Sensory Physiology Survival Guide: Complete Overview

1. Basic principles of Sensory Physiology

1.1. **Entero/Extero** receptors: respond to signals from [within] vs. [outside of] the body

1.2. **Primary/Secondary** sensory cells: [primary = axons & APs, e.g. for smell (olfaction)] vs. [secondary = no axons & direct synaptic transmission, e.g. for hearing, taste]

1.3. Terms

- 1.3.1. **Reception**: Receptor potential = initial electrical response of sensory cell. **Graded**. If primary → APs; if secondary → NT release
- 1.3.2. **Transduction**: Energy from stimulus → Electrical signal for nervous system
- 1.3.3. **Transmission**: Between 2 synapses
- 1.3.4. **Perception**: Information processing in a network (CNS)
- 1.3.5. **Signal amplification**: Allowing to respond to minimal energy quantity
- 1.3.6. **Sensory modality**: Types of sensory info. Low threshold for adequate stimuli, high otherwise; due to structures etc. selectively sensitive to 1 modality

1.4. Response characteristics of a sensory cell

- 1.4.1. **Initial lag phase**: Sensory cell gets activated gradually
- 1.4.2. **Working range**: More or less linear. Nice! Faithful information
- 1.4.3. **Saturation phase**: Receptor potential will no longer increase
- 1.4.4. [**Spontaneous activity**: Avoiding lag phase, directly entering working range]

1.5. **Weber-Fechner**:

- 1.5.1. Law: Logarithmic relation between stimulus intensity and subjective experience
- 1.5.2. Experiment: Pairs of stimuli presented + reduced difference until not perceptible anymore → **Minimal perceptible increment**. Then: Experiment over wide range of intensities: M.P.I. depends on strength of first stimulus: Sensitivity (-) for progressively higher stimulus intensities. → **Weber-Fechner-Law: Logarithmic**
- 1.5.3. General: (+) **Extend working range**; (-) Measure **absolute** stimulus intensity

1.6. Reproduction of timing

- 1.6.1. **Tonic**: Continuous stimulation → Fires continuously
- 1.6.2. **Phasic** Continuous stimulation → Fires at on- and offset. Changes
- 1.6.3. **Phasic-Tonic**: →Initially onset/offset, then proportional (but lower than onset)

1.7. SLIDES: **Sensitivity** of receptor cells: How can we increase working range?

- 1.7.1. Detection: Threshold of cell structures (adjust the threshold?)
- 1.7.2. Spontaneous activity (avoiding the lag phase, directly in working range)
- 1.7.3. Amplification (cascades, etc.)

1.8. **Adaptation**: (+) mode of info encoding, (+) protection against overstimulation. Where and how?

- 1.8.1. Filter level, accessory structure, f.e. close pupil → Info does not even enter
- 1.8.2. Transducer molecules, f.e. visual pigments bleached→Receptor molecules inactive.
- 1.8.3. Inhibition of enzyme cascade, f.e. feedback inhibition by 2nd mess. like cAMP
- 1.8.4. Change of electrical properties, f.e. free Ca^{2+} (+) → Ca^{2+}-sensitive channels (+)

1.9. Feedback and lateral inhibition:

- 1.9.1. **Feedback inhibition**: Inhibitory interneurons project back to receptor cell → Reduce signaling activity to appropriate level
- 1.9.2. **Lateral inhibition**: Neighboring cells often connected via inhibitory interneuron: Local stimulus → Inhibition of surround → Sharpening of contrast **exc**

2. Vision

2.1. Physics

2.1.1. **Photoreception**: Energy of photons → Interpretable signals for nervous system

2.1.2. Visible light: Wavelengths between 400 (blue) and 700 (red) → **Visual spectrum**

2.2. The human eye - Intro

2.2.1. **Cornea**: Initially refracts 70% of total refractory power of incoming light rays

2.2.2. **Lens**: Light rays further bent by lens → Inverted image on retina. Changing shape of lens → Refractory power adjusts to distance of objects → Accomodation: **exc**

2.2.2.1. Ring muscle (-) → Ciliary fibers stretch lens → Flattened shape → Far

2.2.2.2. Ring muscle (+) → Ciliary fibers (-) → Lens back to resting state → Near

2.2.2.3. How? Ring muscle (+) by mAchR→ Near, Ring muscle (-) by Atropine→Far

2.2.3. **Iris**: Controls amount of incoming light. → "Fast iris reflex"

2.2.3.1. Sphincter muscle → [by mAchR] → Closure of pupilla.
mAchR antagonist Atropin therefore blocks fast iris reflex

2.2.3.2. Dilatator muscle → In the dark: Dilatator muscle (+) → reopens pupilla

2.2.4. **Pairs of eye muscles**: Control movement of eye, (again) controlled by oculomotor centers in midbrain. There are 2 types of **involuntary** eye movements

2.2.4.1. Smooth pursuit movement: Response to moving object

2.2.4.2. Saccadic movement: Short sudden jumps. While scanning the world.

2.2.5. **Retina** (part of CNS, extension of Diencephalon)

2.2.5.1. **Photoreceptors** →radially to: **bipolar cells** → [**amacrine cells**] → **RGCs** → form optic nerve → to target neurons in brain

2.2.5.2. **+ Horizontal cells**: Mediate lateral contact between photoreceptors

2.2.5.3. **+ Amacrine cells**: Interconnect bipolar cells and RGCs

2.3. Photoreceptors

2.3.1. **Rods**: b/w vision, dim light vision, light-sensitive, 120 mio, periphery, strong convergence: >1000 rods synapsing onto 1 RGC → light-sensitivity (+), detail (-)

2.3.2. **Cones**: color vision, only under good illumination, 6 mio, fovea, nearly a 1:1 (maybe 6:1)-relationship → light-sensitivity (-), detail (+)

2.3.3. Photosensitive part of the cells: **Outer segment**. Rods: Outer segment filled with flat membrane disks containing rhodopsin (visual pigment); Cones: Pleated outer plasma membrane instead. Both: Unique presynaptic structure: **synaptic ribbon**.

2.4. The visual process

2.4.1. **Rhodopsin**: 7 TM protein **opsin** + **11-cis retinal** fixed to opsin + coupled to G-protein '**transducin'**.

2.4.2. **Dark current**: cGMP bilds to CNG-gated cation (+) channel and keeps it open → cation (+) influx → depolarization of photoreceptors (-30 mV) → "dark current"

2.4.3. **Illumination**: short: Cation-channel closes + potassium efflux → hyperpolarization

(1) 11-cis retinal -> all-trans retinal

(2) All-trans retinal splits off of opsin (transported into pigment cells→regeneration)

(3) Conformational change in opsin -> dissociation and release of G-protein complex

(4) Liberated alpha-subunit activates PDE (phosphodiesterase; its substrate is cGMP)

(5) PDE splits cGMP -> 5´-GMP

(6) Closure of CNG-gated cation (+)-channel -> no longer influx of cations (+) (dark current). Due to ongoing potassium efflux: even hyperpolarization!

2.4.4. **Signal amplification**: 1 rhodopsin activated by light → 500 G-proteins activated → Every molecule of PDE splits 2000cGMP molecules → 1.000.000 * amplification

2.5. Neuronal circuitry

2.5.1. Photoreceptor → via radially oriented bipolar cells → RGCs [+amacrine, horizontal]

2.5.2. **Cone-pathway**: Off-bipolar cells AND on-bipolar cells

2.5.2.1. Donkey(GJ): On/Off-bipolar cell: Name → response in light.

2.5.2.2. Donkey(LV): Photoreceptors themselves: Off-response in light: Therefore off-bipolar cells just have to pass the information (excitatory), whereas in on-bipolar cells, there must be some "inversion" → inhibitory synapse

2.5.2.3. **Off-bipolar cells**: Hyperpolarized in light → excitatory synapse: Ionotropic glutamate receptors

2.5.2.4. **On-bipolar cells**: Depolarized in light → inhibitory synapse: metabotropic glutamate receptors

2.5.2.5. **RGCs** connected to on-bipolar cells → firing (+) under illumination. Connected to off-bipolar cells → firing (-) under illumination. As expected

2.5.3. **Rod-pathway**: Only on-bipolar cells, but on- and off-responses possible

2.5.3.1. **On-response**: Metabotropic glutamate receptors (inhibitory -> see above)

2.5.3.2. **Off-response**: Via amacrine cells → release glycine(-) → hyperpolarization (electrical synapses between amacrine and RGC maintain on-response of RGCs in the rod-pathway)

2.5.4. Advantage of having both off-**and** on-bipolar cells: Input/ Output relationship (+) by combining their 'working range'(good!)-parts of the Input/ Output relationship: Off → high firing frequency for dark areas, on → for illuminated areas.

2.5.5. **Horizontal cells and lateral inhibition**: Connected to neighboring photoreceptor cells- Dark: Release inhibitory GABA. In a light spot: Neighbors influenced via horizontal cells: Hyperpolarization of illuminated photoreceptor, but then: Horizontal cell will reduce GABA-release → reduce the inhibitory effect on NEIGHBORING photoreceptors → Sharpening of contrast **exc** again

2.6. **RGCs** (number of photoreceptors connected to a RGC → its receptive field)

2.6.1. Moving a small spot light on the **center** of the receptive field:
On-center RGC: firing frequency (+)
Off-center RGC: firing frequency (-).
Center-surround antagonism: Center = -(surround) [lateral inhibition]

2.6.2. 3 types of RGCs

2.6.2.1. **Alpha cells** = M-cells: large, wide dendritic tree, coarse structure + movement

2.6.2.2. **Beta cells** = P-cells: small, fine detail analysis

2.6.2.3. **Gamma-cells**: Heterogeneous population, irregular, motion/direction

2.7. Color vision theories

2.7.1. **Trichromacy theory**: 3 primary colors (red, blue, green) sufficient to produce all other colors. Cones: 3 distinct classes of cones with overlapping absorption spectra. Additive and subtractive(?) color mixture consistent with this.

2.7.2. **Color opponency theory**: We see green after-image after looking at red surface and then at neutral white. RGCs: red-green, blue-yellow and black-white color oppenency. BTW: blue+ (green+red) → blue/yellow opponency

3. Visual processing and perception + Pathways

3.1. **Optic tectum** in lower vertebrates and mammals

- 3.1.1. Fish: Total decussation (Überkreuzung) of both nerves, [higher mammals: partial!]. Fish: Retinal axons -> primary visual center in midbrain = optic tectum
- 3.1.2. Optic tectum in mammals: No longer a primary visual center (only 10% of RGC axons), but **coordination center of eye movement**. 2 functionally different parts
  - 3.1.2.1. →**Colliculus superior** (oculomotor input): Controls position of eye ball and therefore of the visual field by innervating the 3 pairs of eye muscles + stable visual scene + multimodal interface
  - 3.1.2.2. →**Colliculus inferior** (auditory input) → see: Hearing

3.2. The human central visual pathway **exc**

- 3.2.1. [Already: light → photoreceptor → bipolar cells → RGCs → form optic nerve →]
- 3.2.2. Visual field(inner) → Retina (outer) → ipsi (BTW Temporal = outer; Nasal = inner)
- 3.2.3. Visual field(outer) → Retina (inner) → contra via Optic chiasm
- 3.2.4. Result: Left visual field info from both eyes→ right hemisphere → for 3D-Vision V1

3.3. **Lateral Geniculate Body** (**LGB**) = **Corpus geniculatem laterale** (**CGL**) = seitlicher Kniehöcker

- 3.3.1. CGL ∈ **Thalamus** ∈ **Diencephalon**
- **3.3.2.** Retinal fibers terminate in CGL. **Both visual fields still separated. Each CGL input from only one half of the visual field**
- 3.3.3. CGL: excitatory synapses (**glutamate**) + precise **topographic** representation of retinal image on thalamus (is this equal to retinotopy?!)
- 3.3.4. Layers: **exc**
  - 3.3.4.1. **Parvocellular pathway**: Color, fine details
    Layers 3-6, beta-RGCs – Fovea → Central retinal region overrepresented.
    Blobs in layers 2/3 of V1; visual field V4
  - 3.3.4.2. **Magnocellular pathway**: Motion detection, coarse structure
    Layers 1-2, Alpha-retinal ganglion cells – Periphery
    Interblobs in layers 2/3 of V1; visual field V5
- 3.3.5. At the level of CGL, the visual pathway is split into
  - 3.3.5.1. Nasal contralateral branch: terminating in **layers 1,4,6 → KIIKIK**
  - 3.3.5.2. Temporal ipsilateral branch: terminating in **layers 2,3,5 → KIIKIK**

3.4. **Thalamo-cortical gating**: Filtering of sensory signals by inhibitory GABAergic interneurons in thalamo-cortical gating in CGL. Controlled by strong feedback projection from layer 6 to V1.

- 3.4.1. Relay neurons: 2 different types of response activity
  - 3.4.1.1. **Highly attentive state**: Regular spiking activity: Tonic firing of AP
  - 3.4.1.2. **Sleeping mode**: Rhythmically bursting: Low-threshold voltage-gated calcium-channels that open in response to IPSP and elicit slow depolarization. After certain level of depolarization, voltage-gated sodium channels also open → burst of APs. Repolarization: Calcium → calcium-sensitive potassium channels open → repolarization.

3.5. **Primary visual cortex = V1** = area 17 = striate cortex (**in occipital lope, 6 layers**)

- 3.5.1. Visual fibers from CGL → stellate cells in layer 4 → pyramidal cells with cell body in layer 5 → pyramidal cells in layer 6 → back to CGL

3.6. Functional organization of the visual cortex:

3.6.1. Most sensitive to **bars** and straight borders → Cortex functionally organized into **laterally aligned columns**, preferring different angle of bar orientation

3.6.2. Cellular basis: Convergent connection **between stellate cells in layer 4 and simple pyramidal cells** → Overlapping receptive field of individual stellate cells is collected on a target neuron → Each simple cell receives excitatory input from CGL cells (via stellate cells) whose on-centers are linearly aligned → Summation of EPSPs, simply cell maximally activated if light falls exactly on this receptive field.

