Chapter 7: Ear trauma

A. G. Kerr and G. D. L. Smyth

Trauma to the ear is on the increase. Society is becoming more violent, urban terrorism with guns and explosives is more widespread, and there are increasing numbers of road and other accidents. In addition, the improved management of those with severe injuries is contributing to the number of patients surviving to require treatment of aural trauma.

The lesions may range from simple blunt trauma to the pinna, without loss of tissue, through simple rupture of the tympanic membrane to transverse fracture of the petrous temporal bone with complete loss of inner ear and facial nerve function.

External ear

Traumatic lesions of the outer ear are discussed in Chapter 6.

Tympanic membrane and middle ear

Traumatic perforations of the tympanic membrane

Rupture of the tympanic membrane may be caused by changes in air pressure, by fluids or by solid objects.

Pathogenesis

Air pressure changes

Although gradual changes in air pressure may result in rupture of the tympanic membrane, this most often occurs from a rapid change. Sudden forceful blows on the ear, which seal the external auditory meatus, can result in sufficient increase in the air pressure in the ear canal to rupture the tympanic membrane. The most frequent single cause is a blow on the ear, usually with the open hand. This may occur in sporting and not so sporting situations. A blow on the ear from a ball or a fall on water are not uncommon causes of trauma to the tympanic membrane.

Blast injury of the ear commonly, and barotrauma uncommonly, may cause damage to the tympanic membrane. Blast injury is considered later in this chapter and barotrauma in Volume 1, Chapter 7.

Eustachian tube inflation, either by the Valsalva manoeuvre or by the use of a catheter, rarely results in perforation of a normal, healthy tympanic membrane, but can rupture one weakened by previous disease. Similarly, pressure changes in the middle ear as a result of nitrous oxide anaesthesia may result in rupture of a weakened tympanic membrane. There have been reports of lighting, with its associated air pressure changes, resulting in perforation of the tympanic membrane.
Perforations due to air pressure changes occur most commonly in the anteroinferior quadrant of the tympanic membrane. It is unlikely that they ever cause perforation of the pars flaccida.

**Fluid**

When syringing an ear, it is important to ensure not only that the fluid is at body temperature, but also that the full force of the jet is not directed onto the tympanic membrane. By directing the jet onto the posterior meatal wall the likelihood of rupture of the tympanic membrane is considerably reduced.

Caloric tests in patients with gossamer-thin tympanic membranes must be undertaken with considerable caution.

In skin-diving, it is possible for perforation of the tympanic membrane to occur not only from air pressure differentials but also from fluid pressure in the ear canal.

**Solid objects**

Although the usual foreign bodies occurring in children rarely rupture the tympanic membrane, attempts at their removal may do so. Matchsticks and hair clips, used to remove wax or relieve itching in the ear canal sometimes cause damage to the tympanic membrane. (It is remarkable how many people stand just inside a door to scratch the ear canal!) Sparks of hot metal, especially in welders, can perforate the tympanic membrane by burning through it. The tympanic membrane may fail to heal after extrusion of a middle ear ventilation tube.

**Management**

Traumatic perforations often occur in the healthy members of the community; generally the prognosis is excellent. The two main factors leading to failure of the perforation to heal are loss of tissue and secondary infection. Welding injuries usually result in both loss of tissue and secondary infection; water sports injuries frequently result in the latter. Consequently, the prognosis for spontaneous healing is reduced in both.

Small perforations are more likely to close spontaneously than large ones. Nonetheless, in the majority of traumatic perforations, the membrane usually heals and the function of the ear returns to normal.

The most effective management is to do nothing. Because of the risk of introducing infection, the ear should *not* be cleaned out unless contaminating material is found in the meatus or there is evidence of active infection. Antibiotic ear drops, in the absence of infection, are of no value and may well introduce organisms. Systemic antibiotics should also not be prescribed in the absence of overt infection unless there is good reason to believe that the ear has been contaminated. The ear must be kept dry.

There are many advocates of an active approach to the tympanic membrane following trauma. They recommend examination under a microscope with eversion of the edges of the perforation. This approach is reasonable so long as it is carried out by a competent person...
under aseptic conditions and, in fact, could be regarded as the ideal. Inverted edges are
certainly undesirable and if infection is not introduced, it is unlikely that any harm will result
from this procedure. However, immediate surgical repair with grafting is not indicated
because of the excellent prognosis for spontaneous recovery. Even subtotal perforations often
heal with an excellent end result.

If the perforation fails to close spontaneously in 3-6 months, surgical closure is
indicated. Special reference should be made to welding injuries. Not only is the perforation
unlikely to close spontaneously but the results of surgical closure are disappointing. In
addition to the loss of tissue and secondary infection, it is likely that there is also some
avascularity of the tissues with dense avascular scarring secondary to the burn.

Complications

The most common complication of a traumatic perforation is secondary infection of
the middle ear. The development of squamous epithelial cysts in the middle ear has been seen
following perforations caused by blast and is due to implantation or inversion of such tissue.
Depending on the force of the injury causing the perforation, there may be ossicular
displacement with conductive deafness, or inner ear damage with sensorineural deafness and
tinnitus.

