Paparella: Volume II: Otology and Neuro-Otology

Section 3: Diseases of the Ear

Part 1: General Problems

Chapter 15: Otologic Trauma

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Because of high-speed travel and activities unknown to past generations, modern man faces an increasing problem of severe injuries and death due to trauma. Correctable and preventable injuries to the ear are of major importance and deserve the reader's closest attention. These injuries are always amazingly unique in their unending variety of origin. For instance, one may see serious ear disorders with causes ranging from being struck by lightning to being hit by a snowball. The most frequent and most acutely complex injuries are those from closed head trauma.

Trauma from Closed Head Injuries to the Temporal Bone and its Contents

Today, trauma of the head is a devastating injury to which we are all susceptible. The more actively one participates in life, the more danger one faces; thus, the young are the most vulnerable.

It is startling to realize that the major health problem in persons between the ages of 1 to 34 years is now automobile accidents! More people are killed and injured by this means than by any other. Because the head is the most mobile part of the body, almost 75 per cent of vehicular accidents involve the head. Furthermore, when the head is severely injured, the ear is the most frequently damaged sensory organ. Much could be said in regard to prevention. Decreasing and enforcing the speed limit and compulsory utilization of seat belts and/or air bags should receive our professional, organizational, and personal support.

The first discoveries of injury to the ossicular chain resulting from skull fracture in the living were made independently in 1956 by Thorburn and the senior author of this chapter. These injuries produced a well-documented pure conductive hearing loss by ossicular chain interruption. It is now realized that these injuries occur quite frequently.

Early Management of the Critically Injured

After a serious head injury, the maintenance of life is the demanding concern. The otolaryngologist is usually not the first physician required to take responsibility unless he or she encounters a situation such as a road-side accident. Nevertheless, by training, he or she is uniquely qualified and should be mentally prepared to organize professional care around some well-recognized and practical standards of successful management. Likewise, because the

structures within this field are so frequently injured, the otolaryngologist should readily join the head injury team with confidence.

An immediate threat to life may result from interference with vital physiologic functions. These involve the respiratory and circulatory systems. Priorities are thus established accordingly.

Airway

The first priority is to establish and maintain a proper airway. Suction clearance of the respiratory tree, laryngoscopy with intubation, or tracheostomy with controlled ventilation is frequently needed to ensure proper oxygenation. A tracheostomy is more necessary than an emergency laparotomy or craniotomy. One must remember the brain's constant and acute need of oxygen. The brain constitutes approximately 2 per cent of total body weight, but it utilizes 20 per cent of the total oxygen uptake. This requirement continues even in sleep. The life centers of the brain are, therefore, more dependent on efficient respiratory function than any other tissue (Hough, 1970).

If one finds that a good respiratory ventilation is difficult to establish, a careful examination for the presence of any of the following should be done immediately: pneumothorax, hemothorax, flail chest, ruptured bronchus, or an open, sucking chest wound.

Circulation

Maintain proper circulation with control of hemorrhage and shock. After a proper airway is established, the circulatory system should be carefully evaluated from the standpoint of blood pressure and cardiac output. Shock and hemorrhage must be controlled rapidly and efficiently.

An important principle that should always be kept in mind in evaluating head injuries is that hypotension appearing after a closed head injury is almost always the result of blood loss and not caused by the head injury itself. If the blood pressure is falling, one must look for the source of blood loss.

Internal hemorrhage may be present in the chest, causing cardiac tamponade or hemothorax. Hemorrhage may also occur in the abdomen from a ruptured viscus, such as a spleen or a kidney. An intracranial vascular break may also be present, requiring neurosurgical intervention. As will be discussed later, this must be evaluated carefully as to site. The most frequent site of hemorrhage is usually not within the head but within the chest or the abdomen. If radiologic assessment is not available in an emergency, an 18-gauge needle inserted into the peritoneal or thoracic space may give valuable information.

Vasoconstrictor drugs should not be used to correct hypotension. Attention should be directed toward replacement of the intravascular fluid volume.

Ongoing Evaluation of the Patient with Temporal Bone Fractures

After the acute emergency of pulmonary or circulatory failure has been overcome, one is in a position to carry out a more detailed investigation with longer periods of observation.

State of Consciousness

Observation of the state of consciousness is extremely important. An improved state of consciousness is promising, whereas a deteriorating state suggests a poor prognosis. The patient may be awake and responsive; this is a good sign even though amnesia may be present. The patient who is confused and restless may yet have a fairly good prognosis, but the unresponsive patient in deep coma whose pupils are dilated has a very poor outlook. A deteriorating state of consciousness is a very ominous sign. The development of a hemiparesis with a dilation of one pupil suggests a developing intracranial hematoma (Hough, 1969).

Neurologic Evaluation

A thorough neurologic appraisal with an evaluation of cranial nerve, spinal cord, cerebral, and cerebellar functions can usually be done quite successfully. Since most of these responses are objective in nature, a practical assessment of the general neurologic system is possible even if the patient is unconscious.

Specialized Tests

Computed tomography (CT) has become an extraordinary aid in identifying and localizing expanding hematomas, and so forth, and in observing positional shifts of major brain structures. This, plus the more recent benefits from nuclear magnetic resonance imaging (MRI) has rendered many other investigative techniques obsolete. These procedures may be life-saving in localizing expanding intracranial lesions such as hematomas. Cerebrospinal fluid examination is of little value in acute head injures and may be misleading or even dangerous.

It must be emphasized that early x-ray examination of the bony skull is elective but can be very helpful. One must remember, however, that the acute problem resides in the effects of the injury on the soft tissues of the brain and the neurovascular structures. Initially, edema and hemorrhage are far more important than is the study of bony fracture lines. Conventional radiographs frequently demonstrate no visible fracture lines; however, laminography and polytomography makes possible more definitive roentgenologic studies that can make possible better localization of fractures through the temporal bones.

Unfortunately, the temporal bone contents are difficult to see on radiographs because of surrounding bone density, which must also be penetrated in order to display the object of the search. Laminography or tomography and, to a greater degree, hypocycloidal polytomography are able to overcome this objection by blurring out the objects proximal and distal to the plane of interest.

The four main types of tomography used in radiology are linear, elliptical, circular, and hypocycloidal (Wright and Taylor, 1973). It behooves the clinician to know which type of tomographic equipment is available in his or her institution for the following reasons:

1. Circular tomography is 3.15 times more efficient than linear tomography in dispersing the blurs (Ross, 1963; Wright and Taylor, 1973), and thus in obtaining sharper and clearer tomograms. Therefore, linear tomography is seldom used for tomography of the temporal bone.

2. Hypocycloidal tomography (polytomes) produces even sharper images, being five time as efficient as linear tomography in dispersing the blur. Thus, hypocycloidal tomography produces the best possible imaging sections of the temporal bone.

Some radiologists recommend four projections at approximately 45 degrees apart. These projections are usually the coronal, axial, sagittal, and the Stenvers projections (Valvassori et al, 1982). Most tomograms are obtained with two views, the coronal (anteroposterior) and the sagittal (lateral). Longitudinal fractures are best seen on the coronal projection (Potter, 1972). Using tomography, Lambert and Brackman (1984) were able to identify the fracture line in a series of 26 longitudinal temporal bone fractures 81 per cent of the time. In contrast, Griffin and colleagues (1979), using conventional radiography, were able to find one or both fractures in only five of 15 patients who had bilateral longitudinal temporal bone fractures. In thee of the five patients in which the fracture was not identified using conventional radiography, they were able to visualize one or both fractures with the use of tomography.

CT functions through an arc of 180 degrees or 260 degrees, depending on the unit. CT scans are taken in an axial plane at a much faster rate by using many detectors that pick up irradiation through different projections and then store this information in a computer. The CT scan takes multiple "pancake slices" through the axial plane; reliable information is then produced concerning the whole head, one slice at a time. One should note that some degree of resolution is lost on reconstructed planes. The fracture line will show best on the CT scan when it is perpendicular to the axial plane. Additional CT scans through the coronal plane (a view from the chin to the top of the vertex of the head) may give further information, but patient positioning for this may be unreasonable for the severely injured. Coronal scans of the temporal bone are particularly informative in visualizing the internal auditory canal, the oval window, and the tegmen (Shaffer and Haughton, 1980).

When the question arises as to whether one should use a CT scan or tomography, the following advantages of each technique should be considered:

CT Scan

1. Will show hematomas within the calvarium; this makes CT the procedure of choice for the acutely injured patient.

2. Can be performed much faster than conventional tomography, giving it preference in

use for the severely injured.

3. Gives less radiation than does conventional tomography, if the plane of the eye can be excluded from the CT scan (Shaffer et al, 1980).

Tomography

1. Less expensive (usually costs one fourth to one third as much as a CT scan).

2. Can be performed through multiple planes, whereas CT can be performed only through the axial plane in the severely injured (the coronal plane can be added in those patients who can lie on their abdomen and extend their head). This provides greater opportunity to find difficult-to-see fracture lines.

Using CT, even the ossicles may be studied with a moderate degree of accuracy. Conventional roentgenography, regardless of the type of projection, has been of little help in assessing the integrity of the ossicular chain or the course of a basilar skull fracture. In emphasizing the importance of hypocycloidal polytomography in the preoperative diagnosis of these injuries, Wright and associates (1969) stated, "Deviation from the normal position of either the malleus or incus or both have been evident in tomography over 90 per cent of the time". Guerier and colleagues (1967) add, "Unfortunately there are no known projections that give good information regarding the status of the stapes".

One must not rely to heavily on roentgenology for diagnosis. In general, one must realize that the other, quite specific signs and symptoms of temporal bone fractures are an excellent foundation for early practical diagnosis and management.

The Head Injury Team

Most of the procedures just described are ordinarily under the domain of those professional colleagues concerned with neurosurgery, neurology, and radiology. The otolaryngologist, however, is very much a part of the head injury team. In about 2000 patients with cranial trauma hospitalized and observed by Guerrier and co-workers (1965-66, 1967), 290 suffered obvious acute otologic disorders; this represents 15 per cent and does not include a large group who may have suffered unrecognized auditory and vestibular disturbances. Long ago Passow stated, "In severe skull trauma, the ear rarely escaped undamaged" (Grove, 1947). Thus, the otolaryngologist is uniquely able to inform and assist his or her professional colleagues, to the benefit of the injured patient. The otolaryngologist's importance on the head injury team is emphasized by his or her credentials. First, by examination of the nose, nasopharynx, throat, and ears, he or she is able to see more deeply into the interior of the head than any other physician. Secondly, he or she is better acquainted with the anatomy and functional tests of the two cranial nerves most likely to be injured (VII and VIII) in severe head injury (Hough, 1970).

Types of Temporal Bone Fractures

To understand the signs and symptoms of various temporal bone fractures and to be able to correct the defects surgically, one must have a good concept of the program and extent of the fracture line through the base of the skull. Injury to the skull may produce fractures of the temporal bone that can be easily classified into three groups: longitudinal, transverse, and mixed. It must be categorically stated that a rigid classification of temporal bone fractures is impossible as to type and direction. One must always realize that no two temporal bone fractures are alike, and the varieties of otologic effects are endless. Nevertheless, it is both convenient and practical to use the conventional, long-established classification of these three groups.

