CHAPTER 1.

UNDERSTANDING THE EAR

Introduction

The ear is a series of three compartments, the external, middle and inner ears. Fortunately, diseases of the ear usually afflict one compartment at a time. Therefore to understand the presentation of disease, one requires an understanding of each compartment's anatomy and functions.

Figure 1: Compartments of the ear. Anatomically the ear divides into external, middle and inner ear divisions. As a rule, disease tends to affect only one compartment at a time. This helps the clinician considerably during diagnosis of ear pathology. eam: external auditory canal; tm: tympanic membrane; ow: oval window.

1. ANATOMY AND PHYSIOLOGY

1.1. EXTERNAL EAR

Figure 2: The normal pinna (auricle), surrounded by the helix, with the Y-shaped antihelix within. The stub of cartilage anterior to the EAC and conchal bowl is the tragus.

The pinna (auricle) and external canal are similar to a hearing horn. In man, the pinna has only rudimentary sound gathering purposes and is largely expendable. The external canal, by contrast, has three functions:
a) Sound Conduction
The external canal permits sound to reach the eardrum and Ossicular chain. Total obstruction, as in congenital atresia, produces deafness as severe as 70-80 decibels. If the canal is blocked by debris, hearing is lost appreciably only when total canal occlusion occurs. A small chink between obstructing Exostoses or around a mass of wax may render the blockage asymptomatic.

b) Defence of the tympanic membrane
i. S-curve. The entrance to the external canal is guarded by an overlap of the conchal bowl cartilage. The canal itself is curved, sometimes markedly so. These factors prevent direct penetrating injuries.
ii. Sensitivity. The canal is progressively highly sensitive towards the tympanic membrane. This helps avoid self-injury during cleaning attempts or other intrusions.
iii. Cerumen. The canal “wax” discourages intrusion by insects and helps collect dust for expulsion.

c) Self-Cleaning
The squamous epithelium of the canal has the unique ability to constantly migrate towards the exterior, originating from the tympanic membrane. This migration carries dust, wax and keratin laterally, preventing “silting-up” of the deep canal.

1.2. MIDDLE EAR

Principal Structures
a) Tympanic membrane
b) Ossicular chain
c) Eustachian tube
d) Mastoid air cells

Figure 3: The middle ear, endoscopic view, showing the v-shaped drum, and the attached malleus-incus-stapes chain. The round window is in the foreground, the tubal orifice to the rear, right.

a) Tympanic Membrane
The eardrum has a shape similar to the shallow, curved cone structure of a loudspeaker, with the centre at the tip of the handle of the malleus. Due to the angulation of the external canal, the malleus appears to pass postero-inferiorly to the observer. The cone shape provides optimal acoustic pick-up. The membrane is normally taut and transparent, but becomes slightly sclerosed or “milky” with age.
b) Ossicular Chain

Figure 4: The Ossicular chain. The ossicles form a lever system and a piston mechanism. The malleus and incus function as a unit, rotating on an axis between the anterior malleolar ligament and the short process of the incus. The shorter incus thus permits greater mechanical leverage on the piston, formed by the stapes

*M: malleus; I: incus; S: stapes.*

The three ossicles (malleus, incus, stapes) function as a lever system (malleus, incus), which rotates to drive a piston (stapes). The malleus handle is 1.3 times as long as the incus, and the total area of the eardrum is approximately 20 times the area of the stapes footplate at the entrance to the cochlea. These two factors combine to enhance sound transmission though the middle ear into the cochlea.

c) Eustachian Tube

The Eustachian tube extends from the post-nasal space to the anterior mesotympanum. Its prime function is to aerate the middle ear, to permit the Ossicular chain, to vibrate as freely as possible. In addition, the tube maintains middle ear pressure as near as possible to the external atmospheric pressure, to minimise tension or strain on the drum.

Figure 5: Anatomy of the Eustachian tube, extending from the PNS on the right, to the middle ear, left. Two thirds is cartilaginous, the rest, bony.

