Biomedical Sensors and Signals

Skriptum

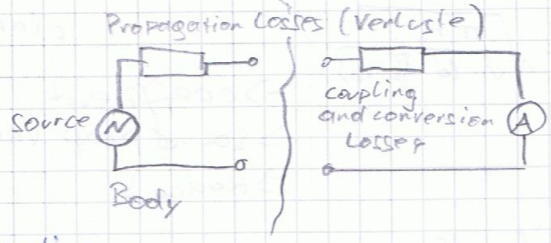
by freiBär

~~freiBär~~ BSS für alle!

Dieses Skriptum beinhaltet hauptsächlich eine ~~A~~bschift der Folien inklusive eine Erklärung der m/M schwierigsten Themen.
Ich erhebe keinen Anspruch auf Vollständigkeit.
Ein paar Folien wurden ausgelassen - ich empfand sie wenig relevant für die Prüfung.
Am besten nach den Folien lernen, und dieses Skriptum zu Rate ziehen.

BSS - Introduction

Biosignal → description of physiological phenomenon
→ irrespective (unabhängig) of the nature of description
→ used in diagnostics and therapy



Classification

Existence → Permanent (EEG | EKG) — Induced (impedance tomogram / Plethysmogram)
(nicht invasive Messung der ↓ Sauerstoffsättigung)

Dynamic → Static (Body temp.) — Dynamic (Heart rate)

Origin of biosignals → Electric (Myogram), Magnetic (Cardiogram)
Acoustic (Heart Sounds), Mechanic (Skin deformation)
Optic (Plethysmogram)

Acoustic Biosignal Example

Heart-sounds

$$P_H = k \cdot \frac{P_0}{r} \cdot e^{-\alpha \cdot r}$$

P_H .. Sound pressure
k → konstant
P_0 .. Power (of Heart)
r .. Radius
α .. Absorbtion coefficient

→ Strong heart sounds → $> p_n$
→ $>r$ → $< p_n$
→ if r high → α is weighted more

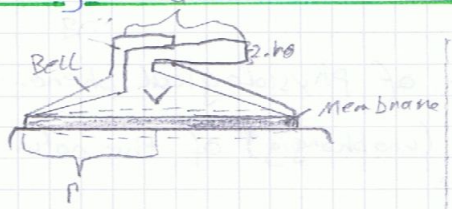
Lounge Sounds

$$P_L = k \cdot \int_V \frac{p(r)}{r} \cdot e^{-\alpha r} \, dV$$

Lounge too big → can't see it as a point
$p(r)$ .. sound power density

↑ Propagation loss →
(tissue)

Coupling and conversation losses



Membrane

$$f_{mn} = \frac{s_{mn}}{2\pi r} \cdot v_M$$

s_{mn} → coefficient

↑ Eigenfrequence

↑ Sound propagation velocity along the diaphragm

→ coefficient
→ sound prop velocity
→ Radius

Bell

$$f_R = \frac{v}{2\pi} \cdot \sqrt{\frac{\pi \cdot r_0^2}{V \cdot (d + \frac{\pi}{2} \cdot r_0)}}$$

v ↓ Sound velocity of air

↑ Resonance frequency

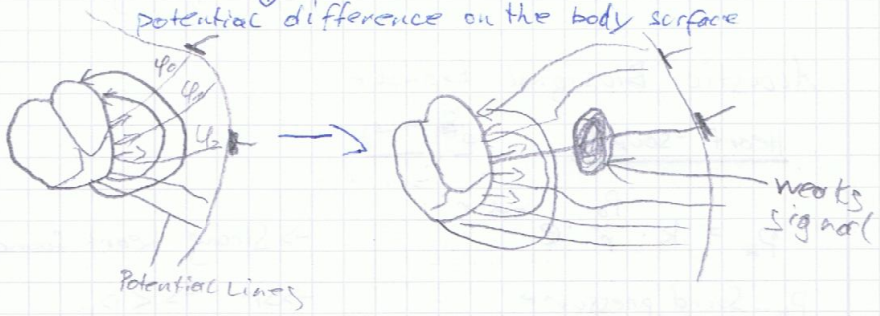
→ Sound velocity of air
→ dimensions of bell

Passive approaches

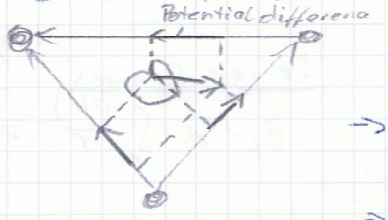
Electrocardiography

→ electrical excitation (Anregung) of heart muscels

↓ potential difference on the body surface



Projection of dipole axis

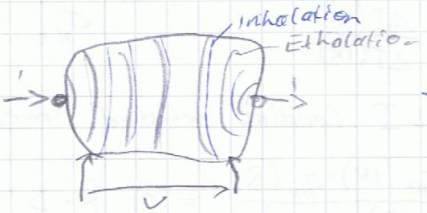


→ Potential difference on body surface
→ projection of total dipole
→ time and space dynamic
→ used for
→ propagation of excitation and back formation PQRST
→ heart position and rate

Electrical field plethysmography (EFPG)

→ Registration of ~~human~~ impedance of human body through applied alternating electrical field

→ Registration of volume and conductivity changes

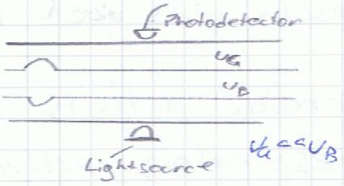


→ Region dependence

→ for cardiac activity
respiratory activity
heart stroke activity, breath volume

→ Optical plethysmography

= Registration of optical transmition of a body part



Absorbtion Law

$$I = I_0 \cdot e^{-\mu \cdot d}$$

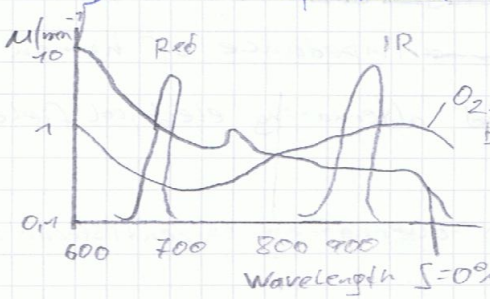
I_0 (Einfallender) incident Light intensity

I_0 gets smaller if μ or d > I - Transmitted Light intensity

μ - Absorbtion coefficient

d - Pathlength of Light

Registration of blood oxigenation S (=oxiometry)



$$\mu = \frac{H}{V}\left(S \cdot \sigma_{HbO} + (1-S)\cdot \sigma_{Hb}\right)$$

↑ Absorbtion

H ... Hematocrit → cellular part of blood

V ... Volume of red blood cell

σ_{HbO} ... Absorbtion area of saturated blood cells

σ_{Hb} ... Absorbtion area of non-saturated blood cells

$$R = \frac{\mu(R)}{\mu(IR)} \approx \frac{\Delta I(R)}{\Delta I(IR)}$$

↑ Ratio

ΔI ... Cardiac related intensity changes

$$\underbrace{S}_{\text{blood oxigenation}} = \frac{R\cdot\sigma_{Hb}(IR) - \sigma_{Hb}(R)}{\sigma_{HbO}(R) - \sigma_{HbO}(R)} = \underbrace{\alpha_1 \cdot R + b}_{\text{constant}}$$

→ Linear Correlation between Ratio $\left(\frac{\mu(R)}{\mu(IR)}\right)$ and ~~Hematokrit~~ blood oxigenation

⇒ gesättigtes Blut lässt rot besser durch, deshalb erscheint es auch roten ♡

Used for: - cardiac activity
- respiratory activity
- oxygen supply

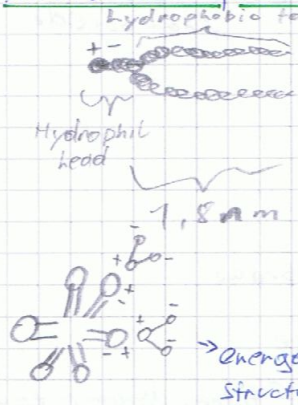
BSS - Physiological Basis

Cellular processes

Membran thickness ~ $8 \cdot 10^{-9}$ m
Cell diameter ~ 1 µm

Length of DNA 2m
→ $3{,}2 \cdot 10^9$ base pairs
$2 \cdot 10^4$ base pairs / gene

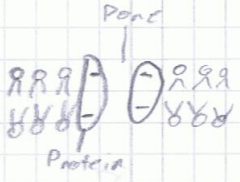
Membrane potential



double lippid layer
→ polar Binding heads - H_2O
→ polar binding heads - heads
→ hydrophobic between tails
→ saturated of stray electric field
„von einem gestreuten elektrischen Feld durchdrungen"

→ energetically optimal structure of lipids in H_2O

Double layer structure is fluid → flip-flop of lipid molecules
→ receptors, enzymes, antigenes and ion channels on outer side
→ Low conductivity
→ high capacity
→ affinity to positive ions (Na^+, K^+)



Channels
→ Pores as channels for material transport
→ passive channels
- diffusion vs electrostatic force

→ active channels
- voltage - controled
- transmitten controlled
- Stress controlled
- temperature controlled

Membrane potential
→ experimental → -70 mV
→ theoretical approach
→ different ion concentration (extracellular / intracellular) → Voltage
→ different membrae permeability for ions

Hier stehen die Annahmen (=Voraussetzungen) für den theoretischen Ansatz
→ die Spannung wird durch unterschiedliche Ionenkonzentr. (intra und extrazellulär) erzeugt
→ zusätzlich werden manche Ionen z.B. K^+ besser durchgelassen als z.B. Na^+

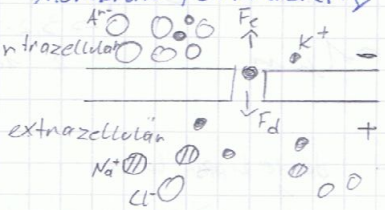
two descriptions / ideas

→ Static equilibrium (Gleichgewicht)

- ignore 2nd constraint

↓

Membran permeability only for K^+



F_d ... diffusion force from insiol

F_e ... electrostatic force from outside

$$F_e = \frac{U}{d_m} \cdot e$$

U .. Membrane potential

d_m - thickness of membrane

e - elementary charge

→ Nernst-equation (because of thermodynamic equilibrium)

~~Thermodynamic~~

$$U = \varphi_i - \varphi_e = \frac{R \cdot T}{F \cdot z} \cdot \ln \frac{[K^+]_e}{[K^+]_i} = 58\,mV \cdot \lg \frac{[K^+]_e}{[K^+]_i} = -83\,mV$$

↑ Potentials

R ... Gas-constant
T ... Temperature
F ... Faraday-constant
z ... number of elementary charges
$[K^+]$ ... ~~co~~Concentration

⚠ -83 mV is nearly -70 mV → Qualitativ right

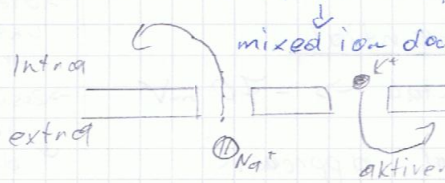
→ it's not exact enough because we have a dynamic equilibrium

→ dynamic-equilibrium

Membrane permeability not only for K^+

→ weak diffusion of Na^+ into cell ⚠

↓ mixed ion double layer



→ not Nernst - equation → Goldmann equation

$U = 58\,mV \cdot \lg \frac{g_{Cl} \cdot [K^+]_e + g_{Na} \cdot [Na^+]_e + \dots}{g_{Cl} [K^+]_i + g_{Na} \cdot [Na^+]_i + \dots}$

$58\,mV = \frac{R \cdot T}{F \cdot z}$

$g_K : g_{Na} : g_{Cl} = 40 : 1 : 0$

↓

Relevant Proportion of ions permeability

$U = 58\,mV \cdot \lg \frac{40 \cdot 5 + 150 + 0}{40 \cdot 150 + 9 + 0} = -70\,mV$ ← that is what we measured

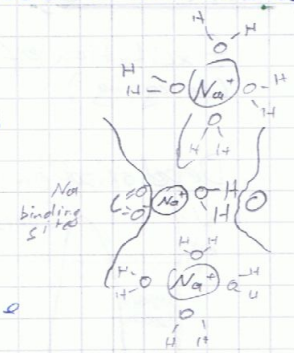
→ Consideration of Na^+ yields lower modulus of U (-70mV vs 83mV)

→ K^+ and Na^+ selectivity of channels

→ H_2O shell (thickness↑ with n&)

→ Specific binding of Na^+

→ K^+ channel - only for small effective diameter (= + shell thickness)



$r_{Na} = 0{,}09\,nm$

$r_K = 0{,}13\,nm$

Auf gut Deutsch

Na^+ sind klein → an Na^+ docken H_2O an → Na^+ sehr groß

K^+ sind groß (im Verhältnis zu Na^+)

↓

K^+ geht nur durch kleine Channels → kann aber nicht durch den Na^+ Channel, weil der selectivist

↓

Na^+ nur durch große, in kleine passts nicht rein

→ So we have a Stationary equilibrium

→ Static U (Voltage)

→ dynamic Ion changes → but they are simultaneous (because of duffesion)

↓

active maintaince of concentration gradient of Na^+ and K^+

Active ion pump - specific active channel

→ Returns K^+ and Na^+ against their concentration gradient by using ATP

→ Active transport and passive diffusion are simultaneous but locally constricted (by different pore-types = unterschiedliche Membranporentypen)

(Brauchen wir, weil ~~sonst~~ sonst der Konzentrationsgradient abnehmen würde)

Nerv-cells

Excitation of Nerve axons (Anregung)

→ Hyperpolarisation $U < -70mV$

→ Depolarisation $U > -70mV$

Depolarisation with $U > -50mV$ (treshold)

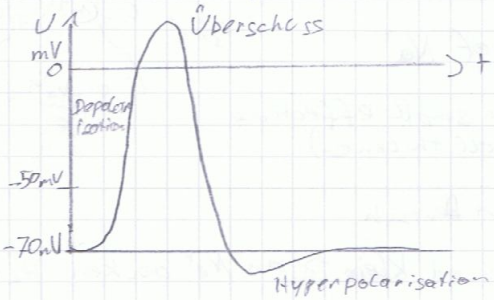
↓

until this treshold the depolarisation decreases on the way

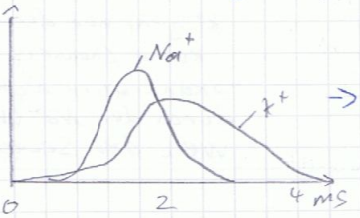
→ Depolarisation $< -50mV$

→ Aktionsimpuls (= Aktionspotential)

→ unabhängig von Weg (Entfernung)



Ions permeability's changes of voltage-controlled channels

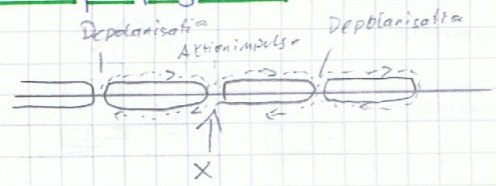


→ Delayed opening

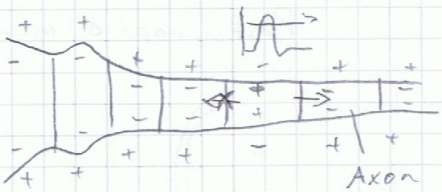
"Überschuss kommt von öffnen der Na^+ Pore und einströmen von Na^+

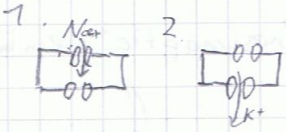
Nochmal ganz einfach: Schwellwert führt zu öffnen der Na^+ Pore, wodurch ein Aktionsimpuls ausgelöst wird. → Pore schließt sich nach 1-2 ms

Axon propagation



1. Excitation on location x → depolarisation
2. This causes a action potential
3. The action potential causes a depolarisation at the neighbours
4. Start again





passive Diffusion um ursprungs-zustand wieder herzustellen

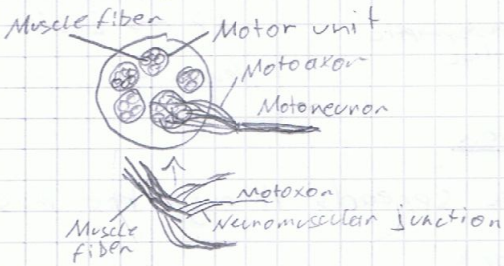
The action impuls propagates only in one direction because a axon has a refractory period of 2ms!

