Chapter 18: Vertigo

A. G. Kerr

Vestibular disorders have been considered in depth in Volume 2. However, every day the otolaryngologist is actively involved in the management of patients with vestibular problems and this volume would be incomplete without a chapter on vertigo. This chapter differs from those in Volume 2 in that it takes a more practical, day-to-day look at the problem omitting much of the detail.

Vertigo tends to be a subject which depresses both patient and doctor. Generally, the patient has difficulty in describing his symptoms and, in the time he has available, the doctor may have difficulty in grasping the picture which the patient is trying to convey.

The patient often feels foolish and thinks that the doctor may be secretly smiling at his naivety in producing these bizarre complaints. On the other hand, the doctor, in approaching this difficult symptom complex, may have a haphazard system, or none at all, and try to make a diagnosis with inadequate information.

Unfortunately, when the conscientious doctor turns to the standard textbooks he may find that he does not get much help. This is not because the information is not available. There are many excellent and comprehensive books on this subject. The problem is that, with few exceptions, the textbooks tend to start with the diagnosis and then give the clinical features. For example, having made a diagnosis of dizziness secondary to vestibulotoxic drugs, there is little difficulty in finding a description of this condition and its management. However, if one starts with the symptoms, but not the diagnosis, it may be necessary to read through most of the textbook to find the appropriate section.

There is only one straightforward way of overcoming this difficulty. That is to start with the detailed symptoms and work backwards, through the differential diagnosis to the identification of the underlying problem.

Despite the bizarre histories which one frequently obtains from patients complaining of vertigo, there are certain characteristic patterns. Many of these are consistent with a specific underlying pathology and, obviously, this pathology should be considered in the management of the patient.

Definition of vertigo

The definitions of vertigo which have been given are often as bizarre as the histories given by the patient. Although the Latin root of the word implies a sensation of turning, in general clinical usage of this term a sensation of spinning or turning need not necessarily be implied. Probably the most effective definition is 'a subjective sense of imbalance'. If this contains a sensation of spinning then one can add the adjective 'rotatory' when describing the symptom.
Types of vertigo

The diagnosis of vertigo is usually made on the basis of the history. Subsequent examination and investigations are normally used to confirm the diagnosis and it is likely that, in 80% of cases, if one does not have an idea of the diagnosis at the end of the history, one is unlikely to have it at the end of the examination and investigations.

Basically, vertigo can be described in one of two ways; either it is rotatory or it is not. If it is rotatory, patients usually have little difficulty in saying so. Where there is no obvious sense of rotation patients have more difficulty in giving a good description. They use various terms but, in essence, the complaint is that they are unsteady. Many will have associated symptoms such as deafness, tinnitus, vomiting or nausea.

Generally speaking, each of the two groups (rotatory or unsteady) can be further subdivided into those where the symptom is episodic and those where it is more or less continuous. When it is episodic it may be very short-lived, that is less than a minute, or very much longer, such as hours or days. Consequently, the vast majority of patients with vertigo can be classified as in Table 18.1. It would be naive to expect every patient to fit neatly into any classification but, remarkably, most do. Broadly speaking, each groups has a basic underlying pathological correlate which is discussed later in the chapter.

Table 18.1. Classification of vertigo

Rotation

(1) Episodic
   (a) seconds
   (b) hours
(2) Prolonged
    weeks

Unsteadiness

(1) Episodic
   (a) seconds
   (b) hours to days
(2) Prolonged
    weeks to months

A certain degree of licence has been taken in that 15 minutes are equivalent to hours.

History

Certain precautions must be observed when taking a history. The first is to ensure that the story given by the patient starts at the onset of the complaint. There is a tendency in any condition which has been going on for some years, for the patient to start the history somewhere other than at the beginning. It is important to establish when the patient was last perfectly steady.
The doctor must control the history-taking. It is through this that he develops a picture of the condition. If he does not have a systematic way of covering all aspects, he will get an incomplete picture. If left to their own devices, patients rarely give the complete story because they will emphasize the aspects which have impressed them most. For example, the periods of freedom from vertigo are as important as those with vertigo, but most patients do not talk about these without prompting.

