Chapter 6: The clinical examination of aural function

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Anamnesis (case history)

As Rosenberg (1972) pointed out in Katz's *Handbook of Clinical Audiology*, the first, and possibly the most valuable, test of all is the case history. In the practice of aural medicine, questionnaires can be of considerable value, as in medicine in general (Bennett and Ritchie, 1975). In aural medicine, questionnaires may be particularly valuable for the assessment of the disability and handicap of auditory (Noble, 1978; Barcham and Stephens, 1980) and vestibular (see Chapter 15) dysfunction. The value of questionnaires in forensic aural medicine, however, is more limited. In such circumstances, recourse is best made to the non-leading question (and answer) approach which is used by lawyers themselves (Hinchcliffe, 1987).

Wilbush (1984) has emphasized the need to distinguish between symptoms and semeions. Symptoms are those sensory experiences which are spontaneously reported to the doctor; semeions are those which are elicited only by direct questioning.

Aural symptoms can be broadly classified into auditory, vestibular and those of aural discomfort. The aural discomfort symptoms can be classified into pain, pressure or fullness, and itching. Aural pain characterizes inflammatory disease of the ear. In the absence of any visible evidence for inflammatory or other disease of the ears, psychological factors must be considered. This consideration, of course, is given after one has ensured that one is not dealing with referred pain from, for example, a pharyngeal lesion.

A pressure sensation in the ear is seen not only where there is cerumen causing obstruction but also with otitis media with effusion. A pressure/fullness sensation is also characteristic of endolymphatic hydrops.

Itching is characteristic of chronic otitis externa.

*Auditory symptoms*

Auditory symptoms can be broadly classified under the paracuses (Table 6.1) and tinnitus. These various symptoms are considered later in this chapter under Tuning-fork tests.

*Equilibratory and vestibular symptoms*

Although dizziness, oscillopsia, ataxia, falls and frank immobility associated with balance disorders embrace many aetiopathological entities, different conditions may be associated with each of these particular symptoms. Consequently the medical history taking will be moulded primarily to the particular manifestations of the balance disorder.
Table 6.1. Classification of auditory symptoms (paracusus)

1. Intensity related dysfunctions
   1.1. Paracusia acris (hyperacusis)
      1.1.1. Oxyacoeia
      1.1.2. Phonophobia
   1.2. Paracusia obtusa
      1.2.1. Hypoacusis
      1.2.2. Anacusis
   1.3. Paracusia perversa (Paracusia willisii)

2. Frequency related dysfunctions
   2.1. Paracusia sclerotica
   2.2. Paracusia duplicata (diploacusis)
      2.2.1. Diploacusis monauralis
      2.2.2. Diploacusis binauralis
         2.2.2.1. Diploacusis binauralis harmonica
         2.2.2.2. Diploacusis binauralis dysharmonica
   2.3. Paracusia isoacustica

3. Localization dysfunctions
   3.1. Paracusia localis
      3.1.1. Dysstereoacusis
      3.1.2. Astereoacusis.

Dizziness

A complaint of dizziness may be associated with dysfunction of a body system other than the vestibular system. Moreover, multiple sensory deficits will often be found to be the basis for complaints of dizziness, particularly in the elderly (Drachman and Hart, 1972). Such patients, who are often diabetic, complain of light-headedness when walking, particularly when turning.

Bezold (1906) pointed out that dizziness due to a vestibular disorder can be differentiated from that due to a non-vestibular disorder by the effect of eye closure. Closing the eyes abolishes dizziness due to the latter disorder.

If the patient's complaint of dizziness or giddiness is found to be equatable with a hallucination of movement, then he (or she, which is more likely) is considered to have vertigo. The occurrence of vertigo, especially when a rotatory sensation is experienced, is more likely to indicate a disordered vestibular system. Schuknecht and Kitamura (1981) have examined the temporal bones from elderly patients who had sustained one or more episodes of acute vestibular failure (marked vertigo associated with nausea and, sometimes, vomiting) unassociated with other vestibular deficits. The results indicated that the pathological basis was a discrete degenerative neuropathy of the vestibular nerve. Moreover, these authors consider that the aetiology in their cases was viral. Nevertheless, an acute cerebellar infarct may mimic acute vestibular labyrinthe disease (Guiang and Ellington, 1977). However, a
central lesion producing vertigo is diagnosed by evaluating the paraphenomena associated with the vertigo rather than by the characteristics of the vertigo itself (Levy and O'Leary, 1947).

The direction of the vertigo may or may not have lateralizing value in respect of the site of the lesion. It would appear that only in the case of cerebellar tumours is the relationship constant (Stewart and Holmes, 1904). With unilateral tumours, the sense of movement of the individual or of external objects is towards the unaffected side.

Oscillopsia

'Oscillopsia' is the term given to the symptom of an apparent visual oscillation of a fixed stationary target. Various types of oscillopsia exist. Oscillopsia which is experienced only on walking or riding is generally due to bilateral dysfunction or, more likely, a function of the vestibular end organ, nerve or nucleus. This symptom reflects the primary function of the vestibular labyrinths, that is to permit man to walk and see. Bender (1965) cited the case of a man who had had a 5-week course of streptomycin therapy for a bacterial endocarditis. The main complained of an unsteady gait and blurred vision. He noticed that, as he walked towards his sister, her features became blurred and indistinct. When he stopped walking, she again became clear. There was no oscillopsia when the eyes moved from side to side or up and down, provided the head was held still and there was no body motion. Examination failed to disclose any nystagmus either on naked eye observation or on fundoscopy. The caloric test produced no response from either side.

Bender also reported that he had observed periodic alternating oscillopsia as an adverse reaction to phenytoin. The patient experienced oscillopsia which alternated between horizontal and vertical movements; the horizontal phases lasted for between 100 and 200 seconds and the vertical for between 15 and 30 seconds.

A constant oscillopsia which persists even when the patient's head and body are at rest is more likely to be due to a brain lesion. If the oscillopsia is localized to homonymous visual fields, then it indicates an occipital lobe lesion.

Both monocular and asymmetrical oscillopsia are associated with disease of the basal ganglia, cerebellum or midbrain.

Just as the patient uses the umbrella term 'dizziness' to cover a variety of dysfunctions other than those referable to the vestibular system, so difficulty in walking will be used to cover a variety of dysfunctions other than ataxias. The complaint of difficulty in walking, or with one leg, may, after examination, be shown to be due not to a coordination disorder but to a motor or sensory neurological disorder, or even a non-neurological disorder.

Examination of aural and related structures

There is considerable variation worldwide in methods for the physical examination of the ear and its adnexa. This applies both to instruments in use and the way they are used. Most of the differences appear to reflect personal prejudices or the place where a doctor was trained. There appears to be little or no scientific basis for dogmatic assertions regarding the 'right' instrument, the 'correct' method of holding it, or the 'best' method of examination.
However, considered worldwide, the most common method for the examination of the ear, nose and throat is still the method which was introduced by von Tröltsch in the nineteenth century. This involves the examination of the cavities of the ear, nose and throat using light reflected from a concave mirror perforated in the centre. Many are now able to supplement this method by the use of instruments where the light source and the optical (mirrors, lenses) system are incorporated in the same instrument, for example the otoscope. Concurrently, the doctor has a choice of instruments giving a range of magnifications, from the simple pneumatic ear speculum through the Blackmore-Hallpike hand-held ear microscope which has a magnification of six, to the larger wall-mounted, or free standing, ear microscope (Lundborg and Lindzander, 1969).

There are also a variety of specula (instruments designed to be inserted into the external orifice of a cavity to facilitate the visual examination of the interior) in use. It is to be noted that doctors who have been trained in British schools tend to use Thudicum nasal specula, and those trained in other schools, Politzer nasal specula.

The examination of body cavities has been considerably facilitated in recent years by developments in fibreoptics.

Lancer and Jones (1986) have drawn our attention to a flexible rhinolaryngoscope which has an external tip diameter of 3.4 mm, a 1.5 radian field of view, a tip movement range of 4 radians and a working length of 255 mm. The instrument can be used to assess both the structure and function of the upper respiratory tract. With this instrument, all areas from the nasal vestibule to the carina may be viewed with precision. Thus the locations which become visually accessible include the ostia of the paranasal sinuses, the roof of the nose and the pharyngeal recess (formerly termed the 'fossa of Rosenmüller'). The pharyngeal opening of the auditory (previously termed 'pharyngotympanic', and prior to that, the 'eustachian') tube may be observed during opening and closure. Palatal function may thus be assessed, the tongue may be observed at rest and during protrusion, and the larynx may be seen at rest, during quiet and deep respiration, and during phonation. Although one can usually examine the larynx and pharynx, including the nasal part of pharynx, by indirect techniques, there are those patients who are overanxious, who may have an easily excitable gag reflex and who may have a narrow pharyngeal isthmus. In such patients, a full and satisfactory assessment may not be possible with conventional methods. It is in the examination of these patients that the flexible fibreoptic rhinolaryngoscope would be of most value. Sedation is, however, required for children under the age of 8 years. Another advantage of this fibreoptic system of investigation is that patients may view their own larynx with a teaching attachment. Moreover, this feedback technique is helpful in the management of certain disorders of speech; students may look through this attachment, and a photographic record may be obtained (Lancer, 1986).

**Clinical tests of auditory function**

The clinical examination of auditory function in the adult and in the schoolchild is based upon tests developed in the nineteenth century. This has permitted time for the accumulation of a considerable data-base and, with the introduction of the modern type of audiometer in the 1920s, the possibility of validating the tests against more precise electroacoustic measures. A hundred years ago, however, reports were appearing of
comparisons of the results obtained with tuning forks, the watch and the whisper tests (Eitelberg, 1886).

The clinical examination of auditory function uses six sound sources, some 30 auditory phenomena and two reflexes. The six sound sources are finger movement, speech, tuning forks, the monochord, the whistle, and the lever pocket watch. It is convenient to classify the tests according to their use of these sound sources.

**Finger friction tests**

Successive rubbing or snapping of a finger and thumb is an acoustic stimulus which is more commonly used by neurologists (for example see DeJong, 1967) than by other specialists. It is a convenient sound source, not only for screening for defects in the threshold of hearing, but also for screening for defects in sound localization (localization encompasses both directionalization and distance perception).

Klingon and Bontecou (1966) reported the use of the finger friction/snapping test to examine localization in the horizontal plane. They examined 156 patients, of whom 154 had a neurological disorder, together with a comparison group of 50 'neurologically normal nurses'. In each case, the thumb and forefinger 'were successively rubbed or snapped until the patient touched them or obviously could not locate them'. Care was taken to have the fingers closer to the patient than any other part of the examiner's arm. The sound was produced in a minimum of three locations. These locations were all in the horizontal plane; two of the locations were in the acoustic axis of the ear being tested, one adjacent to the auricle and the other within the patient's own arm length. The third location was at an intermediate distance but anterior to the acoustic axis and at an angle of just less than one radian subtended at the meatal orifice. Each stimulus was given at least three times. Thirty-three of the 42 patients with supratentorial neurological disease had defects in localization on the side opposite to the involved hemisphere; in three cases the localization defect was bilateral. None of the patients with neurological disease which was not supratentorial showed localization defects. Klingon and Bontecou also reported two other patients with localization defects. One had had the 'external ear virtually amputated because of a basal cell carcinoma'; the other had had 'an external and middle ear removal for malignant parotid tumour'. In the first case, 'gross acuity was bilaterally equal, but the patient could not localize the stimulus on the opposite side'. The second case was 'totally deaf on the operated side', but sound loud enough to be heard on the normal side could not be localized. Among the 50 nurses, only one showed a defect in localization; this was associated with a chronic suppurative otitis media.

Sound directionalization or localization in the vertical plane should also be tested since this function may be impaired when that in the horizontal plane is normal, and vice versa. Different mechanisms are involved for the two planes.

In 1881, Laborde had already suggested that stereoacusis was dependent on normal vestibular function. Both Young (1931) and Wallach (1940) subsequently showed that satisfactory vertical plane directionalization requires the ability to move the head.

Vertical plane directionalization also requires the possession of normal high-frequency auditory acuity. For example, normal horizontal plane directionalization, but impaired vertical-
plane directionalization has been observed in a patient with a bilateral abrupt high-tone loss (90 dB hearing level at 6 kHz; 75 dB at 8 kHz) above 4 kHz. The converse ability was observed in a patient with a total unilateral hearing loss (Butler, 1970).

Vertical-plane directionalization thus depends on the ability to correlate head movement with high-frequency acoustic clues. Neurological cases may similarly show different behaviour in the two planes. For example, a man with a parietal glioblastoma showed normal (or near normal) vertical plane directionalization, but impaired horizontal plane localization. The converse behaviour is sometimes associated with brainstem lesions (Walsh, 1957).

In the present author's experience screening hearing with finger friction or finger snapping noise is an extremely sensitive test, especially with regard to localization in the sagittal plane.

Dys-stereoacusis must be distinguished from what Heilman and his colleagues (1971) term 'auditory inattention'. The latter applies to conditions in which the subject reacts as though acoustic stimuli (whether unilateral or bilateral) were coming from one side only. The condition occurs in certain cerebral hemisphere lesions. In parietal lesions, there is no response to contralateral stimuli; in frontal lesions, there is no response to ipsilateral stimuli.

Speech tests

Clinical tests have been used:

(1) to measure hearing impairment
(2) to detect feigning
(3) to determine the nature of an organic hearing impairment.

Measuring hearing impairment

Since their introduction by Wolf (1871, 1879) and recommendations regarding their quantification by Lucae (1907), clinical speech hearing tests have been subject to criticism (Hartmann, 1877; Fowler, 1947; Trowbridge, 1947). Hartmann said that they were too complicated to ensure accuracy.

Despite early attempts, such as those of Reuter (1904) and Bezold (1897), to standardize the tests, King (1953) ranked the failure to standardize as still the principal source of variability. Reuter advocated limiting the use of speech material to monosyllables. Bezold suggested that only the residual air, that is the volume of air left in the lungs after an ordinary, but not enforced, expiration, should be used for whispering; this characterizes the forced whisper test. Difficulty in controlling the intensity and frequency content of a whisper, lack of control of ambient noise, and differing acoustic properties of test rooms are the other three factors which King pointed to as contributing to the variability of responses to the forced whisper test. The last item, however, may not be responsible for as much variability as was hitherto believed.
Fowler (1947) also pointed to the reflex arising of the voice by examiners when they increased their distance from the patient; such an effect can be responsible for paradoxical results. However, one expects to find that the greater the hearing loss, the nearer must the examiner be to the patient in order to be heard.

In a truly free field, where sound can spread spherically in all directions without encountering any reflecting surfaces, the sound pressure decreases as the distance from the source increases. The form of the relationship between sound pressure and distance is given by the equation:

\[ p_i = p_1 \left( \frac{r_1}{r_i} \right) \]  

(1)

where \( p_i \) = sound pressure at point of measurement, \( p_1 \) = sound pressure at some particular distance, \( r_p \), from the acoustic centre of the source, and \( r_i \) = distance from acoustic centre at which sound pressure \( p_i \) is measured; therefore

\[ p_i = k \left( \frac{r_1}{r_i} \right) \]  

(2)

This equation is the mathematical expression of the inverse distance law. It is a special case of spherical divergence. The intensity of a sound, \( I \), is proportional to the square of its pressure. In other words, the intensity of a sound decreases in proportion to the square of the distance from the sound source (Poisson, 1808). The inverse distance law is therefore sometimes referred to as the inverse square law.

As Young (1957) pointed out, because of reflections from the walls, the sound pressure from a source in most rooms does not decrease as rapidly as would be predicted from the inverse distance relationship. If the law were obeyed the sound level would drop by 6 dB with doubling of the distance. King's (1953) data showed that, with increase in distance from 1 to 5 metres, the sound level of the words in each room fell by 11.5 dB instead of the predicted 14 dB.

King reported that he had attempted to standardize the test:

‘by placing the examinee at 20 ft from the examiner. The eyes are shielded by an assistant to prevent lip-reading, the ear under test is towards the examiner, while the other ear is blocked by light intermittent pressure of the assistant's index finger on the tragus. This does produce efficient masking, which is as important in testing monaural hearing in this as in other tests of auditory acuity. The distance at which the examinee can repeat all words correctly is then found.’