3.6.3. **End-inhibition**: Allows discrimination of bar length, e.g. by feedback loops from layer 6 pyramidal cells that project back via GABAergic interneurons

3.6.4. Complex vs. simple cells: Complex cells next level of abstraction in visual processing. Like simple cells respond best to borders, but respond independent of position in visual field → Some detect edges, some geometrical structures, etc.

3.7. **Ocular dominance columns**: Alternately connected to left or the right eye → Detailed comparison of visual disparity angles

3.8. **Blobs** in visual cortex: Mitochondrial enzyme "cytochrome oxidase" highly accumulated **in layers 2 and 3** of V1 in clusters (blobs). Color-sensitive target neurons grouped together there.

3.9. Projection to higher cortical areas

3.9.1. **Parietal "where" pathway**: motion detection, space perception, multisensory int.

3.9.2. **Temporal "what" pathway**: object recognition, recognition of faces, visual memory

3.10. Cerebral ["das Großhirn betreffend"]/Split brain [We already know that]

3.10.1. **Left**: language analysis, writing, reading, calculating → **procedural** processing

3.10.2. **Right**: spatial construction, geometric pattern analysis, nonverbal ideation, emotional touch, music perception → **declarational** processing

4. Hearing

4.1. **Sound**: Mechanical vibration through air or water. **Frequency range**: 20 Hz – 16 KHz. **Loudness range**: 0 [threshold of audibility] – 130 [threshold of pain]. Subjective perception on **log** scale. Loudness sensitivity depends on **sound frequency**. Highest sensitivity: 2-4 KHz (speaking). **Isophones**: 80 db in 1000 Hz = 135 db in 16 Hz. Pure sounds = sine waves: frequency (pitch), amplitude(loudness)

4.2. Human Ear: Organs of equilibrium + Organs of hearing **exc**

4.2.1. **Overview**: (1) Sounds waves collected in external ear → via (2) external auditory canal to → (3) eardrum: vibration → (4) 3 bones connected to eardrum: vibration → (5) oval window: vibration → (6) fluid inside cochlea: vibration → (7) hair cells (Organ of Corti) fire **(see below!)** → (8) signal via auditory nerve → (9) brain

4.2.2. **External ear**: Funnel: Sound waves → Eardrum (=tympanic membrane). Amplifies sounds in particular frequency range. Directional sensitivity.

4.2.3. **Middle ear**: **Eardrum** (tympanic membrane) **+ 3 bones (malleus, incus, stapes)** + **oval window**

4.3. Detail: Inner ear/Cochlea (contains the Organ of Corti) **exc**

4.3.1. **Scala vestibuli** (Outer): Sound waves travel back through S.T. and leave Cochlea through round window. Perilymph

4.3.2. <- Reissner membrane ->

4.3.3. **Scala media** (Inner): Endolymph (sodium --, potassium ++)

4.3.4. <-Basilar membrane ->

4.3.5. **Scala tympani** (Outer): Perilymph(sodium ++, potassium --)

4.3.6. **ORGAN OF CORTI**: Sort of between Scala media and basilar membrane

4.4. Signal amplification (together x22) **exc**

4.4.1. Funnel effect (x17): size reduction from eardrum to oval window

4.4.2. Lever effect (x1.3): malleus, incus, stapes.

4.5. **Hair cells**: Secondary sensory cells -> no axon [Phil]. Sound input → via hair cells → neuronal signal. In 4 rows on basilar membrane, covered by tectorial membrane (consists of "Schleim"). Elongated with stereocilia, 2 subtypes

4.5.1. **Inner hair cells:** 1 row of cells, sound detection, 90% of auditory nerve, directly responsible for hearing → sound detection. Each hair cell: Numerous afferent nerve fibers. No direct contact with tectorial membrane. Divergent innervation (???)

4.5.2. **Outer hair cells:** 3 rows of cells, connected with 10% of afferent fibers, but strong efferent input, direct contact with tect. membrane. Convergent innervation (???).

➔ **Able to modulate and amplify the sensitivity of sound reception.**

4.6. Hair cells: **Process of sound reception**

4.6.1. Hair cells sensitive to mechanical stimulation (stretch or distortion): (I) Hair bundles [Individual cilia [connected to each other via tip links]] move against tectorial membrane →(II) Cilia bent by the shearing forces.
(1) Mechanical displacement of a hair bundle/deflection of hair cells
→ (2) Mechanosensitive ion (positive) channels open
→ (3) K^+-influx (high extracellular potassium concentration in endolymph)
→ (4) Depolarization → (5) Opening of voltage-gated calcium channels
→ (6) Ca2+-influx →(7) exocytosis of synaptic vesicles → (8) NT release (glutamate).

4.6.2. Repolarization: (A) Voltage-gated potassium channels in lateral membrane open
→ (B) potassium efflux into perilymph fluid.

4.6.3. Inner hair cell: No contact to tectorial membrane, but outer hair cells have.
<60 db, then endolymph stream alone too weak to stimulate the inner hair cells.
→ **Outer hair cells: Voltage-to-force converter**: Able to modulate and amplify the sensitivity of sound reception by modifying the mechanical coupling between inner hair cells and the tectorial membrane: 1000 fold signal amplification by rhythmic cell contraction (up to 20000/s). Protein prestin in lateral membrane of outer hair cells acts as direct voltage- to force converter, allowing fast cell contractions.

4.7. Frequency analysis:

4.7.1. Basilar membrane thicker at the base, more elastic and thin at the apex.
+ selective responses of hair cells to different frequencies of sound.
High frequencies → base | Low → apex: Traveling waves along the whole length of basilar membrane from narrow base to wide apex (Gradient of stiffness)
Extend of membrane displacement → how strong hair cell stimulated

4.7.2. Frequency analysis in auditory nerve

4.7.2.1. Sounds <=1 KHz: Periodicity of APs = length of sine wave (linear)

4.7.2.2. Sounds > 1KHz: Not that linear, because 1:1-correspondence impossible
Phase periodicities of several nerve fibers encode wavelength

4.7.3. Loudness: Heard more close to the base. Frequency discrimination more close to apex. Number of activated fibers per inner hair cell increases with stimulus intensity
Tones of same intensity, but different frequency → perceived as different loudly.
Extra-sensitive: 3000-4000 Hz.

4.7.4. Tuning curves: Describe sound pressure required for producing certain change in membrane potential at various sound frequencies ("best frequency curve"). Slopes more shallow at lower frequencies and steeper at higher ones. Threshold is frequency dependent. Tuning curves reflect receptive field of different frequencies.

5. Hearing – Central auditory processing

5.1. Auditory pathway: (1) Auditory fibers → (2) Neuronal cell bodies in Spiral ganglion → (3) … via 8th cranial nerve → (4) Nucleus cochlearis in brain stem → (5) **splitting into 3 major pathways**

- 5.1.1. (5.1) Slow dorsal "what" path: Pattern analysis, to C.I. –pyramidal cells
- 5.1.2. (5.2.) Fast ventral "that" path: Pre-warning to C.I. – bushy, octopus
- 5.1.3. (5.3.) Ventral "where" path: Superior oliva (sound localization) – bushy, octopus

5.2. **Nucleus cochlearis (Medulla obl. (brainstem)): Heterogenous population** of 2nd order neurons.

- 5.2.1. **Bushy** cells and octopus cells → **On**-response: Maintaining high temporal precision even at higher sound frequencies
- 5.2.2. **Stellate** cells → **Chopper**-response: Reflecting rhythmicity of sounds
- 5.2.3. **Pyramidal** → **Pauser**-response: Providing differentiation of onset and ensuing phases of responses

5.3. **Superior olivary complex**: Information from both ears converge → Binaural sound localization

- 5.3.1. **MSO (Medial superior oliva) <=1.5 KHz**→ Temporal delay. How is it done? → Coincidence detectors use a delay line circuitry (f.e. pathway from left ear is a bit longer than from right ear, s.t. they arrive simultaneously when x degree delayed)
- 5.3.2. **LSO (Lateral superior oliva)** >1.5 KHz→ Difference in sound pressure (intensity of sound). Majority of units ipsilateral excitatory or contralateral inhibitory → Subtraction

5.4. **Colliculus inferior** (in midbrain):

- 5.4.1. Simple auditory processing without memory formation; In the middle of bottom-up and top-down processing of the auditory system. Onion-bulb pattern of isofrequency planes (tonotopy) that are piled up opon each other in discrete cellular layers. A plane corresponds to a place on basilar membrane. Within each plane, target neurons are arranged in **isophone lines**- Neurons with highest sensitivity found in the center, those with lower in the periphery

5.5. Auditory cortex

- 5.5.1. Medial geniculate nucleus in thalamus: Main thalami relay nucleus for auditory information, direct projection into primary auditory cortex (A1) in temporal lobe.
- 5.5.2. A1 organized in isofrequency stripes (containing neurons with identical best frequencies) + tonotopic representation. These stripes subdivided into regions with different loudness, thresholds, etc. Overlapping neuronal maps for detecting
  - 5.5.2.1. Speed or direction of frequency modulation
  - 5.5.2.2. Shape of intensity curves
  - 5.5.2.3. Latency
  - 5.5.2.4. Response speed

6. Sense of balance ((Sense of equilibrium → Inner ear) + Cooperation with other modalities like prop.)

6.1. Semicircular ducts:

- 6.1.1. **3 tubes** (**perpendicular** to each other) filled with endolymph. Base: Each duct widened into **ampulla**. Hairs extend into gel. Mass (=**cupula**), extending to endolymph.

6.1.2. Movement: Semicircular duct rotates → cupula tilted against endolymph, which remains stationary → deflection of hairs → mechanically-gated ion-channels open
6.1.3. Rotation for longer time: Endolymph rotates at same speed → Cupula back in rest
6.1.4. Sudden stop: Bend cupula in opposite direction → dizziness
6.1.5. Hair cells: **Directional** sensitive: Movement of hair bundle towards longest hair: depolarization, other direction: hyperpolarization.

6.2. Macula organs (**Utriculus**: horizontal | **Sacculus**: vertical): Sense of gravity + linear acceleration
6.2.1. Field of hair cells covered by gel. Mass, in which **OTKONIA** are embedded near tips
6.2.2. Sudden movement: OTKONIA lag behind and deflect hair cells in opposite direction
6.2.3. At rest: Otkonia follow gravitation

6.3. Vestibular pathway:
6.3.1. 19000 nerve fibers of each vestibular nerve (**part of 8th cranial**): cell bodies in vest. ganglion → project to 4 vestibular nuclei in medulla→ direct connection cerebellum
6.3.2. Collateral fibers from vestibular pathway system → medullary reticular formation integrated into activities of the reticular systems → Involved in producing symptoms of motion sickness due to overstimulation of vest. System
6.3.3. In cerebellum: Info converges also from muscles via afferent path to spinal cord: tractus spino-cerebellaris: fast conduction velocity (>100m/s) → Proprioception

7. **Proprioception**: Monitoring position of muscles and joints. Measure mechanical forces

7.1. **Tension receptors: Golgi-tendon fibers (Ib-fibers)** measure **tension** in muscles. Synapse onto alpha-motoneuron via inhibitory interneuron (to maintain muscle tension if tendon becomes relaxed).

7.2. **Stretch-receptors**: **Muscle spindles** arranged in parallel with muscle fibers measure **length** of muscle. Respond to passive stretch stimulus with phasic-tonic discharge. Control length of muscle fibers via feedback loop: alpha-motoneurons in spinal cord activated and compensate passive stretch by muscle contraction (**mainstay reflex**)

7.3. **Voluntary movement control**: For voluntary muscle contraction, mainstay reflex has to be overcome. Efferent gamma-fibers from small gama-motoneurons in spinal cord innervate contractile portions of muscle spindles flanking central mechano-sensitive region of intrafusal fibers. Activation of gamma-motoneurons → Initiate stretching stimulus of muscle spindula (pretension) allowing muscle contraction until pretension in muscle spindle is compensated. → Available degree of muscle contraction elicited by alpha-motoneurons is controlled by activity of gamma-fibers.

7.4. **Movement control in the cerebellum**: Movement control by comparing afferent (sensory) info with efferent signal from motor cortex. If: sensory-motor mismatch → Cerebellum sends out correcting signals via Purkinje cell axons with GABAergic nerve terminals. → Strongly engaged in motor learning. 2 prominent pathways to deliver signals to Purkinje cells
7.4.1. **Climbing fibers**: Arise from inferior olivary nucleus. Extensive arborisation + literally climb over Purkinje cell dendrites making a large number of synaptic contacts. Synaptic signals of single climbing fiber → Strong influence on membrane potential of Purkinje cells. Thought to send out **correcting signals in sensory-motor mismatch**
7.4.2. **Parallel fibers**: Axons of granule cells in cerebellar cortex (outnumber any other neurons). Axons split into 2 parallel fibers + make single synaptic contact with large number of Purkinje cell dendrites in series.

