Ear Disturbances Following Head Injury

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It is generally agreed that the ear is frequently involved in head injury and cochlear and vestibular disturbances are present after head injury, particularly in the temporal bone. The object of this paper is to review the ear disturbances following head injury and to present sixty two cases complaining vertigo as a chief symptom after head injury without fracture of the skull.

As our starting point we shall take the fracture of the temporal bone.

I. FRACTURE OF THE TEMPORAL BONE.

According to various clinical studies, it is accepted that from 60 to 80 per cent of the skull fractures are fractures of the base of skull. The temporal bone is involved in over one-third of the cases of basal skull fracture. The labyrinth is involved in 25 per cent.

The temporal bone fractures are clinically divided into main two types (1. longitudinal, and 2. transverse).

In longitudinal fractures the fracture linea courses through the roof of the middle ear and then along the anterior edge of the pyramid, so that the tympanic membrane and external auditory canal are often damaged, and on the other hand the labyrinthine capsule usually remain undamaged. Therefore the vestibular end-organ is rarely damaged, though there is frequent opportunity of injury to the membraneous cochlea (hemorrhages into spiral ligment and scala tympani) especially near the round window.

In transverse fractures the fracture linea courses at right angles to the pyramid, and therefore the following distrubances may be present in the middle and inner ears. 1. The middle ear damage is confined to the medial wall. 2. Blue ear drum may occur and exteral bleeding from the ear is not common. 3. Cerebrospinal ear discharge is more common in the transverse than in longitudinal fracture. The fluid may drain down the Eustachian tube. 4. The cochlea is more vulnerable than the vestibule and the eightth nerve in the internal auditory meatus may be torn. 5. There may occur a tearing of the membranous labyrinth. 6. Bleeding occurs into the endolymph

and perilymph.

In clinical experiences it is kept in mind that pure transverse or longitudinal fractures are rather uncommon than comminution and fragmentation in either type.

As far as prognosis of the temporal bone fractures is concerned the mortality rate varies between five and ten per cent. The first 24 to 48 hours are most critical and persistent indicates increasing danger.

In longitudinal fractures of the skull, about 8 per cent formerly died from meningitis extending from the ear canal. In transverse fractures the death rate was 15 per cent especially if there was a crack through the labyrinthine wall into the middle ear. Since the advent of antibiotics, the prognosis of skull fractures with a complicating meningitis has improved nowadays.

The mojority of otologists agree in thinking that although there is permanent vestibular and auditory function distrubance in transverse fracture, the prognosis in the longitudinal fracture is somewhat better.

II. EAR DISTURBANCES FOLLOWING HEAD INJURY WITHOUT EVIDENCE OF SKULL FRACTURE.

We propose now to consider in detail ear disturbances following head injury without evidence of skull fracture. It is generally accepted that many cases with head injury, without clinical or radioloical evidence of skull fracture, develop cochlear and vestibular disturbances. It may be due to bleeding in the labyrinth (Stenger, 1909[1] Brunner[2], 1925[4]; Schonbauer & Brunner, 1927;) or to degenerative changes in Corti's organ (Wittmaack, 1932; Theodore, 1910; Nassulphis, 1946;) or to tearing of the eighth nerve in the internal auditory canal (Uffenorde, 1924; Klingerberg, 1929;).

Stenger engaged in studies of ear disturbance after mild head injury in animal. Following light blows with a hammer on the skull of white rats, Stenger found bleeding in the cochlea with normal in the vestibular area. He also confirmed that with heavier blows bleeding were more severe in the round window and extended to the helicotrema, and some bleeding in the ampullae. Brunner performed on the similar experiments on guinea pigs and he confirmed Stenger's results. Ogata, our colleague, attempted experimental studies of ear disturbances following head injury in 20 rabbits given a light blow on the temporal area. In sixteen of 20 animals, he confirmed microscopically bleedings not only in the cocholea but in the vestibular labyrinth.