Despite the imprecision of the forced whisper and the conversational voice tests, many clinicians find them useful as clinical screening tests. For example, Sohoel (1956) found the whispered and conversational voice tests detected 75% of audiometrically demonstrable hearing losses in children after otitis media.

A modification of the test which can be performed without an assistant is as follows. The examiner uses one hand to shield the patient's eyes to prevent lipreading and the index finger of the other hand masks the non-tested ear by intermittent pressure on the tragus. With
this method, the maximum distance at which clinical speech tests can be given is about 50 cm. Nevertheless, it would appear that a forced whisper at this distance will screen out subjects with any significant degree of hearing impairment. If the subject fails to respond to a forced whisper at this distance, the hearing impairment may be quantified by an examiner bringing the lips progressively nearer to the subject's ear. The conversational voice test is employed if the subject fails the forced whisper test at 50 cm.

**Detecting feigning**

There are five clinical speech tests which are helpful in exposing a feigned (simulated) unilateral hearing loss, that is Erhard's (1872) loud voice test, Lombard's (1911) test, Hummel's (1898) double conversation tests, Teuber's two-tube test, and Callahan's (1918) test.

Erhard's loud voice test is suitable for suspected feigned total unilateral hearing loss. The test is based upon the watch test of the same name. The test depends on the fact that occlusion of the meatus of a normally hearing ear does not cut out sound completely. Experimental studies indicate that occlusion by firm finger-tip pressure on the tragus would attenuate speech by less than 30 dB.

The suspected malingering is asked to close the eyes and to repeat the words which are heard. The patient is told that the normal ear will be blocked up, and the examiner then presses the tip of the index finger on the subject's tragus. The words are delivered in a loud voice to the suspected ear. Failure to respond indicates malingering. Even with the head shadow effect, there would be insufficient attenuation to prevent the normal ear from hearing.

Lombard's voice reflex test is also appropriate to a feigned total unilateral hearing loss. The test was first described by Bárány in 1910. It depends on the normal monitoring of the sound level of speech by the auditory system; the level is governed by the perceived signal-to-noise ratio. Thus an increase in the ambient noise level results in a speaker raising the intensity of the voice, for example as in a factory. The patient who is suspected of malingering is asked to read a piece of prose. A Bárány noise box is applied to the good ear. After the patient has started to read, the noise box is switched on. If there is an organic hearing loss in the suspected ear, the patient's voice level is appreciably raised. If there were a feigned hearing loss, the normal monitoring would be unimpaired.

Hummel's double conversation test depends upon the confusing effect of different voices giving different messages to the two ears. If one ear is totally deaf, the subject will hear only one speaker. The test is performed with two speakers, each using a speaking tube to one ear. Each speaker asks different questions of the subject.

Teuber (quoted by Müller, 1869) used two tubes, one end of each being coupled to an ear of the patient. The tubes were led to the examiner who stands behind the patient and asks the patient to repeat the words that are heard. The examiner speaks into the free ends of the tubes. During the period of speaking alternate tubes are compressed. The patient, being unable to realize what is happening, often repeats a word that could have been coming only through the tube coupled to the ear with the alleged hearing loss.
The stethoscope test applies to Coggin's (1879) description of a similar procedure using this instrument.

Callahan (1918) based his test upon the phenomenon of binaural fusion which is used in the Stenger test (see below). The test compared responses of the subject to speech delivered monaurally and binaurally via lengths of tubing (see Hinchcliffe, 1981).

### Determining the nature of an organic loss

Information regarding the nature of an organic loss of hearing may be derived from:

1. comparing the relative hearing impairment for low tone words with that for high-tone words
2. comparing results for the forced whisper with the conversational voice test
3. comparing the ability to hear speech with the ability to hear tuning forks.

In disorders characterized by low-frequency hearing losses, the patient will give a relatively poor response to words such as moon, room, rude; with high-frequency losses, there will be a relatively poorer response to words such as seize, six, tease.

Gradenigo (1912) proposed the *index vocalis* as a method for determining the presence or absence or loudness recruitment. The index is the ratio of the distance that a subject hears a forced whisper to the distance that a conversational voice is heard. Ears showing a recruiting hearing loss are considered to have a smaller index than those not recruiting. The frequency pattern of the hearing loss will, however, influence this index. The distance at which a forced whisper can be heard correlates best with the pure-tone threshold at 4 kHz and the conversational voice with that at 1 kHz (Hinchcliffe, 1967). Because of this dependence upon the frequency pattern of the hearing loss, the results for clinical speech tests may show poor correlations with those for other clinical tests, for example watch tests, of auditory function. According to Feldman (1960), this observation had already been made by Frank in 1849.

A patient who shows a very poor performance on speech tests but whose response to tuning-fork tests is not so poor should be suspected of having a neuronal lesion, particularly a vestibulocochlear schwannoma. An apparently normal response to tuning-fork tests but no response at all to speech tests indicates an aphasia.

### Tuning-fork tests

#### The tuning fork

The basic transverse vibration pattern of a straight rod or bar is such that, with both ends free, it vibrates about two nodes (points at rest in a vibrating structure). If the bar is bent on itself, the nodes approach each other at the point of flexion.
In all cases of rectangular bars vibrating transversely, the frequency of vibration \((f)\) is given by:

\[
f = \left(\frac{kt^2}{l^2}\right)\left(\frac{E}{r}\right)
\]

where \(f\) = frequency in hertz; \(k\) = a constant; \(t\) = thickness of bar in metres; \(l\) = length of bar in metres; \(E\) = Young’s modulus of elasticity; \(r\) = density of bar material.

Thus, for a given material, the fundamental frequency of a vibrating rectangular bar is proportional to the square of the thickness and inversely proportional to the square of the length of the bar.

Overtones generated by vibrating bars are not harmonics as in the case of vibrating strings. For a bar clamped at one end only ('clamped-free' condition), the frequencies of the first three overtones, expressed as the ratios of the fundamental frequency, are 6.3, 17.6, and 34.4 Hz. Rayleigh regarded the tuning fork as equivalent to two 'clamped-free' bars mounted on a heavy stiff block of metal (Wood, 1942).

A tuning fork is produced by attaching a U-shaped metal bar to a straight piece, termed the 'stem'. In respect of medical tuning forks, such as the Gardiner-Brown, it is advantageous to have a disc-like portion of the stem for the examiner to grip between the fingers, and an expansion (footpiece) at the otherwise free end; the footpiece provides a suitable surface of the tuning fork for the transmission of sound to the skull and, at the same time, it enables the fork to be applied firmly to the skull without discomfort. The limbs (tines) of the U are termed the 'prongs', the portion of the fork joining these, the 'base', and the point at which the prong joins the base is known as a 'shoulder'. The sides of the prongs which face one another are termed the 'inner normal faces'; the other pair of sides which are parallel to these are termed the 'outer normal faces'; the sides of the prongs which are at right angles to these are known as the 'parallel faces of the fork'. When a force is applied to an outer normal face and at right angles to it, the prongs are set in transverse vibration, and they alternatively approach and separate from each other. The two nodal points which are situated in the shoulders remain stationary and the internodal segment vibrates in the plane of the long axis of the prongs; this vibration is transmitted as a longitudinal vibration, to the attached stem. If struck correctly, the tuning fork produces a comparatively pure tone whose frequency is remarkably constant. Although it is now over half a century since, in the UK, the Section of Otology of the Royal Society of Medicine set up a Committee for the Consideration of Hearing Tests, their findings in respect of the use of tuning forks remain valid to this day. The Committee deliberated for four years before publishing its report in 1933. The Committee recommended, among other things, that the prong should be struck at a point about one-third of its length from the free end. By this means, a pure tone is produced and overtones are kept to a minimum. The Committee also recommended that a prong should be struck sharply against some resistant, but elastic, object, for example a mass of hard rubber; failing the availability of this, testers may strike the tuning fork on their thenar eminence, or over one of their femoral condyles.

When testing hearing by air conduction, the fork should be held as close to the auricle as possible, without touching protruding hairs, and such that the acoustic axis of the fork is coincident with the anatomical axis of the external acoustic meatus. The acoustic axis
corresponds to a line which is perpendicular to the normal faces of a fork and which passes through the point that is equidistant from both the free and the parallel faces of the corresponding prong.

As with any other instrument, tuning forks can, and should, be calibrated. When this has been done, the capital letters A, B and C will be engraved one under the other on the base of the fork. Against each letter there will be a number. The numbers opposite A and B specify the decay rates of the generated tone when the fork is used in the air-conduction mode or the bone-conduction mode, respectively. The number against A or B refers, in each case, to the time in seconds which is required for the sound intensity to fall 3 dB when sound is being transmitted by air or bone conduction, respectively. (The intensity of sound generated by a tuning fork decays exponentially so that the change in decibels is linear with time.) The letter C stands for what Yates (1933) referred to as the characteristic of the fork, and what Hallpike (1933) said would be better termed the 'stem-transmission factor'. This was defined as the number of half-intensity periods for which the fork could be heard by air conduction after hearing by bone conduction had ceased. A half-intensity period is the time required for the sound intensity to fall to a half, that is undergoa3d B change. Thus, if a tuning fork is heard for 42 seconds by air conduction after it has ceased to be heard by bone conduction, and the value of A is 7 seconds, then the characteristic, C, of the fork is 6. This is equivalent to 6 × 3 = 18 dB.

There are cogent arguments for calibrating tuning forks but the values for A and B should now be expressed in decibels per second (dB/s) and the value for C in decibels. Further studies are required to determine the optimum values for A, B and C at specified frequencies. These values should then be standardized.

**Measurement of hearing loss**

Tuning forks can be used, not only qualitatively to determine the type of hearing impairment, but also quantitatively to measure the degree of hearing loss. Rudolph Koenig (1832-1901) had a set of 150 tuning forks ranging in frequency from 16 Hz to nearly 22 kHz (Boring, 1942). Both Fletcher (1925) and Hallpike (1927) proposed a graphic method for representing the results of tuning-fork tests.

In measuring the amount of hearing loss, tuning forks can be accurate to within 10 dB, which is a level of accuracy obtainable with audiometry (Lumio and Arni, 1949). When used to measure the degree of hearing loss by either air conduction or bone conduction, the 1933 report of the Committee for the Consideration of Hearing Tests recommended that the sound be applied intermittently, in the appropriate mode, for durations of one second, with three-second intervals; this would enable the clinician to reduce the effect of any auditory adaptation on the measurement of threshold. Hopefully, the examiners have essentially normal hearing, and can compare the patient's hearing with their own hearing to obtain a measure of the hearing loss. By means of the A and B values on the fork, the time differences between the patient's end-points of hearing and those of the examiner can be converted to hearing levels in decibels for both air- and bone-conducted sound.
Diagnostic tests

There are more than 20 tuning-fork tests available to the clinician to distinguish feigned hearing loss from organic hearing loss, conductive hearing loss from sensorineural hearing loss and cochlear hearing loss from neural hearing loss.

Non-organic hearing loss

Apart from detecting inconsistencies in responses, a bilateral non-organic hearing loss is difficult to diagnose with tuning-fork tests, especially if a total bilateral hearing loss is simulated.

Unmasked bone conduction

Should a patient deny hearing by both air and bone conduction on one side, the vibrating tuning fork is then applied to the opposite mastoid process. Should the patient then acknowledge hearing the fork, it is then applied to the mastoid process on the suspected side. Failure of the subject to acknowledge the sound on this side indicates a non-organic hearing loss. The explanation for this is that there is little or no attenuation across the skull for bone-conducted vibrations (Lierle and Reger, 1946). As a corollary of this, a vibrating tuning fork applied to any point on the skull, is providing an essentially similar acoustic stimulus to both cochleae. Consequently, a masking sound must be applied to the ear not being tested whenever valid bone-conduction hearing measurements are required.

Teal test

Should the patient acknowledge hearing bone-conducted, but not air-conducted, sound on one side, use should be made of the Teal (1918) illusion. The patient is asked to close the eyes, the examiner adding 'so that you can concentrate on hearing the sound better'. Two tuning forks of the same frequency are now used, but only one is set into vibration. The prongs of the vibrating fork are brought up to the ear to deliver an air-conducted stimulus and, at the same time, the footpiece of the other (non-vibrating) fork, is applied to the corresponding mastoid process. If hearing is normal, the subject perceives a sound stimulus and, at the same time, perceives the fork footpiece applied to the mastoid process. Not realizing that two tuning forks are being used, the subject usually does not dissociate the two perceptions, assuming that the sound is coming from the fork whose footpiece is applied to the mastoid process, and therefore acknowledges hearing a sound.

Stenger test

In 1878, Tarchanow reported an experiment in which he placed two earphones one on each side of an observer. He noticed that, with pure tones of equal intensity, the bilateral stimulation 'fused' into a single sound in the median plane. Urbantschitsch (1881) showed that, when the relative intensities in the two earphones changed, the apparent source of the fused sound moved towards the louder side. Bloch (1893) pointed out that this phenomenon could be used as a test for simulated unilateral hearing loss. The application of the phenomenon to the detection of malingering is now known as the Stenger (1900) test.
A pair of tuning forks (256 or 512 Hz) is required for the clinical application of the test. During the course of the test these forks are separately or concurrently disposed on one or other side of the head. Differences in intensity are achieved by placing the forks at different distances from the corresponding ear, that is by making use of the inverse distance law.

The test is performed as follows. The subject is asked to close the eyes and told 'so that you can concentrate on listening to the sounds'. The subject's ability to hear a tuning fork at a distance of, say, 14 cm distant from the good ear is confirmed; refusal to acknowledge anything when the same tuning fork is placed just lateral to the auricle on the 'bad' side is also verified. The test is then repeated using both tuning forks simultaneously, and at those same distances. The subject is asked if anything can be heard. He signifies that a sound can be heard if the hearing loss is real, that is organic. However, if the subject has a normally functioning auditory system, then, because of the Tarchanow phenomenon, a single fused sound will be heard. This will appear to come from the side of the stronger stimulus, that is from the 'deaf' side. If the subject is feigning a hearing loss on that side, he/she will deny hearing anything. Clearly this answer cannot be correct since he/she had previously acknowledged hearing that one tuning fork alone and at the same distance from the good ear.

In practice, when the two vibrating tuning forks are used simultaneously, the examiner would bring the fork on the side of the good ear a little nearer (say at 10 cm) to the head. If the subject previously could hear the vibrating fork at a distance of 14 cm, then there should be no doubt about the subject being able to hear now at a distance of 10 cm.

The Stenger test can be performed with an audiometer provided that this instrument can deliver tones of identical frequency, and preferably cophasic, to the two ears but with separate attenuators for each ear. Frenzel (1932) (cited by Feldman, 1960) in Germany and, subsequently, MacKenzie (1940) in the USA reported the use of an audiometric Stenger test; Cheesman and Stephens (1965) showed how Békésy audiometry can also be adapted for this purpose. Auditory thresholds determined using the principle of the Stenger test are referred to as minimum contralateral interference levels (Martin, 1972).

Bergman (1964) has used what might be termed a 'reversed Tarchanow-Urbantschitsch phenomenon' for monaural threshold determinations.

Chimani-Moos

This test depends on a phenomenon discovered by Wheatstone (1827). In a normally hearing subject, a bone-conducted sound is lateralized to an ear when its external meatus is occluded. It also depends on the presumption that this phenomenon would appear paradoxical to the layman. The test was first reported by Moos in 1869.

Let us consider the case of a man who acknowledges hearing in, say, the right ear but is simulating a total hearing loss on the left side. A 256-Hz tuning fork is applied to the skull and he is asked 'Where do you hear that, left, right, or centre?' If he wishes to demonstrate that he can hear only with the right ear, he may say 'right'. The examiner then says 'I shall now block up that right ear', as firm pressure is applied with the finger to the right tragus. 'Where do you hear the sound now?' Although hearing the sound in the right ear, the subject
may think this unlikely, and deny hearing any sound at all. A truthful person would of course acknowledge the sound in the right ear. In practice, the Chimani-Moos test has not been found to be as helpful as other tests for malingering.

Conductive hearing loss

Wollaston's test

In 1820, Wollaston reported that some hearing-impaired patients have a loss which is predominantly low frequency; others have a predominantly high-frequency loss. Moreover, he showed that these frequency impairments were correlated with conductive and sensorineural hearing losses, respectively. Although this is a good generalization, exceptions spring to mind. Thus endolymphatic hydrops is a low-frequency sensorineural hearing loss and an ossicular chain disruption may give rise to a high-frequency conductive hearing loss.