8. **Olfaction** (Smell) – Detecting airborne molecules. 20.000 more sensitive than taste
    8.1. Structures
        8.1.1. Olfactory cells inside **nasal cavity** (turbinates), covered by olfactory epithelium, covered by thin layer of mucus. Receptors are primary cells with cilia.
        8.1.2. 7-10 different primary odorant classes
        8.1.3. Besides olfactory cells: Population of basal cells allows for self-renewal [2 weeks]
    8.2. Olfactory transduction
        8.2.1. Large family of genes [5%] encoding for about 1000 different receptor molecules. Odorant receptor: 7 TM domain protein with G-protein.
        8.2.2. Binding of the appropriate odorant → dissociation of G-protein complex → activation of adenylylcyclase → increase in cAMP level → opening of CNG-gated ion channel with high Ca^{2+} conductance → Calcium influx → membrane depolarization of receptor cells → Furthermore: calcium opens chloride channels → Chloride flows outward [low chloride concentration in the mucus] → further membrane depolarization [Strong amplification] of the odorant signal is attained.
        8.2.3. Olfactory signal rapidly terminated ← cAMP blocks further G-protein activation (feedback inhibition) + cAMP rapidly degraded by PDE
    8.3. Combinatorial code
        8.3.1. Discrepancy: Huge number of odorants and limited number of odorant receptors available → Combinatorial code: Extends number of odorants that can be identified by the 10000 receptor cells:
        8.3.2. How? Each odorant receptor selectively binds to a certain chemical group within an odorant molecule: Chemical group not unique to 1 odorant molecule but shared by any others. Specific odorant identification performed by population of receptor cells that together recognize the various chemical groups residing in one odorant.
    8.4. Olfactory pathway:
        8.4.1. Axons of olfactory receptors terminate in **Olfactory bulb** (in forebrain)
        8.4.2. Enormous convergence (about 1000 receptor cells) all possessing the same odorant receptor molecules → terminate in a cluster of synapses (**glomerulus**)
        8.4.3. Each of target cells (**mitral cells**) sends highly branched dendrite into one of the glomeruli → receives info selectively from one class of odorant receptor cells
        8.4.4. Axons of mitral cells → form olfactory tract that splits into 3 pathways → (1) thalamus, → (2) amygdala, → (3) hypothalamus

9. Taste

9.1. Basic qualities: (+) Sweet, (+) Salty, (+) Umami, (-) Sour, (-) Bitter

9.2. Physiology

9.2.1. Taste receptors grouped together in **taste buds** arranged on **papillae**. Taste buds in **tissue cavity** with pore on top. Consist of receptor cells surrounded by support cells

9.2.2. Taste bud contains >**25** receptor cells (secondary sensory cells with **microvilli**), most sensitive to one type of stimulus, but also respond to others. Taste info encoded by means of interactions between many elements of different specificities.

9.2.3. **Short life span (1 week)** like olfactory receptors. Secondary sensory cells (?)

9.3. Signal transduction

9.3.1. **Salty**: Readily dissociate in water and sodium ions passively diffuse through sodium channels (not voltage-gated but can be blocked by drug amiloride)-> depolarization

9.3.2. **Sour**: excess of protons. Act through so called PDK2-channels

9.3.3. **Bitter** and **sweet**: act through G-protein coupled receptor molecules: Free nerve endings of the trigeminal nerve responds to very strong stimuli, f.e. spicy-hot.

9.3.4. Taste cells use **ATP** as NT that is not released by exocytosis of synaptic vesicles but via bald channels of gap junctions

9.4. Gustatory pathway

9.4.1. Branch of facial nerve (**7th cranial**) → contacts anterior 2/3 of tong

9.4.2. **9th cranial** + **10th cranial** nerve → innervate posterior 1/3 + pharynx

9.4.3. Carry information to Nucleus solitaries in brainstem (medulla) and then

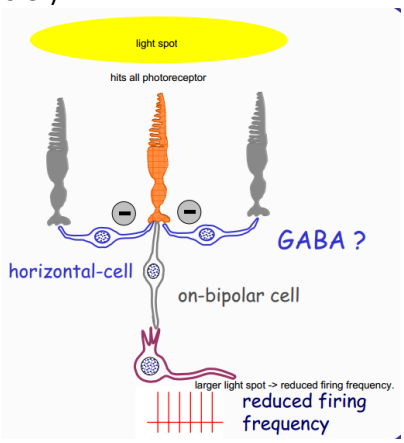
9.4.3.1. → **Thalamus** and **temporal cortex** (close to the somatosensory fields of the face) OR

9.4.3.2. → **Amygdala** and **hypothalamus** (limbic system, conveying emotional aspects of taste

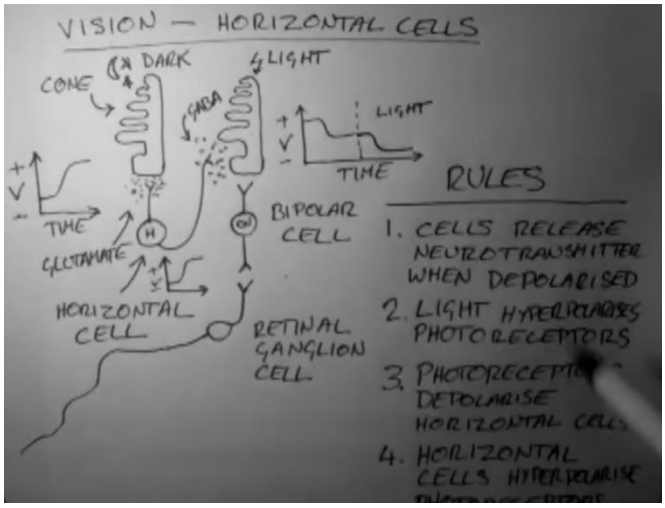
Sensory Physiology Survival Guide: 100% random excursions

(some of the *excursions* are only pictures, but *excursion* sounds cooler)

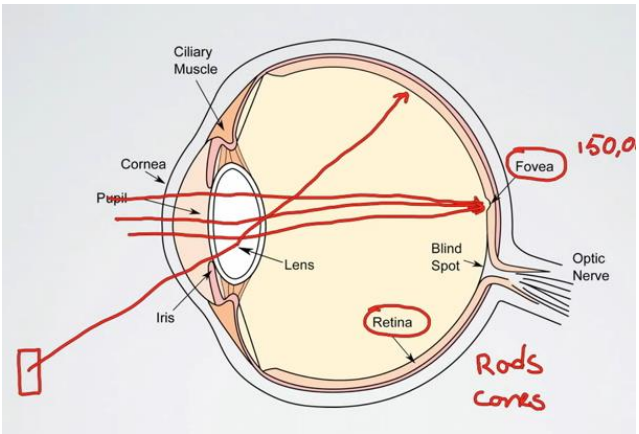
Excursion 1 - Lateral inhibition by horizontal cells:



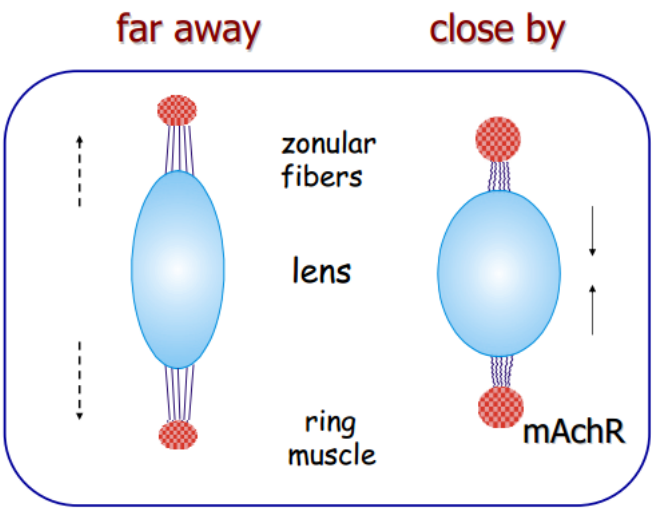
- Effect: Sharpening of contrast; borders; etc. (see: summary)
- Why? Look at the right side: Light hits the photoreceptor in the middle causing the on-bipolar cell to fire. However, the left and right photoreceptors (acting as horizontal cells) are also hit by light and reduce the firing frequency due to an inhibitory influence (~lateral inhibition). If these horizontal cells were not hit by light but in complete darkness, the inhibitory effect would drop out and the firing frequency would increase.
- How exactly? https://www.youtube.com/watch?v=jTEqoefv-pY
  2 cones, left cone is in the dark -> dark current -> depolarization -> glutamate-release (excitatory to horizontal cells) -> depolarization of horizontal cells -> neurotransmitter GABA (inhibitory) released -> cone 2 hyperpolarized.
  Now let´s say: Cone 2 is hit by light, then the cell is mega hyperpolarized -> no neurotransmitter release -> on-bipolar cell depolarized, which is connected to RGCs → Result: If light hits cone 2 (in the center of the on-center off-surround RGC), the RGC will be turned on, when additionally cone 1 (in the periphery of the RGC) is in the dark, the RGC will be turned on even more! The latter situation is a typical contrast situation and the response is increased!



Excursion 2: Human eye:

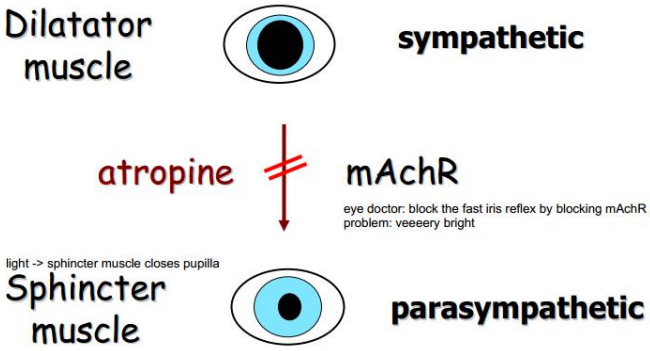


Excursion 3 - Accommodation lens:

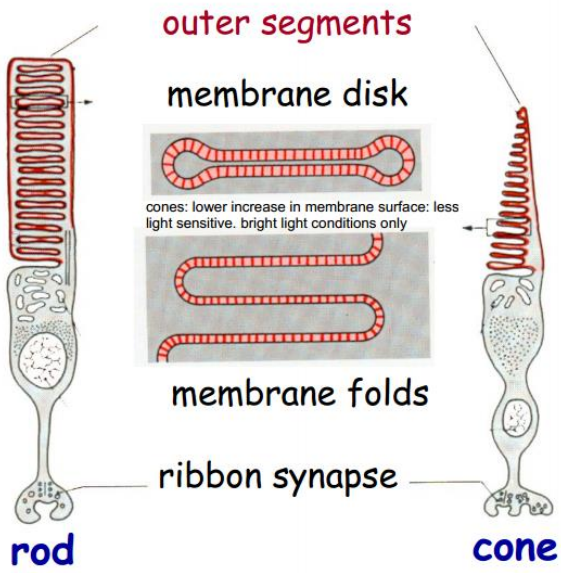


Excursion 4 - Atropin-related stuff in lens accommodation AND fast iris reflex (quite similar):

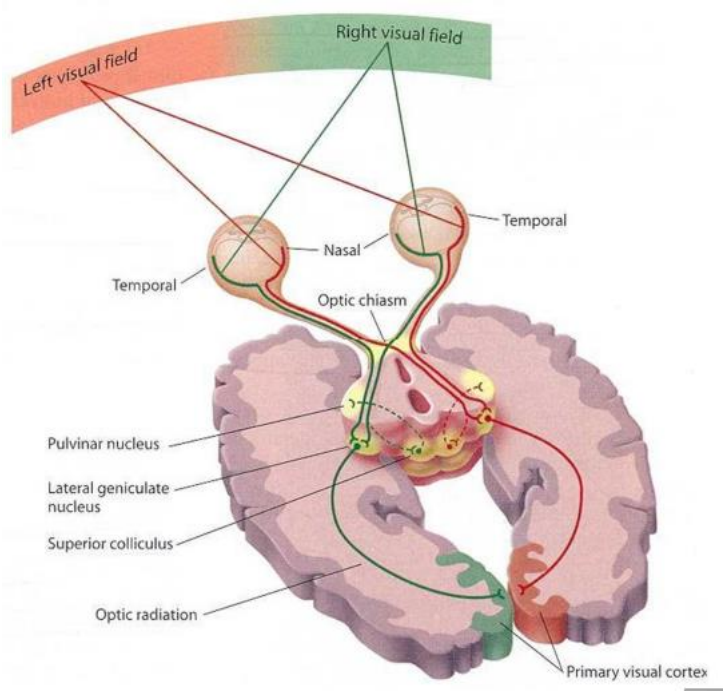
- Lens: Parasympathetic nerve (→mAChR) innervates ring muscles, causing a relaxation of the ciliary fibers allowing the lens to go back to resting state: near accommodation.
  Atropin is the muscarinic antagonist (antagonist to mAcHR), thus relaxes the ring muscles and causes the lens to be stretched by ciliary fibers: far accommodation.
- Fast iris reflex: A sudden closure of the pupilla when exposed to light is caused by a contraction of the sphincter muscle in the iris. This is initiated by cholinergic parasympathetic nerves innervating this muscle. So: Light -> Cholinergic parasympathetic nerve (+) -> Sphincter muscle innervated (+) -> Pupilla closes. Atropin blocks this reflex (doctor inspection)



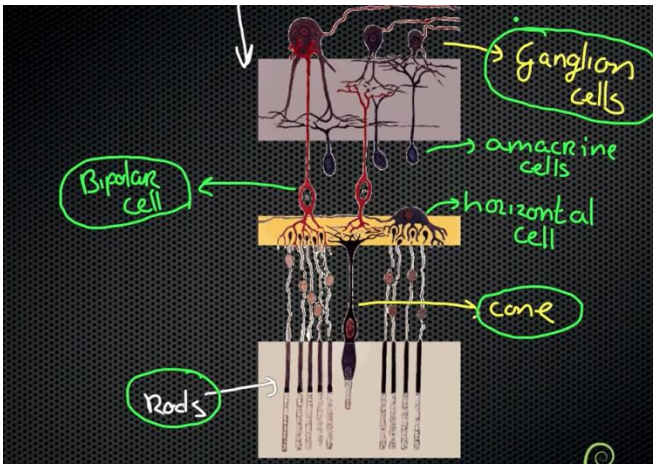
Excursion 5 - Outer segments of rods and cones



