Chapter 1: Examination of the ear

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The practice of medicine demands the taking of an accurate history and carrying out a careful clinical examination. This principle applies to otology as much as to any other branch of medicine, and should not be forgotten in the rush for 'high technology' investigations which may be invasive and add little to the diagnostic process. The symptoms and signs associated with ear disease must be elicited, together with those which the patient may not recognize as being related to disorders of the ear (see also Volume 2, Chapter 6).

History

Symptoms of ear disease include otalgia, discharge, disorders of auditory perception (mainly hearing loss), tinnitus, vertigo and headache.

Otalgia

Pain in the ear may come from pain receptors in the external or middle ear whose afferent fibres lie in the fifth, ninth and tenth cranial nerves and second and third cervical nerves.

As the skin is so closely applied to the meatal and auricular perichondrium, severe pain may be associated with an external otitis having minimal clinical signs. Senturia (1973) stated that severe otitis externa can be one of the most painful disorders known. Note must be taken of long-standing dermatitis, usually of the eczematous type; trauma to remove wax, may predispose to infection but be neglected in the history. Herpes zoster infections of the fifth and seventh cranial nerves or the upper cervical nerves frequently begin with pain, the diagnosis only becoming apparent when the vesicular eruption on the pinna or meatus occurs. Other painful conditions of the external ear include polychondritis helicis (chondrodermatitis nodularis chronica helicis) and squamous carcinoma.

Myringitis bullosa haemorrhagica is a painful condition occurring spontaneously and resolving within several days. A vesicular eruption is seen on the tympanic membrane which may be associated with bleeding and a serious discharge.

Otitis media, although a frequent cause of otalgia, is subject to overdiagnosis (Bluestone and Cantekin, 1979).

Children who have otitis media with effusion (glue ear) may have a history of previous upper respiratory tract infection and earache. Otalgia is said to occur in some children due to eustachian tube dysfunction, when the middle ear pressure is markedly reduced leading to retraction of the tympanic membrane. The pain usually occurs at night when the child has been sleeping and may be due to venous congestion in the eustachian tube area with reduced frequency of swallowing and, consequently, failure of middle ear ventilation. On arising or sitting up the congestion clears, the eustachian tube opens and the pain disappears. Sometimes the child's crying, because of the pain, leads to hyperaemia of the tympanic membrane and misdiagnosis of acute otitis media.
Sudden spontaneous resolution of pain in cases of true otitis media indicates perforation of the tympanic membrane. Pain is not a feature of chronic otitis media unless there is an associated otitis externa, or more ominously dural inflammation. Less frequently, severe pain in a chronically discharging ear may reflect neoplastic change.

Pain may be referred to the ear from other areas supplied by the fifth, ninth and tenth cranial nerves and the upper cervical nerves. Thus when otalgia is a presenting symptom and no local disease is found in the ear, a distant cause must be considered. Usual sources of referred otalgia are dental disease, lesions of the posterior tongue, pharynx and larynx.

**Aural discharge (otorrhoea)**

Otitis externa may present with irritation and a watery odourless discharge. A clear fluid discharge from the ear after trauma may be indicative of a cerebrospinal fluid leak through a dural tear, often over the tegmen tympani and roof of the external auditory meatus.

When discharge contains mucus, it must have arisen from glands within the middle ear cleft, passing into the external auditory meatus either from an open mastoid cavity or through a tympanic membrane perforation.

A thick brown discharge of liquefied wax may occur in an otherwise healthy ear but often heralds an acute otitis media, particularly if it contains blood and pus. In chronic otitis media, the discharge is often long-standing and characterized by a foul smell due to saprophytic organisms. Cholesteatomatous debris may be discharged, such patients frequently presenting because of the embarrassing nature of the smell. In a chronically discharging ear, the onset of bleeding is an ominous sign, indicating the possibility of neoplastic change.

Bleeding from the ear usually follows trauma but in rare cases may occur from glomus tumours or vascular anomalies in the middle ear or external meatus.

**Abnormalities of auditory perception**

Deafness is the term most commonly used by patients to indicate an abnormality or change in their hearing acuity. Some idea of the level of auditory hearing loss can be obtained from the history by asking about their difficulty in varying social situations. Conversation in a quiet environment is conducted around 40 dB hearing level, a door bell output is, on average, 60 dB and conversation on the telephone between 40 and 70 dB within a limited frequency band of 200-1200 Hz. A person with a conductive loss appears to hear better in a noisy environment, usually because the speaker has raised the intensity of the voice. This phenomenon is known as paracusis Willisii and is usually associated with otosclerosis. In sensorineural hearing loss, there is reduced discrimination of speech, particularly in background noise. The ability of a person with sensorineural deafness to discriminate speech is not necessarily helped when the speaker raises the intensity of the voice. Indeed the listener may say: 'Don't shout, I'm not deaf'.

**Recruitment** of loudness is characteristic of a cochlear loss. A relatively small increase in the intensity of the auditory stimulus may cause frank discomfort to the listener. Poor
speech discrimination without recruitment, especially if unilateral, suggests auditory nerve
damage.

