Our educational modules are made for providing information on how the hearing organ works and which test procedures are used to test the functionality of the sound processing elements along the auditory pathway.

With that knowledge the user of our devices should be prepared to use our test modules efficiently.
We have six educational modules

0 Sound
1 Physiology/Pathophysiology of Hearing
2 Audiology (hearing assessment)
3 Hearing Screening (newborn, pre-school/school children, elderly)
4 Tracking
5 Occupational medicine
What is sound?

You can hear it, but you can not see it!
What is sound?

Sound are audible motions of molecules in gaseous, liquid and solid mediums.

The hearing organ is able to percept sound

- via air, that sets the tympanic membrane and the middle ear ossicles in motion and successively the basilar membrane with the auditory sensory cells on it which are embedded into the cochlear fluids

- via bone, that directly sets the cochlear fluids in motion
Physical measures that characterize the sound are:

- sound pressure $p$ [Pa]
- sound pressure level $L$ [dB SPL]
- frequency $f$ [Hz]
- sound velocity $c$ [m/second] depending on the density of the medium

time- $p(t)$, $L(t)$, spectral-function $p(f)$, $L(f)$
Sound pressure $p \text{ [Pa]}$

Sound pressure level $L \text{ [dBSPL]}$

$L \text{ [dBSPL]} = 20 \log \frac{p}{p_0}$

$p \text{ [Pa]} = 1 \text{ N/m}^2$

$p_0 = 20 \times 10^{-6}$
The human hearing organ has a huge dynamic range that covers audible sounds from the hearing threshold with a sound pressure of $p_0=20 \times 10^{-6}$ Pa (at 4 kHz) to the threshold of pain with a sound pressure of $p=20$ Pa. Sound pressure is then one million times higher. For providing a more handy measure, sound pressure level $L$ instead of sound pressure $p$ is used: $L=20\log_{10}\left(\frac{p}{p_0}\right)$. Swinging leaves driven by a tidy wind produce a sound pressure level of 20 dB SPL. When you are sitting in a bath and you move your hands slightly in the water the sound pressure level will be 40 dB SPL. When you speak to your wife the sound pressure level you are producing at a distance of 1 meter to her will be 60 dB SPL. Cars, when carefully driven, emit sounds with a sound pressure level of about 80 dB SPL. A starting air craft produces a sound pressure level of more than 120 dB SPL. In factories sound pressure levels of up to 120 dB SPL can be present. Therefore, factory workers are obliged to wear sound protectors.
Molecules in gaseous, liquid and solid mediums swing around their center.

Displacement $s$ of the particle depends on the pressure $p$ that drives it.
Silence

When there is silence, molecules do not move.
When there is sound, molecules do move.
Sound frequency = Oscillations of the molecules per second

frequency \( f = \frac{1}{T} \) [1/s=Hz]
Molecules, when set into vibration by sound create zones in space where molecules are close to each other (condensation) or apart (rarefaction). Number of these oscillations per second is given by frequency $f$. Cycle period $T$ is the time distance between zones of rarefaction and condensation. Frequency and cycle time are connected by

$$f = \frac{1}{T} \text{ [1/second] or [Hz]}.$$
Sound spectrum: two tones (red, blue) and a transient (green)

Lines indicate frequency (x-axis) and pressure (y-axis) of the tones. Transient stimulus has a broad spectrum.
A sinusoidal signal (a tone) is presented in a sound spectrum as a line. Its place at the x-axis indicates its frequency $f$. Its length on the y-axis gives the sound pressure $p$. In our figure, the pressure of the 1-kHz tone is twice of the pressure of the 2-kHz tone. A transient stimulus (e.g. a click) has a broad spectrum. Due to the transfer characteristic of the electro-acoustic transducer used (head-phones, loud-speaker) its band-width is limited. Due to mass and stiffness, low and high frequency tones are transferred with minor amplitude. The higher the quality of the transducer the broader the bandwidth. For audiology purposes, transducer quality has to be high to guaranty transfer of defined sound pressure at all test-frequencies.
Overview on how the hearing organ’s work works

- Outer ear function/Middle ear function
- Cochlea function
- Neural sound processing and perception
Before talking about the different methods for assessing the functionality of hearing on the different stages of the auditory pathway we will give an overview on the hearing organ’s work. In the following, outer ear and middle ear function (air-conduction), cochlear function (air-/bone-conduction), and neural sound processing and perception will be addressed. First of all, we look at what is happening in the cochlea, when the ear is stimulated by tones (e.g. 2 kHz and 4 kHz) or a transient stimulus (click). After that, we learn about the performance of the human hearing organ with respect to (i) the sound pressure range the ear is able to cover, (ii) the lowest and highest tone frequency the ear is able to hear, and (iii) frequency selectivity.
Tones generate travelling waves on the basilar membrane. High frequency tones lead to displacement near cochlear basis, low frequency tones near cochlear apex. The physicist Georg von Békésy was the first to observe - by means of stroboscopy - that travelling waves are generated on the basilar membrane. Thus, Hermann von Helmholtz’ theory, that tones of different frequency lead to a stimulation at different sites in the cochlea (Einortstheorie) was confirmed. However, Georg von Békésy could show that the cochlea is managing this sound frequency/cochlear-place-transformation not by standing waves but by travelling waves. For this observation Békésy got the Nobel price in medicine in 1961.
Basilar membrane vibration

Travelling wave envelopes on the basilar membrane for 1 kHz-tone, 2 kHz-Tone and transient (click)
Performance of the human hearing organ: audible sound pressure

Sound pressure at hearing threshold = weight of a little mouse

Sound pressure at pain threshold = weight of five elephants

(Peter Dallos 19xx)
The human ear is able to hear 600 tones between 16 Hz and 16 kHz. 1000 Hz and 1002 Hz can be differentiated (0.2%).
Performance of the human hearing organ: audible sound

As already mentioned, the human hearing organ has a huge dynamic range. The mouse/elephant cartoon from Peter Dallos shall illustrate this huge range. In this cartoon, the weight of the mouse represents the sound pressure at hearing threshold, whereas the weight of the five elephants represents the sound pressure at the threshold of pain.

