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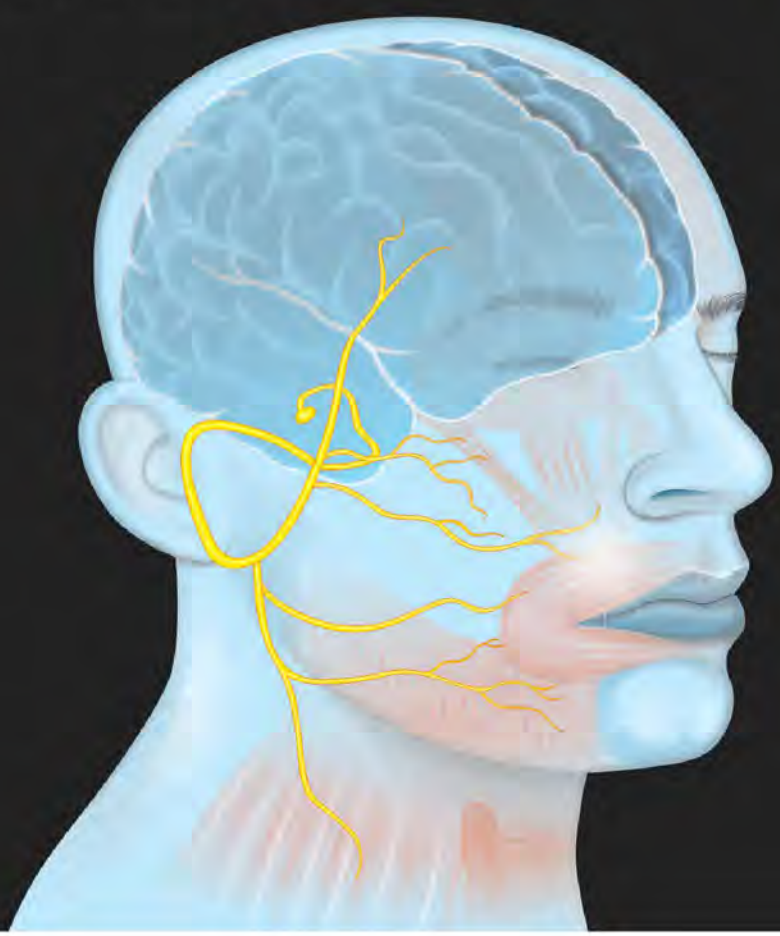
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Symptom Oriented Otolaryngology Head & Neck Surgery

Otology and Pediatrics

Volume 3

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Surgical Physiology of the Ear

2

M Giles

Chapter Overview

Physiology of the Auditory System
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2.2 Definition
2.3 External Ear, Middle Ear, Cochlea
2.4 Retrocochlear Auditory Pathway

Physiology of the Vestibular System
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2.14 Clinical Considerations
2.15 Endolymphatic Duct and Endolymphatic Sac
2.16 Movie Legends

Pearls and Pitfalls

Pearls

- Recent advances in our understanding of the physiology of the cochlea means we are now able explain many acquired and inherited causes of hearing loss.
- Understanding the physiology is essential in clinical examination and testing of the vestibular system.
- In particular, adequate examination of the dizzy patient is impossible without understanding the vestibular-ocular reflex.

Pitfalls

- The patient with bilateral complete vestibular failure (e.g. after gentamycin therapy) will present with no or minimal vertigo and chronic dizziness. Always perform the head impulse test in chronically dizzy patients to avoid missing such cases.

PHYSIOLOGY OF THE AUDITORY SYSTEM

2.1 INTRODUCTION

This section concerns itself with the auditory system from the pinna to the central connections of the auditory nerve. The physiology will be discussed in functional components. The endolymphatic sac (ELS) will be discussed in the section of the physiology of the vestibular apparatus, although dysfunction of the ELS affects the entire inner ear including the cochlea. Some of the physiology of the central auditory pathways is covered in Chapter 4 and this will be pointed out in the text where necessary.

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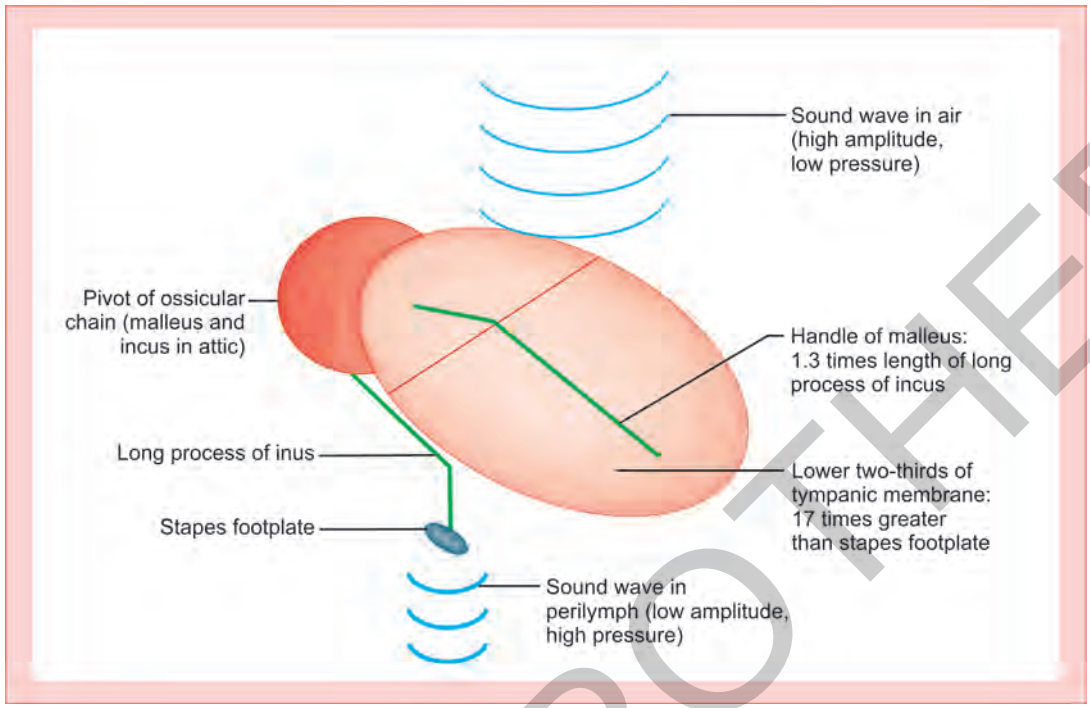


Fig. 2.1: Mechanism of the middle ear.

2.2 DEFINITION

Transduction is a process by which variations in a physical quantity are converted into an electrical signal, or vice versa (e.g. vibration into depolarization of a hair cell).

2.3 EXTERNAL EAR, MIDDLE EAR, AND COCHLEA

Introduction

The elaborate mechanism of the external and middle ear conducts sound from the environment and into the inner ear. Failure of this mechanism produces a *conductive hearing loss*.

