arise?

http://zenstopsigns.blogspot.hu/2012/04/perpetual-oceannasa-goddard-photo-and.html

ENG

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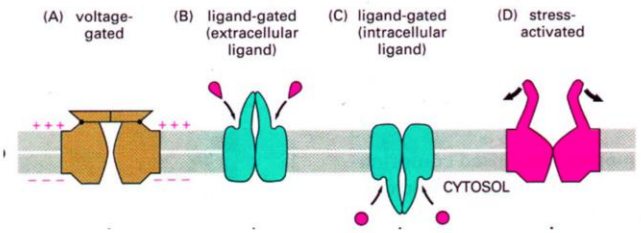
Electric fields produced

- Membrane thickness electric field. (9 MV/m)
- Compare with 1 mm thick spark plug and voltage of 15 kV (15 MV/m)
- The ion separation is established by ion channels

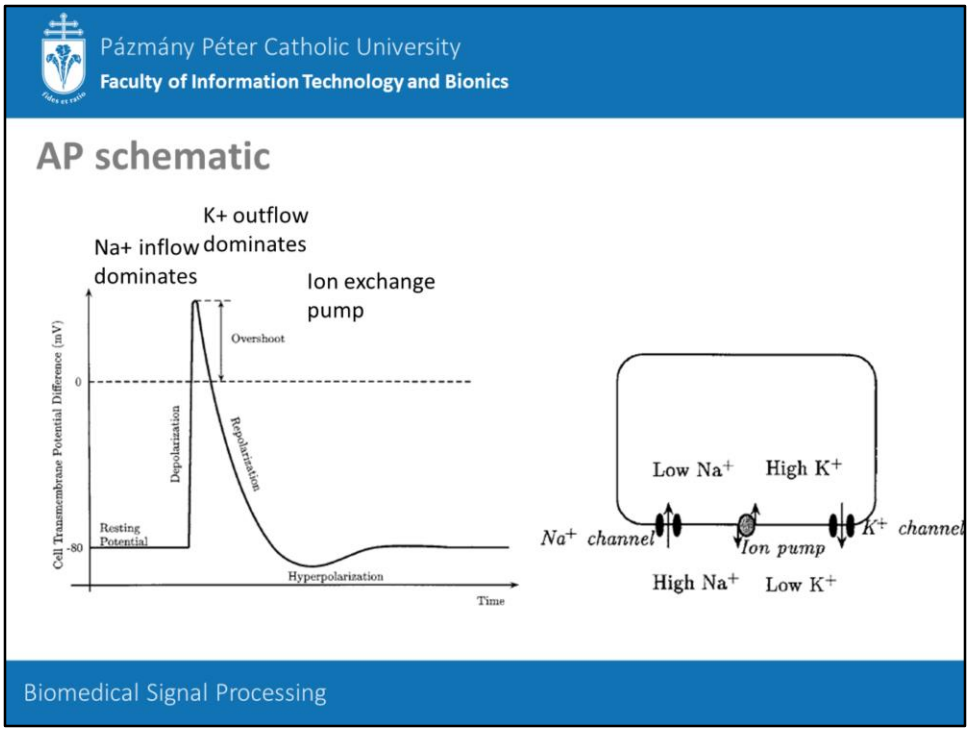


Biomedical Signal Processing

The membrane separates a huge amount of ions compared to its size. Thus a very strong electric field is generated (comparable with a spark plug!), which it endures on a daily basis. The ion separation is established by channels, pores on the lipid bilayer. There are passive (letting in small ions with higher permeability) and active channels, needing some activation. This can be voltage, a ligand causing a rotation of the protein, or motion/stress.

https://en.wikipedia.org/wiki/Voltage-gated_ion_channel
Sodium Na+, Calcium Ca2+, Potassium K+, Chloride Cl-, Proton H+

http://www.boomerpdx.com/baby-boomers-find-their-spark-in-2015/
http://www.mc.vanderbilt.edu/lens/article/?id=177&pg=2



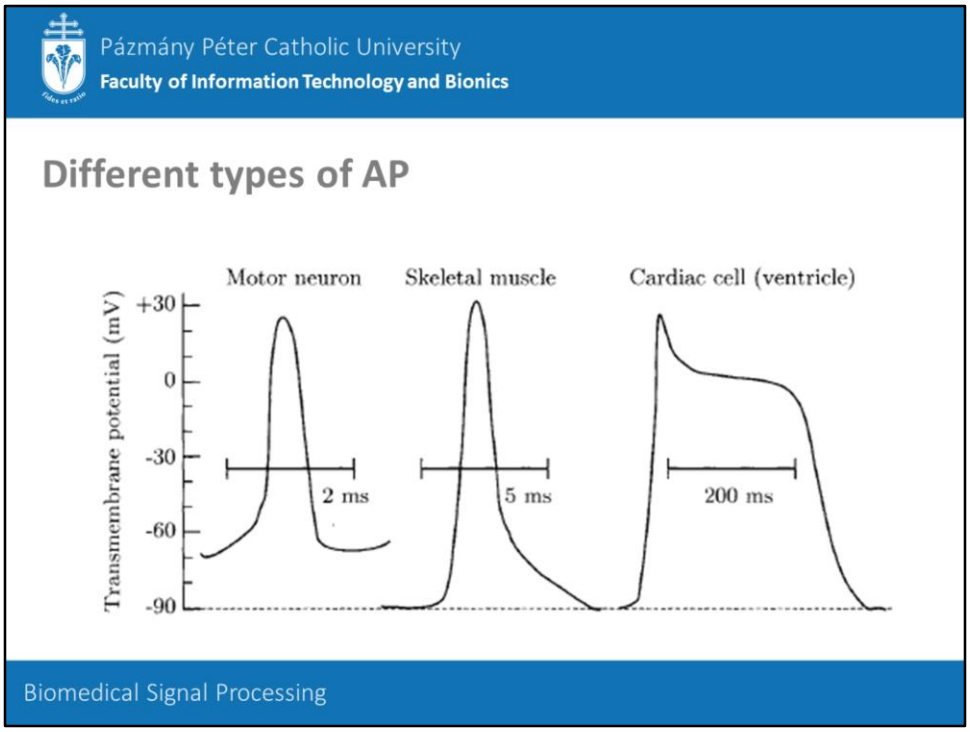
In resting state, wthin the cell (cytosol) there is a lower concentration of Na+ and a higher concentration of K+, compared to the outside, causing a **negative resting potential** compared to the outside of the cell. This potential is usually between -60 and -90mV in a cell.

Flow of primarily Na+, K+, Cl- ions across cell membrane will cause the change of potential.
K+, Cl- leakage across membrane is possible through passive channels, but Na+ permeate x50 less.
(It might be interesting at first! Both Ca2+ and K+ are bigger in size (4th row of periodic table), than Na+ (3rd row), and usually smaller particles permeate easier. However, in Na+ the free electron on the outer layer is closer to the kernel, making its electrically ‚stronger', compared to K+ and Cl+, where the kernel is better shielded from the free electron by the extra electron layer and distance)

The resting potential of the membrane can be disturbed by some current, **stimulated**. When the membrane is the electricity will open voltage-gated Na+ and K+ channels sending Na+ inside and K+ outside of the cell. In the **depolarized** phase under higher voltage a lot more Na+ channel opens than K+, suddenly starting an **action potential**. When Na+ channels get in the refractory period (inactive), the slower K+ channels will **repolarize** the membrane, leading to a **hyperpolarization**, under the resting potential. To balance charge in hyperpolarized state, K+ ions will be resent to the cell,

and **ion pumps** will restore the original ion concentrations. Until the Na+ and K+ channels can be again activized, the membrane will be in **refractory period**.

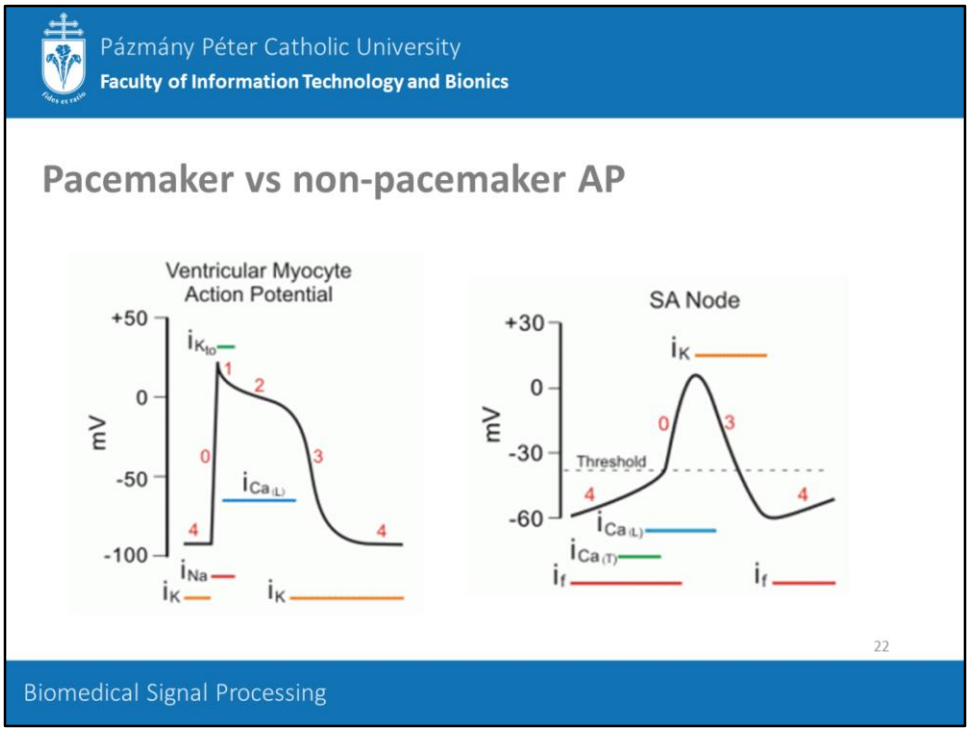
Rangayyan p. 5



The action potential in a **normal skeletal muscle** cell is similar to the action potential in neurons.[56] Action potentials result from the depolarization of the cell membrane (the sarcolemma), which opens voltage-sensitive sodium channels; these become inactivated and the membrane is repolarized through the outward current of potassium ions. The resting potential prior to the action potential is typically −90mV, somewhat more negative than typical neurons. The muscle action potential lasts roughly 2–4 ms, the absolute refractory period is roughly 1–3 ms