Also, the human hearing organ covers a huge frequency range. The lowest sound frequency the ear is able to hear is 16 Hz, the highest sound frequency is 16 kHz. Audible frequencies cover 6 octaves. In this range the ear is able to differentiate about 600 frequencies. Thus, the ear can discriminate two sounds whose frequencies are only 0.2% apart. That means, the ear is able to differentiate a 1000 Hz tone from a 1002 Hz tone. For comparison, two adjacent keys on a piano are approximately 6% apart. See corresponding cartoon from Dallos.
Threshold varies with frequency.
Clinical audiometers assess hearing loss between 125 and 8 kHz only.
The ear’s sensitivity varies with frequency. At mid frequencies - the range of speech - the ear’s sensitivity is highest. Towards the lowest and highest sound frequencies, hearing threshold increases dramatically. Here, the sound pressure to make a sound audible is many times higher than that in the mid frequency region. For clinical diagnosis, hearing threshold is assessed at frequencies between 125 Hz and 8 kHz only. This is done at selected frequencies with a distance of an octave: 125, 250, 500, 1000, 2000, 4000, and 8000 Hz. Additionally, 750, 1500, 3000, and 6000 Hz are used as test frequencies. Sound pressure $p$ and sound pressure level $L$ are indicated on both sides of the graph. For a better reading of the threshold, an up-side down presentation is used where the hearing loss relative to the threshold of normal hearing subjects is used. In doing this, the crooked physiological threshold becomes then a straight line.
A look inside the human hearing organ
outer ear, middle ear, cochlea, neural pathway
A mammalian hearing organ – including that of human beings – consists of three main parts:
- outer ear with pinna and ear canal
- middle-ear with ear drum, malleus, incus, stapes (ossicular chain), and muscles (tensor tympani, tensor stapedius)
- cochlea that looks like a snail shell with $2^{1/2}$ windings having three fluid filled spaces (scala vestibuli, scala tympani, scala media).

Unlike a snail shell, the cochlea has two openings: the oval window with the stapedius footplate in it and the round window. Via oval window sound is transferred from the middle ear to the cochlear fluids. The oval window membrane is necessary for balancing cochlear fluid pressure. Without oval window, there was no stimulation of the sensory cells in the cochlea at all. Before talking about the work of the sound processing elements, a review on famous researchers and their findings to reveal the secrets of hearing is given.
A short look at hearing research history

Platon (428-348 before Christ) - the famous Greek philosopher - thought air molecules to enter the body via external ear canal into the body and to force the liver - which was considered as the hearing organ - to vibrate.

Garbriello Fallopia (1562) and Jean Philippe Rameau (1737) considered the auditory nerves to vibrate like a stringed instrument from which sensations for tones and harmony are delivered to the soul.

Philipp Friedrich Meckel (1724 – 1774) smashed a temporal of a corpse in winter time and found the cochlea to be filled by frozen liquid. Since then, the theory of air to be the carrier for bringing messages to the brain was obsolete.

Alfonso Corti (1822 – 1876) was the first to discover the sensory end organ of hearing – with the help of a microscope he saw the basilar membrane, the outer and inner hair cells. With the knowledge of the phenomenon of electricity in that time it became evident that the transfer of messages to the brain is done electrically and not mechanically.
Hermann von Helmholtz (1821-1894) established the „Einortstheorie des Hörens“: He believed that each single tone has its own place on the cochlea. High-frequency tones excite sensory cells in the basal, low-frequency tones in the apical region of the cochlea.

Georg von Békésy (1909-1972) discovered that not standing waves but travelling waves move the basilar membrane resulting in a frequency-specific displacement at different cochlea sites.

Thomas Gold (1920 – 2004) was the first one to question passive sound processing mechanism within the cochlea. He postulated active, non-linear processes for explaining the huge dynamic range and the high frequency selectivity of the hearing organ.
David Kemp finally discovered in 1978 that the hearing organ is able to emit sound by itself. These otoacoustic emissions (OAEs) confirm the presence of active processes in the cochlea. Later, Brownell (1985), Hudspeth (1989) and co-workers could show that Actin is the basis for the contractile characteristics of outer hair cells. Since then, a lot of researchers dedicate their work in the field of OAEs. Today, transient evoked OAEs (TEOAEs) and distortion product OAEs (DPOAEs) are instruments for diagnosing cochlear function in newborns and children.

Davis and colleagues (1939) initially described auditory evoked cortical potentials seen in raw EEG tracings.

Sohmer and Feinmesser (1967) were the first to measure click-evoked auditory brain-stem potentials (ABRs) from the human scalp.
Jewett, Williston, and co-workers (1970, 1971) definitively identified the origin of far-field scalp recorded ABRs. Since then, a lot of researchers dedicate their work in the field of auditory evoked potentials (AEPs). Today, ABRs, middle and late AEPs are the instrument for diagnosing hearing impairment along the auditory pathway.

Nowadays, auditory steady state responses (ASSRs) elicited by tone bursts of different carrier frequencies are used for assessing the hearing loss frequency specifically.
The hearing organ’s work
Survey of sound processing elements

- outer ear
- middle ear
- basilar membrane
- outer hair cell
- inner hair cell
- auditory nerve
- brain-stem
- cortex

Nervus facialis
Medial olivo-cochlear bundle

Cochlea

Sound
Impedance matching
Frequency dispersion, amplification sensory transduction

PATH medical
What has nature thought up to equip the hearing organ with such enormous capabilities? First of all, the pinna and the outer ear canal receive and amplify the sound signal. The middle ear matches the different impedances of air and fluid. Outer hair cells in the cochlea amplify the sound mechanically and thus make the hearing organ more sensitive. The inner hair cells in the cochlea are the real mechano-electrical transducers. Electrical signals are running on their afferent nerve fibers which are processed at the different stages of the ascending auditory pathway and are finally analyzed and evaluated by the brain: We hear!

Beside this, the hearing organ also provides a descending pathway by its efferent nerve fibers which is made for controlling the hearing organ’s mechanical periphery: Via the fascial nerve middle ear muscles are activated at too loud sounds resulting in an increase of the middle ear’s impedance. Also, the outer hair cell amplifiers are controlled by the efferent system. Their amplification is decreased at a loud sound. Both are done for protecting the ear from injury.
External ear acts like a funnel

- Ear canal
- Ear drum
- External ear

amplifies sound and helps to localize the sound source
External ear acts like a funnel

The external ear works acoustically like a funnel. It receives and amplifies sound. Some non-human mammals have bigger pinna, e.g. rabbits. When enlarging the pinna, sound is more amplified. You can test this by yourself when you extend the receiver area of the pinna by putting your stretched palm on it. If you do that, you can hear quiet sounds you would not hear otherwise.

As you can see, the human pinna has not really the shape of a funnel. The irregular structure inside the pinna develops different acoustical resonators which are set in action when the sound comes from a distinct direction. Thus, the brain gets information about the position of the sound source during unilateral hearing. However, sound localization is primarily done by comparing sound intensity at and travel time to both ears. If sound comes from the right side, the right ear would receive a higher sound pressure and travel time would be lower and vice versa.
Outer ear canal acts like an organ pipe

Sound amplification at resonance frequency

ear canal resonance

\[ f_r [\text{Hz}] = \frac{1}{4} \frac{c}{l_g} [1/\text{s}] = 3430 \text{ Hz} \]
The outer ear canal’s length amounts to about 30 mm. It acts like a short organ pipe. From the organ we know that the shortest pipes produce high-frequency tones whereas the longest pipes produce low-frequency tones. That means, frequency and length are in a reverse order. Assuming your ear canal is 25 mm long. With the sound velocity in air of 330 m per second your ear canal has a resonance frequency of 3430 Hz corresponding to a g4 tone. You can test this, if you blow in a tube of different length while putting your finger tip on the other side. The ear canal’s task is not only to prevent the ear drum from injury but also to increase the ear’s sensitivity in the frequency range of best hearing.
Outer ear canal acts as an organ pipe

\[ f \text{ [Hz]} = \frac{1}{4} \frac{cs}{lg} \text{ [1/s]} \]

- \( cs = 343 \text{ m/s} \)
- \( lg = \text{pipe length} = \frac{1}{4} \text{ wave length} \)

- \( lg = \frac{1}{2} l_o \)

Lg = 2,5 cm
8 mm

Outer ear canal resonance
3430 Hz ~ \( g^4 \)
Middle ear matches impedances (air/fluid) and thus improves energy flow.