Function of the External and Middle Ear Sound Conducting Mechanism

The external and middle ear serve to collect sound waves in air; then reduce their amplitude while increasing their pressure (Fig. 2.1). The process finishes with movement of the footplate of the stapes to produce sound waves in the perilymphatic fluid. This process is called "impedance matching," and is achieved by the external and middle ear without significant loss of acoustic energy.

Imagine a situation in which the external ear was absent, and the oval and round windows presented themselves to the environment on the outside of the skull. Approximately, 99% of acoustic energy would be reflected off the oval window, i.e. the oval window would be an acoustic mirror. This would doom humanity to a permanent conductive hearing loss of approximately 30 dB. This is effectively the case in conductive hearing loss associated with middle ear effusion.

A simple analogy of the mechanism is the stiletto heel, which efficiently concentrates the gravitational pressure of the human body into a much smaller area, enabling the wearer to transmit a deformational pressure into solid objects. Trying to hear without a middle ear and external ear canal is therefore like trying to ruin a vinyl floor while wearing sensible shoes.

The mechanism will be discussed under the functional headings of external ear, tympanic membrane and stapes footplate, and ossicular chain.

External Ear

Pinna

The pinna is a natural "ear trumpet" that funnels sound into the external ear canal. It plays a role in the

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localization of sound, amplifies sound somewhat, and emphasizes high-frequency over low-frequency sounds, which is advantageous when listening to speech.

External Ear Canal

The external canal is a resonance chamber, with a resonant frequency that varies with the anatomy of the ear canal, but averages about 2.5 kHz. This is considered to be a mechanism for accentuating higher frequencies over lower.

Middle Ear

Tympanic Membrane and Stapes Footplate

The tympanic membrane and stapes footplate function as a unit and play a major role in impedance matching. Sound waves in air are collected over the surface of the lower two-thirds of the tympanic membrane (approximately 55 mm^2) and converted into vibration in the much smaller stapes footplate (3.2 mm^2). This reduces the amplitude of the sound wave (in air) to a sound wave (in perilymph) but increases the pressure by a factor of approximately 55/3.2 = 17 (Fig. 2.1).

Ossicular Chain

The ossicular chain acts as a simple lever, because the handle of the malleus is approximately 1.3 times the length of the long process of the incus. The pivot is the incus and malleus head combination in the attic. This will act to decrease amplitude but increase pressure; this time by a factor of 1.3.

The total mechanical effect of the middle ear is therefore approximately 22 (17 times 1.3). The sound wave has its amplitude decreased and pressure increased by 22-fold with no loss of energy. Added to this is a small amount of impedance matching by the pinna and external ear canal, and the phase difference between the movement of the oval window and round window.

Cochlea

Introduction

The cochlea transforms incoming acoustic energy (in the form of a sound wave in the perilymphatic fluid) into bioelectric energy (a change in the membrane potential of the cochlear hair cells). The cochlea has an impressive range of discrimination for pitch (20–20,000 Hz; three orders of magnitude), and response to sound intensity (12 orders of magnitude).

The physiology will be discussed under the following functional components: the fluid compartments of the cochlea, the inner hair cell (IHC) and outer hair cell (OHC), passive tuning (basilar membrane and organ of Corti), active tuning: the OHCs, the stria vascularis, the IHCs and the auditory nerve, summary. The reader interested in more detail of the functioning of the inner ear is referred to the references below.

Fluid Compartments of the Cochlea

The *cochlea* consists of a two and three-quarter spiral, divided into three compartments by the basilar membrane, spiral ligament, Reissner's membrane, and the osseous spiral lamina (Fig. 2.2).

The function of the organ of Corti is best described as if it were uncoiled (Fig. 2.3) or in cross section (Fig. 2.4).

The upper and lower compartments are the *scala vestibuli* and *scala tympani*, respectively, and are filled with perilymph. The *scala media* sits between the scala vestibuli and tympani and contains endolymph.

The scala vestibuli and tympani are continuous at the apex (the *helicotrema*). Sound waves in the incompressible perilymphatic fluid exist only because the compliant round window can follow the movement of the stapes footplate exactly 180° out of phase. This allows vibration of the stapes footplate to propagate sound waves into the scala vestibuli, then across the scala media, into the scala tympani, and ultimately cause motion of the round window (Fig. 2.3).

The *perilymph* is similar to CSF, being rich in sodium and poor in potassium ions. *Endolymph* is a highly specialized fluid, being potassium rich and sodium poor. Cochlear endolymph has a positive electrical potential of +80 mV relative to the perilymph. The ionic environment of the endolymph is partially maintained by the passive barrier function of the basilar membrane and *Reissner's membrane*. This barrier is "leaky" and with time the difference between the endolymph and perilymph fluids would disappear unless it was actively maintained.

To preserve the different compositions of perilymph and endolymph requires *recycling of Na and* K^+ ions by supporting cells and compartments surrounding the OHCs and IHCs, and the *stria vascularis* (*see* later). Some of these passive and active ion flows are depicted in Figure 2.5.

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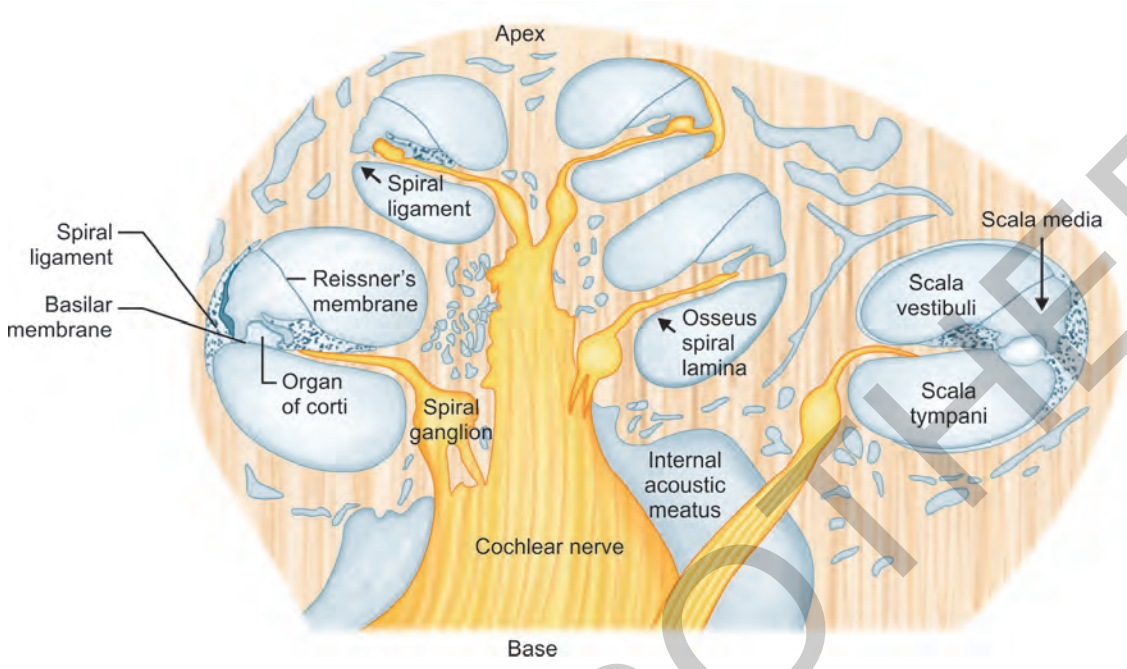


Fig. 2.2: Anatomy of the cochlea.
Source: http://en.wikipedia.org/wiki/Cochlea#mediaviewer/File:Gray928.png [Accessed 4 January 2015].