When seeking a description of the symptoms the patients may need some guidance. One must be careful about leading questions but many find it impossible to describe symptoms without help. It is necessary to know the speed of both onset and resolution.

Throughout the history one must ensure that both the patient and the doctor are on the same wavelength. For example, many patients will say that they are dizzy 'all the time' when they mean 'frequently', but some are, in truth, dizzy 'all the time'. One can ensure that one has an accurate history by asking the same question in two different ways and confirming that consistent answers come back. If not, one must identify where the misunderstanding has arisen and resolve it.

Finally, the patient may well try to interpret the symptoms himself and the doctor must check that both he and the patient are using the same interpretation.

By and large, the history is best taken chronologically with specific details being sought of the first and of a typical recent attack.

Details must be sought of associated symptoms such as deafness, tinnitus, nausea, vomiting, diplopia, blurring of vision, anaesthesia of the face, headaches and loss of consciousness.

**Examination**

In an ideal world, every patient complaining of vertigo would have a detailed neurological examination. Clearly, this is not possible in the average busy otolaryngological outpatient department. Even in vertigo clinics, which are desirable in all sizeable units, this is difficult. However, it is usually possible, within a few minutes, to determine those patients who require a detailed neurological examination.

Following a routine ear, nose and throat examination it is usually possible to make a rapid assessment of cranial nerves III to XII, of cerebellar function and the ability to perform balance tests. It is extremely unusual that the need for a detailed neurological assessment will be overlooked if these are all normal and the history does not point to a central lesion. Although olfactory and ophthalmic symptoms may occur in association with vestibular problems, routine examination of these systems is not required unless some aspect of the history points to them. However, every patient with vertigo should be examined for nystagmus (see below, section on nystagmus). During this examination cranial nerves III, IV and VI will automatically be checked. If there is direction changing or vertical nystagmus, a neurological lesion is usually present and a detailed neurological examination is required. Usually a test of the corneal reflex is sufficient for the assessment of the fifth cranial nerve. Weakness of the seventh cranial nerve will often become apparent simply by looking at the patient while
taking a history. A slower blink on the affected side is usually obvious. Very minor weakness of the facial nerve can be detected by the inability of the patient to bury the eyelashes in tight closing of the eyes.

The auditory part of the eighth cranial nerve will be tested during the routine examination and subsequent audiogram. The ninth and tenth cranial nerves can be rapidly checked by the gag reflex, the eleventh by shrugging the shoulders and, theoretically, the twelfth by having the patient protrude the tongue. Twelfth cranial nerve involvement is uncommon in vertiginous problems which is fortunate as early lesions of this cranial nerve are difficult to detect.

The assessment of balance should have begun when the patient walked into the room. If he walked in steadily there will be no further need to check his walking. A positive Romberg test usually means considerable impairment of function. An extended or tandem Romberg test is more refined. In this the patient is asked to stand in a heel to toe position, first with the eyes open and then closed. One-leg standing, with eyes open or closed, is a further clarification of balance.

**Nystagmus**

Nystagmus is a disturbance of ocular posture, characterized by a more or less rhythmic oscillation of the eyes. The speed of the eye movements may be the same in both directions or one may be quicker than the other. There is little or no interval between consecutive movements which may be horizontal, vertical or rotatory.

The posture of the eyes, and consequently nystagmus, depends on two sets of afferent impulses, visual and vestibular. The visual impulses are concerned with regulation of the position of the eyes in relation to the object of visual interest. It is by means of the vestibular impulses that the position of the eyes is regulated in relation to the position and movements of the head and the remainder of the body. Eye posture is coordinated by a central mechanism that receives these afferent impulses and controls the efferent impulses to the ocular muscles. Nystagmus may result from any lesion involving these afferent pathways, their central connections or, less frequently, the efferent pathways.

