Sudden deafness

A histopathological study

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SUDDEN deafness is a unique sensorineural deafness in that it occurs all of a sudden like 'a thunderbolt from the blue', and that some patients show complete recovery with or without treatment. It is a challenging problem to elucidate etiology and pathogenesis of sudden deafness. Many diseases are known to cause sensorineural deafness of sudden onset. It is still unknown whether sudden deafness is a clinical entity or a syndrome, mainly because of paucity of temporal bone pathology reports. The purpose of this paper is to document the histopathology of the temporal bones from a patient who suffered from bilateral sudden deafness.

Report of a case

A 77-year-old woman noticed a profound bilateral hearing loss at the age of 46 on a day in World War II when three of her eight sons were drafted for military service simultaneously. She was extremely upset and noticed tinnitus and fullness of the ears. That night, she developed profound bilateral hearing loss accompanied by a vertiginous attack.

An audiogram was taken in 1969, 28 years after the onset of the disease. Physical examination showed that the external and the middle ears were normal bilaterally. In October 12, 1973, she died of suffocation. An autopsy revealed that sputum had occluded the larynx.

The temporal bones were removed 9·5 hours postmortem. It was 31 years from the onset of sudden deafness to autopsy. The temporal bones were immersed in 10% formalin solutions decalcified, dehydrated, and eventually brought to celloidine embedding. Serial sections were made by cutting the temporal bone horizontally in 20μ thickness. Cochlear reconstructions were made according to the method of Schuknecht.

Findings

The left temporal bone

The external meatus and the middle ear were normal. The otic capsule appeared normal.

The internal ear showed pathology (Fig. 1). The organ of Corti was atrophic throughout the cochlea. The loss of cochlear neurons was almost complete from the basal end up to the 17 mm. area (Fig. 2). Then, it was

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gradually less toward the apex. The intraganglionic spiral bundle was missing in Rosenthal's canal.

The stria vascularis was completely atrophic and flat except in areas near the apical and basal ends where 25–75% shrinkage of its normal thickness was found. Concretions were occasionally found in it. In the area where the capillary still remained, it protruded into the endolymphatic space (Fig. 3).



FIG. 1.
The left cochlea. There is severe atrophy of the organ Corti accompanied by severe loss of cochlear neurons in the middle and basal turns.

The tectorial membrane showed a variety of pathology throughout the cochlea. Two types of pathology were recognized: (1) flattening of the middle zone, (2) thickening of the limbal zone, (3) droplet formations in the middle and marginal zones. The tectorial membrane was shrunken in the areas from the basal end to the 10 mm. area, and from the 15 mm. area to the apical end. It adhered tightly to Reissner's membrane (Fig. 4). It was devoid of colour by H-E staining. In the 10 to 15 mm. region of the cochlea, the tectorial membrane showed a markedly thick limbal zone (up to 18μ), and the middle and marginal zones showed derangement and spherical-amorphous droplets, the maximum diameter of which mea-

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sured 20μ. The droplet was purple in the H-E stained specimens (Figs. 5 and 6).

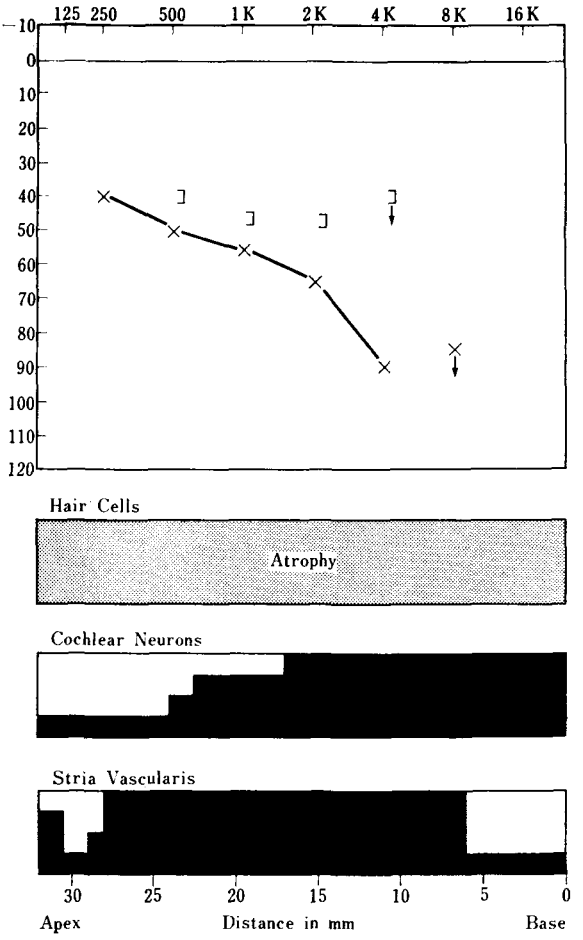


FIG. 2.

Audiogram and histogram showing pathology of hair cells, cochlear neurons, and stria vascularis in the left cochlea.

In the vestibular system the contour of the utricle and saccule appeared normal. The maculae and the cristae of the semicircular canals showed moderate loss of sensory epithelium. The anterior and posterior cristae contained spherical and homogeneous material in the neuroepithelium (*Fig. 7*).

The internal auditory artery had a thick vascular wall. The vascular change was not observed in the blood vessel within the modiolus. The

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strial capillary was missing, and the capillary in the spiral ligament seemed to have disappeared.