Longitudinal Fractures

Classification of the various types of skull fractures depends on the relation of the fracture plane to the long axis of the petrous bone. As the name indicates, the longitudinal fracture runs lengthwise through the petrous pyramid. Longitudinal fractures are by far the most common (70 to 80 per cent) and are usually caused by blows to the temporal or parietal areas rather than to the occipital or frontal areas (Proctor et al, 1956; McHugh, 1959). Although such a fracture is less hazardous to the cochlear and vestibular mechanisms, it is much more damaging to the conductive mechanisms in the middle ear.

This type of fracture classically begins in the temporal squama, extends through the posterosuperior external bony canal wall, runs across the roof of the middle ear, and thence along the carotid canal anterior to the labyrinthine capsule to the middle cranial fossa near the foramen spinosum. Because the tegmen is fractured there will be a cerebrospinal fluid leak if the dura is also torn.

The structures of the middle ear rarely escape injury in a longitudinal fracture. The skin of the external ear canal and tympanic membrane are usually torn; therefore, bleeding from the ear following head injury is considered to be such strong evidence of longitudinal basilar skull fracture that the diagnosis is considered positive unless disputed by other obvious injuries.

The force of the impact may cause a very wide break in the temporal bone with an amazing amount of twisting and torsion, or it may cause only a small crack without bony displacement. This break in the wall of the middle ear and injury to the tympanic membrane produces the second cardinal symptom of longitudinal basilar skull fracture - that is, conductive deafness.

Although the middle ear structures are involved, they are frequently not seriously displaced, and healing often occurs spontaneously without residual conductive deafness. In these cases, a slight or moderate sensorineural loss may be the only residual hearing impairment from the accident. However, in many other longitudinal or mixed fractures, we now know that severe displacement of the ossicles may occur. This results in a marked loss of hearing because of the conductive defect. Fortunately many of these conductive defects are amenable to surgical

reconstruction, as will be discussed later.

In longitudinal fractures, the inner ear frequently escapes direct injury. The fracture line usually extends adjacent to, or around, the hard labyrinthine capsular bone rather than through it. Most frequently the fracture line extends along the carotid canal, running medially to the foramen spinosum or lacerum, but occasionally there is a descending branch that may also extend into the temporomandibular joint. According to Gove (1947), 23 per cent of longitudinal fractures are bilateral.

Facial nerve injuries are present in approximately 20 per cent of patients suffering longitudinal fractures. The facial nerve may be damaged in the tympanic portion distal to the geniculate ganglion and, in rare instances, may be damaged in the vertical portion within the mastoid process. Most longitudinal fractures extend along the fallopian canal instead of across it. As a result, most facial nerve injuries produce a paralysis of delayed onset.

An uncommon type of fracture may be restricted to the mastoid process itself. This may open into the external canal and middle ear and involve the facial nerve in the vertical segments (McHugh, 1949).

Transverse Fractures

A transverse fracture of the petrous pyramid occurs in approximately 20 per cent of those patients suffering temporal bone fractures (Proctor et al, 1956; McHugh, 1959). This fracture extends from the posterior cranial fossa transversely across the petrous pyramid to the middle cranial fossa perpendicular to the axis of the petrous pyramid. The fracture line usually extends between the various foramina in its course across the petrous portion of the temporal bone. It usually begins in the foramen magnum, extending between the various foramina, such as the jugular foramen, to the hypoglossal and occasionally through the internal acoustic meatus and the labyrinthine capsule. It frequently ends in the middle cranial fossa in the region of the foramen lacerum or the foramen spinosum.

As the fracture line passes through the labyrinthine capsule, the vestibular system and cochlea may be functionally destroyed. Because of the course of the fracture, the facial nerve is much more vulnerable to complete lysis, and therefore the injury may cause immediate facial paralysis. The fracture may explode the lateral wall of the labyrinthine capsule (the medial wall of the middle ear) and produces hemorrhages into the middle ear and ruptures of either one or both of the oval and round windows. It is then possible to fracture or dislodge the stapes from the oval window, causing ossicular chain fractures or displacement as well. However, this is uncommon.

These fractures lines are thought to be due primarily to blows on the occipital area. Frontal blows are more likely to produce an anterior plate or an anterior fossa injury, but a crushing blow to the frontal region may cause a parietal buckling, thereby producing a severe mixed fracture of the temporal bone.

In transverse fractures, the important findings are (1) vertigo with spontaneous nystagmus, (2) sensorineural deafness, (3) facial paralysis (the facial nerve is involved in approximately 50 per cent of patients), and (4) frequent hemotympanum behind an intact tympanic membrane.

Mixed Fractures

Multiple fractures may occur from crushing head blows, and the fracture may involve both the middle and inner ears with a tympanolabyrinthine fracture. The fracture lines may include various branches, incorporating several of the previously mentioned routes (Hough and Stuart, 1968).

Ongoing Otologic Evaluation

Examination of the Temporal Bone and its Contents

All the previously mentioned general observations should be made and thoroughly considered during the first otologic evaluation. The specific examination of the temporal bone and its contents with the general physical assessment should be done only as the condition of the patient will allow. Obviously the unconscious patient cannot cooperate, and the reactions to subjective testing, such as are required in most audiometric techniques, cannot be checked.

Nystagmus, facial palsy, and objective hearing loss should be specifically documented. As the examination proceeds, many important signs may become evident. For example, the presence of blood in the external ear canal suggests the possible presence of a longitudinal basilar skull fracture with a torn tympanic membrane and middle ear damage. Ecchymosis over the mastoid area often indicates blood in the pneumatic spaces of the temporal bone (Guerrier et al, 1967). An intact tympanic membrane with blood behind the eardrum indicates an explosive break of the labyrinthine capsule caused by a transverse temporal bone fracture extending across the base of the skull. Cerebrospinal fluid otorrhea may be seen because of a fracture line fistula through the temporal bone with an overlying tear in the dura.

Only two things need to be done as soon as reasonably possible. First, if the ear is filled with debris and blood, which may be an external source of contamination, and if the patient's physical condition permits, the ear should be cleansed; this must be done in the operating room suite under microscopic visualization with absolute asepsis (Hough, 1970). Second, one should institute the prevention of post-traumatic infection with sterile ear care.

Unfortunately the otolaryngologist frequently is not present for the immediate postinjury care of a patient. He is often consulted weeks or even years later because of the patient's facial paralysis, disequilibrium, or loss of hearing. Obviously, education of our colleagues is needed.

Three points should be stressed concerning evaluation of otologic trauma.

1. Every patient with a head injury should have an otologic examination. Many patients live for years with a correctable hearing impairment, unaware that the hearing might be restored. In many of these cases, the primary physician observes only vital signs and is oblivious of the presence of a hearing impairment.

2. The otologic examination should be carried out as soon as possible after the injury. The junior author of this chapter (M. McG) was recently asked to evaluate the ear of a patient who had suffered a fracture of the mandible 3 weeks previously. The patient had surgical repair of the fractured mandible but, unfortunately, did not have an otologic examination. When he was seen, he was found to have complete stenosis of the external ear canal secondary to compound fracture of the anteroinferior external canal wall. This resulted from the condyle of the mandible being pushed through the bony canal wall at the time of injury. It would have been much easier to reduce the fracture shortly after the injury than to correct the stenosis after scar tissue and bone had obliterated the ear canal.

3. The otologic evaluation should be carried out by an otolaryngologist. No other specialist is better acquainted with the anatomy, function, and treatment of the two most common cranial nerve injuries that occur from head trauma. The anatomic and physiologic complexities in this region require the attention of an otolaryngologist.

Analysis of Signs and Symptoms

History

An accurate history, either early or late, to obtain important diagnostic details often is impossible to obtain. The physician must remember that events of childhood are often distorted by poor memory. In addition, the history may be obscured by adult suggestion. Unpleasant incidents are often suppressed because of psychological trauma and occasionally because of a feeling of guilt. Although some patients deny past history of head injury in opposition to the truth, one must remember also that a blow on the head is often related to all kinds of symptoms in the minds of some patients throughout their lifetime.

It was found in an investigation of a large number of patients who presumably suffered otologic disorders secondary to head injuries that 25 per cent gave an erroneous history; that is, they considered head trauma as the probable cause of a hearing impairment. Evidence provided by clinical and surgical findings, however, revealed that the etiologic background was entirely unrelated to the head injury. Nevertheless, one must be extremely wary of disregarding an unsolicited history of head injury when the patient believes a relationship exists between the accident and his hearing loss (Hough and Stuart, 1968).

Conversely, it is not uncommon to find an ossicular chain derangement on exploratory tympanotomy in an individual who steadfastly denies a previous history of head injury. The following case is illustrative of this diagnostic dilemma. In 1960 a 49-year-old woman underwent otologic surgery. She had given a history of progressive hearing impairment since young

adulthood, and her audiometric tests and examination supported a diagnosis of clinical otosclerosis. She did not give a history of head injury at any time. Stapedial surgery was performed on this patient's right ear. The stapes was fixed with an anterior otosclerotic focus, and the functional results following the surgery were excellent. Four years later the opposite ear was surgically entered with the expectation of finding an uncomplicated problem of otosclerosis. A distinct fracture line in the posterior superior bony canal wall was exposed and an ossicular chain derangement with an incudostapedial joint separation was seen. The patient was immediately questioned again about a history of head injury, but she denied knowledge of this. Later inquiry of the patient's mother about her past history revealed that the patient had fallen from an automobile at the age of 2 years and had suffered a significant head injury. This injury was followed by a period of unconsciousness and bleeding from one ear (Hough and Stuart, 1968).

Answers to the following questions will be important for otologic consideration:

1. Was there bleeding from the ear at the time of the accident?

2. Was there unconsciousness at the time of the accident?

3. Was the loss of hearing sudden in onset and was it specifically related to the accident?

4. On what portion of the head did injury occur?

5. Is there evidence of a fracture in the ear canal?

6. Was the hearing loss unilateral?

7. Does the audiometric pattern confirm the historical facts?

8. Were there other neurologic deficits, such as a facial nerve injury?

9. Were there postinjury signs and symptoms of disequilibrium?

In the senior author's experience, the most frequent symptoms of middle ear damage from head injury have been (1) loss of hearing, (2) bleeding from the ear, and (3) unconsciousness. These three symptoms could be called the "traumatic conductive triad". If any of these exist in relation to the injury, one must suspect damage to the conductive mechanism, but if all three symptoms were present at the time of the accident and the hearing loss has persisted at the time of the accident and the hearing loss has persisted, an ossicular chain interruption must be considered to exist until proved otherwise (Table 1) (Hough and Stuart, 1968).

Table 1. The Middle Ear in Skull Trauma (Data from 31 Patients with Ossicular Chain Derangement)

Symptoms	Incidence (%)
Unconsciousness	100
Bleeding from the ear	100
Acute postinjury vertigo	13
Chronic postinjury vertigo	0
Infection following surgery	13
Facial nerve injury	19.3
Residual facial paralysis	13.

Bleeding from the Ear

In a transverse fracture there will usually not be bleeding from the ear unless there are other associated injuries. In a longitudinal fracture, however, bleeding almost always occurs. This symptom is so constant that it is the hallmark of longitudinal temporal bone fractures. The bleeding usually lasts for a very short period and is not profuse. In about 15 per cent of cases, however, the bleeding is more intense. It may originate in the middle ear, with drainage down the eustachian tube, to be displayed from both the mouth and nose, and it may be so profuse as to cause difficulty in keeping the airway of the upper respiratory tract open. Bleeding may continue for several hours or even days.