There are two broad categories of nystagmus depending upon the appearance of the movements. Pendular nystagmus is characterized by ocular oscillations that are approximately equal in velocity in both directions. These are almost always horizontal. There may be a jerk component on extreme lateral gaze. Phasic, or jerk, nystagmus is characterized by rhythmic oscillations in which the movement in one direction is significantly faster than in the other direction. Although the slow movement is pathological, the fast, or corrective, movement is used to denote the direction of the nystagmus.

The position in which the nystagmus is least marked is called the null point.

**Examination for nystagmus**

Nystagmus found at examination may be either spontaneous or induced. Spontaneous nystagmus is said to be present when there are rhythmical eye movements on forward gaze.
Induced nystagmus is said to be present when rhythmical movements are brought about by some specific test. There is an intermediate nystagmus, or gaze nystagmus, which is demonstrated by altering the gaze to the right or left, or up or down. For the purposes of this discussion intermediate or gaze nystagmus will be considered under the heading of spontaneous nystagmus.

When examining the nystagmus it is essential that the patient is in good light. The object at which the patient is asked to look must be within a comfortable range. Ideally, it should be at infinity but a few feet away will suffice, so that excessive accommodation is not required. Care must be taken that, on looking laterally, the nose does not impinge on the field of vision and on looking upwards, that the object remains comfortably in view. Otherwise, searching or nystagmoid movements may be mistakenly interpreted as nystagmus.

Causes of spontaneous nystagmus

Labyrinthine

Labyrinthine nystagmus, which is phasic, has four main characteristics. First, it is usually associated with a sensation of vertigo. Second, the nystagmus is always unidirectional. Third, it is more marked when looking in the direction of the fast phase. Fourth, it is enhanced by removal of fixation by eye closure, Frenzel's glasses or darkness. Following a labyrinthine destructive lesion, the nystagmus decreases with time and clinically has usually disappeared entirely within 4 weeks. However, even then it may still be detected by removal of visual fixation.

Mechanism of vestibular nystagmus

In the normal subject, the tone of the extraocular muscles is to some extent controlled by the vestibular nuclei. The vestibular nuclei drive the eyes to the opposite side. Hence, the vestibular nuclei on the right side influence the left lateral and the right medial recti muscles, moving the eyes to the left side. Normal ocular posture is maintained by a correct balance between the vestibular nuclei on the two sides.

If there is an abnormality of function of the vestibular nuclei on one side there will be a change in the tone in the corresponding muscles. Consequently, stimulation of the vestibular nuclei on one side will result in movement of the eyes to the opposite side. Unilateral inhibition of the vestibular nuclei will result in movement of the eyes towards that side. The effect is to remove the eyes from the point of visual interest. Consequently, there is a correcting movement to restore the eyes to the original position. Repeated deviation of the eyes, and repeated correction, produce nystagmus.

A paralytic lesion involving the right labyrinth, and hence the right vestibular nuclei, will result in slow deviation of the eyes to the right side with the quick correcting movement being to the left side resulting in so-called left beating nystagmus.

In describing unidirectional phasic nystagmus one should state, not only the direction, but also the degree. First degree nystagmus is said to be present when phasic eye movements are detected when looking in the direction of the quick phase. Second degree nystagmus is
present when the phasic movements are seen on looking straight ahead, and third degree when looking in the direction away from the nystagmus.

The logical nature of this is easy to understand when one considers a specific example. A major lesion resulting in reduced function in the right vestibular nuclei will cause grossly reduced tone in the left lateral and right medial recti muscles. Consequently, the opposing recti muscles moving the eyes to the right, will, in comparison, be so hyperactive that there is further drift of the eyes to the right, even when already looking to the right side. As the disproportion between the two groups of vestibular nuclei diminishes as a result of central nervous system compensation, the nystagmus will slowly reduce to second degree, then first degree, and finally nystagmus only when fixation is removed.

Alexander's law states that, in general, if vestibular nystagmus is present it can be enhanced by moving the eyes in the direction of the fast phase and diminished by moving the eyes in the direction away from the fast phase.