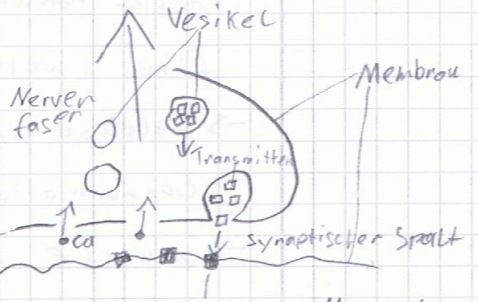
refractory period → temporary inactivation of Na^+ channels

Synaptic propagation

→ From one nerve cell to another (e.g. network)

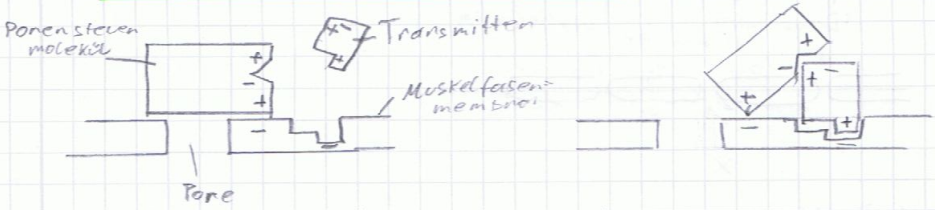
→ From nerve cell to muscle (e.g. Patella reflex)





1. Presynaptic action potential
2. Opening of voltage-controlled Ca^{2+} channels
3. Fusing of neurotransmitter-containing → Vesicles with cell membrane
4. Neurotransmitter diffuses across the synaptic cleft
5. Neurotransmitter binds to receptors
6. Simultaneous opening of transmitter controlled channels for Na^+ and K^+
7. Depolarisation of ~~postsynaptic~~ excitatory postsynaptic potential
8. Equalizing current across the membrane
9. Action potential left/right

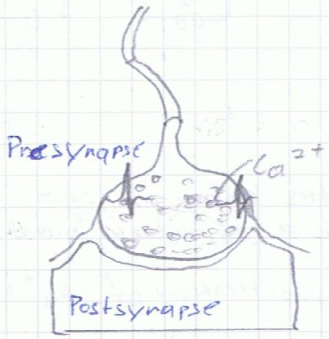
Transmitter controlled channels

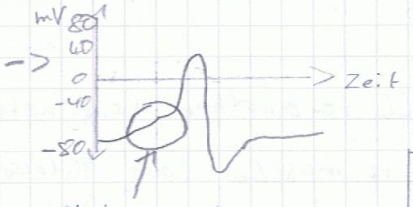


channel closed channel open

→ Complementary conformation and charge between channel control-molecule and transmitter

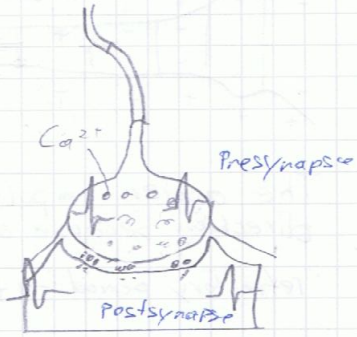
Presynaptic membrane





excitatory postsynaptic potential

Postsynaptic membrane



Postsynaptische Ca^{2+} Signale entstehen durch Ca^{2+}-Einstrom und Ca^{2+} Freisetzung

AP → EPSP → AP

Neuron → Synaptischer Spalt → Muskel

Muscle activation

→ Action Potential spreads through the muscle fiber's network

→ Release of Ca^{+} by voltage controled channels

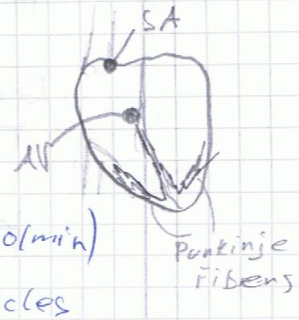
→ Conformation change of myosin and actin

→ muscle contraction

Heart

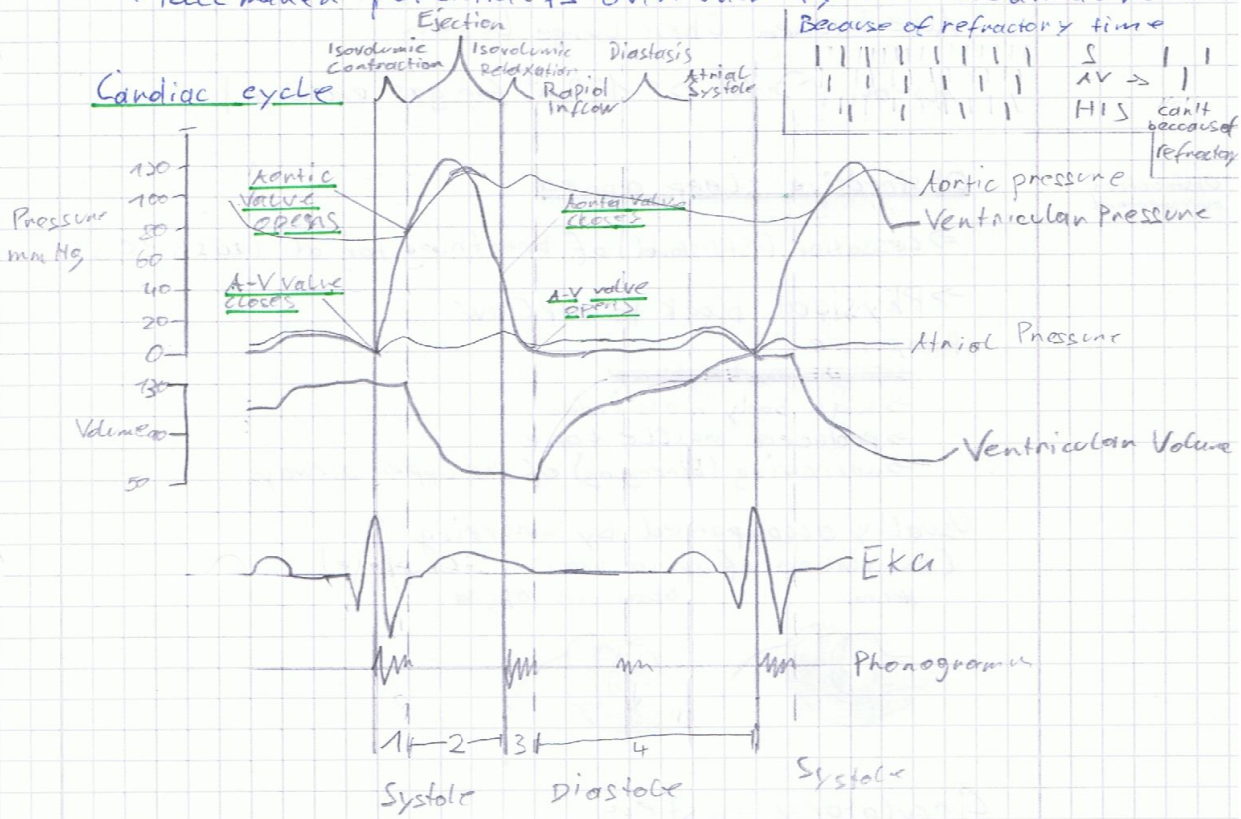
→ alle Sachen über Herz lesen → Anatomie VO sollte aber reichen!

Pacemakers



→ Sinoatrial node (primary ~70/min)
→ Atrio ventricular node (~50/mi)
→ Bundle of His, incl. Purkinje fibers (~30/min)

Atria are electrical isolated from ventricles
→ Pacemaker potential is overridden by the sinoatrial node



Phase 1) Contraction
Phase 2) Ejection
Phase 3) Relaxation
Phase 4) Inflow

Blutdruck = Aortic pressure
→ hier z.B. 120/80
↑ max systole ↖ min Diastole

Contraction + Relaxation → ~~both~~ all valves closed

stiffer valves → high frequence
lower valves → low frequence

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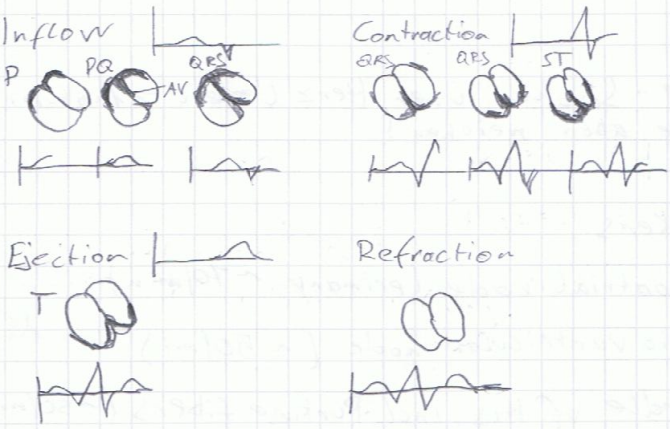


Lung

Alle Sachen über Lunge lesen

Alveoli ($\sim 3 \cdot 10^8 \rightarrow \sim 100 m^2$ for gas exchange)

Obstruktive = versperrend

Obstructive sleep apnea

⇒ cessation (=Stillstand) of breathing for at least 10s

→ Physical block to airflow

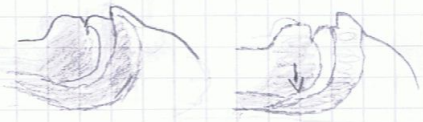
Reasons:

~~→ high muscle tone~~

→ high body mass index
→ reduced muscle tone
→ narrowing (Einengung) of the upper airways

Usually accompanied by snorring
(oscillation of uvula and/or soft plate)

Normal | Obstructiv aprea



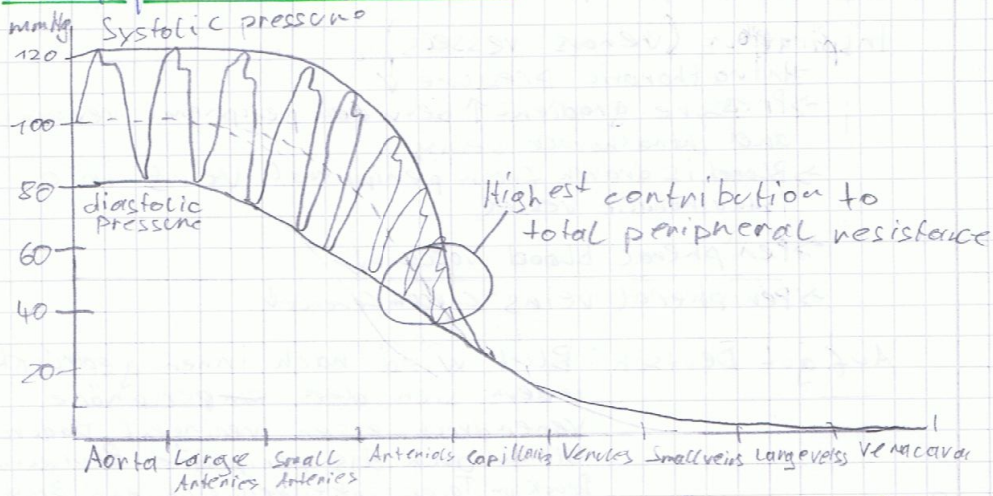
Circulatory system

→ Oxigenated blood
Left ventricel → aorta → arteries → arteriols → capilares

→ Deoxigenated blood
venules → veins → vena cava → right atrium

80% of blood in low pressure systems
veins, right heart and vessels of pulmonary circulation
→ Storage/accumulation
20% high pressure system → blood supply

Blood pressure oscillation



Diameter (mm)	26	8	1	0,02	0,01	0,02	5	16	32
Total cross-section aria (cm^2)	5	20	20	500	3500	2700	100	30	18

p = stroke volume · total peripheral resistance

p ↑ Blood pressure

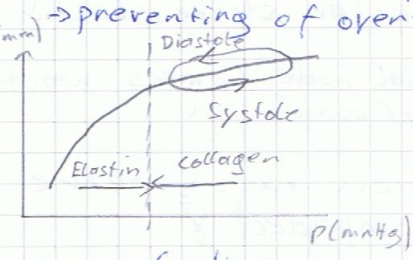
Blood - vessels

- → Stiff collagen
- → compliant elastin (reacting big)
- → Smooth muscle

→ blood pressure controling

→ proximal arteries - elastin dominant
→ distal arteries - collagen dominant

Non Linear relationship between radius r and p
→ preventing of over-distension (Dehnung)

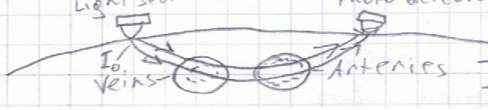


Cardio Mutual Respiratory interrelation
(Beidseitig/gegenseitig) (Wechselbeziehung)

Inspiration: Peripheral venous blood volume and veins circumference (Ausdehnung) ↓

Expiration: → reverse behaviour

Inspiration → Smaller
Expiration → bigger



→ Increase of transmitted Light intensity during Inspiration
Inspiration
Expiration

Respiratory pump

Inspiration (venous vessels)
→ Intrathoratic pressure ↓
→ Pressure gradient ↑ between peripheral venous system and intrathoracic veins
→ Blood is drawn from peripheral veins into intrathoracic vessels
→ Peripheral blood volume ↓
→ Peripheral veins circumference ↓

Auf gut Deutsch: Blut wird nach innen gedrückt indem sich die ~~ver~~peripheren Venendurchmesser verringert. Dadurch sinkt auch das periphäre Blutvolumen. Druck im Torax sinkt, weil sich den Brustkorb hebt

Reverse Thoratic pump

Inspiration (arterial vessels)
→ Intrathoracic pressure ↓, right ventricular strok volume ↑ acording to respiratory pump
→ capacity in the pulmonary vessels ↑
→ Leftward displacement of interventricular septum (Mittelwand dehnt sich nach links aus)
→ Left ventricular stroke volume ↓

Auf gut Deutsch: Volumen von rechten Ventrikeln und pulmonalen Gefäßen erhöht sich → wir wollen ja viel Blut zur Lunge schicke

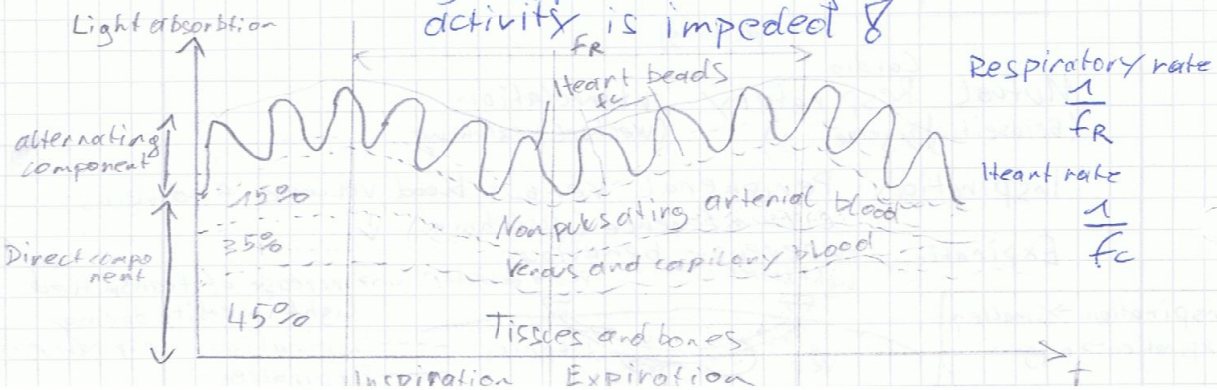
Respiratory Sinus Arrhythmia

Inspiration: Heart rate ↑, diastolic filling time ↓
Left ventricular stroke volume ↓

→ vagus nerve to sinoatrial node lowers heart rate and inhibits the force of contraction

↓↓

During inspiration vagus nerve activity is impeded ↓↓



BSS - Selected Physiological Parameters and their Behaviour

Blood pressure

- Systolic blood pressure P_S
  - → proportional to stroke volume SV (~80 mL) and arterial impedance (e.g. arteriosclerosis limits distention → P_S ↑)
- Diastolic blood pressure P_D
  - → proportional to total peripheral resistance R

$$R = \frac{8 \cdot L \cdot \eta}{\pi \cdot r^4}$$ Allgemeine Formel für den Widerstand in einem Gefäßsystem

$$\Delta P = Q \cdot R \qquad Q = SV \cdot f_c$$

r ... Radius, L ... Length, η ... Viscosity of blood (Zähflüssigkeit)

ΔP ... arterial-venous pressure difference

Q ... Blood flow or cardiac output (~5,6 L/min)

f_c ... Heart rate

→ vasoconstriction (impeded blood flowing, P_D ↑)
→ because of r^4 & 8

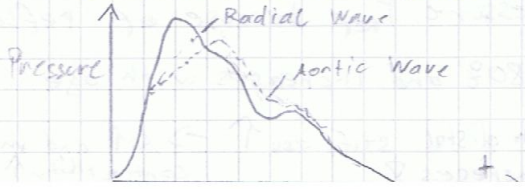
- Mean blood pressure

$$P_M = P_D + \frac{1}{3}(P_S - P_D)$$ approximative

→ P_M < 60 mmHg → ischemic situation

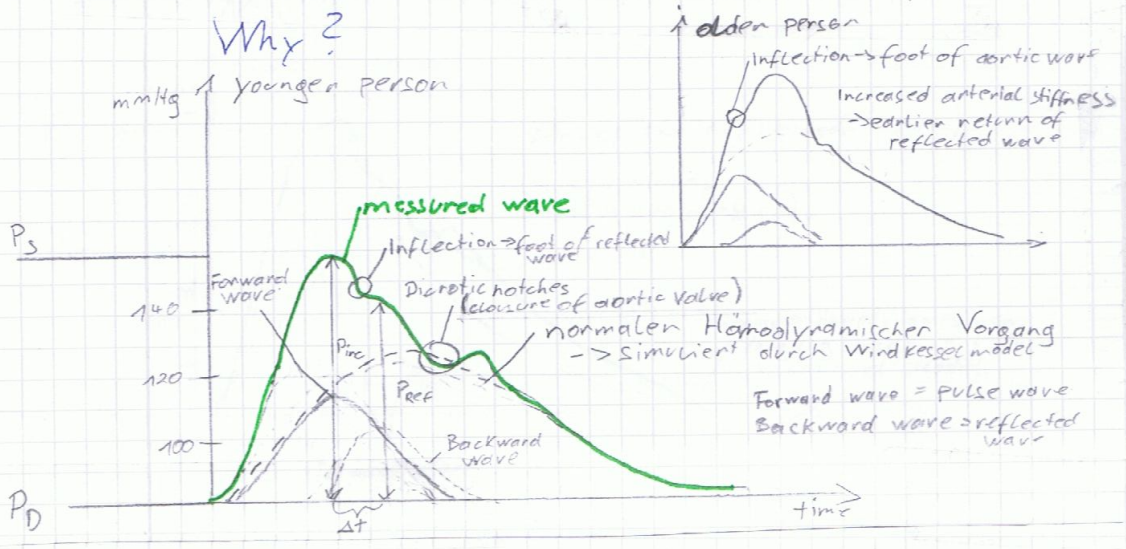
- Local behaviour
  - → Systolic-diastolic blood pressure deflection (Abweichung)
  - → Blood pressure depends on location (stiffness of vessels and pulse-wave reflections) ∇
  - → Aortic wave vs radial wave (Pulse wave)

(measured in aorta) (measured in a. radialis)



→ Erklärung auf nächster Seite

16 → Stiffen vessels Ireduced compliance (Nachgiebigkeit)
→ higher pressure



→ The normal hemodynamic process is simulated by the Windkessel model (= inventierendes Luftgefülltes (oder elastisches) Reservoir dämpft die fluss Pulsation. vgl. Feuerwehrpumpe).

