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Hearing Loss in Skull Fractures

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One hundred three cases of skull fractures in or around the temporal bone were reviewed for hearing loss. Of these, 100 patients had skull series, 66 had computed tomographic head scans, and 44 had polytomographic studies of the temporal bone. Hearing loss in head trauma can be grouped into four categories: conductive hearing loss, peripheral sensorineural hearing loss, central sensorineural hearing loss, and combinations of these hearing losses. The cause of conductive hearing loss and peripheral sensorineural hearing loss was usually identified by the type of temporal bone fracture. However, the cause of the central sensorineural hearing loss was more difficult to correlate with the brain lesions shown in the computed tomographic scans.

In severe head injury, the ear is stated to be the most frequently damaged sensory organ in the human body [1]. With direct lateral or posterior blow to the head, the acoustic nerve is very vulnerable to injury due to acceleration and deceleration movements of the brain within the skull compartments. With the proximal end of the nerve fixed to the brainstem and the distal end to the labyrinth, the acoustic nerve may become stretched and torn [2]. In addition to the cranial nerves and the brainstem, the cerebral and cerebellar hemispheres are also injured in many of these cases. The surface blood vessels of these structures are often injured by traction and friction movements, which may result in intra- or extracerebral hematoma, surface swelling, or cortical necrosis. Although the traditional approach in the study of hearing loss in head injury is centered on temporal bone fractures [4], very little work has been done to correlate the peripheral otologic trauma with the trauma in the auditory pathways located in the brainstem and the cerebral hemispheres.

This paper is a preliminary analysis of the general appearance of the brain and brainstem in cases of fractures in and around the temporal bone in patients who develop hearing loss. The intracranial changes were studied by computed tomographic (CT) scanning; additional examination of the skull and the brain was possible in three of these cases at autopsy. The skull fractures were studied by plain radiographs and tomograms of the skull. Pertinent literature on hearing loss produced by head injury was reviewed.

Materials and Methods

A total of 103 cases of skull fractures involving the temporal bone and/or the contiguous areas of the skull was selected and analyzed for hearing loss. These cases were selected at random by the appearance of the skull fracture or from the results of the known temporal bone fracture proven by polytomography. These cases were collected from patients admitted to the Massachusetts General Hospital (M.G.H.) and the Massachusetts Eye and Ear Infirmary (M.E.E.I.) over a 5 year period. The patients were 3–76 years old (mean, 29 years); 72 were male and 31 were female.

On careful review of the patients' records, it became obvious that the 103 cases were studied inconsistently for head trauma; not all patients with skull fractures were studied with a complete skull series, temporal bone tomography, CT scanning of the head, and audiograms. The availability of these basic studies depended on whether the patient was admitted to the neurosurgical service (M.G.H.) or the otologic service (M.E.E.I.).

The neurosurgical service usually studied these cases with skull films and CT scanning of the head but seldom obtained audiograms or polytomograms of the temporal bone. On the other hand, the otologic service usually studied these cases with polytomography of the temporal bone and audiograms but seldom ordered CT scanning of the head or polytomography of other portions of the skull except for the temporal bone. There were 66 neurosurgical cases, of which 10 were referred to the otologic service for hearing loss. In general, most cases of acute head injuries were handled by the neurosurgical services, whereas head injuries complicated by hearing loss were handled by the otologic service. Most surgical cases were seen immediately after head injury, whereas the otologic cases were seen many months after injury.

Despite the incomplete records of these two groups of cases, it was decided to correlate the type of skull fractures and brain damage with the type of resultant hearing loss. This study was patterned around available radiologic studies in patients with hearing loss after head trauma without the use of electrophysiologic studies (electroencephalograms, and evoked responses of the brainstem). Of the 103 cases chosen for this study, 100 had skull series, 66 had CT, 44 had polytomography, and 40 had audiography. Close clinical and neurologic correlations (table 1) with the radiologic appearance of the skull and brain were not attempted due to the incomplete records in the otologic service.