### Central nervous system lesions

Nystagmus due to central nervous system lesions presents in a multitude of different forms, some of which are not clearly understood. However, most neurological lesions causing nystagmus produce a bidirectional (direction changing) form. Labyrinthine lesions never do this. Similarly, it has been said that labyrinthine lesions never produce a vertical nystagmus which is therefore always indicative of a central lesion. This is not quite true as excitation of the posterior canal crista, as in cupulolithiasis (benign paroxysmal positional vertigo) may cause an upbeat nystagmus and transection of the posterior ampullary nerve may cause a downbeat nystagmus (Gacek, 1985).

Unidirectional nystagmus which is diminished by removal of fixation or which does not vary in amplitude or velocity in different directions of gaze, is almost certainly of central origin.

Textbooks of neurology and ophthalmology describe rare and bizarre forms of nystagmus, for example, monocular, involving only one eye or see-saw, where one eye moves up and the other down. In these and in other central types of nystagmus, most otolaryngologists will wish to seek the opinion of a neurologist!

### Toxic

Many drugs including alcohol, barbiturates, tranquilizers and anticonvulsants, are potential causes of nystagmus. It is likely that, in most instances, the effects of the drug are on the central nervous system. Indeed, it has been suggested that drugs are the most common cause of vertical nystagmus. However, the positional nystagmus which is associated with alcohol, may well be peripheral in its action. Positional alcoholic nystagmus has two phases, in opposite directions. The first phase occurs shortly after the ingestion of alcohol and passes off after a few hours. The second phase occurs about 8 hours later. It has been suggested that the nystagmus is due to partial replacement by alcohol of water-containing components of the semicircular canal ampullae, followed by later metabolism, each phase resulting in abnormal but opposite impulses.
Ocular

Any lesion affecting the macula, especially where peripheral vision is still maintained, may result in nystagmus which is usually pendular. This may occur in amblyopia. In the past, miners' nystagmus also fell into this category although it has been suggested that neurosis played a part in maintaining the disorder.

Central nervous system lesions affecting the ocular pathways may also result in ocular nystagmus.

Congenital

Congenital nystagmus, which is often familial, is usually horizontal and pendular although it may be phasic (jerk). The null point is close to the position of forward gaze. Unlike that in any other condition, horizontal nystagmus continues even when the eyes are turned upwards. Closure of the eyes results in a marked reduction in the nystagmus but the effects of darkness, with eyes open, are variable and in some instances the nystagmus increases.

In the everyday life of the otolaryngologist, the diagnosis is usually verified by the patient confirming that the nystagmus has been present since as long as he can remember.

Causes of induced nystagmus

Nystagmus may be induced by clinical testing or by certain investigations. The most common clinical test inducing nystagmus is the positional test. This is useful in diagnosing positional vertigo and differentiating between the common benign paroxysmal positional type and the uncommon positional vertigo of central origin (Table 18.2).

In the fistula test, nystagmus is induced by altering the pressure in the external auditory meatus, producing both nystagmus and a sensation of dizziness. The Tullio phenomenon is a variation of this cause of nystagmus and is induced by acoustic stimuli of high intensity.

Induced nystagmus in clinical investigations is produced by the optokinetic drum, pendulum tracking and caloric and rotational tests.

Investigations

The only investigation that is required routinely is an audiogram. By and large, if an air conduction audiogram is normal, no further audiometric tests need be done. However, it is the author's routine to do an air and bone conduction audiogram and in addition, a simple speech discrimination assessment (PBmax). There is a danger of over-investigation of patients with vertigo in an attempt to replace ignorance by biochemical screening.

Electronystagmography and caloric testing are not routine. Indeed, there are many who claim that the caloric test is of no practical value. The author disagrees with this conclusion.
but does not feel that it needs to be carried out in the majority of vertiginous patients and especially in the elderly.

Radiological investigations and blood examinations are not required routinely. However, in the light of the clinical features, it may be desirable to exclude an acoustic neuroma or syphilis.