The tube is normally closed, to exclude speech and innate bodily sounds, and also to prevent infection. It is opened during deglutition, yawning or other palatal actions by the tensor palati. This muscle hooks around the pterygoid hamulus, forming a midline raphe with its counterpart in the middle of the soft palate. The raphe provides inferior anchorage, whilst the muscle pulls down on the tubal cartilage to open the lumen of the tube. At the same time, the soft palate is pulled superiorly, shutting off the nasopharynx. This prevents spillover into the nose during opening of the tube, preventing soiling of the middle ear during deglutition.

Air within the middle ear dissolves into the bloodstream. Opening the tube permits a re-supply to enter the ear to maintain normal pressure: failure of this mechanism is the basis for many of the major middle ear disorders.
1.3. INNER EAR

Components

a) Cochlea: hearing
b) Vestibular Apparatus: balance
c) Endolymphatic duct and sac

d) Mastoid Air Cell System

Behind the external canal is the stub of mastoid bone to which the sterno-mastoid muscle of the neck attaches. Within the mastoid is a "honeycomb" of interconnecting air-filled cells, which are into continuity with the middle ear. The mastoid cell provide a much larger surface area to permit resorption of air from the middle ear, and the larger volume provides a buffering effect against sudden external air pressure changes.

a) Cochlea

The "snail shell" of the cochlea contains the array of sound receptor cells. Cells at the basal end receive the higher frequencies (8000cps); those at the apex, the lower tones 250 cps. The cochlea is a combined transducer and "computer terminal". Sound (kinetic energy vibrates the tectorial membrane, causing distortion of the filaments of the hair cells of the organ of Corti. This produces action potentials (electrical energy). These potentials are coded and relayed via the acoustic nerve the central computer, the brain itself.
b) Vestibular Apparatus

The vestibular balance mechanisms have two parts: the utricle and saccule, and the semicircular canals. The utricle and saccule are essentially tufts of hair cells, over which is a layer of gelatinous glycoprotein that is covered by a layer of calcium phosphate crystals – otoconia. Movement displaces the gelatinous layer, stimulating the hair cells that form action potentials that relay to the brain via the vestibular nerves.

The saccule detects vertical accelerations and head tilting in the linear plane.

The utricle detects horizontal acceleration and the effects of gravity.

The superior, lateral and posterior semicircular canals are in an interlinking three-dimensional pattern, and detect rotatory movement in all directions. Each has an ampulla at one end containing a cupula – a gelatinous mass containing sensory hair cells but without the otoconia of the utricle or saccule. They thus have no gravitational action as the cupula has the same weight as the fluid around.

Displacement of the otoconia and their irritation of the ampullary hair cells cause benign positional vertigo.

c) The Endolymphatic Sac

The sac (saccus endolymphaticus) Conjoining tubules from the saccus and utricle form the endolymphatic duct, which passes out of the otic capsule to form the sac, in the dura of the posterior fossa, just medial to the lateral sinus. The function remains somewhat uncertain, but appears to be responsible for the turnover of the endolymph – the fluid that fills the membranous labyrinth – to ensure its volume stability and composition. The fluid is evidently resorbed in the sac by phagocytic action. Failure leads to endolymphatic hydrops.

2. CLINICAL ASSESSMENT OF THE EAR

2.1. HISTORY

Disease in each compartment of the ear presents with one or more of the cardinal ear symptoms:

a) Pain
b) Discharge
c) Deafness
d) Tinnitus
e) Imbalance

A sixth symptom, cranial nerve palsies, especially facial palsy, may also occur.