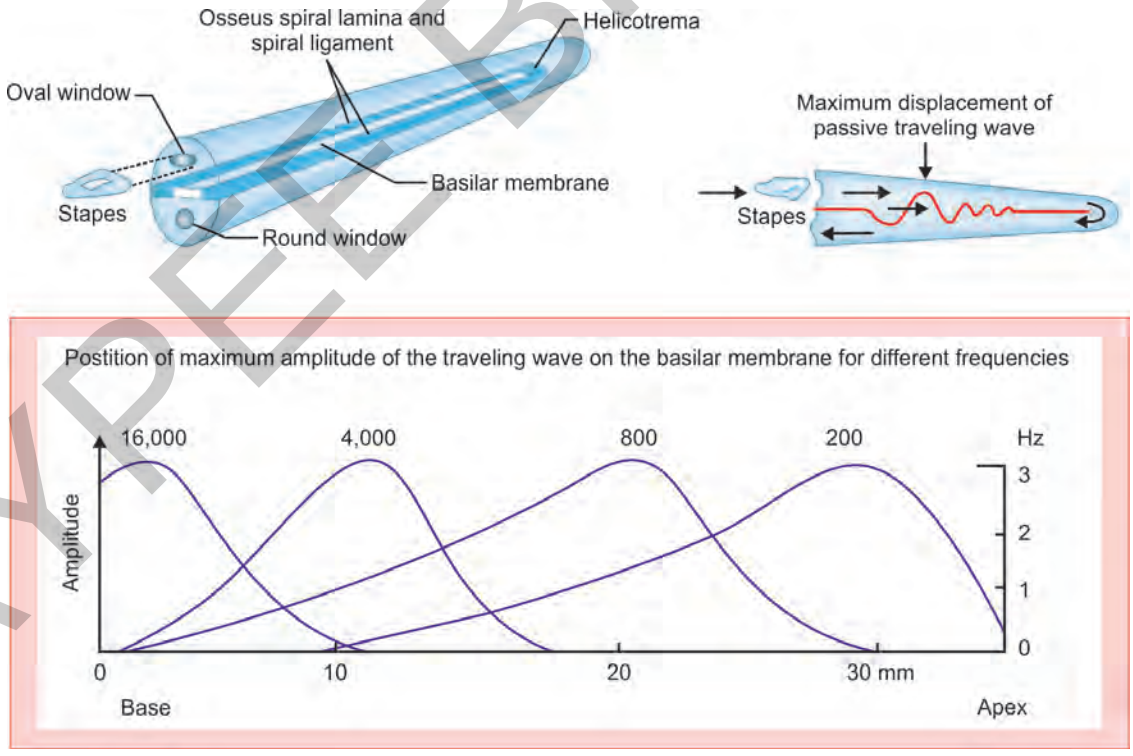


Fig. 2.3: Cochlear mechanics.
Sources: http://www.physics.georgetown.edu/~vincenz/ssm/16/ssm-Lect16.html (top left and right) [Accessed 4 January 2015]; http://www.sfu.ca/sonic-studio/handbook/Basilar_Membrane.html (bottom) [Accessed 4 January 2015].

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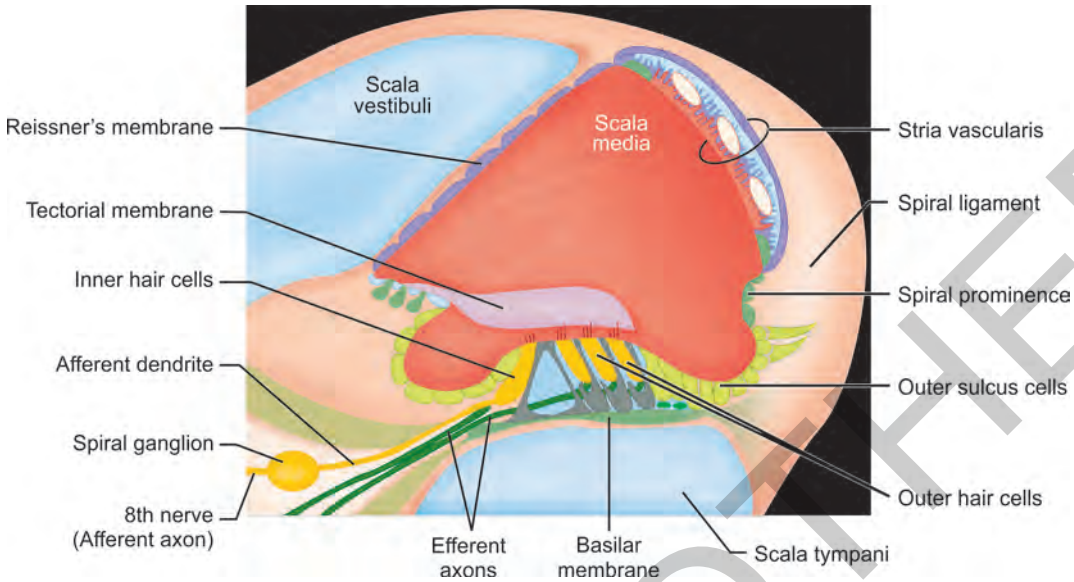


Fig. 2.4: The cochlea in cross section.