Skull Films

Of the 103 cases, skull films were unavailable in three, in which temporal bone fractures were proven by polytomography. Of the other 100 cases, 60 had complete skull series (four to six films) and the other cases had incomplete series (two or three films). Many of these studies were of poor quality due to motion artifacts and rotated projections. Although all cases were specially picked for fractures in or around the temporal bone on either side or the base of the skull, the actual presence of a temporal bone fracture could not be assessed in 29 cases without polytomographic study

TABLE 1: Hearing Loss in Head Injuries due to Motor Vehicle Accidents, Blows, Falls, or Blasts

Possible Presentations and Radiographic Findings
Patient presents with one or more of the following symptoms:
Neurologic:
Normal
Confusion
Cerebral signs
Brainstem signs
Somnolence
Coma
Otologic:
Hemotympanum
Otorrhea
Facial paralysis
Labyrinthine concussion
Conductive hearing loss
Sensorineural hearing loss
Radiographic findings may be:
Skull series:
No demonstrable fracture
Nontemporal skull fracture
Temporal and other skull fracture
Temporal fracture
CT series:
Normal
Contusion
Laceration
Intracerebral hematoma
Extracerebral hematoma

TABLE 2: Plain Film Findings in Skull Fracture

	No. Findings
Type of skull fracture (unilateral, bilateral, multiple sites):	
Squamotemporal	30
Parietal	30
Occipital	21
Frontal	16
Basal	15
Temporal bone fracture (unilateral, bilateral):	
Longitudinal	30
Transverse	10
Oblique	4
Mixed	4
Ossicular dislocation	25

Note.—Skull films were available for review in 100 cases.

TABLE 3: CT Findings in Skull Fracture

Findings	No.
General:	
Normal	18
Swelling	30
Contusion	11
Laceration	8
Hematoma (intracerebral):	
Small	8
Large	5
Epidural hematoma	5
Subdural hematoma	6
Subarachnoid hemorrhage	6
Intraventricular hemorrhage	4
Local:	
Free air	10
Small hemorrhage:	
Cortical	10
Subcortical	8
Basal ganglia	4
Brainstem	2
Cisternal blood	8
Skull fracture	15
Temporal bone fracture	10
Mastoid air cell change	5
Soft-tissue edema extracranial	15

Note.—CT scans were available for review in 66 cases.

of the temporal bone. Many of the cases had more than one fracture line in the skull bone. For generalization, the positions of the largest fracture lines in relation to the ear are listed in table 2.

Some fractures involved more than one bone or were depressed. The long linear fracture line of the parietal or occipital bone appeared to enter the mastoid air cells and involve the external auditory canal in several cases to qualify as longitudinal fracture of the temporal bone; whether the fracture line entered the petrous bone was uncertain in some of these cases without the use of polytomography or special coned-down lateral views of the mastoid bone. The presence of a small temporal or basal skull fracture was not obvious in the skull films in 10 cases studied later with polytomography.

Polytomography of the Skull

Polytomographic study of the temporal bone was performed in 44 patients. Although most of these polytomographic sections covered only the temporal bone, as ordered by the otologists, at least 10 cases had larger areas of coverage in addition to the temporal bone. Another 12 cases had special polytomographic studies of the skull base, sinuses, sella, or orbits when these areas appeared to be involved on a clinical basis. These additional studies were usually performed to search for the site of cerebrospinal fluid (CSF) leak or to explain the involvement of other cranial nerves besides the seventh and eighth nerves. In severe head trauma, frontal, basal, and sagittal 1 mm tomographic sections may be required if a fracture is suspected in the temporal bone or to show the type and entire extent of the one or more fractures.

CT Scans of the Head

Sixty-six patients were studied by CT. In the neurosurgical service, all head injuries with skull fractures in the past 7 years have

been studied by CT. Many of the patients reviewed by us had more than one set of CT scans during and after their hospital stay. With the use of different CT scanners over a 9 year period, the quality of the CT scans varied considerably. The more recent scanners produced better images of the brain, the temporal bone, and the skull base. Motion artifacts were more common with the earlier scanners. As all CT scans in our series were obtained to assess the brain after head injury, scan sections were taken at 1 cm intervals in the axial projections without contrast enhancement; only a few patients were studied further with thinner scan sections, coronal projections, or special reconstructed images. Intravenous contrast material was used occasionally when a brain abscess was suspected. Table 3 lists the abnormal findings in the brain in the 66 cases studied by CT scanning. Many of these cases had two or more changes in the brain.

TABLE 4: **Types of Hearing Loss in Head Injury**

Types of Hearing Loss
Central sensorineural:
Brainstem
Cerebral hemisphere
Vascular
Peripheral sensorineural (28*):
Partial tear of nerve VIII
Transection of nerve VIII
Fracture of labyrinth
Fracture of internal auditory canal
Cochlear damage
Conductive (40*):
Ossicular dislocation
Fracture of ossicle
Fracture in attic
Hemotympanum
CSF fistula

* Represents number of cases found in 103 cases reviewed.