Excursion 6 - Human central visual pathway:



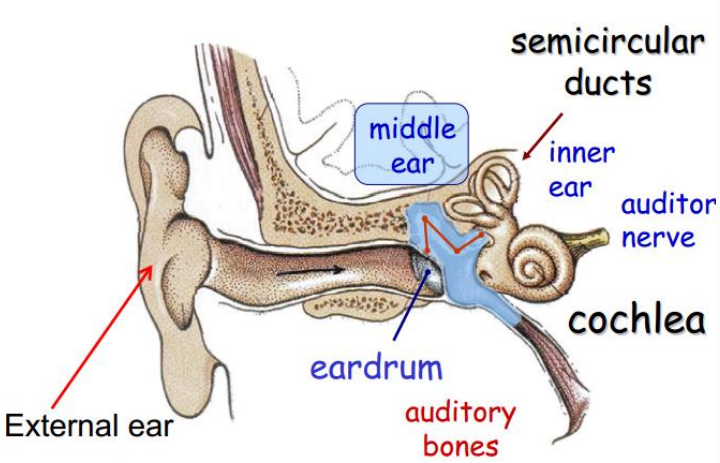
Excursion 7 – Retina:

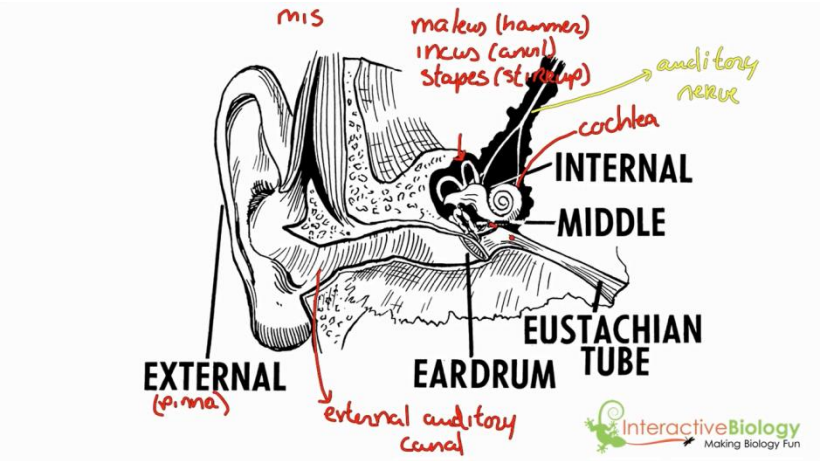


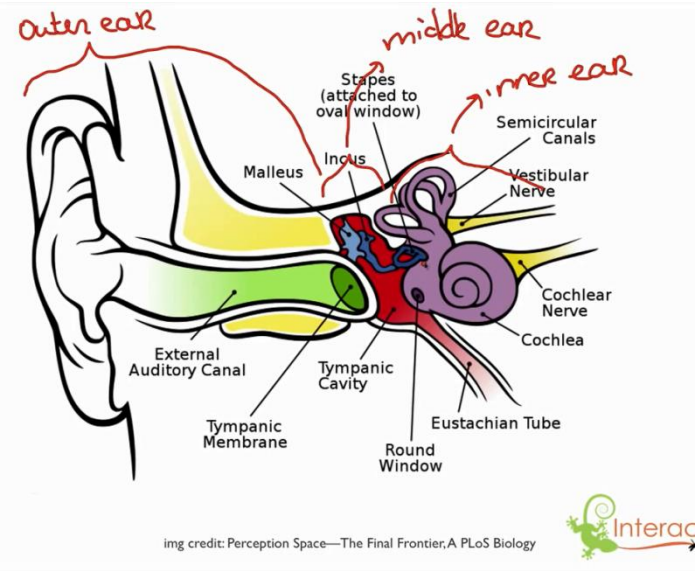
Excursion 8 - The layers (correct?):

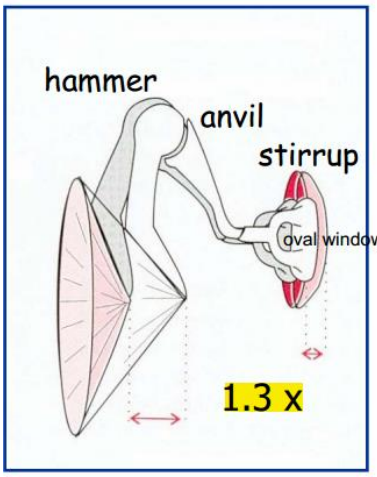
…

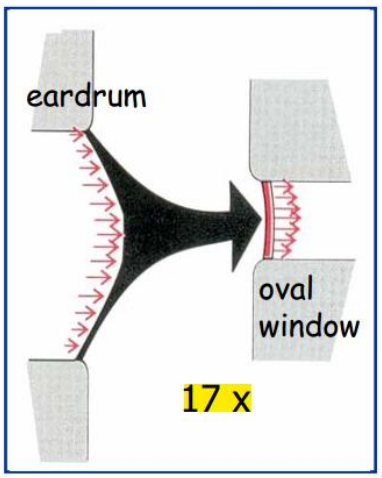
Excursion 9: Visualizations of the human ear, etc.:





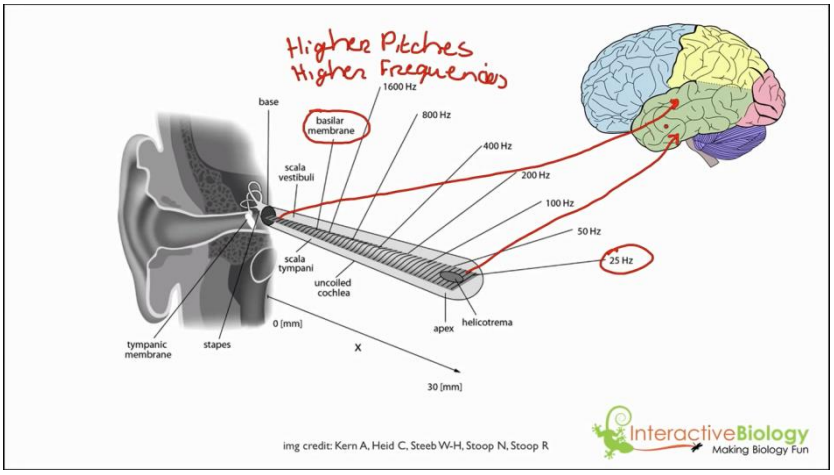


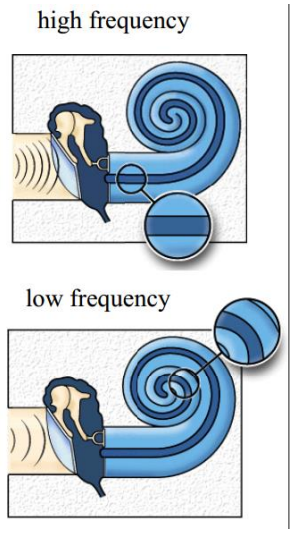


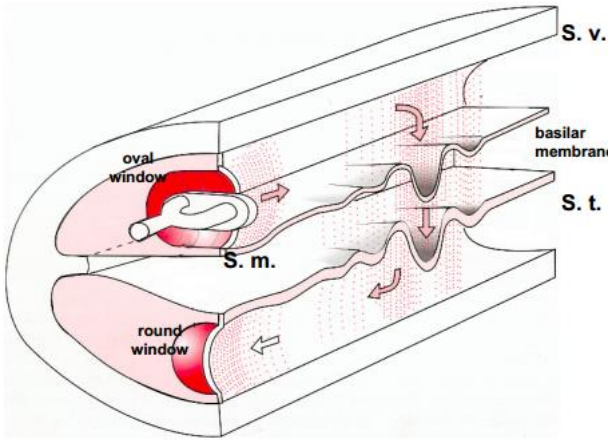


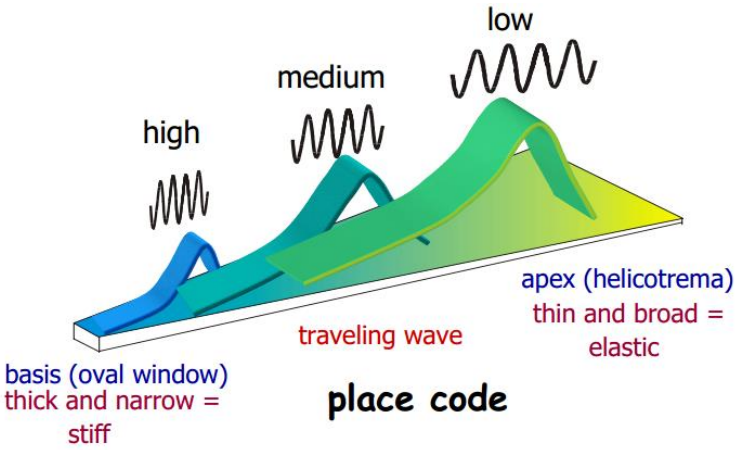
- **Big picture process:** Sound (waves) enter external ear -> focusses the soundwaves into the external auditory canal -> molecules vibrate back and forth -> sound waves strike the ear drum -> ear drum vibrates back and forth -> malleus, incus, stapes vibrate -> oval window vibrates -> in the cochlea (connected via oval window): fluid inside. When the oval window vibrates, the fluid inside the cochlea is going to vibrate, causing a series of vibrations, going to cause a signal in the auditory nerve -> brain
- Harder to get fluid inside the cochlea to vibrate than to get air to vibrate ("acoustic impedance mismatch"): funnel & lever effect help

Excursion 10: Basilar membrane





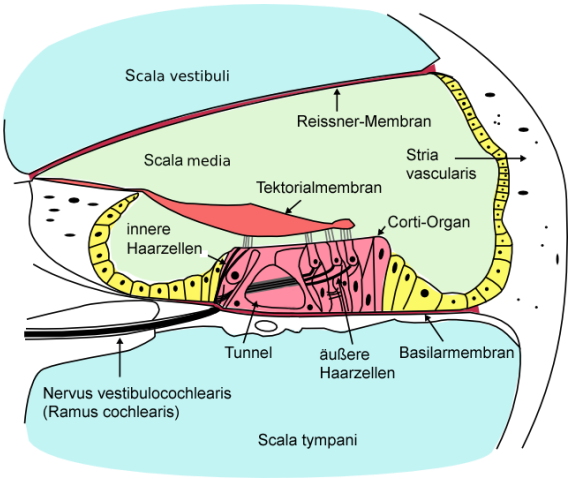


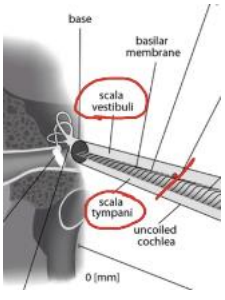


- Cochlea looks like a snail -> roll it out -> see picture
- Thin at base, thick at the apex; Higher frequencies at base, lower frequencies at apex; Easy to move thinner piece of membrane than thicker piece -> greater force needed for lower frequencies

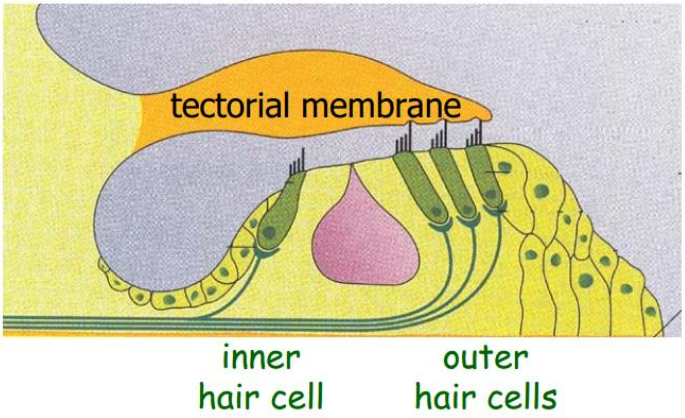
Excursion 11: Visualizations of Cochlea etc.:

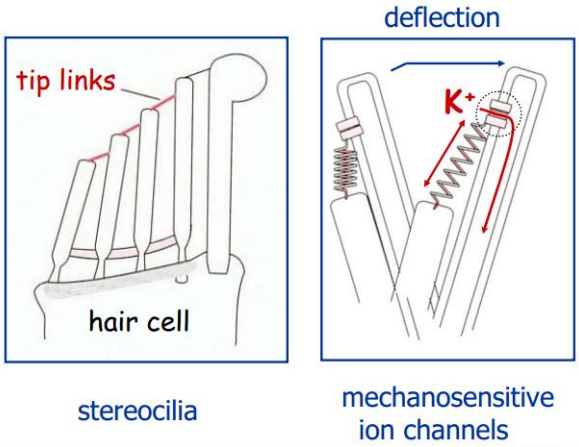
- Entering cochlea through oval window, leaving the cochlea through round window
- Cross section of the cochlea, cut there (see picture at the bottom)
- Basilar membrane up and down -> Organ of Corti up and down -> Tectorial membrane attached only at 1 end -> move in a windshield-wiper-like fashion -> cilia bent -> signal sent to the end ->
- Tectorial membrane causes outer types of the hair cells (cilia) to vibrate -> cilia will bent
- Directly: inner hair cells
- Outer: Help in modulating the response