The **cardiac action potential** differs from the neuronal action potential by having an extended plateau, in which the membrane is held at a high voltage for a few hundred milliseconds prior to being repolarized by the potassium current as usual. This plateau is due to the action of slower calcium channels opening and holding the membrane voltage near their equilibrium potential even after the sodium channels have inactivated.
The cardiac action potential plays an important role in coordinating the contraction of the hear

https://en.wikipedia.org/wiki/Action_potential#Phases
Sornmö p. 10

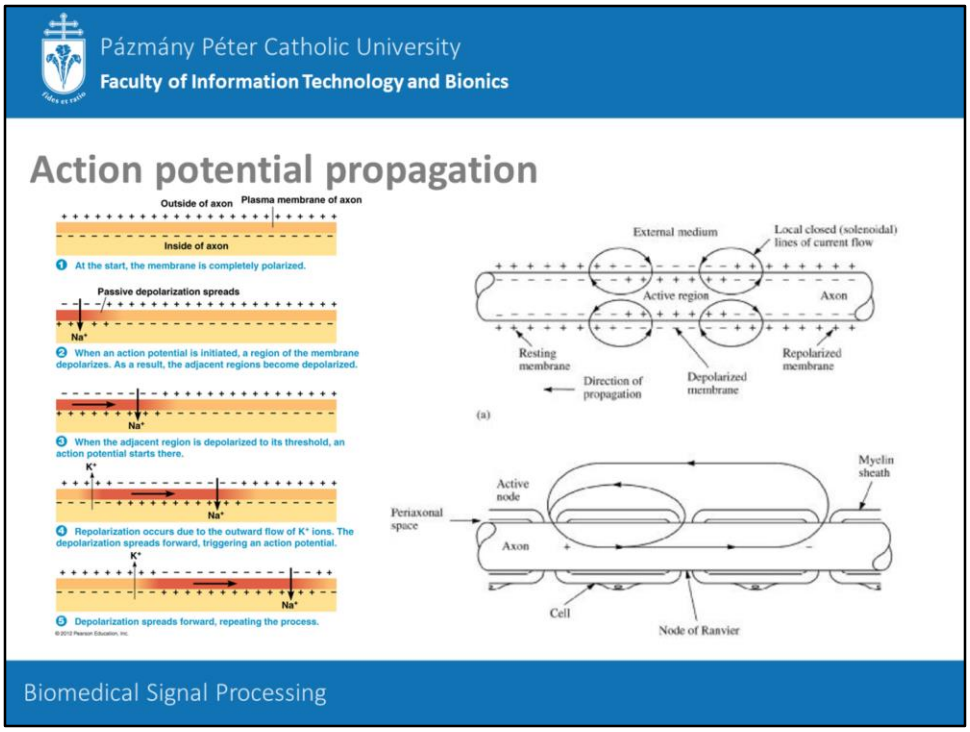


The cardiac cells of the sinoatrial (SA) node provide the **pacemaker potential** that synchronizes the heart. It has a ‚normal' resting potential around -60 mV.
The funny currents (If) are the result of hyperpolarization-activated potassium channels: When the membrane would start to hyperpolarize, this subthreshold current depolarizes, helping the transient and lasting Ca2+ channels to fire a new action potential.

Atrial myocytes, ventricular myocytes and Purkinje cells are examples of **non-pacemaker action potentials** in the heart. Because these action potentials undergo very rapid depolarization, they are sometimes referred to as "fast response" action potentials. They have a long plateau phase as the result of slow Ca2+ channels

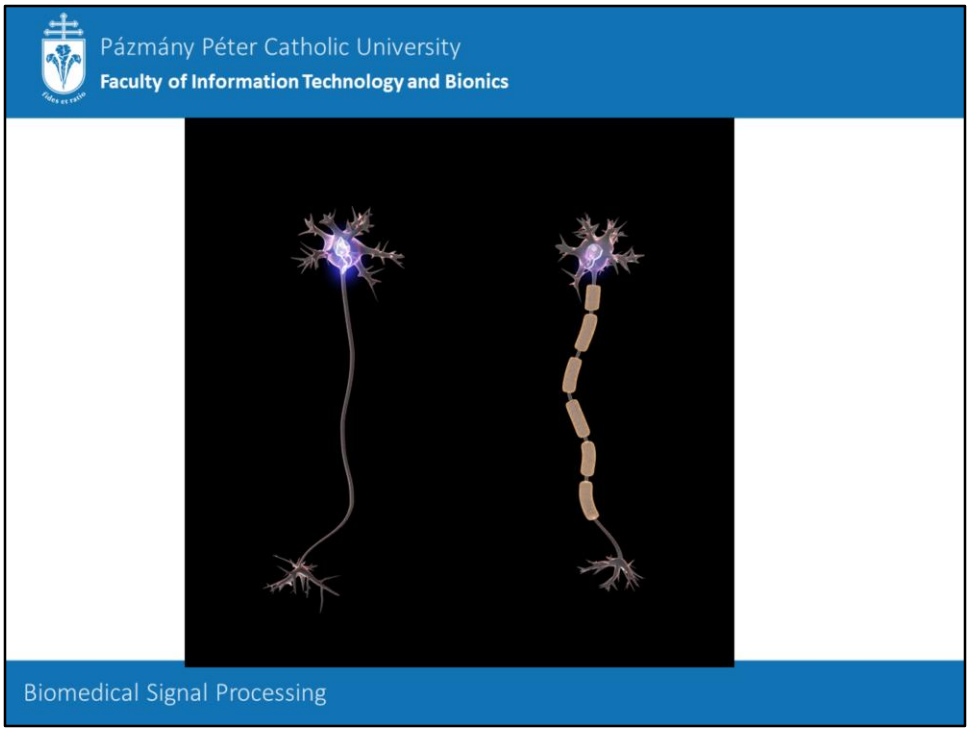
Inward calcium channels
T (transient) : initiate AP
L (long-lasting): sustain AP

http://www.cram.com/cards/61-ventricular-and-pacemaker-action-potentials-2599520



The **action potential** generated at the axon **hillock** propagates as a wave along the axon. The **currents flowing inwards** (flow of current is from positive to negative potential!) at a point on the axon during an action potential creates a **loop** to balance the electric field. In an unmyelated neuron the loop closes on the adjacent patch, in a myelinated on the neighbouring node of ranvier. This will **depolarize the adjacent nodes/patches** of the membrane. If sufficiently strong, this depolarization provokes a similar action potential at the neighboring membrane patches. During repolarization the resting charge-separation will be restored, and until this patch is in its refractory period, it will not be activated, assuring a unidirectional propagation.

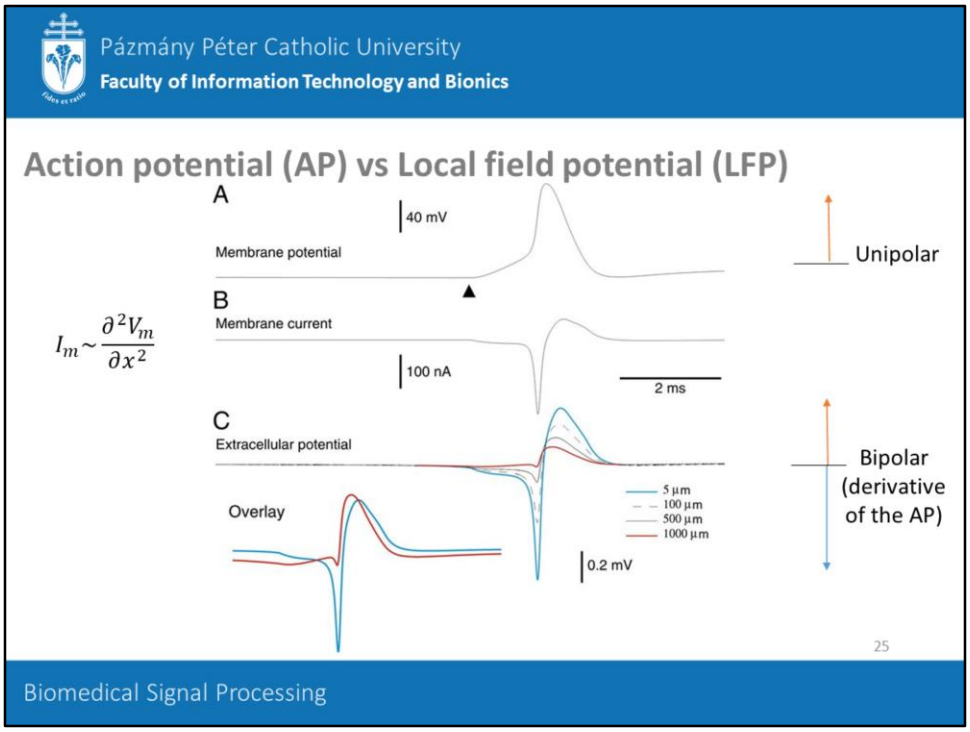
http://www.dokimiscience.com/b---action-potential.html



https://www.youtube.com/watch?v=JTQTa74iDiI

Unmyelinated vs myelinated neuron
In both, you have current loop in extracellular space. Myelin sheath reduces membrane capacitance and increases membrane resistance in the inter-node intervals, thus allowing a fast, saltatory movement of action potentials from node to node. Myelin prevents ions from entering or leaving the axon along myelinated segments. As a general rule, myelination **increases the conduction velocity** of action potentials and makes them **more energy-efficient**. Whether saltatory or not, the mean conduction velocity of an action potential ranges from 1 meter per second (m/s) to over 100 m/s.

https://en.wikipedia.org/wiki/Action_potential#Phases
https://i.imgur.com/odrLrRI.gif

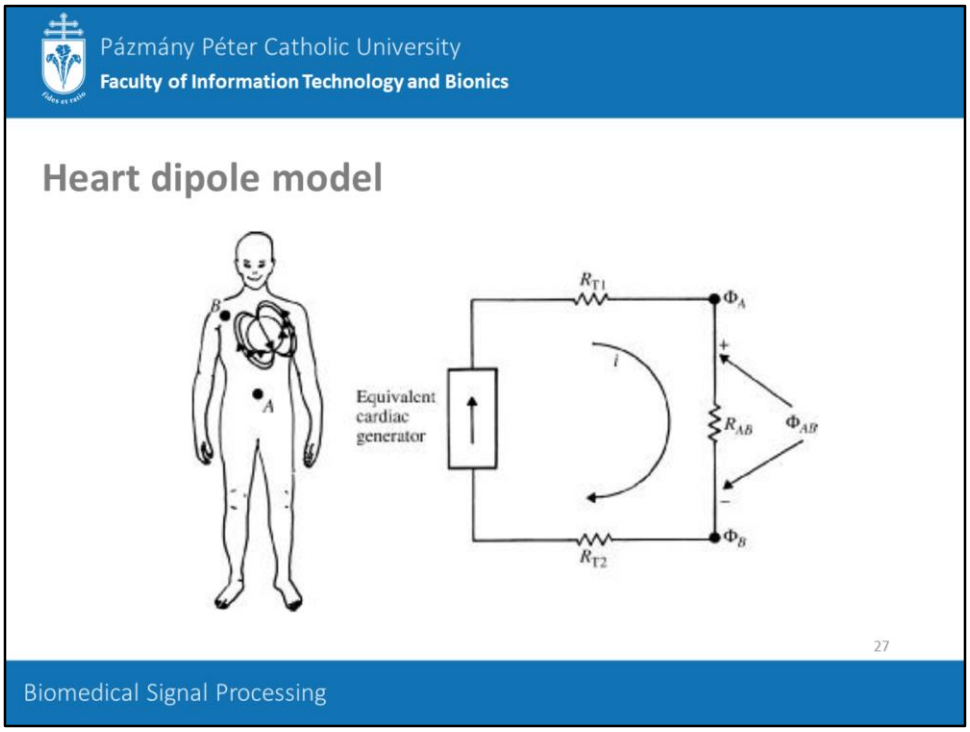


The membrane current is proportional to the second spatial derivative of the membrane potential.

Membrane/action potential approximately unipolar (depolarization is more prominent than the hyperpolarzation).
The **extracellular potential (local field potential, LFP)** can be calculated by derivating the AP -> bipolar.
In addition, there is low-pass filtering of an inhomogeneous **tissue,** which **dampens the sharp spikes**.
Finally, there are several fibers contributing to the local field potential.