Increase of pressure = 17:1 (A1:A2)
Increase of force = 3:1 (l1/l2)
The middle ear is an acoustic transformer. For matching the different impedances of air and fluid, nature provides two mechanisms: increase of pressure and increase of forth. First one is due to the different areas of the tympanic membrane and the footplate of the stapes with a ratio of 17:1, second one is due to the lever mechanism of malleus and incus which have different lengths with a ratio of 3:1. Increase of pressure and increase of force in sum, bring about that 60% of the sound energy is transferred to cochlear fluids.
Cochlea allows for **frequency coding**, **sound amplification**, and **neural transduction**

**Frequency coding:** *frequency-place-transformation + periodicity of nerve signals (action potentials)*

**Sound amplification:** *motility of outer hair cells*

**Neural transduction:** *inner hair cell/cochlear nerve*
Cochlea allows for **frequency coding**, **Sound amplification**, and **neural transduction**

Coming now to the cochlea. As already stated, cochlea’s work is to transfer a mechanical signal (i.e. stereocilia displacement) to an electrical signal (i.e. action potential on the nerve fiber). However, this has to be done such that the cochlea ‘knows’ about the frequency and the intensity of the incoming sound.
Cochlear frequency coding

*Frequency (f)-Place (z)-Transformation*

Due to decreasing stiffness of the basilar membrane from apex to basis, travelling waves are generated. Low frequency tones (e.g. 500 Hz) generate vibrations near apex, high frequency tones (e.g. 2 kHz) near basis.
Cochlear frequency coding

*Discovery of frequency (f) – place (z) transformation*

Look inside the cochlea

*Tectorial membrane, outer + inner hair cells*
Cochlear frequency coding

**Periodicity of neural signals**

With decreasing tone frequency, distance $T$ between action potentials increases.
Travelling wave $A(z)$ and time course of basilar membrane displacement $A(t)$ for click, low- (l), mid- (m), and high-frequency tones (h).
Cochlear sound amplification

*Outer hair cells = sound amplifiers*

There are 3200 cochlear segments. Each segment has 1 inner and 3 outer hair cells. Due to the motility of outer hair cells, displacement of basilar membrane is enhanced actively (cochlear amplifier). Inner hair cells are responsible for the neural coding of sound.
Discovery of cochlear sensory cells by Alfonso Corti → Corti Organ

Bildatlas Innenohr, Duphar Pharma, 1983
Cochlear sound amplification

*Outer hair cells = sound amplifiers*

Outer hair cell amplifiers care for an enhancement of the hearing organ's sensitivity. Due to a feedback mechanism, displacement of the basilar membrane is increased.
Outer hair cells are able to move their body and thus care for additional vibration energy that is feeded back to the basilar membrane. Without outer hair cell function dynamic range of hearing would be halved.
Rocking outer hair cells
*by Jonathan Ashmore (1987)*

Isolated outer hair cell that is moving following the rhythm of "rock around the clock" (youtube video).
Dysfunction of cochlear amplifier
→ Loss of sensitivity and compression
→ Recruitment (hearing loss at low, normal hearing at high sound intensities)

Sensitivity of cochlear sensory cells
→ highly vulnerable

1 nm = 1/1000 000 000 m
For comparison: diameter of a human hair = 100 µm = 1/1000 000

In: Dallos et al. (Editors)
The Cochlea

inner hair cells  outer hair cells
3 – 4 rows

Corti-Organ

1 nm = 1/1000 000 000 m
For comparison: diameter of a human hair = 100 µm = 1/1000 000)
Noise induced hearing loss
Pathologies of the hearing organ

External ear
- Cerumen
- Marble
- Atresia

Middle ear
- TM-perforation
- Otitis media with effusion
- Eustachian-tube dysfunction
- Otosclerosis
- Ossicle fracture or ossification

Basilar membrane

Outer hair cells
- Noise
- Sudden hearing loss
- Menière’s disease
- Presbyacusis
- Ototocicity
- Gentetics
- Barotrauma

Inner hair cells

Auditory nerve
- Akustikusneuroma
- Tumors
- Neurofibromatosis
- Auditory neuropathy
- Multiple sclerosis
- Brain trauma

Brain-stem

Cortex
- Psychological hearing disorders
- Encephalitis
- Meningitis
- Stroke
What means a hearing loss for people concerned

“Oma und das Gewitter” youtube video
Audiology – hearing assessment

Sound processing + sensation

Outer ear
- Otoscopy
- Tympanometry

Middle ear
- Basilar membrane
- Outer hair cells
- Inner hair cells

Auditory nerve

Brain stem
- Carhart Langenbeck
- Stapedius Reflex

Cortex
- Pure-tone
- Speech intelligibility
- Loudness
- Late AEP

Pure-tone audiometry: air/bone conduction
- SISI Fowler
- Otoakoustic emissions (OAE)
- Auditory brain-stem evoked potentials potentials (ABR)

Elektro-Cochleography (ECOG)
Tuning Fork
Pure-tone Audiometry
Speech Intelligibility Tests

Tympanometry
Oto-acoustic Emissions
Auditory evoked potentials
What is louder:
• infront - Air Conduction (AC)
• behind the ear - Bone Conduction (BC)?