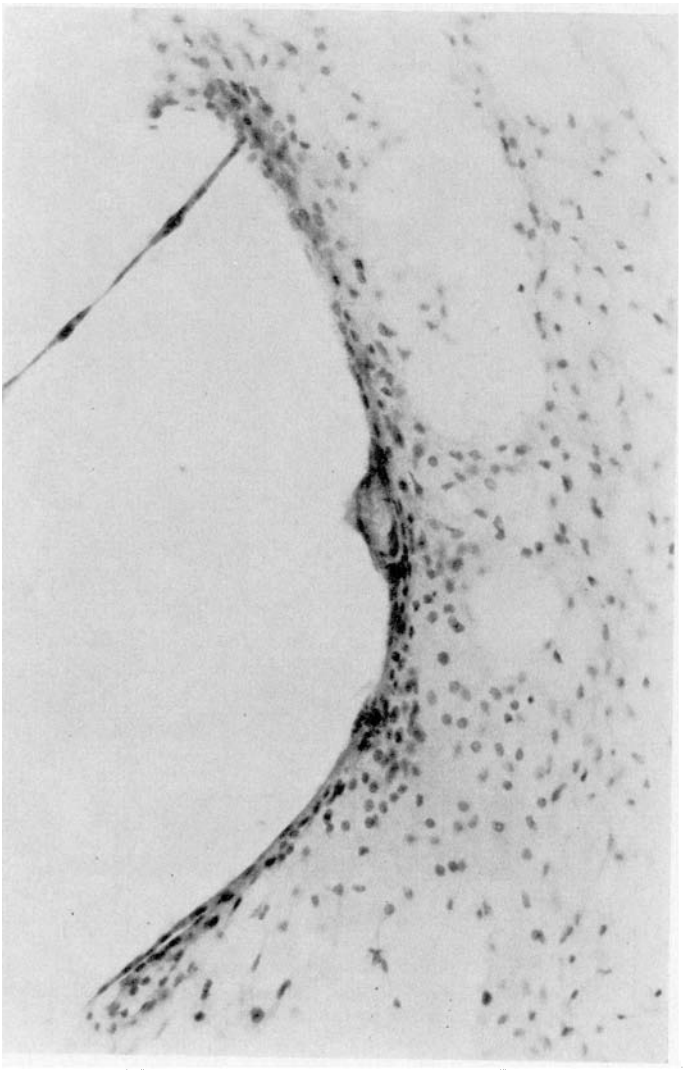


FIG. 3.
Atrophy of the stria vascularis. Stria capillary is also missing. Remnant of degenerating capillary makes a protrusion. 14 mm. area.

The cochlear and vestibular aqueducts appeared normal.

There was a macrophage in the scala vestibuli, close to Reissner's membrane (Fig. 8).

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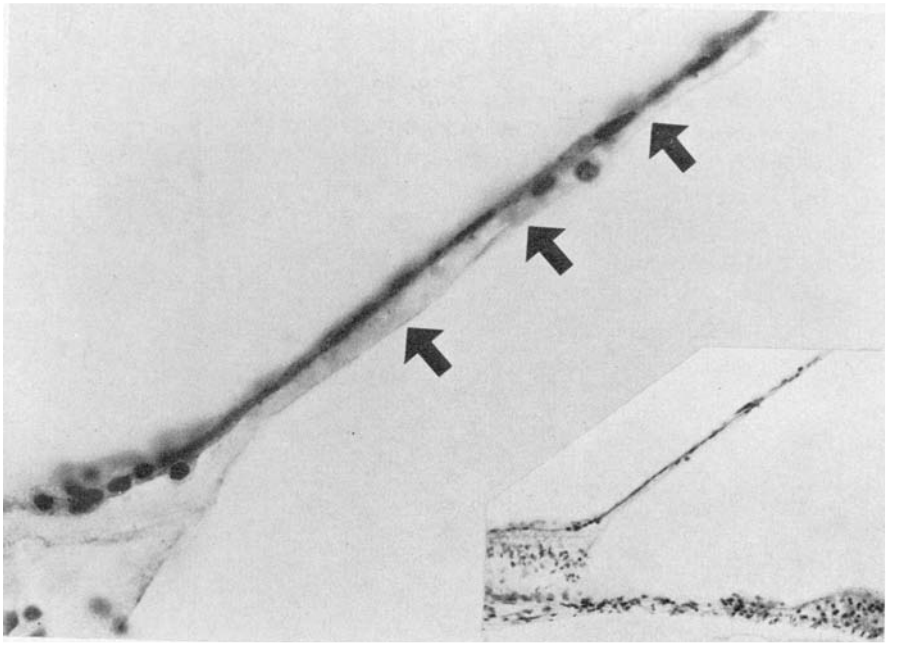


FIG. 4.

The tectorial membrane (arrows) is atrophic and flat. It adheres to Reissner's membrane. Inset shows a low power view.