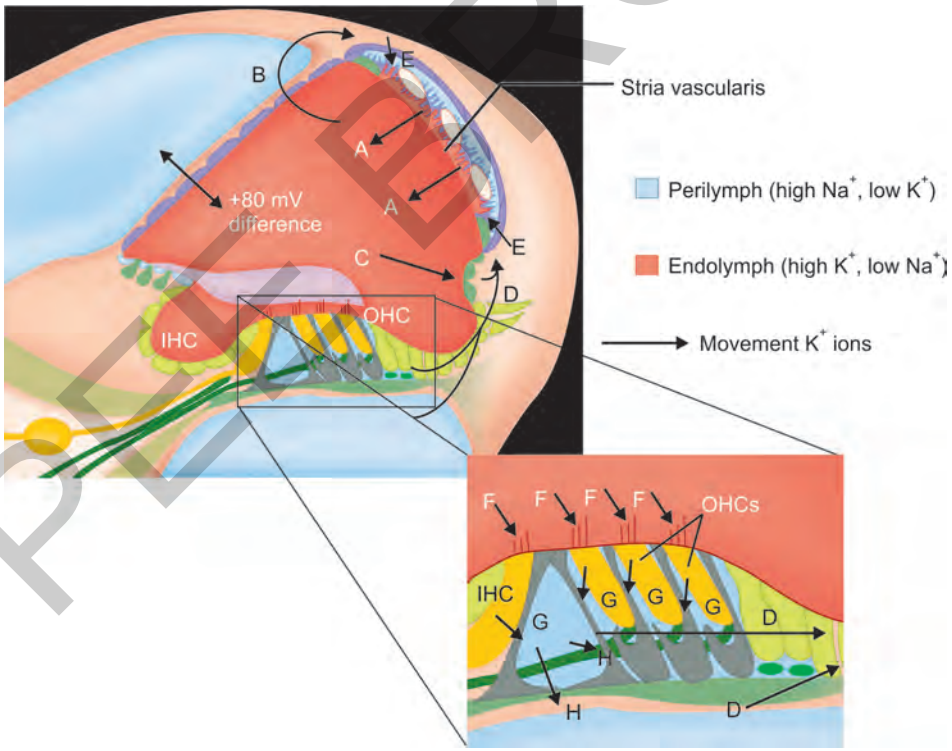


Fig. 2.5: K^+ ion flows within the cochlea. In the close-up of the organ of Corti (lower right), the tectorial membrane has been omitted for clarity. A, active K^+ flow into endolymph from stria vascularis; B, K^+ flow to lateral wall of cochlea via Reissner's membrane and scala vestibuli; C, K^+ flow to lateral wall from endolymph; D, K^+ flow to lateral wall from supporting cells of organ of Corti and scala tympani; E, K^+ entry into stria vascularis from lateral wall; F, K^+ entry via MET channel into OHCs and IHCs; G, K^+ exits OHCs and IHC into perilymph spaces of organ of Corti; H, K^+ entry into supporting cells of organ of Corti and scala tympani. (IHC: Inner hair cell; MET: Mechanoelectrical transducer; OHC: Outer hair cell).
Source: Zdebik, Wangeman and Jentsch (2009).

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Inner and OHCs

There are approximately 11,000 OHCs and 2,500 IHCs per cochlea. A detailed description of the electrophysiology of the hair cells is beyond the scope of this chapter, and the interested reader is referred to Forge and Wright (2002). The IHCs and OHCs are organized within the organ of Corti as shown in Figure 2.4. Displacement of the stereocilia of the IHCs and OHCs causes potassium ions to enter the hair cell from endolymph, leading to depolarization (stimulation) of the cell.

Passive Tuning: The Basilar Membrane and Organ of Corti

From base to apex of the cochlea, the spiral lamina, basilar membrane, organ of Corti, and spiral ligament run from the modiolus to the outer wall of the cochlea.

Previous descriptions of the passive tuning ability of the cochlea emphasized the basilar membrane. The width and stiffness of the basilar membrane changes smoothly from the base to the apex, being shortest and most tense at the base and widest and least tense at the apex. However, the organ of Corti also plays a role in the passive tuning. The organ of Corti increases in height from the base to the apex, thereby changing the mechanical loading of the basilar membrane. The interested reader is referred to de La Rochefoucauld and Olson (2007) for more details.

The scala media compartment is, therefore, passively tuned. The input (sound energy) is converted into a mechanical output—a traveling wave in the basilar membrane (Fig. 2.3). The traveling wave is maximally tuned at a specific point—at the base for high-frequency sound and at the apex for low-frequency sound (Fig. 2.3). "Tuning" means that the wave is not only larger, but also moving more slowly and has a sharper shape.

It is important to stress that the passively tuned traveling wave is markedly enhanced by the active tuning of the organ of Corti (*see* the next section).

Active Tuning: The OHCs

The OHCs are physically "tuned" to the same frequency as the underlying basilar membrane: the height of the cell body and the stereocilia both increase from the base to the apex of the cochlea. When the OHCs attached to the basilar membrane are displaced by a traveling wave, shearing occurs between the stereocilia and tectorial membrane. This displaces the stereocilia, setting in motion electromechanical events that increase the tuning of the travel-ing wave, i.e. increasing the displacement, reducing the velocity and increasing the sharpness of the peak.

The exact mechanism by which OHCs increase the tuning of the traveling wave is still not completely described; but it is known that the +80 mV potential difference between the endolymph and perilymph provides the energy source, and mechanism is dependent on active and rapid alterations in the size of the OHC ("electromotility") following entry of K^+ ions into the OHC (Fig. 2.5). The reader is referred to Ashmore (2008) for a detailed discussion of the current state of knowledge on OHC physiology.

The OHCs are not directly involved with stimulating the acoustic nerve, as only 5% of auditory nerve fibers connect directly to OHCs. The three rows of OHCs provide about 60 dB of amplification of the incoming acoustic signal, and give the organ of Corti fine-tuning for sounds of different frequency.

For sounds below about 60 dB, the function of the OHCs in tuning the traveling wave is vital to creating enough sheering force between the IHC stereocilia and the tectorial membrane to stimulate the IHC. The tuning is so precise that at threshold only about 10 adjacent IHCs are stimulated. Outside this zone, the stereocilia of adjacent IHCs will be displaced, but not enough to cause stimulation.

The OHCs amplify the traveling wave in a nonlinear fashion, the effect being greatest at threshold and progressively less as the signal increases in intensity. Above an intensity of 60 dB, the OHCs do not amplify the amplitude of the traveling wave, but will still sharpen and slow the traveling wave.

Stria Vascularis

Potassium ions are recycled by the supporting cells of the organ of Corti back into the stria vascularis, and from the stria into the endolymph (Fig. 2.5). *Connexins*, also known as *gap junction proteins*, play a vital part in this process. The stria vascularis is responsible for production of endolymph and maintenance of the +80 mV endocochlear potential. It contains specialized secretory cells, which contain numerous mitochondria and Na^+/K^+ pumps that actively maintain the high K^+/low Na^+ environment of the endolymph. The processes involved in the circulation of K^+ within cochlea are discussed in more detail in box.

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K^+ Circulation Within the Cochlea.

The otologist needs a working understanding of K^+ circulation in the cochlea to understand sensorineural hearing loss. The major points are summarized below. The cochlea performs a transduction process, by which a mechanical stimulus (movement of the basilar membrane) leads to depolarization of IHCs. The keys to understanding the transduction are the endocochlear potential (EP), the mechanoelectrical transducer (MET) channel and the circulation of ions. The important ions involved in cochlear function are Na^+, K^+, Ca^{++}, HCO_3^-, and Cl^-. This text box will focus mainly on K^+, as this is the ion that drives the transduction process.