Unconsciousness

In a large series of 211 patients with various types of skull fractures, Grove (1947) reported 193 (91 per cent) had unconsciousness. In Proctor's (1956) series of 57 cases, 54 per cent had unconsciousness related to the injury. It should be noted that these series included a number of skull fractures that were not of the temporal bone. However, in a large series dealing only with temporal bone fractures, Hough and Stuart (1968) reported unconsciousness as a symptom in 100 per cent of patients.

Usually the unconsciousness is of very short duration, but it can last for several weeks. Although unconsciousness may not be present in every case of temporal bone fracture, it must be recognized that any injury sufficient to produce dissolution of the temporal bone is usually sufficient in intensity to cause a period of unconsciousness.

Hearing Loss

Of principal interest is the effect of the head injury on the mechanisms of hearing. If the fracture line extends through the middle ear, as in longitudinal or mixed fractures types, there is always an immediate impairment of the conductive mechanisms. This may be a singular result of either a torn tympanic membrane or be owing to an accumulation of blood in the tympanic

cavity from torn mucous membranes; it may occur with a longitudinal, transverse, or mixed fracture. In all these soft-tissue injuries, there is a good chance for spontaneous recovery of the conductive hearing impairment produced by the skull fracture.

Bone and joint injuries caused by skull fracture are discussed later in more detail, but we wish to emphasize here that these injuries are quite common and are frequently correctable by modern microsurgical techniques. It should also be emphasized that any residual conductive hearing impairment after the ear is healed is probably the result of bone or joint injury. If it is significant in amount, the ear should be surgically explored.

Traumatic injury to the inner ear may be caused by concussion or by a tearing fracture line through the cochlea or the internal auditory meatus. Concussion injury may occur from any blow to the head, with or without skull fracture. It may be seen as an addition to the conductive loss resulting from a longitudinal fracture. There may not be an extension of the fracture to involve the inner ear; yet the concussive forces of the injury may cause permanent damage to the neural mechanisms of the inner ear (Pearlman, 1939; Schuknecht, 1950; Schuknecht et al, 1951; Ward, 1969).

Studies of hearing acuity should be done as soon as the patient is able to respond and his or her physical condition permits. These studies should include tuning fork tests, pure-tone air and bone conduction tests, and speech discrimination and speech threshold determination. Complete objective tests such as impedance audiometry and auditory brain-stem response tests may be helpful in certain problem areas. This thorough study of the acoustic branch of cranial nerve VIII is obviously important in the surgical and medical management of the injured. In addition, the otologist is the individual most responsible for a careful assessment and documentation of the disability for medical purposes (Barber, 1969; Fox, 1969; Hough, 1970; Schuknecht, 1969; Wright et al, 1969).

Vertigo

Loss of equilibrium may occur dramatically with head injury with or without fracture of the temporal bone. The otologist is best qualified to determine neurologically the anatomic location of the lesion and is best qualified to assess its importance. Simple Hallpike bithermal caloric tests or Kobrak ice-water caloric tests can readily determine severe injuries to the labyrinth, particularly if the injury is unilateral. Electronystagmography is more precise and provides better documentation; furthermore, both spontaneous and positional nystagmus are more easily detected. In addition to bithermal caloric tests, a number of other useful electronystagmographic tests can be performed that may identify central lesions. Conclusions from labyrinthine tests may of great value when correlated with other findings. (Obviously, caloric tests with H_2O can only be done when the tympanic membrane is intact.)

Vertigo following head injury is of both immediate and long-term importance. Evidence seems to be mounting that much of the postinjury vertigo may have its origin in sources outside the labyrinth. In whiplash injuries, in which the head is free and never strikes another object,

animal experimentation has demonstrated marked ecchymosis and edema of the brain stem and cerebral and cerebellar cortical areas. It has been observed that vertigo occurring with various head positions is a commonly seen physiologic finding, related perhaps to stimuli originating in the soft tissues of the neck and skull (Barber, 1969; Jongkees, 1969b). Although temporal bone fractures can cause acute disturbance in equilibrium, long-lasting chronic disturbances may not be from these sources (Hough, 1968, 1969, 1970). Physiologic paroxysmal positional vertigo, disturbances in neck structure reflexes, and concomitant labyrinthine hydrops or cerebellar disease may be responsible for vertiginous episodes. These may be completely unrelated to fracture of the temporal bone. In many patients, the dysfunction is outside the labyrinth (Hough, 1970).

To emphasize this, in a series of 35 patients having longitudinal fractures of the temporal bone with sufficient injury to produce derangement of middle ear structures confirmed by surgical intervention, there was little evidence of vestibular damage. In only 13 per cent did the patients even remember vertigo following the accident (Hough, 1968, 1970). In these patients the vertigo subsided before the end of 3 weeks. None of the 35 patients experienced long-term post-traumatic vertigo, either constant or episodic. This is an interesting observation, since the patients were questioned thoroughly regarding this symptom and despite suggestive leading questions, all denied postinjury vertigo past the initial 3-week period.

The definition and evaluation of known disability from the loss of a part, or all, of the vestibular system are the responsibility of the otolaryngologist (this is amplified in the section of pathology). At the time of injury, destructive changes, hemorrhagic areas, lysis of nerves, and edema may occur in the brain stem, in the vestibular centers over the cerebral and cerebellar cortical areas, or in the vestibular labyrinth (Barber, 1969; Hough, 1969; Ward, 1969). The fracture line of the transverse fracture of the temporal bone may directly involve the otic capsule with all or part of the vestibular mechanisms. On the other hand, it may cause direct injury or lysis of the cranial nerve VIII. This, in effect, dismembers the cochlear and vestibular labyrinth and causes total loss of hearing and vestibular function on the side involved. In this event, the patient will experience severe vertigo until central accommodation occurs. In a unilateral injury, this usually takes place over a period of no more than 3 to 4 weeks, and relatively little post-traumatic vertigo is experienced as a residual disability, unless the patient is elderly. An individual with visual impairment or disturbances in proprioceptive senses may accommodate much more slowly. A bilateral loss of vestibular function produces severe disturbances in equilibrium, with only partial recovery over a long period of time, resulting in a definite permanent residual disability (Hough, 1970).

Facial Nerve Paralysis

The facial nerve (cranial nerve VII), so important to expression and personality and to many less obvious but extremely important functions such as taste and lacrimation, is in grave danger during head injury. The facial nerve traverses a confined, inflexible bony canal for a greater distance than any other cranial nerve and is vulnerable to injury from skull fracture regardless of where the head is struck. It is, therefore, understandable that this nerve is the second most commonly injured cranial nerve. Because of its relationships, one may say that when there

is a complete facial paralysis following a closed head injury, there is always an associated fracture of the temporal bone. This sign is diagnostic.

In the senior author's experience, the facial nerve was rendered functionless either temporarily or permanently in 19.3 per cent of longitudinal temporal bone fractures (Hough and Stuart, 1968). Among these patients, one-third recovered spontaneously, and this recovery was complete without surgical decompression. However, of those patients who were seen months or years following longitudinal temporal bone fractures with postinjury facial nerve problems, two-thirds experienced incomplete return of facial nerve function and suffered noticeable facial weakness, synkinesis, facial tics, mass movements, and so forth.

Because the petrous pyramid is fractured across somewhat perpendicularly to the facial nerve in a transverse fracture, there is much greater chance for facial nerve injury than with longitudinal fractures. In approximately 50 per cent of transverse fractures the facial nerve is injured, in contrast to 20 per cent for longitudinal fractures.

A longitudinal fracture of the temporal bone rarely causes total lysis of the nerve. The authors have not seen this and neither has Guerrier (1966, 1969) in a large series of patients, but a transverse fracture is much more likely to produce complete lysis. The nerve may even be injured in several places.

The most important question arising in the surgeon's mind is "Which seventh nerve injury requires surgery?". In those head injuries that are accompanied by facial paralysis, approximately one-half of the patients will have immediate paralysis. In the remainder, paralysis is delayed in onset; in these cases, the prognosis is better. Most recoveries that occur spontaneously will begin to appear within the first 5 days. Griffin reports that in up to 94 per cent of patients with delayed-onset facial paralysis, function is recovered within 6 to 8 weeks without treatment; fortunately, most cases are in this category. However, experience indicates that if recovery does not become apparent after 3 weeks, the chance of complete recovery is greatly diminished. When paralysis occurs immediately at the time of the accident, the chance is much greater that the face will be permanently paralyzed than if the onset is gradual or delayed. If paralysis occurs immediately, the nerve is usually torn completely through and is much more likely to be in need of surgical repair, but the repair must wait until the patient's general condition permits surgical exploration. If paralysis begins to appear secondarily, one must follow its course by testing the nerve's various end-organs so as to determine the appropriate time for surgical intervention.

Tests for Facial Nerve Paralysis

There are two primary questions related to the need of surgical intervention:

1. Will the nerve spontaneously recover?

2. Where is it injured?

Clinical tools such as radiography, the Schirmer test, electrical tests, and audiometric evaluations provide a wealth of clinical information.

Radiography

It has been shown that the most common site of injury of the facial nerve in the longitudinal fracture is in the perigeniculate ganglion (Fisch, 1974; Lambert and Brackman, 1984). The most common site of injury in the transverse fracture is in the labyrinthine segment (Griffin, 1979). Because some temporal bone fractures are missed on radiographs, and because many temporal bone fractures are of the mixed type, one cannot assume that a fracture site, injuring the facial nerve in one part of the temporal bone, will be the only place that the facial nerve will be injured. In Lambert and Brackman's (1984) series of 21 patients with perigeniculate fractures, four also had fractures in either the tympanic or the mastoid segments of the nerve. Thus the otolaryngologist needs more than radiographic data in identifying the site of injury.

Topographic Tests

The two most important topographic tests for consideration are the Schirmer test and the stapedial reflex test. The authors do not recommend electrogustometry or the submaxillary flow test. An excellent summary of latter tests are provided by Alford et al (1974).

Schirmer Test. The Schirmer test, which provides much information, uses two strips of paper folded and placed in the lower conjunctival fornix so that the length of the strip is 5 centimeters. Both eyes are tested for simultaneous tearing for 5 minutes. Paradoxically, it has been shown that unilateral injury to the geniculate ganglion can cause reduction of tearing bilaterally. In 18 patients who underwent unilateral resection of the geniculate ganglion for petrosal neuralgia, seven had bilateral reduction in tearing (Fisch, 1976). Lambert and Brackman (1984) consider the Schirmer test to be positive if lacrimation is decreased 75 per cent or more on the affected side compared with the opposite normal side, or if lacrimation is decreased bilaterally (less than 10 mm for both sides at 5 minutes).

Electrical Tests

Electrical testing will not determine in the immediate postinjury state whether or not the facial nerve has been transected; transection at this time can be ascertained only by direct vision in surgery. The electrical tests offering the most information are the maximal stimulation tests and electroneurography. Of the two, the MST is the oldest and was first described by May and colleagues (1971).

Maximal Stimulation Test (MST). The MST is an outgrowth of earlier work attempting to test facial nerve paralysis using electrical stimulation. Today, this early work is called the minimal excitability test; it tested each branch of the facial nerve and compared the stimulation on the affected side with that of the corresponding branch on the opposite side. If there was a difference of 3.5 mA between the sides, surgical decompression was possibly indicated (Alford

et al, 1970; Jongkees, 1969a; Kraus, 1970). The Hilger nerve stimulator was designed for this use. The Hilger nerve stimulator is also used in the MST in which a setting of 5 mA or the highest setting tolerated is used. The involved side is compared with the normal side, and one of three responses is elicited; equal response, reduced response, or no response. May and associates (1971) showed that the MST was more likely to predict those patients who would not recover complete function of cranial nerve VII when compared with the minimal excitability test. May and co-workers (1976), in a later publication, found that in patients with a normal response to MST, 88 per cent had complete return of facial nerve function; with reduced MST response 73 per cent had complete return, and with no MST response only 27 per cent had return to normal function.