Interrogation of each must be painstaking, following the usual lines:

i. Site
ii. Nature
iii. Severity
iv. Duration
v. Onset
vi. Causative
vii. Aggravating/Relieving factors
viii. Associated factors
ix. Radiation (pain)

a) Pain

Enquire as to precise nature of the pain, duration severity. Particularly ascertain if the pain is within or near the ear e.g. the temporo-mandibular joint. All five cardinal symptoms are covered in detail in other Chapters.
b) Discharge

Ascertain the side/s, any evident causative factor, e.g. URTI, and also the duration, quantity and the nature of the discharge, e.g. watery, foul, bloodstained.

c) Deafness

Question the duration, speed of onset, or any obvious cause. Sensorineural losses are usually high frequency, causing difficulty hearing ringing sound or diminishing speech discrimination.

d) Tinnitus

The quality of the abnormal sounds in the ear readily lead to the origins of tinnitus. Gurgling and rustling are external in origin, popping or crackling the middle ear. Buzzing/ringing/electronic sounds indicate the inner ear. Note that conductive deafness often enhances both normal and pathological tinnitus by excluding ambient noise that would otherwise muffle these sounds.

e) Imbalance

Of all the symptoms the history is most important in imbalance cases. Dizziness presents in several forms that are set out in the chapter on Inner Ear Symptoms. The clinician must determine whether the problems is vertigo, a drop attack, disequilibrium, ataxia, syncope, a confusional episode or a psychological episode, or combinations of several. This may be a daunting task, requiring patience and a detailed interrogation, avoiding diversion. Blackouts, faintness or syncope are CNS in origin; the ear does not cause loss of consciousness.

2.2. EXAMINATION

When inspecting the ear, a clear canal and bright lighting are essential for accuracy. Given an adequate view of the eardrum and a through history, most external and middle ear diseases can be diagnosed readily.

When handling the ear, exert a feather-light touch. Patients appreciate this and the technique helps settle fractious children, the latter especially if accompanied by soothing speech.

The basic observations of inflammation, swelling, and tenderness are necessary.

a) Pinna

Most pinna conditions will be self-evident, but parotid swellings and retro-auricular surgical scars may be less so.

b) External canal

Ensure that the auriscope has a bright bulb, fresh batteries, and begin with a 4mm speculum if possible; this will give a better view. Larger speculae are usually ineffective.

Straighten the canal by drawing the pinna posterolaterally – this reduces the overhang of the conchal cartilage that normally produces the canal S-bend that protects against penetrating injury.

Introduce the auriscope using the index finger against the skull to prevent jabbing discomfort.

Clean the canal as required (see the chapter on Cleaning the Ear).
c) Eardrum

The entire drum is not usually seen through one view of the auriscope and a composite view in the mind’s eye is obtained by moving the field of vision around, using the 4 mm speculum, or smaller if necessary.

i. Shape

The drum should have a shallow curve loudspeaker shape. Local areas of atrophy and retraction are common after infection or grommet insertions.

*Figure 8: The normal eardrum, transparent, with the handle of the malleus passing postero-inferiorly. The incus can be seen posterior to the malleus.*

Chronic Eustachian insufficiency results in retraction, then collapse of the membrane. This collapse begins in the posterosuperior quadrant and extends to involve the entire tympanic membrane (see the chapter on tubal insufficiency). Localised retractions of the pars flaccida are relatively common. However, crusting and debris accumulation in association with such retractions are to be treated with suspicion, as these may indicate the development of cholesteatoma. Granulations on the tympanic membrane are frequently associated with chronic middle ear disease and should be approached with a high degree of suspicion.

ii. Colour

The normal tympanic membrane is a pale grey, transparent shade with perhaps a pink or beige tint. With age, the drum becomes somewhat milky in appearance. Patches of chalky material in the drum are due to tympanosclerosis; in the absence of deafness these are not clinically significant. When an effusion is present, the drum may have a subtle yellow or waxy discolouration; bubbles and fluid levels in retrotympanic fluid may be present, but difficult to see.

iii. Mobility, Autoinflation

A pneumatic speculum may demonstrate drum immobility if an effusion fills the middle ear. However, using autoinflation as a test of Eustachian tubal patency (Valsalva manoeuvre) is unreliable, as many normal people cannot perform this efficiently.
2.3. CLINICAL HEARING

ASSESSMENT

a) Vocal Testing

A preliminary assessment of a patient's hearing can be gained by testing the ability to hear the examiner's voice at graded levels of loudness. The table below gives a rough guide to the patient's ability to hear speech at one metre, in quiet surroundings. The examiner's mouth should be hidden to prevent lipreading and the contralateral ear should be masked with rustling paper.