Tonal changes in auditory perception are usually expressed as 'one ear not sounding
like the other' or 'tinny'. Diplacusis is the apparent difference in the pitch of a tone between
the two ears and is associated with conditions causing endolymphatic hydrops.

Autophony is the abnormal perception of one's own breath and voice sounds and is
often associated with a permanently open, or patulous, eustachian tube. The patient may also
describe it as sounds echoing in the ear, or as if talking in a reverberating chamber. It
indicates eustachian tube dysfunction and examination may occasionally show serous fluid
in the middle ear.

Fluctuant hearing loss may result from diseases causing either conductive or
sensorineural pathology. The fluctuant nature of the hearing loss associated with upper
respiratory tract infections, eustachian tube dysfunction and otitis media with effusion is well
known. Ménière's disease is characterized by a fluctuating sensorineural hearing loss, with the
hearing deteriorating during each attack and recovering between attacks (Hood, 1980). In a
variant of Ménière's disease - Lermoyez's syndrome - the hearing drops before an attack,
recovering as the vertigo begins.

Other features associated with the onset of the hearing loss should be noted. In the
majority of cases of sudden deafness the cause is unknown, though many are assumed to be
due to vascular disease. The deafness may be related to a recent viral infection, as seen in the
classical unilateral mumps deafness. Severe infections such as meningitis and, abroad, malaria
may pre-date the onset of hearing loss. Sudden deafness may be the presenting feature in up
to 10% of patients with acoustic neuroma (Morrison and Booth, 1970) or be associated with
a perilymph fistula resulting from an increased venous pressure due to straining or lifting.

Previous otological procedures should be noted, especially stapedectomy, which may
have been performed many years previously.

It is important to enquire about a family history of hearing loss since this may reveal
a hereditary cause. There may be a history of noise trauma having occurred 20 or 30 years
previously which has potentiated the effect of ageing on the cochlea to produce a hearing
disability. A clear history of occupational noise exposure and military service is required. In
the UK, protection from industrial noise trauma has been supported by legislation for only a
relatively short time so that many such cases are still presenting to outpatient clinics. Social
noise trauma includes pop music, rifle shooting and motor racing. Some people suffer
irreversible hearing loss from relatively minor noise stimuli, whereas others are exposed to
major noise trauma with little effect on their hearing.

The patient's past medical history is important since the aminoglycoside antibiotics,
used for life-threatening infections, and some of the 'loop' diuretics, are potentially ototoxic
(Ballantyne and Ajodhia, 1984). It is generally considered that topical aminoglycoside
antibiotics, as used in ear drops, do not cause hearing loss. More recently, the use of cytotoxic
therapy in oncology has been implicated as a cause of hearing loss. Salicylates bought 'over
the counter' may be ototoxic to the susceptible user, as is quinine which used to be taken as an antimalarial drug.

In children, a history of poor speech development, lack of communication skills and educational retardation may replace hearing loss as a symptom of ear disease.

**Tinnitus**

Tinnitus, like hearing loss, is a common presenting symptom of aural pathology. The nature of the tinnitus may be helpful in locating the lesion in the auditory pathway. A rhythmic beating or pounding tinnitus, synchronized with the pulse, is suggestive of a vascular lesion such as a glomus tumour. A dull, continuous tinnitus is sometimes found in association with a conductive hearing loss. This may represent normal noise levels in the temporal bone which have now become obvious to the patient because of the absence of the masking effect of environmental sound. Successful treatment of the conductive deafness, for example by stapedectomy, may alleviate this type of tinnitus.

Body sounds transmitted via an abnormally patent (patulous) eustachian tube may be reported as tinnitus, and likewise the noise of a live insect in the ear canal.

Most cases are characterized by rushing, hissing or ringing sounds in the ear or head. The source of these is either in the cochlea, neural pathways or cerebral cortex.

Previous noise exposure and a history of having been given ototoxic drugs (aspirin, quinine, etc) are important aetiological factors (Brown et al, 1981; Meyerhoff et al, 1983). Fluctuant tinnitus may be associated with Ménière's disease and usually increases in intensity prior to a vertiginous attack, returning to its resting intensity in between.

Long-term tinnitus sufferers may well be unable to locate the offending noise in the ears and simply perceive head noise. Recognizable sounds such as voice, music and bells may not be considered to be evidence of ear disease, but more psychological, as in schizophrenia. Tinnitus in general may be caused by all of those agents which produce hearing loss and thus a similar history should always be taken.

**Vertigo**

The definition of vertigo is difficult. It may be defined as an 'hallucination of movement' - that is, the patients feel that they or their environment are moving. Elsewhere in this volume (Chapter 18), it is defined as a 'subjective sense of imbalance'.

The history is of paramount importance in making the diagnosis in cases of balance disorder. In many cases, the diagnosis can be made from the history alone. It is essential to elicit from the patient the exact sensation perceived, since the terms 'dizziness', 'vertigo', or 'lightheadedness' mean different things to different people. The patient may have great difficulty describing the actual phenomenon (Hinchcliffe, 1973). This difficulty in description is a reflection of the small cortical representation of balance perception. The patient needs to rely on the mismatch of positional cues and the associated autonomic vegetative effects for their own description. It is important to identify symptoms not attributable to the vestibular
system, such as the lightheadedness, with blurring of consciousness, which accompanies cerebral anoxia. Anxiety states, in which the patient hyperventilates with resulting hypocarbia, may also produce such symptoms (Evans and Trimm, 1966). The sensation of movement associated with vestibular lesions is most commonly rotatory, but can include swaying or tilting of either the patient or the surroundings. If nystagmus is present, the environment is only perceived during the slow phase and since the images traverse the retina in the opposite direction, the environment appears to spin in the direction of the fast component. This may be useful on some occasions in trying to locate the offending labyrinth.