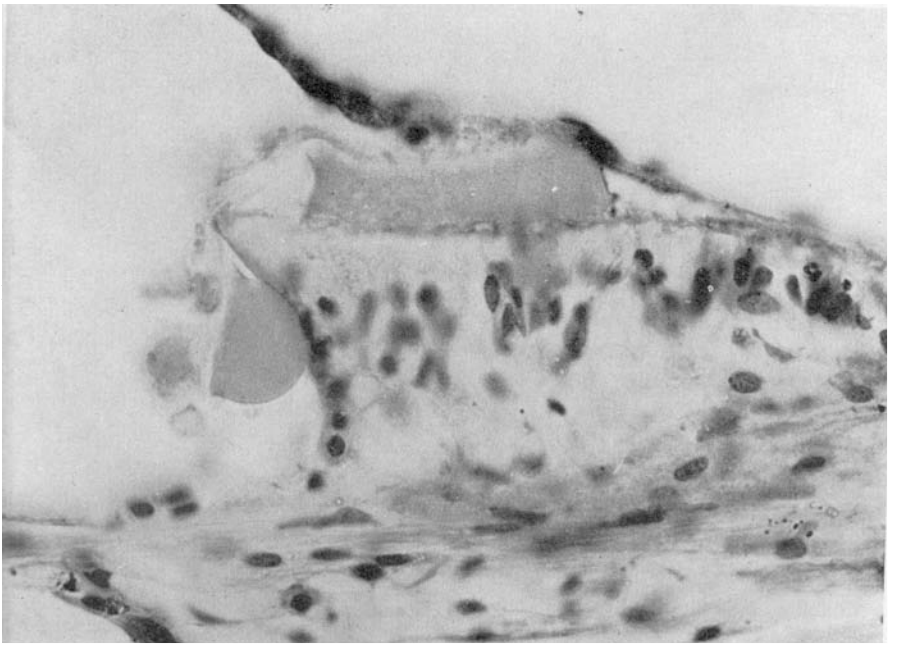


FIG. 5.

Degenerated tectorial membrane. The thin limbal portion is lifted up by a thick mass on the limbus. A large droplet of 20μ in diameter is seen attached to the atrophic and disintegrated middle zone.

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The right temporal bone

The external and middle ears were normal.

The internal ear showed several pathological findings (Fig. 9). The organ of Corti was missing in the lower basal turn, and atrophic in the upper basal turn. In the middle turn, the internal and external hair cells were partly missing and partly showed varying degrees of degeneration (Fig. 10). The organ of Corti in the apical turn indicated atrophy.

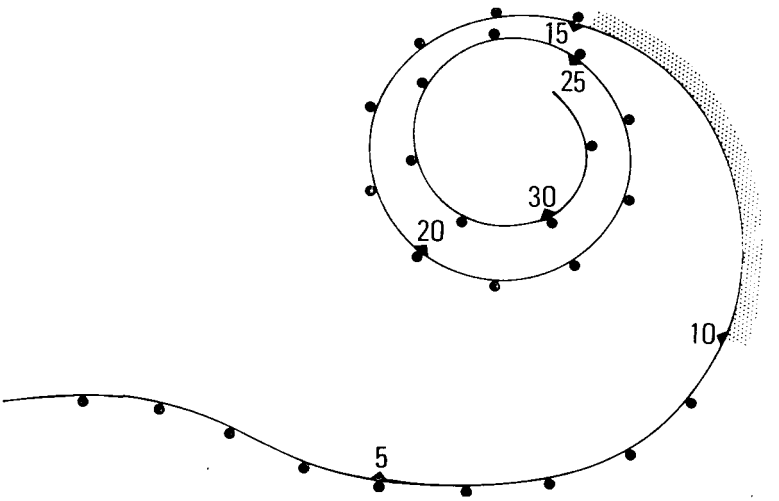


FIG. 6.

Cochleogram of the left ear showing pathology of the tectorial membrane. The membrane is atrophic (black dots) throughout the whole cochlea. There is severe disintegration in the 10–15 mm. area (shaded area).

The cochlear neurons were almost completely missing in the upper middle and basal turns. In the apical and lower middle turns 75 and 25% of the neurons remained respectively (Fig. 11).

The stria vascularis was atrophic and flat in all areas except near the basal end and apical end, where it showed varying degrees of partial atrophy. There was a large vacuole in the spiral ligament near the attachment of Reissner's membrane.

The changes in the tectorial membrane were less pronounced in this cochlea than that found in the other side. In two thirds of the whole cochlear turn, the change was not remarkable. However, in a part of the basal turn, certain peculiarities were observed. There was a spherical and amorphous structure at the marginal zone of the tectorial membrane in the area between 10–15 mm. (Fig. 12). This was also observed on the inferior surface of the middle zone. Furthermore, there were many small 'dots' on the surface of the tectorial membrane (Figs. 13 and 14). These

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were identical in their nature to the spherical structure which was found in the marginal zone.