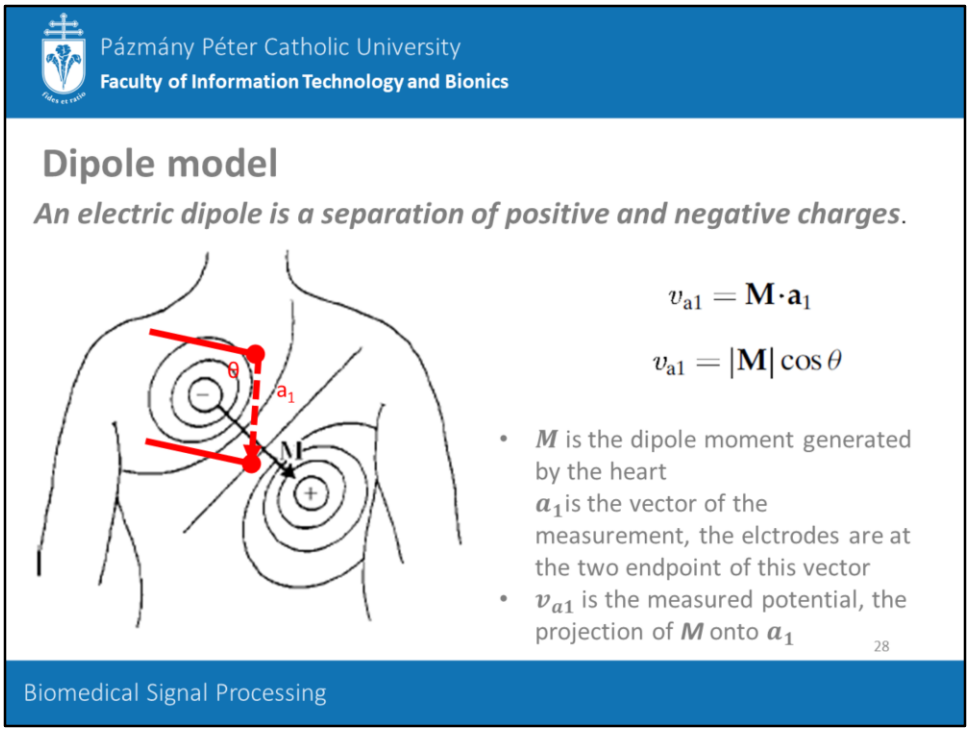
http://www.scholarpedia.org/article/Local_field_potential

https://www.youtube.com/watch?v=RYZ4daFwMa8



Going from LFP to larger, surface potential measurements.
Current sources are added and we can think of one equivalent current source that induces closed current loops
We have lines of current going every which way. This induces a voltage across the two points of measurement.

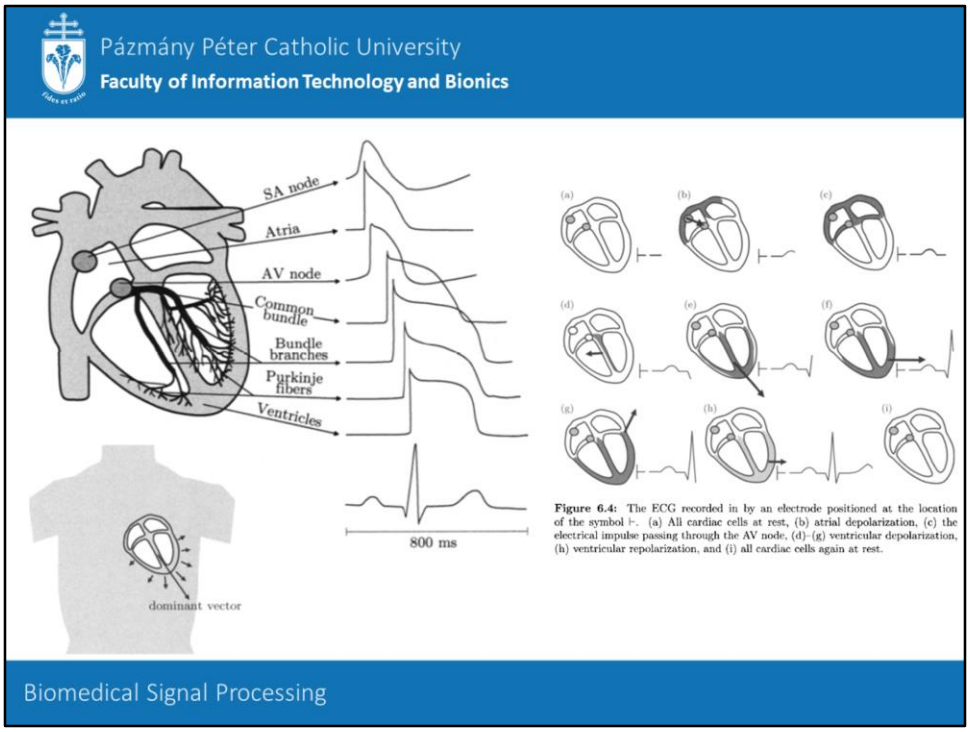
Webster (ed) Medical Instrumentation p. 152



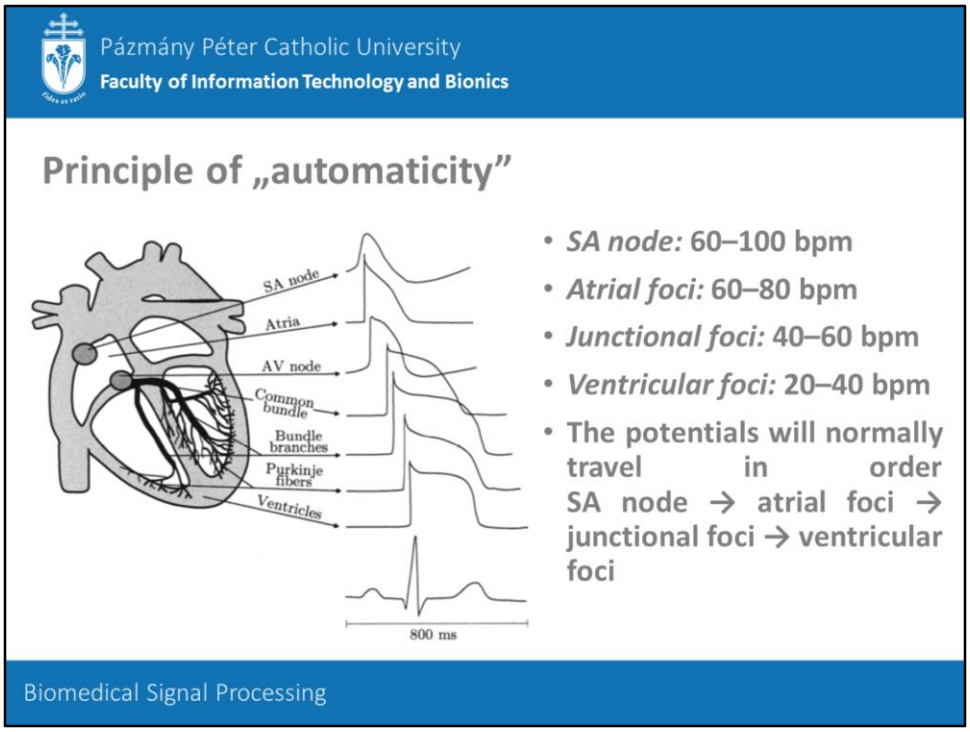
Dipoles can be characterized by their dipole moment, a vector quantity.
The electric dipole moment (M) points from the negative charge towards the positive charge, and has a magnitude (|M|) equal to the strength of each charge times the separation between the charges.

Depending on the angle we measure it, we get different voltages. The measurement v_{a1} is a measurement of M along the vector a_1, so its value will be the projection of M to a_1. This scalar product of the 2 vectors can be calculated equivalently as $|M| \cos(\theta)$, where θ is the angle between M and a_1.
By the way, what is the definition of voltage? Energy to take one unit charge (Coulomb) between two reference points.

Webster (ed) Medical Instrumentation p. 243-245

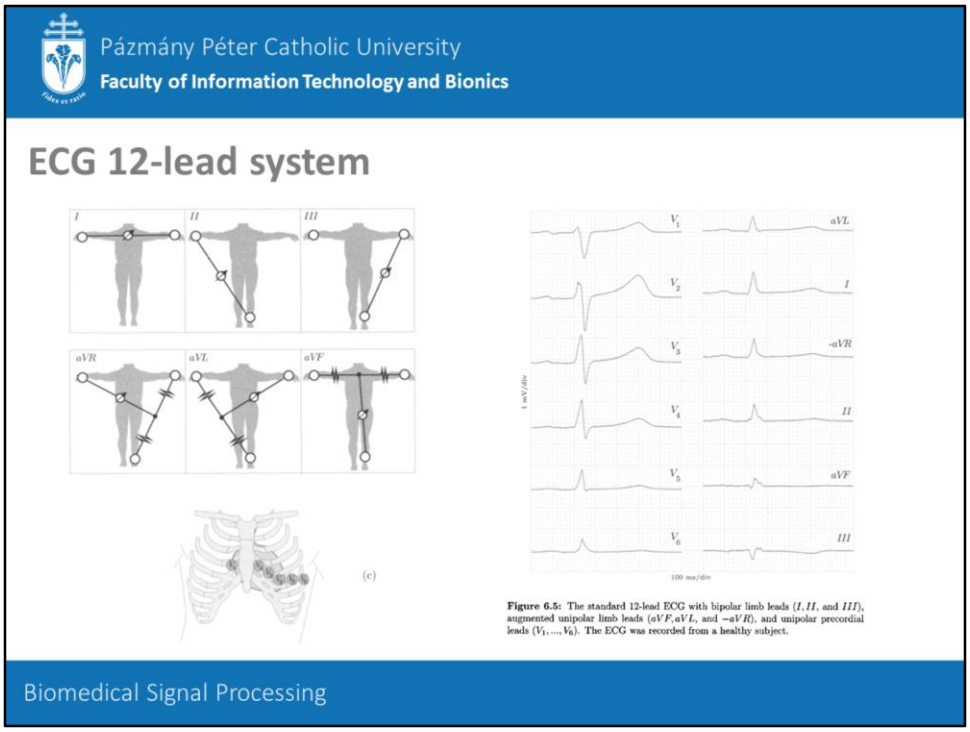


If you check the AP of the heart cells, and sum them simply, they would not add up to a QRS complex.
This is because from each AP generating part of the heart the **current is propagating in a given direction**, so when summing them, this **directional difference** should be considered. Furthermore, QRS will be a local field potential (**LFP**), which is the **derivative of the AP**:



Pacemaker potentials are fired not only by SA node, but also by the other foci. However, the other firing frequencies are slower than the one of the SA node (as seen above). Normally, all the foci will end up firing at the SA node rate, not their intrinsic rate. The other foci attempt to fire at their intrinsic rate, but they are activated by the SA node before they can fire. This rapid firing causes all the foci to fire faster than their intrinsic rates, a phenomenon known as overdrive-suppression. Thus, in the normal, healthy heart, only the SA node intrinsic rate is observable.