Schwabach test

The Schwabach (1885) test is a measure of the threshold of hearing by bone conduction of an unoccluded ear (relative bone conduction). It is thus the clinical equivalent of what is done in conventional bone-conduction audiometry. As Huizing (1975b) pointed out, the method was also described as early as 1846 by Schmalz. The test compares the patient's ability to hear a tuning fork by bone conduction with that of a normally hearing person.

The test is usually performed by placing the footpiece of a vibrating tuning fork on the patient's mastoid process and asking the patient to say when the sound is no longer heard. As soon as he/she indicates that this point has been reached the footpiece of the fork is transferred to the mastoid process of a normally hearing person. Usually, examiners consider their own hearing to be normal and so compare the patient's hearing with their own hearing by bone conduction. If the observer can hear the tuning fork after the patient has ceased to hear it, the Schwabach test is designated as 'shortened'. This is the finding in sensorineural hearing loss. If the examiner cannot hear the tuning fork immediately after it has been transferred to his/her own mastoid process, the sequence of testing is then reversed. One then determines how long the patient can hear the tuning fork after it has ceased being heard by the examiner. Such results may be obtained in conductive hearing losses.

Because of the absence of any appreciable interaural attenuation for bone-conducted sound, the test should be repeated with a Bárány noise box applied to the non-tested ear. This will mask that ear and ensure that, at least for low frequency tuning forks, the Schwabach test measures the bone-conduction hearing level of the ear under test. Unfortunately, some Bárány boxes produce a noise that is so loud that it may mask the tested ear (Dieroff, 1958).

The mechanism of bone conduction is complex (Tonndorf et al, 1966). However, the 'prolonged' Schwabach response on conductive hearing loss is probably due primarily to the exclusion of the masking effects of the ambient noise which applies to usual clinical test situations. The occlusion effect (see later) is probably only of secondary importance in the clinical situation.
The bone-conduction hearing level of the corresponding ear can be calculated by converting the time difference between the two thresholds to a value in decibels using the B calibration on the tuning fork.

As mentioned previously, in order to minimize adaptation effects, the Royal Society of Medicine's 1933 report recommended that the fork be applied intermittently.

**Pomeroy's test**

Pomeroy's (1883) test is a measure of the threshold of hearing by bone conduction of the occluded ear (absolute bone conduction). The value of the test has been stressed by Hallpike (1927) and others. The test is performed in the same manner as the Schwabach test except that the examiner applies firm pressure to the tragus of the ear being tested. The effect is thus to standardize by 'giving everyone a conductive hearing loss' and excluding the masking effects of the usual clinic ambient noise; the latter is the principal factor accounting for the variability of bone-conduction hearing levels determined by tuning forks in the clinic. Because of this attenuation of room masking noise and the operation of the occlusion effect, the Pomeroy test will normally give lower, that is better, thresholds than the Schwabach test. Patients with conductive hearing losses will show either a reduced difference between the thresholds determined by the two tests, or identical thresholds. Like the Schwabach test, a 'shortened' Pomeroy test is indicative of a sensorineural component in the hearing loss.

The Pomeroy test may also be performed as an audiometric procedure. In this case it is usual to occlude the meatus with the earphone used for air-conduction testing.

Aubry and Giraud (1939) stressed the importance of measuring both absolute and relative bone-conduction levels. Judging from the results of studies made with bone-conduction audiometry, it would appear that variability in the results of the Pomeroy test will arise, among other things, from differences in the pressures used to occlude a meatus as well as those used in applying the fork footpiece to the mastoid process.

A number of tuning-fork tests have been described which compare the perception of air- and bone-conducted sound in the same subject. These are the Bing, the Rinne, the Lewis, and the Federici tests.

**Bing test**

Comparison of occluded and unoccluded bone-conduction thresholds in the same subject constitutes what has become known as the Bing or occlusion test. Bing (1891) was the first to report the use of the test for the diagnosis of disorders of hearing although the phenomenon on which the test is based was known to Rinne (Huizing, 1975b).

The phenomenon whereby a bone-conducted sound is perceived to be louder when the ipsilateral meatus is occluded was first described by Wheatstone in 1827. The phenomenon is primarily due to the elimination of the normal high-pass filter effect produced by the unoccluded external acoustic meatus (Tonndorf et al, 1966).
The test may be performed by either a loudness comparison method or by a threshold method. In either case, the footpiece of a vibrating tuning fork is applied to the mastoid process. After ensuring that the patient hears it, the corresponding external acoustic meatus is occluded by pressing a finger tip on the tragus. The patient is then asked: 'Does that make the sound quieter, louder, or no change?' If the subject says that the sound becomes louder, the Bing test is said to be positive; other responses are termed 'negative Bing responses'. An increase in loudness after occlusion of the meatus occurs with normal sound-conducting mechanisms, that is those producing conductive hearing losses, are associated with negative Bing response.

With the threshold method, the tuning fork is intermittently applied to the mastoid until it is no longer heard. The ipsilateral meatus is then occluded by pressure on the tragus. The patient is then asked whether or not anything can be heard. In an assessment of the Bing test using the loudness comparison method, Golabek and Stephens (1979) found that this test could detect conductive hearing losses of 9 dB or more at 512 Hz. Sheehy, Gardner and Hambley (1971) said that they found the Bing test more valuable than the Rinne test in differentiating between a conductive and a sensorineural hearing loss.

The preferred frequency for the tuning fork for both the Bing and the Rinne test is 256 Hz.

**Rinne (or Polansky-Rinne) test**

This is perhaps the best known of the tuning-fork tests used to distinguish conductive form sensorineural hearing losses. Rinne described all the details of the tests in 1855. However, Huizing (1975a) pointed out that the principles of the tests were first described by Polansky (1842), a Viennese otologist.

In the Rinne test, the patient's ability to hear by bone conduction is compared with that by air conduction. The clinician can simply ask 'Which is louder, number one sound' (holding the vibrating fork with the prongs near the ear) 'or number two sound?'. (transferring the same vibrating tuning fork to press the footpiece gently but firmly on the corresponding mastoid process). A normally hearing subject will say 'Number one sound'; so will a subject with a sensorineural hearing loss in the affected ear, unless the loss is marked; a subject with an incipient conductive loss or with a conductive component up to 20 dB in a mixed loss, will also give the same response. The response in which a sound is heard better by air conduction than by bone conduction is referred to as a Rinne positive response.

If the tuning fork is heard better by bone conduction than by air conduction then the test is termed a 'Rinne negative response'. Such cases may be due either to the bone-conduction sounds being heard in the opposite ear (in cases of severe or total hearing loss in the tested ear), or an appreciable impairment of sound transmission in the affected ear, that is a conductive hearing loss in that ear in excess of about 15-20 dB hearing level. The two conditions can be differentiated by applying a Bárány noise bow to the opposite ear. This masking noise raises the threshold of hearing in the non-tested ear to such a level that the tuning fork cannot be heard in that ear by cross-hearing. Thus if the Rinne test is repeated with noise applied to the opposite ear and this abolishes hearing by bone conduction on the tested side, the Rinne response is said to be a false negative Rinne response. If the Rinne
negative response (bone conduction better than air conduction) is still obtained with a noise box applied to the opposite ear, then the result is said to be a true Rinne negative response.

Repetition of the test with contralateral masking is still required in bilateral Rinne negative responses, since such a result may be found in a patient with a pure conductive loss on one side and a total sensorineural hearing loss on the other side. Responses of a subject indicating a bilateral false negative Rinne response indicate a non-organic hearing loss.

In a patient with a unilateral hearing loss showing a negative Rinne response, the direction of lateralization of the Weber test (see below) would indicate to the examiner the nature of this hearing loss without recourse to contralateral masking; in practice, owing to the vagaries of the Weber test, a Rinne test giving a negative response should always be repeated with contralateral masking.

The paradoxical terminology by which the normal response to the test is termed ‘positive’ and an abnormal response ‘negative’ is attributed to Bezold. As early as 1885, a plea (by Politzer) was made to end the confusion but the terminology has stuck (Tschiassny, 1946).

The validity of the Rinne test has been investigated by comparison with both otoscopic appearances (Hinchcliffe and Littler, 1961) and autopsy findings (Polvogt and Bordley, 1936). Studies on a random sample of a rural population in the UK indicated a negative Rinne response was invariably associated with abnormal otoscopic appearances. Exceptions would correspond to otosclerosis. The clinicopathological study showed that a negative Rinne response was associated with one or more lesions involving the ossicular chain in every ear of a sample of subjects who had had impaired hearing during life.

Erroneous true Rinne negative responses may be obtained with tuning forks of frequencies 128 Hz and below. These erroneous responses are due to responses to vibrotactile stimulation (DeWeese and Saunders, 1973; Gelfand, 1977).

The reliability (reproducibility) of the test has also been investigated in a sample of the rural population. A total of 75 individuals were retested one month after the initial test. Twelve of the ears had given a negative response on the first occasion, the remainder a positive response. On the second occasion, one of these 12 showed a positive response, another a neutral response (tuning fork heard as long by air conduction as it is on the mastoid process). All the ears (144) that had given a positive response to the first test gave a positive response on the second test (Hinchcliffe and Littler, 1961).

The sensitivity of the test has been investigated by comparing the test response with audiometric measurements. Bunch (1941) compared the Rinne response for a 512-Hz tuning fork with the difference between the air- and bone-conduction thresholds at the same frequency. The air-bone gap was taken as a measure of conductive hearing loss. The difference between the two thresholds showed two distinct distributions depending upon whether the Rinne response was positive or negative. The two distributions intersected at a point equivalent to about 20 dB of conductive hearing loss, with little or no overlap. Other reports on the sensitivity of the Rinne test using the standard (loudness comparison) technique have given values, at 512 Hz, of 17 dB (Hinchcliffe and Littler, 1961), 25 dB (Crowley and Kaufman, 1966), 15 dB (Sheehy, Gardner and Hambley, 1971) and 19 dB (Golabek and
Stephens, 1979). The differences are not surprising when one considers that different patterns of tuning forks made of different materials were used in rooms of varying acoustic treatment (ranging from untreated clinic rooms to ‘sound insulated and absorbent rooms’). The construction of the fork will clearly influence the characteristics of the fork.

Sheehy, Gardner and Hambley (1971) point to a number of other factors which may account for varying results, that is placement (the footpiece should be over the suprameatal triangle), whether or not the fork touches the auricle, force of application, force with which fork is excited (if too strong, overtones may be produced), and whether or not spectacle frames are interfering with the conduction of the tests. A systematic study of some of these factors has been made by Feldman and Sann (1967).

The sensitivity of the Rinne test can be improved, and quantification provided, by following Bezold’s suggestion (Feldman, 1960) of measuring the time which elapses between cessation of bone-conduction excitation and cessation of air-conduction excitation. For example, if the A value of the particular fork used is 4, and the C value, 6, then a normally hearing person would be expected to hear the fork by air conduction for:

$$A \times C = 4 \times 6 = 24 \text{ seconds}$$

after it had become inaudible by bone conduction. This period of time would correspond to a fall in sound intensity of

$$3 \times C = 18 \text{ dB}$$

A hearing-impaired person who is able to hear the fork for t seconds by air conduction after ceasing to hear it by bone conduction will have a conduction loss given by:

$$H_c = 3 \times \left( C - \frac{t}{A} \right) \text{ dB}$$

where $$H_c = \text{conductive loss in dB}$$, $$A = \text{decay rate of fork in air conduction mode expressed as the time required for a fall in intensity of 3 dB}$$, and $$C = \text{a characteristic of fork}$$.

For a patient with a Rinne negative response, the results can correspondingly be quantified by measuring the time for which the fork can be heard by bone conduction after ceasing to be heard by air conduction. In this case, the degree of conductive loss is given by:

$$H_c = 3 \times \left( C + \frac{t}{B} \right) \text{ dB}$$

where $$H_c = \text{conductive loss in dB}$$, $$B = \text{decay rate of fork in bone-conduction mode expressed as the time required for a fall in intensity of 3 dB}$$, and $$C = \text{a characteristic of fork}$$.

As Golabek and Stephens (1979) have shown, the quantification of the Rinne negative response by this method is a more valid procedure in conductive losses than is quantification of the Rinne positive response.

As indicated by Golabek and Stephens' report, timing of Rinne test responses on a group of conductive hearing losses of varying severity can be used to determine the A, B and
C values for a particular tuning fork. The duration by which hearing by sound transmission in one mode exceeds that in the other mode is plotted against the corresponding audiometric measure of hearing loss. The slopes of the two curves (one for Rinne positive responses, the other for Rinne negative responses) will be measures of the A and the B values, respectively. The point of intersection will correspond to the value of C in decibels.

Gelfand's (1977) paper indicates that the probability of obtaining a Rinne negative response in a group of conductive hearing losses decreases from 0.78 for a 128-Hz tuning fork to 0.06 for a 2048-Hz fork. Unfortunately, it is not clear whether or not this group is representative of conductive losses seen in clinical practice. Although there was audiometric control, it is not clear to what extent this decreasing probability of a negative response is related to the decreasing sensitivity of the test and to what extent it is related to the, in general, decrease in conductive loss with increase in frequency.

**Lewis test**

In the Lewis (1925) test, a vibrating tuning fork is applied to the posterior root of the zygomatic process of the temporal bone. As soon as it ceases to be heard in that location, the footpiece is applied to the ipsilateral tragus. Lewis claimed that the sound of the fork was again heard in all cases of sensorineural hearing loss and of conductive hearing losses except those due to stapedial ankylosis. In the present author's experience this abnormal response is also observed in cases of conductive loss other than those due to stapedial ankylosis.

The Lewis test would appear to be less sensitive than the Rinne test.

**Weber (or Schmalz-Weber) test**

The literature consistently refers to Weber (1795-1878) as the originator of the tuning fork test which bears his name. However, according to Huizing (1973), Weber's (1834) book shows that he described only the occlusion phenomenon and lateralization to the occluded ear.

Huizing pointed out that Schmalz appears to have been the first to apply Wheatstone's lateralization phenomenon to clinical medicine. Schmalz (1846) have a complete description of the test and its application to the differential diagnosis of hearing disorders. Nevertheless, according to Feldman (1960), Schmalz gave credit to Weber for having encouraged him in this work.

The Weber test is valuable in cases of unilateral hearing losses, and usually only in such cases. In this test, the examiner applies the vibrating fork to the patient's head in the midline (forehead or vertex) and asks 'Where do you hear that sound, left, right, or centre?' If the patient has a left hearing loss and lateralizes to that side, then this indicates that the loss is conductive (sound transmission loss) in type; if the patient lateralizes the sound to the normally hearing ear, then the hearing loss on the affected side is sensorineural. Responses obtained with the Weber test should be used only in the context of the results of the battery of hearing tests as a whole; anomalous results, even including lateralizing to a 'dead' ear, are obtained not infrequently.
The mechanism of the test is complex. When a tuning fork is lateralized to a conductively impaired ear, two situations must be distinguished (Tonndorf, 1976). First, with otitis media, for example, there is a combination of ossicular loading, due to the oedema of the tissues covering the ossicles, and increased damping owing to the presence of free fluid in the middle ear, giving the audiometric curve the peculiar shape that was first described by Lierle and Reger (1946). Secondly, with stapes ankylosis, there are considerable phase advances at low frequencies, and some phase lags at higher ones. The cause of this lies in the elimination of the middle-ear bone-conduction components and the consequent prevention of sound leakage through the fenestra ovalis. The phase advance leads, of course, to a lateralization towards the involved ear and is capable of overcoming the loss that, being due to a so-called 'Carhart notch', is still small at low frequencies.

The validity of the Weber test has been assessed by comparing lateralization with the Rinne response for 722 ears of a random sample of a rural population (Hinchcliffe and Littler, 1961). For the purpose of this analysis, lateralization was accepted only if there was lateralization to one and the same side when the fork footpiece was applied to the forehead and the vertex. There was confirmation of the clinical experience that ears which are unequivocably lateralized on the Weber test also tend to give a true unilateral negative Rinne response.