Electroneurography (ENoG). ENoG should not be confused with electromyography (EMG). EMG does not help in evaluation of facial nerve paralysis of recent onset. At the earliest, it will not show denervation potentials until 8 to 10 days after denervation has begun (Essen, 1977). ENoG actually records the compound action potential of the facial nerve after it has been stimulated. Electrodes are placed over the skin at the stylomastoid foramen, and a supramaximal stimulation is applied. Recording electrodes are placed over the skin at the nasolabial fold, and supramaximal stimulation is given to ensure that all the functional nerve fibers are excited. The voltage is increased gradually until an increase in voltage has no effect on the amplitude. The response that is obtained is actually a summation of many motor units recorded as a compound action potential. The side with the normally functioning facial nerve is tested first and the interpeak distance is recorded (amplitude). The opposite (paralyzed) side is then tested and its amplitude is recorded. A comparison of amplitudes from each side is made. The amplitude loss on the involved side is directly proportional to the degree of degeneration. For example, if an amplitude on the paralyzed side is only 10 per cent of that on the uninvolved side, then 90 per cent degeneration is said to have occurred. Essen (1977) has shown that even with up to 90 per cent degeneration, recovery is usually good and only a few such patients will have some residual paralysis. Many surgeons use 90 per cent degeneration as the indication for facial nerve decompression. May (1980) recommended surgery when the amplitude of the compound action potential on the affected side is 25 per cent or less than that on the normal side.

Unfortunately the MST and ENoG tests are not of benefit until at least 72 hours postinjury. This conclusion is based on findings in studies in which cranial nerve VII was sacrificed at the time of acoustic neuroma removal (Gilliat and Taylor, 1959). Other problems with the electrical tests are that both tests are painful, and that inconsistency is frequent, especially with ENoG, because of differences in age, sex, patient cooperation, electrode placement, stimulus factors, and type of filters and test and retest variability.

Audiologic Tests

All patients with facial paralysis following head injury should have a thorough Audiologic evaluation that includes pure-tone audiometry, discrimination scores, and impedance audiometry. It is important to know precisely the hearing status of the patient prior to surgery for nerve VII decompression because sensorineural hearing impairment is a common surgical complication

(May, 1983). Other nerve VII tests such as electronystagmography (ENG) and auditory brain-stem response audiometry should also be performed in most cases.

If the facial nerve paralysis is present in a normal-hearing patient with no discernible middle ear trauma, impedance audiometry is a useful topographic test. If the stapedius reflex is absent, then the lesion site will be between the geniculate ganglion and the nerve to the stapedius muscle. If the reflex is present, then the site of injury is distal to the nerve to the stapedius muscle.

Surgery for Facial Nerve Paralysis

Immediate-Onset Paralysis of Cranial Nerve VII

Kettle (1950) stated that an immediate facial paralysis should be surgically explored as soon as the patient's condition permits. The authors concur with this concept.

The surgeon should be prepared to perform all techniques of nerve exposure through either the postauricular or the endaural route. Exposure should be possible from the geniculate ganglion to the stylomastoid foramen. Similarly - and very important - the surgeon should be prepared to explore the facial nerve from the geniculate ganglion to the internal acoustic meatus via the middle cranial fossa approach. The nerve may be frayed, cut, or compressed by bone chips or hematomas anywhere along its entire course. The surgeon must also be acquainted with, and capable of doing, all surgical techniques which might be required to reconstruct the nerve, such as grafting, rerouting, or decompressing.

Delayed-Onset Paralysis of Cranial Nerve VII

If the nerve has not been transected, but a paralysis has gradually progressed to a total block of the axoplasmic flow, the nerve will degenerate. Once degeneration has occurred, there are two possibilities concerning the nerve: (1) no regeneration will occur; and (2) if regeneration does occur it will be incomplete, resulting in permanent defects that might have been avoided with proper surgical intervention. Therefore, if electrical tests (MTS and ENoG), as determined reasonable by the physician, indicate the probability of degeneration, surgery for nerve VII decompression or repair is recommended.

Surgical Technique

When facial paralysis results from head injury, whether of delayed or immediate onset, the authors prefer to enter the ear through an endaural incision. This allows immediate exposure of the horizontal portion of the facial nerve where injury is most likely to occur in a longitudinal fracture. The tympanomeatal flap is elevated and the facial nerve explored from the geniculate ganglion horizontally across the middle ear. It is then followed around the posterior genu near the pyramidal eminence. It is necessary to remove only a small amount of bone from the ear canal to trace the nerve inferiorly. This bone is rapidly and completely drilled away to expose

the nerve, if necessary all the way to the stylomastoid foramen. This approach can be done rapidly and safely and provides a complete temporal bone view of the entire osseous course of the facial nerve. This transcanal approach visualizes, first, the area most likely to be injured and provides early and complete visualization of the facial nerve throughout the operation (Hough, 1970).

May and Klein (1983) and Graham (1980) have described an extended mastoid approach for the visualization of the geniculate ganglion. Unfortunately, the geniculate ganglion cannot be reached in every patient with the approach these authors used. When it can, however, complications associated with the middle cranial fossa procedure may be avoided. With the middle cranial fossa approach, the geniculate ganglion can be reached in all cases. It must be remembered that the transmastoid decompression must be carried out first and then followed by a middle fossa approach for central decompression.

If there is total inner ear loss, then the translabyrinthine approach is recommended.

Recommendations for Type of Surgery

Condition 1. Sudden facial nerve paralysis at the time of trauma plus total sensorineural hearing impairment.

Recommendation. The authors much prefer a trans-labyrinthine total exposure of nerve VII from the stylomastoid foramen to internal auditory canal in the posterior cranial fossa. This provides excellent visualization for total precise decompression, possible grafting, or rerouting if necessary.

Condition 2. Sudden paralysis of cranial nerve VII at the time of trauma, with ipsilateral hearing still intact.

Recommendation. The authors prefer a progressive approach. First, a transcanal tympanotomy is performed, progressing to an endaural exposure of cranial nerve VII from the distal labyrinthine segment to the stylomastoid foramen, followed by middle cranial fossa approach from the labyrinthine segment of cranial nerve VII to the internal auditory canal.

Condition 3. Progressive or delayed paralysis of cranial nerve VII, with no hearing.

Recommendation. The authors recommend the same operation as described for condition 1.

Condition 4. Progressive or delayed paralysis, with good hearing.

Recommendation. The authors recommend the same surgical procedure as described for condition 2.

Condition 5. Cerebrospinal fluid leaks plus facial nerve paralysis, all types.

Recommendation. The authors recommend the postauricular canal wall-up procedure, with middle fossa approach as needed.

Summary

Unfortunately many physicians outside the field of otolaryngology are unaware of the importance of early facial nerve surgery in the severely injured patient. They should be advised of the benefits that modern otologic microsurgery can offer these patients.

Etiology

A recently reported series of 37 head injuries in which there was sufficient force to produce ossicular chain derangement demonstrates the wide variety of circumstances that may be encountered. The head was injured from being struck by a steel pipe, falling log, or pitched baseball; or the injury resulted from the patient's being thrown from a horse or falling downstairs, off a bluff, out of a house, from a building, out of a car or truck, off an automobile or fender, bicycle; or the patient was struck by a train; and so forth. The greatest single cause was from automobile travel. In 35 per cent of this group, the patient fell out of a moving automobile or truck, and in all instances these were children under the age of 13. The use of seat belts and door safety locks might have prevented these injuries. Moreover, in this group, over half of the injuries were caused by an automobile accident (Hough, 1968, 1970).

Head injury is a major health problem in every civilized nation; it is the cause of the most common neurologic disorders. For example, over 500.000 head injuries occur yearly in Great Britain, and it has been estimated that about 75.000 new patients with persistent postconcussive symptoms are added to the existing number each year in that nation (Barber, 1969).

Age and Sex Distribution

In this culture, the vigorous young man tends to live with more dangerous involvement and is much more likely to experience a significant otologic injury than a young woman of the same age or an adult of either sex. This is proved by the fact that in the series observed by Hough and Stuart (1968), 75 per cent of the patients with skull fractures causing middle ear injuries were males and only 25 per cent were females. Although these were all cases of longitudinal temporal bone fractures, one can reasonably expect other skull injuries to fall into the same pattern with regard to sex distribution. Wright and associates (1969) have also reported a ratio of 3-to-1 in male/female involvement in a group of injuries including those caused by foreign bodies, air compression, and so forth.

One usually considers that children are more prone to injury than adults, and this was borne out in a study of age relationships to head injury producing middle ear damage. Fifty per cent of the injuries occurred in children before puberty (age 13), and an amazingly high

percentage (70 per cent) occurred during the first 21 years of life.

Pathology

A closed head injury may cause isolated areas of damage in any region of the temporal bone, from the external ear canal to the internal acoustic meatus, or it may cause total destruction of all major functional units within the structure of the temporal bone. Although by present means of analysis a rather accurate diagnosis can be made as to the type of temporal bone fracture and the extent of the injury to auditory function, must still lies hidden from the examining otologist. The complexities of vestibular disturbance and the difficulty in assessing values and localizing lesions continue to present problems for which we must yet find the answers. Histopathologic investigation of temporal bones after death, roentgenologic studies (particularly with polytomography), and new surgical experience in the tympanic cavities of previously injured skulls have shed great light to benefit our present knowledge and enhance our ability to manage certain of these injuries. At present, specific restoration of function is available to many patients with middle ear lesions, but repair of damage to the inner ear must depend on natural recovery.

External Ear Canal and Tympanic Membrane

The external ear canal is the first otologic structure to be examined visually. If hemorrhage or discharge is occurring or has recently occurred, this examination should be done with the aid of a microscope with complete surgical sterility. When blood is seen in the external ear, one must realize that this may have been produced from other sources, such as laceration of the external ear, a tear in the skin of the canal by trauma to the concha or injury to the temporomandibular joint, or a rupture of the tympanic membrane caused by a foreign body or air compression. A blow to the mandible may cause a subluxation of the temporomandibular joint, the mandible being driven through the anterior ear canal wall. The external ear canal may be completely occluded by the collapse of the wall. On the other hand, bone fragments from the posterior canal or a protruding ossicle may also cause canal distortion or complete occlusion. It is only infrequently that the external ear canal alone is involved.

An obvious break in the posterosuperior bony canal wall is frequently observed and is the presenting sign of a longitudinal temporal bone fracture. This fracture line can be seen running from its extension into the middle ear near the notch of Rivinus obliquely, posteriorly, and laterally on the canal wall.

The tympanic membrane is frequently torn in the longitudinal temporal bone fracture. If the patient is seen early and the physical condition permits, the edges of the perforation may be surgically replaced at the time of the original microscopic examination (Guerrier et al, 1965-66, 1967). The tearing of this membrane usually is produced by the separating force of the canal bone at the circumferential edge of the eardrum. This usually appears in the posterior half of the membrane and is rarely seen anteriorly.