Windkessel Model: $P = P_0 \cdot e^{-\frac{t}{W/C}}$

P_0.. pressure at the start of diastole
C.. Stiffness
W.. Resistance to flow in microcirculation

→ The pulse wave (forward wave) causes a pressure increase (welle in Flüssigkeit → Druckerhöhung)

This wave is reflected by artery bifurcations (Verzweigungen) and peripheral arteries. → backward wave

Hemodynamic process + forward wave + backward wave
→ Systolic Pressure

Augmentation Index $AI = \frac{P_{REF}}{P_{INC}}$ → Pressure at $t_{peak\ of\ reflected\ wave}$ → Pressure at $t_{peak\ of\ forward\ wave}$
= increase of pressure P_{REF} because of reflection

normal AI = 60-80% and increases with age

P_{REF} ↑ and Δt ↓ with distal stiffness ↑ → AI ↑ and myocardial contractility ↑
→ load on heart increases ▽

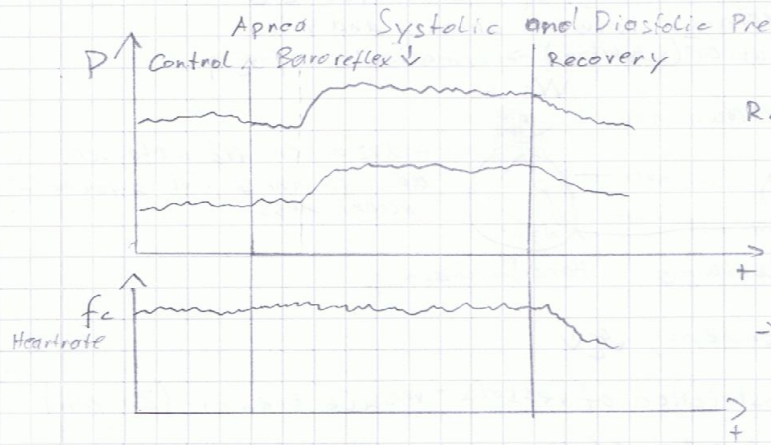
Anmerkung: Vielleicht wundert ihn [illegible] euch, warum die Wendung (Inflection), die ja den Fuß der reflektierten Welle sein soll nicht genau über dem Fuß der reflektierten Welle ist. Ganz einfach: Am Fuß ist die Welle ja noch 0. Deshalb merkt man ihn erst ab einem gewissen Betrag.

- global behaviour
  - Breathing related changes

Inspiration: - Intrathoratic pressure ↓ (weil sich der Brustkorb hebt)
- Right ventricular stroke volume ↑ (damit mehr Blut zur Lunge gepumpt werden kann)
- Leftward displacement of interventricular septum (wenn sich das Septum nach Links verschiebt ist im rechten Ventrikel mehr Platz)
- Left ventricular stroke volume ↓
- P_S ↓ and P_M ↓

Breath hold (=apnea)
hypoxic stimulation of chemoreceptors, reduction of arterial baroreflex ↑ hält den Blutdruck aufrecht

P_S ↑ and P_M ↑



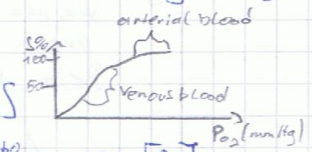
Recovery = ausatmen

→ beim Ausatmen sinkt die Herzrate

Heart rate decreases while respiration
→ respiration → no oxygen → heart rate decreases to save remaining oxygen

Anmerkung bezüglich Baroreflex: Er besteht aus einen NEGATIVE Feedback Schleife d.h. bei hohem Blutdruck senkt er ihn. Wird der Baroreflex also unterdrückt, steigt der Blutdruck

Blood oxygenation



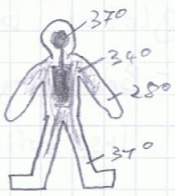
- Hemoglobin oxygen saturation S

$$S = \frac{P_{HbO}}{P_{HbO} + P_{Hb}} \cdot 100 \approx \frac{P_{HbO}}{P_{HbT}} \cdot 100 \; [\%]$$

P is the density of oxyhemoglobin
Hb ... Hemoglobin (Deoxyhemoglobin)
HbO ... Oxyhemoglobin
HbT ... Total Hemoglobin

- Hemoglobin carries O_2 (protein molecule with embedded iron)
- Delivery from plasma to tissue cell by diffusion
  Red blood cell → plasma → tissue cell
- Oxyhemoglobin acts as local oxygen buffer to maintain P_{O_2} (Sauerstoffpartialdruck)

Blood temperature



-Regulatory mechanism

→ core temperature $\vartheta_c = 37°$

→ circadian variation $\pm 0,6°$

$\vartheta_{c\,min}$ at 3 a.m

$\vartheta_{c\,max}$ at 6 p.m

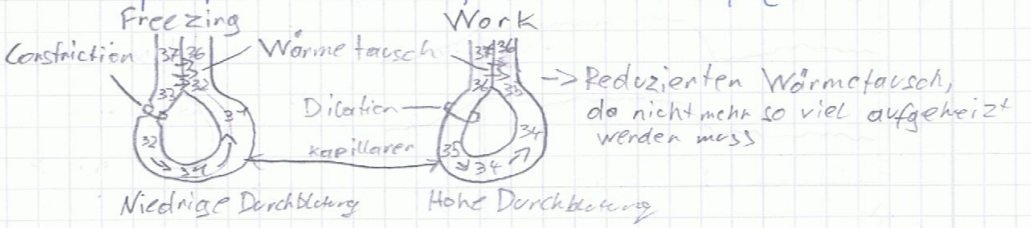
- Readjustment of target value by innen clock

(endogenous CNS rhythm generator
exogenous timer e.g Light)

- Physical work $\vartheta_c \uparrow$

→ dilation (Erweiterung) of vessels → reduced heat exchange between arteries and veins

→ perspiration (schwitzen) → normalization of ϑ_c



- Freezing, tendency $\vartheta_c \downarrow$

→ Vasoconstriction of vessels - muscle tremor (Zittern)

→ normalization of ϑ_c

Erklärung: Bei Arbeit hohe Durchblutung, damit viel Wärme nach außen getragen wird, die dann durch schwitzen abgeführt wird
Beim Frieren umgekehrt

- Feven $\vartheta_c \uparrow$

→ reduced heat dispersion
→ reduced blood circulation in skin
→ vasoconstriction
→ increase heat production due to shivering (Schütteln/Zittern)

Sensation of cold

- Feven $\vartheta_c \downarrow$

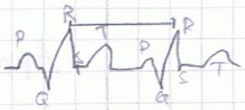
→ increased blood circulation
→ perspiration

Sensation of heat

Sensation = Gefühl

Heart beat

RR-intervall = $\frac{1}{f_c}$



Instantaneous heart rate RR (between R-peaks)

→ controlled by autonomous nervous system

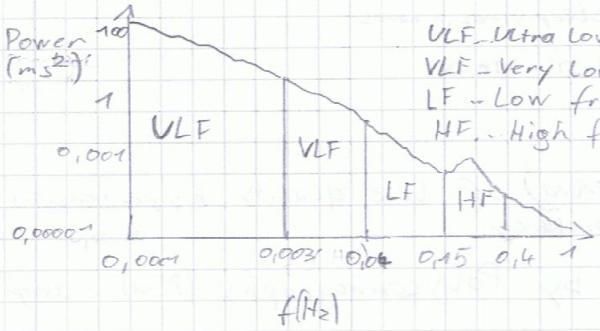
→ Fluctuation of RR → Heart rate variability

SNS = Sympathetic nervous system
fight or flight, hormones and nerves
e.g. f_c↑, inhalation
→ interrelates with LF band of the spectral functions of RR intervall

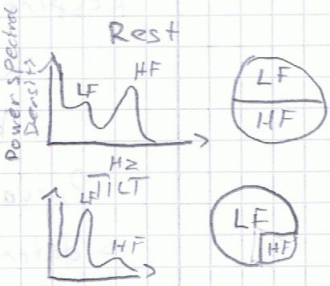
PNS = Parasympathetic nervous system
system break and regeneration, nervus vagus
e.g. fd exhalation
→ interrelates with HF band

→ complementary but NOT ANTAGONISTIC

Heart rate variability



VLF - Ultra low frequency band
VLF - Very low frequency band
LF - Low frequency band
HF - High frequency band



Einfach erklärt: Man macht für ein paar Minuten ein Kardio-tachiogramm. Dann schaut man sich die einzelnen RR-Intervalle an. Liegt die Frequenz zwischen 0,04 und 0,15 Herz → LF Band, 0,15 und 0,4 → HF Band
Die zwei Diagramme rechts zeigen die Dichte der gemessenen Frequenzen. Bei Ruhe hält sich das ganze in den ~~Wage~~ Waage (Fläche unter der Kurve 8), bei Stress hat man 75% LF Band Frequenzen

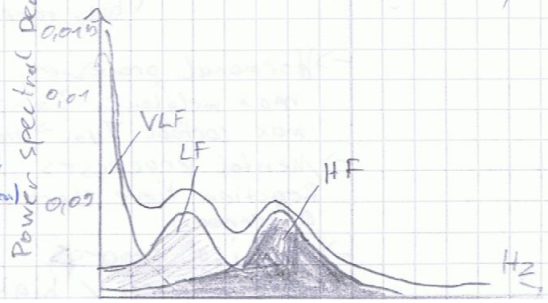
HRV-Index

$$\frac{\text{number of all RR intervals}}{\text{high of histogram of RR intervalls}}$$

(Ich nehme mal an, es ist die Höhe ohne VLF gemeint)

↑ HRV Index → ↑ Survival probability after myocard ~~infarction~~

Histogramm (hier nur für Rest → selbe wie oben, nur genauer)



Respiration

Aphea - cessation of respiration

=> Cessation of breathing for at least 10 s and $\Delta S = -4\%$
-> Abfall der Sauerstoffsättigung

3 Types

- Obstructive (Behindernd) apnea: obstruction of upper airways, loud irregular snoring
- Central apnea: Lacking neural stimulation
- Mixed apnea: CSA and OSA [Central Sleep Apnea / Obstructive sleep Apnea]

Respiratory Disturbance Index

$$RDI = \frac{\text{Sum of apneas}}{\text{total sleep times in hours}}$$

$RDI > 5$ -> disturbance

Prevalence > 1%

-> deterioration (Minderung) of live quality, hypertension decreased Life expectancy

-> usually detected by Polysomnography (PSG) in sleeplab

-> Countermeasures: -> life style
-> medicaments
-> continuous positive airway pressure
-> surgery, appliances

Circadian changes and sleep

- biological rhythms

-> physiological functions reach their minimum in early morning and maximum at late afternoon (blood pressure, heart rate, body temperature)

-> Hormonal processes
max melatonin 3 am -> initiates sleep
max cortisol 7 am -> stress, wake up

-> Mental processes
Reaction time shortest at late afternoon

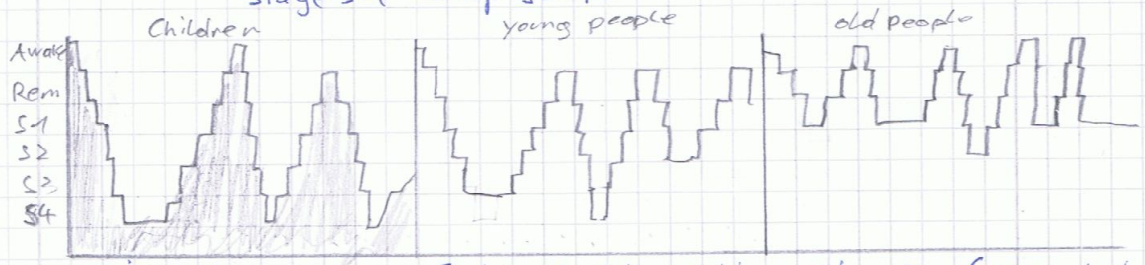
-> Physical process
Work capacity highest 3. am

Seasonal growth, induction	1 year
reproduction	1 month
Sleep-wakefullness	1 day
blood distribution	1 min
peristalsis, blood pressure respiration, heart beat	1 sec
EEG, nerval actions	0,1 sec - 0,001 sec

- Circadian changes
  - extern timers: Light, sensorial inputs, food intak, heat, social field, season
  - internal timers: hormon, body temperature
  - Physiological functions, e.g

→Older people have a lower core temperature

- Sleep
  - → Cycles of ~~rapid~~ eye movements (REM) and NREM phases → classification mainly by EEG
  - → REM - paradoxical sleep, high mental activity and inhibition of muscle movement, DREAMING
  - → NREM - revitalization of the body
    - Stage 1 - beginning of sleep, slow eye movements, partial consciousness
    - Stage 2 - Unconsciousness, awakened easily
    - Stage 3-4 - deep sleep



mainly deep sleep
lot of REM
3-5 sleep cycles

first half of the night
→ deep sleep, second half
→ REM-sleep
3-5 sleep cycles

strongly fragmented sleep, Light sleep

Folien Seite 39 → besser

Sleep is not a passive state → Sleep related changes

→ heart rate and blood pressure ↓ while deep sleep ↑ while REM

→ Respiratory frequency reduced in deep sleep → respiratory volume slightly increased

NREM important for explicite memory tasks (learning vocabulary), REM for implicite (motoric skills, cycling a bycycle)

BSS - Biosignals of Humans

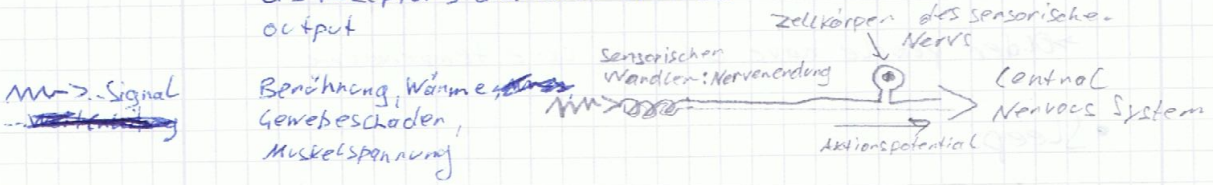
-> Description -> Find in Introduction

Basic principles of sensing

- ~~Primary cells~~ Sensing Organs

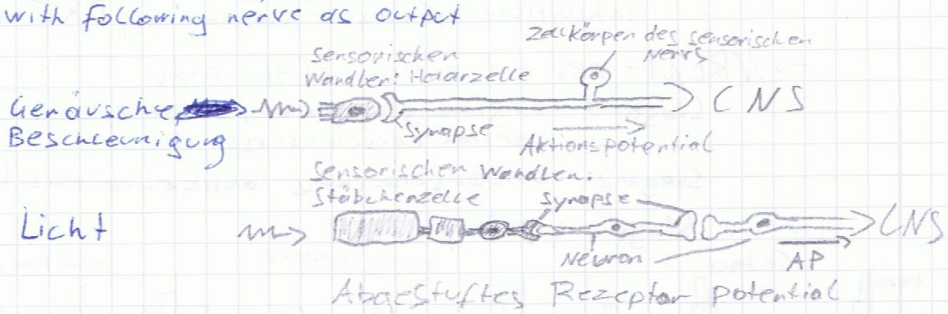
Primary sensing cells

-> bare nerve endings (=dentrites) as receptors and nerve axon as output



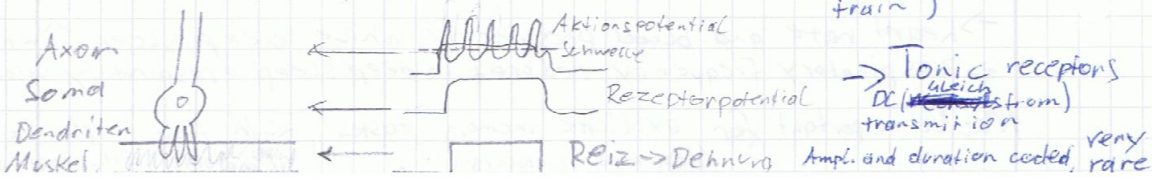
Secondary sensing cells

-> special epithelium cells as receptors and synaptic cleft with following nerve as output



-> Activation energy of secondary sensing cell much lower than energy of membrane potential changes (Verstärken)
  -> Intermediate amplification stages
  -> ultra high sensitivity
  -> ATP energy consumtion
  -> Stimulus of cells only a triggering event!