Peripheral lesions usually produce vertigo of sudden onset which may last for only seconds or up to a few days. In Ménière's disease, the attacks are recurrent and usually associated with fluctuating hearing loss and tinnitus. Movement tends to make vertigo of peripheral origin worse. The best known example of this is the sudden onset of rotatory dizziness associated with certain head movements in patients with benign paroxysmal positional vertigo. Vertigo associated with coughing or sneezing suggests the presence of a perilymph fistula. Tullio's phenomenon is the vertigo caused by loud sounds and may be due to endolymphatic hydrops or a third labyrinthine window, as in a labyrinthine fistula (Kakkar and Hinchcliffe, 1970).

Central lesions tend to produce less intense vertigo. Positional changes have less effect, but the patient tends to have more disturbance of gait.

Vertebrobasilar ischaemia can cause sudden onset vertigo and drop attacks, without loss of consciousness, but usually accompanied by other associated symptoms. A full medical history may reveal long-standing degenerative conditions such as diabetes mellitus or atherosclerosis. Life-threatening infections sometimes require potentially ototoxic antibiotics. Some of these, particularly gentamicin, may damage the vestibular system.

In summary, it is important to ask the patient if he/she remembers the first attack and to describe it accurately. The onset, whether sudden or gradual, precipitating factors, duration of attack and associated symptoms are noted. The frequency and severity of attacks should be enquired about.

**Oscillopsia**

This descriptive term is used when the patient complains that the horizon rotates or jumps in a vertical plane when walking (Ramsden and Ackrill, 1982). Resulting spatial disorientation is corrected by the patient halting, holding on to a solid structure and focusing on a near image. It is due to an imbalance in the vestibulo-ocular reflex, which is necessary to stabilize the retinal image. Oscillopsia may follow loss of peripheral vestibular function, but is also a feature of central lesions, especially when associated with an acquired pendular nystagmus (Rudge, 1984).
Clinical examination

The ear

Congenital absence of the auricle is termed 'anotia' and incomplete development 'microtia'. Anotia is associated with severe malformations of the ear canal, middle and inner ear; with microtia the auricular remnant is usually anteroinferior to the bony ear canal and the presence of a tragus is considered a good prognostic feature for middle ear reconstruction. Accessory auricles may be found and represent separate developments of the second branchial arch remnants. In all cases of congenital external ear dysplasia, a full examination should be made for other features which might allow the identification of a named syndrome.

Acquired lesions on the auricle include gouty tophi, squamous carcinoma, basal cell carcinoma and the painful nodules of chondrodermatitis helicis.

It is important to look behind the auricle for surgical scars. Postauricular incisions may be difficult to see deep in the retroauricular sulcus, and the more posteriorly placed incisions associated with the formation of a Palva flap or excision of an acoustic neuroma may be hidden in the hairline. Endaural incisions can usually be noted in the area between the tragus and helix. Examination behind the auricle may reveal evidence of acute inflammation in the form of erythema, tenderness or abscess formation. In children, a subperiosteal abscess tends to point posteroinferiorly to the external auditory meatus. In adults, the abscess points more posteriorly, reflecting the more extensive mastoid development. Pus from the mastoid may track anteroinferiorly along the sternomastoid muscle presenting as a mass in the neck (Bezold's abscess). Alternatively, it may track medially along the posterior belly of the digastric muscle emerging in the submandibular triangle as Citelli's abscess (Shambaugh, 1967b).

Lymphadenopathy, associated with ear infections, occurs commonly in the preauricular node where it has to be differentiated from an infected preauricular cyst. Swollen, tender postaural nodes also occur, but other sites of primary infection (scalp, etc) should always be considered. Neoplastic infiltration of neck nodes in both anterior and posterior triangles occurs with aural carcinoma and is a grave prognostic feature.

The external auditory meatus is examined using either an auriscope or hand-held speculum and headlight. In the adult, traction on the pinna upwards and backwards helps to straighten the canal and facilitate vision. If the view is obscured by wax this should be removed either by a wax probe or by syringing. Syringing is best avoided if there is a possibility that the tympanic membrane is perforated. It must not be forgotten that syringing can change the appearance of the tympanic membrane. It is often difficult to see the anterior sulcus of the canal because of a prominent anterior meatal wall. Canal stenosis may follow chronic otitis externa or surgery. Meatal osteomata, which are sometimes associated with cold water swimming, appear circumferentially in the bony part of the meatus and may obstruct a clear view of the tympanic membrane.

Tympanic membrane

It is essential to identify the normal anatomical features of the tympanic membrane using either an auriscope or speculum and headlight. The wide-angled lens of a Storz rod may
be helpful and is useful for photography of the tympanic membrane. Some tympanic membranes are difficult to see and in these cases an operating microscope is useful.