Tympanic Cavity

An apparent fracture involving the middle ear should arouse suspicion as to the possibility of its extension into the inner ear, thus presenting a mixed temporal bone fracture. A rupture of an overlying meningeal barrier may be present, causing a fistulous leak of cerebrospinal fluid. This leak usually ceases spontaneously with a fibrous seal of the fracture line; however, persistent leaks lasting over 2 weeks require surgical repair. Purulent otorrhea from superimposed infection is frequent in traumatic recovery (Hough and Stuart, 1978). The pathologic inflammatory involvement in virgin tissue is often severe and, since fracture lines have opened normal structural barriers, intracranial and intralabyrinthine complications may develop rapidly and be difficult to control (Perlman, 1939).

The appearance of a hemotympanum without rupture of the canal wall or tympanic membrane is presumptive evidence of a transverse (tympanolabyrinthine) fracture with a fracture of the medial wall of the middle ear. Laceration and hemorrhage of the mucous membrane of the middle ear may occur without temporal bone fracture or a tear in the tympanic membrane and may be the result of ossicular displacement, buckling of the tympanic walls, and/or aerocompression.

The otologist frequently first examines the patient months or even years after the injury. This late post-traumatic examination may reveal a visible fracture line in the posterosuperior canal with or without a notch at the edge of the canal wall, bony displacements caused by bone fragments, pieces of ossicles, and so forth. The tympanic membrane may demonstrate healed or unhealed fracture lines.

Middle Ear: Ossicular Injuries and Concomitant Disease

The reader is invited to examine the details presented in Table 2. This presents the pathologic observations made at the time of surgery in 31 patients suffering an ossicular chain derangement following skull injury (Hough and Stuart, 1968). This is a study of patients who had been subjected to enough force to dislodge the ossicular chain and thus cause a conductive hearing lesion that would not recover spontaneously. These are, therefore, practical statistics to keep in mind when approaching the middle ear surgically. Since this report was made, the experience of the senior author (J.V.D.H.) continues to approximately the same.

Note that in 66.6 per cent of these patients, the posterosuperior bony canal wall was found to have a visible fracture line at the time of surgery. In 92.3 per cent there was an incudostapedial joint separation, and in 57.1 per cent there was massive dislocation of the incus. A fracture of the stapedial arch was also found to be very common; it was seen in 30 per cent of these patients. Fractures of the malleus were much more rare, being present in only 11 per cent. Ankylosis and fixation of the upper ossicular chain by hyperostosis or by tympanosclerosis was found in a surprisingly high percentage (25 per cent). In one ear the incus was entirely missing, and in another the malleus could not be found. Unusual, multiple concomitant pathologic conditions were found in an amazingly high percentage of 34.5 per cent.

Table 2. Middle Ear Surgical Problems Found in 31 Patients Following Severe Head Trauma

Injury	Incidence (%)
Posterior bony canal wall fracture	66.6
Incudostapedial joint separation	82.3
Massive dislocation of the incus	57.1
Fracture of the stapedial arch	30.0
Fracture of the malleus	11.0
Fixation of the upper ossicular chain	25.0
Multiple concomitant middle ear pathology	34.5.

Concomitant middle ear disorders found in the same group are shown in Table 3. Otosclerosis was found in 6.4 per cent of the patients; tympanosclerosis, in 9 per cent; chronic otitis media and cholesteatoma, in 9 per cent; and concomitant congenital defects, in 9 per cent (Hough and Stuart, 1968).

Table 3. Concomitant Middle Ear Disorders Found in 31 Patients Following Severe Head Trauma

Disorder	Incidence (%)
Otosclerosis (2)	6.4
Tympanosclerosis (3)	9.0
Chronic otitis media and cholesteatoma (3)	9.0
Congenital defects (3)	9.0.

The picture of such a variety of unexpected anomalies and pathologic conditions is certainly fascinating. When one attempts to correct such middle ear injuries, these complexities tax the ingenuity of the surgeon in a unique way.

Middle ear pathologic findings resulting from closed head injuries may be conveniently divided into the following categories: (1) incudostapedial joint separation, (2) marked dislocation of the incus, (3) fracture of the stapedial arch, (4) fracture of the malleus, and (5) hyperostosis and concomitant disease.

In many skull injuries involving severe forces one may see fractures of the tympanic cavity walls and tearing of soft tissues, resulting in a massive hemotympanum or perhaps even rupture of the tympanic membrane. Despite this sudden terrible quake of destruction, a large number of these temporal bones heal spontaneously without functional defects. In many, however, there is enough disorganization to produce continued hearing loss of a conductive nature.

Incudostapedial Joint Separation

The incudostapedial joint is well known to be that part of the ossicular chain most vulnerable to destruction during inflammatory disease (Hough, 1959). Despite its rather protected position under the posterosuperior bony canal wall, this joint is also the portion of the ossicular chain most vulnerable to all the forces that injure the middle ear, regardless of direction or origin. This is the middle ear, regardless of direction or origin. This is true of those injuries occurring with objects or forces passing through the ear canal, such as foreign bodies, air compression, and water; it is also true in those instances in which forces are transmitted through the skull, as in skull fractures. The incudostapedial joint thus becomes the key to the interpretation of the otologic portion of head injuries. In many cases what happens to the incudostapedial joint will determine the method one must use to correct the conductive defects; therefore, a study of the forces and factors involved is very important.

When an object strikes the head with sufficient force, an incudostapedial joint separation frequently occurs. When the skull has been fractured so as to produce a conductive deafness, it has been found at the time of surgical exploration that the incudostapedial joint was separated in 92.3 per cent of the patients (Hough and Stuart, 1968). Our thinking is arrested by the query as to what might possibly happen to cause a separation of this joint during those traumatic moments when the injury to the head is occurring. One can consider the following possibilities, acting either as separate forces or in unison (Hough, 1959, 1968).

Effect of Concussion. An important factor is the effect of concussion. A severe vibratory reaction to an impact sufficient to produce a fracture of the skull may cause momentary separation and weakening of the tissues. The cohesiveness of the tissue is temporarily disrupted as though there had been an explosion among the cells.

Effects of Inertia. The simultaneous jarring effect tends to produce derangement of all movable parts according to the laws of physics related to inertia - that is, the effects of inertia on the head during reaction to acceleration and deceleration. When an object strikes an immovable structure, such as when the head strikes a wall, a very rapid deceleration occurs. Anything movable within the head, such as the brain or ossicular chain, tends to stay in motion, thus tending to produce disorganization. Newton's law of motion related to inertia states that "a body at rest will remain at rest and a body in motion will continue in motion with constant speed in a straight line as long as no unbalanced force acts upon it". Even when the head is stopped, the brain, ossicles, and so forth must be under a strong force to remain in motion in accordance with the laws of inertia. In an opposite way, the same is true when the head is struck by a moving object".

Action of Tympanic Muscles. The action of the tympanic muscles must be considered. The sudden tetanic contraction of the intratympanic muscles may cause severe stress in directions that would aid the forces of destruction rather than those of preservation.

Investigators have attempted to determine the maximum tension of which these muscles are capable. One investigator estimated that the tensor tympani in a rabbit exerted approximately 1.2 grams of pull when stimulated by sound. Other investigators found the stapedius muscle of a cat exerted about 1 gram of tension under experimental conditions. This would represent a substantial force in the human.

Twisting of Incudostapedial Joint. Another factor may be the twisting of the incudostapedial joint. Some authorities consider the ear to have two axes of rotation; one axis is along a line through the short process of the incus and the anterior ligaments of the malleus, and the other extends from the head of the stapes to the footplate.

The incudomalleal articulation divides the two large ossicles into two portions of equal weight. Along this axis is a line where the moment of inertia of the ossicles is at its lowest weight. The weight above and below are equal. The upper mass acts as a counterweight to the lower mass.

By utilizing certain laws of physics it can be demonstrated that the incudostapedial joint acts much like the universal joint of an automobile, because it lies between the two major axes of rotation. As the universal joint in a machine prevents a disastrous play of unbalanced counterforces from producing destruction or distortion during the transmission of energy, so the incudostapedial joint performs this vital role in sound transmission. Unfortunately, the severe stresses produced by change in the rotational axis may center in the incudostapedial joint, making it most easily dislodged when there is motion outside its narrow limits (Hough, 1959).

Torsion Effect. Certainly one of the most important aspects causing incudostapedial joint separation is the torsion effect that the skull fracture causes by shifting of the middle ear and its contents (Hough and Stuart, 1968). When a break occurs in the wall of the middle ear, as in a longitudinal skull fracture, the same powerful energy that caused the splitting of the bony wall is exerted on all the structures attached to, or lying adjacent to, the area of force. This may cause the ossicular ligaments to be torn or the ossicular chain to be pushed or pulled in the direction of the shift of the bony wall. The rather tenuous suspension of the incus between the firmly anchored malleus and stapes does, in fact, make it relatively much more vulnerable to traumatic dislocation.

Torsion is not, however, limited solely to those patients in whom there is fracture of the wall. Guerrier and colleagues (1967) emphasized the deformation of the tympanic cavity walls during concussion or compression injuries without fracture. The concentration of force is thought to center in the epitympanum. It is believed that this deformation without fracture helps to explain those cases of ossicular dislocation in which a skull fracture is not found.

Massive Dislocation of the Incus. The incus has no muscular anchor and has the weakest soft tissue attachments of all the ossicles; it is thus the weakest link and the most easily dislodged. It is the suspended span in the ossicular chain, and tremendous forces work during skull injury to fracture and displace it from its moorings. It may be trapped in all kinds of

awkward and functionless positions.

This type of injury represents much more trauma than an incudostapedial joint separation inasmuch as the articulation of the incus with the malleus, fossa incudis, and stapedial head are all avulsed, and the incus is thrown free from all its attachments. This type of drastic injury has occurred in 58 per cent of the senior author's patients with longitudinal temporal bone fractures producing ossicular chain interruption (Hough, 1968, 1969). The incus has been observed to have been completely subluxated into the middle ear. It also has been observed to turn completely over 180 degrees in the attic! The incus may be partially thrown through the fracture line and may be found to project into the external ear canal; even more dramatic, the incus may be entirely missing (Hough, 1970).

As mentioned, the incus may completely turn over 180 degrees in the epitympanic space. In this event, the tympanotomy view of the incus would show the lenticular process of the incus facing laterally. The short process of the incus would be pointing toward the head of the malleus, and the incudomallear articulating surface would be facing the mastoid antrum. It is interesting to envision this awesome occurrence at the time of skull fracture. Obviously, when the skull is fractured, for a brief traumatic second the fracture line opens wide enough for the body of the incus to be disarticulated from the malleus and to be completely turned over so that the short process of the incus is directed forward toward the malleus. Clearly, a huge opening in the lateral wall of the epitympanum must occur for this complete version to take place. Following this, the fracture line may snap back to an almost imperceptible line.

The incus has also been observed to have been trapped in the fracture line during the version turn so that the body of the incus is actually projecting into the external ear canal. The jaws of the fracture edge closed on the incus during the time that it was turning over.

Fracture and Displacement of the Stapes

The twisting torsion of the incus during skull fracture may often cause severe damage to the stapedial arch. In the previously mentioned series this occurred in 30 per cent of the patients suffering ossicular chain derangement from skull fracture. Severe injuries to the medial wall of the middle ear may be the result of mixed longitudinal and transverse skull fractures. In these patients, the skull fracture frequently involves the inner ear mechanisms but may also cause fractures around the oval window, producing stapedial displacement or breaking of the crural arch from the footplate of the stapes. Usually, however, the fracture of the stapes results from the tremendous commotion of the incus and malleus along with the other factors mentioned in the section for incudostapedial joint separation.