The reliability of the test was assessed by the same authors on 78 unselected individuals from the same sample. These individuals were tested after an interval of one month. For forehead applications of the fork, the initial results were duplicated in 72% of cases; for vertex applications, the results were duplicated in 86% of cases. For cases (about 75% of the sample) where the first test response was the same for both forehead and vertex applications, replication occurred in about 75% of cases. It should be emphasized that these results apply to the general population; higher reliability obtains for a predominantly hearing-impaired clinic population.

The Weber test can, of course, be performed using an audiometer (Bunch, 1943). The bone-conduction transducer is applied to the forehead in the midline. Rubenstein and Klein (1957) used this method to assess the sensitivity of the test. They claimed that patients with an 'air-bone gap' of as little as 5 dB could lateralize easily and correctly. These authors also presented a formula based upon the air-conduction and the bone-conduction thresholds by means of which it is possible to predict the side that will be lateralized. The high predictability has, however, not been borne out by the studies conducted by Golabek and Stephens (1979). These authors, however, determined lateralization with the tuning fork; Rubenstein and Klein (1957) did so with the audiometer. Moreover, for a variety of reasons, Rubenstein and Klein rejected 150 of their initial sample of 250 subjects before analysing the results. Since lateralization involves interaural phase (Christian and Roser, 1957) as well as intensity differences (Spoor, Schmidt and Van Dishoeck, 1957), it would appear unlikely that a simple consideration of air- and bone-conduction thresholds alone could provide a valid prediction of lateralization.

Groen (1962) considered that the results of the lateralization test were not valid for frequencies in the range of 1-3 kHz; paradoxical results often occur in this region.
Analysis of results for the lateralization test in four positions (upper incisor teeth, nasal bridge, forehead, vertex) showed that the greatest consistency occurred with a tuning fork on the upper incisors and the least when on the forehead (Golabek and Stephens, 1979).

**Gellé test**

This test is based upon a phenomenon which was first discovered by Wheatstone (1827). The significance of the absence of the phenomenon was first described by Gellé (1881), a Parisian otologist, who also first reported its application to clinical diagnosis (Gellé, 1885). The phenomenon consists of the decrease in the loudness of a bone-conducted sound when the air pressure in the ipsilateral external acoustic meatus is increased. This effect is found in individuals with a normal sound-conducting mechanism. The phenomenon is absent in patients with stapedial ankylosis.

Tonndorf (1976) said that the change in air pressure mechanically biases the tympanic membrane by displacing it inwards (increased pressure) or outwards (decreased pressure). It is the only test in this series in which lateralization occurs away from the tested ear.

By conducting the test with an audiometer, it may be shown that the absolute bone-conduction threshold is impaired by about 7 dB for frequencies lower than 2 kHz. Arnold and Schindler (1963a) adapted Békésy audiometry to do the test. Nevertheless, doubts are still expressed regarding the validity, reliability, and sensitivity of the test. Dankbaar (1970) found considerable variation. He re-emphasized that special attention needs to be paid to the influence of the occlusion effect and the necessity for masking in the bone-conduction measurement. De Wit and van Dishoeck (1959) emphasized the converse effects on bone conduction of meatal occlusion (Bing test) and meatal pressure (Gellé test). The effects of meatal pressure change are much greater on air conduction than on bone conduction and negative pressure was more effective than positive pressure. Maximum effects were at 250 Hz; a 6 kPa negative pressure at that frequency impaired the air-conduction threshold by 15-25 dB.

There is also the confounding effect of auditory tubal malfunction. With slight degrees of malfunction, negative pressure becomes less effective than positive pressure in shifting both the air-conduction and the bone-conduction thresholds, the effects on air conduction being more marked than on bone conduction (Arnold and Schindler, 1963b).

Judged by changes in the bone-conduction threshold at 1 kHz, the Gellé test is an insensitive one. At this frequency, Jones and Edmonds (1949) found that the test was unable to detect conductive hearing losses unless there was at least a 30-dB air-bone gap. Clarke (1929) also queried the sensitivity of the test when conducted with tuning forks. Moreover, Clarke also reported a third type of response to the Gellé test. This response, which he termed the 'reversed response', was characterized by an increase in loudness occurring with increase in meatal pressure.
Sensorineural hearing loss

If it has been demonstrated that a hearing loss is not feigned and, if organic, not conductive, then it must be sensorineural. A number of tests are available to distinguish a cochlear hearing loss from a neuronal hearing loss.

Cochlear hearing loss

In cochlear disorders there are abnormalities of both frequency coding and intensity coding which may be detectable with tuning-fork tests.

Frequency coding abnormalities

Frequency coding abnormalities in auditory disorders became recognized in the nineteenth century. Tuning-fork tests to recognize these abnormalities were also employed then although their precise pathophysiological bases were not (and still are not) completely understood. Because of the nature of these abnormalities they may also constitute symptoms for which a person, especially one who is musical, may seek medical attention.

A classification of these frequency coding paracusis is shown in Table 6.1. Unlike other auditory tests, and the phenomena underlying them, those relating to frequency coding have no particular eponyms attached to them.

Paracusis sclerotica. Itard (1821) was perhaps the first to recognize the phenomenon of sound distortion which characterizes certain auditory disorders. He wrote:

'... j'ai pu observer chez un acteur qui vint me consulter ... Toutes les fois qu'il voulait chanter dans le haut, les sons de sa voix produisaient sur son oreille une sensation confuse qui le faisait continuellement détonner. Les mêmes sons tirés d'un instrument à vent ou à corde produisaient sur le même effect.'

'I was able to observe an actor who consulted me ... Every time he wished to sing in the upper register, the sound of his voice produced a confused sensation in his ear which made him continually out of tune. The same sounds produced either by a wind instrument or a string instrument had the same effect on him.'

This phenomenon of sound distortion is detected by presenting a vibration tuning fork first to one ear and then to the other ear of the patient. The subject is asked if the musical note sounds the same on the right side as on the left side. If this form of paracusis is present the patient will say that the sound is distorted, harder (hence the term 'paracusis sclerotica'), harsher, rougher or out of tune on one or other side. Daae's (1894) first case, a 32-year-old man who has sustained a direct injury of the left internal ear, perceived tones as a scratching of metallic substances against one another. In this case, as in many other cases, the phenomenon was associated with diplacusis.

Clinically, the phenomenon is characteristic of cochlear disorders. Even the cochlear hair cells of Pseudemys scripta elegans (a turtle) are sharply tuned (Fettiplace and Crawford,
1980) so that paracusis sclerotica, along with other frequency coding abnormalities, probably arises from hair cell dysfunctions.

*Paracusis duplicata.* This was the term used by Sauvages to describe a phenomenon which is now generally known as diplacusis (Itard, 1821). The first of Sauvages' cases

'est celui d'un donneur de cor ... Lorsqu'il donnait de son instrument, il entendait le son qu'il voulait en tirer; plus un autre son du même rythme, quoique tout différent, ce qui lui rendait l'ouie double. Ce n'était pas un écho, puisque les deux sons se faisaient entendre simultanément; ce n'était pas non plus deux consonnats, car ils eussent été agréables.'

'... is that of a horn-player ... When he blew his instrument, he heard the sound which he intended to produce, together with another sound of the same rhythm, which, being completely different, made it sound double. It was not an echo since the two sounds were heard simultaneously; they were not two consonant sounds either, because this would have been pleasant.'

Thus Sauvages clearly characterizes this case as one of diplacusis dysharmonica, distinguishing it from both diplacusis echotica and diplacusis harmonica which came to be recognized by Gruber (1888) and by Gradenigo (1892), respectively. However, from the description, it is not clear whether either of Sauvages' cases had binaural or monaural diplacusis. This distinction was not clarified until Gradenigo's paper in 1892. Nevertheless, from the description of Itard's fourth case it would appear that that was one of binaural diplacusis. Itard wrote

'*... car, en bouchant alternativement l'une et l'autre oreille, elle entend séparément, ou le son naturel, ou le son aigu.'*

'*... because, on blocking-up one ear or the other, she hears separately either the normal sound or the high-pitched sound.'*

*Monaural diplacusis.* In harmonic monaural diplacusis the affected ear perceives not only the fundamental tone but, particularly when the fundamental is not intense, also one of the harmonics, so that in one ear there is a double and synchronous perception of sound (Gradenigo, 1892). In reporting the phenomenon in his first case, that is, that of a 29-year-old violinist and composer 'with catarrhal otitis media' but 'no symptoms pointing to disease of the internal ear', Gradenigo emphasized its dependence upon intensity. He said that when a fork of middle or higher register is held before the ear, the tone is heard single so long as its intensity is great; but as the intensity diminishes a second tone is clearly heard which is usually harmonic and represents a higher or lower major or minor third or quarter. The interval varied with different fundamentals but was constant for the same tone.

Gradenigo's second case was a 28-year-old woman with bilateral 'catarrhal otitis media' and involvement of the internal ear. The forks 'c3, c4 and c5' were heard double in the last 10 seconds before becoming inaudible. The interval between the true tone and the pseudotone was harmonic.
Steinbrugge's (1882) case must also have been one of monaural diplacusis since the hearing was said to be completely lost on one side. Moreover, it was of the harmonic type since the patient observed that 'along with each tone he heard also the major third'.

Shambaugh (1940) reported a 25-year-old male with otosclerosis involving both the stapes and the cochlea, who exhibited what he termed 'diplacusis monauralis dysharmonica'. The pitch from a 1024-Hz tuning fork appeared to be double on one side.

Gradenigo pointed out that the phenomenon can be elicited by bone-conducting testing. He considered that the site of this distortion was in the cochlea. The precise mechanism is, however, uncertain. Nevertheless, even in normal subjects, as Wegel and Lane (1924) first pointed out, a single pure tone generates harmonics because of the non-linear characteristics of the cochlea. Cochlear mechanical non-linearities exist for both moderately high (Tonndorf, 1958; Rhode and Roubles, 1974) and very low (Anderson and Kemp, 1979; Kemp, 1979) sound stimulus levels. Despite his inability to find other evidence for internal ear dysfunction in his first case, it may well be that Gradenigo's phenomenon has its basis in normal low level non-linearities.

Indeed, Flottorp (1953) has speculated that monaural diplacusis is related to the idiophonic effect (pure-tone tinnitus evoked by acoustic stimulation). This in turn shows remarkable similarities to Kemp's stimulated acoustic emissions from the cochlea. More recent biochemical studies by Macartney, Comis and Pickles (1980) have demonstrated the presence of myosin in the stereocilia of cochlear hair cells. It has therefore been suggested that these cochlear acoustic emissions may be the result of actin-myosin interactions as in muscle cells. As with binaural diplacusis, the validity of differentiating between harmonic and dysharmonic monaural diplacusis may be questioned. Interpretations based upon the idiophonic effect would indicate that the presence of absence of consonance would depend, among other things, on the frequencies of the stimulus (test) tone and of the idiotone (Flottorp, 1953), and how many of the latter existed in a particular individual.

Binaural diplacusis. Although, as pointed out previously, Gradenigo (1892) and others have differentiated between harmonic and dysharmonic diplacusis, the value of this distinction is questionable. Daae (1894) pointed out that, in a given patient, discordant double hearing may be present on one part of the musical scale, and harmonic may be present in another part. Moreover, during the course of the disorder, the interval between the tone and the pseudotone may be changed. Indeed, a gradual decrease in the interval may herald recovery. As such, a test of binaural diplacusis may be of prognostic as well as of diagnostic value. Williams (1952) says that it is the first sign to disappear in Ménière's disorder in response to successful treatment.

The diagnostic value of the binaural diplacusis test in Ménière's disorder has been emphasized by Shambaugh. In 1935, he reported three cases of Ménière's disorder in which the patient reported that a tuning fork of a given frequency was perceived as being of a different pitch in the two ears when heard by air conduction. In two of the cases, low frequency tuning forks were heard at a higher pitch on the affected side. Subsequently, Shambaugh (1940) reported 43 more cases where binaural diplacusis was demonstrated with tuning forks. The pitch was invariably perceived to be higher on the affected side.
Studies by Jones and Pracy (1971) showed that there is not normally a difference of more than 4% for a pair of tones that are matched to give the same pitch perception to the two ears. In Ménière's disorder, the difference may amount to 37% (maximum at 250 Hz) and, in high-frequency hearing losses, 17% (maximum at 4000 Hz). These authors also confirmed the upward shift in perceived pitch on the affected side.

Binaural diplacusis occurs in a number of cochlear disorders other than endolymphatic hydrops. Moos (1866) reported a patient who inhaled chloroform for an asthmatic attack. This produced tinnitus, impaired hearing, and 'all the notes of the scale from A appeared doubled'. Diplacusis was observed in another asthmatic who took 35 mmol potassium iodide daily for 6 weeks (Moos, 1882). Spalding (1881) himself sustained binaural diplacusis following exposure to factory noise. In the affected ear the sounds from a flute were observed to be a 'minor third higher'. Subsequently, experimental studies by Elliott, Sheposh and Frazier (1964) on noise-induced temporary threshold shift demonstrated an upward shift in pitch for the frequencies 2.8 kHz and 5 kHz after fatiguing at 2 kHz and 4 kHz, respectively.

Rossberg (1954) reported observing binaural diplacusis in two fenestrated otosclerotics. The tones in the operated ear were of a higher pitch. Bracewell (1966) observed diplacusis as a temporary phenomenon following stapedectomy. At a level of 20 dB sensation level, all subjects with diplacusis matched a 500-Hz tone in the operated ear to a tone of 540 ± 10 Hz in the unoperated ear. The phenomenon was most noticeable 10-14 days following operation, negligible after one month, and had disappeared by 6 weeks. It is of note that the most severely affected case was a patient who developed acute vertigo on the fourth postoperative day. This poststapedectomy diplacusis probably indicates a temporary endolymphatic hydrops which itself reflects a serous labyrinthitis.

The experiments of Brandt (1967) have a bearing on the clinical demonstration of binaural diplacusis even when there appears to be little or no hearing loss. In studies on noise-induced temporary threshold shift, Brandt demonstrated impaired binaural pitch matching even at frequencies which did not show a threshold shift.

The clinician must test for the presence of diplacusis since, as Knapp (1869) pointed out, few patients recognize its presence.

Albers (1965) has reported that binaural diplacusis can be measured conveniently by an adaptation of Békésy audiometry, that is by diplacusimetry.

Echoic diplacusis. On reading the description of his first case of diplacusis, it would appear that Sauvages was mindful of the possible occurrence of diplacusis echotica. However, it will never be known whether or not he had observed such a phenomenon. Nevertheless, the occurrence of diplacusis echotica is clearly referred to by Gruber (1888). The condition was also recognized by Treitel (1891).

In echoic diplacusis, the true tone and the false tone (pseudotone) are separated by an interval of time. The phenomenon is not infrequently associated with binaural diplacusis (Gradenigo, 1892).
Shambaugh (1940) reported a 42-year-old male with acute suppurative otitis media and serous labyrinthitis who perceived the sound from a tuning fork as a distinct echo in one ear.

Flottorp (1953) has suggested that some cases of displacusis echotica, like diplacusis monauralis, may be manifestations of the idiophonic effect. There are indeed broad similarities to Kemp's stimulated acoustic emissions from the cochlea.

Isoacusis (Gk 'equal hearing'). This is a condition in which all frequencies in a particular frequency band are perceived as being of the same pitch. The condition is sought clinically by using a set of tuning forks of different frequencies. Daae (1894) reported the case of an adult male who was afflicted with progressive hearing loss and tinnitus. Examination showed that tones ranging from 60 to 128 Hz were perceived correctly and with equal intensity in both ears. On the left side, however, tones between 128 and 2048 Hz were perceived as one and the same tone, that is $f'$. Above 2048 Hz, the tones were heard alike and correctly on the two sides.

Bunch (1942) reported a patient in which all frequencies above 512 Hz 'sounded exactly alike'. Bunch considered the condition to be a special case of binaural diplacusis. The condition could of course occur not only bilaterally but also to a similar degree on the two sides so that no binaural diplacusis might be demonstrable.

The condition is associated with cochlear dysfunction. The pathophysiological basis could be the loss of 'tips' of the tuning curves of cochlear neural elements (Evans, 1975) with degradation to a very broad tuning.