If possible, the whole of the tympanic annulus should be seen, along with the handle and lateral process of malleus. In most normal tympanic membranes there is the sharp reflection of the auriscope's light spreading anteroinferiorly in a cone shape. The mobility of the tympanic membrane is assessed by using a pneumatic bulb on the auriscope or Siegle's speculum (Siegle, 1864).

Alternating positive and negative pressures in the ear canal result in the normal tympanic membrane moving inwards and outwards. Where there is a perforation of the tympanic membrane, or fluid in the middle ear, there is loss of normal mobility.

The position of pathological features should be noted in relation to the normal anatomy, that is the pars tens and pars flaccida. A perforation may be central, marginal or attic (in the pars flaccida). It is sometimes difficult to differentiate a retraction pocket from a perforation. In such cases, pneumatic otoscopy with bacteriostatic powder blown on to the tympanic membrane helps to make the diagnosis. Assessment of the retraction pocket's adherence to middle ear structures (usually the incudostapedial joint) is important. If a perforation is present, the state of the middle ear mucosa should be assessed for inflammation, infection and oedema. When the stapes is visible through a perforation, its mobility can be assessed using the operating microscope by stimulating the other ear, if the hearing is normal, with a Barany noise box and looking for a crossed stapedius reflex. This technique may be helpful in the preoperative assessment of cases involving tympanosclerosis.

**Eustachian tube patency**

The following manoeuvres indicate patency of the tube, although not necessarily normal function, as they are non-physiological.

**The Valsalva manoeuvre**

The production of a high nasopharyngeal pressure by blowing out against closed lips and nose normally results in an increase in middle ear pressure with the tympanic membrane bulging outwards. It is important to have the auriscope in place before the patient starts blowing as otherwise trauma to the external meatus may occur with head movement.

**Toynbee's manoeuvre**

This occurs when a swallow is made with the lips and nose closed. A negative pressure in the nasopharynx and middle ear results in an indrawing of the tympanic membrane. This should return to its normal position when swallowing again with an open nose.
Frenzel manoeuvre (nasopharyngeal pressure test)

This manoeuvre was described in 1938 by Hermann Frenzel, a prominent figure in German aviation medicine in World War II, and has been found to be more effective than the Valsalva and Toynbee tests (Frenzel, 1950).

'With the nostrils and glottis closed, the air in the nasopharynx is compressed by the muscles of the floor of the mouth and tongue. The opening of the eustachian tube by this method is facilitated by the convexity of the tongue which places the soft palate and parts of the tube orifice to which it is connected into a more favourable position for opening the tube'.

Its advantage is that it can be performed in any phase of respiration and is independent of intrathoracic pressure. The disadvantage is that the procedure has to be learned, but once acquired it soon becomes no longer necessary to hold the nostrils as these close automatically. This has an obvious advantage for those wearing a flying helmet, oxygen mask or both.

The subject has to acquire a feel for voluntary closing of the glottis and Frenzel suggested 'the repeated production of a silent "ah" while expiring after a moderate inspiration'. Davison (1962) suggests:

'... having the subject close the glottis after a moderate inspiration and then attempt to make an oral "ka" sound. If the subject partially compresses his nostrils while performing this manoeuvre, he can feel and hear the rush of air out of the anterior nares, thus demonstrating that the manoeuvre does diminish the volume of the nasopharyngeal space'.

Patulous eustachian tube

This is a not uncommon ear condition which frequently goes undiagnosed and is managed incorrectly (Bull, 1976). The patient complains of a sensation of blockage in the ear, but denies any hearing loss. The sensation of blockage disappears on lying down and may alter with certain positions of the head. Patients may also say that they hear the noise of themselves talking, eating or breathing.

The tympanic membrane is normal, but may in some cases be seen to move with respiration. If the patient is asked to breathe in and out through the nose with the mouth open, air flow through a patulous tube is accentuated and the tympanic membrane movement is more easily visible (O'Connor and Shea, 1981).

The condition is often missed and treated as a eustachian tube obstruction with topical and systemic decongestants. These may make matters worse. The condition is common in people who have lost weight suddenly, usually from strict dieting, those on the contraceptive pill and in pregnancy. It may also be found in older patients given diuretics.

Treatment is usually unnecessary if the condition is explained to the patient and reassurance given that there is nothing seriously wrong. Some people obtain relief after
insertion of a ventilation tube and, in rare instances, injection of Teflon paste around the eustachian cushion may help.

**Fistula sign**

If, following a pressure increase in the external auditory meatus, vertigo and nystagmus result, a positive fistula sign is said to be present. Such a pressure change can be achieved by simply compressing the tragus into the external auditory meatus. A similar effect can be obtained by using a pneumatic otoscope, Siegle's speculum or the air pump of a tympanometer. In cases of chronic suppurative otitis media, a positive fistula sign indicates the presence of a third window into the perilymphatic space enabling gross movement of the inner ear fluids and stimulation of the vestibular end organs (Schuknecht, 1974). In ears with such disease, palpation of a fistula while probing the ear results in a violent vertiginous response.