Squamous epithelial invasion of the middle ear is a rare but recognized complication
of middle ear ventilation tubes, especially when there is a persisting perforation after
extrusion.

Blast injury

Explosive material changes suddenly from solid to gaseous form with a massive
increase in volume and pressure, resulting in a blast wave spreading outwards from the seat
of the explosion. There is a short-lived positive pressure phase, usually of the order of a few
milliseconds and a longer and less marked negative phase, always less than atmospheric
pressure and of the order of tens of milliseconds. The amount of energy in each phase of the
wave is approximately equal. The front of the blast wave is irregular and damage may be
cauased in a capricious fashion.

The factors influencing such damage are:

(1) the rise time, that is the speed with which the pressure builds up

(2) the intensity of height of the peak pressure

(3) the duration of the positive pressure wave

(4) the site of the explosion and the presence of objects which may reflect or deflect
the blast wave.

Some people close to the bomb escape ear damage while others, further away, may
be severely deafened.
Exposure to blast can result in damage to both middle and inner ears. There may be hyperaemia or even subepithelial bleeding in the tympanic membrane. Perforation due to the blast occurs in the pars tensa and the ear facing the bomb tends to be more seriously damaged. However, reflection of the blast wave from a wall, with augmentation of the pressure, may reverse this situation.

Perforations are probably caused by the positive phase of the blast wave. This conclusion is reached by the demonstration of squamous epithelium in the middle ear in post-mortem specimens and by the occasional finding of epithelial pearls in the middle ear. The latter are also sometimes seen in tympanic membranes which have healed spontaneously. Although everted edges are often seen following blast injury, these are probably caused secondarily by the suction effect of the negative phase.

Most blast injuries of the tympanic membrane heal spontaneously with conservative treatment. Kerr and Byrne (1975) reported spontaneous healing of 83% of 66 perforated tympanic membranes from one explosion.

### Radiation injury

The ear is at risk in radiotherapy of any lesion involving the ear itself or in the nasopharynx. The development of middle ear effusion is common in these patients. Osteoradionecrosis has been reported. The management of both these conditions is influenced by the success or otherwise of the radiotherapy in controlling the original malignancy.

There is some circumstantial evidence that sensorineural loss may develop. However, the authors are not aware of any controlled prospective study confirming such damage and, without this, any aetiological connection between radiotherapy and sensorineural deafness must remain speculative.

### Surgical trauma

#### The chorda tympani nerve

In theory, disorders of taste and salivary secretion should follow every instance of surgical trauma to this structure. In practice, although the nerve is frequently stretched, manipulated, dehydrated and even cut, dysgeusia is an uncommon complaint after stapedectomy and hardly ever occurs following tympanoplasty.

Notwithstanding this, because disorders of taste do occur following ear surgery and can considerably diminish the pleasures of the table, sometimes for many months, the surgeon should always handle the chorda tympani with care.

#### The jugular bulb

This structure is occasionally dehiscent in the posteroinferior quadrant of the mesotympanum. In these cases, it is at risk when the fibrous annulus is being elevated from the sulcus tympanicus in creating a tympanomeatal flap during stapedectomy or transcanal tympanoplasty. Brisk venous bleeding at this stage can usually be controlled by promptly
replacing the tympanic membrane and its annulus and applying a small pack. If the bleeding area is then avoided, it is usually possible to complete the procedure with only limited inconvenience.

The facial nerve

In all middle ear surgery, especially for chronic suppurative disease, the safety of the facial nerve will depend upon knowledge of several important landmarks.

In a mesotympanum which is completely filled with granulations or cholesteatoma, it is best to find the landmark which is more resistant to disease than all others - the eustachian tube. From there, it is safe to dissect posteriorly over the promontory as far as the grooves for the tympanic plexus. These grooves can then be followed superiorly to the base of the cochleariform process which marks the junction of the labyrinthine and tympanic segments of the nerve. When the process has been destroyed, a useful alternative guide is the belly of the tensor tympani muscle, which is usually exposed in such cases.

Dissection can then proceed posteriorly following the osseous canal of the tympanic segment of the nerve. In transcanal tympanoplasty, if any doubts develop about the anatomy of the seventh nerve, it is always wise to increase exposure of the area by converting the procedure into a combined approach tympanoplasty. This permits dissection inferiorly over the medial epitympanic wall and the anterior half of the lateral semicircular canal, exposing the tympanic segment of the nerve from its superior aspect where its bony covering is least likely to be deficient. Once the characteristic pink, rounded bone overlying the nerve is compared with the ivory-white labyrinthine bone, the position of the nerve will be apparent, and dissection can proceed over it in all directions.

It should be remembered that there are several possible abnormalities of the facial nerve in this region which may give rise to confusion and possibly disaster if there are not known. The most common of these is the facial nerve which is overlying the footplate of the stapes. For this reason, it is important to identify the nerve positively in its normal horizontal canal before removing soft tissue from the surface of the footplate.

