The cochlea and auditory transduction (and some vestibular)

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This class deals with the perception of sound (balance) and the means by which the brain represents and processes sounds (balance).

Sound is a pressure wave that can propagate in any elastic medium (air, water, steel, . . . ). Sounds are usually produced by vibration of objects, such as the vocal folds, or by other pressure disturbances like turbulence.

The electrical signal resulting from a sound picked up by a microphone is shown below. Sounds have amplitudes and frequencies, as shown. They also have many other properties, discussed as the course goes along.

Frequency is measured as the number of repetitions of one cycle of the waveform per second, in units of Hertz.

Frequency is usually represented logarithmically in the auditory system. log frequency is almost always the correct way to plot frequencies in auditory science.

Amplitude is also measured logarithmically in the auditory system; conventionally, amplitude is given in dB where

$$\text{dB} = 20 \log_{10} \frac{P}{P_0}$$

If the reference pressure $P_0$ is 20 $\mu$Pa (the approximate threshold of human hearing), the dB scale is called dB SPL.

Note that a 20 dB increase in level corresponds to a factor of 10 increase in the sound pressure. The range of hearing is from 0 dB (threshold) to about 100 dB, where sounds become unpleasant to hear.
Sounds are complex patterns in time and frequency (which equates to space within the auditory system). The spectrograms below show the properties of some typical complex sounds. Note the sounds are sums of energy at many frequencies and also have complex temporal patterns.

X-axis - time
Y-axis - frequency, using a scale similar to the cochlea
Z-axis - signal intensity in dB = 20 \log_{10} P

Functionally, the middle ear transforms the pressure in air ($p_T$) to the pressure in the base of the cochlea ($p_V$).

The anatomy of the ear: there are three parts:
1. The **pinna** or external ear collects sound and helps to encode sound source direction.
2. The **middle ear** couples sound from air to the cochlea.
3. The **cochlea** transduces sound to neural activity patterns for transmission to the brain.

The middle ear matches the low acoustic impedance of air to the higher impedance of the cochlea

$$Z = \frac{\text{sound pressure}}{\text{volume velocity}}$$

The ratio of the energy transmitted across an interface to the energy reflected:

$$\frac{\text{transmitted}}{\text{reflected}} = \frac{4r}{(1 + r)^2}$$

$r$ is the ratio of impedances on the two sides of the interface. $r \approx 135$ for air and the cochlea, giving a 15 dB power loss at the air/cochlea interface (factor of 0.03).

The middle ear changes the effective acoustic impedance of the cochlea by the product of the area ratio: eardrum/stapes and the lever ratio (squared): malleus/incus (~29 for the human ear, not quite complete compensation).
The actual motion of the middle ear ossicles is more complex than the lever-ratio model, meaning that the pressure transformation is less than that predicted by the model.

The middle ear transfers sound into scala tympani at the oval window. The sound pressure produces a pressure difference across the basilar membrane, causing it to deflect, indicated schematically below. The deflection is the stimulus to the cochlear transducer cells (the hair cells).

The actual structure of the cochlea and the Organ of Corti.

Sound pressure is coupled into scala tympani (ST). The pressure difference between scala tympani and vestibuli (SV) deflects the basilar membrane, thereby stimulating hair cells (IHC and OHC).

Deflection of the basilar membrane (vertically in the figure below) acts through the geometry of the organ of Corti to deflect the cilia of the hair cells (horizontally in the figure below).

IHC = inner hair cell
OHC = outer hair cell
Forward transduction (shearing motion of IHC stereocilia to electrical current). Transduction channels at the tips of stereocilia are opened by stretching the tip links when the cilia are displaced. The transduction current is $K^+$, driven by the difference in potential between the scala media (+90 mV) and the intracellular potential of the IHC (~-50 mV).

1. At low frequencies, the potential follows the waveform of the signal.
2. At high frequencies, the waveform is filtered away, leaving a depolarization.

At low frequencies, depolarization is in synchrony with the stimulus waveform. At high frequencies, it follows the envelope of the signal.

Inner hair cell receptor potentials in response to short sinusoidal stimuli at various frequencies.

Note two components to the receptor potential:

1. At low frequencies, the potential follows the waveform of the signal.
2. At high frequencies, the waveform is filtered away, leaving a depolarization.

At low frequencies, depolarization is in synchrony with the stimulus waveform. At high frequencies, it follows the envelope of the signal.

The basilar membrane is differentially sensitive to frequency, as shown by the “Best Frequency” scale below. This property arises from variation of the physical properties of the membrane along its length, giving it a resonance to low frequencies at the apex and high frequencies at the base.

Below, the velocity of one point on the basilar membrane is plotted versus the frequency of the sound stimulus. The parameter is the sound pressure at the eardrum. Note:

1. The strong frequency dependence of the response (tuning)
2. The nonlinear amplitude-dependent gain. Gain is larger at low sound levels (the cochlear amplifier)
3. The linearity of the BM post mortem (or after intense acoustic trauma)
Dynamic range compression in basilar-membrane motion (velocity). The motion is linear (slope 1 dB/dB) at low sound levels. It is compressed at moderate levels (22 dB / 60 dB ~ 0.3).

After damage to the cochlea by acoustic trauma (open circles), the motion is linear at all levels.

It is the loss of outer hair cells (OHCs) that causes the disappearance of the cochlear amplifier post mortem.

A similar effect is seen in hearing impairment, where the OHCs may be damaged by loud sounds, ototoxic substances or other insult leading to a loss of cochlear amplification.

OHCs shorten or lengthen in response to their membrane potential. One mechanism is preston, a modified anion transporter that is very dense in OHC membranes. $\mathrm{Cl}^-$ ions move into and out of preston molecules in response to membrane potential, changing the cross-sectional area of the molecule and the length of the cell.

A direct connection between the cochlear amplifier and preston was provided by using chimeric mice between preston knockouts (black mice) and WT mice (brown mice). There was a direct relationship between the lack of preston (abscissa) and the loss of auditory sensitivity (ordinate) in an electrophysiological test.