http://en.wikipedia.org/wiki/Pacemaker_potential



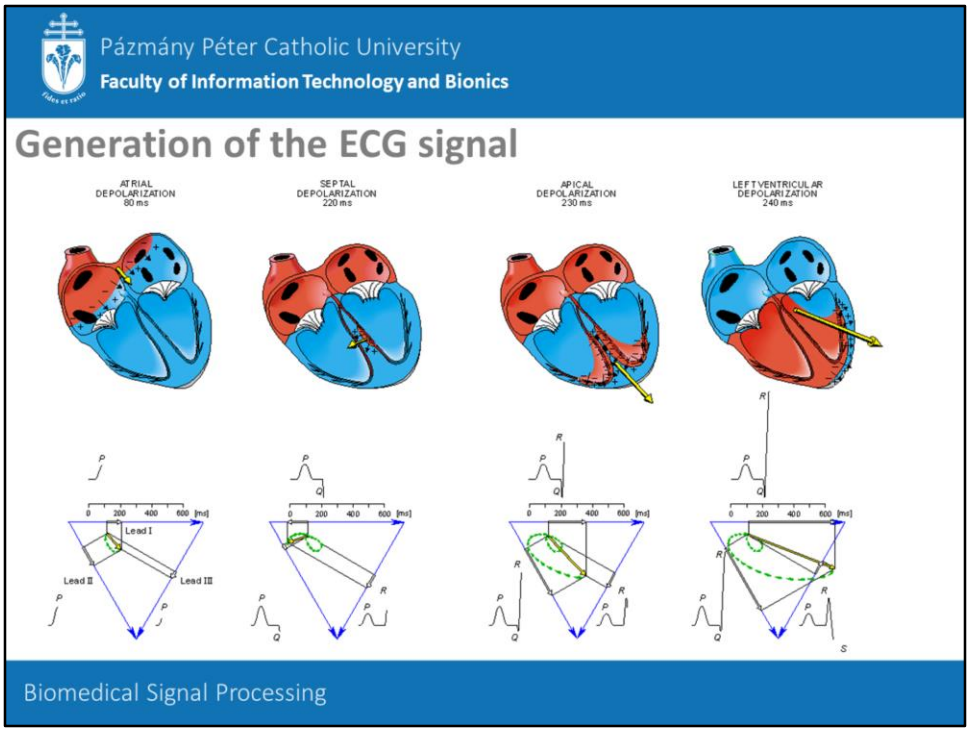
Limb leads: I, II, III
The limb leads form the points of what is known as Einthoven's triangle.
their positioning can be seen on left figure, upper part
I = left arm – right arm
II = left leg- right arm
III = left leg- left arm

Wilson's central terminal V_W, is produced by averaging the measurements from the electrodes RA, LA, and LL (these potentials are measured against the right leg)

Leads aVR, aVL, and aVF are the ***augmented limb leads***. They are derived from the same three electrodes as leads I, II, and III, but they use Goldberger's / Wilson'scentral terminal as their negative pole. Goldberger's central terminal is a combination of inputs from two limb electrodes, with a different combination for each augmented lead

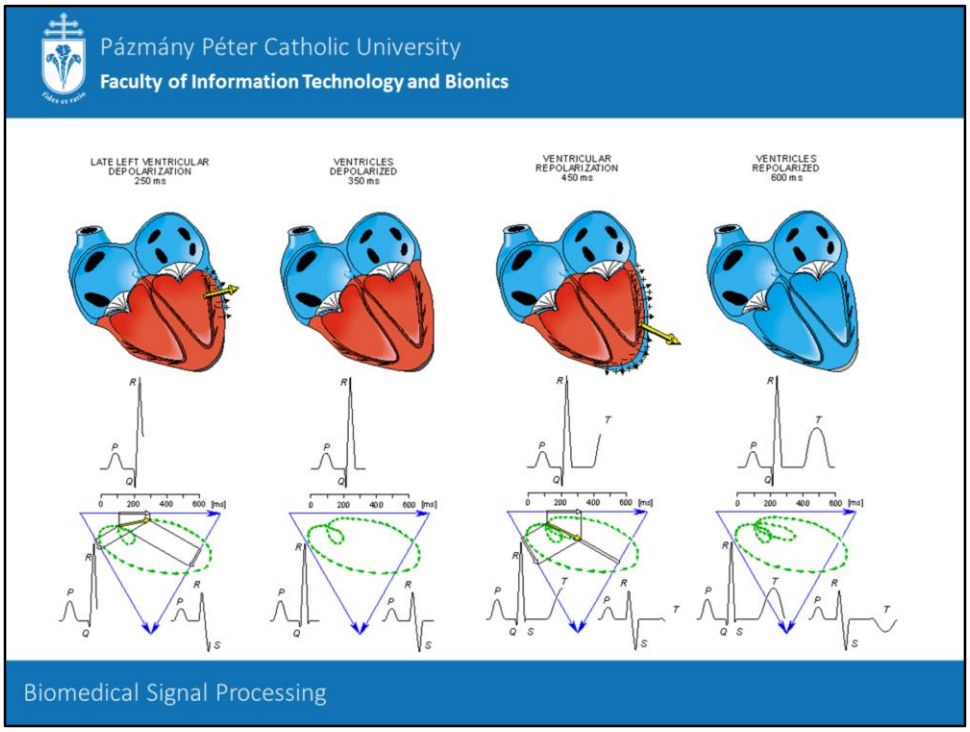
The precordial leads lie in the transverse (horizontal) plane, perpendicular to the other six leads. The six precordial electrodes act as the positive poles for the six corresponding precordial leads: (V_1, V_2, V_3, V_4, V_5 and V_6).
their positioning can be seen on left figure, lower part

Sörnmo and Laguna (2005): Bioelectric Signal Processing, p. 421-423



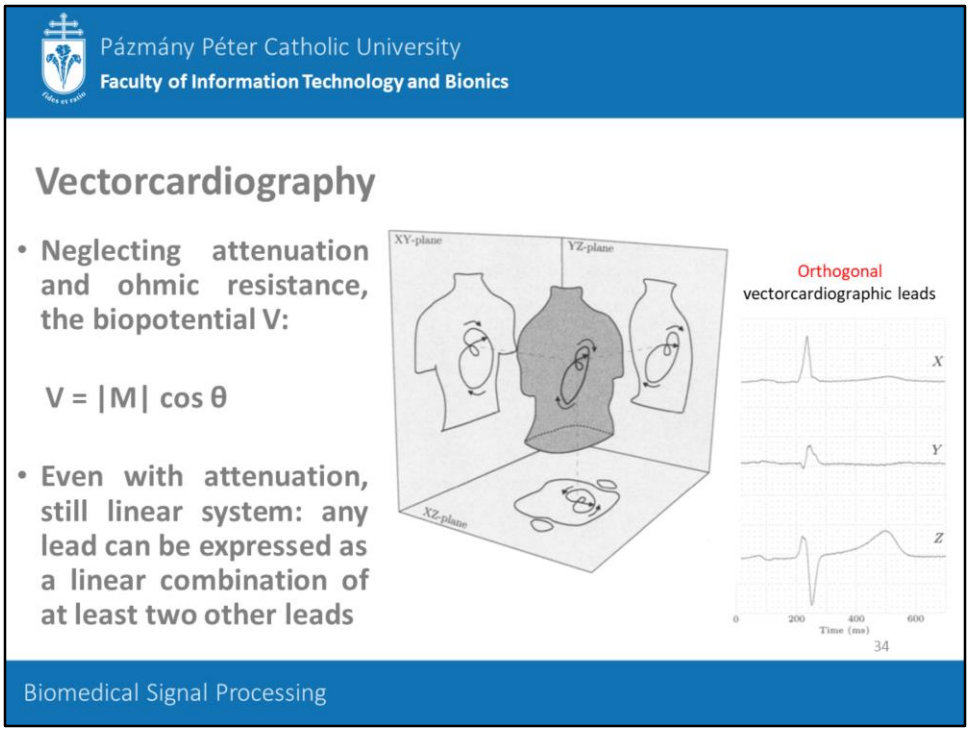
In red the polarized tissue can be seen. The yellow arrow shows the direction of the propagation. On the lower plots the corresponding ECG signal can be read.

http://www.bem.fi/book/15/15.htm
See animation here: http://www.bem.fi/book/15/15x/animati/000.htm



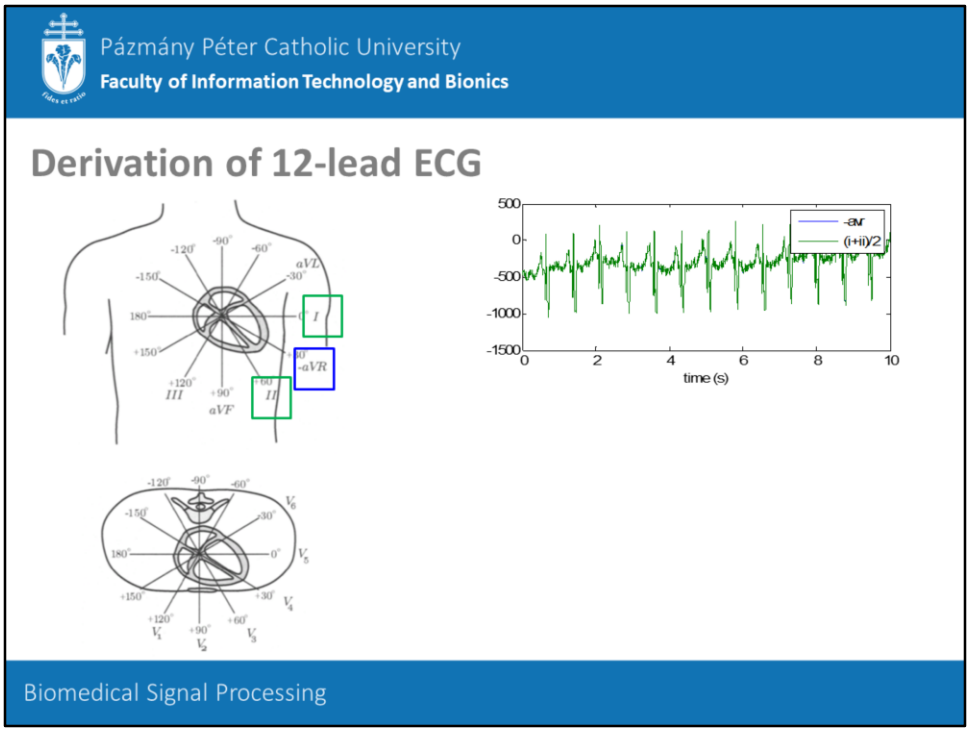
http://www.bem.fi/book/15/15.htm

See animation here: http://www.bem.fi/book/15/15x/animati/000.htm
https://www.youtube.com/watch?v=IS9TD9fHFv0



Using 12 leads is an overcomplete system. The same information can be given using 3 orthogonal signals. It would simplify the eg. storage of the data (but doctors prefer traditional look).

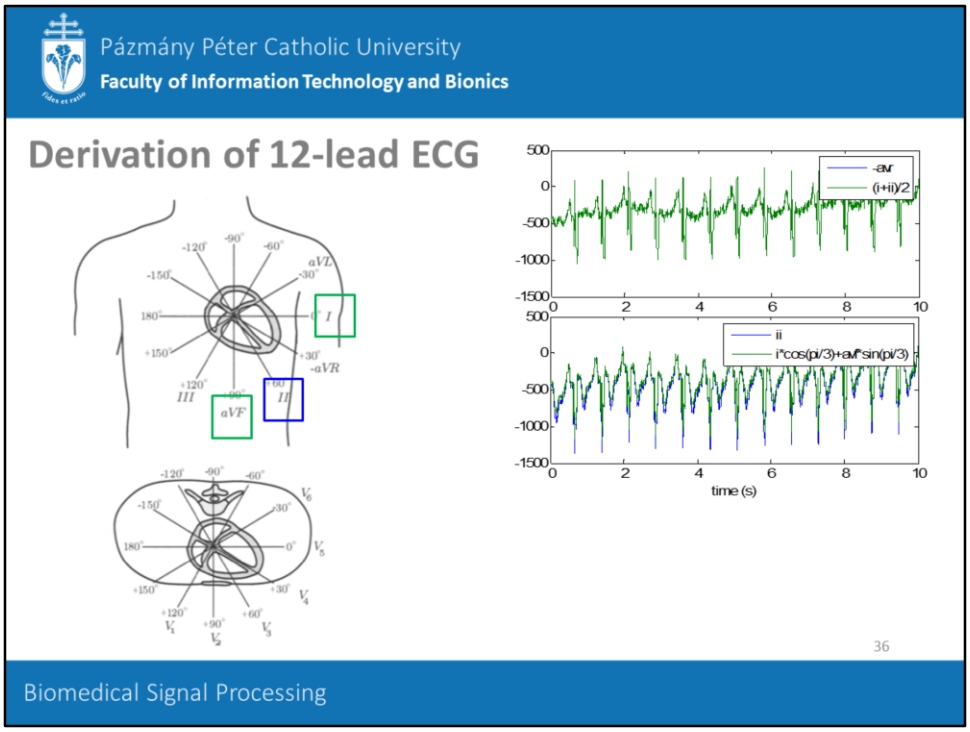
Sörnmo and Laguna (2005): Bioelectric Signal Processing, p. 423-425
Webster, ed. (1998): Medical Instrumentation: Application and Design, p. 235-236