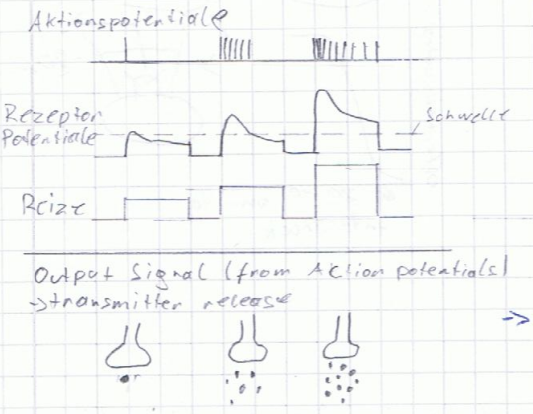
-> conversion of amplitude modulated ~~cells~~ membrane potential into frequency modulated signal (=impulse train)



-> Tonic receptors DC (Gleichstrom) transmission
Ampl. and duration coded, very rare

Tonic-phasic receptors

→ Adaptation (Anpassung), amplitude and ~~de~~ partly duration coded → as long as membrane potential is above treshold

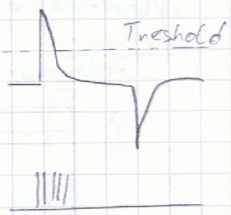


→ jetzt kann man sich denken warum Frequenz-moduliert wird
hohe Frequenz → viele Aktionspotentiale → viele Transmitter

Phasic receptors

→ %AC (wechselstrom) transmission

→ differential behaviour on stimulus slope



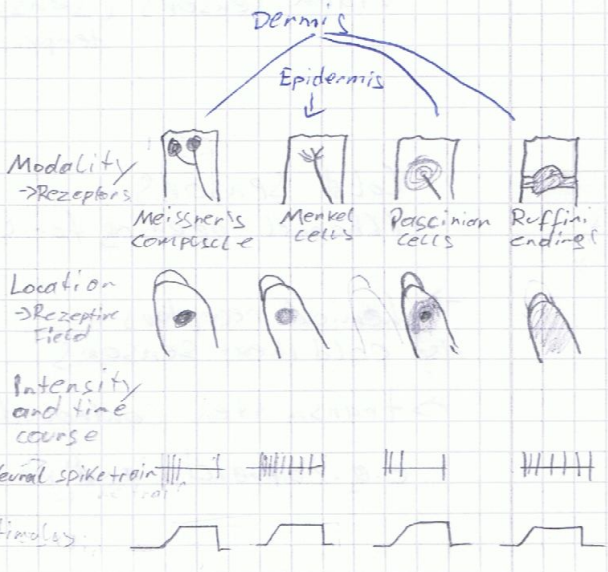
• Humans as sensory creatures

Sensor systems mediate four attributes

- modality (=class of stimulus)
- Location
- Intensity
- timing

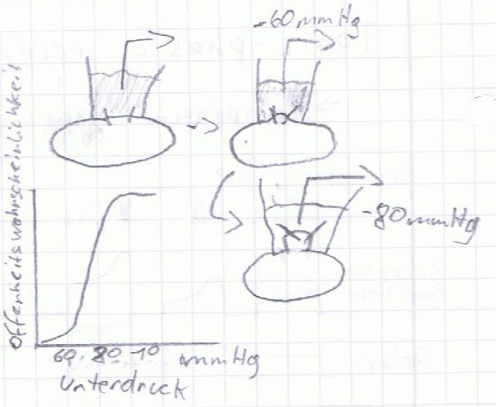
→Touching

- per fingertip 300 receptors
- Mechanical, thermal and chemical receptors
- receptors in dermis and epidermis
- Bare nerve endings as receptor (primary sensing cells)



→ Mechanical receptors

Stress / stretch sensitive ion channels

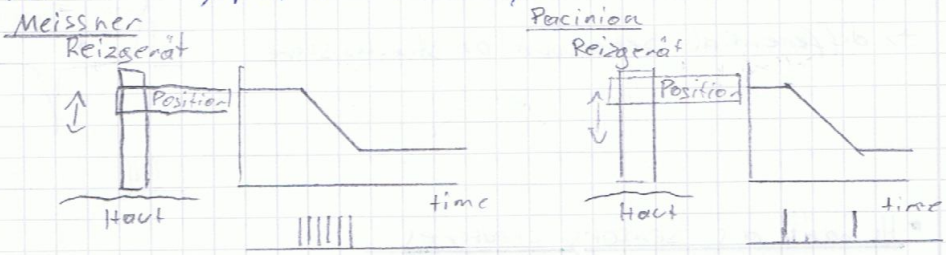


Merkel's disc for steady touch of things
slow → Intensity detector

Ruffini endings for low frequency vibrations and skin sketching
(slow adaption, tonic-phasic)
slow → Intensity detector

Meissner corpuscle for changes in touch stimuli as tops on a keyboard (quick adaption, tonic phasic, 40-150 µm)

Moderately rapid → velocity detector



Pacinian corpuscle for strong vibrations
(phasic receptors, 100-2000 µm)
very rapid → acceleration detector

→ Termal receptors

Warm sensors (sensor opening for highter temperature and capsaicin, component of pepper)
↓
heat perception if in contact

Cold sensors
(channel opening for lower temperature and menthol)
↓
cold perception in contact

→ Chemical receptors
also cold / heat sensors

→ transmitter controled receptors (see Physiological Basis)
e.g. capsaicin and menthol as transmitter molecule

→ Pain perception

Pain is feeling, sensation, protection, illness

→ tissue undendurness or damaged

nociceptors → primary cell (=bare nerve ending as receptors)

→ Tenmo-sensitiv channels

→ Mechano-sensitive channels

→ Acid-sensitive channels
(tissue damage, inflamation, ischemia, pH↓, pit channel open)

Nerve fibres

Aδ-fibres → quick and sharp pain

C-fibres → slow and dull pain

→ sensitivity of nociceptors may increased by inflamation or by duration pain
(chemical sensibilisation of post-Synaptic Channels + multiplication of the channel by additional DNA-reading)

→ temporal ignorance of pain by endorphins/enkephins in brain (adjust the activity of brain neurons processing pain)

Referred pain
⇒ pain from various organs felt in certain skin areas
⇒ convergence of neurons

Phantom pain
in the spine

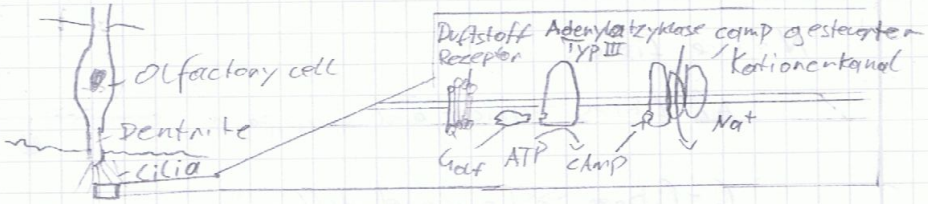
Phantom pain
severed sensory nerves or intermediate spinal nerves continue to send pain

Chronic pain
→ still active neurons ine the spine (Rückrat)

Nociceptors are nearly permanently activated as well as the corresponding inhibitory systems

→ The sense of smell

- → Receptors to smellable chemicals
- → closely allied with taste
- → ~~5~~ $5 \cdot 10^6$ olfactory receptors as neurons
  - → Lifetime 5–6 weeks
- → 10^3 different receptors
- → neurons build a direct Line to the brain
- → primary sensing cells, phasic receptors



- → numerous receptors with low specificity
  (Die meisten receptoren reagieren auf viele Staffe)

→ The sense of taste

Each of the thousands of tastes
is some combination of five primary tastes

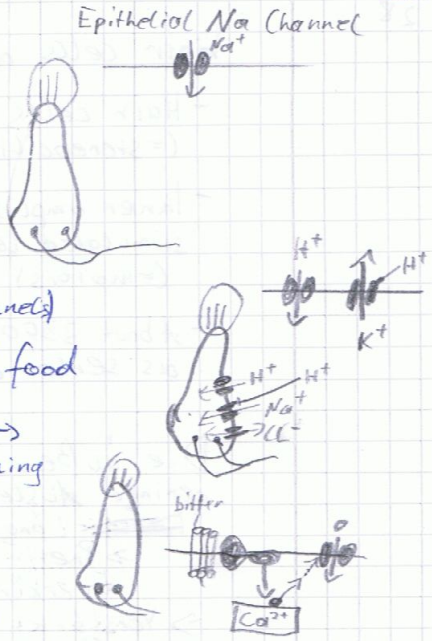
- → sweet, sour, bitter, salty, umami
- → taste has also survival value → many noxious substances taste bitter and spoiled (rotten) or has „off" flavour

Taste → secretion of digestive (verdaungs) enzymes
to break down food into nutrients

- → chemical receptors (of substances dissolved in Salvia (speichel)
- → Regional distribution of receptors
- → but no topographical information !

- → secondary sensing cells

Primary tastes



-> Salty ($NaCl$ -> Depolarisation -> opening of Ca^{2+} channels -> Synaptic propagation)

-> Sour (pH detectors, H^+ channels + blocked K^+ channels + activated Na^+ channels)
-> signal of spoiled (verdorbenes) food

-> bitter (numerous taste receptors -> activation of enzymes -> opening cation and Ca^{2+} channels)
-> signal of potential dangers

-> sweet (similar to bitter receptor)
-> signal to intake of food rich in calories

-> umami (similar to bitter receptor)
-> signal to protein intake

-> Sense of hearing

Basics -> AnatomieVO

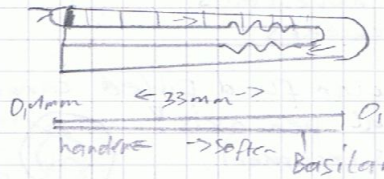
Frequency resolution -> action potential up to 500 Hz -> no direct coding?

Human Frequency resolution 20 - 16.000 Hz

-> Transmission of frequency informations into spatial information

High pitched sounds vibrate basilar membrane at its thinnen end

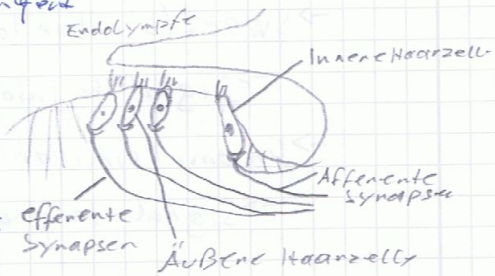
Low pitched sounds vibrate the thicken parts



hair cells as mechanical receptors

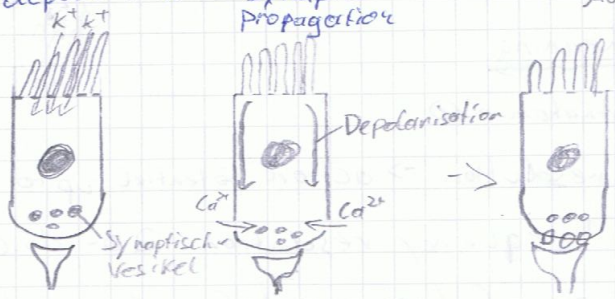
- Hair cells (about 16000) with sensory hairs (= stereocilia, about 100/cell, cell Ø 7 µm)
- Inner amplification of sounds (~ factor 1000) by stimulated length changes (~ 5%) of outer hair cells (= motors)
- About 3500 inner hair cells (= secondary sensing cells) as sensor along the basilar membrane, frequency coding

Die äußeren Haarzellen reagieren schon auf sehr geringe Auslenkungen ihren Haarbündel mit einer ~~Kontraktion~~ Längsänderung ihres gesamten Zellkörpers
→ Protein (Protein)^2-Motoren → potentialabhängig
→ verkürzt oder verlängert
→ Verstärkt
→ erhöht Frequenzselektivität



Innen Haarzell

deflection → opening of K^+ channels
→ depolarisation → Synaptic propagation



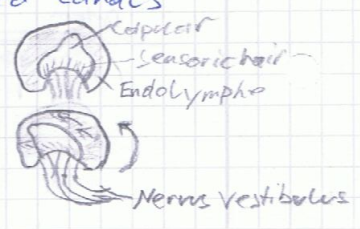
Ohne Auslenkung sind übrigens auch K^+ Ionen kanäle geöffnet (oben nun ein paar) führt zu einen mittelmäßigen Erregung
Haarzellen beschädigt → mehr Kanäle offen → Tinitus!

→ Sence of balance

→ Innen ear, vestibular apparatus (Vorhof + Labyrinth)
→ acceleration sensors (= secondary sensing cells)
For: - Rotation movements
- Linear movements

→ rotation: → 3 semicircular fluid filled canals
→ one canal per axis?

- Canals closed by jelly-like mass (= cupula)
- cupula moves by inertness (Trägheit)
- Sensoric ~~cells~~ hairs (stereocilla) inside



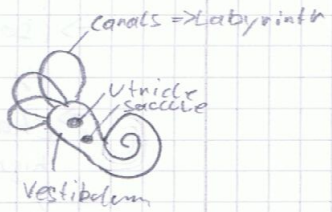
Die 3 halbrunden Kanäle im Querschnitt

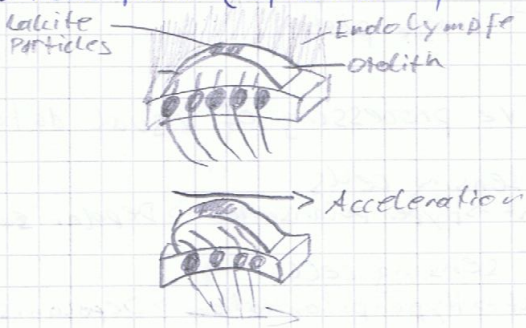
→translational and gravitational acceleration

2 macula organs

→ Utricle for degrees of tilting the head

→ Saccule for movement in vertical plane (up and down)





→Light and vision

- Visible light 300nm-700nm
- Colours because objects reflect some wavelength and absorb others
  - →Black (=total absorbtion) →white (=total reflection)
  - →composite colours

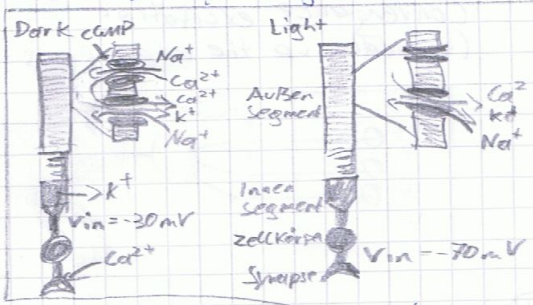
Retina
- Photoreceptors are clustered at the near
- Cones (Zäpfchen), Rods (Stäbchen)

~~7~~ ↓ ~~100~~ $\cdot 10^6$
3 types
→concentrated over forela
→colon
→high resolution
→1:1 connection of receptors and neurons

↓
$120 \cdot 10^6$
→no color
→high sensitive to light
→detects shift in light

forela = free of blood vessels

Photoreceptors
→secondary sensing cells



~~Light~~ Dark: depolarization because of Na^+ inflow trough transmitten (cGMP) controlled channel

Bright: cGMP concentration reduced es, [illegible] transmittion stops

Na^+ inflow eigentlich permanent
→deshalb -30mV
→wenn kein cGMP vorhanden stopt ein Teil des Na^+ einstroms
→ $V_{in} = -70mV$

Light absorbing molecule: rhodopsin

→ isomerization (= rearrangement) of molecule by photon absorbtion

→ chained enzymic reactions with very high amplification

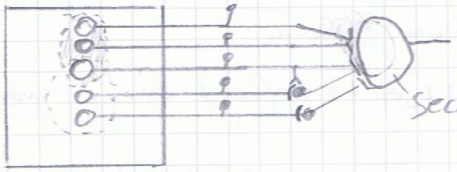
(e.g. 250.000 deactivated cGMP-molecules per single photon)

Excessive processing of visual data

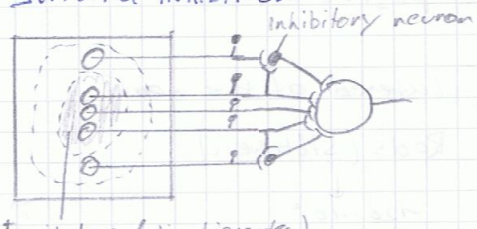
- ON sensing cells
  (Light → Hyperpolarisation → Depolarisation, with synaptic inverter)
- OFF sensing cells
  (Light → Hyperpolarisation → Depolarisation, with synaptic NON-inverter)

→ Spatial (Räumliche) Resolution

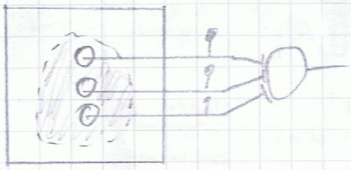
Lateral inhibition to increase contrast



Surround inhibition



Convergence excitation
(by adding up the output of reds



Das ganze dient zur Datenkompression, da es 100 mal mehr Photorezeptoren als Ganglionzellen gibt

→ Es werden gleichfarbige Bereiche zusammengefasst (Convergence E.), Kanten erkannt (Surround inhibiton) und den Kontrast durch Überlappung

-> Other receptors, e.g. osmoreception

Osmosis: water diffuses across the semipermeable membrane in direction of the higher concentrated solution (osmotic pressure >0)

-> Identical concentration of impermeable solutes (e.g. NaCl), osmotic pressure=0

Concentrated solution outside blood cell -> Hypertonic

Equilibrium = Gleichgewicht -> Isotonic

Dilute solution outside -> Hypotonic

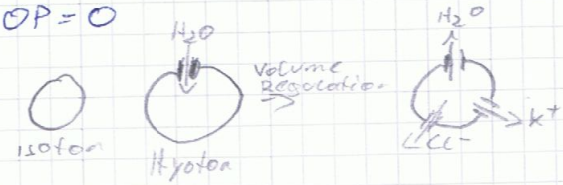
-> Volume and salt concentration regulation (e.g NaCl concentration ≈ 0,9%)

-> Osmotic Pressure >0 -> H_2O flows into cell -> swelling

-> opening of mechanical receptors for K^+ and Cl^-

-> salt concentration in cell decreases -> H_2O inflow stops

-> OP = 0



Limitations

Ultraviolet, magnetic field, high frequency sound, ... Spider senses, X-ray vision, Sensitivity to the force ....

-> Say that to get extra points at the exam ;-)

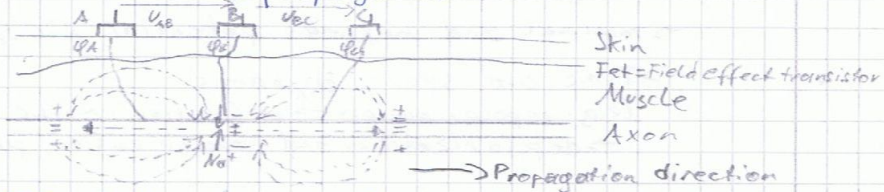
Electric Biosignals

-> ECG (Electrocardiography)

Passive approach!

Generation of electrical biosignals

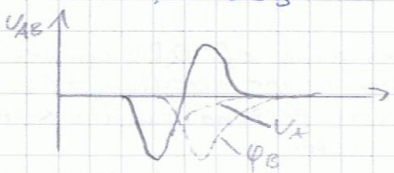
-> Local excitation, equalizing current
-> Quasi-continious propagation



=> Darstellung ~~des~~ einer Messung des Aktionspotentials einen Nervenfaser, φ -> Dämpfung durch Gewebe

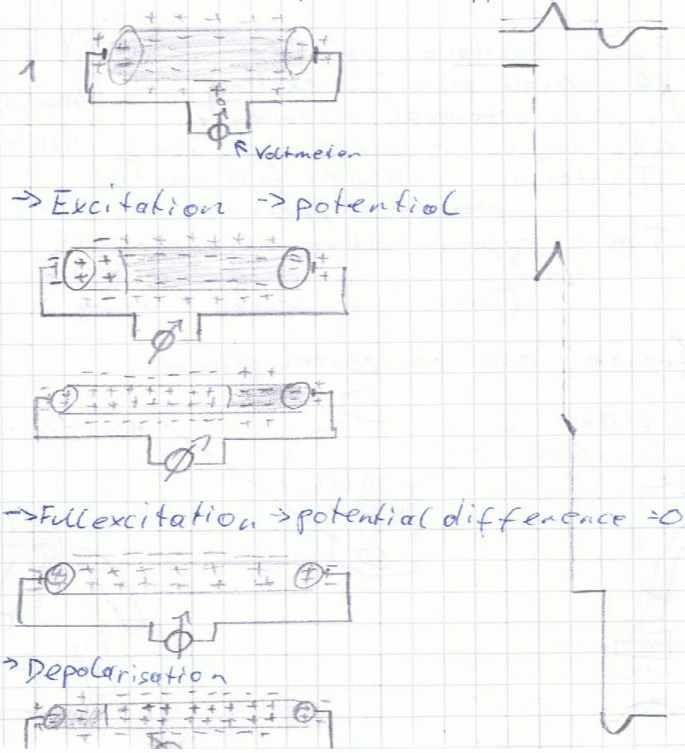
-> Nur eine Propagationsrichtung wegen Refractory period

-> course of biosignals



Depolarization of muscle membrane

-> No excitation -> potential difference = 0



-> Excitation -> potential

-> Full excitation -> potential difference = 0

-> Depolarisation

Depolarization of heart

= Registering of electrical excitation of heart muscle

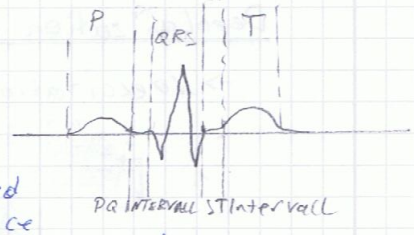
→ potential difference on the body surface

→ see Introduction, page 2

- Origin of ECG
  - → potential differences on the body surface
  - → projection of total dipole on the electrode axis
  - → minimal and maximal potential difference
  - → Time and space dynamic
    (e.g. repolarization → deflection in the same direction
    → depolarization and repolarization pathway differ!)