**Table 18.2. Comparison of the positional nystagmus of benign paroxysmal positional vertigo (BPPV) with that of certain lesions of the central nervous system (CNS)**

<table>
<thead>
<tr>
<th></th>
<th><strong>BPPV</strong></th>
<th><strong>CNS</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Latent period</td>
<td>A few seconds</td>
<td>Nil</td>
</tr>
<tr>
<td>Distress</td>
<td>Present: may be severe</td>
<td>Nil</td>
</tr>
<tr>
<td></td>
<td>with patient clutching</td>
<td></td>
</tr>
<tr>
<td></td>
<td>at couch or examiner</td>
<td></td>
</tr>
<tr>
<td>Direction of nystagmus</td>
<td>This is usually rotatory and is anticlockwise with the right ear down and clockwise with the left ear down. (When the nystagmus is horizontal it is towards the undermost ear.)</td>
<td>Variable</td>
</tr>
<tr>
<td>Duration of nystagmus</td>
<td>Less than 30 seconds</td>
<td>Persists while position maintained</td>
</tr>
<tr>
<td>On sitting up again</td>
<td>Similar events with nystagmus in opposite direction</td>
<td>Nystagmus stops</td>
</tr>
<tr>
<td>Fatiguability</td>
<td>Nystagmus and dizziness stop with repeated testing</td>
<td>Nystagmus persists with repeated testing.</td>
</tr>
</tbody>
</table>

**Pathological correlates**

*Table 18.3* shows the suggested pathologic correlates for the different types of vertigo.

**Table 17.3. Suggested pathological correlates for the different types of vertigo**

*Rotation*

(1) Episodic
   (a) seconds: short-lived stimulation or depression of the labyrinth
   (b) hours: physiological failure of the labyrinth or central connections

(2) Prolonged
   weeks: destructive lesion of the labyrinth or central connections

*Unsteadiness*

(1) Episodic
   (a) seconds: physiological overload of the vestibular system
   (b) hours to days: temporary impairment of the central connections or decompensation of the vestibular system

(2) Prolonged
   weeks to months: vestibular inadequacy.
**Rotatory**

(1a) Episodic rotatory vertigo, lasting for only seconds, would be expected from short-lived depression or stimulation of one of the labyrinths or its central connections. The main causes of such vertigo are benign paroxysmal positional vertigo, labyrinthine fistula, the caloric effect and alternobaric vertigo (Table 18.4). Also included in this group, although not in such pure form, are the post-concussional syndrome, vertebrobasilar insufficiency and cervical vertigo.

**Table 18.4. Short-lived episodic rotatory vertigo**

<table>
<thead>
<tr>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benign paroxysmal positional vertigo</td>
</tr>
<tr>
<td>Labyrinthine fistula</td>
</tr>
<tr>
<td>Caloric effect</td>
</tr>
<tr>
<td>Alternobaric vertigo</td>
</tr>
<tr>
<td>Post-concussional syndrome</td>
</tr>
<tr>
<td>Vertebrobasilar insufficiency</td>
</tr>
<tr>
<td>Cervical vertigo</td>
</tr>
</tbody>
</table>

These short-lived episodic types of vertigo can recur frequently, many times each day, depending on the frequency of the stimulus.

(1b) The episodic types of vertigo lasting from a few minutes to less than 24 hours, are due to a physiological or metabolic failure of the labyrinth (Table 18.5). They include Ménière’s disease, syphilitic labyrinthitis and other endolymphatic hydrops of immediate or delayed type. Also included in this group are decompensation of a previously compensated lesion and the dizziness which may occur in the first 24 hours after middle ear surgery.

**Table 18.5. Physiological failure**

<table>
<thead>
<tr>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ménière’s disease</td>
</tr>
<tr>
<td>Syphilitic labyrinthitis</td>
</tr>
<tr>
<td>Delayed endolymphatic hydrops</td>
</tr>
<tr>
<td>Decompensation of previous vestibular lesion</td>
</tr>
<tr>
<td>Following middle ear surgery.</td>
</tr>
</tbody>
</table>

Physiological failure and recovery can recur frequently but not as often as the short-lived attacks.