Altered pitch-intensity function

Pitch is primarily a function of the frequency of a tone (Galilei, 1638). However, it is secondarily a function of the intensity of the tone. This phenomenon was recognized by Urbantschitsch (1881) long before Zurmühl (1930), followed by Stevens and Newman (1935) and by Morgan and Garner (1947), put it on an experimental basis. Urbantschitsch observed that, even in normally hearing subjects, a given tone seemed lower in pitch as it became more intense and higher as it grew weaker.

Gradenigo (1892) reported that his first patient with monaural diplacusis showed an accentuation of this pitch-intensity relationship. The monaural diplacusis affected both ears, the left more than the right, where the hearing was almost normal. However, the altered pitch-intensity relationship affected the right ear only. 'The tone of $g^2$ which was heard normally for 45 seconds by the right ear seemed more than a half-tone higher as its intensity diminished.'

Intensity coding abnormalities

Intensity coding abnormalities in cochlear disorders are classically held to show themselves in the phenomenon that was first described by Pohlman and Kranz (1924). The phenomenon is generally termed 'loudness recruitment' (Fowler, 1965). However, de Bruine-Altes (1946) referred to it as regression, since the term described what happens to the hearing loss which regresses, that is decreases, with increase in loudness. Moreover, to the
neurophysiologist, the word 'recruitment' has a specific neurological connotation. The phenomenon may be defined as a change in the growth of loudness of a tone as a function of sensation level in which the growth is more rapid than normal. Thus notwithstanding that a patient may have a unilateral hearing loss of say 30 dB at a given frequency, at 80 dB hearing level the tone may appear equally loud in the two ears.

**Chandler's test.** Chandler (1958) described his test for loudness recruitment as follows. The tuning fork is struck to produce threshold, or near threshold, sound in the ear under test. The two ears are then compared by presenting the fork to the other (better) ear. The sound is heard much louder in the better. The fork is then maximally activated and immediately presented to each ear in turn. The patient is asked: 'In which ear is the sound louder now?'. If the patient answers that the loudness is the same, or similar, in the two ears, then loudness recruitment has been demonstrated.

Paradoxically, audiometric methods for examining loudness recruitment were reported at an earlier stage than the tuning-fork method. A manual audiometric procedure for demonstrating the phenomenon was presented by Fowler in 1936; this was termed the 'alternate binaural loudness balance test'. Self-recording (Békésy type) audiometric methods for measuring loudness recruitment were reported by Miskolczy-Fodor (1964). The phenomenon can also be demonstrated using acoustically evoked potentials (Knight and Beagley, 1969; Portmann, Aran and Labourge, 1973).

Dix, Hallpike and Hood (1948) demonstrated that the phenomenon of loudness recruitment was characteristic of cochlear, as opposed to neural, auditory dysfunction. In this connection, one must distinguish between the 'site of the auditory defect' and the 'site of the causative lesion'. Failure to do this has brought some hearing tests into disrepute in audiological diagnosis, for example failure to recognize that a cochlear auditory defect may be associated with a tumour of, or pressing on, the vestibulocochlear nerve.

A current interpretation of loudness recruitment is that it is due to loss of the 'tips' of the tuning curves of the neural elements (Evans, 1975). However, such a mechanism could not account for the recruitment of the conductive element which is sometimes seen in early otosclerosis. A simpler, and more likely, explanation to account for many recruitment results is the one based upon the 'summation' principle (Simmons and Dixon, 1966). As the intensity of a low-frequency tone increases, longer and longer segments of the basilar membranes are excited, with extension towards the basal turn. Thus, loudness recruitment is characteristically associated with low-frequency hearing losses, for example endolymphatic hydrops, or early otosclerosis with a 'stiffness tilt' of the audiogram. It may not be irrelevant that early (reversible) hydrops also has an essentially mechanical basis.

At specific locations on the basilar membrane, and for lower levels of stimulation, abnormal loudness growth may be due to increased firing rate of neurons which are tuned closely around the stimulus frequency (Coles and Johnstone, 1974).

As well as being able to demonstrate loudness recruitment, Chandler's test might also be used to demonstrate loudness derecruitment (Fowler, 1965; Davis and Goodman, 1966). This is the converse of loudness recruitment, that is it is the condition of an abnormally slow growth in loudness. The phenomenon has also been referred to as loudness reversal (Dix,
and loudness decrement (Simmons and Dixon, 1966). In contrast to loudness recruitment, loudness decrement is characteristic of vestibulocochlear nerve lesions, particularly those due to tumours, or demyelinating disorders.

Uncomfortable loudness levels. Chandler (1958) mentioned the common observation that a maximally activated tuning fork produces obvious discomfort when it is presented to an ear with a cochlear disorder.

In normal ears when a 1000-Hz tone reaches a level of about 100 dB sound pressure level it becomes uncomfortably loud. Such levels are within the range of tuning forks since, when struck forcibly against the heel of the shoe without regard to generating distortion produces, 110 dB sound pressure level may be reached (Chandler, 1958). An audiometric threshold of uncomfortable loudness was first used systematically by Watson (1944). Subsequently, Bangs and Mullins (1953) recommended the audiometric measure as an index of the recruitment phenomenon. As such, Hood and Poole (1966) emphasized the value of the measurement in distinguishing Ménière's disorder from vestibulocochlear nerve disorders. Patients with cochlear disorders show similar uncomfortable thresholds (measured in sound pressure level) to those of normal subjects. The measurement has been variously referred to as the uncomfortable loudness level or the loudness discomfort level. Clearly, however, there are a number of such levels. The loudness discomfort level abbreviation of LDL is one used by physicians for low-density lipoproteins; it would, therefore, be more appropriate to refer to this measurement as the threshold of uncomfortable loudness.

Audiometric studies have shown that there is considerable variation in the threshold of uncomfortable loudness (Stephens, Blevgad and Krogh, 1977). It depends on the noise experience of the individual (Niemeyer, 1971) and, unlike the most comfortable loudness level, it is influenced by psychological measures (Stephens and Anderson, 1971; Fuller, H. and Stephens, S. D. G., 1981, unpublished data).

Corradi's test. Stephens (1947) recounted how Lord Rayleigh (J. W. Strutt) demonstrated the phenomenon of auditory adaptation at threshold to Helmholtz in 1881. Auditory adaptation may readily be demonstrated at high frequencies even in normally hearing individuals. Corradi (1890) was the first to describe the phenomenon with bone-conducted sound from a tuning fork.

Later manual audiometric studies by Schubert (1944) in Germany and by Carhart (1957) in the USA and Békésy type studies by Reger and Kos (1952) and by Jerger (1960) in the USA and by McLay (1959) in the UK showed that marked auditory adaptation at threshold is characteristic of neuronal lesions.

Abnormal auditory adaptation may be demonstrated by a tuning fork as follows. The audibility of an ear to air-conducted sound is tested in the usual way with the tuning fork. The patient is asked to raise a hand as long as a musical note is heard. As soon as it stops the patient is told to lower the hand immediately. Should a note be heard again then the hand must be raised immediately, and so on. As soon as the patient indicates that he/she no longer hears the sound, the tuning fork is brought briskly away from the ear to a distance of about half a metre and then quickly back again to its original position with the prongs of the fork near to the meatus. The sequence is repeated until such time as the patient indicates that
he/she can no longer hear the fork. At the same time that the patient first indicated that the fork could no longer be heard, a stop-watch was started. As soon as the patient indicates that the fork can no longer be heard when the tuning fork has been restored to its testing position, the watch is stopped. A quantitative measure of auditory adaptation is thus available.

**Monochord test**

With recent emphasis on the importance of high frequency hearing threshold levels in audiological diagnosis (Dieroff, 1976; Osterhammel, 1979), it is possible that clinicians may now retrieve from museums the monochord and the whistle. These are the two clinical instruments which were formerly used to measure the upper frequency limit of hearing.

Struycken's (1913) monochord consists of a steel string under tension. In contrast to the tuning fork and musical string instruments which are activated to generate transverse vibrations, the monochord is activated to generate longitudinal vibrations. The frequency of the sound generated is controlled by varying the tension and/or the length of the wire. The monochord can be used to measure the hearing by both air and bone conduction.

Struycken reported that, using the monochord, the upper limit of hearing for the age range 10-19 years was in the range of 16.5-21 kHz when measured by air conduction, and between 17.8 and 26 kHz when measured by bone conduction. These values fell with age until, for the age range 60-90 years, the minimal and maximal air conduction limits were 10.6 kHz and 14.8 kHz, while the upper bone-conduction levels were in the range 11.8-14.2 kHz.

Larsen (1939) described how he used Struycken's monochord:

'It was rubbed with cotton wool, which was moistened with a liquid containing equal quantities of benzol and terpentine. I noticed that it is of importance to use a new piece of cotton before each test, and to moisten the cotton fairly with the liquid. If the cotton is too lumpy and not moist enough, the tone becomes less intense, and this may give rise to erroneous determinations. If there was reason to suppose that the hearing for monochord was different on the person's two ears, he was requested to close the ear opposite to the one being tested with his fingers. The determination of the upper limit was performed with an accuracy of 1000 vibrations. The determination was made partly by air conduction and partly by bone conduction. The difficulty of monochord determinations is that the test person should be able to state whether it "pipes" or whether it "frictionizes" only. That is why trial determinations were made of values which are essentially below the limit.'

Equipment is now available for determining high-frequency hearing threshold levels in the range 8-20 kHz and data have been reported on normal thresholds in respect of sex and age (Northern et al, 1971; Osterhammel and Osterhammel, 1979). The means are, therefore, now at hand for assessing the validity and sensitivity of the monochord and other measures of high-frequency hearing sensitivity.

**Whistle tests**

The Galton whistle is essentially a closed organ-pipe fitted with an obturator which enables the length of the contained air column to be varied. This manoeuvre changes the
resonant frequency. A sound (whistle) is generated by propelling air through the tube by means of an attached rubber bulb. As Feldman (1960) points out, the Galton whistle was introduced into medicine by Burckhardt-Merin (1885). Using the Galton whistle, Alderton (1896) reported on the upper tone limit in normal and abnormal ears. Sonnenschein (1933) considered the monochord the instrument of choice in clinical determinations of the upper frequency limits of hearing.

**Lever pocket-watch tests**

Prior to the introduction of the quartz watch, a lever pocket watch was used by patients and doctors alike to test the hearing. For some years after the Second World War, ex-servicemen in the USA were compensated on the basis of watch and whisper tests of hearing (Suter and von Gierke, 1976). In his textbook *The Neurological Examination*, De Jong (1967) says, ‘but for more critical evaluation a watch is used’.

Of equal importance to Tarchanow's fusion property of binaural hearing is the property of sound separation. This property was reported by Weber in 1848. Weber observed that, when two watches were placed on either side of the head of an observer, each could be heard separately and could be referred to its correct side of the head. A watch, like finger friction, can be used to test sound localization.

The ability to hear a lever watch is particularly sensitive to high-frequency hearing losses. The author was recently consulted by a 38-year-old woman who complained that she was unable to hear a watch ticking in one ear. Pure-tone thresholds of hearing in that ear were normal up to 4 kHz. Had the auditory examination covered only that range of hearing an erroneous diagnosis of cophophobia (fear of losing hearing) might have been made. Sweep frequency Békésy-type audiometry showed that threshold sensitivity of the ear fell off above 4 kHz.

Miyazaki (1975) used a watch in an industrial screening test. Eight per cent of 4393 adult male workers in a metal industry in Japan were found to have a hearing loss. The loss was confirmed by audiometry in 96% of cases.

Not only can the watch be used to obtain a quantitative measure of hearing loss (Knapp, 1898; Keen, 1929), but it can also be used to distinguish between a conductive and a sensorineural hearing loss. Stephens (S. D. G., 1979, personal communication) points out that Astley Cooper's (1801) test may prove invaluable if one is caught off-balance by an unexpected domiciliary visit. In referring to the auditory status of a young male patient, Astley Cooper wrote:

‘The auditory nerves, however, were perfect; for he could distinctly hear the beating of a watch if placed between the teeth or against the side of the head; and he never had perceived any buzzing in the ears.’

In 1827, Tortual described the occlusion phenomenon using a pocket watch. He had performed the test with the watch in his mouth.
A watch test for feigned unilateral hearing loss was described by Erhard (1872) and is similar to the loud voice test that bears his name. Erhard observed that a chiming pocket watch could be heard at a distance of 3 metres even when the meatus was occluded.

**Reflexes**

In suspected feigned bilateral deafness, the clinician must have recourse to physiological tests. The cochleopalpebral reflex (blinking in response to a sudden loud sound) was known to Müller (1838) and used by Gault (1916) in the First World War to identify feigned bilateral deafness. The cochleopupillary reflex (transitory pupillary contraction followed by more sustained dilatation in response to loud sounds) was described by Holmgren (1876) and has been used by Unger (1939) and others.

The cochleostapedial (acoustic stapedius) reflex, which cannot be detected clinically, can be detected by acoustic impedance technique (see Chapter 7). This technique was introduced as a test for feigned hearing loss by Jepsen in 1953.

**Clinical tests of vestibular and related functions**

**Gait**

The examination of gait begins with the observation of unstressed, natural walking. If this is normal, the examiner will then proceed to use various tests which are designed to show up balance disorders when walking.

As Hodkinson (1980) said, gait abnormalities due to deformities usually present no diagnostic difficulties. Abnormal posture may call attention to spinal deformities, that is kyphosis and scoliosis. A dipping gain may be due to shortening of a leg due to an old fracture or severe osteoarthritis of the hip. Bizarre gaits, where one grossly externally rotated leg leads and the other follows behind it, may result from bilateral osteoarthritis of the hips. An unsteady wobbling gait, where the affected knee is stabilized in a knock-kneed apposition to its healthy fellow, will result from the lateral instability of a grossly disorganized knee incurred either by advanced osteoarthritis or, less frequently now, a Charcot joint.

A gait which Hodkinson likens to walking like a pair of protractors, that is progression by small limited rocking movements, characterizes hip stiffness due to severe osteoarthritis.

The anamnesis will have indicated if an intermittent limp is due to pain or intermittent claudication.

Hobbling gaits are the consequence of painful feet due to corns, bunions or just ill-fitting shoes. As Jahss (1971) pointed out, of the entire musculoskeletal system, the foot is perhaps the foremost in showing the ravages of time, abetted by the static stresses imposed by weight-bearing and by shoes. Long-standing deformities whether congenital or acquired are often at first flexible and asymptomatic but gradually become fixed and rigid. Consequently, painful exostoses and callouses ultimately develop.
Limping may be due to bone pain from Paget's disease, metastases or an undiagnosed fracture.

Marked lurching and staggering without the patients falling and hurting themselves, and especially when the patients are knowingly being observed, points to a psychological cause.

More sensitive tests are indicated if the subject appears to walk normally. Fregly and Graybiel (1968) developed a battery of such tests. This battery is termed the 'floor ataxia test battery'. The battery incorporates a number of tests - and all with assigned acronyms. Perhaps the most useful for clinical purposes is WOFEC (walk on floor eyes closed). The task requires a subject to walk as straight as possible with 10 steps heel-to-toe behind the first two starting steps. The results for this heel-to-toe test with the eyes closed should be compared with the results for the heel-to-toe test with the eyes open.

Drachman and Hart (1972) reported a useful test for patients with dizziness due to multiple sensory defects. They said that the dizziness in patients with this syndrome is most closely reproduced by walking, or by walking and turning quickly. Touching the examiner's finger lightly provides sufficient additional sensory information to relieve symptoms markedly in most patients.

**Stance**

A psychological cause should be suspected where the patient cannot stand, let alone walk, and where there appears to be no neurological or other cause for the condition (abasia-astasia). The terms 'abasia' and 'astasia' have tended to be restricted to inabilities to walk or stand, respectively, where no neurological or other organic defect can be demonstrated. However, it is more convenient, and etymologically correct, to use these terms in their literal interpretation.

If the patient is able to stand with the eyes open the Romberg (1846) test should be performed. As Rogers (1980) has pointed out, the nature and purpose of this simple test is frequently misunderstood. A subject's ability to stand erect with the eyes closed is compared with the ability to do so with the eyes open. If, on closing the eyes, the subject immediately becomes unstable and falls to the ground, then the test is positive. Such a result indicates a spinal cord posterior column lesion, for example tabes dorsalis.