Fracture and Displacement of the Malleus

Fracture of the neck of the malleus is somewhat unusual during longitudinal temporal bone fractures, but it has been found in 11 per cent of patients in the senior author's experience (Hough and Stuart, 1978). These are usually accompanied by extreme fractures or dislocations

of the incus and stapes. In one patient, the neck of the malleus had been fractured and the head completely displaced out of the epitympanum into the middle ear; the stapedial arch as well as the footplate of the stapes had been fractured and the facial nerve injured. In another instance, the head of the malleus was completely missing; in still another, the head of the malleus was projecting into the intracranial cavity through a break in the tegmen.

Fibrous Replacement of Ossicles

It is generally understood that ossicular malfunction causes a low frequency air-bone gap and that this gap decreases as the frequencies tested increase. This is generally true, whether the loss is purely conductive or a mixed conductive-sensorineural hearing impairment. Exceptions to the above may be seen occasionally when fibrous tissue connects ossicular discontinuity (Anderson and Barr, 1971) and when there is an isolated fracture of the handle of the malleus (Harris and Butler, 1985). These injuries tend to cause a greater air-bone gap in the higher frequencies than the lower frequencies.

Concomitant Disease

Hyperostosis

Massive healing is required to repair the damage produced by a longitudinal skull fracture. The break in the bony wall of the epitympanum along with the tearing of ossicular ligaments and the disruptive effect of the ossicular manipulation leaves a broken, bleeding epitympanic space. The normal reparative processes of healing may be altered, and this is perhaps responsible for some of the unusual findings seen in the ears of patients with skull fractures.

Hyperostosis from osteogenic attempts to repair bony defects may cause the production of a mass of gnarled bone that encompasses the upper ossicular chain. It has been seen to extend down the long process of the incus, involving the entire body of the incus and the head of the malleus and producing bony fusion of these structures to the wall of the epitympanum (Hough and Stuart, 1968).

Tympanosclerosis

Tympanosclerosis is frequently found alone or mixed with hyperostosis. In some cases this is thought to be a post-fracture sequela; on the other hand, it may be an additional concomitant pathologic entity. Ankylosis of the ossicular chain, particularly attic fixation, is common with this condition. Its presence may obscure or confuse the etiology of morphologic changes found at the time of surgery. The author has found this double problem of fracture and tympanosclerosis to be present in 18 per cent of the ears explored for this reason. In several patients, tympanosclerosis caused marked attic fixation of the ossicular chain and epitymapnic ossicular chain erosion (Hough and Stuart, 1968).

Inflammatory Disease (Acute and Chronic Otitis Media, Meningitis, and Cholesteatoma)

Inflammatory disease of the tympanic cavity and mastoid may occur as an acute complication of skull injury or it may be a late, superimposed condition. It is the most serious concomitant pathologic process than can occur with, or following, head injury. Fracture lines or breaks in the meningeal barriers may provide easy channels for intracranial spread of infection. Breaks in the enchondral bone may form permanent fistula lines to the vestibule, semicircular canals, and cochlea. The enchondral and endosteal layers of the otic capsule are notorious in their tendency toward slow and incomplete repair, whereas the external periosteal layer may heal rapidly and completely (Perlman, 1939). Ward (1969) stated, "Previous studies of temporal bone fractures showed only fibrous union in fracture lines where there was insufficient periosteal covering". Such a union may not be an adequate barrier to the spread of infection; therefore, a potential route for infection to the subarachnoid space remains open and may be available for repeated travel to the meninges for years to come.

Cholesteatoma of the tympanic cleft or mastoid may develop following a skull fracture. This apparently results from either a traumatic fracture implantation of squamous epithelium, a fracture cleft retraction pocket, or an invasive attempt of squamous epithelium to heal a dehiscence of the tympanic membrane or attic.

Since natural barriers are sometimes broken and pneumatic cells are frequently open to rapid invasion, cholesteatoma in these cases can be more difficult to manage surgically.

Otosclerosis

The history of hearing ability prior to head injury is a very important differential point. The possibility of finding ossicular discontinuity from a previous head injury must be constantly remembered when doing a tympanotomy during stapedectomy or tympanoplastic procedures.

Congenital Defects

A skull fracture involving the middle ear in the presence of a congenital malformation may present a difficult surgical or diagnostic dilemma (Hough, 1958). One should be prepared mentally for this unexpected finding. For instance, the disappearance of the incus following severe head injury has been reported by Does and Bottema (1965). This was repeated in the senior author's experience in a patient in whom a positive history of skull fracture was given (Hough and Stuart, 1968). When the ear was entered, the incus was found to be absent. Exploration of the epitympanic region did not reveal its presence. The stapes was malformed, apparently from congenital causes, as was the facial nerve.

Inner Ear

The inner ear may be injured by either concussion or direct fracture or by both in severe closed head injuries. The pathologic finding of non-fracture injuries to the inner ear may be explained on the basis of concussion. By the same laws of acceleration and sudden deceleration mentioned in the section on middle ear injuries, the movable structures of the inner ear may undergo violent displacement. A blow to the head may produce concussion with cochlear damage occurring even on the opposite side. A violent shock wave may cause peripheral sensorineural hearing impairment by damaging the organ of Corti. Schuknecht (1969) believes this to be high-intensity vibratory energy transmitted to the inner ear and creating a hearing loss most severe in the high frequencies. The histopathologic evidence of damage to the cochlea both in experimental animals and in human autopsy findings is similar to that caused by prolonged exposure to high-intensity noise (Ward, 1969). The threshold center for mild hearing loss is around the frequency of 4000 Hz. As the energy is increased, the spectrum widens (Schuknecht, 1950, 1969; Schuknecht et al, 1951; Ward, 1969).

As previously mentioned, the vestibular labyrinth may likewise be damaged by violent displacement. Schuknecht (1969) belies the otoconia may be dislodged and henceforth become free-floating stimulators to the remaining functional end-organs, thus producing episodes of paroxysmal positional vertigo. The senior author's experience does not support this as a common possibility. These symptoms were totally absent in a rather large series of temporal bone fractures; therefore, true assessment of vertigo in its amount, anatomic origin, and relation to actual injury must still be clarified (Hough, 1968, 1969).

Severe damage to various portions of the vestibular end-organ system by a disruptive concussion would not only produce a partial functional labyrinthectomy but could also allow an unbalanced labyrinthine disharmony of the remaining functional units. A direct fracture onslaught of the inner ear usually destroys both by mechanically separating vital tissues and by the concussion mentioned previously.

A sensorineural hearing loss of varied degree is, therefore, an extremely frequent finding in head injury. Indeed, if studied well, some sensorineural hearing loss is most likely to be present in all cases of injury sufficient to produce a skull fracture.

Surgical Management of Ossicular Chain Derangement Following Injury

Because of the tremendous number of variables in ear injury, such as area and force of injury, anatomic variations of individuals, and concomitant pathology such as tympanosclerosis and otosclerosis, operative techniques cannot be followed in a steplike fashion but must be personalized. Despite the lack of uniformity, however, general classifications of injury effects can guide the surgeon, and known surgical methods may be done successfully. The following classification has been given in the previous discussion of pathology.

Incudostapedial Joint Separation

Realignment by Repositioning

The most frequent ossicular chain injury is separation of the incudostapedial joint. Frequently the incus is displaced, but it may be loosened, manipulated, and replaced to its original position so that its articulation with the stapes is restored. Even when there is massive dislocation of the incus, with disruption of all three articulations, the incus may often be replaced. A small amount of bone from the posterosuperior canal wall may be removed to provide a moderate exposure of the epitympanum. A gelatin sponge (Gelfoam) bed is provided in the attic and the incus is replaced in its original position. This cannot be done in all patients because of anatomic limitations.

Simple realignment and repositioning of the incus to its original site is the most physiologic operation that can be accomplished.

Sculptured Ossicular Transplant

Fractures of the long process of the incus or concomitant disease in the attic, such as tympanosclerosis or hyperostosis, may make it necessary to bypass the upper ossicular chain. In such cases, removal of the incus remnant from the attic can be done so that it can be used as a bone graft. After careful sculpturing of the body of the incus, the newly prepared graft may be tightly fitted over the head of the stapes and clamped around the handle of the malleus. This sculpturing should be done precisely with small cutting burs while the ossicle is firmly held with a Sheehy bone forceps. If the incus body cannot be used, the neck of the malleus may be cut with a House-Dieter malleus nipper, and the head of the stapes removed from the epitympanum and sculptured as a graft. If neither of these bones is usable, a suitable incus or head of malleus homograft may be used, with equally good results. Careful measurement and sculpturing are absolutely necessary so that the newly designed ossicle will fit snugly in place. If the stapes is in good condition, one may expect a high rate of success with any one of these procedures.

Marked Dislocation of the Incus With an Intact Stapes

The previously discussed techniques are also applicable when this more serious displacement of the incus has occurred. This condition, representing an increase in force and resultant injury, is usually associated with more drastic alteration of other ossicular elements as well as the walls of the middle ear.

Fracture of the Stapedial Arch

A fracture of the stapedial arch presents an entirely new group of requirements in reconstruction. The twisting torsion of the incus as it responds to a fracture force frequently causes severe damage to the stapedial arch. Complete subluxation of the entire stapes out of the oval window is possible; usually, however, the crural arch fractures at its weakest anatomic point

- that is, where the arch meets the footplate.

In such an instance, the footplate of the stapes is still intact and mobile, but sound energy is not reaching it. One must reconstruct the conductive mechanism from the footplate of the stapes to the incus, malleus, or the tympanic membrane. In the senior author's experience, the use of a prosthesis from the incus to the footplate is less satisfactory and more hazardous than the use of an ossicular bone graft reconstruction from the stapedial footplate to the handle of the malleus. Likewise, the use of an artificial prosthesis of either plastic or metal from the footplate of the stapes to the handle of the malleus and/or the tympanic membrane is unsatisfactory. In the senior author's opinion, this procedure should be condemned. It causes numerous complications and produces inferior functional results. As one might expect, natural materials (sculptured human ossicles) have proved their ability to outlast and outperform artificial materials.

It is now the senior author's practice to reconstruct the ossicular chain from the mobile stapedial footplate to the handle of the malleus by bridging the distance with a sculptured ossicular bone graft. This may be the patient's own incus, if available, or a homograft.

In utilizing bone grafts, the procedure is to remove the incus from the epitympanum. If the long process is intact, a carefully measured sculptured ossicle is then form with cutting burs. The notched end is then snugly fitted around the handle of the malleus, and the long process of the incus is rested on the mobile footplate of the stapes. The position is usually secure but may be further stabilized with a gelating sponge (Gelfoam) cast.

Fortunately, the distance between the tip of the long process and the superior margin of the body of the incus is always sufficiently long to provide enough bone for sculpturing so that the graft may adequately form a secure bridge from the stapedial footplate to the handle of the malleus. This technique of incus transposition with some modification in sculpturing has been used by the senior author since 1957, and has been a foundation in the tympanoplastic surgical armamentarium since that time (Hough, 1960).

On rare occasions, the footplate of the stapes is fixed with concomitant disease. In this instance the footplate may be removed and a tragal perichondrial graft used as a seal for the oval window. The long process of the above-mentioned sculptured incus may then be placed in the center of the perichondrium and the proper connection made to the handle of the malleus. Perichondrium is unique among oval window grafts in that it is firm enough, if used properly, to allow this type of bone graft reconstruction without subluxation into the labyrinth.

