Section of Otology

President Philip Reading MS

Meeting May 2 1969

Fractures of the Temporal Bone and Associated Middle and Inner Ear Trauma

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Surgical Aspects of Temporal Bone Fractures

More young adults are now killed and injured from automobile accidents than from any other cause. No longer is disease or war the greatest threat to life. Of these injuries, more than 75% are head injuries. 'In severe head injury, the ear is the most frequently injured sensory organ in the body' (Hough & Stuart 1968). Temporal bone fractures or basilar skull fractures are extremely common in any injury to the head. Since the temporal bone is the domain of the otologist, it is his responsibility to help prevent and treat these devastating injuries.

The middle ear effects of skull trauma were little known before 1956 (Ballantyne 1962). The operating microscope and the introduction of Rosen's tympanotomy incision have led to the discovery of many conductive lesions previously considered extremely rare. Until recently it was thought that nothing could be done to correct the hearing loss due to head injury, because the damage was considered to be always in the inner ear and, being sensorineural, was irreparable (Hough 1959).

It was not until 1956 that ossicular chain injuries due to skull fractures were discovered in the living (Hough 1958, 1959). Thorburn (1957) was the first to describe this in the scientific literature. My experience was a short time later. During the next ten years only 45 cases were reported (Ballantyne 1962, 1966). This would lead one to believe that this problem is indeed quite rare, but it is not. I have personally operated on 37 ears with ossicular chain derangement due to head injury since 1956.

Initial Management of Acute Head Injuries

Injuries involving the respiratory and cardiovascular systems may require urgent treatment. Laryngoscopy with intubation or tracheotomy and suction may be needed immediately to ensure proper oxygenation. The brain weighs only approximately 2% of the entire body weight, but utilizes 20% of the total oxygen uptake of the body; furthermore, it continues to use this much energy even during sleep. The life centres in the brain are, therefore, more dependent on efficient respiratory function than any other tissue. Another factor of importance is that the brain not only needs good oxygen exchange, but must receive a more abundant circulation than the remainder of the body. It normally receives only 15% of the blood supply to the body, but requires that 20% of the body's oxygen supply be delivered to its neurones. Thus, there is a greater extraction of oxygen from each unit of circulating blood through the brain than elsewhere. This increases the brain's vulnerability to hypoxia whether caused by circulatory or pulmonary embarrassment.

Therefore, after a proper airway is established, the circulatory system should be carefully evaluated from the standpoint of blood pressure and cardiac output. Shock and hæmorrhage must be controlled rapidly and efficiently. Hæmorrhage from the head may require immediate surgical intervention. For instance, severe intractable hæmorrhage from the external ear canal may occur from rupture of a major blood vessel, such as a dural sinus or the middle meningeal artery. Less severe bleeding from the external ear canal is a very frequent symptom of temporal bone

fracture occurring from a fracture of the bony canal and/or a laceration of the tympanic membrane. Surgical control in these cases is unnecessary, but the amount and consistency of discharge and, if possible, the condition of the tympanic membrane should be carefully recorded. Much care should be exercised in keeping the external ear canal clean and uncontaminated. Cerebrospinal fluid otorrhœa may be present, predisposing to meningitis. Furthermore, if the ear becomes infected, a later surgical reconstruction may be hindered.

Internal hæmorrhage may be present within the chest (cardiac tamponade, hæmothorax), or the abdomen (ruptured viscus, such as spleen or kidney). An important principle which should always be kept in mind is that hypotension in closed head injuries is almost always the result of blood loss and usually not caused by the head injury itself. This hæmorrhage occurs most frequently within the chest or the abdomen. An 18 gauge needle may easily demonstrate its presence.