As with all tests, there is the need, as specifically pointed out by Edwards (1973) for this test, to properly explain the test and gain the patient's confidence. When this is done, a normal subject may sway slightly with the eyes open and somewhat more with the eyes closed. Conversely, if it is suggested to the patient that he/she may well fall, then he/she will frequently do so. Indeed, this forms the basis of Hull's (1933) body sway test for suggestibility. Thus the Romberg test can be linked to other balancing tests for purposes other than diagnosing a posterior column lesion. In particular, quantitative measures of body sway, when the subject is standing with the feet set apart, provide an index of postural stability. In addition, the subject's ability to stand with the feet in tandem (heel-to-toe) would be influenced, among other things, by the integrity of the vestibular mechanism (Fregly and Graybiel, 1968).
**Limb posture test**

There are a number of versions of a test, variously attributed to Bárány, Güssich, Hautant and Quix, in which the spontaneous deviation of the outstretched upper limb (or limbs) is observed. Depending on the particular version employed, the test is conducted with the subject lying in bed, sitting, standing with the feet apart or standing with the feet together. The subject is asked to extend the arms to the front of the hands closed but the index fingers also extended so that they nearly touch those of the examiner. The subject is then asked to close the eyes and the deviation of the limb(s) is observed. Quix's (1925) version, where the subject stands with the feet apart, has been studied extensively by Hart (1980) on 100 neuro-otological patients. Hart observed that the arms deviated to the left in 39 out of 47 left-sided peripheral balance disorders, and to the right in seven out of 17 right-sided peripheral balance disorders. No arm deviation was observed in eight left peripheral lesions and six right peripheral lesions. In eight out of 10 left-sided central lesions, the arms deviated to the left, but in three right-sided central lesions, the arms also deviated to the left. Hart's results would thus suggest that, with this method of testing, arm deviation to the right would be indicative of a right peripheral lesion. Arm deviation to the left would indicate that, with a peripheral lesion, a left-sided affection is most likely and, with central lesions, a left-sided affection is more likely than a right one. The emergence and pattern of the sinistrality phenomenon in Hart's study is of considerable interest. One wonders whether or not these intriguing results would be substantiated by further studies.

In a group of 33 cases of unilateral acute cerebellar injuries, Holmes (1917) observed that, in all except one case, the homolateral arm swung out following eye closure and came to rest slowly. The exception was a case where the limb was so asthenic that it could not be raised. In slight injuries where deviation was not observed, it could be brought out by shaking or tapping the affected limb. In a few patients who suffered from extensive unilateral lesions, the contralateral arm tended to deviate inwards, but this deviation was always slight and neither constant nor regular. Holmes found that the tendency to deviation of the lower limbs was less common, but, when this occurred, the homolateral leg swung inwards. Thus not only the direction, but also the pattern of spontaneous deviation of the limbs, can be of diagnostic value.

These spontaneous extended arm deviation tests can be combined with Bárány's past-pointing test. If there is no deviation of the outstretched hands with the eyes closed, the patient is then asked to bring the hands slowly down to the sides and then back to the original position. In some cases, the test is positive when there has been no spontaneous deviation of the upper limb. Normally, a subject can regain the target relatively well on each attempt. When the test is positive, the patient's index finger deviates constantly to one side; if the test is repeated, the deviation initially increases with each attempt. The test can be quantified and, at the same time, the subject 'reassured', by the use of a tape-measure, which the examiner holds horizontally in front of the patient. By doing this, the error of each movement can be measured and, by allowing the patient's finger to touch the tape-measure, he/she is unable to ascertain whether or not there has been an error. This test will also disclose any dysmetria, that is overshooting, which is indicative of cerebellar dysfunction.

In association with these arm deviation tests, it is convenient to perform the 'finger-to-nose' test and look for tremor.
Fukuda's (1959) vertical writing test is another way of detecting an imbalance affecting the upper limbs. The square drawing test of Sekitani and his colleagues (1975) is perhaps a more convenient development of this test. With these tests, the examiner must not allow any part of the patient's upper limb to come in contact with the writing surface, otherwise the results are vitiated.

The stepping tests of Unterberger (1938, 1962) and others form a group of tests which are intermediate between those for testing stance and those for testing gait. They are, however, an extension of the deviation test since this is what they are designed to measure. As performed by Scherzer (1968) and others, the test is performed by asking the subject to mark time on the spot (the examiner demonstrates) with the arms outstretched in front and the hands clasped together. The subject is asked to close the eyes. The direction and degree of rotation is then noted. In addition to measuring the angular rotation in degrees in the stepping test, Peitersen (1967) measures the linear displacement of the patient from the starting position and conducts the test in a quiet, darkened room with the patient blindfolded and with arms hanging by the sides.

Erroneous previous criticisms of the invalidity of these deviation or turning tests regarding lateralizing vestibular lesions were partly because of the failure to recognize the existence of recovery phenomena (central compensation). In this state it is possible to have a nystagmus, termed a 'recovery nystagmus' (Erholungsnystagmus), which is directed towards the affected vestibular labyrinth or nerve (Stenger, 1959). Thus, in assessing the validity of these limb deviation and stepping tests, the results should be compared not with the side suspected to be involved, but with the direction of any spontaneous or provocation nystagmus.

**Eye movements**

As Uemura and his colleagues (1977) pointed out, before searching for nystagmus, the eye movements themselves should be examined for conjugate movements, limitation of gaze and convergence. But before these examinations are made, the resting position of the eyes should be observed. Observations should then be made of each eye during both conjugate gaze and uniocular gaze, that is then the other eye is covered.

Yap, Loong and Nei (1975) found that 44% of stroke patients seen within 48 hours of admission showed an ocular motor abnormality. The mortality rate of such patients appeared to be double that of patients without ocular motor abnormalities.

Conjugate deviation of the eyes towards the side of a unilateral hemisphere lesion may be observed in stuporose patients. Unlike other motor phenomena, this defect disappears within a few hours of the patient regaining consciousness (Cogan, 1970). In acute unilateral cerebellar lesions, the eyes at rest are initially deviated away from the side of the lesion. Although this deviation is also more marked in unconscious patients, it may be observed in conscious patients (Holmes, 1917).

Squint (strabismus) is the pathological condition of chronically misaligned visual axes. This is frequently genetically determined and is rare in coloured races. Minor degrees of squint may be recognized clinically from the asymmetrical positions of the bright corneal light reflections relative to their respective pupil margins. There are two principal types of squint,
that is concomitant and paralytic squints. Concomitant squints are so named because the eyes retain their relative positions in all directions. In paralytic squints, the difference in eye positions is greatest when gaze is in the direction of the normal action of the paralysed muscle. Concomitant squints are due to a disorder of the sensory component of the reflex arc or its central connection; paralytic squints are due to damage to the motor component of the arc. Persistent squints can result in an amblyopia ('lazy eye'). Experimental studies on monkeys have shown this to be due to shrinkage of cells in the lateral geniculate nuclei (von Noorden, 1974).

Robinson (1975a) has presented a static eye position model. The model offers, for the first time, a quantitative estimate of the multitude of ways in which muscles can interact and interfere with each other when they hold the globe. The results are quite interesting. Because muscles have different lengths and sizes, their innervational participation in a movement can appear to be quite different from their mechanical participation. From an innervational standpoint, the vertical recti and obliques participate equally in vertical gaze. Muscles interfere with each other a good deal and necessitate changes of innervation to counteract these cross-couplings.

Skew deviation of the eyes is characterized by a deviation of gaze of one eye above the other, the angle of which may or may not be fixed for all directions of gaze. It is due to a lesion other than one involving the extraocular muscles, their motor neurons or local mechanical factors in the orbit. The eye on the side of the lesion is usually hypotropic, that is the lower one. Holmes (1917) observed skew deviation in five cases of unilateral gunshot wounds of the cerebellum. The homolateral eye was directed downwards and inwards while the other looked upwards and outwards. This lack of parallelism in the optic axes disappeared, however, on fixation when this could be obtained; consequently diplopia did not result. Skew deviation was observed only during the first week or so after an injury of the cerebellum except in two cases in which rapid destruction and compression of the structure occurred owing to abscess formation. Skew deviation is thus seen in patients with acute asymmetrical cerebellar disease and represents a dysfunction of the vertical vergence mechanism. Although rare in demyelinating disease, it has been reported in cerebellar and pontine artery thromboses, in platybasias and in vestibulocochlear schwannomas ('acoustic neuroma'). It has also been produced experimentally in animals with lesions of the vestibular nuclei and their connections with the oculomotor nuclei (Oloff and Korbsch, 1926). Intermittent skew deviation is associated with intermittent vertebrobasilar insufficiency (Walsh and Hoyt, 1969).

Having considered eye deviations at rest, squint and skew deviations, the examiner can then consider abnormal eye movements in general.

After muscular and neuromuscular causes of ocular motor nerve pareses have been excluded, it will be found that vascular disease is the commonest assignable cause of such pareses (Rucker, 1958). In considering individual ocular motor nerves, a neoplasm will be the commonest assignable cause of abducent palsies, an aneurysm of oculomotor, and vascular disease of trochlear palsies. An isolated trochlear palsy is unlikely to be due to a neoplasm. In the case of abducent palsies due to a neoplasm, the most likely lesion would be a primary other than a meningioma or one involving the hypophysis (pituitary). In the case of oculomotor palsies due to a neoplasm, the most likely lesion would be a metastasis or a hypophyseal tumour.
Of particular interest to the neuro-otologist is an internuclear ophthalmoplegia. This condition is characterized by adductor palsy on attempted conjugated lateral gaze. In the primary position of gaze, the eyes are usually directed straight ahead. There is usually an associated, predominantly uniocular, nystagmus of the abducted eye. The condition is due to a lesion of the medial longitudinal bundle ipsilateral to the adductor palsy. With lesions of the posterior part of the bundle (medullary part), convergence is unimpaired; with anterior bundle lesions (midbrain part), convergence is impaired (Cogan, 1970). Bilateral internuclear ophthalmoplegias, which are almost pathognomonic of disseminated sclerosis, are more common in younger patients; unilateral internuclear ophthalmoplegias are more common in older patients.

In pseudo-ophthalmoplegia (supranuclear palsy), command eye movements are absent but eye position deviation occurs with head movements (oculocephalic manoeuvre - see later) or with vestibular stimulation.

Ocular dysmetria is best demonstrated by asking the subject to look from an eccentric position of gaze back to the midline position (Cogan, 1954). This movement is normally executed with remarkable precision but, in the presence of cerebellar disease, there is a characteristic overshoot (occasionally undershoot) with several pendular excursions of the eyes before final fixation is attained. Goldstein and Cogan (1961) reported that saccadic (fast eye movement) overshoot dysmetria sometimes occurred only with saccades towards the side of the cerebellar lesion.

Clinical evidence indicates that this dysmetria is due to dysfunction of the cerebellar vermis. The control system abnormality in saccadic overshoot dysmetria is an abnormally high gain in the brain's feed-forward path. Selhorst and his colleagues (1976) suggest that the cerebellum continuously modulates saccadic gain, although it may not lie directly on the visual ocular motor (feed-forward) path.

**Ocular spasms**

Ocular spasms may be classified into those of an oculomotor nerve or an extraocular muscle, those of conjugate gaze and those of convergence.

The features of muscle spasm, or overaction, are basically the converse of those of palsy. If the sound eye is used for fixation, the affected eye shoots beyond it in the direction of action, its movement being quicker and its total excursion greater than those of its fellow. As Duke-Elder and Scott (1971) pointed out, the differential diagnosis between a spasm and palsy of the contralateral synergic may be difficult but may not be necessary since the two conditions frequently coexist.

The spasm of conjugate gaze that has perhaps been most studied is that which characterizes oculogyric crises (chronic eye fits). These consist of conjugate spasmodic deviations of the eyes, usually upward. The eyes remain open and there are usually associated movements such as rhythmic contractions of levator palpebrae superioris and of orbicularis oculi, as well as head and neck movements. Although commonly a sequel of postencephalitic parkinsonism (Hohman, 1925), the phenomenon has also been reported in neurosyphilis (de Nigris, 1933; Krabbe, 1936) and following trauma (Kaslín, 1936). Ablation and electrical
stimulation studies indicate that the motor and premotor cortex are involved in the generation of these crises (Klemme, 1941). The crises can be abolished by coagulative lesions of the posterior limb (pathway of the corticospinal tract) of the internal capsule (Gillingham and Kalyanaraman, 1965).

As Guiloff, Whiteley and Kelly (1980), say, convergence spasm is not always an hysterical manifestation. They point out that this condition may be associated with organic disease affected the cerebral hemispheres, the brainstem or both. In order of probability, these organic disorders are: head injury (de Morsier and Balavoine, 1949), labyrinthine lesion (Borries, 1926), encephalitis (Margulis and Model, 1926), tumours (including epidermoid cysts, pinealomata and schwannomata) (de Morsier and Balavoine, 1948), Wernicke's encephalopathy (Thompson and Lynde, 1969) and ocular muscle imbalance (Bagshaw, 1963), as well as various other disorders.

**Uniocular oscillations**

Abnormal spontaneous uniocular oscillations encompass not only nystagmus proper but also pseudonystagmus, which is represented by superior oblique myokymia (Susac, Smith and Schatz, 1973). This condition is characterized by intermittent, small amplitude, uniocular torsional eye movement in otherwise healthy adults. The disorder reflects phasic contraction of the superior oblique muscle. Affected subjects suffer from oscillopsia. The condition usually responds to carbamazepine.

Uniocular nystagmus is a rare condition (Duane, 1905; Cogan, 1963). There is a varied pathological basis. In the elderly, the more likely cause is a medial longitudinal bundle lesion with a vascular basis. As mentioned previously, this produces a predominantly uniocular nystagmus of the abducting eye.

**Binocular pseudonystagmus**

Apart from the searching movements of the blind, binocular pseudonystagmus comprises six conditions - opsoclonus, ocular flutter, macrosaccadic oscillations, macrosquare wave jerks, ocular myoclonus and ocular bobbing.

**Opsoclonus**

As Daroff (1977) points out, opsoclonus is characterized by most bizarre and dramatic ocular oscillations. The name was first suggested by Orzechowski (1927) who referred to the phenomenon as 'chaotique'. The term 'saccadomania' (Daroff and Hoyt, 1971) is perhaps more descriptive. The eyes exhibit involuntary, rapid, unpredictable saccades which are multivectorial: horizontal, vertical, diagonal or circular. There have been clinical reports of opsoclonus in association with ocular dysmetria and ocular flutter (Ross and Zeman, 1967; Ellenberger, Campa and Netsky, 1972). Ellenberger and his colleagues therefore consider that these three ocular movement disorders are related and represent dyskinesias of the saccadic oculomotor system. The morphological basis for opsoclonus is damage to the dentate nucleus (Ross and Zeman, 1967) or its connections (Gilbert, McEntee and Glaser, 1963).
Ocular flutter

Although Daroff (1977) regards 'ocular flutter' as a wastepaper basket term to cover many diverse types of oscillation which clinically have similar appearances, Cogan (1954) restricts the term to episodes of three or four rapid pendular oscillations of the eyes in the horizontal plane which last no more than a few seconds. Affected subjects complain of momentary blurring of vision during attacks.

Ocular myoclonus

Ocular myoclonus is characterized by continuous, pendular oscillations which are synchronous with the rhythmical movements of other midline structures (soft palate, tongue, facial muscles, pharynx, larynx) which are involved in the disorder. The frequency of the eye movements is in the range 1.5-5 Hz and the direction is usually vertical. Only the coexisting movements of the other structures distinguishes ocular myoclonus from a pendular nystagmus (Tahmoush, Brooks and Keltner, 1973). The pathological basis for the myoclonus is pseudohypertrophy of the inferior olivary nucleus which follows an acute lesion of the dentate nucleus or its associated structures.

Ocular bobbing

Ocular bobbing (Fischer, 1961; Susac et al, 1970) is characterized by spontaneous abrupt, erratic downward jerks of the eyes followed by a slow return to the mid-position. Typically affected individuals also have absent spontaneous or reflex horizontal eye movements.

Fischer, and Susac and colleagues, classify ocular bobbing into binocular bobbing and monocular bobbing. The binocular bobbing is further subdivided into typical and atypical bobbing.