In the mastoid segment, the following landmarks are of service: the lateral semicircular canal, the fossa incudis, and the digastric ridge. The posterior semicircular canal lies on the medial aspect of the mastoid segment of the nerve. In performing a mastoidectomy or combined approach tympanoplasty, the first landmark is the mastoid antrum. Korner's septum may give rise to confusion and lead the surgeon in an inferior and anterior route into the mastoid segment of the facial nerve. This can be avoided by:

(1) awareness of the risk

(2) noting the level of the tympanic membrane as compared to the medial wall of the 'false antrum'.

In combined approach tympanoplasty, posterior tympanotomy is performed by cutting a groove downwards from the tip of the short process of incus towards the mastoid tip. The groove should be parallel to the expected course of the facial nerve, and the bone should be
thinned gradually with diamond burrs so that the nerve can be seen before it is uncovered. Bleeding from the nerve sheath is often an excellent warning sign. Once the facial sinus has been entered, further enlargement of the posterior tympanotomy is carried out by removing bone inferiorly and laterally. For this, the position of the chordal eminence and the chordal ridge should be known and understood. Exposure of the hypotympanum requires removal of the styloid eminence.

Abnormalities of the facial nerve rarely occur in this area but they should be known. The most common of these is the nerve that passes posteriorly, but bifid facial nerves below the genu have been reported. The digastric ridge and the position of the chorda tympani nerve can be used as guides to the nerve when required.

**Treatment**

Surgical trauma to the facial nerve in temporal bone surgery may result in loss of continuity and loss of substance. In these situations, direct anastomosis does not necessarily produce the best result and it is now agreed that grafting with a piece of the great auricular nerve is the preferred method in most instances.

Grafting should be delayed for 3 weeks after the injury by which time axoplasmal regeneration is optimal. The results are better when tension can be avoided and a grafting technique is carried out with the use of magnification. Excessive proliferation of connective tissue in an anastomotic area can be reduced by the removal of several millimetres of epineurium from the stumps. The ends of the stumps should be approximated after they have been cut in an oblique direction in order to increase the surface areas in contact. Foreign body reaction with connective tissue proliferation is reduced by the avoidance of suturing material. The natural self-adherence of the nerve tissue is often sufficient to retain the position of the nerve graft in the mastoid and tympanic segments. If there is any doubt about the stability of the graft, however, one or two 8/0 monofilament nylon sutures should be inserted.

In conclusion, avoidance of facial nerve trauma in advanced chronic middle ear disease will be enormously enhanced by the recognition of important landmarks such as the processus cochleariformis, the lateral semicircular canal and the oval window. The great importance of surgical skill, anatomical knowledge and awareness of variations in normal anatomy, were summed up by Fowler (1961) when he said: ‘Although traumatic facial palsy is more likely to occur when a surgeon is inexperienced, it can and does occur with the most skilful and experienced otologic surgeon, especially when the course of the nerve is anomalous’.

There is a natural tendency to want to terminate the operation once one is aware of having damaged the facial nerve. This should be resisted. The damage has been done and the patient's interests are not furthered by dealing inadequately with the original disease. The patient has already paid a high price. It is all the more important that the original surgical objectives be attained.
Temporal bone trauma

Fractures

The clinical features of fractures of the temporal bone can only be understood by considering their anatomy and pathology. Fractures involving the temporal bone can be classified into longitudinal, transverse and mixed, depending on the relationship of the fracture line to the long axis of the petrous temporal bone. In many, the lesion is confined to the squamous temporal bone, in which case it could be regarded as an incomplete or partial longitudinal fracture. Often the diagnosis is made purely on clinical grounds as the fractures do not always show on routine skull radiographs. Rarely, the diagnosis is delayed until the appearance of discoloration of the skin over the mastoid (Battle's sign).

Pathology

Longitudinal fractures

Most (80%) temporal bone fractures are longitudinal and usually result from blows to temporal or parietal areas (Proctor, Gurdjian and Webster, 1956). The fracture begins in the squamous temporal bone and extends along the roof of the bony external auditory meatus, tearing the meatal skin and the tympanic membrane and, crossing the roof of the middle ear, reaches the petrous temporal bone. If then runs anterior to the labyrinthine capsule, through the carotid canal, to end near the foramen spinosum or foramen lacerum.

Damage to the skin of the external auditory meatus and the tympanic membrane result in bleeding from the ear. In the absence of any other obvious cause, bleeding from the ear following a head injury can be presumed to indicate a fracture of the base of the skull, usually longitudinal, despite negative X-ray findings. Displacement of the bone is very rare but a gap may be present at the fracture line.

Middle ear structures are always involved in longitudinal fractures but, in most cases, this is not serious and healing occurs spontaneously without residual conductive deafness. However, should there be persistent conductive deafness, the possibility of ossicular dislocation must be considered.

Because a longitudinal fracture usually runs anterior to the hard bone of the labyrinthine capsule, only rarely is the inner ear directly involved, but there may be concomitant inner ear concussion with high tone sensorineural hearing loss. Although the underlying damage in the sensorineural hearing loss of longitudinal fractures is probably more often to be found in the cochlea itself, experimental and clinical evidence has been presented to suggest that some of the deafness is central in origin, secondary to neural damage (Makishima, Sobel and Snow, 1976).