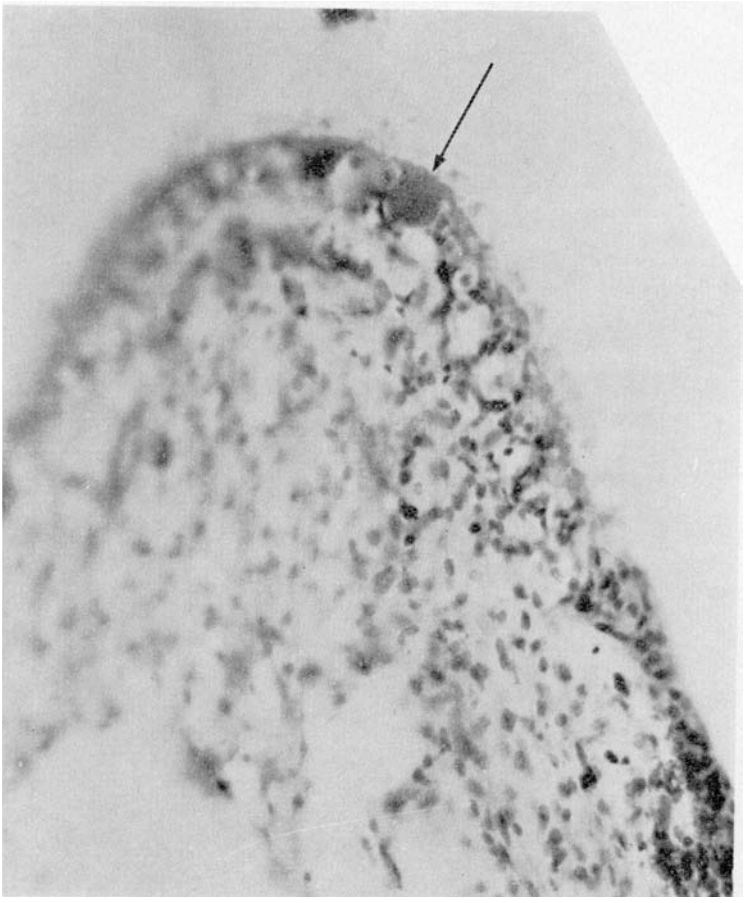


FIG. 7.
The posterior crista contains a spherical and amorphous mass in the neuroepithelium (arrow).

The cristae of the three canals did not show any remarkable change except a moderate loss of sensory epithelium. There was a cyst or gland-like structure near the transitional epithelium in the anterior and posterior cristae (Fig. 15). The macula utriculi and its nerve fibres showed moderate degeneration.

The saccular wall had a perforation of about 750μ in diameter (Fig. 16) It was not collapsed. The perforation was located in the posterior part of the wall. At the edge of the perforation, the epithelium was round, indicat-

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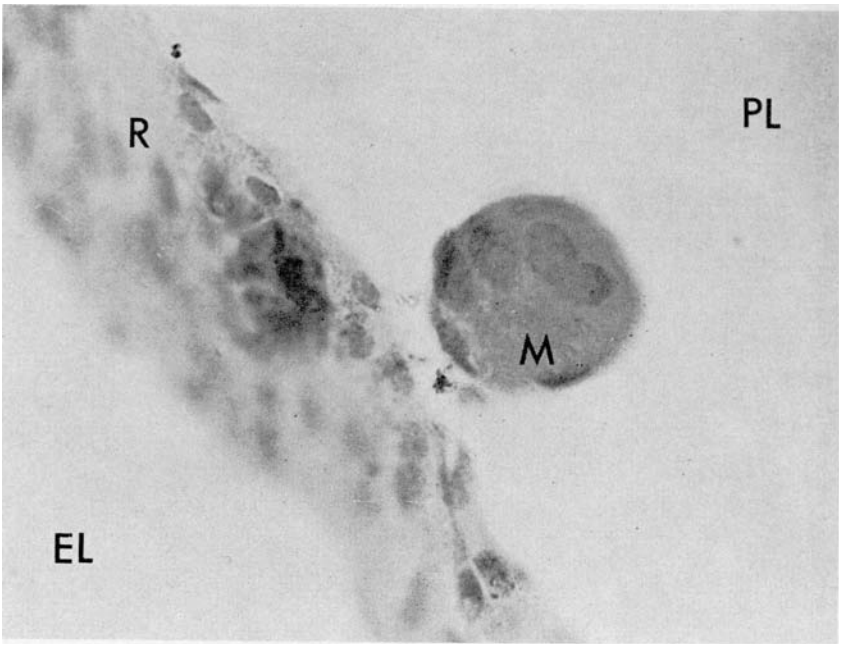


FIG. 8.
A macrophage (M) in the scala vestibuli.
R: Reissner's membrane, PL: perilymph, EL: endolymph.