**Hennebert's sign**

Hennebert's sign occurs when there is a positive fistula test with an intact tympanic membrane and no evidence of middle ear disease. The pathophysiology of this sign is unclear, but is thought to be due either to adhesions in the vestibule or to the presence of a third window somewhere in the labyrinth caused by osteitis (Schuknecht, 1974). It is seen most commonly in congenital or late tertiary syphilis, but is sometimes found in other conditions causing endolymphatic hydrops such as Ménière's disease. Hennebert's sign is seen most clearly using the slow, sustained negative pressure change of the tympanometer.

The normal caloric response due to air currents that occurs during suction to the meatus or mastoid cavity should be recognized and not interpreted as a fistula sign.

**Auscultation of the ear and temporal bone**

This part of the examination is useful in some cases. The stethoscope is used, placing the bells over the ear canal and then lightly on the mastoid process; bruits from vascular anomalies or glomus tumours may be heard (Moffat and O'Connor, 1980). Recently, perception of cochlear emissions, which may or may not be associated with subjective tinnitus, have been reported (Harrison, 1986). In cases of patulous eustachian tube, a stethoscope end inserted into the meatus will pick up the transmitted voice sounds from the nasopharynx.

**Examination of the eyes**

Inspection of the eyes may reveal features, such hypertelorism or coloboma, associated with congenital hearing disorder syndromes. The presence of blue sclera (osteogenesis imperfecta) and interstitial keratitis (congenital syphilis, Cogan's disease) should be noted.

**Fundal examination**

Examination of the fundus of the eye must be performed when there is a possibility of an intracranial lesion. Papilloedema may be seen with a space-occupying lesion, such as
a cerebellopontine angle tumour or temporal lobe abscess, and also in otitis hydrocephalus
where it is often chronic (O'Connor and Moffat, 1978). Optic nerve atrophy follows
demyelinating conditions which may present with auditory or vestibular disturbances.

When looking at the fundus it is important to use optimum conditions, including a
darkened room and mydriatic drops in order to see the fundus clearly. Remember,
ophthalmologists regularly use these conditions; it would be wise for the infrequent
'ophthalmologist' to do the same.

**Eye movements**

Nystagmus is involuntary eye movement. Patients with nystagmus may describe an
inability to focus on a still object or, when associated with rotational vertigo, movement of
the visual field in the same direction as the nystagmus. Nystagmus is most easily seen in good
light with the patient looking to the front (spectacles on, if usually worn!) and the observer
viewing slightly from the side. Visual fixation is obtained by placing a finger central to the
eyes and at least 45 cm from the nose. The presence or absence of nystagmus is noted and
the finger moved laterally in the same horizontal plane 30° to either side, asking the patient
to follow the finger.

Congenital nystagmus is characteristically pendular in type, when viewed in the
'neutral' central position and usually associated with visual defects.

Vestibular nystagmus may be horizontal or rotatory and has two components, a slow
phase with a fast corrective phase in the opposite direction. The slow phase reflects an
imbalance of input to the vestibular nuclei and the fast phase is a central righting response.
The direction of the nystagmus is conventionally defined in terms of the direction of the fast
phase. The intensity of the nystagmus is described in terms of the direction of gaze. Thus, a
first degree nystagmus is visible only when the eyes are deviated to the side which is also the
direction of the fast phase. A second degree nystagmus is visible in the above position and
also with the eyes in the 'neutral', straight ahead position. A third degree nystagmus means
that the nystagmus is present in all directions of gaze. If nystagmus changes in direction with
the gaze it is termed 'direction changing' and may be indicative of a central lesion. Visual
fixation should then be reduced using Frenzel's glasses (spectacles having a focal length of
infinity, and illumination so that the eye movements are clearly visible to the observer). It is
also possible to abolish fixation by using a dark room where eye movements may be seen
with an infra-red viewer. In general, removal of visual fixation enhances the nystagmus due
to peripheral lesions but reduces that due to central lesions. These clinical techniques correlate
well with the findings when electronystagmography is used.

If spontaneous nystagmus as described above is absent, nystagmus may be induced by
positional changes, rotational or caloric stimulation. Nystagmus induced by changes in
position may be associated with benign positional vertigo and is characterized by a brief delay
in onset following the change of position (latent period), rotational or horizontal nystagmus
directed towards the undermost ear, which lasts no longer than 20-30 seconds and is
fatiguable. It is thought to be due to a 'benign' pathological process affecting the peripheral
vestibular system (Schuknecht, 1969). A positionally induced nystagmus, that has no latent
period, remains present while the patient is in the provocative position and is direction
changing according to head position, is suggestive of a central lesion. If a benign positional nystagmus has atypical features, the presence of a central lesion must be considered.

Caloric stimulation may be achieved in the clinic with cold water. Ten millilitres of ice-cold water are introduced into the deep external auditory meatus by means of a soft Portex tube (intravenous infusion catheter). Nystagmus is viewed using Frenzel's glasses and, if present, indicates significant labyrinthine function, the direction of the nystagmus being away from the stimulated ear. This technique cannot differentiate a canal paresis from a directional preponderance.