AC is louder than BC àRinne +

BC is louder than AC → Rinne -

Where do you hear the sound, in the left or in the right ear?

normal middle ear

hearing loss

tone is heard in the better inner ear

tone is heard in the worse middle ear
### Tuning folk

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**Normal hearing**

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**Middle ear dysfunction left**

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**Cochlear dysfunction left**
Hörweitenprüfung

50 dB Hörverlust

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<td>10 cm</td>
<td>Flüstersprache (m)</td>
<td>20 cm</td>
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<tr>
<td>1 m</td>
<td>Umgangssprache (m)</td>
<td>4 m</td>
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40 dB Hörverlust

Flüstersprache 6 m normales Gehör

--- vier-und-zwan-zig ---
Pure-tone audiogram
Pure-tone air and bone conduction

Normal hearing

Bone conductor

Headphones

Outer-/Middle-
Cochlea/Neural ear pathway

Hearing loss

125 Hz frequency 8 kHz

0 dB HL
Pure-tone audiogram

Air conduction deficit

125 Hz frequency 8 kHz

0 dB HL

Hearing loss

air-bone conduction difference

Bone conductor

Headphones

Headphones

Outer-/Middle-ear disease

Cochlea/Neural pathway

Outer-/Middle-ear disease

Hearing loss

0 dB HL
Pure-tone audiogram

Air conduction deficit (middle ear dysfunction)

Otitis media with effusion

Otosclerosis

Bone conductor (BC)

Head phones (AC)

left ear

right ear

X — X

> .... >
Pure-tone audiogram

Air and bone conduction deficit

125 Hz frequency 8 kHz

0 dB HL

Hearing loss

Air and bone conduction shift

Bone conductor

Headphones

normal disease

Outer/Middle ear Cochlea/neural

Sensory/neural dysfunction
Pure-tone audiogram

Air and bone conduction deficit

Noise induced hearing loss

Menière’s disease

Bone conductor (BC)

Head phones (AC)

left ear

right ear
Menière's disease
Noise induced hearing loss
Presbyacusis
Otoxicity
Sudden hearing loss

Pure-tone audiogram
Cochlear/neural hearing loss

air = bone conduction
Presbycusis
hearing loss at high frequencies

Median air conduction hearing loss according to ISO 7029
a) 30 years  b) 50 years  c) 70 years
10 Zweistellige Zahlen (10 Gruppen)
1. 98 22 54 19 86 71 35 47 80 63
2. 53 14 39 68 57 90 85 33 72 46

Startschallpegel 25 dB über Hörschwelle bei 500 Hz, dann Startschallpegel +5 dB

20 Einsilbige Worte (20 Gruppen)
1. Ring Spott Farm Hang Geist Zahl Hund …
2. Holz Ruß Mark Stein Glied Fleck Busch …

Startschallpegel 65 dB, dann +15 dB bis 100% Verstehen erreicht wird.
(bei Lärmgutachten: Startpegel 60 dB, dann +20 dB bis 100% Verstehen erreicht wird.)

Ergebnis:
Hörverlust für Zahlen: 28 dB
Maximale Einsilberverständlichkeit: 100%
Physiological tests

- Tympanometry
- Otoacoustic Emissions
- Evoked Potentials

- External/middle ear
- Cochlea
- Neural pathway

- Acoustically
- Acoustically
- Acoustically
- Acoustically
- Acoustically
- Electrically
Physiological test procedures reflect the mechanical and neural function of the auditory system. Therefore, physiological tests are used for reliably detecting the site of impairment along the auditory pathway. Tympanometry (Tymp), oto-acoustic emission (OAE) and auditory evoked potential (AEP) are able to differentiate between middle-ear, cochlear, and neural disorders. In case of Tymp and OAE, both, stimulus and response are acoustic signals. In contrast, in case of AEP there is an acoustical input-signal and an electrical output-signal.

In view of an adequate therapy of a hearing impairment, it is important to known which stage of the auditory pathway is concerned. Tymp, OAE, and AEP allow for selectively assessing middle-ear, sensory (cochlear) and neural disorders. Behavioural testing is less reliable. This is true, especially, in infants and other non-cooperative patients, where psycho-acoustical tests cannot reliably be performed.
Tympanometry

Probe tone 226 Hz, 667 Hz or 1000 Hz

Sound probe

Outer ear canal

Probe tone 226 Hz, 667 Hz or 1000 Hz

Pump for ear canal pressurization

Tympanic membrane

Malleus

Incus

Cochlea

Apex

Stapes

basilar membrane 35 mm
The function of the middle ear is to minimize the loss of acoustic energy that appears when sound is transferred from air in the outer ear canal (low density) to fluid in the inner ear (high density). Without the specific middle ear features, approximately 99.9% of the sound energy would be reflected at the fluid due to the different densities. The middle ear helps to improve the energy balance by increasing sound pressure and force. The increase of sound pressure is simply due to the fact that the tympanic membrane area is seventeen times larger than the area of the footplate of the stapes which is the connecting link between middle and inner ear. The increase of sound pressure becomes clear when looking at the physical equation, which defines pressure as force divided by area \( p = \frac{F}{A} \). Thus, with reduced area and same force the pressure increases. The increase of force is due to the different length of the malleus and the incus providing a lever action of the ossicular chain (malleus, incus, stapes). Both mechanisms yield an impedance matching which allows for a transmission of 60% of the sound energy to the inner ear.
Tympanometry

normal

226 Hz Ytm

-300 -200 -100 0 100 200 daPa

ECV: 1.31 ml - TW: 70 daPa
Peak: 1.17 ml / -3 daPa

tube dysfunction

226 Hz Ytm

-300 -200 -100 0 100 200 daPa

ECV: 0.51 ml - TW: 70 daPa
Peak: 0.50 ml / -202 daPa

otitis media effusion

226 Hz Ytm

-300 -200 -100 0 100 200 daPa

ECV: 0.66 ml
No Peak in -300 ... 200 daPa
In middle ear diagnostics, typically the admittance is evaluated. Admittance is determined by compliance (= 1/stiffness, spring load), mass, and friction or resistance. Mathematically, the admittance $Y$ is a complex value consisting of conductance $G$ (real part) and susceptance $B$ (imaginary part), i.e. $Y = G + jB$. Friction influences conductance, whereas compliance and mass influence susceptance. Conductance (friction) is independent of frequency, whereas susceptance (compliance, mass) is dependent on frequency with compliant susceptance being inversely proportional to frequency and mass susceptance being directly proportional to frequency. With increasing frequency, the total susceptance progresses from positive values (stiffness controlled) towards 0 mmho (resonance) to negative values (mass controlled). The resonance frequency is directly proportional to the stiffness of the middle ear, i.e. with increasing stiffness the resonance frequency increases (e.g. at otosclerosis), and inversely proportional to the mass of the middle ear, i.e., with increasing mass the resonance frequency decreases.
Tympanometry

**chain interruption**

- ECV: 1.21 ml
- TW: --
- Peak: --

**otosclerosis**

- ECV: 1.12 ml
- TW: 40 daPa
- Peak: 0.16 ml

226 Hz Ytm
Tympanometry is usually performed at low test-tone frequency (220 or 226 Hz). At low frequencies, the normal-middle ear system is stiffness-controlled and susceptance (stiffness element) contributes more to overall admittance than conductance (frictional element). Typically, static air pressure varied from +200 daPa to -200 daPa. The result is a graphic display called a tympanogram which plots middle ear admittance over static air pressure. Different middle-ear pathologies exhibit different tympanogram shapes. In case of normal middle-ear function the tympanogram shape corresponds to a Gaussian bell curve with its maximum being around zero static pressure, i.e., maximum energy is transferred into the cochlea. In case of Eustachian-tube dysfunction the peak of the Gaussian bell curve is shifted towards negative pressure. In case of otosclerosis, the peak of the Gaussian bell curve is small (due to decreased mobility), however located within the zero static pressure range. In case of an interruption of the ossicular chain (due to increased motility) there is a open curve.
Tympanometry, acoustic reflex

Reflex pathway

ipsi-lateral
contra-lateral

central auditory pathway
The middle ear is able to increase its impedance for providing protection against loud sounds. In case of a sound higher than about 80 dB HL, the middle ear muscles (stapedius muscle and tensor tympani muscle) are activated resulting in an increased stiffness of the middle ear. As a consequence, the energy transmitted to the inner ear is lower.