Facial nerve injuries are uncommon in longitudinal fractures and when they occur, are usually delayed in onset.
Transverse fractures

Transverse fractures usually result from frontal or occipital blows and account for approximately 20% of temporal bone fractures (Proctor, Gurdjian and Webster, 1956). These patients usually suffer more severe neurological injury than those with longitudinal fractures.

The fracture line extends transversely across the petrous pyramid, passing through the vestibule of the inner ear. Although the fracture can be demonstrated radiologically in about 50% of cases, the diagnosis is essentially clinical. In a pure transverse fracture there is a haemotympanum but, as the tympanic membrane is not damaged, there is no bleeding from the ear. The severe general injuries of the patient may dominate the clinical picture but, if sought, there is evidence of severe or complete sensorineural deafness on the affected side, usually accompanied by tinnitus. The deafness is permanent.

Very severe rotatory vertigo with nausea and vomiting, due to severe damage to the vestibular apparatus on the affected side, occurs initially. Nystagmus is usually present with the fast component to the opposite side. Unfortunately, the significance of the severe dizziness, vomiting and nystagmus, is often missed by those responsible for the management of the head injury. It may not become apparent that the patient has suffered vestibular damage until he is allowed out of bed a week or two after the injury. The patient is then surprised to find that he is extremely unsteady and unable to walk without support. Central compensation develops in the subsequent weeks and months.

When the fracture line involves the vestibular aqueduct, delayed secondary endolymphatic hydrops may develop (Rizvi and Gibbin, 1979).

Facial nerve injuries occur in about 50% of these patients and the onset is usually immediate.

Mixed fractures

In severe head injuries there may be a combination of longitudinal and transverse fractures.

Management

The importance of avoiding the introduction of infection into the middle ear cannot be overemphasized and, unless there are signs of active infection, it is better to leave untouched any blood clot in the external auditory meatus. A cerebrospinal fluid leak must be treated by a sulphonamide and an antibiotic. The management of the head injury usually takes precedence and indeed it may be some days before the otolaryngologist is asked to see the patient.

Cerebrospinal fluid leak

Most cerebrospinal fluid leaks close spontaneously within 7-10 days. If this does not occur, more active treatment will be necessary. The introduction of a spinal drain at this time may well be all that is required to allow the leak to close but many neurosurgeons are
unhappy about this approach. They feel that, although closure may occur at the time, some leaks will recur later or, even without a recurrence of the leak, there may be an ascending intracranial infection from a subsequent otitis media. If the leak is profuse or if it fails to close promptly, with or without spinal drainage, exploration and surgical closure are indicated. These leaks usually arise from the middle cranial fossa and the help of a neurosurgeon is desirable. The middle cranial fossa is explored, the dura elevated and, after exposure of the tear, the defect is covered with a graft of fascia lata or temporalis fascia.

Some otologists, perhaps chauvinistically, recommend a mastoid approach to this problem. Undoubtedly this is possible and the operation may be shorter and less major. However, access to the site may be impaired and the positioning of the graft is less secure.

**Meatal damage**

Tears in the meatal skin may heal with the formation of fibrous bands in the depths of the meatus, resulting in pockets which collect epithelial debris. If these cannot be cleaned adequately and effectively via the meatus, or if repeated cleaning will be necessary over many years, surgical removal of these bands, perhaps with grafting of the tympanic membrane, may be required.

A wide fracture line predisposes to invasion of the middle ear cleft by squamous epithelium and cholesteatoma development has been reported. However, the vast majority of cholesteatomata diagnosed for the first time after a head injury have not been caused by the trauma. Medico-legal problems can arise but in the absence of a wide fracture line, and especially with a sclerotic mastoid, the cholesteatoma can reasonably be presumed to have been present before the injury. The degree of pneumatization of the temporal bone is usually a guide to the pre-existence of the cholesteatoma. When a cholesteatoma occurs, secondary to a fracture, in a well pneumatized temporal bone, it can rapidly become very extensive and subsequent surgical control may be difficult.

**Deafness**

The conductive deafness which follows a longitudinal fracture is, in most instances, due to a tear in the tympanic membrane and blood in the middle ear and it usually recovers spontaneously. Failure to regain normal middle ear transmission is usually due to ossicular dislocation or the formation of adhesions. Ossicular damage can also result from a head injury in the absence of a skull fracture.

The most commonly affected ossicle is the incus, as the malleus and stapes are relatively more stable; the most frequently found defect is a dislocation of the incudostapedial joint (Hough, 1970). All other lesions are uncommon. These include fracture of the stapedial crura, dislocation of the stapes footplate, total dislocation of the incus, dislocation of the malleus, fracture of the malleus handle, fixation of the malleus head in the epitympanum by fibrous tissue or bone and total destruction of the ossicular chain. Delayed necrosis of the long process of the incus has been described.

In these cases there is usually a conductive deafness with an air-bone gap in the region of 40 dB. Exploration of the middle ear is indicated. Ossiculoplasty is carried out on the
general principles of tympanoplasty, usually with better results than in chronic suppurative otitis media.