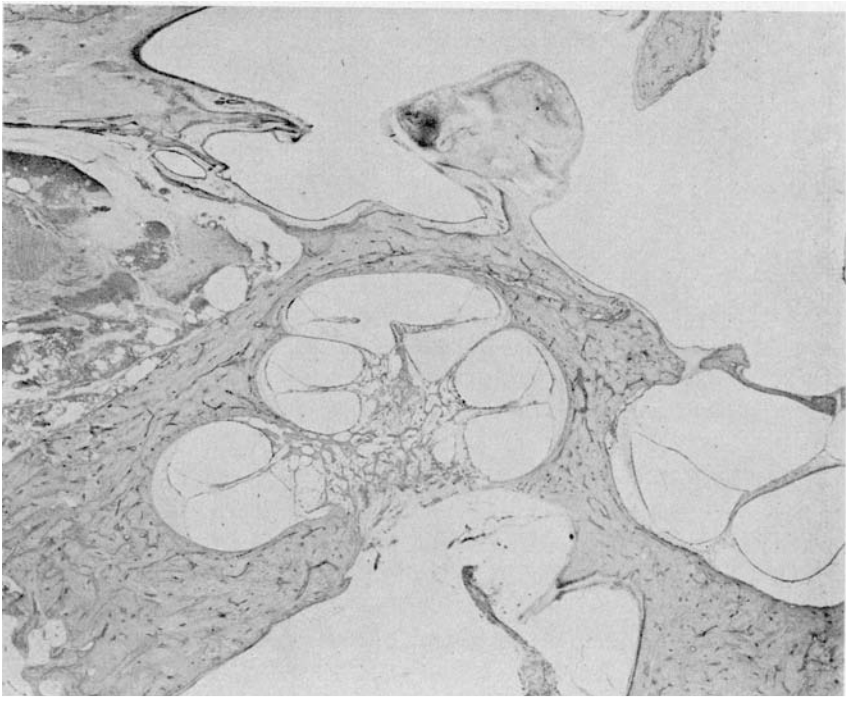


FIG. 9.
The right cochlea. There is atrophy of the organ of Corti and the stria vascularis.

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ing that the change might have been a vital process. The macula sacculi showed about 50% loss of the neuroepithelium.

In the internal auditory canal there was no pathology. Amylacea bodies were found scattered in the meatus. The wall of the internal auditory artery was thick. The blood vessels in the labyrinth were not pathologic except for the missing strial capillaries. The cochlear and vestibular aqueducts revealed no pathology.



FIG. 10.
The organ of Corti in the middle turn.
The external hair cells are missing in the first and second rows.

Discussion

The etiology of sudden deafness has not been elucidated yet. There are several theories concerning its etiology: 1. vascular theory, 2. viral theory, and 3. membrane rupture theory. There are many diseases which may show severe sensorineural hearing loss of sudden onset. However, sudden deafness of unknown etiology will be discussed here.

We have been doing a nation-wide survey of sudden deafness under the sponsorship of the Ministry of Health and Welfare. As it is still under way, we do not have a definite figure of incidence in our country. However, a rough estimate of the number of patients is 3000 per year. As the criteria for making the diagnosis of the disease, the following principal and additional symptoms were selected: 1. hearing loss of sudden onset, 2. a profound sesorineural hearing loss, 3. unknown cause. The additional

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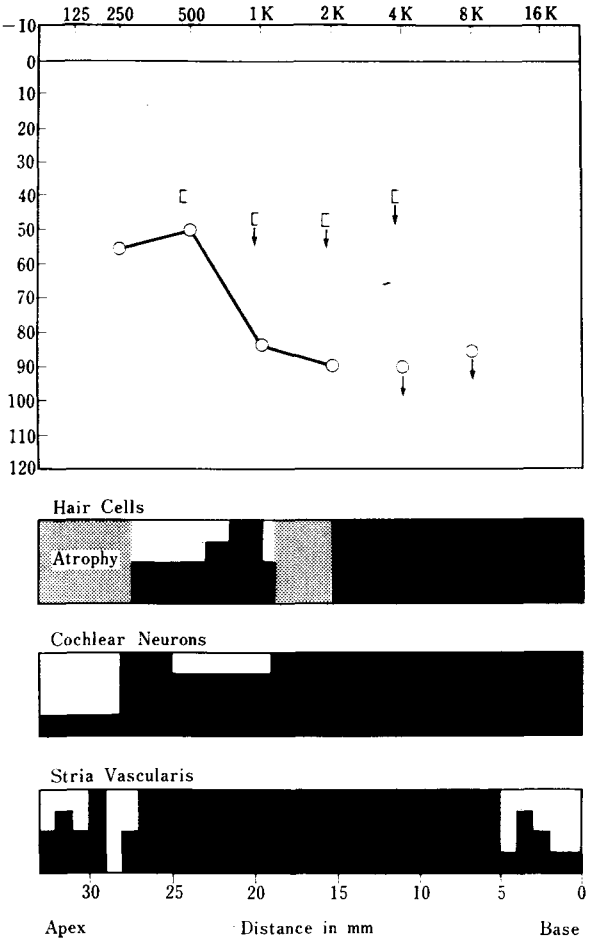


FIG. 11.

Audiogram and histograms showing pathology of hair cells, cochlear neurons, and stria vascularis of the right ear.

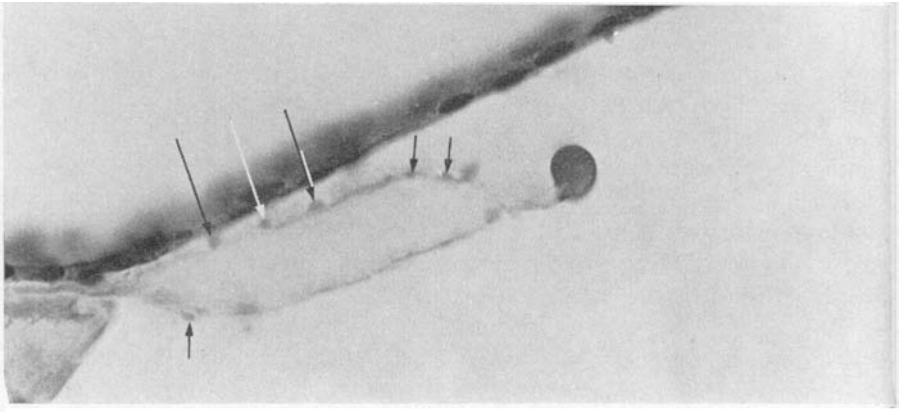


FIG. 12.