Vasoconstrictor drugs should not be used to correct hypotension. Attention should be directed towards replacement of intravascular fluid volume. To accomplish this, a double intravenous infusion should be started, using large needles. A physiological solution (Ringer's lactate) should be given until blood grouping and cross-matching can be done. If no more than 750 ml of blood has been lost, it is preferable to give up to 2,000 ml of Ringer's solution until the hypotension is controlled rather than to subject the patient to a possible transfusion reaction. If hypotension is not controlled before cross-matching, O type blood should be given without reservation even before cross-matching.

Evaluation of the Patient with Temporal Bone Fractures

After good pulmonary and circulatory function has been established, the patient must be continuously observed while the more detailed investigation continues. The state of consciousness is extremely important. 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 and whose pupils are dilated has a very poor outlook. A deteriorating state of consciousness is very ominous. The development of hemiparesis with dilatation of one pupil suggests a developing intracranial hæmatoma.

Cerebral angiography is the single most valuable procedure in evaluating the patient with a severe head injury and the more recent and very safe ultrasonic echo encephalography is very valuable in determining a shift of the brain structures from the midline. Both these procedures may be lifesaving in localizing expanding intracranial masses, such as hæmatomas. Spinal fluid examination is of little value in acute head injuries and may be misleading or even dangerous.

X-ray examination of the skull is elective, but may 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 neurovascular structures. Initially, œdema and hæmorrhage are far more important than the study of bony fracture lines.

Most of the things mentioned above are ordinarily the concern of the neurosurgeon and physician. The otolaryngologist is, however, very much a part of the head-injury team. In about 2,000 cases of cranial trauma hospitalized and observed by Guerrier *et al.* (1965–6) and Guerrier *et al.* (1967), 290 (15%) suffered injuries to the ear. Long ago Passow stated: 'In severe skull trauma, the ear rarely escapes undamaged.' The otolaryngologist is indispensable because of his special methods of examination of the nose, nasopharynx, throat and ears, and his better acquaintance with the functional tests of the two cranial nerves most likely to be injured, the VII and VIII.

Examination

In the first examination of the ear as much specific information as possible should be obtained, but never at the expense of the general welfare of the patient by requiring over-exertion or unnecessary movement.

One should determine as far as possible the functional ability of the cranial nerves. Nystagmus, facial weakness or paresis, and objective hearing loss should be specifically observed. Ecchymosis over the mastoid area often indicates blood in the pneumatic spaces of the temporal bone. The presence of blood in the ear canal suggests the possible presence of a longitudinal basilar skull fracture with a torn tympanic membrane and middle ear damage. An intact tympanic membrane with blood behind the drum indicates an explosive break of the labyrinthine capsule caused by a transverse temporal bone fracture extending across the base of the skull. Cerebrospinal fluid otorrhœa may occur 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, the ear should be cleaned, with absolute asepsis, under magnification. Secondly, systemic antibiotics should be given to prevent infection.

Facial Nerve Paralysis

This most unfortunate problem may be immediate or delayed, partial or complete. If facial paralysis

is immediately present, repair must wait until the patient's general condition permits surgical exploration. If facial paralysis appears later, one must follow its course by the appropriate tests.

When first seen, if feasible, the patient should receive the full gamut of VII nerve investigation, including nerve excitability, salivary flow, taste and lacrimation tests. With this battery of clinical tests, one can usually determine the site of the lesion as well as obtain good prognostic information.

Pressure on the nerve may be from hæmatoma, œdema, infection or bone fragments. If nerve excitability tests show sudden or progressive deterioration, the ear should be surgically explored early and without hesitation, even though the exact cause is speculative. Surgical delay in reconstruction of the ossicular chain or tympanic membrane to restore hearing may not affect the end-result, but the matter of facial paralysis is entirely different.

Kettel (1950) believes that an immediate paralysis should be surgically explored as soon as the patient's condition permits. I concur with this if nerve excitability tests demonstrate a poor prognosis, but would delay if, and as long as, reactions to stimuli are bilaterally symmetrical.