Fig. 1.—Longitudinal fracture of petrous bone with ossicular dislocation producing conductive hearing loss. Lateral polytomographic section through ossicles of middle ear shows longitudinal fracture of petrous bone with fracture line (*small arrows*) involving anterior part of external auditory canal and entering attic of middle ear. Malleus and incus show dislocation (*large arrow*).

Audiograms

Only 40 patients had formal audiograms in their records. Although the audiograms were routinely used by the otologists in head trauma, they were seldom used by the neurosurgeons, who usually tested hearing clinically. In many of their cases, testing for hearing was difficult due to the presence of hemotympanum, CSF otorrhea, neurologic deficits, and lack of cooperation. Although it is stated that hearing loss is greatest at the high-frequency range in head trauma [5], this could not be assessed without the use of audiograms. Despite many severe head injuries in this series, most patients did not appear to complain of hearing loss during the convalescent period.

Results and Discussion

The radiologic findings of the skull and brain seen after head trauma are listed in tables 1–3. The type of hearing loss resulting from head trauma is grouped into three main categories: central sensorineural hearing loss, peripheral sensorineural hearing loss, and conductive hearing loss (table 4). The hearing loss may be of mixed etiology. Although conductive hearing loss totaled 40 cases, some of these were mixed with peripheral sensorineural hearing loss as shown by the type of temporal bone fracture.

Although the aim of this study was to correlate the type of temporal bone fracture and the appearance of the brain damage with the type and degree of hearing loss, this was difficult to do because of incomplete radiologic and electrophysiologic studies. Despite these deficiencies it was possible to conclude from the data that the peripheral sensorineural and conductive hearing loss could be correlated with anatomic changes in the temporal bone. On the other hand, it was impossible to correlate the brain lesions as shown by the CT scans with central sensorineural hearing loss.

In cases of CT-demonstrated cerebral contusions and intracranial hematoma, most patients did not appear to complain of gross hearing loss after the surgical removal of the hematoma or after the contusion subsided, unless there was a fracture of the temporal bone. This observation suggested that in order to develop central sensorineural hearing loss, considerable damage to the brainstem and temporal lobes is required. Patients who developed posttraumatic hearing loss usually showed occult temporal bone fracture. The transverse and oblique fractures of the petrous pyramid involving the labyrinth or the internal auditory canal all developed severe or complete sensorineural hearing loss in this series. The hearing

loss is usually permanent. On the other hand, longitudinal fracture of the temporal bone does not always produce a hearing loss. If it does, it may be conductive or sensorineural hearing loss or both.

It is important to study longitudinal fractures of the petrous bone in detail with several views because the fracture line may have multiple prongs to involve the ossicular chain, labyrinth, or the internal auditory canal. The type of hearing loss can be predicted by the extent of the longitudinal fracture. The recognition of ossicular dislocation with or without a longitudinal fracture is important (fig. 1), since considerable improvement in hearing is possible if the dislocation is corrected.

Free air or hemorrhage about the cerebellopontine angle cisterns or around the brainstem in head trauma on CT scans often suggests the presence of temporal bone fracture. Without further study of these cases with thinner CT sections and additional views, a fracture of the temporal bone is seldom demonstrated conclusively. With a third-generation CT scanner, a transverse fracture of the petrous bone is readily seen, especially if the fracture line is long (fig. 2) or gaping and the petrous bone is not excessively pneumatized (fig. 3). Longitudinal fracture of the temporal bone is more difficult to show with CT scans taken in axial or coronal views. Although newer CT scanners [6] can demonstrate considerable anatomic detail of the middle and inner ear compartments, special attempts must be made to study the temporal bone fractures; multiple views, thinner sections of the scans, and special positioning of the head may all be required. At present, the otologists still depend on polytomography to demonstrate the temporal bone fractures; CT scanning for temporal bone fracture is seldom used, although this practice may change in the future.

The three autopsy cases in this series of 103 patients showed that plain skull films are inadequate to show all basal and temporal bone fractures. Even polytomographic studies did not show the true extent and type of fracture of the skull base and temporal bone if incompletely applied. The surface lesions of the cerebral hemispheres and the brainstem were often inadequately demonstrated on CT; surface lesions such as small hemorrhages, lacerations, and necrosis, which were seen at autopsy, did not always correlate with the "almost" normal-appearing scans. Except for a suggestion of slight swelling or faint increased or decreased surface changes, the surface lesions were inconclusively shown in the CT scans.