A droplet at the marginal zone of the tectorial membrane. The surface of the membrane has small dots (arrows.)

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symptoms were: 1. tinnitus may appear simultaneously, before or after the onset of hearing loss, 2. vertigo may occur with or without nausea and vomiting. However, it does not occur repeatedly. 3. No cranial nerve symptom is noted except for the VIIIth nerve.

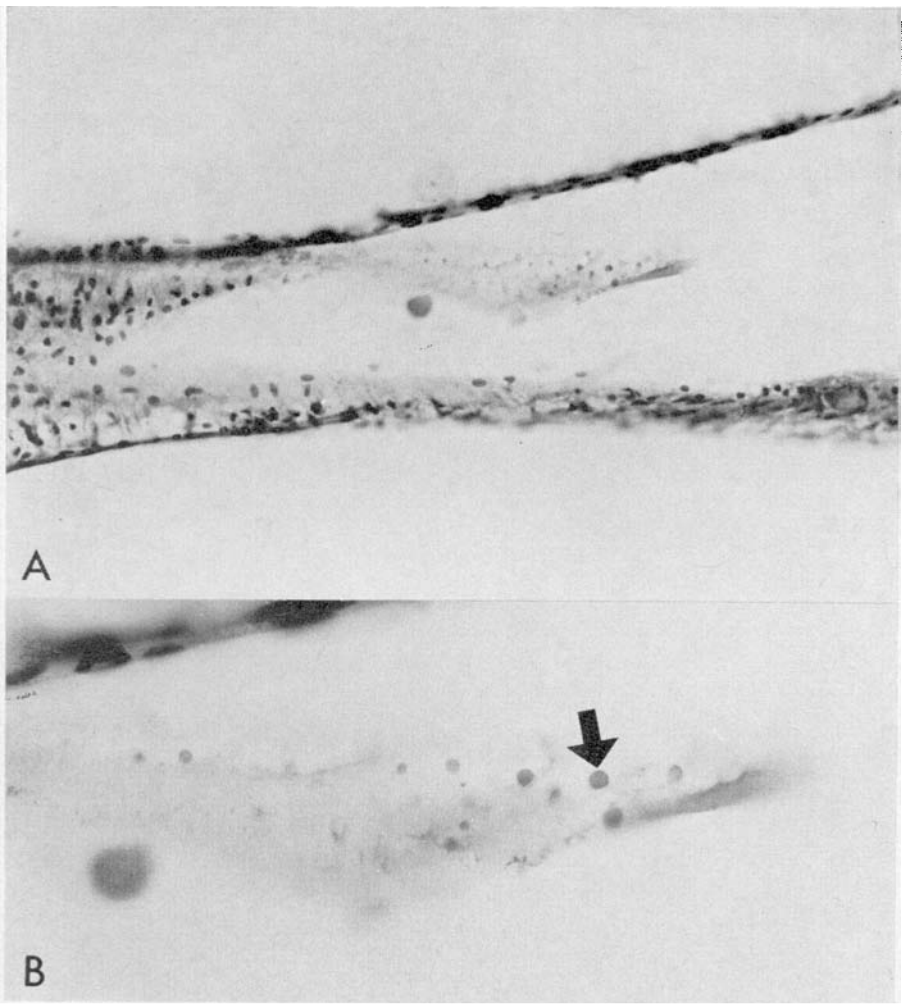


FIG. 13.
A. Many dots are found on the surface of the tectorial membrane.
B. A high power view. An arrow indicates one of the dots.

Strictly speaking, it is hard to make the diagnosis of the sudden deafness, even if we follow the above criteria. It is a well known fact that in 10 per cent of patients with vestibular schwannoma, the initial symptom was a sensorineural hearing loss of sudden onset. Some patients with

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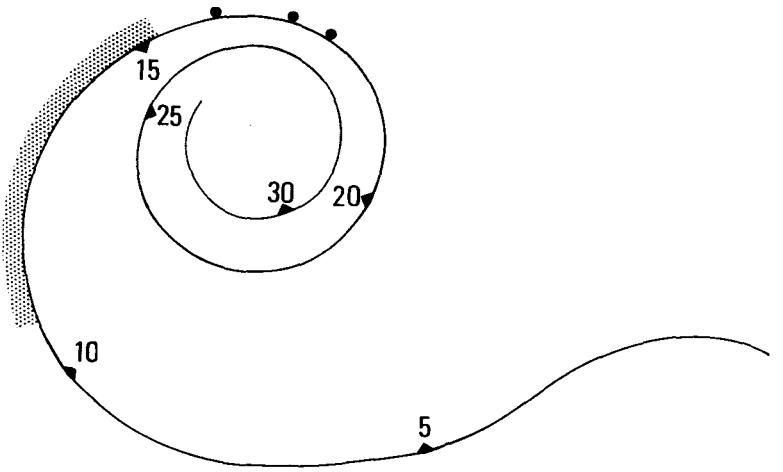


FIG. 14.

Cochleogram of the right ear showing pathology of the tectorial membrane. There are small dots on the surface of the tectorial membrane in the 11–17·5 mm. area. In addition, a large droplet is seen in the 11–15 mm. area (shaded area).