- electrical axis of the heart
  Defined as mean direction of the total dipole during QRS complex, R peaks are not concurrent in the leads!

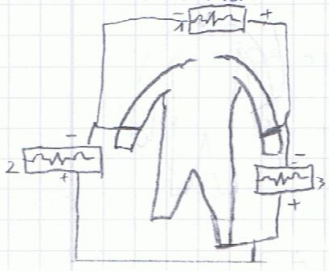
Determination of net area of QRS complex
area > 0 → heart vector in the direction of the Lead
area ≈ 0 → heart vector perpendicular (im Rechten Winkel) to Lead vector
area < 0 → HV opposite to Lead vector



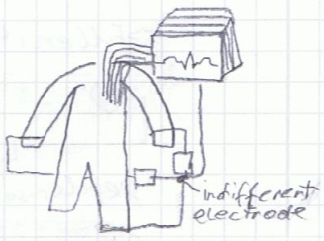
P wave: excitation of atria
PQ intervall: atria full excited
→ no potential difference
QRS complex: propagation of the excitation in ventricles
ST interval: ventricles full excited
→ no potential difference
T wave: repolarisation of venticels
→ deflection in the same direction
(Twelle in den selben Richtung wie R...)

→ frontal and horizontal plane

Frontal plane
→ Einthoven Derivation

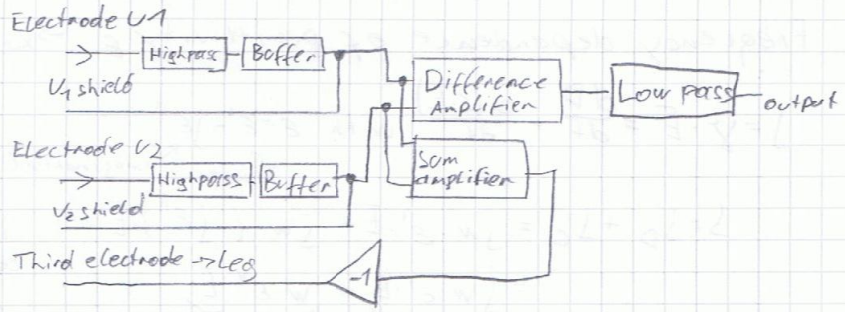


Horizontal-Derevation



- Application issues of ECG

Hardware priciple



Clinical for use

-> Propagation of excitation and back formation

-> Heart position and rate

But not for

->pumping capacity

->mechanical contraction of heart

Normal and pathological ECGs and Advanced ECG signal analysis halte ich nicht für so wichtig

-> Folien seite 9

(Tachycardia, atrial premature contraction, ventricular premature contracten)

Electric field plethysmography

-> Active Approach

Medium impedance as biosignal origin

->Constant electrical field

-> Ωm

- Inhomogeneous distribution of resistivity ρ (local characteristics)
- Resistance R (global Resistance)

ρ ... Rho

$$R = \frac{\rho L}{A}$$

L .. Länge des Leiters

A ... ~~the~~ Querschnitt des Leiters

-> Alternating electrical field

Frequence depending of conductivity γ (= 1/ρ) (Local characteristics)

$$J = \gamma \cdot E$$

J ... current Density (A/m^2)

E .. Electric field (V/m)

$$\frac{dD}{dt} = \frac{d(\varepsilon E)}{dt}$$ with γ

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Frequency dependence of conductivity γ → $(1/\rho)$ → Spule

Frequency dependence of permittivity ε → kondensator

$$J = \gamma \cdot E = \frac{dD}{dt} = \frac{d(\varepsilon \cdot E)}{dt} \quad \text{with} \quad \varepsilon = \varepsilon' - j\varepsilon''$$

(ε' ↑ Realteil, ε'' ↖ Imaginärteil)

$$J = J_D + J_C = j\omega \cdot \varepsilon' \cdot \bar{E} + j\omega \cdot (-j\varepsilon'') \cdot \bar{E}$$

$$= \underbrace{j\omega \cdot \varepsilon' \cdot E}_{\text{Dielectric current density } J_D} + \underbrace{\omega \cdot \varepsilon'' \cdot E}_{\text{Conductance current density } J_C}$$

Increased conductivity $\Delta\gamma = \omega \cdot \varepsilon''$

ω... Frequency

j ... imaginär

Aus den Formeln ist es schwer schlau zu werden.
Grundsätzlich ist das aber so: Verschiedene Materialien (Gewebe haben verschiedene Impedanzen (Wechselstrom widerstände)
Um jetzt die Stromdichte an einem bestimmten Punkt zu berechnen, muss man beachten, dass die Impedanz von der Frequenz abhängt.
Haben Gewebe unterschiedliche Impedanzen sind sie unterschiedlich Leitfähig

elektrische Leitfähigkeit $\gamma = \frac{1}{\rho}$ .. Leitungswiderstand

Die Stromdichte J = elektrische Leitfähigkeit γ · elektrischen Feld E

Die Stromdichte J = elektrische Flussdichte D abgeleitete nach der Zeit
(Flussdichte zu einem Zeitpunkt t = Stromdichte)

Die elektrische Flussdichte D ist wiederum die Durchlässigkeit für Elektrische Felder ε multipliziert mit E

Das bedeutet wiederum, dass die elektrische Leitfähigkeit γ vom selben Faktor abhängt wie die Durchlässigkeit für elektrische Felder, und das ist .... die Frequenz!

Jetzt können wir sagen, dass sich die Stromdichte J aus der Stromdichte ~~für~~ abhängig von der Permeativität J_D (Dielectric current density) und der Stromdichte abhängig von der Leitfähigkeit ist

$$J = J_D + J_C \qquad (J_C = \omega \cdot \varepsilon'' \cdot E = \Delta\gamma \cdot \varepsilon'')$$

Wie ein kondensator — Spule

Oder noch einfacher:

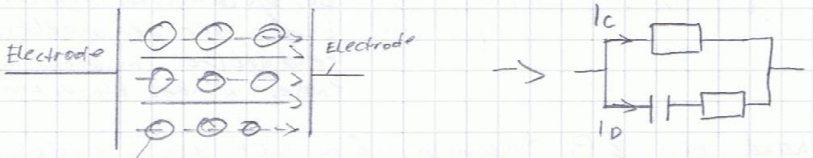
Der Widerstand von Gewebe gegenüber Wechselstrom (Impedanz) ist abhängig von der Frequenz. Diesen Widerstand kann durch 2 Bauteile modelliert werden:

Kondensator $\dashv\vdash$ I_D → Stromdichte abhängig von der Durchlässigkeit von elektrischen Feldern Permeativität ε

Spule $\sim\!\square\!\sim$ I_C → ~~Stromdichte~~ Stromdichte abhängig von der elektrischen Leitfähigkeit Conductivity γ

Verschaltbar

High frequency (impedance $Z\downarrow$, wide cross-area $A\uparrow$)

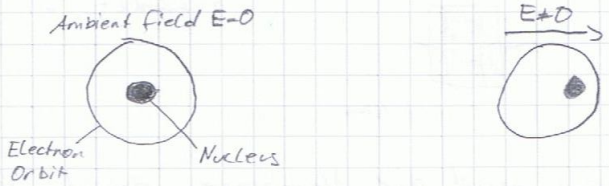


Tightly packed cells — Low frequency (impedance $Z\downarrow$, narrow cross area $A\downarrow$)

→ Dispersion mechanisms:

- Displacement polarization (for $f < 100000$ GHz)

Dispersion ... Abhängigkeit einer Größe (→ Permitivität) von der Wellenlänge oder Frequenz



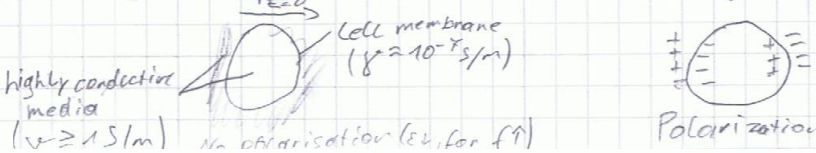
No polarization ($\varepsilon\downarrow$ for $f\uparrow$) — Polarization ($\varepsilon\uparrow$ for $f\downarrow$)

- γ dispersion, Orientation polarization ($f < 30$ GHz, H_2O 10–15 GHz)

→ zelluläre Flüssigkeiten (stark wässriges Gewebe Blut, Muskelgewebe)

No polarization ($\varepsilon\downarrow$ for $f\uparrow$) — Polarization ($\varepsilon\uparrow$ for $f\downarrow$)

- β dispersion, Cell membrane polarization ($f < 100$ MHz)



No polarization ($\varepsilon\downarrow$ for $f\uparrow$) — Polarization ($\varepsilon\uparrow$ for $f\downarrow$)

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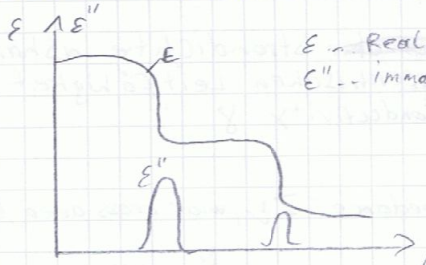
Die Dispersion soll eigentlich nur erklären, warum biologische Medien eine Frequenz abhängige elektrische Leitfähigkeit besitzen. ~~Diese wird~~
Dabei ergibt sich bei steigender Frequenz eine sinkende Permittivität (stufenweise!)



ε' .. Realteil
ε'' .. Imaginärteil

↑ geglättete Stufen

Die relative Permittivität ist übrigens allgemein komplexwertig, da sich bei Dielektrika Polarisationsfelder bilden, der der angelegten äußeren Feldgröße um einen gewissen Phasenwinkel hinterherhinkt!

Anhand der β-Dispersion lässt sich auch sehr schön zeigen, warum wir den Gewebswiderstand mit einem Kondensator modellieren können

Zellmembran → Isolator

„Normaler" Kondensator → wem jetzt nichts auffällt...

Dielektrikum

→ Des weiteren sind die Membranen durch Verschiebungsströme überbrückt und der Stromweg ist durch bessere Leitfähigkeit der Flüssigkeit möglich ~~[illegible]~~

~~[illegible]~~
→ Unterhalb der Gamma Dispersion ist die Membran Kapazität zu groß → ~~[illegible]~~, da keine Gamma Dispersion mehr

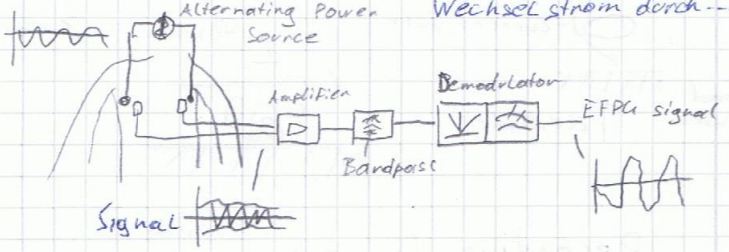
→ Die Kapazität eines Kondensators ist ja Frequenzabhängig
→ Die Spule ergibt sich halt aus der Formel...

→ Modelling of a single dispersion mechanism

→ lassen wir, immerhin waren das jetzt 3½ Seiten ;-)

Methodological issues

Registration of impedance of human body through applied alternating electrical field → wir schicken Wechselstrom durch ...



Signal components

→ Nearly constant component U_0 (>90% of U) due to initial state of medium impedanc

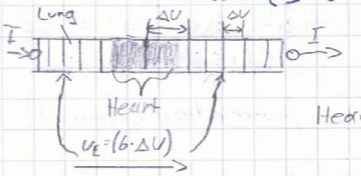
→ Alternating component $\mu_\sim$ (<10% of μ)
- respiratory and cardiac activity
- displacement of organs / Liquids
- blood volume changes and flow velocity changes

Physiological correlates

- Registration of volume and conductivity changes
- Inhalation vs Exhalation (see Introduction Page 3)

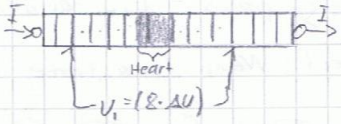
Cardio respiratory activity

Exhalation



Heart = conductiv tissue

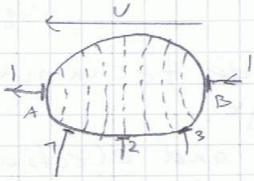
Inhalation ($U_I > U_E$)



- Systole vs Diastole

Late Systole

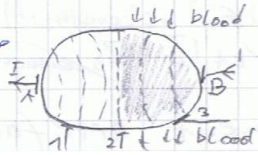
U_S



$U_S (= 2{,}4 \cdot \Delta U)$

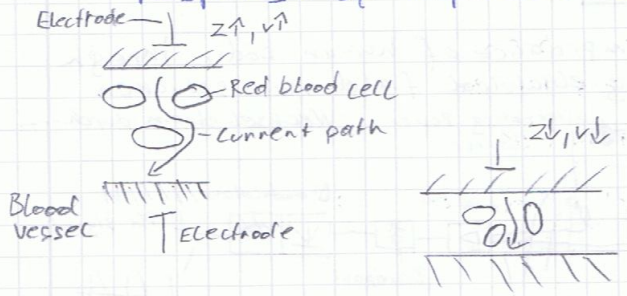
Late diastole

$U_D (< U_S)$



$U_D (= 1{,}8 \cdot \Delta U)$

- Response of impedance Z to flow velocity



V changes
↓
Z changes of <10%

Recording System

- Frequency f
  1) 20-100 kHz → flat course of ε and γ
  2) >20 kHz to avoid stimulation of nerve system
  3) <100 kHz due to dispersion → if f↑↑ then ε, γ of tissue, lung, and blood are identical!
- Current amplitude I
  1) ~1 mA
  2) Probability of neural stimulation increases with increasing of I and decreasing f
  3) Thermal effects increase with increasing I
  4) Signal-to-nose ratio of alternating component (Ansatz)

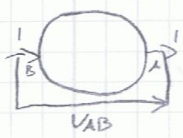
     $u_\sim$ (= information carrier!) increases with ↑I
- Difference Amplifier - Bandpass - Phase-insensitive modulator - High pass - Anti-aliasing filter!

Alias-Effect: Wenn Abtastrate zu gering ist

- Two electrode technique

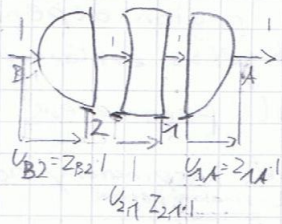
  Disadvantage
  1) Contact and electrode impedance (even if I is constant)

     $Z_{BA} = U_{BA} / I = Z_{B,\text{contact}} + Z_{BA,\text{Tissue}} + Z_{A,\text{contact}}$



  2) Movement artefacts

Four electrode technique



Anmerkung für nicht Elektrotechniker:
→ Durchs Voltmeter fließt kein Strom
→ deshalb gibts auch keine Impedanz der Kontakte, da Strom nur von B zu A fließt
→ dort wird aber nicht gemessen!

Kein Strom zwischen B und 2 bzw A und 2 fließt!

Advantages

1) No influence of $Z_{A,contact}$, $Z_{B,contact}$ (if I constant) ↗ Weil zwischen

2) No influence of $Z_{1,contact}$, $Z_{2,contact}$ and Z_{skin} (if voltmeter is ideal)

$$Z_{21}=U_{21}/I=Z_{1,Tissue}$$

↓ Weil zwischen 1 und Contact bzw 2 und contact kein Strom fließt

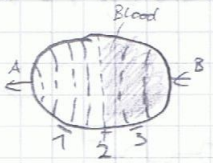
Local measurements

Voltage vs current application

Normal

Voltage Application

$\Delta U_{21} \neq 0$ i.e. "no blood" yields voltage $\Delta U_{32}' \neq 0$

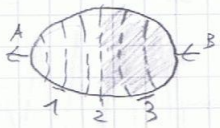


Current Application

→ Allows Local assessment of conductivity changes → higher sensitivity

$\Delta U_{21} = 0$ i.e. "no blood" - no voltage

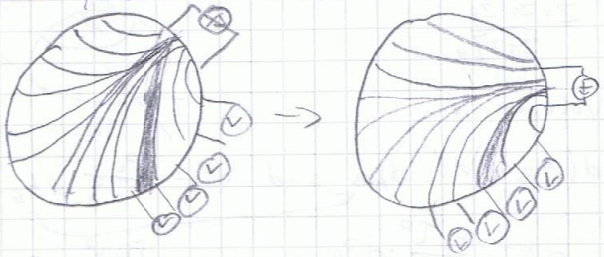
$\Delta U_{32}'' \neq 0$ and $\Delta U_{32}'' > \Delta U_{32}'$



Modified version of EFPG

→ resolution of about 1cm

→ up to 16 frequencies (10kHz – 1 MHz) used to account dispersion, used $I \approx 1mA$

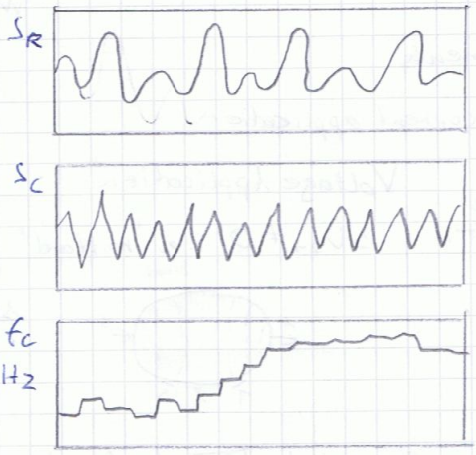


Application issues

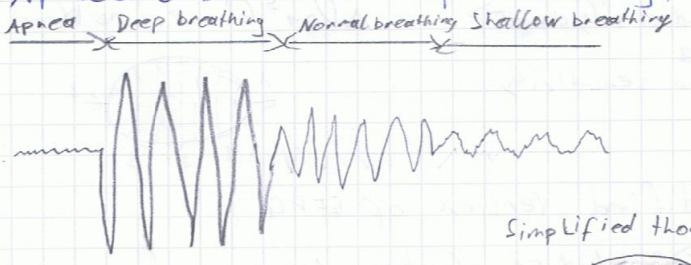
-> Signal and region dependance
  -> Name ist selbst erklärend ...