Proctor et al[3] examined 14 cases of head injury with unconsciousness

without skull fracture and obtained the following results. Four had normal hearing; eight had a high tone loss and two presented a low tone loss. They stated that it suggests a rather high incidence of inner ear damage. Also they examined 32 cases of head injury without unconsciousness or skull fracture, and confirmed that normal audiogram were obtained in seven cases, a mild degree of bilateral nerve deafness was present in 12 cases, in five cases severe degree of bilateral nerve deafness was present in two of which there were a previous history of acoustic trauma.

As to disturbances of vestibular function in the cases following head injury without skull fracture, the majority writers accord that vertigo is most common sequelae of head injury and it may arise from disturbances in the vestibular sense organs, the primary vestibular nuclei and probably the central vestibular pathway.

As a sequelae of head injury, two types of vestibular disturbances may occur: One is the peripheral type; the other is the central type in orgin.

Proctor, et al,[3] emphasized that the peripheral type usually reveals a true turning vertigo such as staggering and room movement, and spontaneous nystagmus beating to the opposite side and disturbance in balance may be noted. Accompaning involvement of the autonomic system also may be present with nausea or vomiting. These effects are mediated through connections of the vestibular nuclei with the vagus. On examination abnormalities of caloric responses are seen in the majority of cases.

McNally indicated that vertigo arising from disturbances in the central nervous system may be difficult to differentiate from an attack of peripheral labyrinthine vertigo. If tinnitus and hearing loss combined with vertigo and autonomic disturbances are present, the likelihood of a peripheral origin is more certain.

Grove[4][5] believed that off-balance sensation such as tilting of the bed, a rising of the floor, a falling of the ceiling of a room, a feeling of uncertainty or of being drunk, is to be of possible vestibular origin if was accompanied by an abnormal caloric response.

In the central type of vestibular disturbance, the term "blacking out" is sometimes used by patients to express the attack of vertigo which may be initiated by sudden movements, such as stooping, straining, head turning and upward movement of the eyes. Rowbotham stated that these momentary blacking-outs are due to instability of the cerebral circulation consequent upon injury to its vasomotor apparatus, rather than to labyrinthine dysfunction. He suggested that a momentary ischemia of the brain, due to circulatory lability, is the essential cause of the vertigo in cases with central type of vestibular disturbances.

Van Egmond, Groen, Hulk and Jongkees described, as proof of a central origin in vestibular disturbances, the following symptoms: 1. Spontaneous nystagmus towards a disturbed labyrinth; 2. Rotatory nystagmus lasting long period (unsually for a few months); 3: Vertical nystagmus; 4. Normal turning reactions without caloric excitability; 5 Monophasic reactions without quick phase in eye movements. 6. Somnolence in deviations of the positions of the head.

We come next to describe and analyse our own cases with the ear disturbances following head injury without skull fracture. Our present analysis of clinical features based upon the study, made possible through the courtesy of our colleagues at Dept. of Otolaryngology, Yamaguchi University Hospital, of sixty two cases. These we have examined in the course of last three years. Age and sex distribution are shown in Table 1. It will seen that the disorder chiefly affects the age group 20 to 60 and in the great majority, 50 of the 62 cases, patients are male.

Table 1.

Age Distribution

AGE (Years)	10-19	20-29	30-39	40-49	50-59	60-69	70-79	Total
Number	6	12	18	10	8	7	1	62

Sex Distribution

Male	50		
Female	12	Total	62

As to interval from accidents to our examinations, it is full of variety, such as 13 of these 62 cases are within one month, 17 are 2 to 3 months, 13 are 4 to 6 months and 19 are 1 to 5 years. Our all cases are divided into the following three groups by causes of head injury: 1. Car accident in 38 (61.3%); 2. Falling in 17 (27.4%); and 3. Blow in 7 (11.3%). We find over a half of cases are due to car accident.