The sensorineural deafness caused by head injuries is, unfortunately, untreatable. Although there may be some spontaneous recovery of the high tone sensorineural loss that often accompanies longitudinal fracture, there is no likelihood of recovery of any useful hearing following transverse fractures.

Labyrinthine damage can occur without any clinical or radiological evidence of temporal bone fracture. In these cases it is presumed that labyrinthine concussion is responsible for any associated auditory or vestibular symptoms, although there is the possibility of underlying damage in the brain. The deafness usually affects the high frequencies.

Generally speaking, an injury insufficiently severe to cause loss of consciousness does not damage the hearing. However, this is not always the case and difficult medico-legal problems can arise. These are usually settled on the basis of circumstantial evidence such as when the patient first noticed the deafness and the problems that he describes. For example, the damage due to trauma is usually immediate and a severe hearing loss, first noticed 6 months after the injury, cannot reasonably be attributed to that injury.

**Vertigo**

Vertigo is common following head injuries and, as with deafness, can occur without a skull fracture. The most common form is that associated with the post-concussional syndrome. These patients have vague unsteadiness, especially when getting up from sitting, and usually associated with frequent severe headaches. The unsteadiness generally settles in a matter of 6-12 months and when it is prolonged beyond this time, the question of a post-concussional neurosis must be considered.

Following transverse fracture of the temporal bone, there is severe incapacitating vertigo making it impossible for the patient to walk unaided for a length of time which varies from 1 to 4 weeks, depending on factors such as age, motivation and other injuries. In the immediate period after a head injury the symptoms can be relieved by labyrinthine sedative drugs. There is a slow but gradual improvement; young patients recover to fairly normal balance in a matter of weeks and elderly patients in months. However, the convalescence is often complicated by other injuries and, especially in the elderly, associated brain damage may prevent full compensation.

Benign positional vertigo may follow as a complication of head injuries, with or without fracture of the temporal bone. Schuknecht (1969) has postulated that this results from damage to the utricle with destruction of the otootic membrane, the otoconia of which become adherent to the cupula of the posterior semicircular canal. This is then stimulated by movement of the head, especially when the affected ear is placed undermost. The dizziness is short-lived, associated with transient and fatiguable rotatory nystagmus, and always precipitated by head movement; between these induced episodes the patient is perfectly steady. This is usually a self-limiting condition, although it often takes up to 2 years before the vertigo settles.
When the vestibular aqueduct is involved, delayed secondary endolymphatic hydrops may develop. In the unlike event that there is any remaining vestibular function this can result, years later, in episodic rotatory vertigo, similar to that seen in Ménière's disease (Rizvi and Gibbin, 1979).

Perilymph fistulae may also be a cause of post-traumatic vertigo and will be discussed later.

**Facial paralysis**

Facial paralysis following fracture of the temporal bone is classified broadly into two groups - immediate and delayed. Immediate paralysis usually indicates tearing of the facial nerve, impaling of the nerve by bone, or entrapment in a fracture line. Early surgical exploration is indicated if there is to be any reasonable prospect of good functional recovery.

Delayed onset of facial paralysis confirms that, anatomically, the facial nerve is intact, and that there has not been any direct gross trauma. The management of delayed traumatic paralysis is similar to that of idiopathic facial paralysis.

**Penetrating injuries of the temporal bone**

Injuries from bullets, missiles, and explosions may result in lesions involving any part of the body. When the temporal bone is involved, almost any lesion can occur. In most cases, the other injuries predominate and it may be some time after the injury before the otolaryngologist is asked to see the patient.

The lesions of the temporal bone are difficult to classify because of their variability; the management of each patient depends on the specific circumstances. Often the other injuries necessitate compromise in the otological management.

Patients have been reported where gunshot has remained in the temporal bone for many years without any complications. In one patient, the tympanic membrane was largely destroyed, gunshot remained in the middle ear cleft and brain herniated through a damaged tegmen tympani into the attic (Kerr, 1967). The patient lived for 40 years after the injury and died from other causes. Despite such cases, if there is gunshot in the middle ear cleft, with the possibility of infection, surgical exploration and removal are indicated. If, on the other hand, the gunshot has been adequately buried for some time, without any infection or likelihood of infection, and is not causing symptoms, action is not required.

**Whiplash**

The term 'whiplash injury' used in an unpublished paper in 1928, was first recorded in 1945 and has been a source of controversy ever since. Many object to the name but all agree that it implies an acceleration-extension injury of the neck; some also include deceleration injuries and forward or lateral flexion movements in this syndrome. The diverse symptomatology and prolonged litigation that follows these injuries has led to considerable scepticism about this condition. Nonetheless, it has been shown that many patients continue to have symptoms, sometimes disabling, related to whiplash injuries, not only when other
simultaneous severe injuries have become symptom-free, but even years after litigation has ended and compensation has been paid (Gotten, 1956). While symptoms can occur following forward and lateral flexion injuries, the vast majority arise from the acceleration-extension injuries which occur in rear-end collisions. In many instances the initial injury seems trivial, with severe pain in the neck developing only some hours later.