Sörnmo and Laguna (2005):
Bioelectric Signal Processing, p. 423

Data from PhysioNet PTB diagnostic ECG database (http://www.physionet.org/physiobank/database/ptbdb/) acquired with Matlab WFDB toolbox (http://physionet.org/physiotools/matlab/wfdb-swig-matlab/) using command

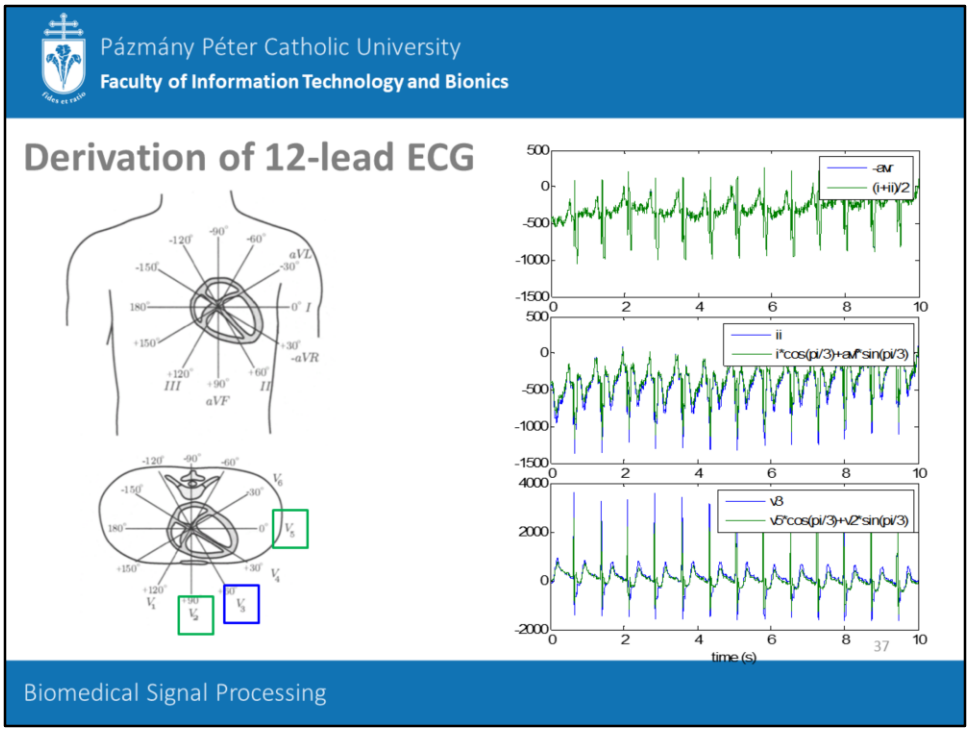
```
y = rdsamp('ptbdb/patient001/s0010_re','begin','00:00:00','stop','00:00:10');
```



The blue signal (II) can be composed from the two green (I and aVF) signals → their linear combination.

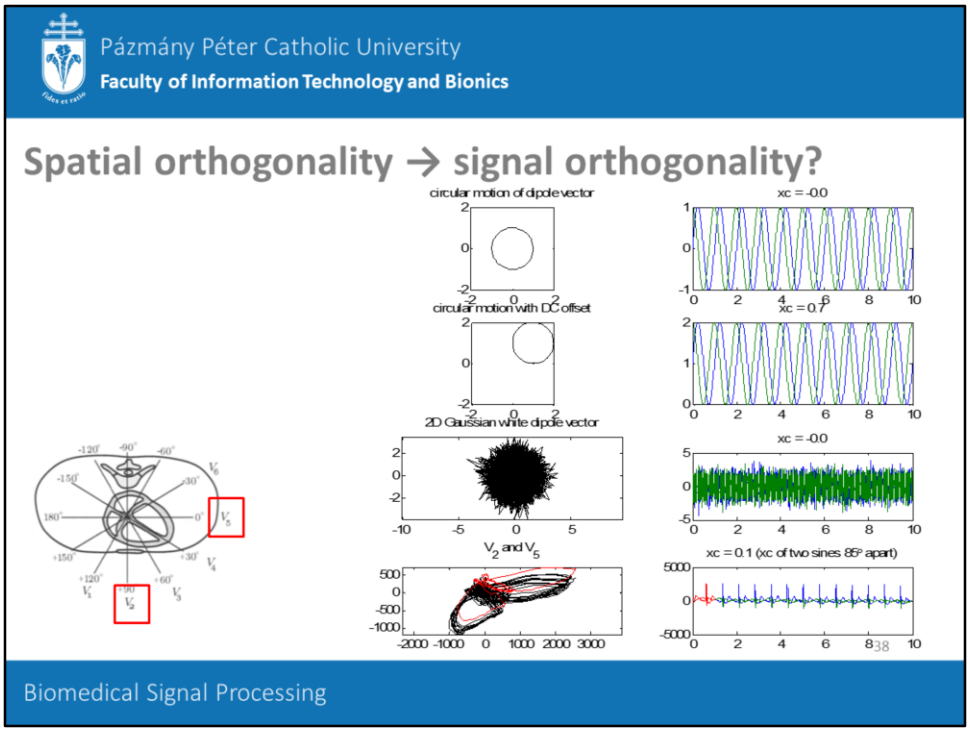
Sörnmo and Laguna (2005):
Bioelectric Signal Processing, p. 423
Data from PhysioNet PTB diagnostic ECG database (http://www.physionet.org/physiobank/database/ptbdb/) acquired with Matlab WFDB toolbox (http://physionet.org/physiotools/matlab/wfdb-swig-matlab/) using command

```
y = rdsamp('ptbdb/patient001/s0010_re','begin','00:00:00','stop','00:00:10');
```



Sörnmo and Laguna (2005):
Bioelectric Signal Processing, p. 423
Data from PhysioNet PTB diagnostic ECG database (http://www.physionet.org/physiobank/database/ptbdb/) acquired with Matlab WFDB toolbox (http://physionet.org/physiotools/matlab/wfdb-swig-matlab/) using command

```
y = rdsamp('ptbdb/patient001/s0010_re','begin','00:00:00','stop','00:00:10');
```



V2 is orthogonal to V2. Does it mean, that these measurements will be decorrelated?

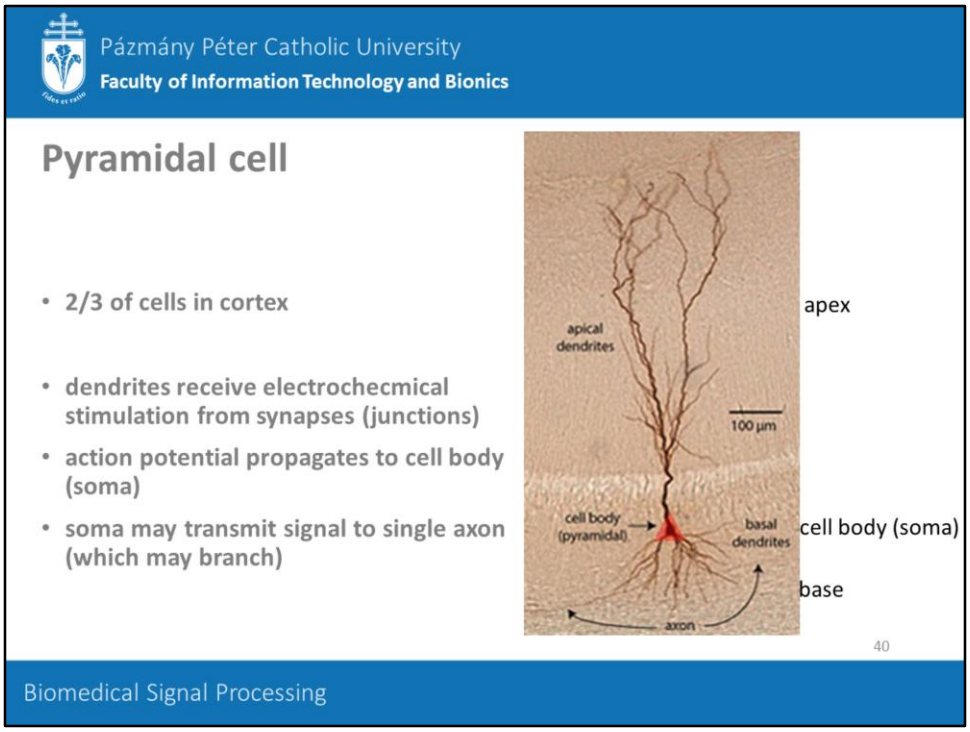
Does spatial orthogonality mean zero cross-correlation? The lead signals are projections of a vector signal, but the components of the vector may in some instances be correlated (it is not necessariliy the orthogonal coordinate system of the signal). In the case of the ECG signal, however, they are quite decorrelated (similarly to a rotating vector, which is not unlike the motion mapped by the vector during ventricular depolarization)

Sörnmo and Laguna (2005):
Bioelectric Signal Processing, p. 423

Data from PhysioNet PTB diagnostic ECG database (http://www.physionet.org/physiobank/database/ptbdb/) acquired with Matlab WFDB toolbox (http://physionet.org/physiotools/matlab/wfdb-swig-matlab/) using command

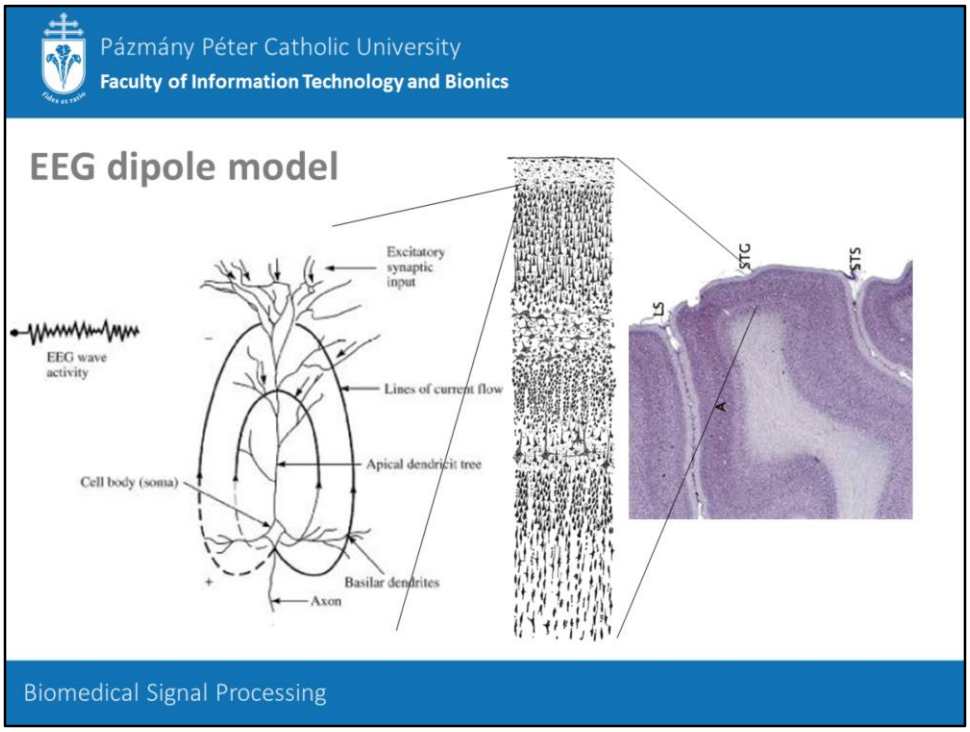
```
y = rdsamp('ptbdb/patient001/s0010_re','begin','00:00:00','stop','00:00:10');
```