All our 62 cases complained of vertigo such as feeling of off-balance in 40 (64.5%), gyratoric movements of body in 8 (12.9), sensation of blackout in 5 (8.0%), tilting of the bed in 4 (5.4%) and raising of the floor in 3 (4.8%). From these figures we find the majority of cases suffer from sensation of off-balance (forty of 62) which is aggravated by head movements of all kinds and walking.

Localization of injury in our 62 cases is given in Table 2. It will be seen from Table 2 that the majority cases with vertigo following head injury (49

Table 2. Localization of Injury.

Temporal area	27	43.5%
Occipital area	22	35.5
Parietal area	4	6.5
Frontal area	2	3.2
Unknown	7	11.3
Total	62	100

of 62 cases) were damaged in the temporal or occipital areas, and on the other hand in cases blown in the parietal or frontal areas only a few cases complained of vertigo. From these experiences it seems, beyond any doubt, to confirm that the vestibular apparatus is more disturbed by blowing in the temporal or occipital areas than in the parietal or frontal areas.

Araki (1954) is the first to describe clinically the classification of head injury in our country. The head injury is divided into four major types by him. According to his description type I is a form of simple in charactor such as no unconsciousness with feeling top heavy, type II comes under commotio cerebri with unconsciousness lasting a few minutes, type III is suitable to concusion cerebri and type IV means hemorrage in the brain.

In our 62 cases with head injury, classification by Araki is given in Table 3. It will be confirmed in Table 3 that the majority cases are included in type II or III and even in type I (simple type without unconsciousness) there are 12 cases (19.4%) blowed in the temporal area.

When we come to investigate spontaneous, positional and caloric nystagmus, the following abnormalities are present (See Table 4).

Table 3. Types of Head Injury. (By T. Araki)

Type Ⅰ	12	19.4%
Ⅱ	26	41.9
Ⅲ	16	25.8
Ⅳ	6	9.7
Unkown	2	3.2
Total	62	100

1). Spontaneous nystagmus.

In twenty seven of the 62 cases (43.5%) we observe spontaneous nystagmus which reveal in the majority of cases horizontal or rotatory in character with exception of vertical nystagmus towards upwards in 5 cases. Spontaneous nystagmus towards the normal side are seen in twenty of 27 cases and remaining seven cases show towards the disturbed side in direction.

It is generally accepted that horizontal or rotatory nystagmus is due to disturbances in the peripheral vestibular apparatus confined to the inner ear and vertical one is elicited from central vestibular lesions, particularly in the brain stem. It is of reasonable to understand from the above mentioned data that in our 62 cases at least five cases with disturbances of the central vestibular apparatus are included. Judging from direction of the nystagmus the majority of cases present hypo-function of the vestibular apparatus.

2). Positional nystagmus.

In nineteen of the 62 cases (30.6%) we can see positional nystagmus showing horizontal or rotatory in character without exception. Positional nystagmus towards the normal side are observed in sixteen of 19 cases and remaining three cases reveal towards the disturbed side in direction. Positional nystagmus of all 19 cases are considered to be "direction fixed" in type, that is to say its direction does not change with changes in the position of the head. In our experiences the direction-changing nystagmus frequently elicited on postural tests suggests disturbances in the vicinity of the vestibular nuclei and on the other hand the positional fixed one may attribute to the peripheral vestibular lesions.

3). Abnormalities of caloric responses.

Caloric tests were carried out in accordance with the technique described by Fitzgerald and Hallpike. A description of caloric abnormalities in all cases is given in Table 4. In thirty three (53.2%) of the 62 cases abnormalities of caloric responses are observed and in 28 they are unilateral; and in 5 they are bilateral. In the majority cases (in 30 of these) canal paresis (CP) are present, remaining three cases show directional preponderance (CP). It is of important to keep in mind that all of our cases following head injury complain of vertigo at period of our examinations, nevertheless in twenty nine of 62 cases no abnormalities of caloric responses are present. Normal caloric responses in 29 cases may be explained by the ability of the vestibular nuclei to compensate for the unbalanced impulses which one deranged labyrinth may initiate.