**Clinical features**

It is not uncommon for the passengers in a car involved in a rear-end collision to be entirely symptom-free immediately after the injury. However, during the succeeding minutes or hours, an ache begins to develop in the neck which increases within a short time to severe pain. Although the most common complaint is pain in the neck, the otolaryngologist becomes involved when the symptoms include dizziness, tinnitus, deafness and dysphagia.

Typically, the onset of dizziness does not occur until some days after the injury. The symptoms tend to be diverse but the most prominent is a generalized sensation of unsteadiness which increases, and may take the form of rotatory vertigo, in association with certain head and neck movements. Routine clinical examination is frequently unremarkable, although positional nystagmus may be demonstrated. However, with the aid of electronystagmography, nystagmus may be demonstrated even when it is not present on clinical examination. Published reports on the findings in the caloric test frequently refer to abnormalities but, unfortunately, many of these do not eliminate those patients with head injuries and, furthermore, do not include controls. However, it is the authors' view that the weight of evidence is that abnormal electronystagmography and caloric tests are often seen following uncomplicated whiplash injury.

There are four theories to explain these features. First, the problem may be neuromuscular with abnormal proprioceptive impulses causing the dizziness. Second, it may be neurovascular with abnormality of the cervical sympathetic nervous system. Third, there may be a mechanical vascular problem with kinking of the vertebral artery. Fourth, brain damage may occur as a result of the whiplash injury.

The typical history is of injury followed by a short symptom-free period before the development of pain in the neck. During the next week or so dizziness develops and may persist for years. Although this settles in the vast majority, in Gotten's (1956) series it persisted in 12% for up to 2 years after litigation has ended.

Tinnitus is a frequent complaint in the early stages. It is unlikely that there is any direct damage to the ear. The tinnitus may be due to a concussive effect on the brain; alternatively, and in the authors' opinion more probably, the stress of the injury may result in unmasking of potential tinnitus due to pre-existing sensorineural deafness.

Although deafness has been reported in some publications on this subject there is little substantiating evidence. It seems likely that deafness does not occur in the absence of an associated head injury and that whiplash injuries, of themselves, do not result in hearing loss.
Treatment

In the early days of the recognition of the clinical entity of acceleration injuries of the neck, they were most commonly the result of catapult-assisted take-offs from aircraft carriers. The injury was prevented by extending the back of the seat so that the pilot's head was supported. Many modern car seats are now similarly designed and the widespread use of head restraints should reduce the incidence of this syndrome.

Whiplash injuries are best treated during the acute phase. If the nature of the injury and the patient's symptoms suggest this condition, immediate and adequate splinting of the neck is required, accompanied by bed-rest to relieve the neck of the weight of the head. Macnab (1971) has pointed out that the persistent complaints following acceleration-extension injuries do not occur in side collisions where lateral flexion is the predominant lesion. Similarly, wrists and ankles, injured in the same accident become symptom-free long before neck symptoms settle down. He has suggested that the persistence of symptoms in these patients is not because these people are by nature litigious but that the initial injury is inadequately treated. He emphasized that if the neck needs to be splinted, it needs to be done adequately and to have the weight of the head removed from it by bed-rest. Unless the patient is relatively symptom-free within 24 hours he recommended bed-rest for one week. The time for a collar is now and not 6 months later. Heat and massage may make the patient feel more comfortable but probably do nothing to speed resolution of the underlying lesion.

Treatment in the chronic phase is difficult. If the patient appears to be developing disabling symptoms due to functional overlay, do not over-treat or over-investigate. There is no evidence that prolonged immobilization is of benefit at this stage, or that heat and massage do more than imprint the symptoms on the patient's mind. Neck traction and muscle strengthening exercises may be of benefit but must not be instituted until it has been established by flexion and extension radiographs that there is no joint instability.

A frank and open discussion with the patient about the possibilities of disordered function is the best approach, once the chronic stage has been reached. The response from tranquilizers and labyrinthine sedative drugs is variable and unpredictable.

While many patients improve with time, especially after litigation has been settled, this is not always the case; do not be misled into thinking that all these patients are malingers.

Perilymph fistula

In a perilymph fistula, not to be confused with labyrinthine fistula, perilymph may leak from the inner into the middle ear. The leakage may occur either from rupture of the stapediovestibular joint, fracture of the stapes footplate or tearing of the round window membrane. Clinical reports indicate that these are more often from the oval than the round window. Typically, there is a history of a head injury, middle ear surgery or some other event associated with raised intracranial pressure, such as coughing, straining or exertion, or sudden changes in middle ear pressure.

The clinical features are variable but there are certain characteristics. The most common symptom is unsteadiness, usually with a marked positional element and often with
a disproportionate degree of ataxia. The dizziness due to direct trauma to the vestibular apparatus in a head injury usually improves dramatically over a period of weeks. On the other hand, the dizziness associated with a perilymph fistula tends to persist until the fistula is closed either by spontaneous healing or by surgery. The main differential diagnostic problem arises with benign positional vertigo.

Sensorineural deafness and tinnitus often occur in association with a fistula but are not constant features. The deafness may fluctuate.

A high index of suspicion is necessary for the diagnosis of this condition and one must look in the history for a predisposing cause.