1. Endolymph has a high K^+ concentration and a positive electrical potential (EP) of +90 mV compared to perilymph. Both of these are actively generated by the stria vascularis.
2. The stria vascularis is the energy source for the transduction process.
3. The high EP plus the negative intracellular potential of the intracellular fluid of OHCs and IHCs creates a driving force favoring K^+ entry into the HCs.
4. The MET ion channel is situated in stereocilia of HCs. When open, it allows K^+ to enter the HC. There are approximately 100 MET channels per HC (Fig. 2.6).
5. Displacement of the stereocilia of the HCs modulates entry of K^+ into the HCs by opening and closing the MET ion channels.
6. Entry of K^+ causes the HC to depolarize.
7. In OHCs, depolarization leads to motility of the OHC, enabling active tuning of the basilar membrane (*see* above).
8. In IHCs, depolarization causes release of glutamate into the synaptic cleft at the base of the IHC, and change in the firing of the associated auditory neuron. This process is mediated by Ca^{++}.
9. K^+ leaves the HC into extracellular fluid, and then is returned to the stria vascularis by inter- and extracellular pathways (Fig. 2.5).
10. This recycling of K^+ uses ion channels, ion transporters and gap junctions between nonsensory cells of the cochlea.
11. Electrical and ionic insulation of the endolymph is essential to maintain the high K^+ concentration and EP.
12. Once K^+ enters the stria, it is moved back into the endolymph by an active process. A $K^+/Na^+/2Cl^-$ cotransporter is vital in this process.

Sources: Zdebik, et al. (2009); Shi (2011).

IHC and Auditory Nerve

The IHC is depolarized by sheer forces acting on the stereocilia. This leads to depolarization of the OHC. This in turn triggers Ca^{++} mediated release of neurotransmitter (glutamate) into the synaptic cleft between the IHC and associated auditory neurons at the cell base, altering the electrical activity of the neurons (Fig. 2.6).

There are two types of neuron in the auditory nerve, type I and (imaginatively) type II. The *type I neurons* are myelinated and bipolar with the cell body in the spiral ganglion. The IHCs are joined to type I auditory neurons by synaptic boutons (Fig. 2.6), with about 10–20 boutons per IHC, each bouton connecting to a different auditory neuron. Type I fibers have a natural resting rate of discharge, and the typical effect of IHC stimulation at their characteristic frequency is to increase the firing rate.

Type II neurons are nonmyelinated and their role is not understood. They will not be discussed further.

Summary of Cochlear Physiology

If the response of the inner ear to incoming sound is considered as a whole, then the effects of the passive tuning of the basilar membrane, the active tuning of the OHCs, the function of the IHCs, and auditory neurons can be visualized in Flowchart 2.1.

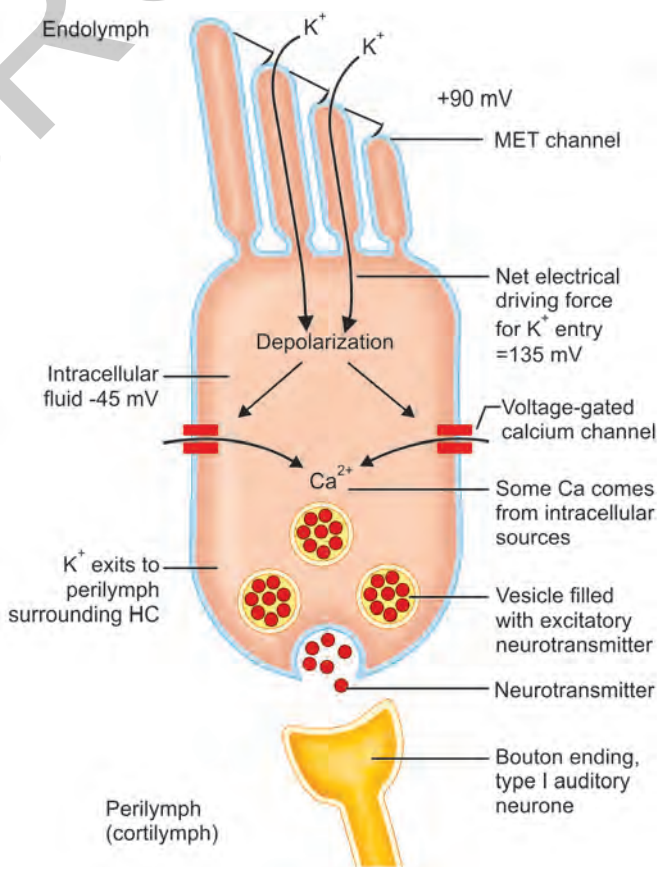


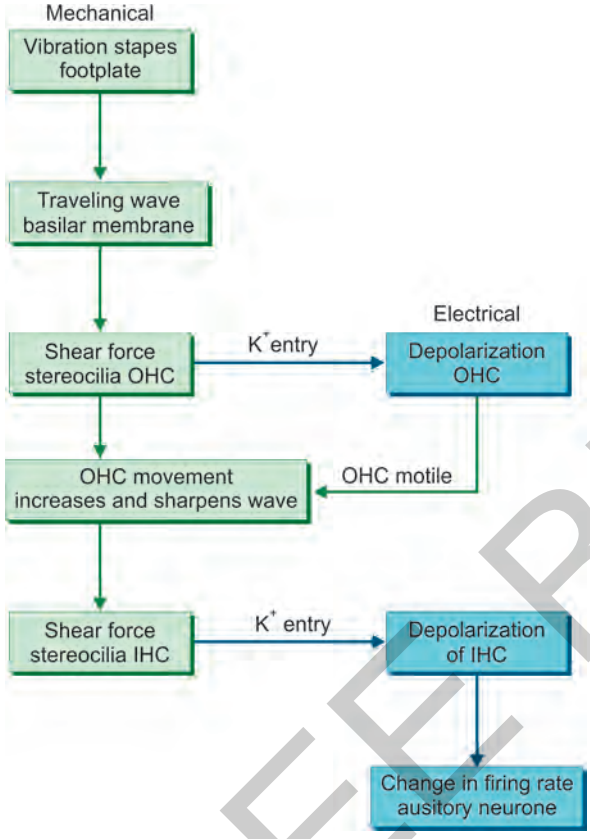
Fig. 2.6: IHC and auditory neuron. Again, the tectorial membrane has been omitted for clarity. (IHC: Inner hair cell; MET: Mechanoelectrical transducer).