The temporal bone fracture itself very rarely causes total paralysis of the facial nerve; I have not seen it and neither has Guerrier in a large series (Guerrier *et al.* 1965–6, Guerrier *et al.* 1967, Hough & Stuart 1968). A temporal bone fracture usually produces hæmorrhage by breaking the vessels of the fallopian canal causing an outflow of blood or œdema within the sheath so that there is cessation of axioplasmic flow. If continued, paralysis is inevitable and frequently irreversible unless decompression is done. To reduce œdema, chorda tympani neurectomy should probably be accomplished with nerve decompression.

Technique of Decompression

I prefer to enter the ear through an endaural incision. I lift the tympanomeatal flap and explore the facial nerve 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 is far superior in time and safety and provides a complete temporal bone view of the entire osseous course of the facial nerve.

Unfortunately, many of our medical and surgical colleagues are unaware of the importance of early facial nerve surgery in the severely injured patient and of the therapeutic benefits possible with modern aural microsurgery. The chance of facial nerve injury is much less in a longitudinal fracture than in a transverse fracture of the temporal bone. The VII cranial nerve is damaged in fewer than 20% of the cases with longitudinal fracture and in approximately 50% of the patients with a transverse temporal bone fracture. In my experience, recovery without decompression is common, but among those who recovered spontaneously before I was able to see them, two-thirds had incomplete recovery with residual weakness, synkinesis, facial tics, &c. (Hough & Stuart 1968).

Vertigo

Loss of equilibrium may occur dramatically with head injury with or without fracture of the temporal bone. Disruptive changes, hæmorrhagic areas, paralysis of nerves and œdema may occur in the brain stem, vestibular centres, over the cerebral cerebellar cortical areas, or in the vestibular labyrinth. The fracture line of a transverse fracture of the temporal bone may directly involve the otic capsule with all or part of the vestibular mechanisms involved. It may also cause a direct injury to the VIII nerve. This in effect dismembers the cochlea and vestibular labyrinth with total loss of hearing and vestibular function on the side involved. In this event, the patient will experience severe vertigo until central accommodation occurs. This usually takes place in about two or three weeks and relatively little post-traumatic vertigo is experienced as a residual disability.

Concussion causes shock waves through the membranous labyrinth (Schuknecht 1969). Cochlear injury manifested by a sensorineural hearing loss in the high frequencies very often occurs in any severe head injury. The vestibular end-organs are subject to similar injury although probably to a much lower degree. It is thought that this might be the result of the intense acceleration and sudden deceleration causing the otolithic membrane of the utricular and saccular structures to lose otoconia by detachment into the endolymph. Schuknecht speculated that these free floating particles of otoconia might intermittently move with certain motions to contact and stimulate the intact end-organs of the various semicircular canals, thus producing sudden episodic attacks of positional vertigo.

Evidence is accumulating that many cases of post-injury vertigo may be due to central and not labyrinthine lesions. In whiplash injuries, where the head is free and never strikes another object, animal experiments have demonstrated marked ecchymosis and œdema of the brain stem and cerebral or cerebellar cortical areas.

Barber (1969) and Jongkees (1969) have shown that vertigo occurring with various head positions is a common physiological finding. It may even be related to stimuli originating in the soft tissues of the neck and skull. Temporal bone fractures and perhaps whiplash injuries can cause acute disturbances in equilibrium; long-lasting chronic disturbances may not come from these sources. Physiological paroxysmal postural vertigo, disturbances in neck structural reflexes and concomitant labyrinthine hydrops or cerebellar disease may be responsible for vertiginous episodes. These are completely unrelated to fractures of the temporal bone and their aftermath.

In my series of 31 cases having a longitudinal fracture of the temporal bone with sufficient injury to produce derangement of the middle ear structures, there was very little evidence of vestibular damage (Hough & Stuart 1968). In only 13% of cases did the patients even remember vertigo following the accident. In these, the vertigo subsided before the end of three weeks. None of the 31 patients experienced long-term post-traumatic vertigo either constant or episodic. We believe this to be an interesting observation, since the patients were questioned directly regarding this symptom and, despite the suggestiveness of a leading question, all patients denied post-injury vertigo after the initial three weeks recovery period.