The acoustic reflex (or stapedius reflex, attenuation reflex, auditory reflex) is an involuntary muscle contraction. This includes contraction of the stapedius and tensor tympani muscle. The stapedius muscle stiffens the ossicular chain by pulling the stapes away from the oval window of the cochlea and the tensor tympani muscle stiffens the ossicular chain by loading the eardrum when it pulls the malleus in toward the middle ear. As a consequence the transmission of vibrational energy to the cochlea is decreased. The pathway involved in the acoustic reflex is complex and can involve the ossicular chain itself, the cochlea, the auditory nerve and the brainstem. Acoustic reflex is elicited at different test-frequencies: 500, 1, 2, 3, and 4 kHz.
Tympanometry, acoustic reflex

**tympanogram**

**acoustic reflex**

Reflex threshold = 70 dB HL
Reflex at different test-frequencies

**Tympanometry, acoustic reflex**

- **Ipsi**: 2000 Hz, 80 dB HL

![Graph showing reflex at different test-frequencies](image)
Oto-Acoustic Emission (OAE)
Discovered by David Kemp in 1978

The discovery of oto-acoustic emissions (OAEs) by David Kemp in 1978 has produced a fast, powerful, and versatile tool for diagnosing cochlear integrity. OAE measurements are today a standard part of the audiometric test battery. OAEs are elicited and measured by means of electro-acoustic transducers (loudspeaker and microphone) within an ear probe placed in the outer ear canal. There are spontaneous (SOAEs) and evoked OAEs (EOAEs). EOAEs are the by-product of the non-linear sound amplification process in the cochlea. OAEs are low-level sound emissions generated by the outer hair cells (OHCs) within the cochlea. OAE levels depend on the number of functioning outer hair cells given a normal middle-ear function. OAE levels depend on the ear canal volume. Because of the smaller ear canal volume, OAE amplitude in newborns and infants is higher compared to that in adults. Thus, OAEs are easier to measure and thus provide a suited tool for newborn-hearing screening and follow-up diagnostics.
Transient Evoked Oto-Acoustic Emission (TEOAE)

- Ear canal
- TEOAE
- Ossicles
- Sound probe
- Apex
- Basilar membrane
- Tectorial membrane
- Basilar membrane
- IHC
- OHC motility
- Cochlear amplifier
- Afferent NF

- Frequency [kHz]:
  - 0.125
  - 0.25
  - 0.5
  - 1
  - 2
  - 4
  - 8
  - 16
- Distance from apex [mm]:
  - 0
  - 800
  - 1600
  - 2400
  - 3200
- Sensory cell rows:
  - 0
  - 8
  - 16
  - 24
  - 32
Transient Evoked Oto-Acoustic Emission (TEOAE)

![Diagram of TEOAE](image)

- **TEOAE**
  - Frequency domain: $f \ [Hz]$
  - Sound pressure level: $L \ [dB SPL]$

- **Outer hair cell response**
  - Time domain: $t \ [ms]$
  - Pressure: $p \ [Pa]$

- **Evaluation window**
  - Recording window:
    - 1ms
    - 2ms
    - 4ms
    - 8ms
    - 16ms
    - 32ms

- **TEOAE noise**
  - TEOAE: 8/8
  - Noise: 0%
  - Artifact: 0%
  - Stability: 100%

**Legend:***
- Green bar: TEOAE
- Blue bar: Noise

**Audiology:**
- Page 85
Transient oto-acoustic emissions are elicited by transient acoustic stimuli (clicks or tone-bursts). TEOAEs represent the sum of acoustic impulse-responses of OHCs along the cochlea. At click-stimulation, almost all OHCs along the cochlear partition are set into movement. When using tone-bursts of different carrier-frequencies a specific part of OHCs along the cochlea is stimulated. Due to cochlear frequency dispersion, TEOAE components can be directly traced to a specific place. As the basilar membrane at basal sites moves faster than at more apical sites, high-frequency TEOAE components stem from basal cochlear sites, whereas low-frequency TEOAE components come from more apical ones. As a consequence, basal responses appear at the beginning and apical responses at the end of the TEOAE time function. TEOAEs thus provide a rough frequency-specific estimation of cochlear hearing loss. TEOAE’s sound pressure level is very low varying from about 10 to -30 dB SPL. Thus, measurement and averaging of several hundred signal-epochs are necessary for extracting TEOAE response from noise.
Transient Evoked Oto-Acoustic Emission (TEOAE)

28 years old adult, normal hearing

SNR criteria: 
- 22 dB
- 26 dB
- 22 dB
- 22 dB
- 9 dB

Overall criterion: 5 / 5

Stimulus Optimized
Level [dB peSPL] 85
Averages 533
Transient Evoked Oto-Acoustic Emission (TEOAE)

A normally hearing adult exhibits TEOAEs having high levels and a broad signal spectrum. Signal-to-Noise-Ratio (SNR) is high (more than 20 dB with the exception at 4 kHz (9 dB) in the case example). SNR criterion for accepting a response as valid was set to 9 dB. In contrast, in a newborn at birth with a temporary sound conduction deficit due to amniotic fluid in the tympanic cavity (see case example), TEOAE components have lower levels SNRs (10, 13, 18, 17, 7 dB). However, SNR criterion is fulfilled at all test frequencies with exception at the highest test frequency (4 kHz). The reason for this is a high frequency hearing loss due to a reduced sound conduction because of the amniotic fluid. In contrast, in a six days old newborn no TEOAEs could be measured. SNRs are below the selected criterion (< 9 dB). There was a normal tympanogram (not shown). In this case, a dysfunction of outer hair cells is most likely. TEOAEs already disappear at mild hearing losses and are therefore commonly used in hearing screening programs.
Transient Evoked Oto-Acoustic Emission (TEOAE)

2 hours old newborn, amniotic fluid in tympanic cavity
Transient Evoked Oto-Acoustic Emission (TEOAE)

6 days old newborn: no TEOAE $\rightarrow$ OHC dysfunction
Distortion Product Oto-Acoustic Emission (DPOAE)