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Consequences of Clinical Importance

1. *Loss of OHCs* (e.g. ototoxicity or noise induced hearing loss) can produce a hearing loss of about 40–60 dB. However, above that intensity there is enough displacement of the basilar membrane for the IHCs to respond. However, the cochlea will have a *reduced dynamic range*, with a reduced span of perceptible sound between "just heard" and 'too loud." For sound stimuli above 60 dB, the OHCs do not increase the amplitude of the traveling wave but still act to reduce its speed and increase its sharpness; such patients will therefore also have *reduced ability to discriminate pitch.*
2. The reduced dynamic range and loss of fine tuning can be a considerable source of disability to patients with (for example) noise-induced hearing loss—even the loss is relatively mild and isolated to high frequencies.
3. Excessive fluid (*hydrops*) within the endolymphatic compartment of the cochlea (as in Mèniére's disease) will cause ballooning of Reissner's membrane. In the apical region, the basilar membrane (which is at its most lax at the apex) will also distend. The mechanical properties of the basilar membrane will change, reducing the ability of the organ of Corti to respond to low-frequency sound. This is suggested as the mechanism behind the fluctuating low-frequency sensorineural hearing loss seen in *Mèniére's disease.*
4. Disruption of the microcirculation of the stria vascularis is capable of interfering with the endocochlear potential and circulation of K^+, producing hearing loss. This has been proposed as a mechanism in: noise-induced hearing loss, Mèniére's disease, presbycusis, and sudden hearing loss. This is also the rationale for treating some inner ear disorders with vasodilators such as β-histidine therapy in Menière's disease (Shi, 2011).
5. The various protein molecules that enable the organ of Corti to function may be involved in some forms of *inherited deafness* such as Pendred's syndrome, and connexin-related deafness types 26, 30, and 31. Some of these are listed in Table 2.1 (Forge and Wright, 2002; Zdebik, et al., 2009).

Flowchart 2.1: Green, mechanical process; blue, electrical.



(OHC: Outer hair cell).

Table 2.1: Some of the proteins involve in K^+ recycling in the cochlea and the effects of mutations in the genes encoding those proteins.

Gene	*Encoded protein*	*Effect of genetic defect*	*Clinical diagnosis*
KCNQ4	Ion channel OHC	Reduced exit K^+ OHCs	DFNA2
Barttin	Cl^- transport stria V	Inability to generate ECP	Barrter's syndrome with deafness
KCNQ1/KCNE1	K^+ ion channel stria V	Reduced K^+ recycling	Jervell-Lange-Neilsen syndrome
Pendrin	$Cl^-/HClO_3^-$ ion transporter	Acidification of endolymph	Pendred's syndrome with deafness/enlarged vestibular aqueduct
Connexins 26, 30, 31, 43	Gap junction proteins	Reduced K^+ recycling from HCs to stria	Autosomal recessive nonsyndromic deafness. Connexin 26 mutations = DFBN1
Claudin	Tight junction protein	Loss electrical insulation and ECP	DFNB29

(DFNA: Autosomal dominant deafness; DFNB: Autosomal recessive; OHC: Outer hair cell; ECP: Endocochlear potential).

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6. Loop diuretics such as frusemide are potent inhibitors of the $Na^+/K^+/Cl^-$ co-transporter channel essential to the function of the stria vascularis. Overdose can cause changes in the ECP and ionic environment of the hair cells. This may be the cause of apoptosis of HCs and permanent hearing loss seen with these agents.

2.4 RETROCOCHLEAR AUDITORY PATHWAY

The cell bodies of the *auditory neurones* lie in the spiral ganglion in the base of the osseous spiral lamina. The bipolar neurones have a short peripheral process that synapses with one IHC, and a long axon that runs along the *modiolus* of the cochlea to reach the spiral perforations in the base. It then joins the other axons arising from the spiral ganglion to form the acoustic nerve. The anatomy of the acoustic nerve in the internal acoustic meatus (IAM) and Cerebellopontine angle (CPA) has already been described in Chapter 1. The first-order nerve fibers of the acoustic nerve enter the inferior cerebellar peduncle at the lower border of the pons, and synapse at the cochlear nucleus in the brainstem.

The central connections of the cochlear nuclei are described in Chapter 4, in the section on *Auditory Brainstem Response*. They are of interest to the otolaryngologist because the connections contribute to the auditory brainstem response as discussed in that chapter.

ACKNOWLEDGMENT

The author would like to thank Professor Peter Thorne (School of Population Health, Auckland University, New Zealand) for his considerable help and support in writing this article, and for inspiring me to develop an interest in the fascinating subject of inner ear physiology.

PHYSIOLOGY OF THE VESTIBULAR SYSTEM

2.5 INTRODUCTION

Knowledge of the physiology of the vestibular system is essential to understanding the examination of the patient with dizziness and poor balance, and the management of these patients. This chapter should, therefore, be read prior to the chapters on examination of the patient and management of the patient presenting with dizziness and balance problems.

Otolaryngologists are in a key position to know what life is like with bilateral vestibular system failure, as they are called upon to diagnose vestibular loss after gentamicin treatment for sepsis. The disability caused can be extreme and intuitively seems out of proportion to the limited conscious awareness we have of vestibular sensation. The degree of disability becomes easier to understand when some of the major outputs of the vestibular system are considered (Table 2.2).

Table 2.2: Outputs of the peripheral vestibular system.

Structure	*Output*	*Importance*	*Consequences of failure*
Oculomotor nuclei Cranial nerves III, IV, and VI	Vestibular-ocular reflex (VOR)	Smooth pursuit Gaze stabilization	Osscillopsia Nystagmus Reduced dynamic visual acuity
Cerebellum	Direct output to cerebellum from both vestibular nuclei and the vestibular nerves	To assist in co-ordination of head, eye, and body movements	Ataxia, postural instability Abnormal smooth pursuit
Cervical spinal motor neurons	Vestibulocolic reflexes	Maintain stability of head in relation to body	Ataxia, postural instability Osscillopsia and reduced dynamic visual acuity during locomotion
Lower spinal motor neurons	Vestibular-spinal reflex	Maintain stability of trunk Assist with gait	Ataxia, postural instability Increased fall risk Abnormal gait

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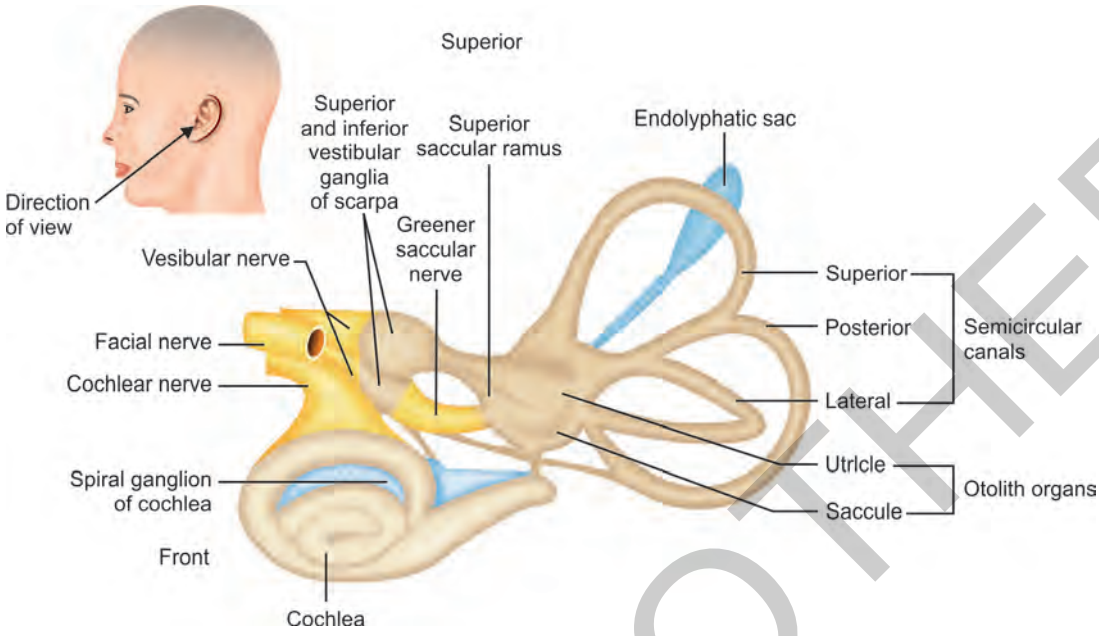