On some occasions the long process of the incus is found to be fractured or, even more rarely, the incus may be completely lost due to injury. In this event, a homograft incus may be sculptured and used as outlined above with equal success. Likewise, and even more preferable, a homograft handle of the malleus may be used as demonstrated. This is particularly advantageous if the oval window is extremely narrow or if mechanical advantages seem to indicate that this ossicle would function more satisfactorily. The homograft handle of the malleus is sculptured and fitted as shown.

Stapedectomy with Preservation of The Posterior Crus and Perichondrial Graft

This procedure may be indicated when the footplate of the stapes is fixed in the oval window by complicating lesions, such as otosclerosis, tympanosclerosis, or post-traumatic osteogenesis. At present, the authors would use tragal perichondrium to cover the oval window. The posterior crus of the stapes can be placed nicely in the center over the oval window perichondrial graft.

Because of our general tendency to think in terms of total stapedectomy with the use of a prosthesis, one might be inclined to simply remove the stapedial arch and use a replacement prosthesis. This, of course, is totally unnecessary and should not be done in such a case.

Fracture of the Malleus

Fracture of the neck of the malleus is somewhat unusual during a longitudinal temporal bone fracture but has been found on occasion. Fracture of the head of the malleus is usually accompanied by severe fractures or dislocations of the incus or the stapes. Although the head and the neck of the malleus may be dislocated even through the tegmen or down into the middle ear, the handle of the malleus is usually still adherent to the tympanic membrane in its original position and can be utilized as a base for reconstruction of the ossicular bridge from the eardrum to the oval window. The sculptured incus or a sculptured homograft handle of the malleus is ideal for this repair.

Total destruction of the ossicular chain may leave one without the stapes superstructure, the incus, or even the handle of the malleus attached to the tympanic membrane. Reconstruction of the tympanic membrane and ossicles must be accomplished in such a case. A fascial graft placed medial to the annular rim and drum remnant, and medial to the tympanomeatal flap, will readily solve the tympanic membrane problem. One may then use a homograft handle of the malleus and the medial surface of the tympanic membrane graft and reconstruct with ossicular graft transplants from the footplate of the stapes to the oval window to the above-mentioned handle of the malleus. This is more difficult to do because of graft instability and may require a two-stage procedure.

Results

From the standpoint of hearing restoration in these cases of conductive deafness caused by middle ear ossicular derangement, the results following surgical repair have indeed been excellent. Considering all methods used in a series of 31 consecutive cases, 78 per cent of the ears were restored to within 10 dB of the preoperative bone conduction in the three speech frequencies (512, 1024, and 2048 Hz). In 45 per cent, the hearing was equal to or better than the preoperative bone conduction threshold in the three speech frequencies (closed or overclosed the air-bone gap). In one case the hearing was worsened (Hough and Stuart, 1968).

Water Sports Injuries

Water sports must be recognized as a major cause of middle ear injuries. Obviously the geographic location will partially determine the frequency with which this occurs; however, in all areas this problem increases with the increase in popularity of water sports.

In the senior author's experience, diving was responsible for the largest number of injuries (48 per cent), and water-skiing was responsible for 30 per cent. However, although many more people swim than water-ski, we must recognize that water-skiing is proportionately a much greater danger to the ear than any other water activity. Since prevention is a professional responsibility, it should be emphasized that all these injuries could have been completely prevented by the use of ordinary earplugs.

In the author's experience, these accidents caused perforation of the tympanic membrane in all cases. The perforation is probably produced by the hydraulic compression of air against the tympanic membrane as the water is forced into the external ear canal. This could conceivably cause other damage such as ossicular chain separation, rupture of the round window membrane, and lysis of the annular ligament in the oval window (see the section on Air Pressure Injuries). These two latter complications have not been observed, probably because of the perilymphatic protection behind the windows.

When injury to the tympanic membrane occurs from water pressure, there seems to be no particular design or specific area where the membrane is ruptured. Perforations appear in all sizes. In the senior author's series, 30 per cent occupied two-thirds to one-half of the tympanic membrane; in 20 per cent, less than one-half of the drum area was involved. These perforations appeared in all quadrants of the membrana tensa, but none occurred in the membrana flaccida. There was no uniform characteristic as to perforation shape.

An important complication must be emphasized in this group of patients. Because of water contamination of the middle ear, one should expect many to suffer an immediate postinjury otitis media. In addition, in water sports injuries there is an amazingly high incidence of chronic as well as acute post-traumatic suppurative otitis media. In approximately 50 per cent there will be chronic otorrhea, and in many of these the infection will persist for years. In all these patients it is necessary to institute careful specific medical therapy in order to abolish inflammation prior to surgical repair. These inflammatory sequelae are probably responsible for the striking finding in the senior author's experience of an incudostapedial joint separation from necrosis of the incus in over 50 per cent of patients suffering water sports injuries to the ears!

The possibility of squamous epithelium transplants or a misdirection of squamous epithelium producing cholesteatoma must be considered. This occurs in approximately 5 per cent of patients and is probably due not to the fact that water injuries are mechanically unique but rather to the tendency for otitis media to develop as a postinjury complication.

Welding Injuries

The advantage provided by the anatomic structure of the ear is clearly demonstrated when one studies welding injuries to the ear. The small, narrow, tortuous tube guarded by hair and a coating of cerumen at its exterior entry provides excellent protection of the delicate middle and inner ear sound receptors. As a result, hot beads of metal that constantly burst from the point of the weld are trapped in the external ear canal and thus are usually prevented from reaching the tympanic membrane. Although localized burns on the canal are painful, ordinarily there is no permanent damage in this location. Because of the frequent industrial necessity for welding, it is not too uncommon to see a patient who has suffered the experience of the hot metal bead finding its way down the ear canal and landing on the tympanic membrane, causing a perforation of the tympanic membrane.

It is generally believed by most otologists that these perforations are resistant to repair. This is thought to be caused by a loss of tissue vascularity due to the cautery effect of the burn, followed by healing with a dense avascular scar. Thus, an increased susceptibility to both graft rejection and perhaps superimposed infection is thought to exist.

Although little has been written on this subject, the author's experience with these injuries emphasizes several significant characteristics.

1. A welding burn to the tympanic membrane and mucosa of the middle ear causes it to be unusually vulnerable to infection. In 85 per cent of patients the ears became chronically infected. This does not always occur immediately but usually appears within 3 months of the injury. In 25 per cent, the mastoid and epitympanum became chronically infected because of superimposed secondary infection prior to surgery. Despite the extent of the infection, after adequate medical control and surgical repair good restoration of hearing has been accomplished.

2. The actual loss of tissue caused by the burn involves only the tympanic membrane. In the author's experience an ossicular chain involvement was not observed as the result of a welding injury.

3. Since the advent of the interior temporalis fascial grafting technique, the authors have found no predisposition to graft failure among these patients. Improved methods of infection control and better techniques of tympanic membrane grafting place these patients in a good position as candidates for successful surgical repair.

Air Pressure Injuries

Barotrauma

Barotrauma in the context of this chapter refers to physiologic or pathophysiologic effects that result from the changes in the ambient pressure that affects the air spaces of the temporal bone. These changes occur because of the inability of the eustachian tube to respond to the pressure difference that naturally occurs secondary to the laws of physics on the change of altitude. Boyle's law states that, at a constant temperature, the volume of a gas varies inversely with the pressure. As the pressure increases (atmospheric), the volume decreases. Barotrauma is most commonly seen in divers and airline passengers and pilots.

Barotrauma from SCUBA Diving

Since its invention in 1948, scuba diving (*s*elf *c*ontained *u*nderwater *b*reathing *a*pparatus) is ever increasing in popularity. It has been estimated that there are 2 to 4 million sports divers, with 250,000 divers being trained each year (Gunby, 1981). Lundgren and associates (1974) sent questionnaires to 4000 members of the Swedish Sport Diver's Association and to 200 members of the Royal Swedish Navy. Approximately 50 per cent indicated that they had experienced vertigo at some time while diving. These figures do not include other symptoms that occur in barotrauma such as hearing loss, membrane ruptures, and pain. When the trauma does occur, it may take place in the ear canal, middle ear, or inner ear. Of the three, middle ear barotrauma occurs most commonly.

Ear Canal Barotrauma

For ear canal barotrauma to occur, there must be a blocked external ear canal so that trapped air is present within the canal. This may occur with cerumen impactions, use of earplugs, or with a stenosis of the external ear canal. In this event, the volume of air or pressure will vary according to Boyle's law. The phenomenon of ear canal barotrauma is frequently referred to as "reverse ear squeeze".

Pain is the most common symptom. Blebs of the ear canal and tympanic membrane are frequently seen. There may be petechial hemorrhages of the skin, and the tympanic membrane may rupture. Treatment includes gentle cleansing of the ear canal and removal or correction of the underlying cause. The tympanic membrane is repaired as with any other perforation.

Middle Ear Barotrauma

Most diving disorders due to barotrauma occur in the middle ear. When barotrauma occurs on the descent and involves the middle ear, this is called "middle ear squeeze". This is the most common diving injury (Dickey, 1984) and occurs when there is failure to equalize the pressure in the middle ear with the ambient environment. The deeper the diver descends, the greater the pressure. The symptom first experienced by the diver is usually a "pressure feeling". If the

eustachian tube does not open to equalize the pressure difference, then barotrauma will occur. The tympanic membrane is pushed medially and pain begins. If the diving continues, blood vessel rupture and mucosal tears will occur in the middle ear, and the tympanic membrane may rupture. Pressure differential from 100 to 500 mm Hg (4.3-17.4 feet of salt water (FSW)) can cause tympanic membrane rupture (Farmer and Thomas, 1976). Middle ear squeeze occurs in 30 per cent of neophyte divers during their first compressed-air dive, and in approximately 10 per cent of experienced divers (Dickey, 1984).

Of the divers who terminate a dive because of pain, more than 75 per cent do so within the first 33 FSW (Spivak, 1980). 33 FSW represents 2 atmospheres at sea level; thus, at this depth, the volume of the middle ear space will be cut in half compared with that present at sea level. If the tympanic membrane does rupture, water enters the middle ear, and this may result in vertigo due to caloric stimulation of the vestibular labyrinth. This type of vertigo, which occurs on descent, is not to be confused with the most common cause of vertigo in divers - alternobaric vertigo, which occurs on ascent (discussed later).

If vertigo does occur on descent secondary to middle ear squeeze, the diver may be totally disoriented, and his life may be endangered. For this reason, scuba diving should never be performed alone.

It is imperative for all divers to learn to equalize middle ear pressures by way of the eustachian tube. If the eustachian tube is functioning improperly, symptoms first appear at a depth of 2.6 FSW (60 mm Hg). In order for the diver to continue to descend, equalization via the eustachian tube must be done every 2 to 3 feet. If the pressure differential extends to 90 mm Hg, the eustachian tube becomes "locked", due to the nasopharyngeal valve effect (Stucker and Echols, 1971; Taylor, 1959), and the eustachian tube will not open.

Inner Ear Barotrauma

Although not as frequent as middle ear barotrauma, inner ear barotrauma is more serious, having the potential to cause permanent damage. It is almost always associated with the inability to equalize the pressure in the middle ear, causing damaging changes in the hydrodynamics of the inner ear fluids. It has been postulated that sudden pressure changes in cerebrospinal fluid pressure can cause this effect (Becker and Parell, 1979; Freeman and Edmonds, 1972; Strauss, 1979). The former would create an implosive force at the oval window or round window; the latter would result from a transmission of the CSF pressure through the cochlear aqueduct, causing an explosive force in the cochlea that could cause a rupture of the round window, the oval window, or the intracochlear membranes.