DPOAEs are cubic distortions of outer hair cells (OHC) when stimulated simultaneously by two tones $f_1$ (lower frequency) and $f_2$ (higher frequency). DPOAEs arise directly from the frequency-selective compressive nonlinearity of OHCs. The two primary tones interact in the cochlea within the region of overlap of the traveling waves of the two primary tones close to the characteristic place of $f_2$. Thus, DPOAEs can be applied as a probe for frequency-specific assessment of cochlear dysfunction at the $f_2$ place. In humans, both quadratic ($f_2-f_1$) and cubic distortion products ($2f_1-f_2$) can be detected. The cubic distortion component $2f_1-f_2$ yields the highest amplitude and is therefore primarily used for diagnostics. DPOAE amplitudes typically range from about 20 dB SPL down to about -30 dB SPL. DPOAEs are measurable at a cochlear hearing loss of up to 40 to 50 dB HL. DPOAEs provide quantitative and frequency-specific information about the range and operational characteristics of the cochlear amplifier, i.e., sensitivity, compression, and frequency selectivity of the hearing organ.
Distortion Product Oto-Acoustic Emission (DPOAE)

- Frequency [kHz]: 0, 8, 16, 24, 32
- Distance from apex [mm]: 0, 800, 1600, 2400, 3200
- Sensory cell rows: 0, 8, 16, 24, 32

- Primary tones
- 2f1 – f2
- DPOAE
- Distortion Product Oto-Acoustic Emission

Diagram showing:
- Outer ear canal
- Ossicles
- Cochlear apex
- Basilar membrane
- Tectorial membrane
- IHC (Inner Hair Cells)
- OHC (Outer Hair Cells) motility
- Afferent NF (Neuronal Fibers)
- Cochlear amplifier
Distortion Product Oto-Acoustic Emission (DPOAE)

The number of OHCs contributing to DPOAE generation depends on the size of the overlapping region, which is determined by the primary tone levels $L_1$ and $L_2$, and the frequency ratio $f_2/f_1$. A frequency ratio of about 1.2 has been found to be optimal.

A primary tone level setting, which accounts for the different compression of the primary tone traveling waves at the $f_2$ place, is the scissor paradigm (see below) DPOAE-grams plot the DPOAE level $L_{dp}$ as a function of $f_2$ (the main DPOAE generation site) for a selected combination of primary-tone levels $L_1$ and $L_2$.

DPOAE-grams reflect the sensitivity of the cochlear amplifier (CA) best when recorded at close-to-threshold stimulus levels. In normal hearing (normal CA), DPOAE-grams are close to each other at high and more separated at low stimulus levels reflecting cochlear non-linear sound processing. In cochlear hearing loss ears (impaired CA), DPOAE-grams are more separated even at high stimulus levels, revealing loss of CA compression.
ABSTRACT

The purpose of the study was to evaluate whether mDPOAE measurements can be done in both ears simultaneously without mutual influence of primary tone pairs in the ipsilateral and the contralateral ear. The mDPOAE paradigm recently reported (Dziendziel, 2012, unpublished doctoral dissertation). Protocol using automatic pass/refer criteria and a preset multiple primaries, their frequencies have to be at least one octave apart. When stimulating both ears contralateral DPOAE suppression can occur, especially for primary tone levels higher than 65 dB SPL.

RESULTS

For L<sub>2</sub> = 65 dB SPL, the mean DPOAE and noise levels were almost identical for two testing conditions.

For L<sub>2</sub> = 45 dB SPL, the mean DPOAE levels were lower, mostly at f<sub>2</sub> = 1.5 and 2 kHz, for binaural stimulation than those for monaural stimulation.

For L<sub>2</sub> = 65 dB SPL, the mean DPOAEs were lower, mostly at f<sub>2</sub> = 4 and 8 kHz, for the mDPOAE condition than those for the single-pair presentation.

The mDPOAE testing resulted in elevated noise levels at 4 kHz for both L<sub>2</sub> values and at 3 kHz for L<sub>2</sub> = 65 dB SPL.

There was a small decrease of DPOAE levels measured with L<sub>2</sub> = 45 dB SPL, especially at 1.5, 2, and 4 kHz.

For L<sub>2</sub> = 65 dB SPL, an increase of noise levels at 3 and 4 kHz and slightly reduced DPOAEs were observed.

DISCUSSION

Binaural presentation

In general, the binaural presentation of single-pair stimuli had subtle effects on DPOAE levels. The difference of 2 dB between mean DPOAE levels collected with binaural versus monaural presentations for f<sub>2</sub> = 1.5 kHz and L<sub>2</sub> = 45 dB SPL was the only one that reached statistical significance. All other data measured with the two paradigms were very consistent. Thus, the effects of contralateral inhibition created by binaural stimulations are negligible. Similar results for DPOAEs collected using the ILO Otodynamics Analyser 922 system at L<sub>1</sub> = 71 and L<sub>2</sub> = 60 dB SPL have been previously reported (Dziendziel, 2012, unpublished doctoral dissertation).

The mDPOAE paradigm

The effect of applying one additional pair of tones with an octave spacing on DPOAE levels was quite small, with the largest decrease of 1.3 dB for f<sub>2</sub> = 4 kHz and L<sub>2</sub> = 65 dB SPL. Slightly lower DPOAE levels obtained with the mDPOAE method than with single-pair stimulation may result from small mutual suppression of cochlear nonlinearities. The noise levels were elevated at 3 and 4 kHz in mDPOAEs measured at L<sub>2</sub> = 65 dB SPL. Most likely those results are due to the frequency response of the transducers requiring quite different gains when mixing mid- and high-frequency stimuli.

Could mDPOAE data be collected in both ears simultaneously?

In general, the mean DPOAE and noise levels collected with mDPOAEs and binaural presentation conditions were highly reproducible when compared to those obtained with the single-frequency monaural paradigm. Thus, multi-frequency and binaural measurements could be applied to reduce DPOAE testing time considerably. A protocol using automatic pass/refer criteria and a preset maximum measurement time for each data point and lower-level primaries, e.g., L<sub>2</sub> = 45 dB SPL, is expected to be efficient, for example in patients with beginning cochlear hearing loss due to noise overexposure or ototoxic drug administration.
Distortion Product Oto-Acoustic Emission (DPOAE)

176 measurement points between 1.5kHz and 5kHz
Distortion Product Oto-acoustic Emission (DPOAE)

DP-gram → OHC-dysfunction

DP-gram → normal OHC-function
Distortion Product Oto-Acoustic Emission (DPOAE)