Table 4. Spontaneous, Positional and Caloric Nystagmus.

Localization of injury	Spontaneous N.	Positional N.	Abnormal of caloric N.
Temporal area 27	14	12	15 (cp 13, dp 2)
Occipital area 22	9	6	12 (cp 11, dp 1)
Parietal area 4	0	1	1 (cp 1)
Frontal area 2	0	0	1 (cp 1)
Unkown 7	4	0	4 (cp 4)
Total 62	27(43.5%)	19(30.6%)	33 (53.2%)

4). Abnormalities of equilibrium.

Results from equilibrium tests in our 62 cases are given in Table 5. As shown in Table 5, on Mann's test abnormalities of equilibrium are present in forty two (67.7%) of 62 cases, on goniometer test in thirty two (53.2%) and on gait test in twenty eight (45.2%). In the majority cases body sway or falling towards the disturbed side is present: thirty five of 42 cases on Mann's test; twenty nine of 33 cases on goniometer one; and twenty three of 28 cases on gait. These directions of body sway or falling and nystagmus are reversible in all cases.

It is true in all probability that abnormalities of equilibrium which arise from disturbances in the central nervous system may be difficult to differentiate from the peripheral labyrinthine disequilibrium, because equilibrium depends upon a number of impulses, such as: labyrinthine

Table 5. Abnormalities of Equilibrium.

Localization of injury	Mann's test	Goniometer test	Gait test
Temporal area 27	20	15	15
Occipital area 22	13	10	9
Parietal area 4	2	3	2
Frontal area 2	2	1	2
Unkown 7	5	4	3
Total 62	42(67.7%)	33(53.2%)	28(45.2%)

discharge from the maculae and cristae; proprioceptive impulses from muscles, tendons and limbs; exteroceptive impulses; and visual impulses from the retina. The correlation of the impulses takes place in the high centers of the central nervous systems.

5). Abnormalities of optokinetic nystagmus (O K N) and eye tracking test (E T T).

We come, finally, to review results from optokinetic responses and eye tracking test in our cases. Test of optokinetic nystagmus were carried out in accordance with the technique described by Suzuki (1962). Results obtained by these tests are given in Table 6.

Abnormalities of optokinetic responses are seen in seventeen (27.4%) of the 62 cases and of eye tracking responses are sixteen (25.8%).

Table 6. Abnormalities of Optokinetic Nystagmus (O K N) and Results of Eye Tracking Test (E T T).

Localization of injury	OKN	ETT
Temporal area 27	13	10
Occipital area 22	3	3
Parietal area 4	0	1
Frontal area 2	0	0
Unkown 7	1	2
Total 62	17(27.4%)	16(25.8%)

Examinations of optokinetic responses and eye tracking are carried out as tests for disturbed vision and visual acuity, and also are useful to differentiate ocular nystagmus from other spontaneous one. Judging from the results above mentioned, it is suggested that seventeen of our cases show disturbed vision which may be due to the central oculomoter lesions.

Optokinetic nystagmus is of great interest to ophthalmologists, neurologists and otologists since last century. For clinical purpose it was first used by Wright. In otological clinic the use of optokinetic nystagmus was introduced by Barany who studied the influence of induced optic nystagmus upon a spontaneous nystagmus using a turning optical wheel. Sommer studied the interference of spontaneous nystagmus with induced ocular and induced labyrinthine nystagmus. He found that any spontaneous ocular nystagmus can easily be influenced by an induced ocular nystagmus,

but not so easily by a labyrinthine (caloric) nystagmus. On the other hand, a spontaneous nystagmus of central labyrinthine origin can easily be influenced by a caloric nystagmus, but not so easily by an induced optic nystagmus.

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