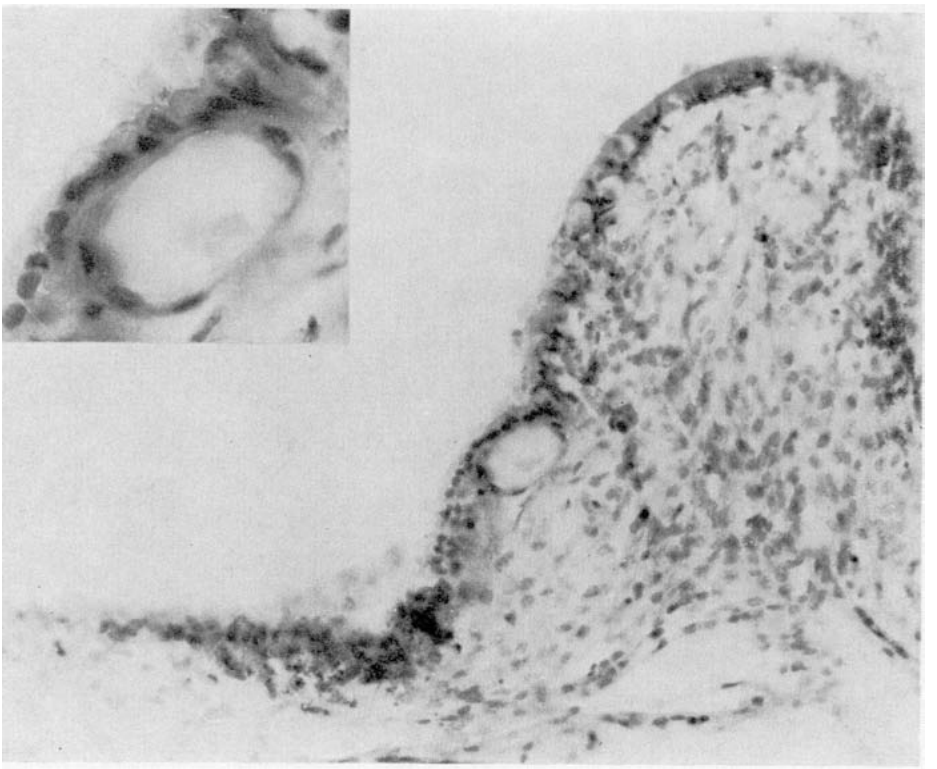


FIG. 15.

Posterior crista. There is a cyst under the transitional epithelium. Inset shows high power view.

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Sudden deafness

Ménière's disease may show sudden hearing loss as the first symptom. Therefore, a long term follow-up is required to confirm the definite diagnosis of sudden deafness.

It is important to make a differential diagnosis from Ménière's disease. In this regard, Takahara *et al.* (1974) reported a patient with