Types of Temporal Bone Fractures

Temporal bone fractures may be classified in three groups: (1) Longitudinal fractures. (2) Transverse fractures. (3) Mixed fractures.

(1) *Longitudinal fractures* are by far the most common (70–80%) and are usually caused by blows to the temporal or parietal areas. Frontal or occipital injuries do not usually cause this fracture. As the name indicates, the longitudinal fracture runs lengthwise through the petrous pyramid. It usually extends from the squama through the posterior superior external bony canal wall, across the roof of the middle ear and thence along the carotid canal anterior to the labyrinthine capsule to end in the middle cranial fossa near the foramen spinosum. The injury may extend anteriorly across the eustachian tube and into the fossa mandibularis of the temporomandibular joint.

The structures of the middle ear rarely escape injury. The skin of the external ear canal and the 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 strong evidence. The force may cause a very wide break with an amazing amount of twisting and torsion or it may cause only a small crack in the bone without displacement. In either severe or mild fracture, the ossicular chain is frequently separated or fractured producing the second cardinal symptom of longitudinal basilar skull fracture; that is, conductive deafness.

The inner ear usually escapes direct injury in longitudinal fracture because the line of injury is adjacent to, or around, the hard labyrinthine capsular bone rather than through it.

The facial nerve is in an extremely vulnerable position in the temporal bone and is injured in approximately 20% of longitudinal fractures. Cerebrospinal fluid otorrhœa may also occur, through the ear canal or into the nasopharynx.

(2) *Transverse fractures* are thought to be due primarily to blows to the occipital area. Frontal blows are more likely to produce an anterior plate or anterior fossa injury, but a crushing blow may cause a parietal buckling and produce a severe mixed fracture of the temporal bone.

A transverse fracture usually extends from the posterior cranial fossa from the foramen magnum across the petrous pyramid to the middle cranial fossa. The fracture line usually runs between the various foramina, such as the jugular foramen, the hypoglossal, occasionally through the internal acoustic meatus and the labyrinthine capsule and ends in the middle cranial fossa in the region of the foramen lacerum or foramen spinosum.

As it passes through the labyrinthine capsule, the vestibular system and cochlea may be functionally destroyed along with the facial nerve. The fracture may explode the lateral wall of the labyrinthine capsule and produce hæmorrhages 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.

In transverse fractures, the important findings are vertigo with spontaneous nystagmus, sensorineural deafness, facial paralysis and hæmotympanum without rupture of the tympanic membrane.

(3) *Mixed fractures* may occur from crushing head blows and the fracture may involve both the middle ear and the inner ear with a tympanolabyrinthine fracture.

Reconstructive Surgery of the Temporal Bone

Since in this discussion we are concerned only with the surgical aspects of temporal bone fractures, the further discussion will not include transverse fractures, which have caused sensorineural hearing impairment, or destruction of the vestibular labyrinth. The initial emergency surgical management has been briefly described; unfortunately, at present, there is little else to do for these patients except to wait hopefully for spontaneous resolution and adjustment.

The surgical management of facial nerve injuries and the emergency management of the acutely injured patient with aural hæmorrhage has also been previously discussed. In addition, there remains one type of injury to the temporal bone which can now be treated successfully by modern surgical methods. I refer to techniques which are now available to correct most defects in the middle ear caused by temporal bone fractures (Hough & Stuart 1968, Hough 1969).

The middle ear effects of skull trauma were almost unknown before 1956 (Thorburn 1957). Kelemen (1944) had reported ossicular separations found at autopsy following crushing head injuries, but the first discoveries of injuries to the ossicular chain due to skull trauma in the living were made independently by Thorburn in 1956 and shortly thereafter by me (Thorburn 1957, Hough 1958). These injuries produced a pure conductive deafness by interruption of the ossicular chain.