Ocular bobbing may occur in Wernicke's encephalopathy and be responsive to thiamine medications.

Binocular nystagmus

Typically spontaneous nystagmus is an involuntary, sustained quasi-periodic, symmetrical and conjugate eye movement with a frequency in the range 1-5 Hz, and where the excursions of the centre of the cornea are observed to be predominantly linear. As indicated previously, exceptions to this pattern occur. Other patterns include voluntary nystagmus, circumduction nystagmus, dissociated nystagmus, retraction nystagmus and periodic alternating nystagmus.

Voluntary nystagmus

Voluntary nystagmus is typically a high frequency horizontal, pendular, conjugate oscillation of the eyes initiated and maintained by willed effort (Blair, Goldberg and von Noorden, 1967). Uniocular cases may occur.
Shults and his colleagues (1977) have shown that voluntary nystagmus consists of a series of to-and-fro saccadic eye movements. To explain this pattern of eye movement, Zee and Robinson (1979) postulate that the saccadic pulse generator in normal subjects is inherently unstable. In physiological terms, they consider it conceivable that some subjects may learn to generate voluntary nystagmus by inhibiting their pause cells which would, in turn, permit their burst neurons to oscillate.

**Circumduction nystagmus**

Circumduction nystagmus is characterized by the centre of a cornea describing a circular or an elliptical path. This rare condition has been reported in multiple sclerosis and degenerative cerebellar lesions (Strubel et al, 1980). It was also said to be the pattern of nystagmus in some cases of that rapidly disappearing occupational disorder, miners' nystagmus, where it could also be of the dissociated type (Duane, 1905).

**Dissociated nystagmus**

The term 'dissociated nystagmus' is used to designate rhythmic oscillations that are different in two eyes. The range of movement may be detectably greater in one eye than the other but in the same direction; it may be heterodirectional, or it may differ in both respects. Dissociated nystagmus is a sign of a posterior cranial fossa lesion.

Cogan (1963) considered that uniocular nystagmus is a special case of dissociated nystagmus. The syndrome of uniocular nystagmus and impaired lateral conjugate deviation due to contralateral adductor palsy was termed 'ataxic nystagmus' by Harris (1944). As mentioned previously this is due to a medial longitudinal bundle lesion.

**Seesaw nystagmus**

Seesaw nystagmus is a special type of dissociated nystagmus. Characteristically this is a conjugate, pendular, torsional oscillation with a superimposed disconjugate vertical vector in which the intorting eye rises and the extorting (opposite) eye falls. Repetition of the sequence in the reverse direction provides the seesaw effect (Daroff, 1965). The most likely site of the causative lesion is in the parasellar region or suprasellar region anterior to the third ventricle. Most commonly the lesion is a tumour. Schurr (1963) described such a case in a 51-year-old woman which was due to a chromophobe adenoma of the hypophysis cerebri. Sano and his colleagues (1972) have reported abolition of this nystagmus by stereotactic destruction of the interstitial nucleus of Cajal on one side (this nucleus is a collection of cells situated in the lateral wall of the third ventricle immediately above the cranial end of the cerebral aqueduct; it also lies at the cranial end of the medial longitudinal bundle).

**Abducting eye nystagmus**

Stroud (1974) has described an abducting eye nystagmus in older people. This nystagmus is observed in lateral gaze and can either be diminished or increased by tonic deviations such as those caused by vestibular stimuli whether produced by rotatory or caloric stimulation.
**Retraction nystagmus**

Retraction nystagmus (Koerber, 1903; Salus, 1913; Elschnig, 1913) is characterized by irregular jerks of the eye backwards into the orbit when the patient attempts to look in one direction or the other.

Gay, Brodkey and Miller (1963) reported retraction nystagmus on attempting upward gaze in a 59-year-old man. The man had experienced pain in the neck of sudden onset. Although conscious, he was unable to see, speak, swallow or move his extremities. Recovery subsequently occurred. Basilar artery insufficiency was diagnosed as the cause of the disorder. Smith and his colleagues (1959) observed retraction nystagmus evoked by command upward eye movements in a 72-year-old man who had complained of intermittent diplopia and dizzy spells. Examination showed total paralysis of upward gaze (command, pursuit, passive, lid closure and optokinetic movements) and a left homonymous hemianopia. The patient was hypertensive. A right posterior cerebral artery occlusion was diagnosed.

The paralysis of upward gaze, which was observed in the last case, is termed 'Parinaud's syndrome' (Parinaud, 1883). This syndrome, together with retraction nystagmus and dissociated or total pupillary areflexia, make up the Koerber-Salus-Elschnig cerebral (Sylvian) aqueduct syndrome.

Daroff (1977) explains retraction nystagmus as due to anomalous co-firing of extraocular muscles. If medial rectus firing is greater than external rectus firing, a convergence retraction nystagmus is observed; the converse firing pattern produces a divergence retraction nystagmus.

**Periodic alternating nystagmus**

'Periodic alternating nystagmus' is the term applied to a spontaneous nystagmus that alternates in direction and does so in regularly recurring cycles. The period of these cycles is usually in the range 200-300 seconds.

Only three cases of periodic alternating nystagmus have been studied postmortem. In each case, multiple lesions of the brainstem were found (Towle and Romanul, 1970; Towle, 1971; Keane, 1974).

Halmagyi and his colleagues (1980) report that both acquired period alternating nystagmus and its associated oscillopsia, but not congenital periodic alternating nystagmus, respond to baclofen medication.

After these atypical and infrequent patterns of nystagmus have been sought, what remains is the major body of spontaneous binocular nystagmus, which may be variously classified as congenital or acquired, or occurring in the primary gaze position or being gaze-deviation elicited. In addition, one may have rebound nystagmus and, perhaps, even rebound-rebound nystagmus.

Finally, mention will have to be made of physiological nystagmus.
**Congenital nystagmus**

This type of nystagmus, which is typically asymptomatic, persists throughout life. It may thus generate some initial confusion when it is first observed in an adult who is coincidentally afflicted with a disorder of balance.

Barber and Stockwell (1980) point out that congenital nystagmus shows three features:

1. nystagmus on upward gaze is virtually always horizontal and not vertical
2. it is reduced, if not abolished, on convergence
3. it is observed only at, or very near, the primary position of gaze.

However, a vertical congenital hereditary nystagmus has been reported (Jung and Kornhuber, 1964). Characteristically, congenital nystagmus is unaffected by abolishing optic fixation (Hood, 1967).

**Fixation nystagmus**

There is considerable variation in what is meant by fixation nystagmus. Holmes (1917) defined fixation nystagmus when describing the signs of acute cerebellar injuries. This nystagmus is seen as a rule only when the patient fixates (looks at) an object.

'It is on looking towards the injured side that the nystagmus is most pronounced; then it consists of wide, slow deviations towards the middle line, or more correctly, towards the rest point, and forcible jerks of large amplitude, slow in rate (2.3-3 Hz) and fairly regular in rhythm, towards the point to which the eyes should be voluntarily directed ... Both movements are as a rule strictly horizontal.'

The rest point is the point to which the eyes, when at rest, tend to deviate. It is usually at 175-525 mrad (10-30°) to the unaffected side of the midline. In such a position, no nystagmus is observed. Associated with this nystagmus towards the affected side is a:

'nystagmus on fixing on an object to his unaffected side, but it is more rapid, finer in range and less regular. Here too, the slow deviation is towards the rest point and the movements are most commonly horizontal ... One convergence, both eyes often tend to deviate away from the side of the lesion and are brought back to their proper position by irregular jerks of small range ... Spectacles with high, convex lenses ... were placed in front of the patient's eyes and it was then found that when he moved them to order the oscillations were considerably less marked, or did not occur on deviation in certain directions.'

Nowadays, the term 'fixation nystagmus' is often used to refer to any nystagmus which disappears or changes direction or form after optic fixation has been abolished. Moreover, the nystagmus which Holmes described in cerebellar lesions is frequently referred to as gaze paretic nystagmus.
**Pendular nystagmus**

This is an oscillatory eye movement in which fast and slow phases cannot be distinguished; the waveform is sinusoidal, with a frequency of about 4 Hz. Pendular nystagmus is also a fixation nystagmus. Congenital nystagmus may or may not be pendular. Pendular nystagmus may result from central vision being lost in early life. Acquired pendular nystagmus is probably due to a lesion of the cerebellar nuclei or their connections in the brainstem (Nashold, Slaughter and Gills, 1969; Aschoff, Conrad and Kornhuber, 1974). This acquired form is manifest as oscillopsia.

**Vestibular nystagmus**

This type of nystagmus exhibits both a slow and a quick phase; the waveform is thus like a sawtooth. There is usually a rotatory as well as the linear (usually horizontal) component to this nystagmus. It is most marked when the patient looks in the direction of the quick phase, and least marked on looking in the direction of the slow phase. Indeed, the nystagmus may be visible only when the patient's gaze is directed towards the side of the quick phase. This type of vestibular nystagmus is termed 1° (first degree) nystagmus. If the nystagmus is observed while in the primary gaze position also, it is termed 2° (second degree). If the nystagmus is also evident with gaze in the direction of the slow component, it is termed 3° (third degree).

A peripheral vestibular nystagmus is enhanced by abolishing optic fixation whether by the use of Frenzel glasses or recording with the eyes closed or eyes open in darkness using electrical methods (see Chapter 9). A central type of vestibular nystagmus is diminished or abolished by removing optic fixation.

Frenzel (1925) glasses consist of a pair of +20 dioptre biconvex lenses. These not only interfere with fixation by the patient but also, by magnifying, assist observations by the observer. The provision of illumination improves conditions for observing the subject's eyes. Unfortunately the source of illumination may also provide some facility for the subject to fixate. Frenzel glasses are, therefore, not as effective as observing eye movements in complete darkness, for example by means of an infrared viewer or by electrical registration methods.

**Gaze-deviation elicited nystagmus**

In many instances, a spontaneous nystagmus may not be observed in the primary position of gaze. The nystagmus becomes evident only when the patient looks in one or other direction of gaze. Gaze-deviation elicited nystagmus comprises various types of nystagmus. For example a mild degree (1°) of vestibular nystagmus may be present only when the patient looks in the direction of gaze of the quick component.

**Rebound nystagmus**

This type of nystagmus was first reported by Bárány (Kornhuber, 1975) and was subsequently clearly demarcated by Hood, Kayan and Leech (1973). Hood and his colleagues point out that the features are such that, in the initial examination of the eyes in the primary
position of gaze, no nystagmus is observed. With a subsequent gaze deviation to, say, the
right, a brisk nystagmus with its fast component to the right appears. After about 20 seconds,
the nystagmus fatigues and may even reverse direction. If, at this time, the eyes are returned
to the primary position, nystagmus to the left, not present initially, occurs and this too fatigues
with time. Hood and his colleagues reported that this type of nystagmus is associated with
chronic cerebellar disease and was found in 6% of neurological patients referred for a neuro-
otological examination.

Halmagyi and his colleagues (1979) have reported a rebound-rebound nystagmus
which replaced a periodic alternating nystagmus treated with baclofen.

**Physiological nystagmus**

Daroff and Dell'Osso (1979) pointed out that there are three types of spontaneous
physiological (normal) nystagmus, that is unsustained end-point nystagmus, sustained end-
point and fatigue nystagmus.

Unsustained end-point nystagmus is the most common type of physiological
nystagmus. This occurs with gaze deviations of 500 mrad (about 30°) or more. On initially
attaining gaze deviations in excess of this angle, a few beats of nystagmus may occur. This
nystagmus is typically a horizontal jerk nystagmus, with a quick and a slow component and
with the quick component directed in the direction of the gaze. Unsustained end-point
nystagmus is frequently bilateral and commonly symmetrical.

Sustained end-point nystagmus again starts immediately the eyes attain an extreme
lateral gaze position, or within a few seconds of attaining such a position. This nystagmus is
again typically horizontal, bilateral and symmetrical. Under illuminated conditions the
amplitude is within the range 15-50 mrad (about 1-3°) and with a frequency in the range 1-3
Hz. Changes occur in this pattern under conditions of darkness. Moreover, there is a distinct
intrasubject variability (Schmidt and Kommerell, 1976). Of 11 normal subjects studied by
Daroff and Dell'Osso using an infrared recording technique, six developed a sustained gaze-
evoked nystagmus with gaze deviations of 700 mrad (40°); one subject developed the
nystagmus at a gaze-deviation of only 350 mrad. In all cases, recordings of the nystagmus
showed the slow phase to be linear.

Fatigue nystagmus begins during extended maintenance of an extreme gaze deviation.
This type of nystagmus becomes increasingly rotary with more prolonged and extreme gaze
deviations (Nylén, 1922). Bárány (1906b) reported that the condition occurred in 60% of
normal subjects with maximally deviated gaze maintained for periods in excess of 30 seconds.
It would appear that the occurrence of this nystagmus is a function of the duration of the
maintenance of extreme gaze deviation. Schmidt and Kommerell reported one subject where
the nystagmus began after a latency of 90 seconds.

There is probably a continuum between many forms of physiological nystagmus and
forms of pathological nystagmus. For example, both types of end-point nystagmus show
remarkable similarities to gaze-evoked spontaneous nystagmus. The nystagmus of myasthenia
gravis is probably an exaggeration of fatigue nystagmus.
Optokinetic nystagmus and slow pursuit eye movements

Purkinje is said to have observed a nystagmus in bystanders who were watching passing cavalry. A nystagmus may also be observed in passengers in railway trains who gaze at the passing countryside (hence the term 'railway nystagmus'). This nystagmus is similar to vestibular nystagmus in that, although of optical origin, it shows a fast and a slow component.

Robinson (1975b) has sought to distinguish between optokinetic nystagmus and pursuit nystagmus. The optokinetic system has little or nothing to do with following moving targets. It is concerned, phylogenetically, with estimating self-rotation and the generation of eye movements appropriate to self-rotation. Robinson illustrated the biological need for such a system to supplement the vestibular system by citing a fish which would swim around its territory once every 10 seconds. This would give an angular velocity of 628 mrad/s. Accepting a cupular time constant of 4 seconds, there would be no vestibular-ocular reflex left after 12 seconds, so that retinal images would slip at 628 mrad/s and the fish would be unable to see anything. Thus the real purpose of the optokinetic system is not to track a moving visual environment while the object is stationary, but to use vision to help the vestibular system to assess self-rotation within the environment and generate appropriate eye movements.

Vestibular and optokinetic signals are probably combined in the vestibular nuclei (Henn, Young and Finlay, 1974) or in the prepositus hypoglossi nuclei of the dorsal medullary reticular formation (Baker and Berthoz, 1975; Uemura and Cohen, 1975).

Stimulation of the entire visual field is the appropriate stimulus for generating optokinetic nystagmus. The subject should therefore be within a rotating cylinder. If the subject does not perceive self-rotation, the optokinetic system is probably not being stimulated. Robinson argues that rotating hand drums or moving stripes in front of the subject test the visual pursuit system and not the optokinetic system. However, for a number of years clinicians have used Bárány’s vertically striped hand drum to elicit a nystagmus which has provided useful diagnostic information.

In 1926, Fox and Holmes showed that a directional preponderance of this optically induced nystagmus occurred with lesions of the inferior part of the parietal lobe. In those patients where the directional preponderance occurs, the fast component of the nystagmus that is directed towards the side of a lesion is relatively more marked than that directed to the contralateral side. The actual change is a suppression of the nystagmus which is directed away from the side of the lesion. This is because the foveal pursuit system is impaired (Balogh, Yee and Honrubia, 1979). The integrity of the slow pursuit system for objects going away from the side of a lesion would permit the fast phase of this nystagmus to be retained towards the side of a lesion.

Since Fox and Holmes’ initial observation, this optically induced nystagmus has been shown to be disturbed by pathological processes in a variety of locations in the brain (Enoksson, 1956). Thus, even if the localizing value is less specific than Fox and Holmes thought, the test has lateralizing value, a directional preponderance being directed towards an involved cerebral hemisphere. Bárány (1907) first reported the abolition of the quick phases of both optically induced and vestibular nystagmus in supranuclear lesions. Dix and Hood
(1971) reported that two cases of progressive supranuclear palsy showed abolition of the fast components both of this optically induced nystagmus and of vestibular nystagmus (induced by simultaneous bilateral caloric stimuli) in the vertical plane in both cases. Tonic deviations of the eyes occurred in the direction of the slow component of both optically induced nystagmus and vestibular nystagmus in both cases. Doll's head movements (see below) were intact in both cases in the vertical and horizontal planes.