Fig. 2.7: The static and kinetic components of the vestibular apparatus.
Source: http://instruct.uwo.ca/anatomy/530/530notes.htm [Accessed 4 January 2015].

2.6 VESTIBULAR APPARATUS

Introduction

The vestibular apparatus detects linear acceleration and rotatory motion, plus the direction of gravity. Like the cochlea, it detects mechanical stimuli and transforms them into a change in neural activity. Detection is achieved by a set of 10 "push-pull pairs" of structures, and there is no equivalent of the OHCs of the organ of Corti to amplify the stimulus. It functions to assist with balance and motion via reflex pathways, and sometimes to provide conscious awareness of motion. It is divided into two functional parts: the *otolith organs* (saccule and utricle) detect linear acceleration and gravity; and the *semicircular canals* (horizontal, anterior, and posterior) detect rotation (Fig. 2.7). Among its most important actions is to assist visual fixation while the head is in motion; it does this via the *vestibular-ocular reflex* (usually abbreviated to VOR).

The vestibular apparatus will be discussed under the following functional headings: endolymphatic fluid and hair cells, otolithic organs, and semicircular canal system. The vestibular neurons, the vestibular-ocular reflex, and other central connections will be discussed in the following section.

Endolymphatic Fluid and Hair Cells

Like the cochlear endolymph, the *vestibular endolymph* is rich in potassium. This plays a similar role in hair cell response to stimulation, as it does in the organ of Corti. Specialized dark cells, distributed around the lining of the vestibular apparatus, maintain the high concentration of potassium. There is no equivalent of the +80 mV endocochlear potential in the vestibular apparatus.

The *vestibular hair cells*, like those found in the cochlea, are specialized neural cells surmounted with *stereocilia*. The vestibular hair cells, however, have a *kinocilium*. Deflection of the stereocilia toward the kinocilium allows potassium ions to enter the hair cell, and the cell depolarizes. The opposite movement causes hyperpolarization of the hair cell. The approximate number of hair cells per vestibular sensory organ is as follows: utricle 33,000, saccule 19,000, and each semicircular canal ampulla 8,000. Each hair cell has bouton synapses from one or more vestibular neurons at its base, both afferent and efferent.

Otolith Organs (Saccule and Utricle)

The *saccule* and *utricle* (also called the otolith organs) detect linear acceleration and gravity. Their sense organs (maculae) consist of plaques of neurosensory hair cells,

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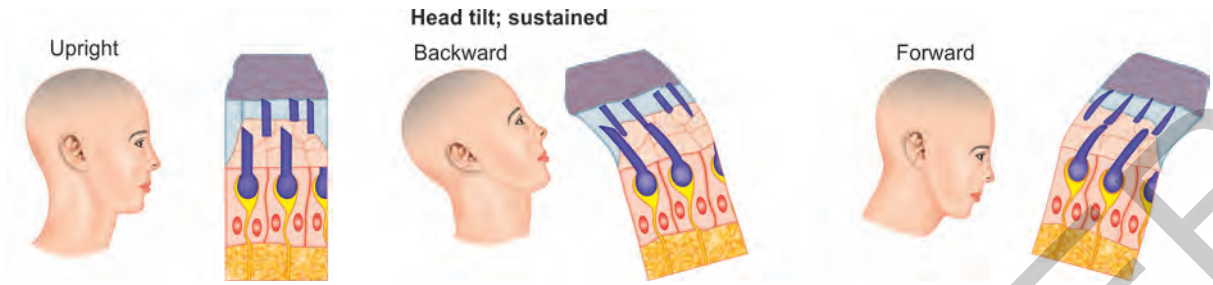


Fig. 2.8: A typical response of the otolith organs to gravity.

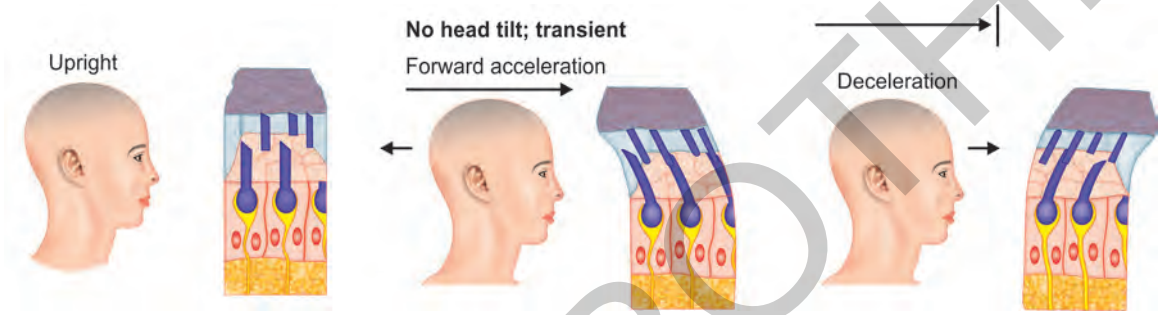


Fig. 2.9: A typical response of the otolith organs to linear acceleration.

with the stereocilia being embedded in a gelatinous matrix studded with crystals of calcium carbonate, the "otolithic membrane." Because the otolithic membrane is denser than the endolymph it forms an inertial mass, which will be displaced by linear acceleration and gravity. Movement of the membrane causes sheering forces on the stereocilia of the underlying hair cell, leading to either depolarization or hyperpolarization.

The maculae of the utricle and saccule are at 90° to each other, with most of the utricular macula lying in the horizontal plane and most of the saccular macula in the vertical. The orientation of the hair cells in the maculae of the utricle and saccule varies, and the macule and saccule for the left and right ears are mirror images of each other in this respect. This increases the sensitivity of the vestibular system; the left and right utricle and the left and right saccule acting as "push-pull pairs." In any given position of the head, on one side of the head some of the otolith organ hair cells will depolarize and some hyperpolarize, and the exact opposite will happen with the contralateral otoconial membrane and hair cells. The interested reader is referred to the references for more detail.