It had previously been considered that any hearing loss following skull trauma was necessarily sensorineural in nature and, therefore, not amenable to corrective surgery. During the following thirteen years, only a relatively few cases have been reported in the scientific literature (Ballantyne 1962, 1966, Hough 1959, Hough & Stuart 1968). This would suggest that these ossicular injuries are not common. In order to dispel this impression, may I say that I have operated on 36 ears where longitudinal fractures had caused ossicular chain separation in the middle ear. This would indicate that the condition is relatively common and, therefore, deserves our most serious investigation and discussion.

Surgical Management of Longitudinal or Mixed Fractures with Middle Ear Derangement

Because of the tremendous number of variables involved, such as area and force of injury, anatomical variations between individuals, and concomitant pathology (such as tympanosclerosis and otosclerosis), operative techniques must be tailored to the needs of each case. To repair the conductive mechanism of the middle ear, therefore, one needs not only an organized procedure in mind but also an understanding of the function of each component part of the mechanism.

Despite the lack of uniformity, there are general classifications of injury effects which can guide the surgeon and there are known surgical methods which may be used successfully. None of these techniques is presented as a final answer. Hopefully, most will be improved in the future or discarded in favour of better methods. I will present several examples from these groups to illustrate in general, first, types of tympanic injuries which may be expected from temporal bone fractures and, secondly, surgical methods I have found helpful in correcting these defects.

(1) *Incudostapedial Joint Separation*

Case 1

L H, man aged 34

This patient sustained a severe skull injury when he was 16. While he was riding in a convertible automobile an accident occurred and the car turned over. He was thrown from the vehicle and was unconscious for a short time. His left ear bled and on regaining his senses he noticed a unilateral hearing impairment.

I first saw this patient eighteen years later, in 1956. He had a unilateral purely conductive hearing loss of 78 dB for pure tones in the three speech frequencies (500, 1,000, 2,000). Speech tests demonstrated the same impairment, but he retained excellent discrimination. A small niche was observed in the posterior superior bony canal at the edge of the tympanic membrane.

This was my first experience with ossicular chain injury due to skull fracture. At that time there were no reports in the scientific literature of non-fatal head injuries with ossicular derangements and I was unaware of Thorburn's experience. I approached the middle ear through a transcanal tympanotomy incision. Had I been aware of Thorburn's experience I would probably have followed his technique – a Type III tympanoplasty through an endaural incision (Thorburn 1957).

On entering the tympanum, an incudostapedial joint separation was observed. To correct this became the immediate problem. The use of artificial prostheses had not been introduced at that time and no other technique had been defined. Because I had noted the viability of bone fragments and loose pieces of ossicles in the tympanic cavity on re-exploring ears which had had previous fenestration of the horizontal semicircular canal, the thought occurred that perhaps a chip of bone could be used to bridge the gap between the joint surfaces of the incus and

stapes. A curette was used to chip off a bone fragment from the edge of the posterior superior bony canal wall. This in turn was placed between the two freshened joint surfaces. Amazingly enough, hearing was totally restored and has remained excellent for the past twelve years. This happy experience introduced me to the possibility of conductive deafness following head trauma. Furthermore, it not only influenced my concepts regarding head injuries and their effects, but also encouraged me in subsequent years to use autogenous bone or ossicles as grafts in tympanoplasty. The use of the patient's own ossicular substance has extended to techniques in stapedial surgery as well.

I have used this method of bone graft repair of the incudostapedial joint many times since and have found it uniformly successful when the two bones were properly aligned for sound transmission.

Case 2

S S, man aged 56

This patient was seen, not long after the previous case, following a skull fracture which resulted from a load of heavy fencing material falling on his head. He sustained multiple body fractures. He had bleeding from both ears, unilateral hearing loss and a partial facial paralysis, and was unconscious for several days. Hearing loss was of a mixed type in the left ear with a predominant conductive component. He had a rather marked sensorineural impairment in the high frequencies in both ears, perhaps from cochlear concussion or presbycusis.