(2) Prolonged rotatory vertigo, lasting for more than 24 hours and usually for less than 3-4 weeks, can be expected when there is some destruction of the labyrinth or the central connections (Table 18.6). This is a large disparate group where the clinical picture is of severe incapacitating rotatory vertigo associated with nausea and vomiting.

Vestibular neuronitis, bacterial or viral labyrinthitis and vascular lesions of the inner ear fall into this group. Traumatic lesions, either accidental as in head injury or inexpert ear surgery, or planned as in labyrinthectomy or vestibular neurectomy, are also included in this
group. A rare but definite clinical entity is secondary metastatic deposits in the
cerebellopontine angle, from primaries in the breast, kidney, lung and prostate gland.

**Table 18.6. Destructive lesions**

| Vestibular neuronitis
| Trauma
| head injury
| ear surgery
| labyrinthectomy
| vestibular neurectomy
| Labyrinthitis
| bacterial
| viral
| Vascular lesions
| Metastatic deposits in cerebellopontine angle.

In many cases of naturally occurring destructive lesions the damage is incomplete and
the vertigo may recur. A destructive lesion cannot recur if it is complete. The central
neurological compensation which follows such lesions may, however, break down in the
future as a result of stress or some intercurrent illness.

**Unsteadiness**

In those patients with unsteadiness, the picture is usually not quite so clear-cut. However, it still bears some attempt at pathological classification.

(1a) The unsteadiness that lasts for only seconds can be due to a physiological
overload of the vestibular or central processing systems (Table 18.7). The central processor
deals with impulses not only from the labyrinth, but also from the visual and proprioceptive
systems. If the central processing system is overloaded, imbalance will be experienced. This
may occur for any one of three reasons. First, there may be excessive input, as can happen
in a normal person with very rapid movements. Second, it may occur as a result of abnormal
input, especially from the visual apparatus. Third, it may result from minor inadequacies in
the visual, proprioceptive or labyrinthine systems, when the central processor cannot keep
pace with all the signals that are arriving, so that short-lived unsteadiness occurs.

**Table 18.7. Physiological overload of central processor**

| Rapid movement
| Abnormal input - especially visual
| Minor inadequacies
| visual
| vestibular
| proprioceptive.

In young people, this is seen in the later stages of recovery from the post-concussional
syndrome or benign paroxysmal positional vertigo. It may also be seen in those who have
compensated for a destructive labyrinthine lesion. It is probably seen most often in the elderly, in such activities as rising quickly to the feet or turning rapidly. The dizziness is usually only momentary.

By its nature, this short-lived unsteadiness can occur many times each day.

(1b) The unsteadiness which lasts from hours to days may be due to temporary impairment of the central vestibular connections or decompensation of the vestibular system (Table 18.8). Drugs are a common cause of the temporary impairment. They may be self-inflicted as in drug overdose or alcohol. They may be iatrogenic where drugs such as tranquilizers or anticonvulsants are used, which, even in normal therapeutic dosage, can cause unsteadiness. Travel sickness also produces unsteadiness and is included in this category.

Table 18.8. Unsteadiness for hours to days

<table>
<thead>
<tr>
<th>Drugs</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>self-inflicted</td>
<td>iatrogenic</td>
</tr>
<tr>
<td>Travel sickness</td>
<td>Perilymph fistula</td>
</tr>
<tr>
<td>Active chronic suppurative otitis media</td>
<td>Decompensation of pre-existing lesion</td>
</tr>
<tr>
<td>Hyperventilation</td>
<td>Functional</td>
</tr>
</tbody>
</table>

It is difficult to categorize the dizziness due to perilymph fistula, but it can reasonably be inserted here, even though the unsteadiness may persist for weeks or months. Similarly, the unsteadiness sometimes seen in active chronic suppurative otitis media, which resolves with control of the infection, can also be included here. When it is prolonged it falls into the next category.