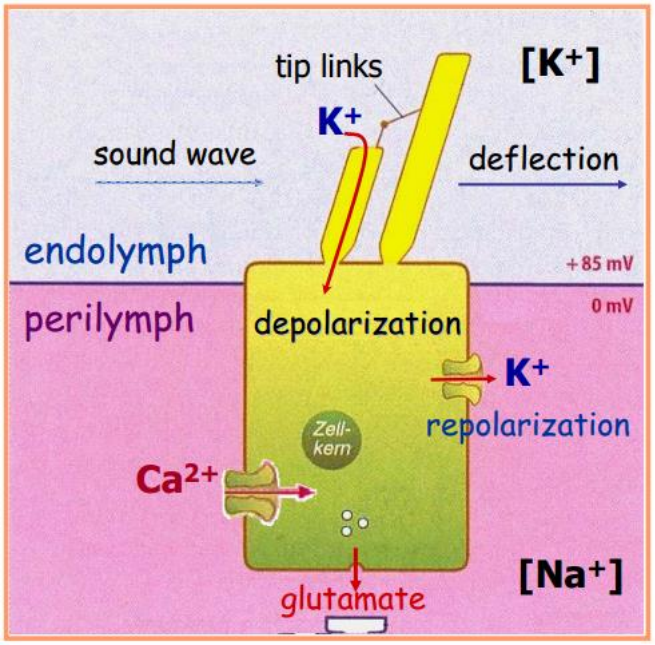




Excursion 12: Hair cells & the process of hearing





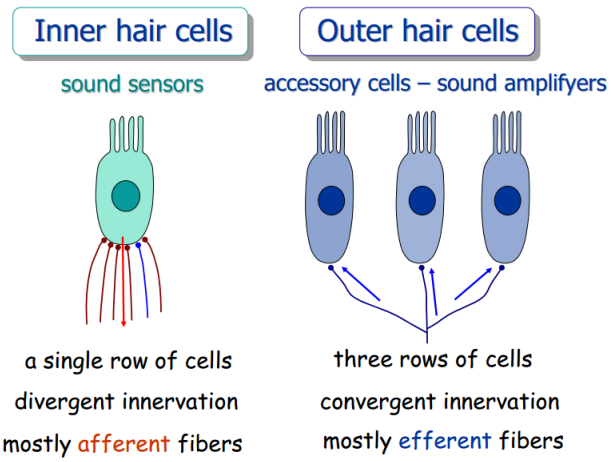


Deflection of hair cells -> opening of mechanically gated cation-channels -> K+-influx -> depolarization of hair cell -> voltage-gated calcium channels open -> Calcium influx -> Neurotransmitter release (Glutamate)

Repolarization by opening of voltage-gated K+-channels

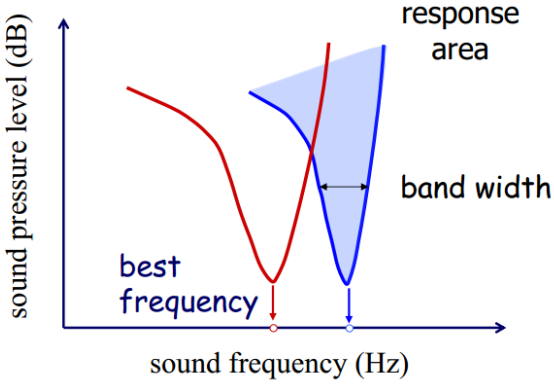
Excursion 13: Cooperation: Inner & outer hair cells:

- Hair cells sit on basilar membrane, covered by tectorial membrane consisting of 'Schleim'
- Hair cells are ABLE TO MODULATE AND AMPLIFY THE SENSITIVITY OF SOUND RECEPTION. How?
- By modifying the mechanical coupling between the inner hair cells and the tectorial membrane. The outer hair cells can contract or relax in response to their sensory input. 1000-fold signal amplification by rhythmic cell contraction (up to 20,000 times/second). Below 60 db, the endolymph stream alone is too weak to stimulate the inner hair cells. Protein prestin in the lateral membrane of outer hair cells acts as a direct voltage to force converter, allowing extremely fast cell contractions.



Excursion 14 – Tuning curves:

- Intuitive understanding of the curves: The blue one only needs a very low sound pressure to reach the threshold that is sufficient to produce a certain change in the membrane potential to say "Nice, I received the signal at frequency x" for frequencies around the "best frequency"-x-position. The amount of pressure needed to register frequencies that are



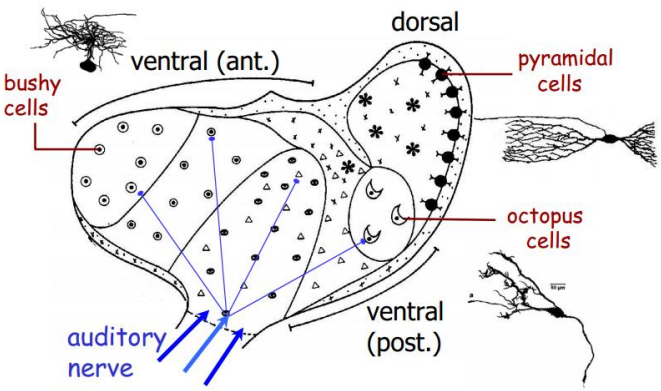
not “best” gradually increases and the slopes of the curves is usually more shallow towards the lower frequencies and steeper towards the higher frequencies. The red thing has a different response property, simply shifted more towards the lower frequencies

- Summary: Different cells have different response properties – those can be captured by tuning curves. These cells always have a best frequency, i.e. there exists a frequency towards which they are particularly sensitive to, i.e. only very little sound pressure is needed to reach the critical threshold, i.e. even very quiet sounds in these frequencies will be detected, whereas to detect higher or lower frequencies, the sound has to be louder in order to be perceived.
- Big picture: Think back of the basilar membrane; the hair cells at the one side are sensitive towards very low, on the other side towards very high frequencies; plotting these 2 extreme response properties would result in tuning curves comparable to the blue and red ones from above (only a bit more extreme)

Excursion 15: Frequency analysis in the auditory nerve

- Sounds <= 1KhZ: Periodicity of bursts reflect the length of a sine wave (phase locked stimulus response); discharge of acoustic fibers are synchronized with the phase of the tonal stimulus
- Sounds > 1KhZ: 1:1-correspondence between sound waves and electrical signals prevented. At these frequencies, not the periodicity of bursts or APs encode the wavelength of a sound but the phase periodicities of several nerve fibers?

Excursion 16: Cochlear nucleus



The Cochlear Nucleus

Channelling of sound features

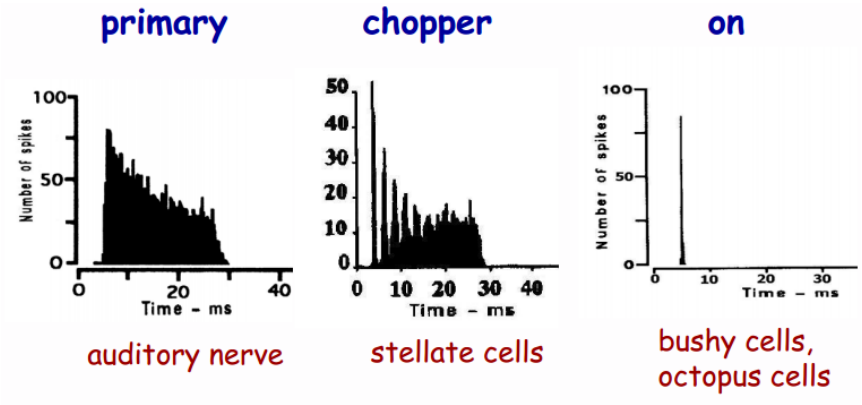
Dorsal part : pyramidal cells

What pathway : detailed sound analysis

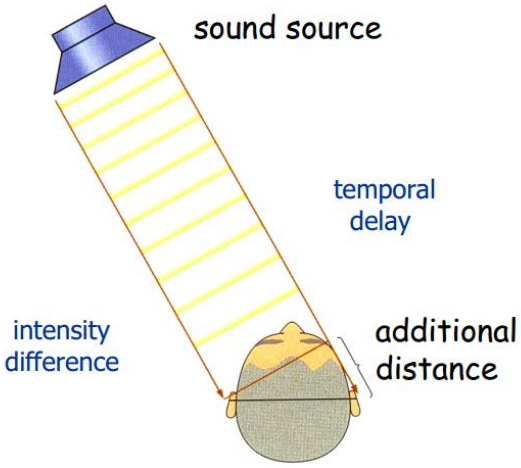
Ventral part : bushy cells, octopus cells

That pathway : prewarning to the inferior colliculus

Where pathway : sound localization in the superior oliva

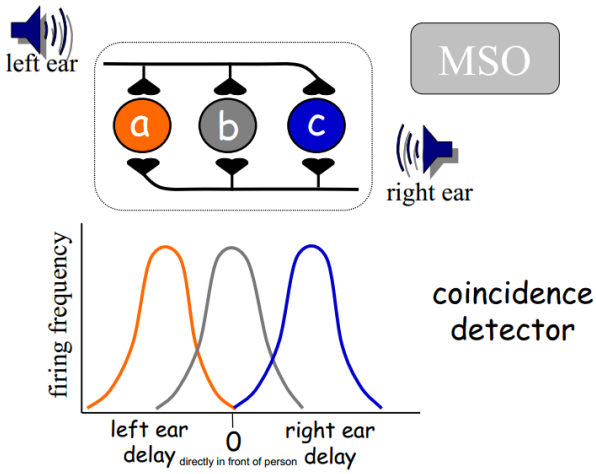


Excursion 17: Sound localization



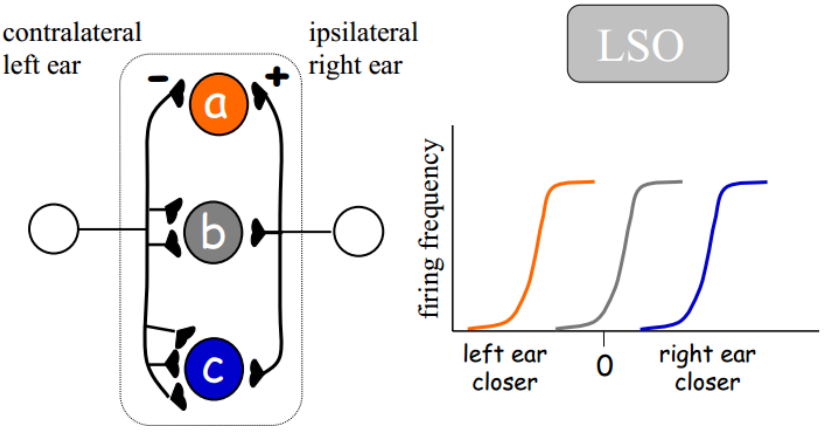
1. MSO (medial superior olivary nucleus):

- 1 to 1.5 KhZ: temporal delay, coincidence detection (EE); minimal perceptible delay = 10 microseconds (1-3 angle)
- How it works: a b and c only fire when they receive input from both sides at the same time. For a, this is the case when there is a left ear delay, for c, when there is a right ear delay, for b if it is perfectly timed and the sound´s origin is directly in front of you. The logic here is that the temporal delays are fully accounted for by the length of the fibers.
- Script "Target neurons with identical best frequencies operate as coincidence detectors using a delay line circuitry: thereby the different relative lengths of axons from the 2 cochlear nuclei to the MSO act as temporal delay lines, that compensate for the delayed arrival of sound waves.

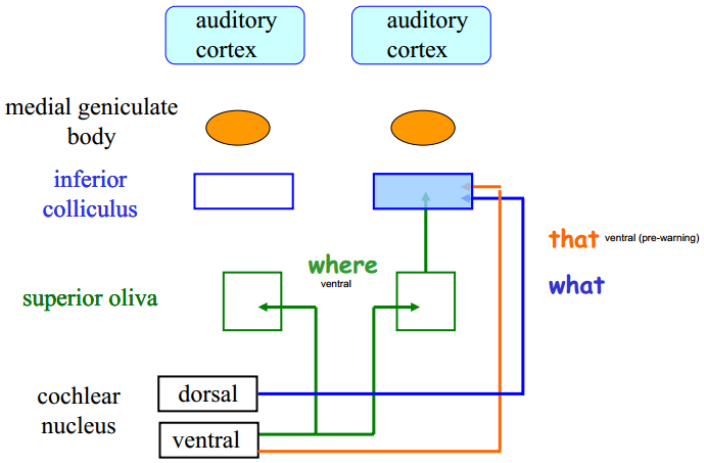


2. LSO (lateral superior olivary nucleus):

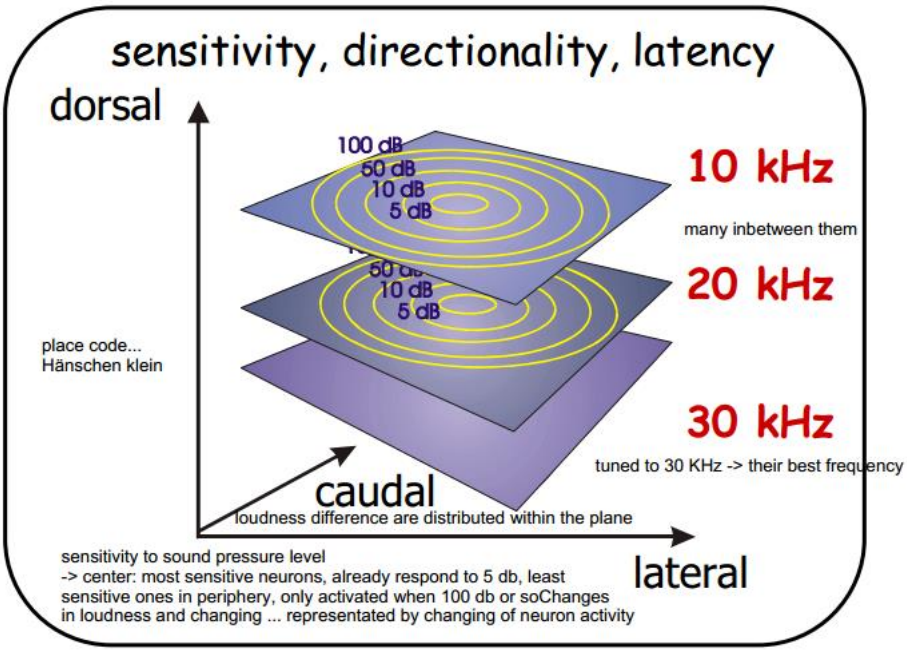
- Above 1.5 KhZ: intensity differences (>= 1db), ipsilateral excitatory input from bush cells of VCN an contralateral inhibitory input (glyice(IE)):
- Script: "Sound pressure differences of sound waves from both ears are used for sound localization. Majority of units are ipsilateral excitatory and contralateral inhibitory, generating a subtractive response. The strength of inhibition varies topographically.