EEG



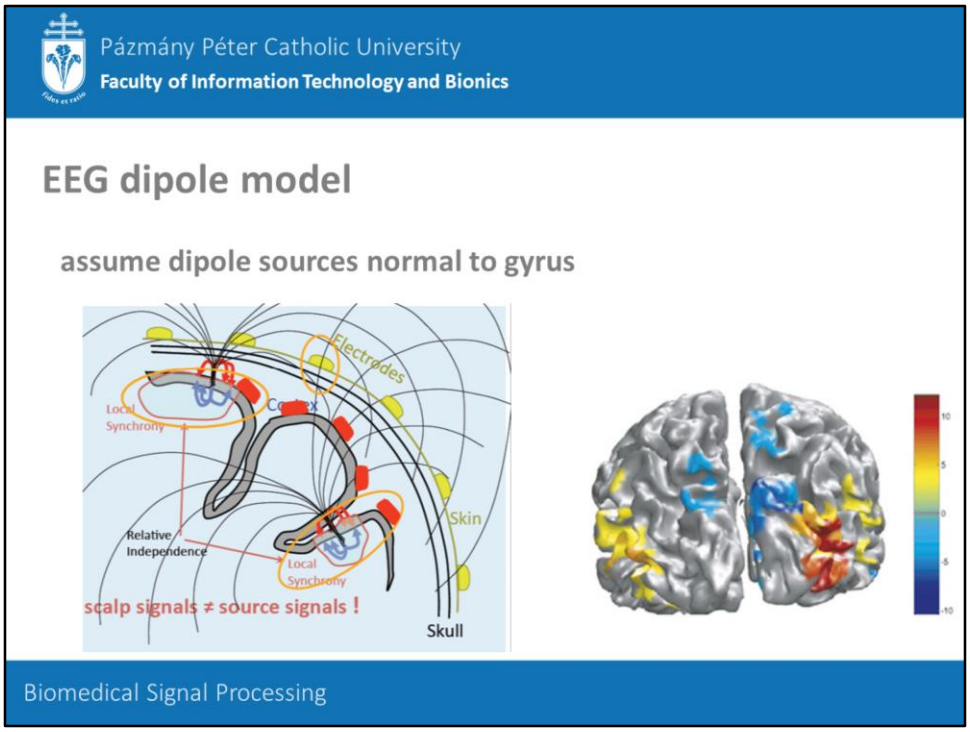
pyramidal neurons, are a type of multipolar neuron found in areas of the brain including the cerebral cortex, the hippocampus, and the amygdala.

http://www.scholarpedia.org/article/Pyramidal_neuron
http://www.cell.com/current-biology/pdf/S0960-9822(11)01198-5.pdf



When there is an **excitatory** input, current flows towards base. A loop formed in the extracellular matrix that then flows the other way,
so that extracellular medium near apex acts as sink (-), extracellular space near soma is source (+). reversed if **inhibitory** input. This will be the dipole model of the cell. These cells are oriented perpendicularly to the cortex.

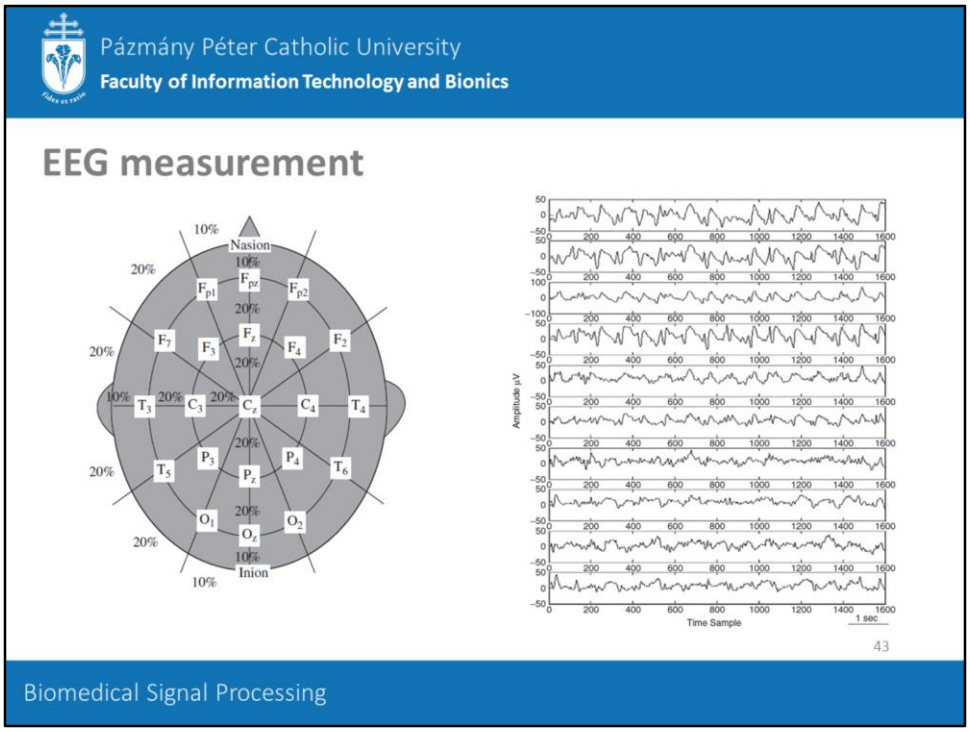
Webster, ed. (1998): Medical Instrumentation: Application and Design, p. 163-165
http://en.wikipedia.org/wiki/Cerebral_cortex



On the left in red is the positive side, blue is the negative side of the charge separation (depends on inhibitory/excitatory nature). The dipole points from the positive to the negative side. The black lines are the loops closing around the dipole, on this line the electrodes will measure the potential difference. Those lines, which are perpendicular to the skull, will overdominate those, which lie within the fissure and are tangeltial to the skull.

This orientation means, that dipoles in cortex paralell with the skull will be measurable from the scalp, but where the cortex has a groove (sulcus), the dipoles will be paralell to the scalp, thus will not be measureable.

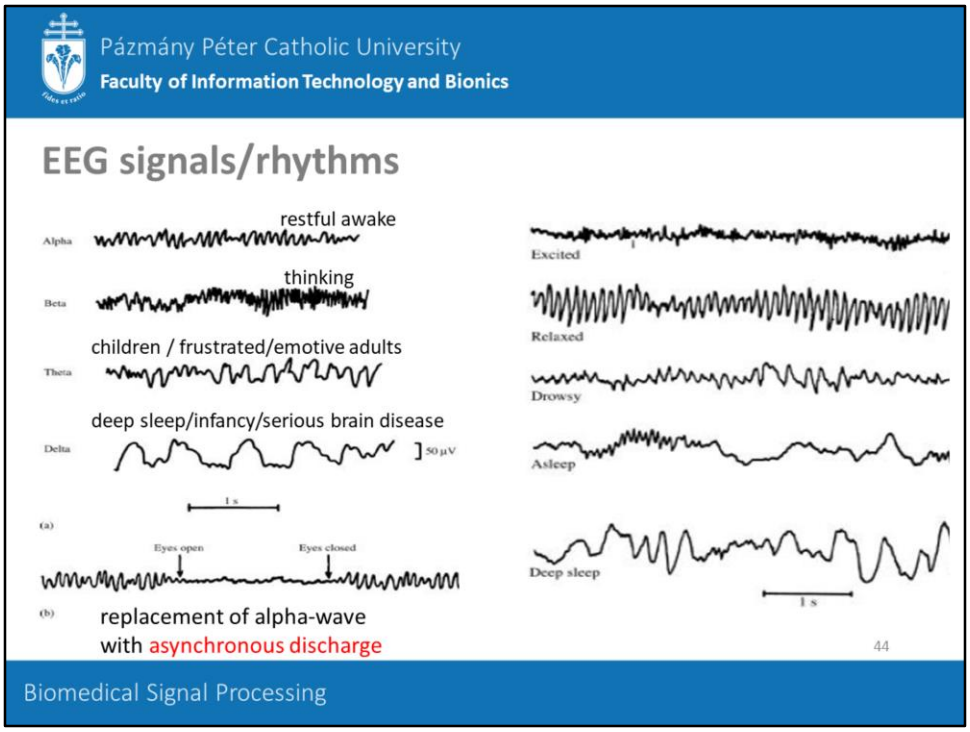
ftp://sccn.ucsd.edu/pub/bcilab/lectures/02_EEG_Basics.pdf
http://ars.sciencedirect.com/content/image/1-s2.0-S0013469497001168-gr1.gif
Grova et al. (2008): Concordance between distributed EEG source localization ...



The 10–20 system or **International 10–20 system** is an internationally recognized method to describe and apply the location of scalp electrodes

reference placed at left or right earlobe or both. The "10" and "20" refer to the fact that the actual distances between adjacent electrodes are either 10% or 20% of the total front–back or right–left distance of the skull.

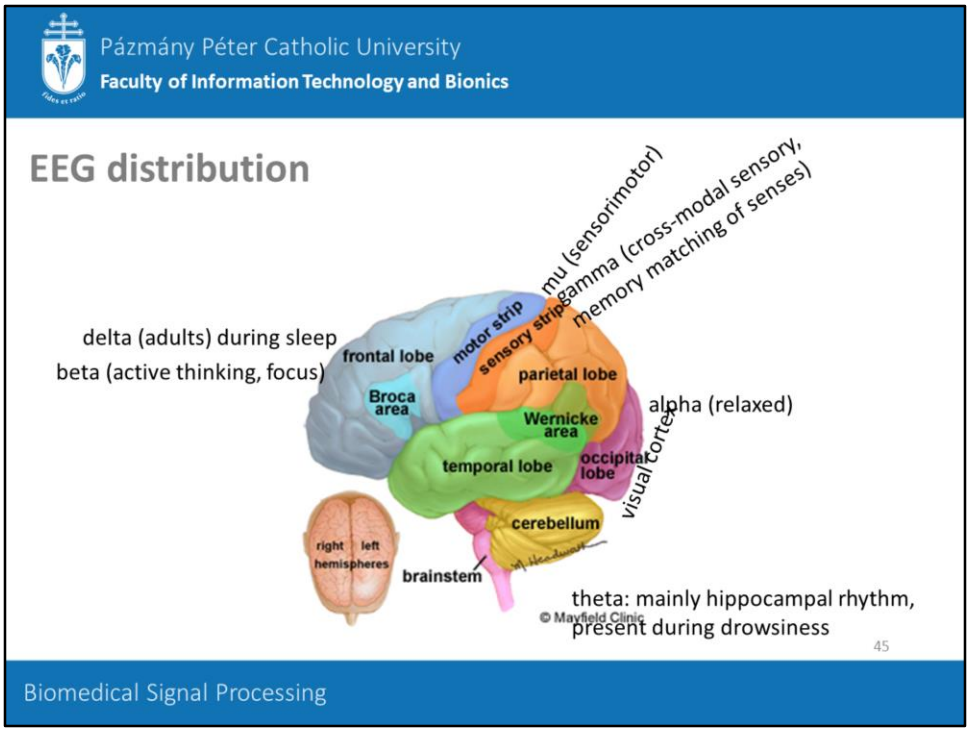
Sanei and Chambers (2007): EEG Signal Processing
https://en.wikipedia.org/wiki/10–20_system_(EEG)



Delta: 0.5-4 Hz, Theta: 3-8, Alpha: 8-13, Beta: 13+, Gamma: 30+,
They will be further dicussed next lecture

Webster (ed): pp. 172-177

Deep sleep music?
https://www.youtube.com/watch?v=IIv__ux2f70
If you hear taps in the range of 0-10 Hz, you can hear the individual taps. If the taps are greater than 60 Hz, they produce a pitch. However, in the range of 10-60 Hz, it is hard to interpret.
http://www.phys.uconn.edu/~gibson/Notes/Section5_5/Sec5_5.htm
https://mynoise.net/NoiseMachines/binauralBrainwaveGenerator.php
http://www.szynalski.com/tone-generator/



https://en.wikipedia.org/wiki/Electroencephalography
https://www.mayfieldclinic.com/PE-AnatBrain.htm
http://www.springer.com/gp/book/9783540879183

EMG

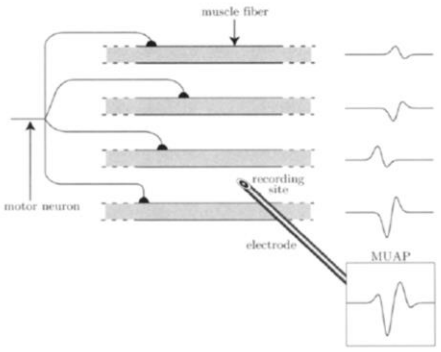
Pázmány Péter Catholic University
Faculty of Information Technology and Bionics

Motor unit action potentials (MUAP)

- **Biphasic LFP**
- Motor neuron and fibers to which it connects known as ***motor unit***
- **MUAP:** summation of contribution of single fibers.