The unsteadiness due to stress, both in normal subjects and in those where decompensation of a pre-existing lesion occurs, can also be included. This may be precipitated by hyperventilation.

(2) Prolonged unsteadiness, lasting for weeks or months, is usually due to vestibular inadequacy (Table 18.9). This is most often seen in the elderly. It may also be seen in the group who continue to take the offending drugs, usually unaware that they are the cause of any unsteadiness. Vestibulotoxic drugs such as gentamicin or streptomycin, which cause permanent damage to the labyrinth, also produce this problem.

There are many central nervous system lesions producing chronic vestibular inadequacy but, usually, these are accompanied by other neurological symptoms or signs which indicate the nature of the problem and point to the site of the lesion. Large unilateral or smaller bilateral acoustic neuromata fall into this group.

There is a large group of patients whose dizziness is thought to be psychogenic and who have in the past been categorized as 'floating females'. These patients need not
necessarily be female! On examination and caloric testing they are normal so that they are not, technically, suffering from any identifiable vestibular inadequacy. However, the unsteadiness may persist for months. Remarkably, this rarely interferes with everyday activities.

Table 18.9. Vestibular inadequacy

The elderly
Drugs
  metabolic effect, eg, anticonvulsants
  destructive effect, eg, gentamicin
Central nervous system lesions
'Floating females'
Toxic products from chronic middle ear infections.

Head trauma

Head trauma causes such a diversity of vertigo that it is an aetiological factor in many of the tables. The six different causes of vertigo following head trauma are listed in Table 18.10.

Table 18.10. Vertigo following head injury

Post-concussional syndrome
Benign paroxysmal positional vertigo
Destructive labyrinthine lesion
Perilymph fistula
Delayed endolymphatic hydrops
Functional.

The principles of treatment

In the management of vertigo there are certain basic principles which can be applied to most conditions. Once enumerated they appear to be basic common sense, but then that applies to much of medicine.

Treat or eliminate the cause

Treating or eliminating the cause is a perfectly obvious way of dealing with any problem. John Masefield knew as well as anyone that those who do not go out on the sea do not suffer from seasickness, and those who stay at home and do not go down to the sea, do not suffer from car sickness!

Unfortunately, many of the causes of vertigo are either untreated with our present knowledge or irreversible by the time the patient presents and consequently this mode of management is not always applicable.
Suppress the vestibular system

In labyrinthine vertigo, the sensation of dizziness is due to a disproportion between the activity in the two sets of vestibular nuclei. In an acute labyrinthine destructive lesion, the equal and opposite activity in the vestibular nuclei is lost. In an effort to correct this, the cerebellum imposes an attempt at a shut down of electrical activity in the vestibular nuclei, the so-called 'cerebellar clamp'. This reduces the disproportion between the two sides. This can be augmented by the use of labyrinthine sedative drugs including cinnarizine, cyclizine, dimenhydrinate and prochlorperazine. These drugs are of particular value in acute situations, not only reducing dizziness but also exercising an antiemetic effect. Diazepam also is a potent labyrinthine sedative drug and has, in addition, anxiolytic properties. However, if the problem is already due to vestibular inadequacy, as in the elderly, these labyrinthine sedative drugs will simply increase the unsteadiness.

Suppress the patient's emotional reaction

Vertigo is a most distressing symptom especially if it is accompanied by vomiting and is occurring in a random and unpredictable manner. There are few patients who do not develop some emotional reaction to severe, episodic labyrinthine vertigo. This may result in aggravation of the symptoms as it may disturb even further the dysequilibrium between the two sets of vestibular nuclei.

These patients need strong reassurance both about the nature of their dizziness and the prognosis. In addition, a short period of suppression of the emotional reaction with a mild tranquilizer may be desirable. However, it is important to be alert to the possibility that the central effects of tranquilizers, especially in high dosage, can aggravate the dizziness.