Excursion 19: Paths

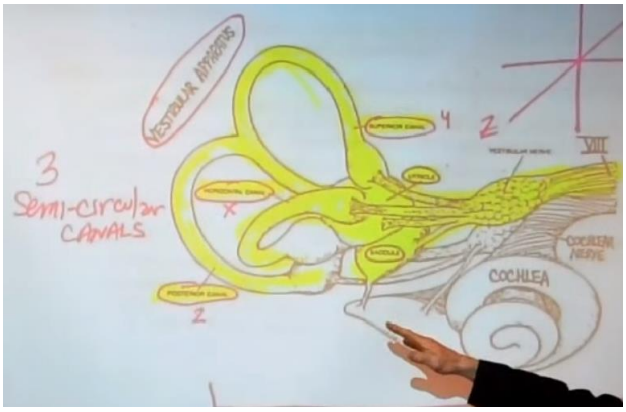


Excursion 20: Inferior colliculus: Isofrequency planes etc.

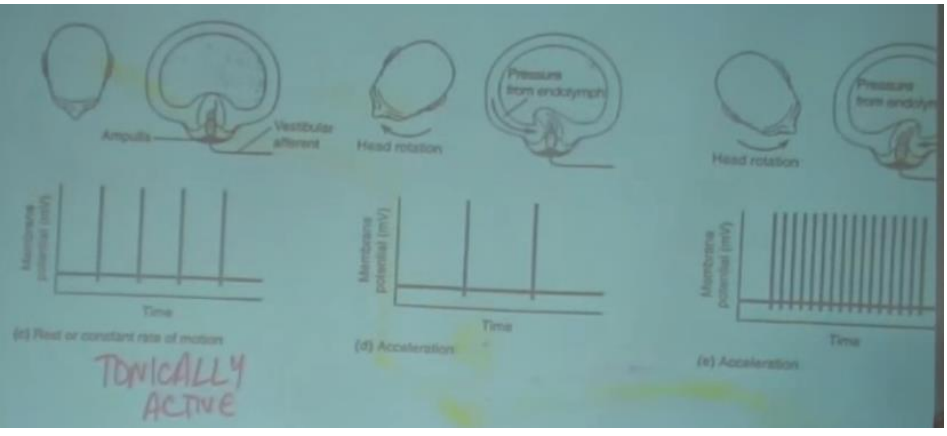


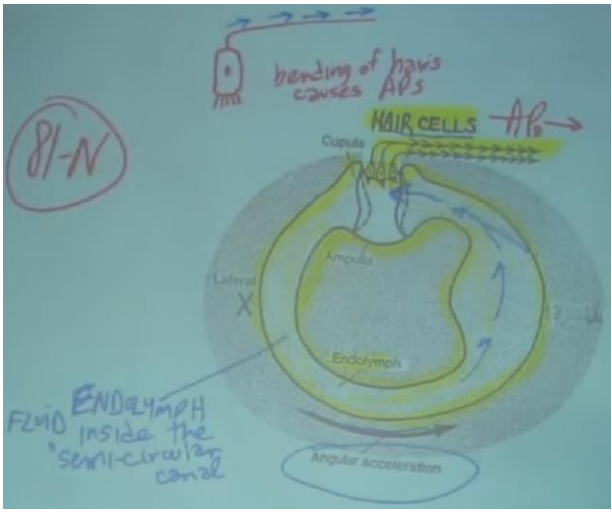
"Die zentralen Bereiche der Colliculi inferiores haben eine tonotope Organisation, wobei die Frequenzachse von dorsal (tiefe Frequenzen) nach ventral (hohe Frequenzen) verläuft."

Excursion 21: The vestibular apparatus for Balance & Equilibrium (Prof. Fink lecture): Semicircular canals:



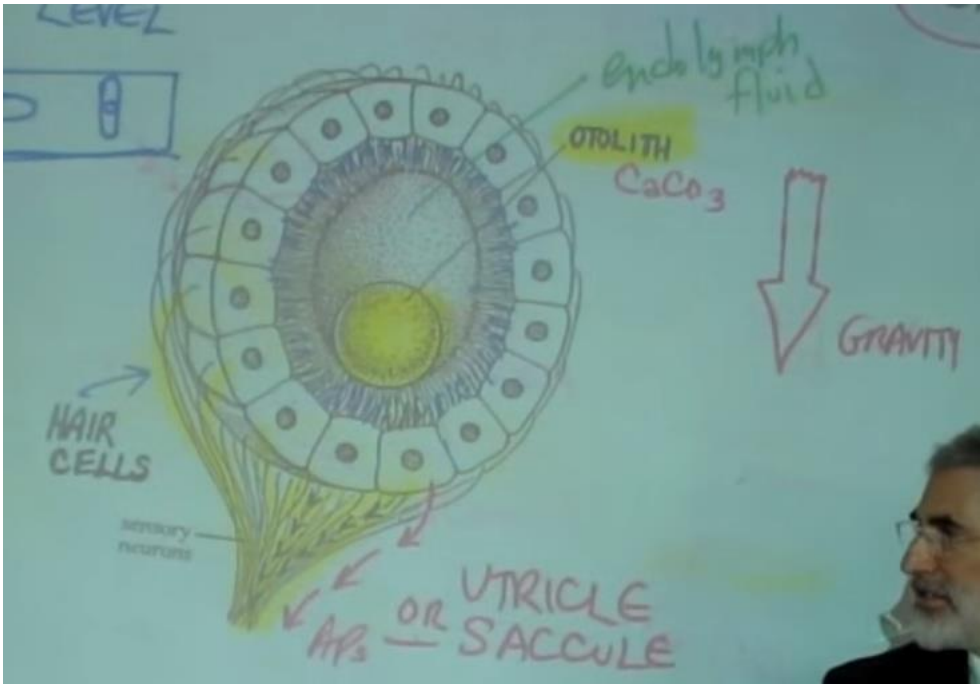
- Oriented in 3 different planes/dimensions; contain endolymph
- Rotational movement of the fluid inside the semicircular canals bends the hairs of the hair cell sensory neurons activating them to send APs to the brain
- Example: turning your head, spinning in a circle, etc.
- If you start spinning in a clockwise direction, this fluid is starting to flow in a clockwise direction; if moving in the opposite direction? How does the brain know in what direction we move?
- Hair cells send APs at constant rate and fire off action potentials at a certain frequency; when rotating the head in 1 direction, the frequency increases but decreases when going into the opposite direction; if stationary: in the middle.



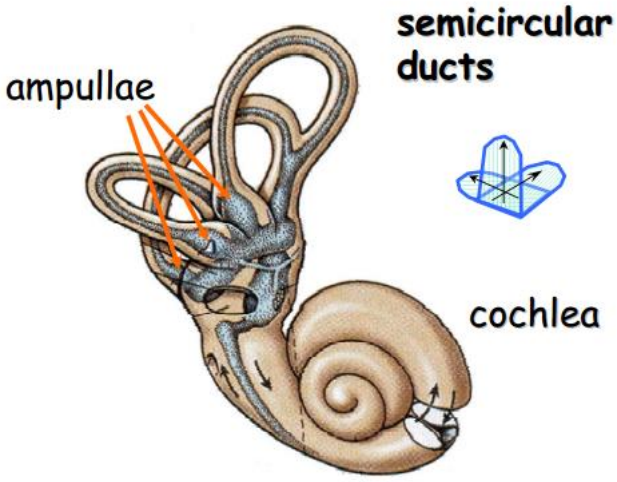


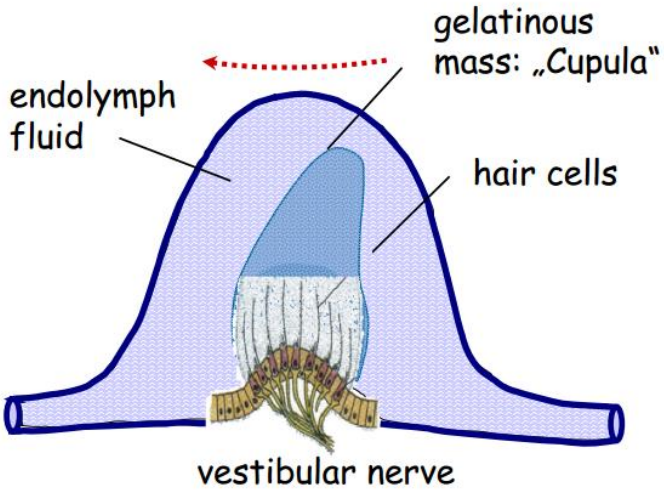
Excursion 22: Utricle and Saccule (Prof. Fink)

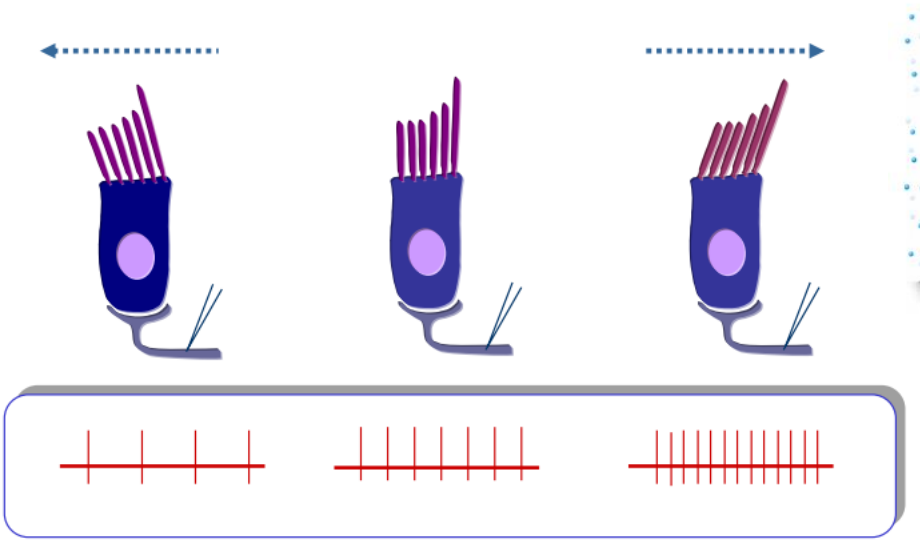
- Oriented horizontally and vertically
- Gravity & linear motion (acceleration of de-acceleration) moves the otoliths inside the utricle and saccule; The otoliths cause bending of the hairs activating the hair cells to send APs to the brain
- Cavity filled with endolymph fluid; gravitational detector
- Gravity pulls things down; rock pulls down bending these hair cells sending ATs to the brain
- Turn you upside down; rock goes up, those hair cells bent,

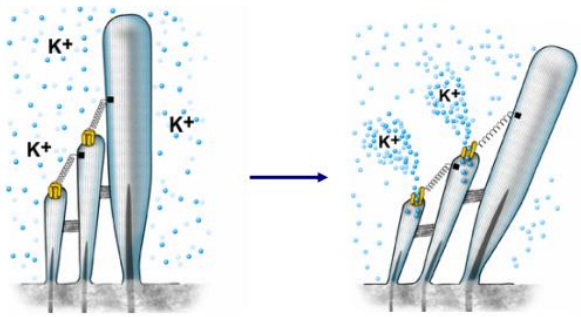


Excursion 23: Sense of Balance now again from Gunnar´s point of view:





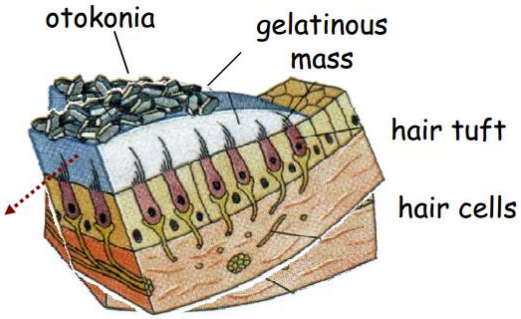




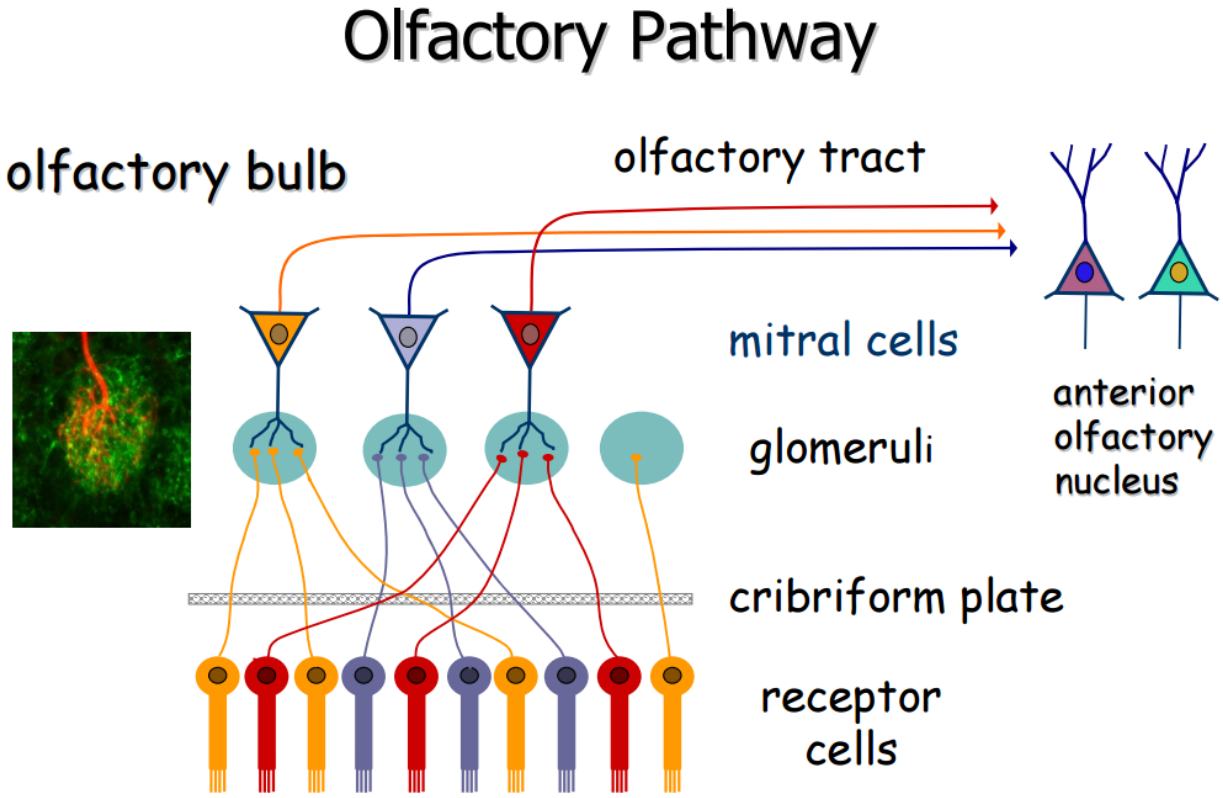
- Semicircular ducts = 3 tubes filled with endolymph; perpendicular to each other
- Base of the ducts: widen into ampulla (see picture 1)
- Wall of ampulla: Patch of sensor cells. Hairs extend into gel. mass (=cupula), which extends into endolymph
- Swinging door principle: Head movement -> Semicircular duct rotated -> Cupula tilted against endolymph fluid (fluid remains stationary due to physical inertia) -> Deflection of hairs -> mechanically-gated ion channels open.
- Longer time: Endolymph fluid not stationary anymore but starts to rotate at the same speed -> Cupula back in resting position. Sudden stop -> Cupula bent into opposite direction -> dizziness
- Directional sensitivity (see picture 3,4): movement of hair bundle towards the longest hair (kinocilium) -> depolarization; opposite direction -> hyperpolarization. Changes in membrane potential due to opening/closing of mechano-sensitive ion channels in the hair membrane

Macula organs (Utrculus: horizontal, Sacculus: vertical):

- Harbor sense of gravity + linear acceleration
- Hair cell covered by gel. mass into which OTOKONIA embedded (near hair-tips)
- Sudden movement -> otokonia lag behind -> deflect hair into opposite direction
- Rest: Otokonia follow gravitation



Excursion 24: Olfactory Pathway



Sensory Physiology Survival Guide: Questions (sorted by category)

Vision

1. Welche Aussagen zum primären visuellen Cortex der Säuger sind vollständig richtig?

XO Orientierungsempfindliche Neuronen besitzen balkenfömige rezeptive Felder (Balken & V1 immer gut – aber VORSICHT wenn es nicht um V1 sondern CGL o.ä. geht. Übrigens: Das mit den Balken liegt an der Konvergenz von stellate cells aus layer 4 auf pyramidal cells in layer 5, weil die receptive fields von stellate l4 überlappen und dann die epsp´s aufsummiert werden)

XO Die starke Rückprojektion zum CGL stammt aus Schicht 6 (ja, CGL -> s4 -> p5 -> p6 -> some: CGL back)

FO Der primäre visuelle Cortex ist im Temporallappen lokalisiert (nope, occipital lobe)

XO Der primäre visuelle Cortex ist aus 6 unterschiedlichen Schichten aufgebaut (ja, alle)

FO Die Fasern aus dem CLG terminieren auf Pyramidenzellen in Schicht 4 (nein, STELLATE cells in layer 4 synapse onto pyramidal cells in layer 5)

FO Im primären visuellen Cortex treten keine farbsensitiven Neuronen auf (Doch, blobs in V1: layer 2 und 3)

2. Welche Angaben zu den Photorezeptoren der menschlichen Retina sind komplett richtig?

Xo Stäbchen werden bei Dunkelheit depolarisiert. (dark current: Stäbchen=rods)

Fo In der Retina treten sehr viel mehr Zapfen als Stäbchen auf. (umgekehrt, 6 vs 120 Mio.)

Xo Zapfen sind farbempfindlich, weil sie unterschiedliche Opsine besitzen. (genau, Opsine sind die Basis von color vision)

Fo Stäbchen sind über **off**-Bipolarzellen mit amakrinen Zellen verbunden. (Genau lesen, rods: **nur** on-bipolar)

Xo Zapfen sind entweder mit off- oder on-Bipolarzellen verknüft.

Fo Stäbchen sind lichtempfindlicher als Zapfen; sie treten vor allem im Zentrum (Fovea) auf. (1. Häfte richtig, aber rods treten gehäuft in der periphery auf)

Fo you can get lazer vision, if you remove the retina and replace it by lazers (es geht offenbar nicht um diese Laserbehandlung, sondern die Vorstellung mit Lasern zu sehen -> Science-Fiction-Trickfrage)

Xo on-bipolar cells possess metabotropic glutamate receptors, evoking an epsp when photoreceptors are illuminated (Genau. Die Reaktion von den Photorezeptoren auf Licht ist ja erstmal negativ. Damit wir on-bipolar cells haben, müssen wir die Logik quasi invertieren. Um das zu erreichen, brauchen wir inhibitory shit. Welchen inhibitoren shit? Glutamat -> metabotropic glutamate)

3. Welche Aussagen über das Corpus geniculatum laterale (CGL) des Menschen sind komplett richtig?

Fo **β**-Retinaganglienzellen terminieren in den Schichten 1 und 2 (ne in 3,4,5,6)

Xo Zielneuronen im CGL besitzen **konzentrische** rezeptive Felder (ja, immer noch konzentrische Felder)

Fo das CGL ist Bestandteil des Mesencephalons (diencephalon)

Xo ipsilaterale retinale Fasern terminieren in den Schichten 2, 3 und 5 (Jeserichs „donkey bridge“: KIIKIK – I´s sind an den Stellen 2,3,5 -> stimmt also)

Fo Das CGL ist **retinotop** strukturiert, wobei die **peripheren** Retinabezirke überrepräsentiert sind (Peripherie unterrepräsentiert, Fovea überrepräsentiert

Xo im CGL ist noch kein rämliches Sehen möglich (genau, die Augeninputs sind noch getrennt, ne?)

4. Wie reagieren off-center Retinaganglienzellen bei punktfömiger Belichtung

a) des Zentrums: A decrease in firing frequency of RCG is observed.

b) der Peripherie der rezeptiven Felder: An increase in firing frequency of RCG is observed.

c) Wie läst sich dieses Verhalten mit der zelluläen Verschaltung innerhalb der Retina erklären?
Lateral inhibition by horizontal cells

5. Welche Darstellung über den Verlauf der Sehbahn des Menschen ist richtig?
…

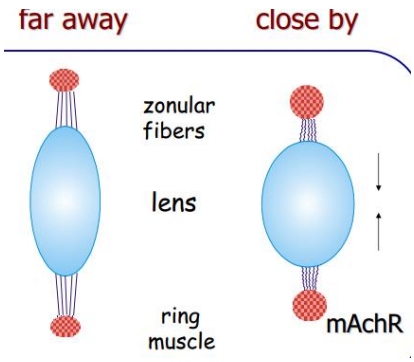
6.1. Welche beiden Theorien des Farbsehens kennen Sie?
A: Trichromacy theory : 3 primary colors (red green blue) are sufficient to produce all colors; 3 types of cones with different wavelength sensitivity; additive color mixture.
B: Color opponency theory : after images; color opponent; ganglion cells
6.1.2. Durch welche Verschaltung in der Retina kommt es zur Entstehung blau-gelb antagonistischer Retinaganglienzellen?
blue + (green +red)) -> blue/yellow opponency

6.2. Welche 3 Typen von Retinaganglienzellen (a,b,c) kennen Sie? Worauf sind diese jeweils funktionell spezialisiert?
a) alpha-cells/M-cells. Function: detect coarse structures/shape (+ movement of objects)
b) betha-cells/P-cells. Function: fine detail analyse & color vision
c) gamma-cells. Function: motion/direction

6.3. Nennen Sie 4 Typen von Interneuronen in der Retina. Warum bilden diese Zellen keine Aktionspotentiale? --> they lack in axons and therefore cannot generate **action** potential
-bipolar-cells (off-bipolar, on-bipolar)
-photoreceptors
-amacrine cells
-horizontal cells

7. Wie funktioniert die Fernakkomodation der Linse, wie die Naheinstellung (Skizze)

Far accommodation: Ring muscle relaxes (-) -> Lens stretched. (flattened shape) by ciliary fibers (+)
- Near accommodation: Ring muscle (+) -> ciliary fibers (-) -> Lens to resting state with high refractory power
- Imagine: ring muscle contracts -> gets smaller ->fibers are stretched -> make lens very flat -> far accommodation, v.v.



8. Which statement about the human visual system are completely correct?
Xo pattern recognition in the primary visual cortex(V1) relies in principle of **convergent** wiring. Thus target neurons possess bar-shaped receptive fields (after long searches we decided it's right! Convergence in context of bar-shaped receptive field for simple cells in slides)
FO Movement detection occurs in cortical field V4 (No! It's not that clear what it is for (V4). Probably: V5 (= MT) is responsible for movement detection stuff)
XO **colour sensitive neurons reside in blobs** of layers two and three (genau)
FO **Object localisation** is performed in the temporal cortex. It is conneced via the dorsal **where pathway** with the visual cortex. (No, w**hat**-pathway!)
FO The human visual cortex consists of **eight** cellular layers. Visual fibers terminate in layer four (No! It's

composed of 6 cellular layers(like all other as all other cortical fields)! Second statement is correct)
XO In the parietal cortex a multimodal integration of sensory information takes place. (Seems to be right, where path)

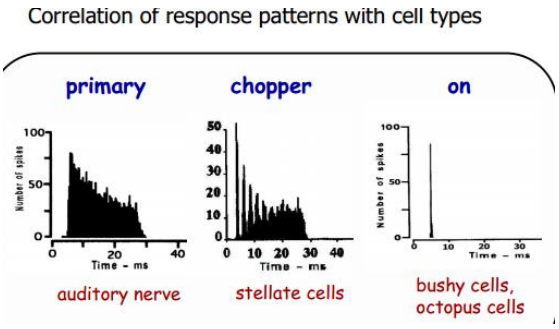
9. What is the function of the superior colliculus? In which brain region is it localized?
- Efferent fibers from the colliculus superior indirectly innervate the three pairs of eye muscles which control the position of the eye ball and hence the position of the visual field. (direction of gaze, nystagmus to keep the visual field constant, visual field selection).
- Multimodal interface
- General spatial tasks?
- located in midbrain (mesencephalon)

10. Which statement about the human laterale geniculate body (LGB) are completely correct?
FO the LGB is part of the mesencephalon (it´s part of the thalamus, which is part of DIencephalon)
FO B-retinal ganglion cells terminate in layers 1 and 2 (no, they connect with the top four parvocellular layers , 1 and 2 are connected with alpha cells (me: easy to remember: there is a parvo and a magno … pathway. As we know, parvo 4 layers in total and magno 2layers: beta – parvo, alpha – magno. Therefore, 1 and 2 -> alpha and 3,4,5,6 beta)

Fragenkatalog – Hearing

1. Welche Mechanismen der Signalverstäkung gibt es im Mittelohr?
Funnel-effect(x17): size reduction from eardrum to oval window
Lever-effect (x1.3): hammer, anvil + stirrup [malleus, incus, stapes]
All in all x22
1.1. Where does the amplification take place?
1.2. At which side does a signal amplification occur?[weird, what do you want from me?]

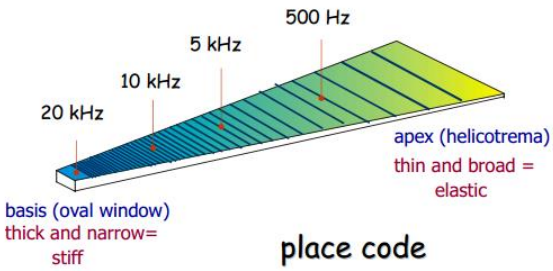
2. Welche Funktionen hat der Nucleus cochlearis? In welcher Hirnregion liegt er?
The nucleus cochlearis is to be found in the medulla oblongata in the brainstem.
Due to the heterogeneous population of second order neurons in the nucleus cochlearis sound signals are transformed.
A) bushy cells and octopus cells in the ventral region often generate an “on-response” maintaining a high temporal precision even at higher sound frequencies.
B) stellate cells of the ventral part often show a “chopper *response”,* reflecting rhythmicity of
sounds.
C) pyramidal cells in the dorsal part typically exhibit “build-up” or “pauser *responses”*
providing for a differentiation of the onset and ensuing phases of responses.