Especially, extrapolated DPOAE I/O functions allow for assessing loss of cochlear sensitivity and compression. The number of OHCs contributing to DPOAE generation depends on the size of the overlapping region, which is determined by the primary tone levels $L_1$ and $L_2$, and the frequency ratio $f_2/f_1$. A frequency ratio of about 1.2 has been found to be optimal for yielding highest amplitudes. A primary tone level setting, which accounts for the different compression of the primary tone traveling waves at the $f_2$ place, is the scissor paradigm. Due to the steep slope of the traveling wave towards the cochlear apex, the maximum interaction site is close to the $f_2$ place in the cochlea. To preserve optimum overlap of the primary tone traveling waves at a constant frequency ratio, the primary tone level difference has to be increased with decreasing stimulus level. This results in a decrease of $L_1$ being lower than the decrease of $L_2$ (scissor paradigm: $L=0.4L_2 + 39$ dB SPL).
Hearing threshold estimation by means of extrapolated DPOAE pressure I/O-function: intersection between L2-axis and linear regression line.
**Distortion Product Oto-acoustic Emission (DPOAE) Threshold**

DPOAE level I/O-functions plot the DPOAE level $L_{dp}$ as a function of primary-tone level $L_2$ for a selected $f_2$ and thus reflect cochlear amplification at the $f_2$ place. In normal hearing, in response to low-level stimuli, DPOAE level I/O-functions exhibit steep slopes, while at high stimulus levels slopes decrease, thus mirroring the strong amplification at low and decreasing amplification (saturation) at moderate sound levels. However, this is only true when a specific stimulus level setting is used which accounts for the different compression of the primary-tones at the $f_2$ place (scissor paradigm).

DPOAE pressure I/O-functions plot the DPOAE pressure $p_{dp}$ (instead of the DPOAE level $L_{dp}$) as a function of the primary-tone level $L_2$. Due to the logarithmic dependency of the DPOAE level on the primary tone level there is a linear dependency between DPOAE pressure $p_{dp}$ and primary tone level $L_2$. Thus, DPOAE data can easily be fitted by linear regression analysis. The intersection point of the linear regression line with the $L_2$-axis at $p_{dp} = 0$ Pa can then serve as an estimate of DPOAE threshold.
Distortion Product Oto-Acoustic Emission (DPOAE)

Hearing Threshold Estimation using extrapolated DPOAE I/O-functions
Distortion Product Oto-Acoustic Emission (DPOAE)

When converting DPOAE level from dB SPL to dB HL (hearing level), estimated DPOAE thresholds can be plotted in an audiogram form (DPOAE audiogram). DPOAE audiograms can be applied in babies due to Eustachian tube dysfunction and/or amniotic fluid in the tympanic cavity or to confirm a persisting cochlear hearing loss in follow-up diagnostics. In case of mild and moderate hearing loss DPOAE audiograms are an alternative method to behavioural audiometry or auditory brainstem responses (ABR, ASSR). Especially in infants where the conditioned free-field audiogram does not reliably reflect hearing threshold. DPOAE audiograms may assess cochlear hearing loss more precisely than behavioural tests. Moreover, unilateral hearing loss can be detected. DPOAE audiograms are able to quantitatively assess the hearing loss at distinct frequencies in a couple of minutes. Predicting hearing loss at five frequencies by tone burst ABR or ASSR may take half an hour and more. Thus, DPOAE audiograms can serve as a suited tool for bridging the gap between screening and behavioural testing in paediatric audiology.
Distortion Product Oto-Acoustic Emission (DPOAE)

DPOAE- Threshold Estimation

$f_2 = 3000$ Hz

<table>
<thead>
<tr>
<th>$f_2$</th>
<th>thres</th>
<th>$L_2$ min</th>
</tr>
</thead>
<tbody>
<tr>
<td>1500</td>
<td>8</td>
<td>20</td>
</tr>
<tr>
<td>2000</td>
<td>4</td>
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<tr>
<td>6000</td>
<td>12</td>
<td>20</td>
</tr>
<tr>
<td>8000</td>
<td>17</td>
<td>25</td>
</tr>
</tbody>
</table>

normal hearing

Full I/O-function-based estimation for right ear

Hearing threshold estimation based on lowest detected DPOAE (right ear in red)

Full I/O-function-based estimation for left ear

DPOAE could be recorded for this frequency. Hearing threshold is probably above 50 dB (right ear in red)

Gray symbols if some levels were skipped
Distortion Product Oto-Acoustic Emission (DPOAE)

DPOAE- Threshold estimation → cochlear hearing loss

- Full I/O-function-based estimation for right ear
- Full I/O-function-based estimation for left ear
- Hearing threshold estimation based on lowest detected DPOAE (right ear in red)
- No DPOAE could be recorded for this frequency. Hearing threshold is probably above 50 dB (right ear in red)

<table>
<thead>
<tr>
<th>$f_2$</th>
<th>thres</th>
<th>$L_2$ min</th>
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</thead>
<tbody>
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<tr>
<td>8000</td>
<td>40</td>
<td>55</td>
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</tbody>
</table>
Distortion Product Oto-Acoustic Emission (DPOAE)

DPOAE- Threshold estimation

6 months old infant

3 months old infant

- hereditary hearing loss
- discrepancy between behavioral and physiological threshold
Distortion Product Oto-Acoustic Emission (DPOAE)

100 newborns < 2.5 days old

Estimated hearing loss dB HL

1 kHz 2 4 8

100 left ears
100 right ears

6 years old child

DPOAE Threshold

Newborn study, see T. Janssen "Otoakustische Emissionen" in: Lehnhardt/Laszig "Praxis der Audiometrie" Thieme 2009 (ISBN 078-3-13-369009-6)
Oto-Acoustic Emission

Clinical applications

- Newborn hearing screening and Follow-up
- Pediatric audiology

- Clinical Diagnostics
  - Confirmation of cochlear hearing loss (topological diagnostic, report noise induced hearing loss)

- Simulation und Aggravation
  - Difference between pure-tone threshold and OAE

- Follow up: Sudden Hearing Loss and Ototoxic Medication
Oto-Acoustic Emission

Clinical applications

OHCs are reported to be impaired by sound overexposure, ototoxic drugs (e.g. therapeutic antibiotics), infections (e.g. meningitis, mumps, materno-fetal infection), and anoxia (e.g. birth trauma), or to be partly missing in genetic hearing loss. OHC impairment results in a loss of sensitivity and frequency selectivity of the hearing organ. OAEs, as a by-product of cochlear non-linear sound amplification, then appear with reduced amplitude or disappear. Since OAEs are a by-product of the non-linear sound amplification process of OHCs in the cochlea they can only serve as a measure for evaluating OHC integrity. Lesions of inner hair cells or retro-cochlear defects (e.g. neural defects, auditory processing disorders) are not detectable by means of OAE. In sound-conductive hearing-loss, both the stimulus and the response amplitude are reduced. OAEs are not present even at a mild hearing-loss. At a cochlear hearing loss exceeding 20-30 dB HL (TEOAE) or 40-50 dB HL (DPOAE) no OAEs are measurable. In these cases, tympanometry, ABR, and ASSR should be performed to determine type and degree of the hearing loss.
If there is a suspicion of a hearing disorder, OAEs should be used first. It is fast and helps to confirm normal middle-ear and cochlear function. This is the case if OAEs are present over a wide frequency range. If OAEs are absent, the presence of a middle-ear or cochlear (OHC) pathology is likely. OAEs then should be followed by tympanometry. If the tympanogram is normal and OAEs are absent, then a cochlear disorder is likely. If the tympanogram is abnormal, a sound-conductive hearing-loss is likely. If there is an indication for a hearing disorder and both the tympanogram and OAEs are normal, ABR/ASSR may reveal if there is a cochlear (inner hair cell) or neural pathology. For example, in auditory neuropathy, where synchronization of neural activity is malfunctioning (either due to inner hair cell synaptic or neural dysfunction), normal OAEs and abnormal ABRs occur.
Oto-Acoustic Emission