Exploration of the left ear revealed a huge fracture line in the posterior superior bony canal wall representing the outer extension of a longitudinal temporal bone fracture. There was also traumatic dislocation of the incus with separation of incus from stapes. The long process was pushed off the stapedial head posteriorly and the entire incus subluxated inferiorly. It was possible in this case to loosen the incus and to replace it in its original position. This realignment produced complete closure of the air-bone gap.

These two cases represent the most common type of middle ear injury produced by trauma; that is, separation of the incudostapedial joint. This joint is well known to be the part of the ossicular chain most vulnerable to destruction during inflammatory disease. Despite its rather protected position under the canal wall, it is also the most vulnerable portion of the ossicular chain to all forces that injure the middle ear regardless of direction or origin. In my experience, this joint was disrupted in 83% of ears operated on for deafness following temporal bone fractures. The incudostapedial joint thus frequently becomes the key to the interpretation of head injury. It also is usually the key to what must be done in tympanoplastic reconstruction. In many cases what happens to the joint will determine the method one must use to correct conductive defects.

Incudostapedial joint separation can easily be treated either with an autogenous bone graft between the joint surfaces or a replacement of the incus to its original position. Obviously one must ensure that the stapes and malleus are mobile before finishing the operation.

(2) *Massive Incus Displacement*

This type of injury represents much more trauma than an incudostapedial joint separation inasmuch as the articulations of the incus with the malleus, the fossa incudus and the stapedial head are all avulsed and the incus thrown free from all its attachments. This type of drastic injury has occurred in 58% of cases of longitudinal temporal bone fractures seen.

In one case the incus was completely subluxated into the middle ear. In another it was completely turned over 180 degrees in the attic. In still another it was partially thrown through the fracture line projecting into the external ear canal and as the fracture line closed the incus was trapped. In yet another case it was entirely missing.

The incus is the suspended span in the ossicular chain and tremendous forces work during skull injury to fracture and displace it from its moorings.

Case 3

D L, woman aged 42

This patient was severely injured during an automobile accident. She was unconscious for several days, bled from both ears and awakened with bilateral hearing impairment. She had no post-traumatic vertigo and did not have a facial paralysis. Audiometric tests demonstrated a bilateral mixed deafness with the conductive element markedly predominant. Each ear canal showed evidence of a fracture line in the posterosuperior aspect. In the right ear, the body of the incus could actually be seen projecting into the external ear canal and one could clearly see the chorda tympani nerve draped over it. The incus had obviously been dislodged and thrown out of the middle ear just as the fracture line opened. When the body of the incus was partially through the fracture opening, the jaws of the fracture closed suddenly and trapped it in this position.

Exploratory tympanotomy required that the body of the incus be freed by removing canal bone from around it. The incus was then removed and the remainder of the ossicular chain examined. The stapes had survived this devastating experience without injury of any kind. The malleus was also intact and mobile.

A small saucer-shaped depression was then drilled on the lateral surface of the body of the incus and the entire incus placed in the middle ear on a gelatin-foam bed so that its body fitted on the stapedial head. The short process was placed against the medial surface of the handle of the malleus.

This type of incus transposition has been helpful in many cases and usually provides excellent results. I should like to emphasize the importance of preserving the full length of the long process of the incus even though it is not functionally needed when in this position. It provides a second weapon which may be used later if one needs to span the distance from the stapedial footplate to the tympanic membrane and malleolar handle.

(3) *Fracture of the Stapedial Arch*

Twisting torsion of the incus during skull fracture may often cause severe damage to the stapedial crural arch. This occurred in 30% of ears suffering from ossicular chain derangement from skull fractures.

Case 4 B H, man aged 38

This man fell from a high bluff, following which he was unconscious and bleeding was noted from his right ear. A fracture line was seen in the ear canal in the postero-superior portion.