Each auditory nerve fiber splits into numerous branches and has multiple representations in the cochlear nucleus thereby projecting onto various cell types.
Thus a conversion of the input signal into various different output responses takes place. (each being an abstraction of one particular feature of the auditory input signal).

3. Wie sind unterschiedliche Schallfrequenzen auf der Basilarmembran der Cochlea repräentiert?

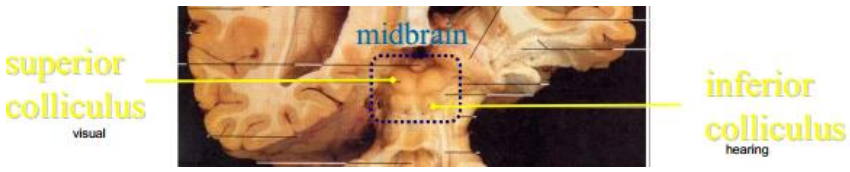
The basilar membrane is more thick, stiff and narrow at the base and gets more elastic, broad and thin at the apex.
Different frequencies of sound provide a different mechanism for selective response of hair cells.
High frequencies cause great vibration at the base of the cochlea, while low frequencies cause maximal deflection of hair cells in the apex of the cochlea. This means low frequencies move as traveling waves along the whole basilar membrane.

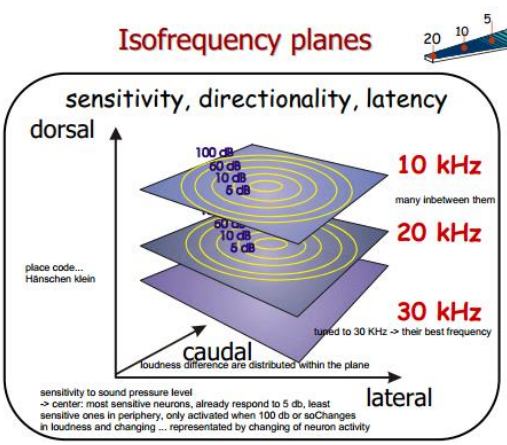


4. Welche Beziehungen gibt es zwischen Lautstärke- und Frequenzwahrnehmung in der menschlichen Hörkurve?

Tones of the same intensity, but of different frequency, are perceived as being of different loudness.
For sounds near 3000 to 4000 Hertz, the ear is extra-sensitive; these sounds are perceived as being louder than a 1000 Hertz sound of the same intensity.
At frequencies lower than 300 Hertz, the ear becomes less sensitive; sounds with this frequency are perceived as being less loud than a sound of the same intensity and 1000 Hertz frequency. The loss of sensitivity gets bigger as one goes to lower frequencies.

5. Welche Funktion(en) hat der Colliculus inferior? In welcher Hirnregion liegt er?





The inferior colliculus in the midbrain performs simple auditory processing without memory formation. It lies in the middle of bottom-up and top-down processing of the auditory system.
It has an onion-bulb pattern of isofrequency planes (tonotopy) that are piled up upon each other in discrete cellular layers.
A plane corresponds to a place on the basilar membrane. Within each plane target neurons are arranged in isophone lines.
Neurons with highest sensitivity are found in the center; those with lower sensitivity reside in the periphery.

6. Erlätern Sie die Reizaufnahme und Signaltransduktion in einer Haarzelle des menschlichen Innenohrs (evtl. mit Skizze).

When a sound hits the basilar membrane, the stereocilia of the hair cells get deflected.
If the sound stimulus is below 60dB, first only the outer hair cells get depolarized, start to contract thanks to pristine which is a voltage to force-converter (but with its 12 transmembrane segments is similar to a transporter protein) and make the tectorial membrane vibrate.
This way the stereocilia of the inner hair cells also get deflected.

This deflection causes the tiplinks via which the stereocilia of a hair cell are connected to stretch and this stretching causes mechanically-gated K+-channels to open.
Because this happens in the Scala media which is filled with endolymph which is rich in K+, K+ ions can stream into the hair cell.
This causes a depolarization which activates voltage-gated Ca2+ channels.
When they open, Ca2+ streams into the cell and exocytosis of glutamate is initiated.
This further depolarization caused through Ca2+ influx also opens K+-channels, such that K+ can flow out of the cell into the perilymph.
Sound hit basilar membrane -> stereocilica deflection Inner hair deflection -> tiplink stretch -> mechanically gated K+ channels open -> K+ influx into hair cell -> depolarization -> Ca2+ channels open -> Ca2+ influx ; exocytisis of glutamate -> further depolarization -> K+ channels from the inside open -> K+ efflux to the perilymph -> repolarization b) Warum ist diese Art der Signaltransduktion fü das Hören besser geeignet als eine enzymatische Signaltransduktion wie sie bei Photorezeptoren vorkommt?

6. English version: Explain the molecular processes underlying olfactory reception
1000 different odorant genes. odorant cells/ receptors = seven- transmembrane domain proteins and they're Gprotein coupled
Binding of odorant: G-protein dissociates --> activation of adenyly cyclase --> cAMP production--> cAMP opens CNG channels --> Ca2+ influx --> depolarization + opening of Cl− channels --> chloride efflux (further depolarization + therefore amplification) terminates as cAMP blocks further G-protein activation + is degraded by phosphodiesterase

7. Welche Eigenschaften sind charakteristisch für die inneren Haarzellen des Innenohrs?
Fo Sie stehen mit einer Vielzahl efferenter Axone in Kontakt (me: eff???, Axone???)
Xo Sie besitzen mechano-sensitive Ionenkanäe.
Xo Sie sind primär für die Schallaufnahme verantwortlich
Fo Sie können rhythmisch kontrahieren.
Fo Sie bilden drei parallele Zellreihen. (me: outer hair cells)
Fo Sie haben eine hohe Regenerationsfäigkeit.
Fo Sie stehen in direktem Kontakt mit der Tectorialmembran.

8. In which two brainstem centres is binaural sound localization taking place? Which sound feature are used for that in each case?
-The superior olivary complex consists of the medial superior oliva(MSO) and lateral superior oliva (LSO). The MSO takes low frequencies up to 1.5 kHz, and can detect differences of only 10 microseconds in the arrival of signals.
-LSO takes sound pressure differences (in the horizontal plane down to 1dB discrimination)
The main units are ipsilateral and contralateral inhibitory, generating a subtractive response. The strength of inhibition varies topographically

9. Which statment about human hearing are completely correct?
FO in the auditory nerve sound pitch is encoded by the frequency, while loudness is encoded by the amplitude of actions potentials
XO the axons of the auditory nerve terminate in the cochlear nucleus. Their cell bodies reside the spiral ganglia.

XO High frequencies are heard at the beginning of the basilar membrane, lower frequencies at the end (helicotrema).
XO High frequencies cause the greatest vibration at the base of the cochlea
XO low frequencies maximally in the apex, very low frequencys move as traveling waves along the whole length of the basilar membrane
XO Sound waves enter the scala vestibuli via the oval window. The scala vestibuli is filled with perilymph fluid
FO inner hair cells of the cochlea are in contact with several axons, in order to widen the spectrum of frequencies in the auditory nerve. (meiner meinung nach falsch)
FO Sound amplification in the middle ear is primarily accomplished by a lever effect mediated by three auditory ossicles(bones). (It's just partially (by a factor of 1,3) the primary amplification is done by the size difference of the eardrum and the oval window(factor 17))

10. Which relationship exists between loudness and frequency in the human hearing curve?
Higher frequencies are perceived louder
higher frequency → higher tone
higher amplitude → higher loudness

11. Why is this type of signal transduction more appropriate for hearing than an enzymatic signalling cascade as found in photoreceptors?
– it is faster

12. What is a tuning curve? Explain it with a sketch --> ?

Fragenkatalog – Taste and Smell

1. Welche Nerven sind an der Übertragung von Geschmacksreizen beteiligt? In welche primären Zielgebiete im ZNS projizieren sie?
The nerves which transmit taste stimuli are:
- facial nerve (7th cranial nerve, anterior two thirds of tongue)
- glossopharyngeal nerve (9th cranial nerve, also touch and temperature sensitivity)
- vagus nerve (10th, innervate the posterior third and the pharynx) Information is send over the Nucleus solitarius in the brainstem (medulla oblongata) to either
The Goal-areas are:
- thalamus and temporal cortex (close to the somatosensory fields of the face)
- to the amygdale and the hypothalamus (limbic system, emotional aspect of taste).

2. Welche Gemeinsamkeiten besitzen olfaktorische Rezeptorzellen, die in dasselbe Glomerulum projizieren?
- They all posses the same odorant receptor melocules b) Wie heißen deren Zielneuronen im Bulbus olfactorius?
- They are called mitral cells

3. Beschreiben sie die molekularen Prozesse der Signaltransduktion olfaktorischer Sinneszellen.
There are1000 different odorant genes.
Odorant sensory cell are 7 transmembrane domain proteins and Gprotein coupled

→ disassociation of G- Protein complex.alpha segment binds to adenylyl cyclase
--> cAMP production (level increase)
→ cAMP opens CNG channels
--> Ca2+-influx --> depolarization opening of Ca2+-gated Cl- -channels
--> chloride efflux
--> further depolarization
amplification of other channels --> feed forward

4. Welche Aussagen üer Geschmacksrezeptorzellen sind richtig?
XO sie haben eine kurze Lebensdauer und werden ständig erneuert (so jede Woche)
FO es handelt sich um primäre Sinneszellen (sekundäre)
FO sie reagieren jeweils selektiv auf Geschmacksqualitäen (though they are most effective to a single stimulus, also respond to several)
XO Geschmacksknospen enthalten unterschiedliche Rezeptorenzelltypen (25☺)
FO die Rezeptormoleküe sind auf Cilien lokalisiert (microvelli carry receptor molecules (last slide last lecture))
FO gustatorische Rezeptoren sind stets G-Protein gekoppelt (bitter and sweet but not the others)

5. Shortly explain the molecular processes underlying olfactory reception
Odorant receptors are seven transmembrane domain proteins g-coupled.
Binding of a odorant causes dissociation of the G-protein,--> activation of adenylyl cyclase
→ cAMP level increases CNG-channel -->Membrane depolarization, Ca2+ opens chloride--> Membrandepolarisation++

6. Which statement about the human chemical senses are completely correct?
FO nerve fibers of the taste pathway directly reach the cortex, without contacting the thalamus before
XO olfactory information is encoded by a population code, each receptor cell responding to a particular structure within an odorant substance
FO taste receptors are primary sensory cells. They produce action potentials themselves
XO smell receptor cells have a high capacity for regeneration. Their receptor molecules are localized on the cilia
FO Mitral cells are the target neurons of olfactory receptor cells. They integrate the smell signals of different receptor cell types (false because its the same cell receptor type!)

Fragenkatalog – The rest: General sensory processing, Brain, Cerebellum, Hemispheres, Proprioception, etc.

1. Welche beiden Hauptfaserprojektionen zu den Purkinjezellen im Cerebellum kennen Sie? Welche der beiden dient der Korrektur eines senso-motorischen Ungleichgewichts?
- The cerebellum exerts movement control by comparing afferent information with efferent signals from the motor cortex. The two pathways sending, signals to the Purkinje cells, are climbing fibers and parallel fibers.
- Climbing fibers are the one which send out signals via the Purkinje cell axons, which have GABAergic nerve terminals, to correct the sensory-motor mismatch.

2. Linke und Rechte Hemisphäe des menschlichen Cortex zeigen eine charakteristische funktionelle Spezialisierung. Welche Arten kognitiver Leistungen werden bevorzugt in der *rechten* linke Häfte prozessiert?
o Rechnen
Xo Musik höen
o Sprechen
o prozedurale Verarbeitung
o Schreiben, Lesen
Xo geometrische Musteranalyse
• The right hemisphere turned out to be specialized for spatial construction, geometrical pattern analysis, nonverbal ideation, emotional touch, music perception (collectively designated *declarational processing).*
• The left hemisphere turned out to be specialized for language analysis, writing and reading, doing calculations (collectively designated *procedural processing).*

3. Welche Funktion haben gamma -Motoneuronen?
γ-Motoneurons regulate the gain of the stretch reflex by adjusting the level of tension in the intrafusal muscle fibers of the muscle spindle. This mechanism sets the baseline level of activity in α-motoneurons and helps to regulate muscle length and tone.

4. Worin unterscheiden sich primäre von sekundären Sinneszellen?
Primary sensory cells have axons and can generate action potentials.
Secondary sensory cells do not have axon and hence cannot generate action potentials. They transmit signals through their cell body.

4.2. Welche der untenstehenden Zelltypen sind sekundäre Sinneszellen?
Xo Geschmacksrezeptoren
o Geruchsrezeptoren
Xo innere Haarzellen
o amakrine Zellen

5. Name the most relevant target of y-motoneuron axons!
– Muscle spindle

6.Which statements about the cerebellum are completely correct?
FO parallel fibers are the axons of granule cells. They make multiple en passant synapses with a single purkinje cell dendritic tree
XO climbing fibers send out a correcting signal in the case of sensory-motor missmatch
XO the cerebellar cortex is composed of six cellular layers
FO longterm depression is involved in motor learning in the cerebellum. On a molecular level LTD is induced by the elevation of intracellular cAMP in purkinje cell dendrites. – LTD is involved but not via cAMP
FO Purkinje cells are the major output source of the cerebellum. They use glutamate as neurotransmitter. – They've GABA neurotransmitter
XO The cerebellum is a major target area to integrate vestibular and proprioceptive signals