The sensorineural hearing loss that results may be total or partial. Fistulas from barotrauma are like other fistulas in that the longer they are present, the more likely that hearing will deteriorate. Vertigo in patients with fistulas resulting from barotrauma is not a frequent finding; Freeman and Edmonds (1972) reported that only 20 per cent of these patients had vertigo.

Most investigators believe that once the diagnosis of a fistula has been made, immediate surgery should be carried out, as this offers the best opportunity for optimal recovery (Becker and Parell, 1979; Caruso et al, 1977; Freeman et al, 1974; Pullen et al, 1979). Others (Goodhill et al, 1973; Singleton et al, 1978) have recommended bed rest for a few days and exploration later if there is no improvement.

Barotrauma from Airplane Travel

The same pathophysiologic events occur in barotrauma from airplane travel as with scuba diving. Most barotrauma from plane travel occurs on descent. However, alternobaric vertigo (discussed in the following section) occurs in ascent. Barotrauma from diving occurs with pressure changes of only a few feet, while airplane travel pressure changes are related to thousands of feet. For example, 18000 feet above the sea level is only one-half of an atmosphere change, whereas going to a depth of only 16.5 feet in water produces the same amount of atmospheric change (1/2). Once barotrauma has occurred in airplane flight, the signs and symptoms are the same as with barotrauma while scuba diving.

Alternobaric Vertigo

Alternobaric vertigo occurs with both airplane travel and water sports diving. It is different from other types of barotrauma in that it occurs on ascent. It is short-lived, lasting from a few minutes up to several hours, and hearing loss and tinnitus usually do not occur

Ingelstedt and colleagues (1974) showed that this condition is caused by relative overpressure in the middle ear, and that it occurs only when one middle ear has a relative overpressure while the contralateral eustachian tube is functioning normally.

These investigators found that by ENoG testing during pressure changes that alternobaric vertigo occurs only on ascent after one eustachian tube has opened passively while the other remains closed, causing a relative overpressure in that middle ear.

In a questionnaire to divers by Lundgren (1965), vertigo of some type was experienced by 26 per cent. Of these divers, 73 per cent had vertigo only on ascent (alternobaric), whereas 19 per cent had vertigo on descent; the remainder had a combination of the two.

Alternobaric Cranial Nerve VII Paralysis

Similar to alternobaric vertigo, alternobaric cranial nerve VII paralysis occurs on ascent and is rare and usually ephemeral. It occurs in diving as well as airplane travel and is temporary and reversible. The mechanism of this paralysis could be an ischemic neuropraxis of the facial nerve produced by excessive pressure in the area of dehiscence so commonly found in the horizontal portion of the fallopian canal.

Injuries Due to Sudden Pressure Changes (Blast Injuries)

These accidental injuries have a variety of causes. Victims' experiences are often quite dramatic as well as tragic. In the senior author's experience, one young man had a home-made bomb of his own design explode in his hand, causing loss of his hand, a huge perforation of the tympanic membrane, and permanent cochlear acoustic trauma. Another was injured when a motor exploded. Furnace explosions, pressure chamber intolerance, shell blasts, barotrauma from aircraft altitude changes, and even a blow from a snowball have been responsible for other injuries in this series. Syringing for wax removal and even a eustachian tube inflation may cause a rupture of the tympanic membrane. However, it is doubtful that either syringing or inflation will produce a perforation unless the tympanic membrane has already been weakened by a previous injury or disease.

The most common etiology, in the author's experience, was a human adversary. Slightly over 50 per cent of patients gave a history of having been struck by another person.

Unlike water injuries and welding burns, most of these patients did not experience purulent drainage from the ear in the long-term postinjury period (66.6 per cent). Furthermore, the results with tympanoplastic repair were better in this group than in those with water sports or welding injury. This is logical, in that these ears were not subject to inflammatory disease as often as the water injury group and did not suffer avascular scarring as did the welding burn group.

Ossicular chain injury, either fracture or separation, is extremely rare even in severe air blast injuries. This is in sharp contrast to the frequent ossicular destruction found in ears damaged by foreign bodies and the ossicular damage (usually necrosis) caused by water sport injuries. Rupture of the round window and/or oval window membranes due to disproportionate pressures may occur on rare occasions. This may be caused by excessive negative or positive air pressures within the tympanic cavity, with or without changes in fluid pressures within the inner ear. Rapid changes in altitude, lack of proper underwater decompression, and unusual abdominal straining are among those factors believed to cause accidental rupture of the membrane of the oval or round windows. The symptoms may be sudden loss of hearing and vertigo. Tympanotomy with visualization of the window membranes should be considered. If a membrane defect actually exists, a soft tissue graft should be used to seal the opening.

In the senior author's experience, the perforations of the tympanic membrane caused by all air pressure changes, whether gradual or sudden, occurred in (1) the anteroinferior quadrant in 42 per cent, (2) the posteroinferior quadrant in 17 per cent, (3) the anterior half of the tympanic membrane in 17 per cent, and (4) superiorly alongside the handle of the malleus in 8 per cent. The tympanic membrane (membrana tensa) was totally destroyed in 8 per cent, and the posterosuperior quadrant was involved in only 8 per cent. This pattern suggests that the anteroinferior portion of the tympanic membrane is more vulnerable to perforation from sudden air pressure changes in the external ear canal. The membrana flaccida was not ruptured.

Otologic blast injuries from explosions may be primary, secondary, or tertiary. Primary blast injuries are the most common and involve injury secondary to the effects of the blast wave alone. Secondary blast injury results from flying debris striking the head or the ear. Tertiary injury results from the patient being bodily moved by the blast, with ear injury occurring on impact with the environment when the patient comes to rest.

When an explosion occurs, there is a very short positive-pressure phase, in the order of milliseconds, in which pressures of a hundred or even thousands of pounds per square inch are reached (Kerr and Byrne, 1975). This is followed by a longer, negative phase, in the order of tens of milliseconds, which cannot be greater than the atmospheric pressure. This positive and negative phase is referred to as the Friedlander curve.

Kerr and Byrne (1975), reporting on the Abercorn Restaurant bombing that occurred in Belfast in 1972, observed that children suffered less tympanic membrane trauma than adults even though exposed to the same pressure environment. Of the 60 tympanic membrane perforations that occurred, 81.7 per cent healed spontaneously; 4 per cent had squamous epithelium transplanted into the middle ear. Seamen and Nowell (1971), in their series of 110 patients, reported 12 per cent had squamous epithelium in the middle ear, strongly suggesting that it is the positive phase of an explosion that produces perforation of the tympanic membrane.

Almost all individuals exposed to severe blasts suffer temporary deafness, frequently quite severe. In most cases, the sensorineural loss recovers quickly; however, when the blast is of severe degree, permanent loss may result. Teter and coworkers (1970) observed four audiologic categories in patients suffering sensorineural hearing impairment: (1) 36 per cent had a dip at 1000 Hz; (2) 15 per cent had a dip at 1000 Hz; (3) 20 per cent had a dip at 4000 Hz; and (4) 28 per cent had a sloping loss. It is interesting that most observers found very little recruitment in those patients with sensorineural loss resulting from blast injuries.

Foreign Body Injuries

As previously mentioned, the conductive mechanism in human hearing is carefully protected from foreign bodies by a long and tortuous canal housed in bone. Furthermore, in order for an instrument of destruction to reach the tympanic membrane, it must pass over an area provided with one of the most generous sensory nerve supplies found in the body. In spite of these protective mechanisms, however, the ear is subjected to frequent injury by foreign bodies from the exterior. This is so common that almost every persons learns the old adage, "Don't put anything in your ear smaller than your elbow".

The list of offending foreign bodies that may injure the ear is endless. It includes almost anything small enough to enter the external ear canal - beads, wheat kernels, corn seeds, bullets, pencils, erasers, Q-tips, stones, twigs, wooden matches, beads, insects, hairpins.

Age and Sex Incidence. Foreign body injury, as one would suspect, is primarily an affliction of childhood. In the senior author's series, 73 per cent of the patients suffered injury

during childhood; the mean age was 6.4 yrs. Prevention by education, is therefore, difficult in this group. Among the adult patients (27 per cent) the injury was caused most frequently by accidentally bumping the arm while the ear was being cleaned with an instrument.

Tympanic Membrane Perforations

Almost all the perforations resulting from foreign body trauma occur in the posterior half of the tympanic membrane; most of them are quite large. In the senior author's experience, 87.5 per cent of the patients suffered a loss of over one-half of the tympanic membrane area as a result of foreign body injury.

Ossicular Chain Injuries

Foreign bodies may cause not only injury to the tympanic membrane but often severe middle ear and occasionally inner ear destruction. It is interesting to note that in all the ears surgically repaired because of foreign body injury, the senior author has found severe ossicular chain injuries in over one-half. These injuries are usually related to the incudostapedial joint, the tapes, and the long process of the incus. Repair is accomplished in the same manner as described for closed skull trauma and fracture cases.

In one unusual case, a Q-tip was accidentally forced into the ear, resulting in fracture and subluxation of the stapes into the vestibular labyrinth. The tympanic membrane healed spontaneously after the original injury, and only a slight conductive hearing impairment remained. It is interesting that this patient had a patent perilymphatic fistula for approximately 1 year before it was finally repaired. During this time, he gave no history of the symptoms that are commonly attributed to perilymphatic fistula.

Removal of Foreign Body

An inert foreign body may only partially occlude the external canal and may be easily removed with forceps or a right-angle hook. Other foreign bodies may be large enough to occlude the canal completely, making the removal more difficult. This is especially true of hygroscopic vegetable foreign bodies, which swell and may also produce edema with an inflammatory reaction.

It is a common experience among otologists that, in almost all childhood injuries, some type of amateur (parental or unskilled professional) removal was attempted prior to the time the patient was seen by the otologist. In many children, the unskilled attempt at removal was responsible for much damage to the conductive mechanism. The public and our professional colleagues should be made more aware of such dangers and of the importance of careful microscopic removal of foreign bodies from the ear.

Difficult-to-remove or slippery foreign bodies, particularly in children, should be removed under general anesthesia. In addition, a local anesthetic agent combined with a generous amount

of epinephrine should be injected into the skin of the ear canal. This prevents excessive bleeding during manipulation, which is likely to occur because of the natural vascularity of the skin of the external canal plus the inflammatory reaction to the foreign body.

An axiom in foreign body removal is that one must "get behind" the foreign body. A simple armamentarium of straight forceps, various-sized suction tips, and a large right-angle hook is usually all that is needed for foreign body removal. Occasionally, however, a more complicated problem arises. One should, therefore, be prepared technically to proceed immediately to a more involved technique. When the canal is completely occluded by an expanding foreign body and is surrounded by marked edema and inflammation, a mastoid canal approach may be most reasonable. Similarly, a perimeatal tympanotomy may be called for when the foreign body has entered the middle ear.

The many facets of the subject of trauma to the ear have been described only generally; however, two conclusions are manifest. First, otologic trauma is as fascinating in its complexity and its widespread ramifications as it can be devastating to the well-being and even life itself of an individual. Second, the management of the care of the injured and the surgical repair of devastated or destroyed anatomic otologic structures challenges the otologist in every area of expertise. Indeed, if one can master otologic trauma, one is a master in the profession.