Figures 2.8 and 2.9 depict the response of a small part of the macule of the utricle in response to gravity (Fig. 2.8) or linear acceleration (Fig. 2.9). This change in position may be stable (the effect of gravity) or dynamic (the effect of linear acceleration). The exact opposite response will occur in the contralateral macule.

Semicircular Canals

The semicircular canals detect head rotation. The three semicircular canals—lateral (or horizontal), anterior (or superior), and posterior—have the same basic structure and are perpendicular to each other (Fig. 2.7).

Each semicircular canal in one ear will have a pair on the contralateral side in the same plane, making a "canal plane pair." These pairs are as follows:

1. The two lateral semicircular canals (abbreviated to RL LL)
2. The right anterior and left posterior (RALP) canals
3. The left anterior and right posterior (LARP) canals.

In describing the function of the semicircular canals it is convenient to discuss them in these plane pairs. It will also be assumed that the endolymphatic fluid "flows" within the semicircular canal, when it does not (the canal is sealed at one end). The displacement of the cupula discussed below occurs through hydrostatic pressure, not actual flow. This inaccuracy does, however, make the discussion easier to understand—at least it does for the otolaryngologist writing this chapter!

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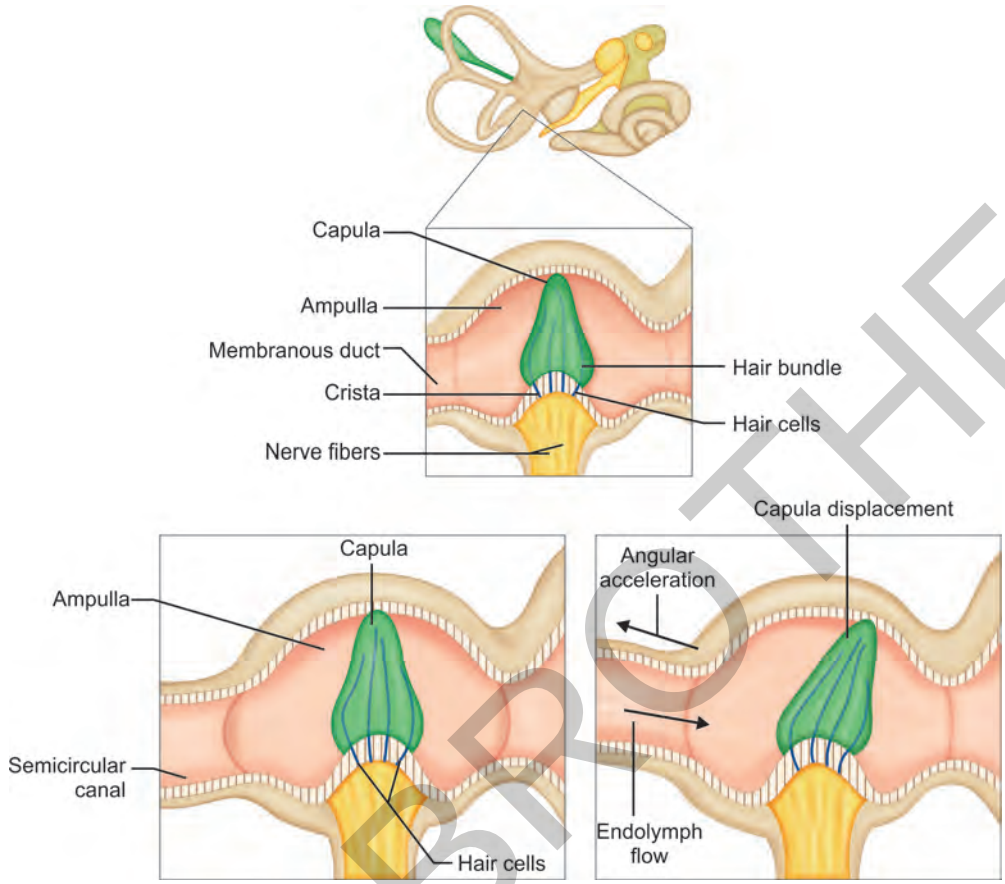


Fig. 2.10: The ampulla of a semicircular canal.
Source: http://what-when-how.com/wp-content/uploads/2012/04/tmp15F77.jpg [Accessed 4 January 2015].

Semicircular canal anatomy: Each canal consists of a smooth duct, open at one end into the remainder of the vestibular apparatus and sealed at the other by a gelatinous plug (*cupula*) and a mound containing hair cells and vestibular neurons (*crista*) located inside a swelling (*ampulla*) (Fig. 2.10).

Basic function: The endolymph within the duct of the semicircular canal has inertia, and is not attached to the walls of the duct. Rotating the head in the plane of a semicircular canal will cause the endolymph in that canal to lag behind movement of the canal, and the net effect is to push or pull the cupula in the opposite direction to the head rotation (Fig. 2.10).

It is convenient to label the fluid movement in any given semicircular canal at any given moment either toward the ampulla (*ampullopetal*) or away (*ampullofugal*). If the endolymph in any given semicircular canal is in an ampullopetal direction, the fluid in the other canal of that plane pair will be moving in an ampullofugal direction—the two canals of the plane pair forming a *hydraulic* "push-pull pair" as shown in Figure 2.11.

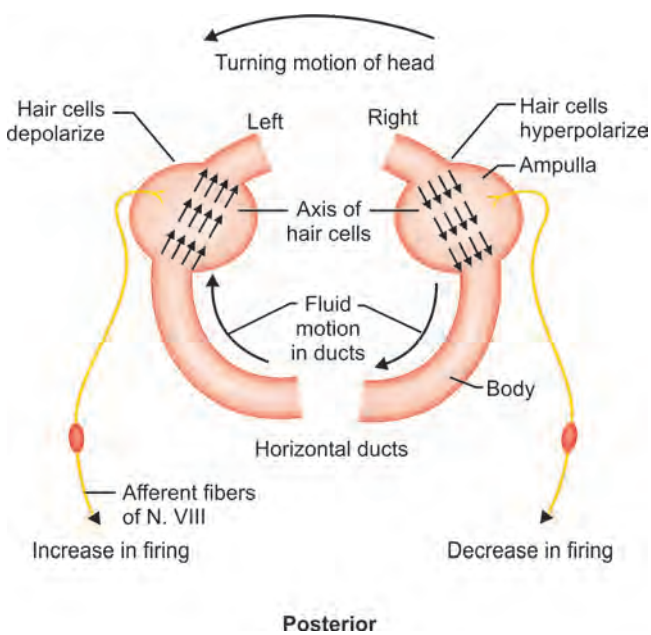


Fig. 2.11: The mechanical and electrical "push-pull pair" of the lateral semicircular canals.

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He has received Presidential Citations from the American Head and Neck Society and NZSOHNS. He has delivered keynote Lectures in several International meetings, including Eponymous Lectures in the UK, USA, and New Zealand.

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