The fist and only sign of an internuclear ophthalmoplegia may be dissociation of this optically induced nystagmus (Smith and David, 1964).

In peripheral vestibular lesions, a directional preponderance may be observed with the 'passive' stimulation but not with the 'active' optical stimulation.

**Oculocephalic reflex**

The Roth-Bielschowsky or doll's head phenomenon (*Puppenkopfphänomen*) refers to compensatory eye movements in response to passive turning of the head. The phenomenon is also termed the 'oculocephalic reflex', and the method of inducing it, the 'oculocephalic manoeuvre'. The manoeuvre is performed by briskly turning the subject's head from side to side, and then flexing and extending the neck. In each case, conjugate deviations of the eyes in the direction opposite to the direction of movement constitute a positive (normal) oculocephalic reflex. In such a case, the nuclear and infranuclear oculomotor pathways must be intact. When conscious patients are examined, they should be told to fix their gaze on an object in the primary position of gaze. In such a case, the manoeuvre tests the fixation mechanism (position maintenance system), the slow pursuit (following mechanism) and the vestibular mechanism. With comatose patients, only the last system is tested. Roth (1901) and Bielschowsky (1903) reported the occurrence of the phenomenon in patients who had lost the ability to make conjugate deviations of the eyes in one or other direction. This pattern of ocular response is referred to as the Roth-Bielschowsky syndrome. Vertical eye movements are preferentially affected. The syndrome is associated with lesions of the basal ganglia or tectum of the midbrain. As mentioned previously, Dix and Hood (1971) reported preservation of doll's head movements, but abolition of the fast components of both optically induced nystagmus and of vestibular nystagmus, in patients with progressive supranuclear palsy. In a series of 100 stroke patients seen within 48 hours of admission, Yap, Loon and Nei (1975) observed that five had an absent oculocephalic reflex. Four of these five were dead within 4 weeks.

**Counter-rolling**

The phenomenon of counter-rolling of the eyes was first observed by John Hunter in 1786, and subsequently described, under the name *Gegenrullung*, by Bárány (1906a). In the French literature, this phenomenon is referred to as *la contrerotation oculaire*. Nelson and Cope (1971) refer to it as the ocular countertorsion reflex. Normally, static head tilts of up to 785 mrad (45°) produce compensatory counter-rolling of the order of only 90 mrad (about 5°) (Miller, 1962). Vestibular disorders may be associated with either a reduction in the degree of counter-rolling or an accentuation, with rotations of up to 400 mrad (23°).
Ocular counter-rolling is absent in 'deaf mutes' (Miller and Graybiel, 1963), but may be preserved in cases of streptomycin intoxication where both caloric and rotatory stimuli failed to elicit a vestibular response. Nelson and House (1971) reported that the degree of counter-rolling was halved in subjects who had undergone unilateral labyrinthectomy or a vestibular nerve section.

Counter-rolling may be augmented during vertiginous episodes (Bárány, 1960a).

A postural abnormality involving ocular counter-rolling, skew deviation of the eyes and head tilting, is termed 'ocular tilt reaction' (Rabinovitch, Sharpe and Sylvester, 1977). Halmagyi, Gresty and Gibson (1978) have reported the occurrence of this reaction following inadvertent destruction of a vestibular labyrinth during stapedectomy.

Halmagyi and his colleagues offer the most likely explanation for ocular counter-rolling never being fully compensated, that is the failure to realign the visual with the earth-fixed axes. They point out that, if there were full oculomotor compensation for head tilt, then the visual and earth-fixed axes would be matched despite the fact that the head might no longer be vertically aligned in space. A mismatch of visual, vestibular and proprioceptive information could be a disadvantage during natural movements. However, an incompletely compensatory response generates a continuous visual error signal to stimulate correct realignment of the head with respect to the earth-fixed axes.

Ocular counter-rolling is best observed by fundoscopy.

**Positioning nystagmus and positional nystagmus**

There are a group of tests, sometimes referred to as the positional tests for nystagmus, in which the nystagmus is elicited by means of a variety of either cervical posture or head positioning tests. In the routine examination for nystagmus, nystagmus should be sought not only with the head in the primary position but also when flexed, extended, laterally inclined or rotated. These constitute the cervical posture tests. Nystagmus produced, or accentuated, by these manoeuvres is attributed to the influence of cervical proprioceptors and/or vertebral artery compression.

The positional test described by Dix and Hallpike (1952) to elicit a nystagmus consists of firmly grasping the head of a patient, who is sitting on a couch, and then briskly taking the patient back into the critical position. This position is one in which the head is rotated 785 mrad (45°) to one side and extended 500 mrad (nearly 30°) over the edge of the couch. The test has consequently been criticized in that at least three nystagmogenic factors are operating. These are what are referred to in the French literature as l'élément cinétique (kinetic factor), l'élément cervical (cervical factor) and l'élément spatial (truly positional factor). It is, however, precisely because of these multiple factors that the test, performed in this manner, is a convenient screening test for 'positional nystagmus': thus in the French it is referred to as une épreuve de dépistage, literally a 'tracking down' test (Aubry and Pialoux, 1957). It is nevertheless doubtful whether the cervical factor is important in the production of nystagmus under these conditions, especially when the nystagmus is sought either by the naked eye or by Frenzel glasses. Dix and Hallpike retested a group of subjects who demonstrated
nystagmus with this procedure by examining them on a tilt table; this would abolish any cervical factor. There was no appreciable change in the findings. In any case, subsequent manoeuvres can be conducted to eliminate one or other potential nystagmogenic factors. One can, as Nylén (1950) and others have reported, perform the positional test by asking subjects to assume the critical position slowly of their own accord. Thus any nystagmus produced by this procedure will be truly positional (Lagenystagmus) and not positioning (Lagerungsnystagmus). The latter type of nystagmus broadly corresponds to the 'benign paroxysmal positional nystagmus' of Dix and Hallpike, and the former type, to the central type of 'positional nystagmus'. The paroxysmal type is much more common than the central type. The term 'benign' has been objected to by Lindsay (1962) and others because some of the cases, albeit a small proportion (probably less than 1%), are associated with an intracranial tumour (Cawthorne and Hallpike, 1957; Riesco McClure, 1957; Harrison and Ozsahinoglu, 1972). Thus these cases are not representative of the clinical condition. Similarly, because of the paucity of histological material available, it is doubtful if this material is representative of positioning nystagmus in general. Moreover, there is disagreement among the experts regarding the interpretation of the histological material (compare Cawthorne and Hallpike, 1957; Lindsay, 1962; Schuknecht, 1962). Nevertheless, the histopathological studies of Schuknecht (1969) indicate that at least one pathological basis for positional nystagmus is a condition termed 'cupulolithiasis'. Cupulolithiasis encompasses the presence of an inorganic deposit on the cupula of the posterior semicircular duct; this renders the organ sensitive to gravitational force and therefore subject to stimulation with changes in head position. In some cases the cupulolithiasis may be due to dislodged otoliths (otoconia). These are minute crystalline bodies (calcite) which normally occur in the gelatinous otolithic membrane of the maculae of the utricle and saccule.

Dayal and his colleagues (1977) pointed out that paroxysmal positional nystagmus is the most frequently seen type of nystagmus prior to its disappearance in end-organ-induced nystagmus. Following a study of stapedectomized patients and drug-induced nystagmus, these authors considered that the type of spontaneous or provoked vestibular nystagmus was of little diagnostic value in localizing the site of a lesion; the various types reflected different degrees of vestibular compensation that had occurred.

A possible pathophysiological explanation for positioning nystagmus is that it represents an imbalance of a pair of vertical semicircular duct systems. It should be noted that, in the positional test, the head is taken back in a position wherein the head is rotated 785 mrad, that is the movement is in the plane of a pair of vertical semicircular ducts (Stenger, 1959; Frenzel, 1960; Hallpike, 1967). Specifically, the ipsilateral posterior and the contralateral superior (anterior vertical) ducts are implicated. Stimulation of this pair of ducts would be expected to produce a nystagmus with a rotary component when the eyes are viewed by the observer facing the patient. This is the usual finding. It is to be noted that Ledoux (1958), in animal experimental studies, demonstrated that the resting action potential discharges in the nerves from the vertical semicircular ducts were appreciably affected by head positioning. Duensing (1967) found that cells in the lateral vestibular nucleus, which received vertical duct signals, also show a marked interaction with gravity receptor mechanisms. Baloh, Sakala and Honrubia (1979) analysed the electrically recorded eye movements associated with paroxysmal positional nystagmus. They concluded that the characteristics were consistent with a burst of excitatory activity originating in the posterior duct of the ear that is undermost at the end of the positioning manoeuvre.
Patients with persisting paroxysmal vertigo usually respond to a system of head and balance exercises (Dix, 1979; Chapter 15). The modus operandi for this treatment is by a habituation mechanism. Intractable paroxysmal vertigo not responding to this method of physical treatment may be treated by transection of the posterior division of the vestibular nerve in the foramen singulare (Gacek, 1974). If there is no useful hearing in the involved ear, labyrinthectomy may be performed.

**Head-shaking nystagmus**

Head-shaking nystagmus or Kopfschüttelnystagmus is provoked by rapid side-to-side movements of the head (Vogel, 1932). Twenty of such movements provide a suitable stimulus. The eyes are then observed for nystagmus immediately after this manoeuvre. In the case of peripheral lesions this nystagmus is usually directed to the contralateral side. This may be the case even when a recovery nystagmus is present (Frenzel, 1961). If such a discordance is evident, it may be due to the influence of neck cervical proprioceptors in the head-shaking procedure. However, it is equally (perhaps more) likely that such a discordance can be explained on the basis of the existence, within a deranged vestibular system, of something analogous to 'recruitment' in the auditory system.

Kamei and Kornhuber (1974) failed to demonstrate any head-shaking nystagmus in a group of 40 normal subjects. However, in a group of patients with central vestibular lesions, head-shaking nystagmus under Frenzel glasses was the only nystagmus detectable. The test is, of course, contraindicated in cases of raised intracranial pressure or retinal detachment. Cervical spine disorders may also preclude its use, or require caution.

**Rotation tests**

If a patient with a suspected vestibular disorder has not shown any spontaneous or provocation nystagmus in an examination conducted so far, he/she may then be investigated with rotatory tests (Bárány, 1907; van Egmond, Groen and Jongkees, 1948; Greiner, Conraux and Collard, 1969; Wall, O'Leary and Black, 1978; Wolfe, Engelken and Olson, 1979). However, the major problem with rotation tests is that both vestibular labyrinths are stimulated.

**Caloric tests**

The caloric test is an attempt to simulate a one-sided rotatory stimulus. Thermal stimuli applied to one ear under test tend to produce convection currents in the semicircular ducts, that is a tendency to move the fluid relative to the walls of the duct, so exerting a deflecting force on the cupula concerned. It has been said that the mechanism of the caloric test will need reassessment following experiments conducted under zero gravity conditions. However, the ability to produce a response under such conditions appears to have come on after a quiescent time span. It is therefore highly likely that what one has observed has been the re-adaptation of the vestibular system to a changing physical environment.

The magnitude of nystagmic response (duration, total number of beats or maximum velocity of slow phase) in the caloric test can be specified by expressing either canal paresis
or directional preponderance as a percentage. Thus the percentage canal paresis, $E$, can be expressed as:

$$E = 100 \frac{((B+D) - (A+C))}{(A+B+C+D)}$$

where $A = $ response to warm stimulus applied to the right ear,
$B = $ response to warm stimulus applied to the left ear,
$C = $ response to cold stimulus applied to the right ear, and
$D = $ response to cold stimulus applied to the left ear;

and the percentage directional preponderance, $F$, is expressed as

$$F = 100 \frac{((A+D) - (B+C))}{(A+B+C+D)}$$

Thus, if $E$ is positive, a right canal paresis is the case, if negative a left one is the case. If $F$ is positive, the directional preponderance is to the right, if negative, to the left.

The first equation is a measure of the preponderance of one vestibular labyrinth, or, more specifically, the lateral duct, ampullary crest and nerve, over the contralateral corresponding structure. Consequently, some authors prefer to use the term 'labyrinthine preponderance' instead of canal paresis. It can also be pointed out that the use of the term 'canal paresis' might lead to irrational utterances in certain cases, for example in demyelinating disorders, where the patient may have completely normal labyrinths. More importantly, a semicircular canal, being a bony structure, cannot be paralysed. However, the term 'labyrinthine preponderance' is itself not free from criticism since 'labyrinth' is a synonym for the internal ear, and cochlear function in so-called 'labyrinthine preponderance cases' may be entirely normal. Objections would also arise in respect of the terms 'duct preponderance' or 'ampullary preponderance'; abbreviation of the former would give confusion with directional preponderance and the abbreviation of the latter would give confusion with action potential. The matter must therefore rest until vestibulometric terminology has been standardized. Nevertheless, the term 'lateral ampulloneural preponderance' might be suggested since the term would emphasize a relative caloric response in respect of the two ears and also emphasize that the test is primarily one of the lateral semicircular duct and its sensor (ampullary crest) together with its pathway (nerve). Thus an impaired response (to both warm and cold stimuli) for the left ear would be designated a right lateral ampulloneural preponderance; the converse response would be termed a 'left lateral ampulloneural preponderance'.

One of the perception associated with response to the caloric test (and many other vestibular stimuli) is the sensation that a particular point in visual space is moving. This illusion is termed the 'oculogyral illusion'. The use of this as an index of caloric test response was suggested by van Dishoeck and Nihoff in 1953. A clinical caloric test based on this method has been shown to be both simple and reliable (Arroyo and Hinchcliffe, 1977). These authors concluded that, when used in conjunction with other tests for detecting vestibular imbalance, for example examination for spontaneous and provocation nystagmus using Frenzel glasses, the oculogyral illusion caloric test should provide a convenient procedure for the clinical examination of vestibular function.
The oculogyral illusion caloric test has the supreme advantage that, not only does it not require any special apparatus for the recording and registration of nystagmus, but it can be done without requiring the examiner to be able to recognize nystagmus. However, it does require the availability of thermostatically controlled water baths which are used to fill the 50-mL syringes used to deliver the stimulus.

A simple test, but one which does require the observer to be able to recognize nystagmus, involves the use of, initially, 0.2 mL of ice water. Nelson (1969) has modified this method by using Frenzel glasses to abolish optic fixation and so enhance the induced vestibular nystagmus. Nelson pointed out that the features of his test which recommended it for clinical use include the universal availability and portability of the test materials since only a paper cup with water, three ice cubes, a plastic tuberculin syringe, a watch and the Frenzel glasses are required. The principal drawback of both the Linthicum and the Nelson methods is that they use water at one temperature only. Norrè (1975) has drawn attention to the errors which may result from using other than a bithermal caloric test.

Simultaneous binaural caloric testing

If, with the subject supine, the two ears are irrigated simultaneously with water at the same temperature, a nystagmus will result which, at least in theory, will reflect the difference in response to the two stimuli given separately. Thus, if a response of the same magnitude is obtained with water at 303K (30°C) when the ears are separately stimulated, then the responses will cancel out when the ears are simultaneously stimulated. No nystagmus would then be observed. If a water at 303K produces, when given monaurally, a greater response from the right ear than from the left ear, simultaneous (binaural) stimulation with water at the same temperature should produce nystagmus to the left.

Simultaneous bilateral caloric stimulation may also be used to test cristoneural function in respect of the vertical semicircular canals. For such purposes, the subject's head is placed in the vertical position. Downward-directed nystagmus occurs with binaural warm caloric stimuli; upward-directed nystagmus occurs with binaural cold caloric stimuli. Dix (1970) has used this procedure in the investigation of patients with supranuclear ophthalmoplegia.

Conclusions

It is clear that there are a multiplicity of clinical tests for the investigation of aural function. Clearly not all of these will be needed in a given case. The selection of tests will be governed by many factors, including not only what aspects of aural function are deranged but also what appears to be the general psychological and medical condition of the patient. Nevertheless, it will be clear that simple clinical procedures can offer a lot before recourse is made to more sophisticated methods of investigation.