Examination of the ear itself is usually unremarkable. The amount of perilymph leaking is usually small and one does not expect to see a fluid level in the middle ear. The fistula test is usually, but not always, negative. (This is not surprising since this test is for a third opening into the inner ear.) However, some authors have reported a positive fistula test in this condition.

Examination of the vestibular system may show positive Rombergism.

It is in positional testing that the main features are to be seen. Singleton et al (1978) have identified the following characteristics which differentiate the positional nystagmus from that in benign positional vertigo.

(1) There is either a short or no latent period.
(2) The nystagmus is not as violent as in benign positional vertigo.
(3) The duration tends to be longer with the nystagmus fatiguing slowly or not at all.
(4) The nystagmus rarely reverses direction when the patient is brought to a sitting position.
(5) The nystagmus does not necessarily beat towards the involved ear.
(6) The nystagmus is only occasionally rotatory.

Audiometry confirms a sensorineural hearing loss. Repeated testing, from day to day, may show minor degrees of fluctuation. Fraser and Flood (1982) have reported small improvements in the hearing after 30 minutes in the horizontal position, with the affected ear uppermost. The speech reception threshold and the speech discrimination scores may be depressed more than one would expect from the degree of sensorineural hearing loss.

**Diagnosis**

The diagnosis of this condition can only be made with certainty by surgical exploration of the ear. A perilymph fistula may be undiagnosed because it was not considered in the differential diagnosis, because the ear was not explored or because a fistula healed
spontaneously before exploration took place. On the other hand, false positive diagnoses may occur because of a failure to understand the anatomy of the round window niche at surgical exploration, because a serous middle ear exudate has been mistaken for perilymph, or because the surgeon has fulfilled his own predictions either by probing or drilling in the region of the round or oval window niches. Unfortunately, it is unlikely that there will ever be a reliable diagnosis of this condition until some foolproof method has been devised for confirming the presence of perilymph in the middle ear.

**Medical management**

If the diagnosis is made soon after the injury, bed-rest may be all that is required. The patient should be kept in bed for 5 days with the head of the bed elevated 30-40 degrees. Sedation and faecal softeners should be prescribed.

After 5 days, if the symptoms have settled, it is recommended that the patient continues to limit his activity for a further 10 days, still sleeping with elevation of the head of the bed and avoiding any exertion.

**Surgical management**

Surgical intervention is recommended if medical treatment fails or if the symptoms have persisted for over one month. After elevation of a tympanomeatal flap it is important to inspect the middle ear carefully before disturbing any of the middle ear structures. Trauma to the middle ear mucosa can, of itself, produce a serous ooze which may be mistaken for a perilymph fistula. If the procedure is performed under local anaesthesia and a fistula is not readily apparent, the Valsalva manoeuvre may help to identify a leak.

It is important to ensure that one does not create a fistula at this stage, thus establishing a self-fulfilling prophecy. Care must be taken in probing the stapes. The round window membrane can never be seen in its entirety without removal of the bone of the round window niche and, in many cases, can barely be seen at all without removing some bone. Once again, at this stage, there is a high risk of creating the fistula for which one is looking.

It is important to remember the anatomy of the round window membrane. It faces inferiorly and care must be taken that one does not confuse mucosal folds in the round window niche for the round window membrane. This is the explanation for some of the published reports which make statements such as 'the round window membrane was in tatters'.

Having identified a fistula, the area around it should be denuded of mucosa. In round window membrane fistulae it may be desirable to drill away the bony overhang. A graft, either of fascia or perichondrium, should be applied to the denuded area and packed in place with gelfoam. There is considerable doubt about the efficacy of fat (Seltzer and McCabe, 1986). In some cases of fistula in the oval window, especially with fracture of the stapes footplate, it may be desirable to carry out a total stapedectomy, seal the oval window with a fascial or perichondrial graft and place some sound conductor between the long process of the incus and the graft. This sound conductor may be the stapes itself, which is especially suitable with a thin graft, or it may simply be one of the prostheses used routinely in stapedectomy.
The results of surgery are frequently dramatic in alleviating the patient of his vertigo and ataxia. Unfortunately, the recovery of the sensorineural hearing loss is rarely so dramatic and, with regard to the hearing, the prevention of further deterioration can usually be considered to be a satisfactory outcome.

**Blast injury**

It has been said in the past that rupture of the tympanic membrane has a significant protective effect on the inner ear. This is probably not the case and, although it is difficult to prove, a survey has shown that sensorineural deafness is no less severe in those whose tympanic membranes have been ruptured (Kerr and Byrne, 1975).

There is still considerable doubt about the underlying pathology in sensorineural deafness secondary to blast. It tends to be most marked immediately after the explosion with a natural tendency to spontaneous improvement. There may be complete bilateral deafness just after the explosion but there is, in the present authors’ experience, always some recovery. Initially, the rate of recovery is rapid so that patients, unable to hear at all at the site of the explosion, are able to understand loud speech without difficulty one hour later. In some, the hearing may have returned to its former level within 48 hours, and in others, although there is permanent sensorineural deafness, this may continue to show slight improvement for up to 6 months.