Clinical applications

Typical clinical applications of OAEs are: hearing screening, follow-up diagnostics after newborn hearing screening, confirmation of cochlear hearing loss (together with tympanometry and ABR), quantitative evaluation of hearing loss and recruitment for providing hearing aid fitting parameters, early detection and monitoring of OHC impairment after noise over-exposure or ototoxic drug administration, topological diagnostics, as well as identifying subjects simulating a hearing loss.
Far-field Auditory Evoked Potential (AEP)

Late Latency AEP (Cortex)
Baucaud et al. (1953), Gastaut (1953), Davis and Yoshie (1963), Davis et al. (1967), Davis (1971, 1976), Keidel (1976)

Middle Latency AEP (Sub-Cortex)
Goldstein and Rodman 1967, Thornton 1975, Gibson 1978

Brain-stem evoked response (Brain-stem)

Auditory study State Response (Brain-stem, Subcortex)
Lit
Neural Auditory Pathway

- Nucleus cochlearis
- Olivo-cochlear bundle
- Lemniscus lateralis
- Colliculus inferior
- Corpus geniculatum
- Auditory cortex

NC → OB → LL → CI → CG → AC

auditory nerve
Auditory Evoked Potentials, Origin

**Auditory Brain-stem Response (ABR)**
- I - V

**Middle Latency Response**
- Na/Pa

**Late Latency Response**
- N1/P1
Auditory Brain-stem Response (ABR)

Eliciting and measuring ABR
Auditory Brain-stem Response (ABR)

U [nV]

stimulus

masking noise for avoiding crossover of sound

Σ

+ -

I II III IV V

IPL

t [ms]
Auditory Brain-stem Response (ABR)

Normal hearing

Latency of wave V within normal range. Response detectable at 10 dB stimulus level
Auditory Brain-stem Response (ABR)

*Middle-ear disorder*

Latency delay of wave V and threshold shift
Auditory Brain-stem Response (ABR)

HF-cochlear hearing loss (presbyacusis)

Latency delay of wave V and threshold shift
Auditory Brain-stem Response (ABR)

HF-cochlear hearing loss (ototoxic)

Latency delay of wave V and threshold shift
Auditory Brain-stem Response (ABR)

Pan-cochlear hearing loss

Normal Latency of wave V and threshold shift
Normal Latency of Wave I and delayed Latency of Wave V = increased Inter-Peak-Latency (Wave V – Wave I) > 4,4 ms
→ Akusticus neuroma (side difference)
→ Tumors, Neurofibromatosis (no side difference)
Auditory Brain-stem Response (ABR)

Retro-cochlear (neural) dysfunction

Normal latency and amplitude of wave I

Latency shift and amplitude decrease of wave V
Auditory Steady State Responses (ASSR) – Stimulus

- Chirps presented with jittered repetition rate
- Rate is randomly changed four times per second
- Rates are equally distributed between
  - 40 – 42 Hz (40-Hz-ASSR)
  - 82 – 84 Hz (80-Hz-ASSR)
- Frequency resolution: 12.2 µHz
- → reduce sensitivity to certain electrical interference (e.g. periodic interferer)
Auditory Steady State Responses (ASSR)

carrier frequency $f_c$ e.g.: 1 kHz

$1/fm = 12.5 \text{ ms}$

Stationäre Stimulus $\rightarrow$ frequency specific stimulation of the cochlea

Amplitude modulation $\rightarrow$ Triggerung of Onset-Neurons in brain -stem,

$fm = 80 \text{ Hz (12.5 ms)}, \text{ in subcortex, } fm = 40 \text{ ms}$
Auditory Steady State Responses (ASSR) – Chirp stimuli

- Exponential chirps \( f(t) = f_{\text{start}} \left( \frac{f_{\text{start}}}{f_{\text{stop}}} \right)^t \), bandwidth: half octave

  \[ \rightarrow \text{compensation for time delay on basilar membrane} \]

\[ f = 1 \text{ kHz} \]
\[ f_m = 84 \text{ Hz} \]

Activation pattern on basilar membrane

<table>
<thead>
<tr>
<th>f [Hz]</th>
<th>f_{\text{start}}</th>
<th>f_{\text{stop}}</th>
</tr>
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<tbody>
<tr>
<td>500</td>
<td>385</td>
<td>650</td>
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<tr>
<td>1000</td>
<td>870</td>
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<tr>
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<td>2300</td>
</tr>
<tr>
<td>4000</td>
<td>3480</td>
<td>4600</td>
</tr>
</tbody>
</table>
Auditory Steady State Response (ASSR) – Recording View (binaural, 4f/ear)

- Right ear
- Left ear

Detection criterion
Stimulus level [dB nHL]
Carrier frequency [Hz]
Progress bar
EEG background noise
Study in normal hearing and hearing-impaired subjects

Threshold estimation (17 adults, 40-Hz-ASSR):
- 20 dB down/10 dB up procedure, start at 60 dB nHL
- Stop threshold determination if two consecutive responses are not present

Mean test duration (both ears, 4 frequencies per ear):

*normal hearing* (9): 15 minutes  
*hearing-impaired* (8): 23 minutes
Conclusion

• Resource efficient signal processing and jittered stimulus presentation make ASSR recording achievable on a handheld device

• Short overall test time
  – Chirps to compensate delay on basilar membrane
  – Independent response analyzers for fast test sequence
  – High resistance against electrical interference

• Record ASSR anywhere: Child‘s bed, surgery, audiometry booth, etc.
Purpose of physiological tests

- Tympanometry, Stapedius reflex → Middle-ear, Cochlear recruitment

- TEOAE - not frequency specific, not quantitative, HV < 30 dB → outer hair cell function (Screening)

- DPOAE - frequency specific, quantitative, HL < 50 dB → outer hair cell function (Audiology)

- ABR - transient neural response, not frequency specific → middle-ear function, cochlea function, neural function up to the brain-stem (Screening, Audiology)

- ASSR - tonal neural response, frequency specific → brain-stem and sub-cortical sound processing

- Middle- and late-latency response → sub-cortical and central sound processing
Do you hear the bees flying around the flowers? If not - you should go to somebody to test your hearing using Path medical products!