**Examination of cranial nerves III-VI**

Voluntary eye movement is examined by exercising all six oculomotor muscles in turn, observing any paresis and enquiring about diplopia associated with specific movements. Failure of lateral gaze with diplopia indicates an abducens nerve palsy. This nerve has a long intracranial course and it is therefore often difficult to localize lesions affecting it. Paralysis of the abducens nerve may be associated with lesions of the petrous apex such as congenital cholesteatoma or Gradenigo's syndrome (aural discharge, pain and sixth nerve palsy).

**Pupillary reflexes**

The reflexes mediated through the Edinger Westphal nucleus of the parasympathetic system are examined by asking the patient to look to infinity and then focus an object at their normal focal length. This process - accommodation, should cause pupillary constriction. This is followed by stimulating the eye with a bright light and once again looking for pupillary constriction. In congenital or tertiary syphilis, the pupil constricts in response to accommodation but not to light - the so-called Argyll Robertson pupil.

**Visual fields examination**

The visual fields of each eye are examined clinically by asking the patient to observe the movement of the clinician's fingers placed at the focal distance, with the eyes in the neutral position. The fingers are then moved to map out the visual fields. Central visual field loss may be associated with papilloedema (perhaps due to cerebellopontine angle lesions or otitic hydrocephalus). With an otogenic temporal lobe abscess, an homonymous quadratic hemianopia may be found due to disruption of the optic radiation.

**Corneal reflexes**

The cornea is touched from the side with a fine wisp of cotton wool (note - it is sufficient to touch the sclera). The normal response is a blink. The response of each cornea should be noted and compared. The loss of corneal reflex is said to be the most sensitive indication of a lesion involving the trigeminal nerve, but is usually a late sign in acoustic neuroma, indicating a large tumour which has expanded sufficiently so as to compress the nerve.
**Facial nerve (VII)**

**Motor function**

It is important to differentiate between an upper and lower motor neuron lesion. An upper motor neuron lesion paralyses only the lower part of the face, the forehead being spared as it has bilateral cortical representation. A lower motor neuron palsy involves all of that side of the face. The patient is asked to frown with the observer's thumb placed firmly in the midline to prevent muscle movement from the other side simulating movement of the affected side. The patient is then asked to close and open the eyes, bearing in mind that the levator palpebrae muscle is partially innervated by the oculomotor nerve. The midface is examined by nose twitching and the lower face by smiling or showing the teeth. Various conversations have been suggested to quantify partial facial palsy, but all are open to observer differences. However, it should always be clearly stated whether the palsy is partial or complete.

**Somatic sensory function**

Touch sensation of the floor of the external auditory meatus has been noted to be absent in some cases of acoustic neuroma (Hitselberger's sign).

**Parasympathetic secretomotor function**

Fibres passing in the nervus intermedius and then in the greater superficial petrosal nerve to the lacrimal gland may be tested by the tearing on a strip of filter paper placed over the lower lid (Schirmer's test). Only a gross difference between abnormal and normal sides is significant.

**Special sensation**

Taste from the anterior two-thirds of the tongue is examined, either by the use of test substances (salt, sugar and citric acid) or by electrogustometry. In this technique, a quantitative assessment may be obtained in terms of the electric current needed to elicit a metallic taste in the mouth; both methods are prone to false positives due to the hyposensitivity of many patients' taste buds.

**Cranial nerves IX-XII**

Glossopharyngeal nerve (IX) function is tested by touching the wall of the oropharynx or posterior one-third of the tongue. This is the afferent arm of the gag reflex whose efferent arm is mediated through the vagus, producing elevation of the palate and generalized movement of the oro- and hypopharynx. The palate should be examined in cases of clicking tinnitus in order to exclude palatal myoclonus.

Indirect laryngoscopy will permit an assessment of vocal cord movement (vagus nerve - X). To test accessory nerve (XI) function, the patient is asked to rotate the head against the observer's hand and the tension in the contralateral sternocleidomastoid muscle is felt. Shoulder shrugging is tested on each side.
Following hypoglossal nerve (XII) palsy, protrusion of the tongue from the mouth may lead to deviation towards the side of the lesion.

**Examination of the nose and throat**

A full examination of the nose and throat must always be carried out. Inspection of the nose may reveal rhinitis or sinusitis which is responsible for eustachian tube dysfunction. The postnasal space is examined using a mirror placed in the oropharynx. This necessitates the use of a tongue depressor and head mirror or headlamp. The development of the fiberoptic rhinopharyngoscope permits the nasopharynx to be examined from the posterior choanae. Where there is evidence of a middle ear effusion, without an obvious explanation, the postnasal space must be examined, even if a general anaesthetic is needed.

The rest of the upper air and food passages should be examined for the cause of a referred otalgia, for example carcinoma of the pyriform fossa.

**Clinical tests of hearing**

During the history taking and examination, the clinician should be making an assessment of the hearing threshold. The clinician should alter the voice level and avoid giving visual clues, and in this way gain an impression of how well the patient hears. This is of special importance in patients who are thought to have a non-organic hearing loss. An estimation of the hearing thresholds in each ear may be obtained with masking of the contralateral side by gently rubbing the orifice of the external auditory meatus with a finger.