In view of the tendency to rapid spontaneous recovery, it is difficult to control any trial of treatment for blast-induced sensorineural deafness. Numerous regimens have been advocated, often without much supporting evidence, including vasodilating drugs, corticosteroids, intravenous low molecular weight Dextran, anticoagulants and carbon dioxide inhalations. Other injuries may preclude some or even all of these forms of treatment. As there is doubt about the efficacy and as some of these treatments carry risks, it is preferable to leave untreated all cases of mild or moderate deafness and to reserve the multi-drug approach only for severe cases where there are no contraindications.

Tinnitus is a common complaint in those exposed to blast. The severity of the tinnitus tends to reflect the sensorineural deafness. Initially, the tinnitus may be severe and, although it persists as a big problem to some patients, it tends to decrease. When there is permanent sensorineural deafness the tinnitus may never disappear entirely but usually ceases to be a burden to the patient. In the uncomplicated case where the hearing returns to normal, one can expect complete disappearance of the tinnitus.

**Surgical trauma**

Injury to any of the structures which lie within the petrous bone is an inherent risk in every ear operation. Although it is certainly true that the introduction of microsurgical techniques in otology has been followed by a reduction in the incidence of surgical accidents such as dislocation of the stapes, opening of the labyrinth and damage to the facial nerve, nevertheless, the possibility of postoperative labyrinthine dysfunction after any operation on the ear, including myringoplasty, is still everpresent, even for the most experienced surgeon. Cochlear losses are reported more frequently than imbalance, but the two can occur together and indeed vestibular defects might be recognized more often were they routinely sought.
Although trauma to the facial nerve and major blood vessels is always possible if anatomical knowledge and surgical expertise are lacking, nowadays, the majority of surgical injuries to the ear affect the labyrinth and follow the creation of a fistula of the oval window, or hydraulic effects on the membranous inner ear. Cellular damage from infection, circulatory changes and alterations in the dynamics of the inner ear fluids are probably responsible for most of the functional damage which complicates microsurgery of the ear.

**Labyrinthine trauma in tympanoplasty**

In tympanoplasty the principal causes of cochlear loss are:

1. The removal of cholesteatoma matrix and granulations from a labyrinthine fistula, most commonly involving the lateral semicircular canal, in the presence of infection during an initial procedure. Rupture of the membranous labyrinth, or labyrinthitis, frequently result in total loss of inner ear function.

2. Incautious removal of granulations, tympanosclerosis or cholesteatoma from the oval window, with fracture of the stapes footplate or rupture of its annular ligament creating a fistula between the mesotympanum and the vestibule of the inner ear. Prolonged perilymph loss or labyrinthitis may follow.

3. Excessive movement of the stapes footplate while removing disease from the oval window or from any part of an intact ossicular chain. Similar risks also pertain during reconstruction of the transmission mechanism and the tympanic membrane.

4. Contact between a toothed rotating burr and any part of an intact ossicular chain (most commonly the body of the incus) in combined approach tympanoplasty. Extensive hair cell damage may result (Paparella, 1962).

In a series of 1680 chronic ear operations, Palva, Karja and Palva (1973) reported a 4.5% incidence of sensorineural deafness after operation, mainly limited to the frequency range 4000-8000 Hz. In 81% the ossicular chain was maintained intact throughout the operation. Smyth (1977) reported various degrees of sensorineural hearing loss in 2.5% of 3000 tympanoplasty operations.

Apart from the special risk of cochlear trauma in combined approach tympanoplasty from contact of the burr, transmitted through the intact ossicular chain, which occurred in 5.6% of such ears, labyrinthine trauma did not appear to be related to any particular surgical technique. However, it should be noted that 1.3% of all myringoplasties (transcanal tympanoplasty with an intact chain) were complicated by a depression of cochlear function of greater than 10 dB averaged through the frequencies 500-4000 Hz, or a greater than 10% loss in speech discrimination score.

Trauma arising from the removal of diseased tissue from the isolated stapes was considered to be responsible for one-third of the casualties; of these, in one-third the dissection of tympanosclerotic plaques had been noted to cause overmanipulation or fracture of the stapes footplate. In another third of the damaged inner ears in this series, the cause
appeared to be excessive movement of the stapes footplate during attempts to reconstruct the ossicular chain.

More recently, Tos, Law and Plate (1984) analysed the incidence and characteristics of postoperative sensorineural hearing loss in chronic ear surgery performed in 2303 ears. Sensorineural hearing loss occurred in a total of 1.2% of cases; 0.5% became totally deaf and 0.7% acquired a high tone loss, most often at 4 kHz only. The incidence was highest in congenital malformations, granulating otitis and cholesteatoma, and in mastoidectomy, especially of the canal wall down type. In this series, the most common causes of anacusis were removal of cholesteatoma from the lateral semicircular canal and removal of the fistula membrane.

One of the lessons to be learnt from these reports is that extreme caution is always necessary when instrumentation in the oval window is required in tympanoplasty. Another lesson is that the considerable risks of severe iatrogenic sensorineural deafness in ears with labyrinthine fistula can only be avoided by the use of a technique in which the matrix over the fistula is meticulously preserved (Gormley, 1986).

**Labyrinthine trauma in stapedectomy**

This is considered in Chapter 14.