Characteristics:

- Amplitude: 0.25-5 mV
  Low: myopathy
  Large: neuropathy
- *Polyphasic:* misalignment of AP
- Duration: 2-10 ms. Increases with number of fibers



Biomedical Signal Processing

motor unit action potential - muap

Sörnmo and Laguna (2005): Bioelectric Signal Processing, p. 339

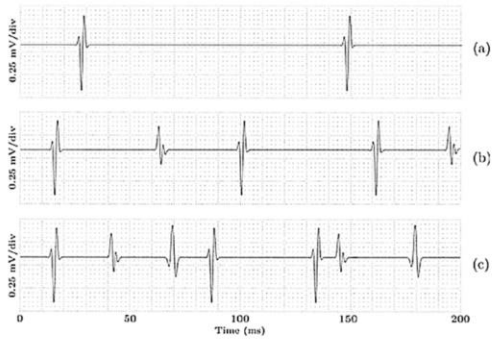
Pázmány Péter Catholic University
Faculty of Information Technology and Bionics

Motor unit recruitment

credit question: which type of recruitment is seen below?

Increment of muscular contraction achieved by:

- **Spatial recruitment:** activation of new motor units with increasing effort
- **Temporal recruitment:** increase firing rate of each motor unit with increasing effort



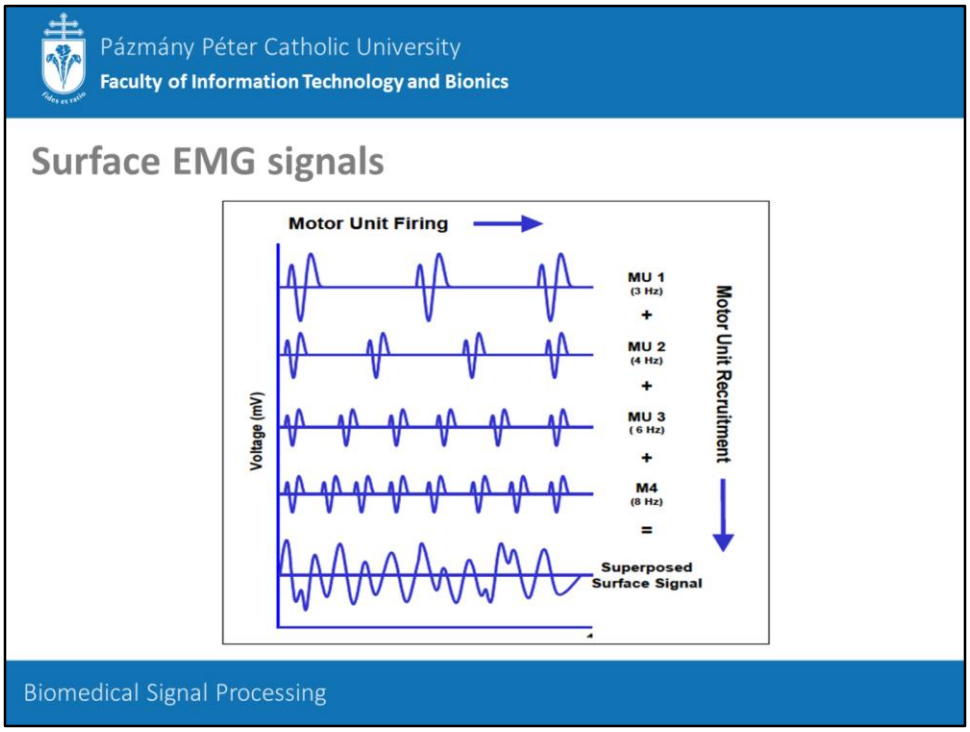
48

Biomedical Signal Processing

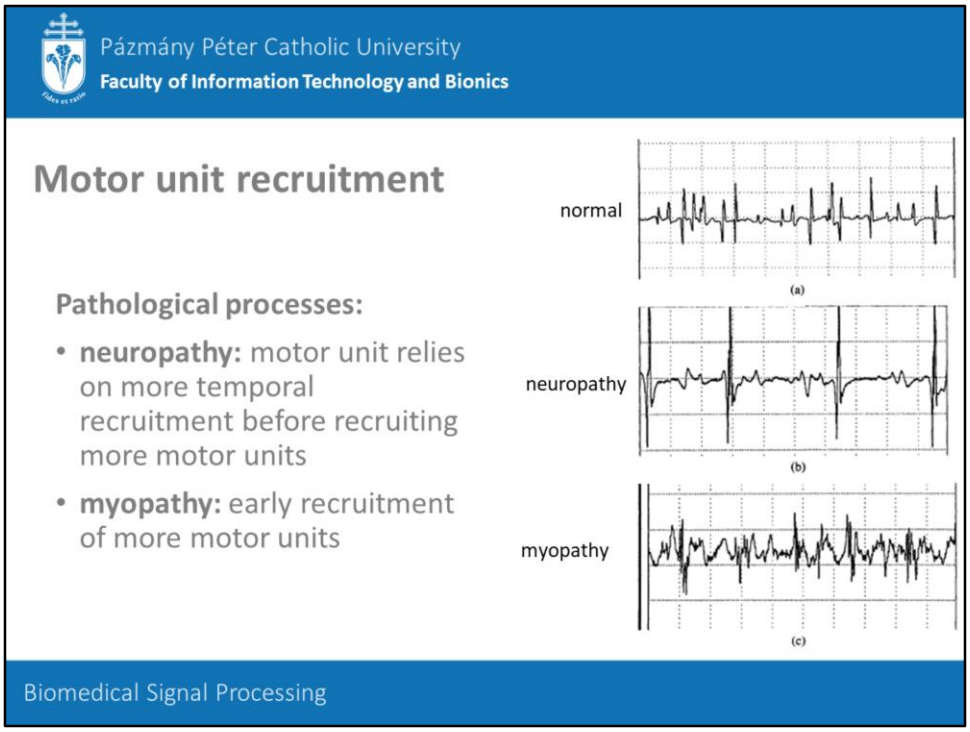
When larger muscle force is needed, either more Mus are involved, or the firing rate will be higher.

In the plot both kind of recruitments can be observed:
Spatial recruitment: Waves with different shape are coming from different MUs.
Temporal recruitment: Frequency of same waneform is higher

Sörnmo and Laguna (2005): Bioelectric Signal Processing, p. 340
Rangayyan p. 15



The ABC of EMG -- http://www.noraxon.com/docs/education/abc-of-emg.pdf?sfvrsn=0



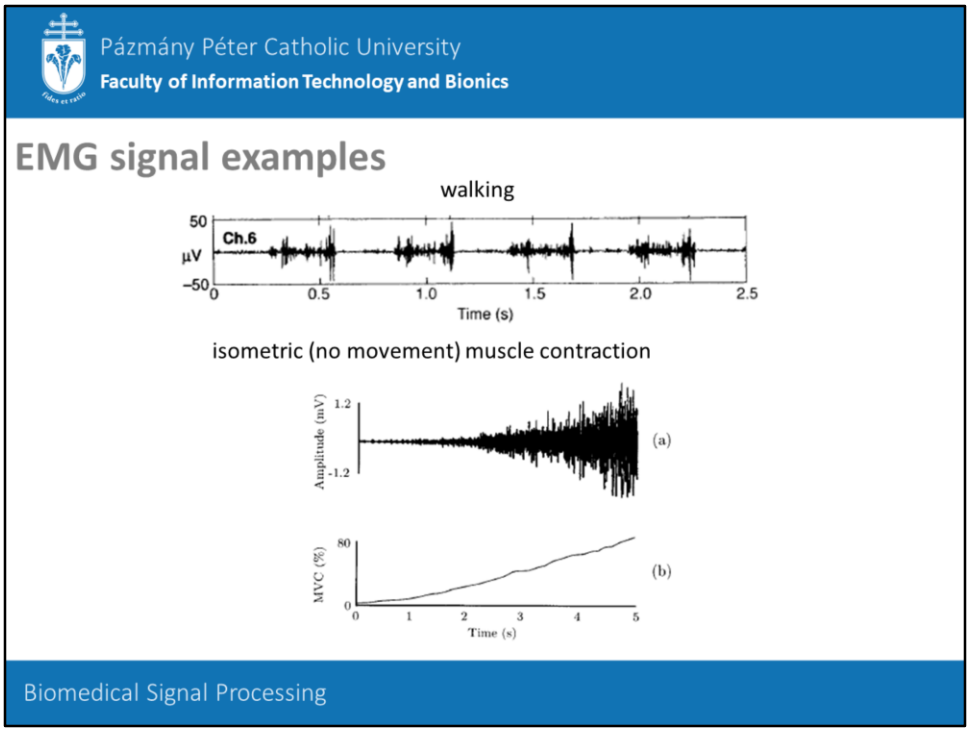
„Figure 1.8. Examples of SMUAP trains.

(a) From the right deltoid of a normal subject, male, 11 years,, the SMUAPs are mostly biphasic, with duration in the range 3-5 ms. (surface detected MUAPs are biphasic)

(b) From the deltoid of a 6-month-old male patient with brachial plexus injury (neuropathy),, the SMUAPs are polyphasic and large in amplitude (800 uV), and the same motor unit is firing at a relatively high rate at low-to-medium levels of effort.