-> Physiological parameters
  - cardiac and respiratory activity

↓ Signal separation necessary



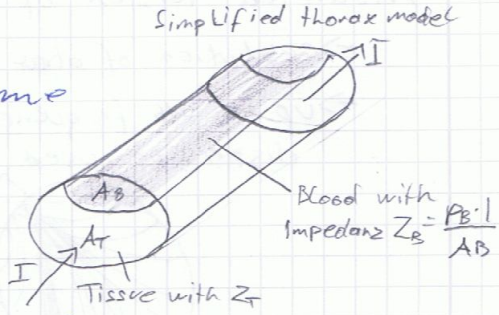
-> Apnea (= secession of breathing)



-> Heart stroke volume

Total Impedance Z

$$Z = \frac{Z_T \cdot Z_B}{Z_T + Z_B}$$



Stroke Volume

$$SV = |\Delta V| = \frac{\rho_B \cdot L^2}{Z^2}$$

Blood Volume V

$$dV = d(A_B \cdot l) = l \cdot dA_B \text{ and } dZ_B = -\frac{\rho_B \cdot l}{A_B^2} dA_B$$

$$dV = -\frac{\rho_B \cdot l^2}{Z_B^2} \cdot dZ_B \text{ or } dV = -\frac{\rho_B \cdot l^2}{Z^2} \cdot dZ$$

Ejection Time

$$\Delta Z = \left|\frac{dZ}{dt}\right| \cdot te$$

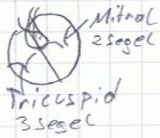
BSS - ACOUSTIC BIOSIGNALS

Generation of Sound

→ Regions of origin, e.g.

- Heart sounds through closing of heart valves
- Lung sounds through air turbulences

→ Heart sounds



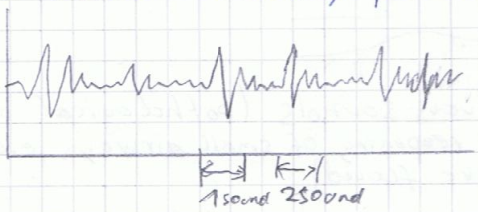
1. sound → closure of mitral and tricuspid valves
(tension of valves, deceleration of blood)
(Spannung) (Bremsung)

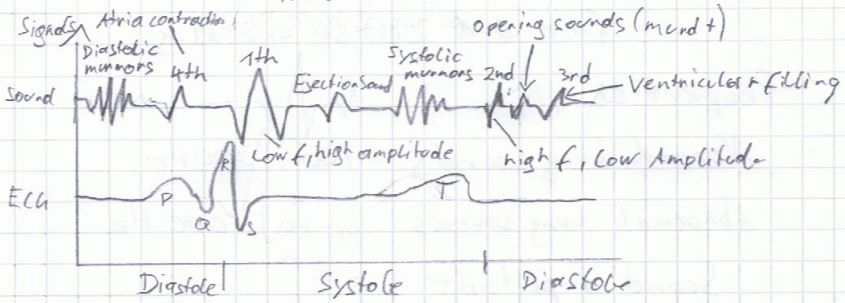
Loudest, longest (140 ms), low frequencies

2. sound → closure of more rigid aortic and pulmonic valves

Lower intensity, snapping quality, duration about 110 ms

Other sounds → usualy pathologic, e.g. opening of valves

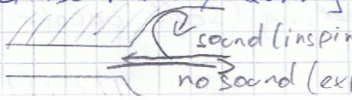




→ Lung sounds

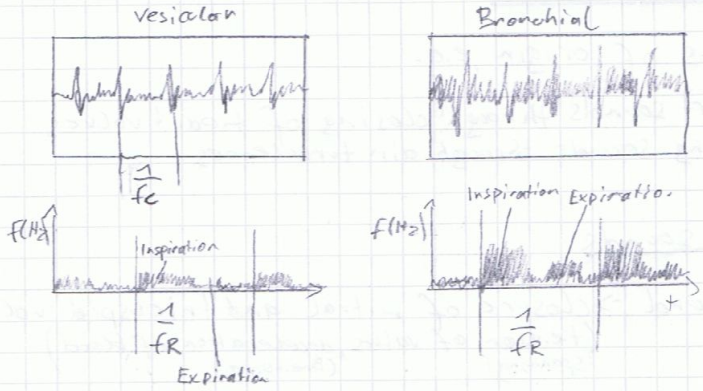
Classification based on

- Location of auscultation (Abhören) region
  - ~~Vesicular~~ sounds
    (at peripheral lung fields, air turbulences arise mainly during inspiration, distributed sources)



- Bronchial sounds

(over large airways, e.g on the neck, turbulent airflow induce vibration of airway walls, central source)



- Sound types

  - Continous sounds (normal and pathological)
    (narrowing and constrictions of airways)
    i.e. wheezes, rhonchi etc
    keuchen/pfeifen

  - Discontinous sounds (pathological only)
    (explosive reopening of small airways, e.g. because of excesive flood)

    i.e. ..fine and course crackles

- Properties

  Normal lung sounds: 100 .. 500 Hz

  Abnormal lung sounds: up to 1000 Hz

  Sound amplitude $S \sim F^n$
  F.. airflow, $n \geq 2$

→ Snoring sounds

Classification based on

- Location of origination (region)

  - Nasal snoring (uvula oscillation)
  - Oral snoring (soft palate oscillation)

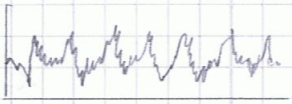
- Type of generation
  - Normal Snoring
    (flow limitation, narrowing of airways, during inspiration, reduced tone of muscles due to stress, alcohol)
  - Obstructive snoring
    (narrowing and temporal occlusion of airways due high compliance of airway walls and masses obstruction airways)
    → Apnea

Normal snoring

Obstructive snoring

- Distinct signal waveform
  - Simple waveform snoring
    (sinusoidal waveform, oscillation without closure)

  - Complex waveform snoring
    (trainstructure, temporal colliding of airways)



- Properties

  Normal snoring = 100 - 800 Hz

  Obstructive = up to 2000 Hz

Transmission of sounds

→ Propagation

→ concentrated sound sources (e.g. heart)
→ distributed sound sources (e.g. lung lobes)

→ Sound waves

- Interrelation of time and frequency domain

$$\lambda = \frac{v}{f}$$

λ .. wavelength
v .. sound velocity
f .. sound frequency

- Determination of v by propagation medium

$$v = \sqrt{\frac{K}{\rho}} = \sqrt{\frac{1}{\rho \cdot D}}$$

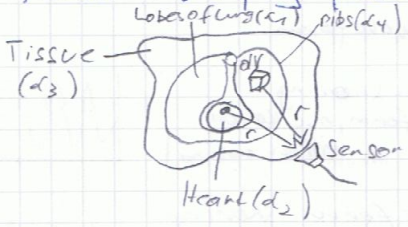
K module of volume elasticity (Pa)
ρ .. density, D .. compliance $= \frac{1}{K}$

→ Pathway depends on f, thus v depends of $f(\frac{P}{P_0})$

- Low frequency (<300Hz)
  → coupling from airways into mediastinum (parenchyma, airways as non-rigid tubes, absorbing sound energy
- High frequency - sound remain within tubes/airways traveling into branching structure

→ Attenuation (Abschwächung)

- Concentrated / distributed sound sources
- Geometrical damping factor $\frac{1}{r}$ (= inverse square law)
- Medium damping factor $e^{-\alpha \cdot r}$



$\alpha_1 \neq \alpha_2 \neq \alpha_3 \neq \alpha_4$

Heart sounds

$$p_H = k \cdot \frac{P_0}{r} \cdot e^{-\alpha \cdot r}$$

p.. Sound pressure
P_0.. Power
α.. Absorption coefficient
r.. Radius, k.. constant

Lung sounds

$$p_L = k \int_V \frac{\rho(r)}{r} \cdot e^{-\alpha \cdot r} dV$$

ρ Sound power density
V Volume

- Volume effects
  - Inner friction (Reibung / Spannung)
    (due to differences in local sound particle velocities)
    $\alpha_F \simeq$ medium viscosity, $a_F \simeq f^2$
  - Thermal conduction
    (propagation ~~[illegible]~~ linked with local variations of temperature, balancing of which withdraws energy from sound wave)
    $\alpha_T \simeq$ difference of specific heat capacities at const. pressure and volume $\alpha_T = f^2$

- Molecular relaxion
(vibration of molecules starts with some delay at the expanse of rotational energy (of atoms) and translational energies (=gas pressure) | delayed setting yielding energy losses)

α_M = difference of sound ~~energy~~ velocities before v_0 and after v_∞ ($>v_0$) relaxation
maximal α_M at relaxation frequency, in human acoustical range $\alpha_M \sim f^2$

$\alpha = \alpha_F + \alpha_T + \alpha_M$

Absorbtion = innere Reibung + lokale Temperatur Schwankungen + "Übergang der Moleküle in Grundzustand"

↓ medium Viscosity

↓ difference in local heat capacities

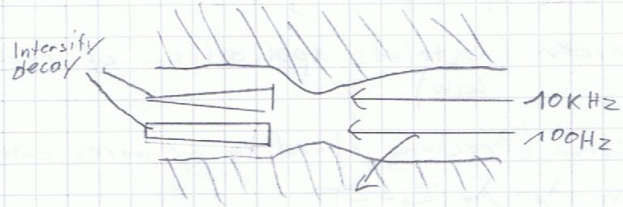
↓ difference in sound velocities before and after relaxation

Gewebe nimmt Energie von den Schallwellen auf -> wird in Termdenergie umgewandelt
-> dabei kommt es natürlich darauf an, wieviel Wärme das Gewebe aufnehmen kann -> kapazität

Durch die Rotationsenergie und Gasdruck beginnen die Moleküle mit einen Verzögerung zu Schwingen -> Energieverlust

-> Propagation and attenuation in short

- Highly inhomogeneous propagation medium
- Frequency dependant sound pathway
- Frequency dependant attenuation

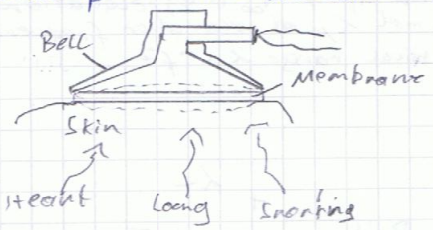


->Bei Hohen Frequenzen
->hohe Dämpfung
-> Bleibt in Luftwegen
->tiefe Frequenzen
->geringe Dämpfung
->Frequenz wandert durch das Gewebe

47 **Coupling and recording of sounds**

→ in short

- Coupling of sounds
- Chestpiece as acoustic amplifier
- Microphone as acoustic - electric converter



→ Coupling

- Inhomogenity effects
  - Reflection (tissue - air interface)

$$R = \frac{Z_A - Z_T}{Z_A + Z_T}$$

R .. Reflection coefficient
Z (= p·v) Sound radiation impedance
Z_A - Air
Z_T .. Tissue

Tissue $Z_T \approx 1{,}4 \cdot 10^6\ kg\,m^{-2}\,s^{-1}$

Air $Z_A \approx 340\ kg\,m^{-2}\,s^{-1}$

→ $R \sim 0{,}998$ → More than 99% of incident sound pressure p is reflected

Limitation of this simplified approach

1) Skin is true multilayer (higher transmittion rate since $|Z_2 - Z_1| < |Z_A - Z_T|$)

2) λ and size of reflecting objects are in the same order (restricts application of reflection law)

- Refraction (Brechung, = bending of waves entering air)

→ $v_A < v_T$, $\lambda_A < \lambda_T$, $Z_A < Z_T$

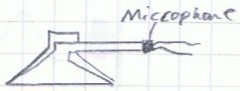
$\frac{v_A}{v_T} = \frac{\sin(\beta_A)}{\sin(\beta_T)}$ β .. refracting angle → flattened wave form

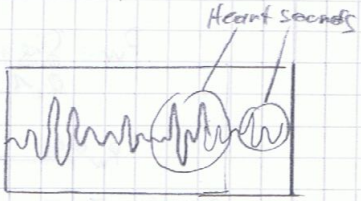
- Membrane and bell

Introduction, page 2

-> Recording

- Microphone as converter

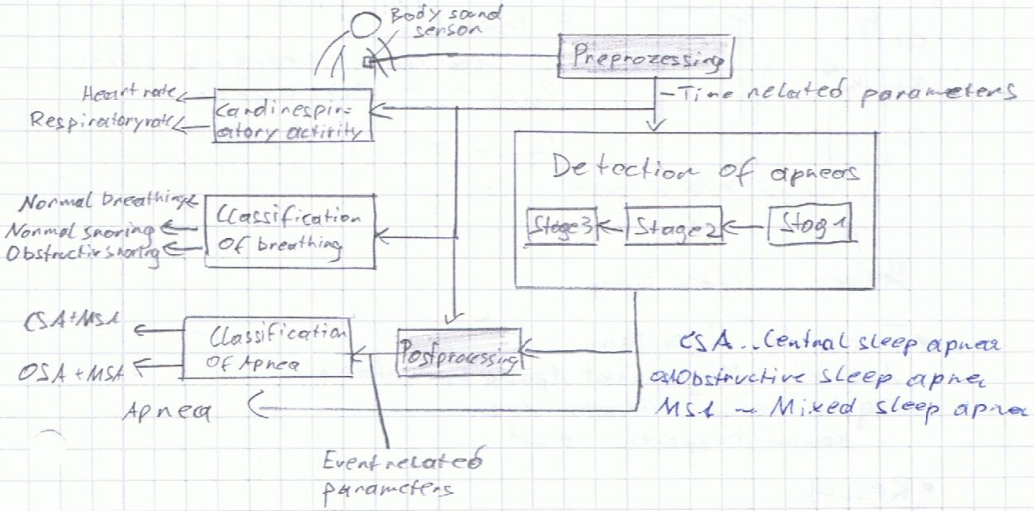




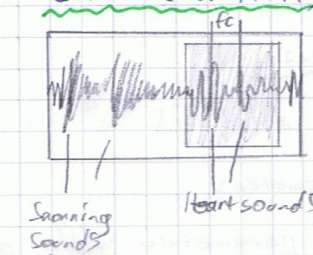
- Spatial distribution of sounds
  -> wuascht...

Application issues

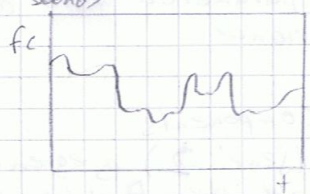
-> Multiparametric monitoring

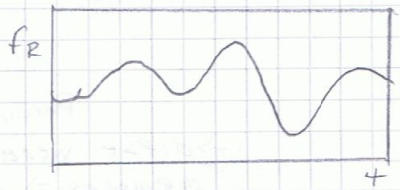


- cardiac activity



- respiratory activity

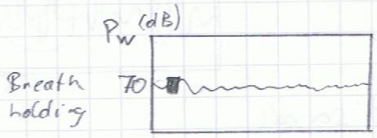


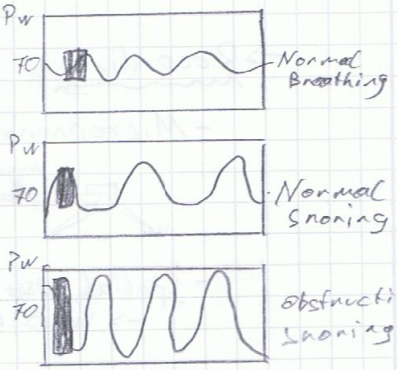


- classification of breathing by

- Amplitude of P_w deflection
- Power ratios

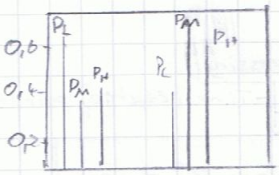
P_w .. Signal power in range 0,1 .. 2 kHz





- detection of apneas

- Assessment of apneical properties



P .. Probability
P_L – Signal Power in range 0 .. 0,3 kHz
P_M – " – 0,3 .. 0,8 kHz
P_L .. – " – 0,8 .. 2 kHz

No increase | >6% increase

↑ Before | ↓ After apnea

- Treshold Algorithm
  Auch eine Möglichkeit Apnea zu entdecken
  → gibt keine literaten dazu
  → kommt hoffentlich nicht
- Result
  .. auch keinen Plan

- classification of apneas

→ Principle Component Analysis

21 event based parameters

→ PCA → 21 parameter with descending variance

↓

Pricipal Components

→ diese werden (jeweils 2) gegeneinander geplotet → anhand der Dichte kann man jetzt den Typ abschätzen (SA, OSA, MSA ~