On reviewing the medical literature, hearing loss is frequently encountered with skull fractures [1, 7, 8]. The hearing loss may be conductive due to blood or CSF in the ear compartments or to disruption of the ossicular chain. The cause of a sensorineural hearing loss is usually explained by concussion of the labyrinth [2, 5] or damage to the acoustic nerve from fracture of the labyrinth or internal auditory canal. The position of trauma to brainstem and
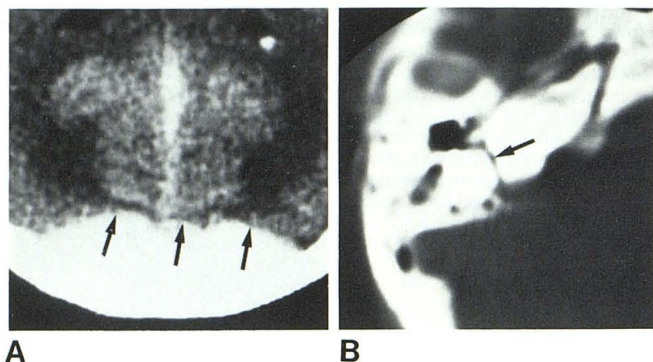
Fig. 2.—Transverse fracture of petrous bone with epidural hematoma and occipital bone fracture producing peripheral sensorineural hearing loss. **A,** Acute epidural hematoma (*arrows*) is shown relative to occipital fracture. **B,** Through posterior fossa. Transverse fracture line (*arrow*) enters internal auditory meatus and involves vestibule of inner ear.

Fig. 3.—Transverse fracture of right petrous bone as part of long basal fracture line in patient in coma. **A,** Long basal skull fracture line (*arrows*) extends through floor of posterior, middle, and anterior cranial fossae and enters facial bone. Transverse fracture of petrous bone (*arrowheads*) involves internal auditory canal. **B,** Axial section through mid-supratentorial region. Compressed lateral ventricles with blood in both occipital horns (*long white arrows*) and deep intracerebral hematomas (*short white arrows*) in frontal lobe. Small extracerebral hematoma and contusion (*black arrow*) in anterior part of right frontal lobe next to fracture.

cerebral hemisphere as a cause of sensorineural hearing loss is seldom mentioned in the early otologic literature. Recently, with the use of auditory-evoked response studies, new interest has developed in the analysis of the brain for abnormalities in head trauma. Hall et al. [9] showed that head trauma that causes electrophysiologic changes in the brainstem can result in central sensorineural hearing loss. In 80% of their cases, there was central nervous system dysfunction in the brainstem in the early phase of head injury. The abnormalities in the brainstem ranged from prolonged latency and reduced amplitude of the wave to total absence of brainstem auditory activity. Hearing loss in head injury can be temporary or permanent or partial or complete, and hearing may or may not recover with time.

Past researchers have correlated the types of skull and temporal bone fractures to the type of brain injury [9–13]. Experimental head trauma was induced in animals by Denny-Brown and Russell [14], Schuknecht et al. [5], and Makishima and Snow [2] to show the type of traumatic changes in the cerebral hemispheres and the brainstem in one end and local changes in the acoustic nerve and the cochlea in the other end. Other workers [15, 16] have experimentally studied the various traction, friction, and rotational movements of the brain that result in head trauma. The direct and indirect forces that produce brain lesions are related to the abutment of the brain to the inner surface of the skull vault with its sharp tentorial and osseous edges. At the histologic level in these trauma cases, Strich [17] demonstrated the presence of extensive degeneration of the white matter resulting from shearing of the nerve fibers in the cerebral hemispheres and the brainstem in head trauma.

Howe and Miller [18] recently described a patient who became deaf after a head injury. Postmortem examination revealed bilateral lesions of the lateral lemnisci and inferior colliculi. They stated that cortical auditory impairment is not commonly associated with cerebral trauma, even when there is contusion of both temporal lobes. This is probably due to the association of marked cerebral edema, unconsciousness, and other more profound deficits that mask auditory impairment.