**Tuning fork tests**

These tests are a most important part of any clinical examination of hearing and should be performed carefully. They are discussed in considerable detail in Volume 2. The tuning fork used most commonly has a frequency of 512 Hz. The note of the higher frequency forks tends to decay quickly, not allowing sufficient time for the Rinne test to be performed. The lower frequency forks tend to enhance perception by vibration sensation.

**The Rinne test**

Essentially this test consists of comparing the auditory acuity of each ear to bone and air conduction. The tuning fork is struck gently so as not to produce overtones and dysharmonics (usually by striking it on a bony prominence, belonging to the examiner not the patient!). The fork is placed firmly on the mastoid with the observer's hand steadying the head. Care is taken, especially in children, to have the fork placed firmly on bone and not on the sternomastoid muscle. The patient is asked to indicate when the sound disappears and the fork is then immediately placed erect and in line with the external auditory meatus about 2 cm from the orifice. If the patient still hears the note when the fork is placed in front of the ear the patient is termed 'Rinne positive' (air conduction being better than bone conduction). If he/she does not still hear it the patient is 'Rinne negative' (bone conduction better than air conduction). Alternatively, and more usually, in routine clinical practice, the patient is asked to compare the sound intensity of the fork in the mastoid position (bone conduction) with that in the meatal position (air conduction). If there is a significant sensorineural deafness, the fork
will not be heard by bone conduction at all, but only by air conduction, and obviously in severe cases not by air conduction either. A conductive deafness of greater than 25 dB usually gives a negative Rinne test with a 512-Hz fork. However, with a 256- or 128-Hz fork, this may be reduced to 10-15 dB and, with the higher frequency forks (1028, 2048 and 4096 Hz), the conductive deafness needs to be greater than 25, 30 and 35 dB, respectively (Shambaugh, 1967a).

**False negative Rinne**

This is an important concept and its possibility should never be missed by the otologist. If the patient has no hearing in the test ear, the bone conduction stimulus may be perceived by the contralateral (non-test) ear, although the patient often says that he/she hears it in the test ear. As there is no hearing by air conduction, the test result is labelled Rinne negative suggesting that the deafness is conductive in nature. This mistaken impression of function in a non-functioning ear is called a false negative Rinne. In such cases the diagnosis is given by a combination of the Rinne and the Weber test. In addition, the non-test ear can be masked by a Barany noise box (a clockwork-driven sound generator of about 90 dB). This phenomenon occurs because the interaural attenuation for bone conduction is less than 5 dB, that is sound passes freely across the skull stimulating both ears equally, regardless of where the tuning fork is placed.

**Weber test**

The tuning fork is struck and the base placed on either the forehead, vertex or upper incisor teeth. The patient is asked where the sound is heard loudest. In a normal hearing person, the sound is related to the midline. In a patient with unilateral sensorineural deafness, it is referred to the good ear and in a patient with a conductive deafness to the affected ear. In cases of asymmetrical mixed (conductive plus sensorineural deafness, no definite rules can be made, but the result interpreted in conjunction with the Rinne test. Obviously the Weber test is a great help in recognizing a false negative Rinne as it will be referred to the good ear. In long-standing cases of sensorineural deafness, the Weber test tends not to lateralize. A lateralized Weber in a conductive deafness may indicate a hearing loss of only 10-15 dB.

It should be understood that the abnormal test results in conductive deafness are not explained by the lack of environmental masking, as they also occur in anechoic (soundless) chambers. Several theories have been put forward and it seems likely that the explanation differs in different types of conductive loss (Tonndorf, 1966).

**Modified Schwabach test (absolute bone conduction test)**

This compares the bone conduction of the patient with the bone conduction of a normal hearing person. The tuning fork is placed on the patient's mastoid with the meatus blocked and, when the patient no longer hears it, the fork is placed on the normal hearing person's mastoid (usually the examiner's), again with the meatus blocked. If the examiner hears the note, the patient's bone conduction is said to be reduced. The Schwabach test is carried out in the same way but without occluding the meatus.
Several other tests are available which use the principle that, in a normal ear when the sound conducting mechanism of the external and/or middle ear is reduced, the bone conduction stimulus will be enhanced. If there is already a conductive deafness, there will be no change in the perception of the bone conduction stimulus.

**Gellé test**

The air pressure in the external auditory meatus is altered using a Siegle's speculum. In the normal individual, or those with a sensorineural loss, increasing the meatal pressure results in a decreased sensation of loudness from a bone-conducted stimulus. No alteration of bone-conduction thresholds indicates fixation of the stapes.

**Bing test**

Increased loudness for bone-conducted stimuli, less than 2 kHz, occurs in the normal patient or those with a sensorineural loss when the external meatus is occluded without altering meatal pressure. There is no change when a conductive deafness is present.

*Tuning fork tests in non-organic deafness*

**Stenger test**

Principle: if sounds of identical frequency but different intensity are presented simultaneously to each ear, only the louder sound will be perceived. The test can be performed either with a pure-tone audiometer or tuning forks.