BSS - Optic Biosignals

Light interaction with tissue

-> Light absorbtion

Absorbtion law

$I = I_0 \cdot e^{-\mu_a \cdot d}$

I_0 .. Incident light intensity

I .. Transmitted light intensity

d .. Pathlength of light

Absorbtion coefficient

$\mu_a = \sigma \cdot \rho$

σ .. Absorbtion cross section

ρ .. Number density chromophores

Chromophor = den Teil des Farbstoffes, der für das Vorhandensein der Farbe sorgt

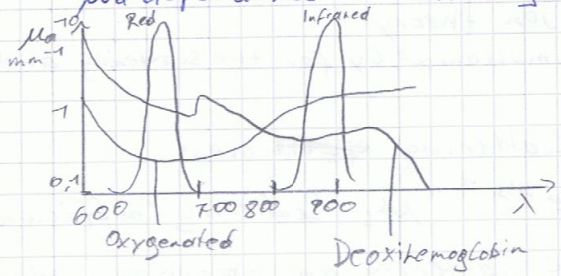
μ_a reciprocal (Gegengesetzt) to d

$e^{-\mu_a d}$ -> probability of survival of the photon after d

μ_a -> key parameter for spectrometry (e.g. blood oxygenation)

d -> key parameter for plethysmography (e.g pulsatile blood volume changes)

μ_a dependence on wavelength λ of hemoglobin



-> Sauerstoffreiches Blut besonders hell

-> Sauerstoffarmes -> dunkel

Sauerstoffreiches Blut ist deshalb so hell, weil es rot nicht absorbiert, sondern [redacted] durchlässt

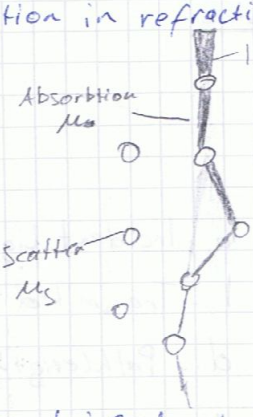
$$S = \frac{\rho_{HbO}}{\rho_{HbO} + \rho_{Hb}} \cdot 100[\%]$$

ρ_{HbO} .. Density of Oxihemoglobin

ρ_{Hb} .. Density of Hemoglobin

5.1 → Light scattering

- Disperson of light ~~effect~~ due to chaotic variation in refractive index



→ Increased pathlength of photons → increased probability of absorbtion
→ By far the most dominant tissue-photon than the absorbtion

- relevant if structure sizes matches λ of the photon (range 600-1000nm)
- Scattering biological structures
  - Lipid water interface of membranes (9nm) → size < λ, Rayleigh isotropic scattering
  - Water-protein periodicity of collagen fibrils (70nm)
  - scattering magnitud scales with $\left(\frac{r}{\lambda}\right)^4$
  - protein aggregates, mitochondrion (1µm) → size ≥ λ Mie-anistropic scattering
  - Collagen fibre bundle (3µm)
  - Scattering magnitude scales with $\frac{r}{\lambda}$
- Photon diffusion theory (to account simultaniously for the scattering and absorbtion effects)
  - Single scattering ~~effect~~ event

    $I = I_0 \cdot e^{-\mu_s \cdot d}$ μ_s.. Scattering coefficient
  - Multiple scattering and absorbing media ($\mu_s > \mu_a$ and low anistropy)

Anistropic → Richtungsabhängigkeit d.H. Licht geht nur in eine Richtung wie z.B. bei einem Laser. Sonnenlicht wäre isotrop

$$\frac{I}{I_0} = K \cdot \frac{e^{-d\alpha}}{4 \cdot \pi \cdot D \cdot d}$$

α.. Total attenuation coefficient
D.. Diffusion length (m)
μ_s'.. Reduced scattering coefficient
g.. anistropy coefficient K.. konstante

$$D = \frac{1}{3 \cdot (\mu_a + \mu_s')}$$

$$\alpha = \sqrt{3 \cdot \mu_a \cdot (\mu_a + \mu_s')}$$

$$\mu_s' = \mu_s \cdot (1 - g)$$

Vermutung...
$K' = K \cdot \frac{1}{4 \cdot \pi \cdot D \cdot d}$
→ wird auf nächsten Seite gebraucht

- μ_s' describes effective isotropic scattering
- g describes asymetry of scattering
  - Forward scattering $g=1$
  - Isotropic scattering $g=0$
  - Backward scattering $g=-1$

$$\mu_s' = \mu_s \cdot (1-g)$$

$g = \cos(\theta)$

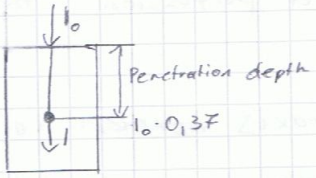
- biological tissues
  - $\mu_s' >> \mu_a$ i.e. relatively low μ_a → deep penetration within the tissue
  - $g > 0{,}7$ i.e. collimated light becomes diffuse after a few millimetes

→ Penetration and probing
(Durchdringen) (forschen/sondieren)

~~Please~~ → Both describe depths to which the light penetrates the tissue

- Penetration depth
  - Depth at which I_0 has fallen by $\frac{1}{e}$ (~63%) → $-\mu_a \cdot d = -1$
    → $d = \frac{1}{\mu_a}$
  - Given by $\frac{1}{\mu_a}$ or $\frac{1}{\alpha}$
  - Key issue for transmittion of light!

Single scatter $I = I_0 \cdot e^{-\mu \cdot d}$
Multiple scatter $I = I_0 \cdot k' \cdot e^{-d \cdot \alpha}$



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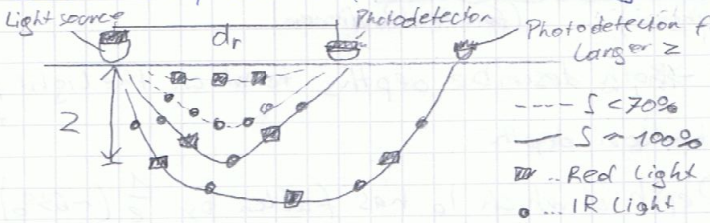
→ Probing depth

- Depth z at which the changes in optical properties have sensitivity lower than 5% of I measured by the photo detector
- Given by source-photodetector separation distance d_r and μ_a, μ_s' $\quad z \approx \frac{0{,}4 \cdot d_r^{\frac{3}{2}}}{(\mu_a \mu_s')^{\frac{1}{2}}}$

Auf gut Deutsch: Probing depth ist die Tiefe, bei der noch 5% aller Photonen als richtig detektiert werden, die von der Quelle kommen (Sensitivität) 95% aller Photonen, die von der Quelle kommen werden ~~nicht~~ falsch detektiert. Das heißt ~~[illegible]~~, wenn man weiß, um welches Gewebe es sich handelt (μ_a, μ_s') und den Photodetektor dort ansetzt, wo die Intensität nur mehr 5% von I_0 beträgt, kann man sich ausrechnen, wie tief das Licht ins Gewebe ~~ein~~ eindringt.



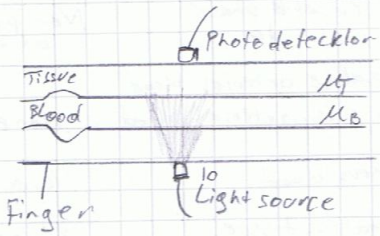
- Nonlinear dependence of μ_a on λ and S
- If blood perfussion ↑ than z↓ ($\mu_a^{Blood} >> \mu_a^{Tissue}$)

→ In den Praxis werden natürlich fixierte Photodetektoren verwendet, die, je nach Einsatzort (als Ort, bei dem man die Tiefe der Blutgefäße kennt) einen bestimmten Abstand d_r ~~[illegible]~~ aufweisen. μ_a kann dann ganz normal mit $I = I_0 \cdot e^{-\mu_a \cdot d_r}$ berechnet werden

Sensor operation

→ Principle

- Optical plethysmography, ie. registration of optical transmittion of blood



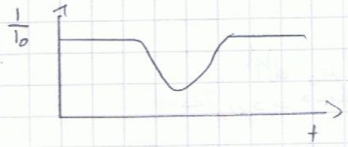
$I = I_0 \cdot e^{-\mu_a \cdot d}$

$\mu_T \approx 0{,}02\ mm^{-1}$ $\mu_B \approx 0{,}4\ mm^{-1}$

for wave length of 800nm

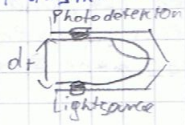
$\mu_T << \mu_B$

- Registration of volume and absorption changes

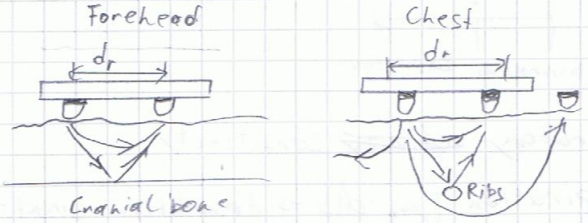


→ Transmittion vs reflection

- Transmittion → Standard



- Reflection → Novelty (Neuheit)
  - Scattering is advantageous, no direct light path
  - Light path is less defined



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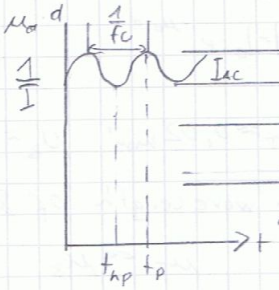
Application aspects

-> Physiological considerations

-> Light absorbers - compartmental (model of tissue)

$$I = I_0 \cdot e^{-\mu_a \cdot d}$$



Pulsatile phase at $t = t_p$ | Non pulsatile phase at $t = t_{np}$

Pulsatile arterial blood

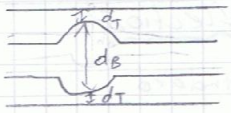
Non puls. arterial blood | Non puls. arterial blood

Venous blood | Venous blood

Bloodless tissue | Bloodless tissue

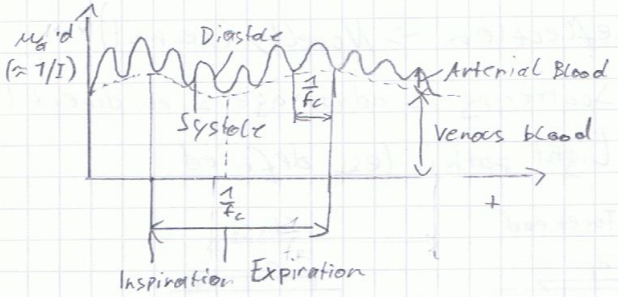
-> Cardiac sensitivity

- Systole: the product $(\mu_a \cdot d)\uparrow\uparrow$ because of both $\mu_a^{Blood} >> \mu_a^{Tissue}$ and $d\uparrow$



-> das Produkt $(\mu_a \cdot d)$ steigt, da μ_a von Blut größer ist, und das d von Blut sich vergrößert
-> lokal mehr Blut

-> Diastole: $(\mu_a \cdot d)\downarrow$



-> Respiratory ~~activity~~ sensitivity

- Inspiration $(\mu_a \cdot d)\downarrow$ -> Absorbtion sinkt -> Intensität steigt -> weil Sauerstoffsättigung $\uparrow$ und $d\downarrow$
  - Arterial blood: Left ventricular stroke volume $\downarrow$ and arterial $d\downarrow$
  - Venous blood: peripheral venous blood volume $\downarrow$ and venous $d\downarrow$

→ Registration of blood oxygenation S (= oximetry)

- $\mu_{a,p}$ as absorbtion due to pulsatile arterial blood

$$\mu_{a,p} = \rho \cdot \sigma = \frac{H}{V} \cdot \left(S \cdot \sigma_{HbO} + (1-S) \cdot \sigma_{Hb} \right)$$

H.. Hämatokrit
V.. volume of red blood
σ.. σ of red blood cells containing oxy/deoxyhemoglobin

- Application of the compartmental model of tissue

$$\ln\left(\frac{I}{I_0}\right) = -\mu_a \cdot d = -\left(\mu_{a,p} \cdot d_p + \mu_{a,np} \cdot d_{np} + \mu_{a,v} + \mu_{a,t} \cdot d_t\right)$$

- Alternating intensity component I_{AC} (from $dI/dd_p \approx \Delta I/\Delta d_p$)

I_{AC} .. Pulsierende Anteil der Intensität

$$I_{AC} = \Delta I = -I_0 \cdot \mu_{ap} \cdot e^{-\mu_{a,p} \cdot d_p - \mu_{a,np} \cdot d_{np} - \mu_{a,v} \cdot d_v - \mu_{a,t} \cdot d_t} \cdot \Delta d_p$$

Zur Erinnerung μ_{ap} ist irgendetwas gegen ~~0,5~~ $\frac{1}{mm}$ $_{0,1mm}$ und d wird auch nicht ~~viel~~ recht groß sein (irgendetwas im ~~mm~~ Bereich) → $\mu_{ap} \cdot d$ ist also ein Faktor ($mm/mm = 1$) kleiner 1

→ meist so gegen 0,05

- Direct intensity component $I_{DC} \approx I$ because I_{AC} only up to 5% of total I (wegen dem 0,05 Faktor)

the ~~AC-DC~~ AC & DC ratio r can be given as

$$r = \frac{I_{AC}}{I} = -\mu_{a,p} \cdot \Delta d_p \approx \frac{I_{AC}}{I_{DC}}$$

- Ratio R given by ratio of r at red wavelength (Index R) to r at infrared wavelength (index IR)

$$R = \frac{I_{AC}(R) / I_{DC}(R)}{I_{AC}(IR) / I_{DC}(IR)} \quad \text{or} \quad R = \frac{\mu_{a,p}(R)}{\mu_{a,p}(IR)}$$

- Finally, the relationship between R to measure and S to estimate

$$S = \frac{R \cdot \sigma_{Hb}(IR) - \sigma\sigma_{HB}(R)}{[\sigma_{HbO}(R) - \sigma_{Hb}(R)] + R \cdot [\sigma_{Hb}(IR) - \sigma_{HbO}(IR)]} \rightarrow$$

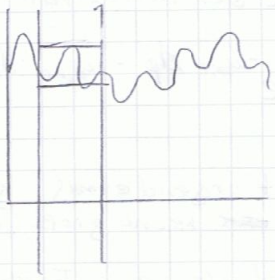
$$\Downarrow$$

$$0$$

$$\Rightarrow S = \frac{R \cdot \sigma_{Hb}(IR) - \sigma_{Hb}(R)}{\sigma_{HbO}(R) - \sigma_{Hb}(R)} \approx -0{,}26 \cdot R + 1{,}11$$

→ Wie würde man jetzt von der Messung zu S kommen?

1) Intensitätsmessung I mit ~~Rot und IR~~ Rot und Infrarot machen

2) 

Dann nehmen wir von einer kurzen Periode (1-2 Herzschläge) $I_{s,s}$ (1 Spitze-Spitze) Differenzen → I_{AC}, Rest ist I_{DC}

3) R berechnen → $R = \frac{I_{AC}(R)/I_{DC}(R)}{I_{AC}(IR)/I_{DC}(IR)}$

4) $S = -0{,}26 \cdot R + 1{,}11$

Multiparametric monitoring

→ Cardiac sensitivity
from pulsatile light intensity (f_c)

→ Respiratory sensitivity
from non-pulsatile light intensity (f_R)

→ Blood oxygenation

- measurement of R by the use of two wavelength
- Reflectance oximetry on the chest
(- accelerated assessment of relative S changes)

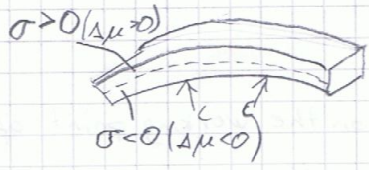
BSS - Mechanic Biosignals

Operation of skin curvate sensor

-> Principle

-> Single magnetostrictive Layer (ML)

- Neutral bending plane ($\sigma = 0$) remains in the centre of ML
- Total $\Delta\mu \approx 0$

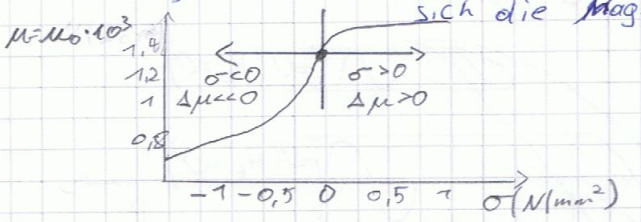


μ.. Permeability

σ.. Mechanical stress

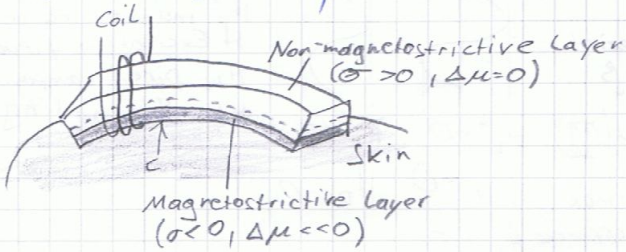
c.. Curvature

Magnetostriktiv => durch Druck und/oder Zug ändern sich die ~~Magnetischen~~ Eigenschaften



-> Bilayer structure

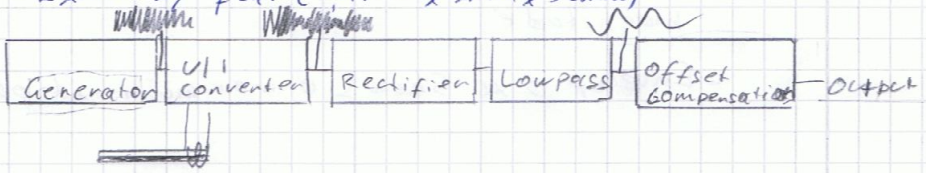
ML affixed to a non magnetic (non-magnetostrictive) counter Layer (CL)



-> Magnetostrictive Layer as senson element

-> Coil for the establishment of ~~the~~ an electrical senson signal

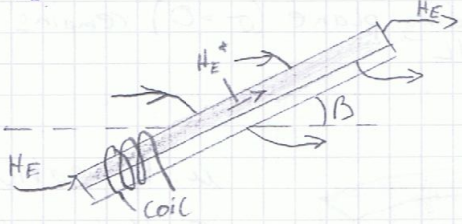
-> Extremely flat (0,1mm x 3mm x 50mm)



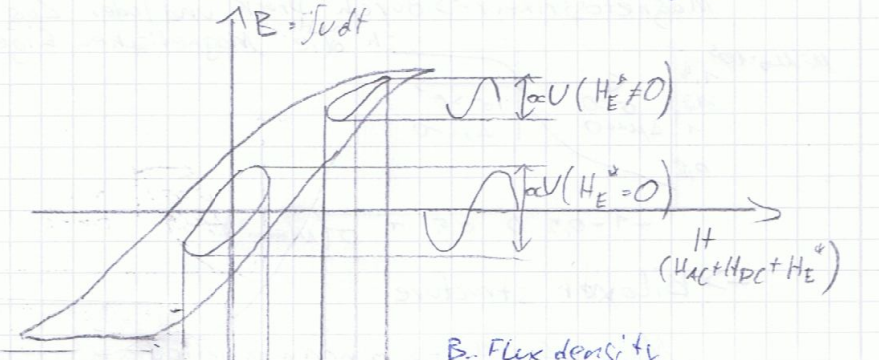
→ Working point optimisation

- Bilayer orientation within the earth magnetic field H_E

- H_E induces within the ML a magnetic field H_E^* ($<< H_E$)
- H_E^* increases with decreasing orientation angle β