(c) From the right biceps of a 17-year-old-male patient with myopathy,, the SMUAPs are polyphasic and indicate early recruitment of more motor units at a low level of effort. The signals were recorded with gauge 20 needle electrodes. The width of each grid bx represents a duration of 20 ms,, its height represents an amplitude of 200 uV. […]"

Sörnmo and Laguna (2005): Bioelectric Signal Processing, p. 340
Rangayyan p. 15

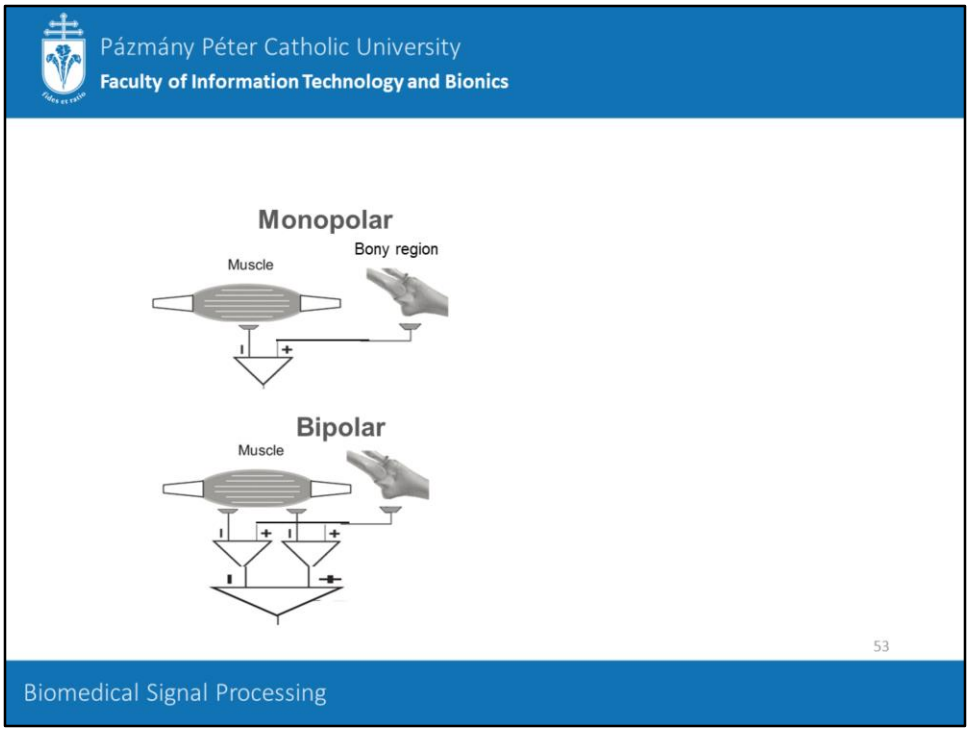


Upper: Surface EMG recorded during walking at natural speed. The electrodes were positioned on the leg over the tibialis anterior muscle.

Lower: (a) The EMG signal and (b) the force curve during force-varying isometric muscle contraction, expressed as a percentage of maximal voluntary contraction”

Sörnmo and Laguna (2005): Bioelectric Signal Processing, p. 346, 386

EXTRA MATERIAL

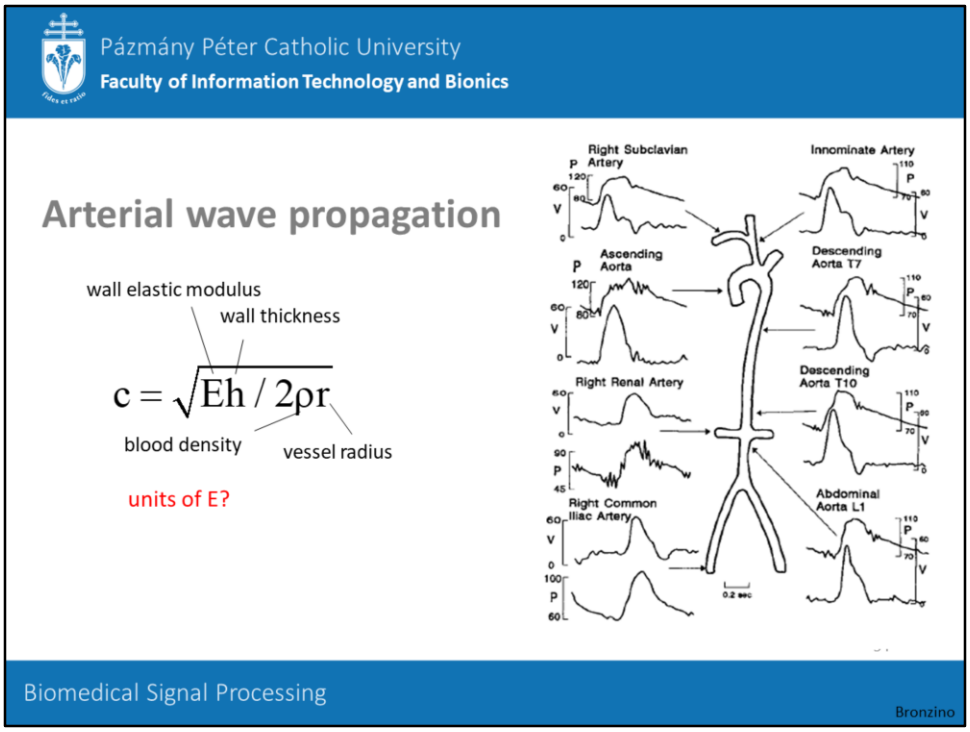


Detection area?

For larger muscles a larger electrode should be used

What is the difference between a monopolar and a bipolar electrode?

Monopolar EMGs correspond to the electrical potential detected on the surface of the skin, immediately above the muscle tissue, with respect to that measured with a reference electrode located at bony regions on the. It might also record interferences from outside sources (for example power line) or the activity of sources (for example distant muscles) other than the muscle investigated. The latter phenomenon, known as crosstalk, is likely reduced with the use of bipolar montages. The potentials of distant MUAPs and deep MUs appear with the same amplitude in the monopolar EMG. Given that a bipolar results from the difference between two monopolar EMGs, the common-mode voltage embedded in both signals from interfering source, appear with very similar amplitudes on both electrodes and, then, is fairly attenuated in the differentiated signal. The degree of cancellation depends on the characteristic of differential amplifiers, and on the unbalance in the electrode-skin impedances between the two recording sites. While bipolar recordings are less sensitive to interference and cross-talk, they reduce the «detection volume» and attenuate the contribution of deep MUs to the surface EMGs.



Vessel elasticity (storage) means waves can propagate (waves require storage of energy via a reaction force, transfer of energy, and inertia)
Reflection

At wavelength λ_1, $I_1 = I_{in1} 10^{-(\alpha_{o1} C_o + \alpha_{r1} C_r) l}$

At wavelength λ_2, $I_2 = I_{in2} 10^{-(\alpha_{o2} C_o + \alpha_{r2} C_r) l}$

ion

where

- C_o is the concentration of oxyhaemoglobin (HbO_2)
- C_r is the concentration of reduced haemoglobin (Hb)
- α_{on} is the absorption coefficient of HbO2 at wavelength λ_n
- α_m is the absorption coefficeint of Hb at wavelength λ_n

assuming attenuation only due to HbO_2, Hb

$$R = \frac{\log_{10}(I_1/I_{in1})}{\log_{10}(I_2/I_{in2})}$$

$$SaO_2 = \frac{C_o}{C_o + C_r} = \frac{\alpha_{r2} R - \alpha_{r1}}{(\alpha_{r2} - \alpha_{o2}) R - (\alpha_{r1} - \alpha_{o1})}$$

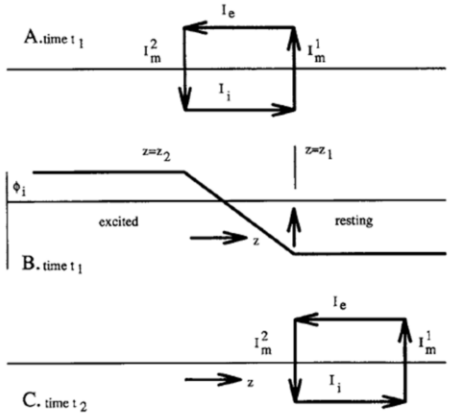
$$R = \frac{\log_{10}((I_{dc+ac})/(I_{dc}))_{\lambda 1}}{\log_{10}((I_{dc+ac})/(I_{dc}))_{\lambda 2}}$$

accounting for attenuation through tissue and veins

55

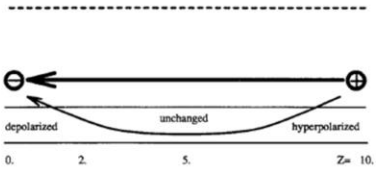
Neil Townsend (2001): Pulse Oximetry (from Medical Electronics lecture course). http://www.robots.ox.ac.uk/~neil/teaching/lectures/med_elec/notes6.pdf

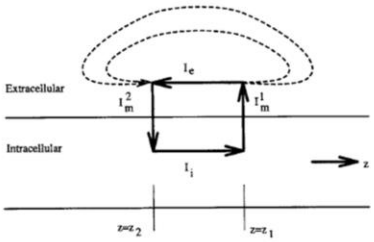
Propagation of AP



Extracellular biopotential

- Usual propagation of sources: waveform remains unchanged
- Here, propagation to observer of propagation along line
- Difference between intracellular and extracellular processes





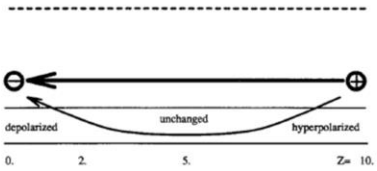
Extracellular biopotential

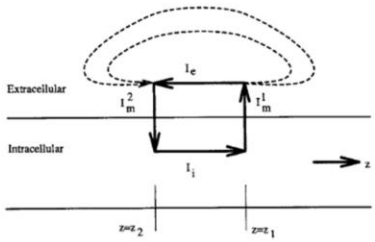
$$\frac{\partial \Phi_i}{\partial x} = -I_i r_i \,, \quad \frac{\partial \Phi_o}{\partial x} = -I_o r_o$$

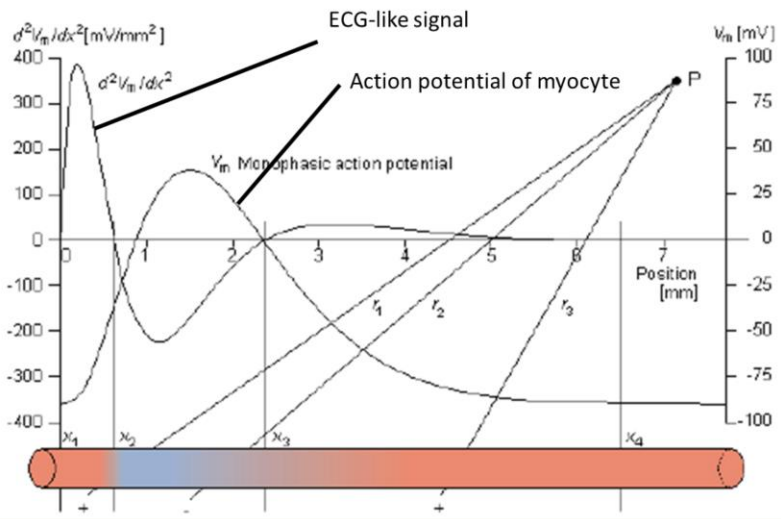
$$i_m = -\frac{\partial I_i}{\partial x} = \frac{\partial I_o}{\partial x}$$

$$i_m = \frac{1}{r_i}\frac{\partial^2 V_m}{\partial x^2}$$

$V_{ext} \sim i_m$



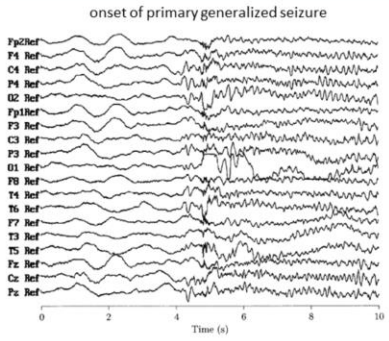


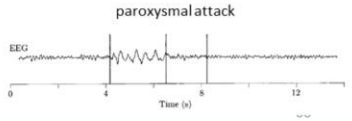


Ponsey: Bioelectromagnetism

Non-stationarity of EEG

- Underlines importance of time-frequency analysis
- Segmentation necessary to identify different segments of activity (S&L p. 125, R p. 431)





Sörnmo and Laguna (2005): Bioelectric Signal Processing, p. 43, 131