Dix and Hood [19] presented nine cases of proven lesions of the brainstem (tumors and vascular and degenerative changes) that resulted in bilateral hearing loss. These cases showed that multiple lesions of the brainstem are needed in head trauma with or without involvement of the cortical representation in the temporal lobes to produce various degrees of hearing loss.

In the future more correlational studies are needed in head trauma cases where one can compare the pathohistologic changes of the auditory tract in 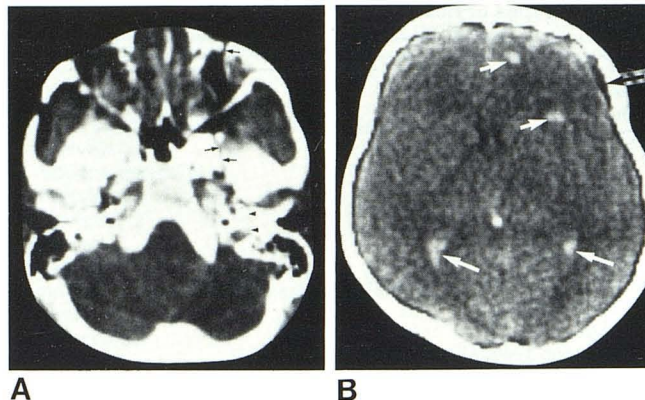the brainstem and the cortical representation in the temporal lobes with the degree and type of hearing loss. Further work is also needed at the radiologic and electrophysiologic level to demonstrate the contribution of brainstem and cerebral cortex damages to the production of hearing loss in head trauma whether or not there is damage to the temporal bone.

REFERENCES

1. Hough JVD. Fractures of the temporal bone and associated middle and inner ear trauma. *Proc R Soc Med* **1970**;63:245–262
2. Makishima K, Snow JB. Effects of head blow on the development of hearing loss. *Laryngoscope* **1976**;86:971–978
3. Gurdjian ES, Webster JE. *Head injuries.* Boston: Little, Brown, **1958**
4. Toglia JJ, Katinsky S. Neuro-otological aspect of closed head injury. In: Vinken PJ, Bryn GW, eds. *Handbook of clinical neurology,* vol 24. Amsterdam: North Holland, **1976**:119–140
5. Schuknecht, HF, Neff, WD, Perlman HB. Experimental study of auditory damage following blows to head. *Ann Otol Rhinol Laryngol* **1951**;59:331–357
6. Virapongse, C, Rothman SLG, Kier EL, Sarwar M. Computed tomographic anatomy of the temporal bone. *AJNR* **1982**; 3:379–389, *AJR* **1982**;139:739–749
7. Browning GG, Swan IRC, Gatehouse S. Hearing loss in minor head injury. *Arch Otolaryngol* **1982**;108:474–477
8. Nelson JR. Neuro-otologic aspect of head injury. In: Thompson RA, Green JR, eds. *Advances in neurology,* vol 22. New York: Raven, **1979**:107–128
9. Hall JW, Huang-Fu M, Gennarelli TA. Auditory function in acute severe head injury. *Laryngoscope* **1982**;92:883–890
10. LeCount ER, Apfelbach CW. Pathologic anatomy of traumatic fractures of cranial bone. *JAMA* **1920**;74:501–512
11. Pearson BW, Barber HO. Head injury: some otoneurologic sequelae. *Arch Otolaryngol* **1973**;97:81–84
12. Grove WE. Skull fracture involving ear, 211 cases. *Laryngoscope* **1939**;49:678–869
13. Podoshin L, Fradie M. Hearing loss after head injury. *Arch Otolaryngol* **1975**;101:15–18
14. Denny-Brown D, Russell R. Experimental cerebral concussion.

Brain **1941**;64:93–163

15. Pudenz RH, Shelden CH. The Lucite calvarium, a method for direct observation of the brain. *J Neurosurg* **1946**;3:487–505

16. Proctor LR, Gurdjian ES, Webster JE. The ear in head trauma. *Laryngoscope* **1956**;66:16–61

17. Strich SJ. Shearing nerve fibers as a cause of brain damage due to head injury-pathological studies of 20 cases. *Lancet* **1961**;2:443–448

18. Howe JR, Miller CA. Midbrain deafness following head injury. *Neurology* (NY) **1975**;25:286–289

19. Dix MR, Hood JD. Symmetrical hearing loss in brain stem lesions. *Acta Otolaryngol* (Stockh) **1973**;75:165–177