The examiner stands behind the patient. A tuning fork is struck and held 20 cm from the 'good ear' - the patient hears the sound. The fork is then removed and placed 5 cm from the 'bad ear' - the patient denies hearing the sound. Another fork is then held 15 cm from the good ear without the patient noticing. If there is a genuine hearing loss the patient will hear the fork in the good ear, but if there is a non-organic hearing loss the patient will not be able to hear the fork in the good ear because the fork which is closer, and therefore of louder intensity, is being heard in the bad ear.

**Chimani-Moos test**

This is a modification of the Weber test. When the tuning fork is placed on the vertex, the patient indicates that he hears it in the good ear and not in the deaf ear. The meatus of the good ear is then occluded. A genuinely deaf patient will still lateralize the sound to the good ear, the malingering will usually deny hearing the sound at all.

Both of these tests should be used in conjunction with the clinical history (Is there a question of litigation? Was trauma involved?), and the clinical assessment of hearing during the examination.
Clinical tests of balance

Normal body position is a function of the neural input into the cerebellum and brainstem from the receptors in the semicircular canals, the macula of the utricle, the proprioceptive and joint position sensors and the eyes. Thus, in the clinical examination, each component of the system should be tested individually. If hypofunction of one input occurs, then compensation by the others usually takes place. However, when such compensation is removed, for example by closing the eyes, the resultant deficiency usually becomes obvious.

Romberg's test

The patient is asked to stand erect looking forwards with the feet together. If the patient is stable, he/she is asked to close the eyes. With a labyrinthine lesion the patient will sway often to the side of the lesion, a feature which is accentuated by closing the eyes. A central lesion in the cerebellum results in symmetrical swaying that is less affected by eye closure. If the patient falls backwards in a rigid pose, but is able to regain balance before falling to the ground, there is a non-organic disturbance such as malingering or hysteria.

Unterberger's test

This test aims to reduce the input from the proprioceptive organs. The patient is asked to stand as for the Romberg test, but with the hands outstretched, and march on the spot with the eyes closed. The patient will rotate towards the side of a paralytic labyrinthine lesion. In the presence of an active irritative lesion, the balance disturbance is so significant that the patient cannot perform the test for more than a few seconds.

The gait test

The patient is asked to walk in a straight line between two points and then quickly turn to return on the same line. Patients with labyrinthine lesions deviate to the side of the lesion whereas marked imbalance on turning indicates a cerebellar lesion. The sensitivity of the test may be increased by asking the patient to walk on a bed of foam.

Caloric test

The minimal cold water caloric test has been described previously. The classical Fitzgerald-Hallpike bithermal caloric test is the generally accepted method of evaluating vestibular function by caloric stimulation (Fitzgerald and Hallpike, 1942).

The patient is placed supine on a couch with the head elevated to an angle of 30° to the horizontal. This brings the lateral semicircular canal into the vertical plane. Both ears are checked for wax or the presence of a perforation, as the latter would preclude caloric testing by this technique. Each ear is irrigated by water at 44°C and 30°C (7°C above and below normal body temperature) for 40 seconds. Warm water is used first and the tympanic membrane checked for an hyperaemic blush which indicates adequate irrigation. The volume of water used is checked and should be about 300 mL. The eyes are observed for nystagmus with the patient focusing on a near object. The end point of the nystagmus is noted and its
duration recorded. Frenzel's glasses are then used to reduce visual fixation and, if the nystagmus reappears, the new end point is noted. A normal caloric reaction results in nystagmus being visible between 90 and 140 seconds after the onset of irrigation, and prolongation by a further 60 seconds following the reduction of visual fixation. The affected ear is stimulated with warm water, then with cold, and the test concluded by cold water irrigation of the affected ear. Between each irrigation a rest period of 7 minutes is allowed.

Cold water produces a nystagmus away from the stimulated ear (away cold = AC) and warm water towards the stimulated ear (towards hot = TH), thus the mnemonic ACTH.

Following bithermal caloric stimulation of a paretic labyrinth, nystagmus may be absent or decreased in amplitude and duration. Care should be taken to look for the end point prior to the reduction of visual fixation, which may prolong the nystagmus into the normal range. When the nystagmus in one direction is significantly greater after bithermal testing, it is termed 'directional preponderance'. The significance of this is not fully understood.

**Other tests for cerebellar dysfunction**

**Dysmetria and past pointing**

The patient is asked to touch his nose and the examiner's finger alternately. The examiner's finger should be placed in front of the patient at a distance which necessitates very full extension of the patient's arms. The target finger is moved around. Failure by the patient to touch the examiner's finger or his own nose suggests the presence of a cerebellar lesion. If the test is performed satisfactorily the patient is asked to close the eyes and continue pointing. Straying from the targets now suggests a peripheral vascular lesion (Marshall and Attia, 1983).

**Asynergia**

The patient is asked to tap the back of each hand in turn with the other hand. With the cerebellar lesions the accuracy of the tap and the discrete area of contact are lost.

**Dysdiadochokinesis**

In cerebellar lesions, asymmetry occurs when the patient is asked to pronate and supinate the hand on the side of the lesion.

**Rebound**

The patient's hands and arms are held out rigidly in front and the examiner pushes from above on one hand and from below on the other. The hands are then released. With a cerebellar lesion the patient's arms are unable to compensate for the change in resistance and move wildly.