On exploration of the middle ear, the incus was observed to be completely turned over. The articulating face of the lenticular process of the incus was facing laterally instead of medially towards the stapedial head. The short process of the incus was pointing anteriorly towards the malleus rather than posteriorly towards the mastoid antrum and the incudomalleolar articulating face of the incus was facing posteriorly. The stapedial arch had also been fractured at the footplate and was violently displaced posteriorly. This meant that when the skull fractured, the fracture line opened wide enough for the body of the incus to be disarticulated from the malleus and completely turned over 180 degrees in the epitympanic space, so that the short process of the incus was directed forwards towards the malleus. The distance between the horizontal canal and the lateral wall of the epitympanum is simply not enough to allow this to happen in the normal state. It was obviously necessary for a huge opening to occur during the injury time so that the complete incus version could take place. In this particular instance, it was interesting to note how completely the bony wall returned to its original position. Only a very thin fracture line was observed at the time of surgery. An imaginary picture of this occurrence is certainly awesome.

To correct this complete dismemberment of the entire central portion of the ossicular chain, the stapes superstructure was removed and the incus removed from the ear. With the Lempert malleus nippers the short process of the incus was cut off at the body and the long process placed against the centre of the footplate of the stapes. The articulating face of the incus was rested anteriorly against the handle of the malleus.

It has been my continued observation that this long dimension of the incus, amazingly enough, is invariably the exact length to reach from the centre of the footplate of the stapes to the tympanic membrane. Furthermore, the malleolar articulating face of the incus has precisely the right little cup-shaped depression to fit against the handle of the malleus.

I have used this technique of reconstruction in these and similar types of problems in tympanoplasty since 1957 and find it to be permanently successful in most cases.

Discussion

Three basic types of ossicular chain injury and four basic types of surgical repair have been mentioned.

The types of ossicular chain injury most frequently observed are: (1) Incudostapedial joint separation. (2) Massive incus displacement. (3) Fracture of the stapedial arch. Other injuries may occur but basically one, or a combination of the three, is seen. In one case the incus was entirely missing. In another it was extruded into the external ear canal. In another the head of the malleus was in the hypotympanum. In 35% of cases with temporal bone fracture, concomitant disease such as tympanosclerosis, otosclerosis or congenital deformity exists.

In surgical management operative flexibility is certainly required. The surgical team must be prepared to use a variety of prostheses, ossicular homografts or autogenous tissue from outside the ear; nevertheless, the four basic techniques are: (1) Bone grafts. (2) Ossicular repositioning. (3) Incus body transplantation to the stapedial head. (4) Incus span of the long process of the incus from the footplate of the stapes to the handle of the malleus. These should be utilized if at all possible. Other techniques employing prostheses, homograft or autograft tissues from outside the patient's own ear should be given last consideration. It is in an unusual circumstance such as a missing incus or malleus that the above-mentioned alternatives can be used. Fortunately, as a substitute, these other techniques do provide a worthy chance of success.

Results

In the entire experience in this field I have used autogenous bone (ossicles or cortical grafts) to reconstruct the ossicular chain in 75% of cases. Now it is possible to use human material (either autogenous or homogenous) in almost all instances. Despite the fact that this problem has reached its present state in such a short period of

time and despite the fact that in some early cases a technique was used that would now be considered obsolete, the overall results from the standpoint of hearing have been excellent. In my series, including all cases and all methods used, hearing has been restored to within 10 dB of the pre-operative bone conduction in the three speech frequencies (512, 1024, 2048 cycles) in 78% of ears. In 45% hearing acuity has equalled or surpassed the pre-operative bone conduction in these frequencies (closed or overclosed the air bone gap). In 90% the speech reception threshold was improved significantly. In only one case was hearing made worse.