Wait for compensation

Nature has a marvellous way of compensating for any imbalance, not least in the activity of the vestibular nuclei. In the early stages there is the 'cerebellar clamping' of the activity of the vestibular nuclei. In time, spontaneous activity is generated in the affected vestibular nuclei with gradual restoration of comfortable balance. Thus, following a labyrinthectomy in a young person, the simple passage of time may result in a return of balance function which is normal for most everyday activities. In peripheral lesions, time tends to be on our side. Unfortunately, this is not so common with the dizziness due to lesions of the central nervous system. In many instances the process of compensation can be accelerated by the performance of Cawthorne-Cooksey exercises.

Eliminate the offending labyrinth

Although nature can compensate readily for the complete loss of function of a labyrinth, it may have some difficulty in compensating for incomplete loss and will have considerable difficulty in compensating for fluctuating loss. Labyrinthectomy, or vestibular nerve section, may be the treatment of choice in any condition where labyrinthine function is fluctuating or where nature has failed to compensate, despite the passage of an adequate length of time. Unfortunately, labyrinthectomy always, and vestibular nerve section occasionally, results in loss of all auditory function in the affected ear.
Acceptance of the problem

There are some conditions where, despite the application of the appropriate methods of management outlined above, there is persisting imbalance. It is desirable that the physician should understand the prognosis and, where appropriate, explain to the patient that some degree of imbalance will have to be accepted. With the explanation, appropriate aids should be recommended. A walking stick is the first and most obvious but it is often overlooked. Very often this is all that is required to restore the patient's confidence and enable the resumption of reasonably normal everyday activity. In severe cases a walking frame and strategically placed hand rails will be needed.

Surgery for vertigo

It is only occasionally that surgery has any part to play in the management of vertigo. Most conditions where surgery is indicated have been discussed elsewhere in this volume.

In Ménière's disease, surgery may be seen as first, correcting the underlying problem, for example saccus decompression or cervical sympathectomy; second, having a metabolic or even placebo effect, for example saccus decompression or 'cortical mastoidectomy'; or third, ablating all vestibular function in an offending labyrinth, such as vestibular neurectomy or labyrinthectomy.

The management of labyrinthine fistulae is discussed in the section on chronic suppurative otitis media (see Chapter 12). Active chronic suppurative otitis media may be associated with vague imbalance, even in the absence of a labyrinthine fistula. It is possibly due to the absorption of toxins. Although the mechanism is obscure, the alleviation of the unsteadiness is often dramatic when the infection is dealt with by either open or closed cavity techniques.

When a labyrinthectomy is carried out in the presence of an open cavity, there may be persisting imbalance, thought to be due to stimulation of vestibular nerve endings. Subsequent vestibular nerve section may be necessary to control this unsteadiness.

Any incomplete destructive lesion of the labyrinth may be followed by persistent vertigo. In some cases this may be due to delayed endolymphatic hydrops. A complete drill-out labyrinthectomy, or even a vestibular nerve section, may be required.

Perilymph fistulae are discussed in Chapter 7.

The surgery of benign paroxysmal positional vertigo deserves special mention as this is not considered elsewhere. This condition tends to be self-limiting and is only rarely sufficiently severe to warrant surgery. However, in the small proportion of patients whose daily life has become crippled by dizziness, or whose symptoms have persisted for some years, surgery may be indicated.

Gacek (1985) reported section of the posterior ampullary nerve and this is now the operation of choice. In this procedure, the posterior ampullary, or singular nerve, is exposed via the middle ear. The bone overhanging the round window membrane is removed. Drilling
of the bone inferior to the round window membrane will expose the nerve at a depth of 1-2 mm. The nerve is divided with a hook and the canal sealed with absorbable gelatin sponge. Unfortunately in a small proportion of patients the nerve is inaccessible under the basal turn of the cochlea.

The main risk of this procedure is sensorineural hearing loss, which occurs in about 10% of patients. There is also the risk of opening into the labyrinth, causing persistent unsteadiness. Very infrequently, a cerebrospinal fluid leak may occur from the singular canal. This is a difficult procedure which is only rarely indicated, but, in competent hands, produces excellent results.