<table>
<thead>
<tr>
<th>Vocal Loudness Level</th>
<th>Approximate level of Hearing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Whisper</td>
<td>0-15 db</td>
</tr>
<tr>
<td>Soft Voice</td>
<td>25-30 db</td>
</tr>
<tr>
<td>Conversation</td>
<td>35-40 db</td>
</tr>
<tr>
<td>Loud Voice</td>
<td>45-55 db</td>
</tr>
<tr>
<td>Shout</td>
<td>60-70 db</td>
</tr>
</tbody>
</table>

b) Tuning Fork Testing

Two tests are performed: the Weber, and the Rinne. A third alternating test is handy to compare ears. Generally, a heavier weight 512 cps fork is used, as this is suitable to detect conductive losses that are often maximal at this frequency. In all tests the fork is activated by bouncing the middle of the side of one prong off the hard bone of the knee. This gives a pure tone, vis a vis tapping the prong on a hard surface, where an atypical frequency may result.

Figure 9: Normal tuning fork responses. The Weber is heard in both ears, and the Rinne is heard with the fork opposite the ear, less when applied to the mastoid (Rinne positive).

i. Weber test

This is performed by pressing the vibrating fork to the midline of the skull (forehead or vertex, or to the chin of a clenched jaw). In normal subjects, or those with symmetrical sensorineural deafness, the sound is heard “in the middle” or “all over”.

Figure 10: Sensorineural deafness (shaded side). The Weber refers to the better ear. The Rinne test is positive in both ears.

When a unilateral sensorineural loss is present, the sound is heard in the better ear.

The sound is heard in the deaf ear in conductive deafness.
ii. Rinne test

This test has two steps. The patient is asked to listen carefully to two sounds. Firstly, the resonating fork is held with the prongs in alignment about 4 cm out from the ear. The fork is then quickly transferred and held firmly against the mastoid, clearing away hair that may dull the sound. The patient is asked which is the louder sound.

The first sound is louder (Rinne positive) in normal ears or in sensorineural deafness. The second sound is louder (Rinne negative) in conductive deafness.

Figure 11: Conductive deafness. The Weber is heard on the (shaded) deaf side, and the Rinne test is negative in that ear (heard louder on the mastoid).

In sensorineural deafness, a false negative response may be due to the sound vibration of the contralateral better ear, although the patient may offer that the sound is heard in the opposite ear. If this is suspected, the better ear is masked with rustling paper, in which case the deaf ear returns a positive Rinne response.

iii. Alternating test

Commonly, patients confuse a blocked feeling in the ear with true deafness, To roughly test this, the tuning fork is held out from each ear in rapid succession and the patient is asked to judge
whether a difference is present; this is surprisingly effective.

2.4. CLINICAL BALANCE TESTS

a) Hallpike manoeuvres
Otoconia from the utricle or saccul e may work loose causing benign positional vertigo. To assess this the patient is sat up on a couch positioned such that when laid down the head extends beyond the top of the couch. Whilst sitting, the head is turned hard right, and then the patient is rapidly laid back on the couch with the head dependant as far as possible. The test is then repeated with the head to the left instead. If BPV is present, vertigo is provoked by one move or the other, as otoconia shift in the posterior semicircular canal. Concurrently, nystagmus is seen in the eyes.

b) Unterberger test
Assessment for a vestibular dysfunction. The patient stands with the eyes closed then walks “on the spot”. With a vestibular weakness turning or veering to the affected side occurs.

c) Romberg test
This checks the patient’s proprioceptor function (in ataxia this is impaired). Given eyesight and vestibular function, one can compensate for lack of joint control. The patient is stood, eyes shut and feet together for 30 seconds. Ataxic (CNS-damaged) patients will sway and perhaps fall - a positive result.