- Influence of H_E^* on the working point of the ML hysteresis (since ML is a soft magnetic material)

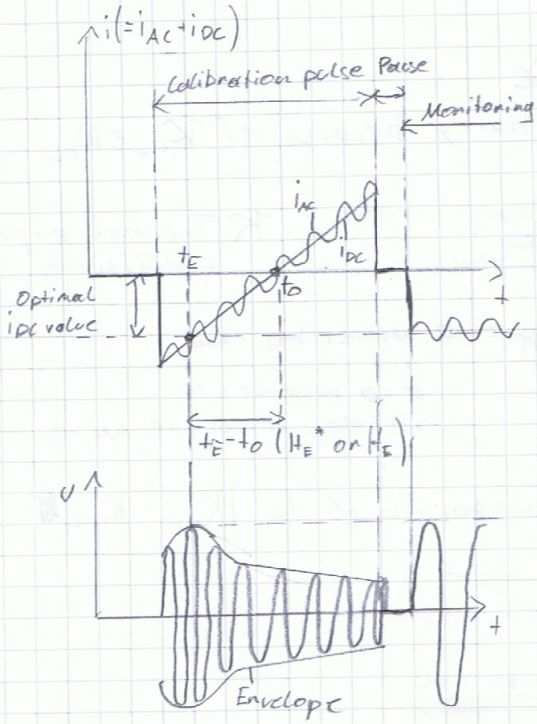


B.. Flux density
μ Coil voltage
H_E^* Induced earth magnetic field
H Excitation field strength
H_{DC} Direct component
H_{AC} Alternating component

Oder einfach: Wirkt das Erdmagnetfeld H_E auf den ML wird ein Magnetfeld H_E^* induziert. Ändert sich das Magnetfeld in einer Spule wird Strom erzeugt. Wird der Winkel β zum Erdmagnetfeld kleiner, wird H_E^* stärken. Wird er größer (bis 90°) sinkt H_E^* bis auf 0. Ist $H_E^* = 0$ erzeugt H_{AC} das maximum an Spannungsdifferenz ΔU, bei steigenden Einfluss von H_E^0 wird ΔU kleiner

$H_E^* = 0$	$H_E^* \neq 0$
$H_{DC} = 0$	$H_{DC} = 0$
$H_{AC} \neq 0$	$H_{AC} \neq 0$

- Calibration pulse of coil current i
- Sawtooth-shaped i_{DC} and i_{AC} with constant magnitude
- Envelop o μ shows a maximum at optimal i_{DC}



μ Coil voltage
H_E^* Induced earth magnetic field
i_{DC} Direct component
i_{AC} Alternating component

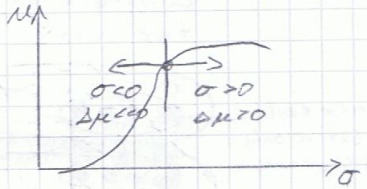
i ... Coil Current

Einfach erklärt: Ändert sich das Magnetische Feld innerhalb einen Spule wird Strom erzeugt. Bei dem „Calibration Pulse" wird ~~die~~ der Sensor angelegt, das heißt, der Sensor muss sich einmal (den Grundform der Haut anpassen. Dabei wird Strom induziert bzw. ändert sich wieder die Ausrichtung zum Endmagnetfeld.

→ Recht viel mehr kann ich als nicht elektrotechniker auch nicht dazu sagen

Application aspects

- Bending sensitivity



  - σ > tension
  - σ < 0 for compression
- 2D mechanical Stressdistribution → egal (Folien seite 11)

→ finite length bilayer → strongly inhomogeneous stress distribution

- quantitative assessment of ~~stress~~ bending sensitivity S

$$S = \frac{\Delta \mu_r}{\Delta c} = k_e \cdot k_m$$

→ magnetoelastic factor k_e
(depends on material properties of ML

$$k_e = \left(\frac{d\mu_r}{d\sigma_{ML}}\right)_{H=const}$$

→ mechanical factor k_m
(depends on material properties of ML and CL as well as BL-dimensions

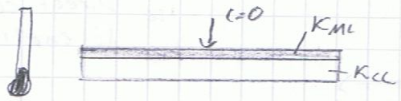
$$K_m = \frac{\Delta\sigma_{ML}}{\Delta c} = \frac{E_{ML} \cdot E_{CL} \cdot d_{CL} \cdot (d_{ML} + d_{CL})}{2 \cdot (E_{ML} \cdot d_{ML} + E_{CL} \cdot d_{CL})}$$

→ S is proportional to E_{CL}/E_{ML} and d_{CL}/d_{ML}

→ stiffen / thicken CL Layer - higher S

- Thermal sensitivity

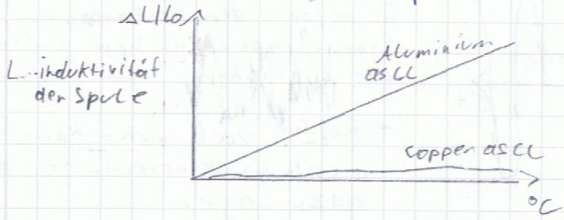
- Non-zero sensitivity because of $K_{ML} \neq K_{CL}$



K.. Thermal expension coefficient

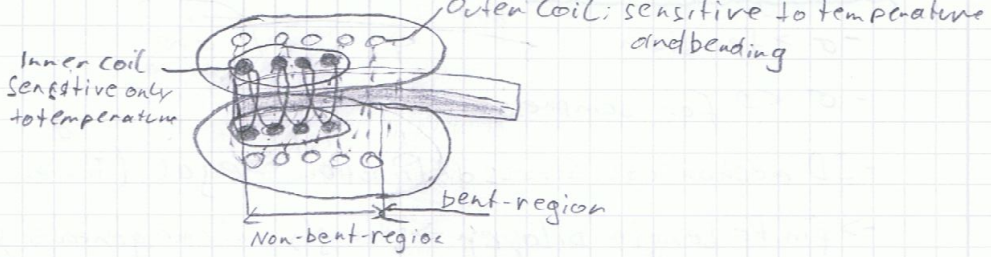
$\varepsilon >> 0$ because $K_{CL} > K_{ML}$
and
$\sigma < 0$ because $\varepsilon > 0$
→ in total $\sigma > 0$ and $\Delta\mu > 0$

- Sensitivity is proportional to $|K_{ML} - K_{CL}|$



- Cross-elimination of bending/thermal sensitivity

→ Elimination of thermal sensitivity using two coils



- Elimination of bending sensitivity using trilayer

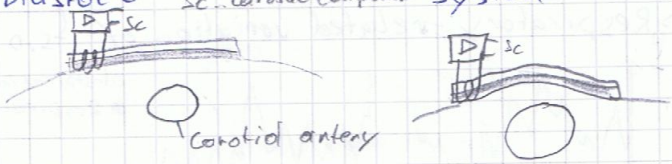
→ Biegen gleicht sich aus

Monitoring on the neck

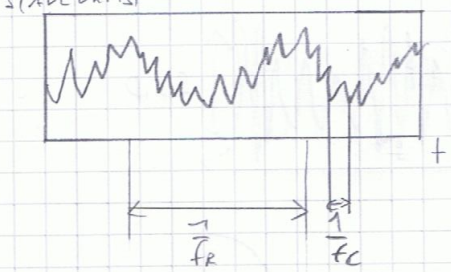
→ cardiac activity

- Systole vs diastole

Diastole Sc...cardiac component Systole



- Skin curvature signal



- pure cardiac component during apnea

→ Respiratory activity

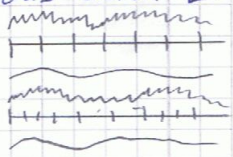
- Linear filtering (fixed cutoff)

→ alles was innerhalb von einem $\hat{f_C}$ ist (also zwischen Start und Endpunkt) wird rausgeschnitten und linear interpoliert. Somit bleibt nur mehr S_R übrig

- Adaptive filtering (adaptive cut-off)
hier wird f_C geschätzt, und ~~zwischte~~ das selbe wie bei Linear filtering gemacht

→ Der Unterschied ist einfach, das beim LF eine Herzrate genommen wird

und beim AF immer die aktuell geschätzte -> ist halt einfach genauer...

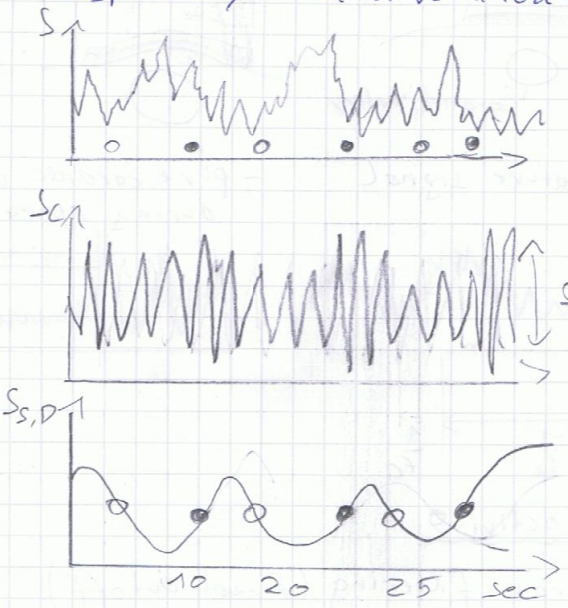


-> Blood pressure (bis jetzt unser einziges Biosignal dafür!)

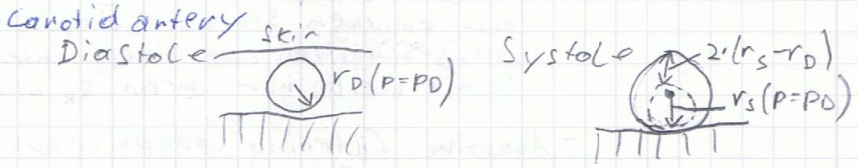
- Motivation: similar wave courses of S_C and p

S_C .. Cardiac component of S $S_{S,D}$ .. Systolic-diastolic Deflection in S_C
S .. Signal
p .. Blood pressure

-> Respiratory-related variation of $S_{S,D}$ and p_S



- Establishment of a model



~~Rod~~

-> Systolic-Diastolic deflection $p_{S,D}$

Radial deformation $\Delta r \approx r_D$

$$\Delta p = K \cdot \frac{\Delta A}{A} = K \cdot \frac{2 \cdot \Delta r}{r} = K \cdot \frac{2\Delta r}{r_D}$$

A ... Artery cross-section area

r_D .. Diastolic radius

K .. Elasticity module

$\Delta r \sim \Delta S$ & $K = f(\Delta r)$, i.e. radius related stiffness

$$v = \sqrt{\frac{K}{\rho}} \text{ thus } \Delta p = \frac{\rho \cdot v^2 \cdot 2 \cdot \Delta r}{r_D} \approx C_1 \cdot \frac{\Delta S}{\tau^2}$$

ρ .. Density, v Pulse wave velocity

τ Pulse transit time, C Constant

$$P_{S,D} = C_1 \cdot \frac{S_{S,D}}{\tau^2} + C_2$$

$S_{,D}$ .. Systolic-diastolic deflection in S_C

->Die restlichen Herleitungen sparn wir uns mal...

Diastolic Pressure P_D

$$P_D = C_3 \cdot \frac{1}{\tau^2} + C_4$$

Systolic Pressure

$$P_S = C_5 \cdot \frac{S_{S,D}}{\tau^2} + P_D + C_6$$

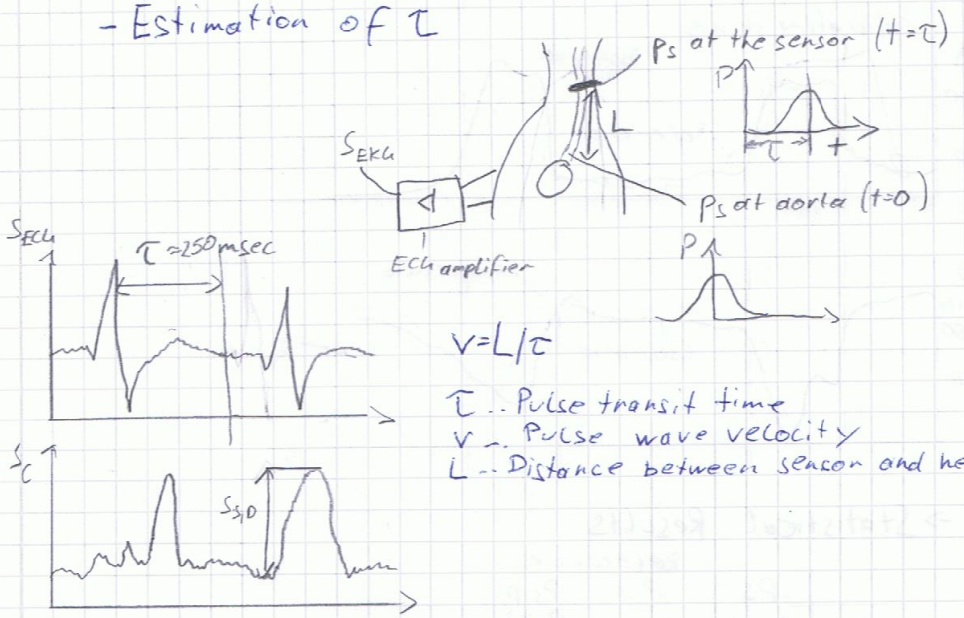
Is nämlich elogisch, dass Diastolische Blutdruck + den Abweichung der Systolische sein wird.

Mit dieser Methode lässt sich übrigens nun den Verlauf des Blutdrucks feststellen. Misst man ihn vorher mit einer anderen Methode lassen sich die Konstanten ausrechnen!

-> Interessant ist, das ~~den~~ eine Blutdruckänderung nur von der Pulse Transit time abhängig ist (τ)!

↓

- Estimation of τ

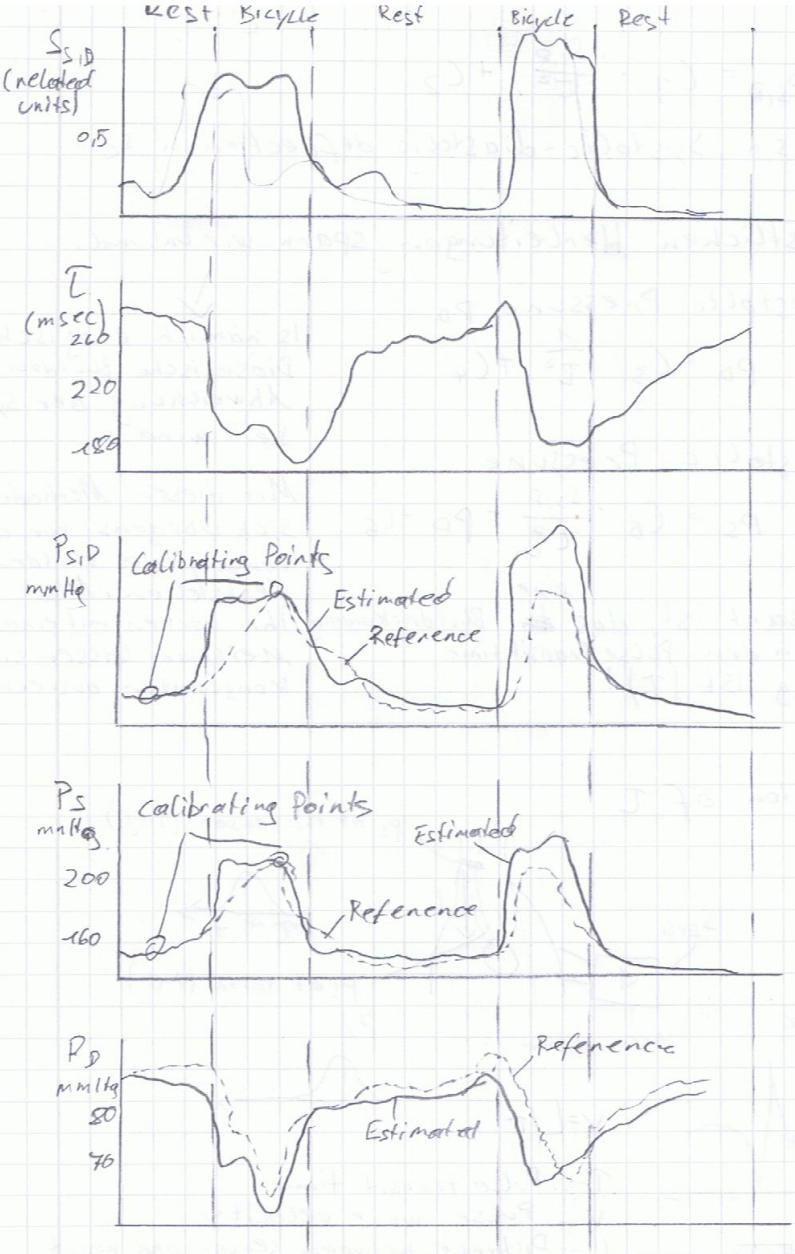


$$v = L/\tau$$

τ .. Pulse transit time
v .. Pulse wave velocity
L .. Distance between sensor and heart

- Estimation of $P_{S,D}$, P_S, P_D

-> nächste Seite



→ Statistical Results

Model		Reference P_S	Reference P_D	Reference $P_{S,D}$
	$S_{s,D}$	0,74	0,42	0,73
	τ	0,73	0,9	0,83
	V	0,74	0,9	0,84
	P_S	0,86	0,42	0,84
	P_D	0,74	0,92	0,85
	$P_{S,D}$	0,81	0,54	0,82

Cross-correlation coefficients

→ Restrictions

→ Inert (zögerliche) changes of stiffness (Elastizität)

→ After temporal increase of P_S value of K decreases slowly with some delay during a subsequent decrease of P_S