Summary

The aural surgeon should be an integral part of the head-injury team. His opportunities to help the patient begin on the emergency basis of providing the injured person with an airway and in the control of hæmorrhage and shock. A proper otoneurological analysis of the temporal bone and its contents provides the team with information that allows: (1) Prevention of contamination of the cerebrospinal fluid and tympanomastoid spaces. (2) Early detection of the need for facial nerve decompression. (3) A responsible approach to the management of post-injury vertigo.

Successful therapy for many disabilities caused by temporal bone fractures such as severe inner ear and facial nerve injuries is still unavailable. Nevertheless, many effects of temporal bone injuries, such as ossicular derangement, may now be corrected by modern microsurgical techniques. These injuries are not uncommon and the results in hearing restoration are gratifying.

The wide variety of conditions encountered as a result of temporal bone fractures has produced a challenge and stimulus which has resulted in the discovery of new techniques applicable to other otological problems.

REFERENCES

Ballantyne J C
(1962) *J. Laryng.* **76**, 661
(1966) *Proc. roy. Soc. Med.* **59**, 535
Barber H O (1969) *Ann. Otol. (St Louis)* **78**, 239
Guerrier Y, DeJean Y & Galy G
(1965–6) *Cah. Oto-rhino-laryng.* **1**, 11
Guerrier Y, DeJean Y & Serrou B
(1967) *Montpellier chir.* **7**, 483
Hough J V D
(1958) *Laryngoscope (St Louis)* **68**, 1337
(1959) *Laryngoscope (St Louis)* **69**, 644
(1969) *Ann. Otol. (St Louis)* **78**, 210
Hough J V D & Stuart W D
(1968) *Laryngoscope (St Louis)* **78**, 899
Jongkees L B W (1969) *Trans. Amer. laryng. rhin. otol. Soc.* p 851
Kelemen G (1944) *Arch. Otolaryng.* **40**, 333
Kettel K (1950) *Arch. Otolaryng.* **51**, 25
Schuknecht H F (1969) *Ann. Otol. (St Louis)* **78**, 253
Thorburn I B (1957) *J. Laryng.* **71**, 542

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Radiological Investigation of Injuries of the Temporal Bone

Fractures of the temporal bone may occur either as an isolated fracture or more frequently as an extension from the vault or base of the skull. In the former instance the primary clinical presentation is usually an otological problem, but in the latter group the extent of the associated intracranial injury is usually such that the otological aspects are only recognized when complications involving the mastoid bone develop or when a defect of hearing is noted later. Radiology can only demonstrate radiopaque structures; however, rupture of the drum, one of the most frequent results of injury to the temporal bone, needs radiological exclusion of associated bony injury or injury to the ossicular chain.

Radiological demonstration of fractures of the temporal bone may be considerably hampered by the fact that the patient's clinical condition precludes the necessary radiographic positional manœuvres necessary to obtain adequate radiographs of the temporal bone. This is particularly unfortunate, as skull injuries associated with facial paralysis necessitate comprehensive radiological survey of the facial canal as soon as possible after the original injury.

Methods of Examination

The radiological methods of visualizing the temporal bone vary from those produced by the use of very sophisticated and expensive machines, specially designed for this work, e.g. the Princeps Mimar X-Ray table, to a simpler conventional radiographic skull unit. If, however, long cones and accurate coning are used remarkably good detail can be seen by this means. It is important, however, that the radiographic projections be adjusted to visualize the suspected site and type of fracture, and no one radiographic position can be advocated at the expense of others.

We have ourselves used particularly the trans-orbital and the Chaussé III position and these are extremely useful as they can be achieved with minimal disturbance to a seriously injured patient. Lateral oblique projections of the mastoid process are, however, necessary when facial nerve injuries are being investigated, and these views are difficult or even impossible to obtain in a gravely ill patient. It must be remembered that only one in four of fractures involving the temporal bone can be seen by this method according to Grove (1947).

It must be confessed, however, that for detailed investigation of the extent of temporal bone fractures complicated tomographic machines