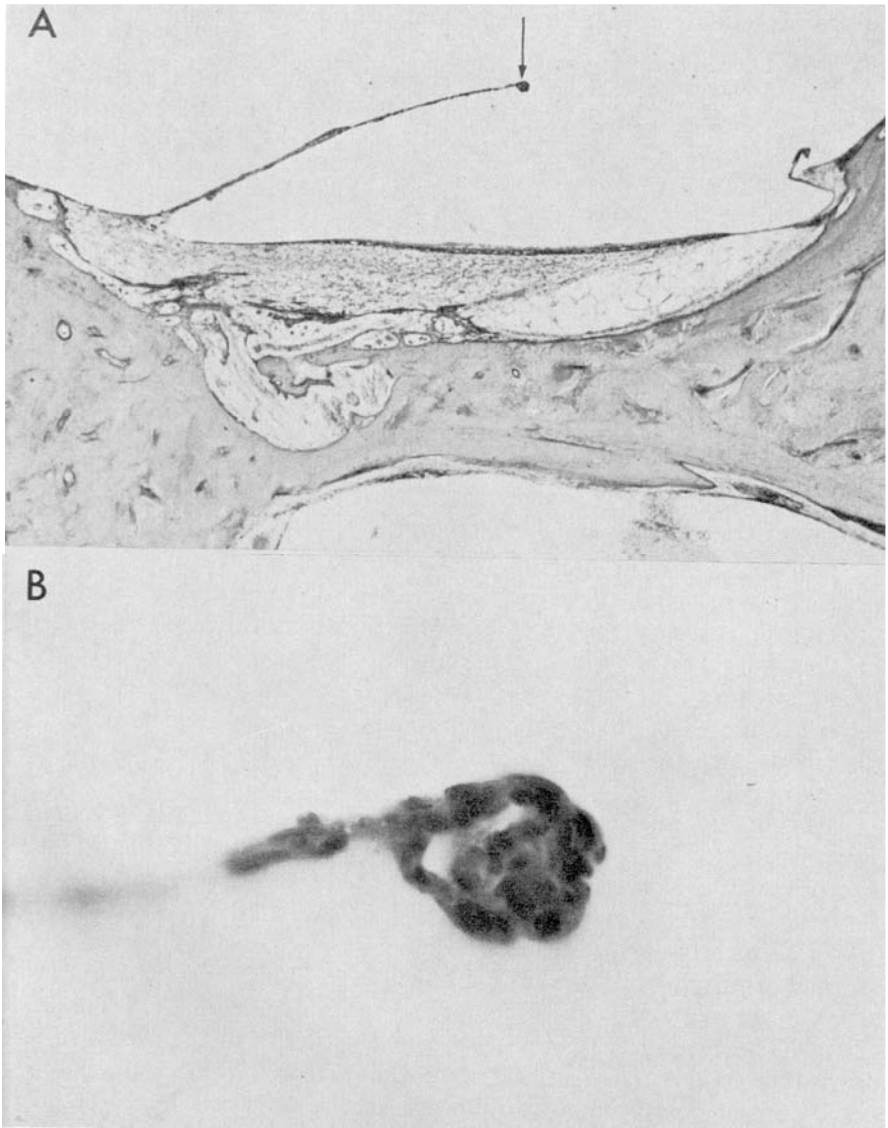


FIG. 16.
A. A perforation in the right saccular wall.
B. The edge (arrow in A) of the perforation shows a rolling.

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sudden deafness, whose temporal bone showed idiopathic endolymphatic hydrops. They reported a case of an 80-year-old male who developed sudden sensorineural hearing loss of the right ear when he suffered from acute nephritis. He had never experienced a vertiginous attack, nausea or vomiting. He died of rupture of a dissecting aneurysm 21 years after the episode of deafness. The temporal bone showed a combination of the idiopathic endolymphatic hydrops and neural and metabolic type presbyacusis.

We (1962) reported bilateral sudden deafness in a patient with Buerger's disease. The patient developed sudden hearing loss of the right ear when he suffered from thromboangiitis obliterans of the radial artery. He lost his hearing on the other side when he had intermittent claudication in the left leg. The sudden deafness of vascular lesions occurs in association with known systemic vascular disease.

The clinical courses of the disease favour the viral theory rather than vascular. If vascular insufficiency is the cause, sudden deafness might occur repeatedly even after recovery. Saunders (1965) was of the opinion that an unusual second attack of sudden deafness after recovery was due to immunity development.

Viral infection of the labyrinth may be caused either by way of the blood stream during a viremia or by direct extension from encephalitis or meningo-encephalitis through the internal meatus and the cochlear aqueduct (Hemenway and Bergstrom, 1967; Lindsay, 1973).

In the case of viral infection by way of the blood stream, the endolymphatic system tends to be primarily affected. Lindsay and Hemenway (1954) proposed a concept of endolymphatic labyrinthitis to viral infection of the inner ear in which the route of infection was by way of the blood stream. The stria vascularis is a site of predilection in viral labyrinthitis. The tectorial membrane has been degenerated and detached from the organ of Corti. Ganglion cells are preserved except in the basal coil.

In contrast, in spread of inflammation from encephalitis or meningo-encephalitis to the labyrinth, structures within the perilymphatic system are primarily involved. Neural elements tend to degenerate. The endolymphatic system may become affected secondarily. Therefore, the tectorial membrane may show degenerative changes. Experimentally, intralabyrinthine and intracranial inoculation of influenza A, mumps and measles viruses were performed in newborn hamsters by Davis *et al.* (1975). They found that inner ear structures differ in their vulnerability to viral infections. The tectorial membrane was free from viral antigen.

Based on postmortem examination of the temporal bones of people with sudden deafness, Schuknecht *et al.* (1973) thought that viral particle circulating in the cerebrospinal fluid found a more ready access to the inner ear through the large cochlear aqueduct.

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Schuknecht *et al.* reported pathology of eight temporal bones of sudden deafness in 1973. The principal pathological changes were atrophy of the organ of Corti, tectorial membrane and stria vascularis. They judged that the findings were more like those occurring in labyrinthitis of known viral etiology such as mumps, rubella and measles than those following vascular lesions observed in animal experimentation. In the vascular obstruction experiment, the tectorial membrane did not show any remarkable changes.

One of the common temporal bone pathologies in sudden deafness was atrophy of the stria vascularis. Almost complete atrophy of the stria vascularis found in the present case could be partly due to metabolic presbyacusis. It is also reasonable to assume that the atrophy resulted from the healing process of a virally infected stria. The autopsy of the 77-year-old woman revealed that the degree of atrophy was severe compared to that observed in temporal bones of patients of the same generation. It is uncertain, however, whether sudden deafness in this case occurred in association with viral infection, although it is probable that the disease may occur by viral infection without clinical evidence.

Emotional episodes apparently had a great influence on the development of the disease in this patient.

The tectorial membrane is divided into three zones: the limbal, middle and marginal zones (Lim, 1972). The outer zone is formed by the marginal band and a marginal net. These two structures are designated as the marginal complex, which is continuous with the cover net on the upper surface of the membrane, and with the homogenous basal layer on the undersurface of the membrane. The thickness of the tectorial membrane is divided into three parts according to Lim (1972): the cover net, the fibrous main body and the homogeneous basal layer or Hardesty's membrane, or both. The main body of the tectorial membrane is formed by fine fibrils and an amorphous ground substance. According to biochemical data, these fibrils are more like keratin than collagen. Histochemical studies further show that this membrane is richly endowed with mucopolysaccharides. Whether or not these mucopolysaccharides are sulphated is a matter of dispute. Lim's study (1972) indicates that the membrane is formed by fibrillar elements and a homogenous ground substance, but cannot conclude whether this dense, amorphous substance of the specialized sub-structures, such as the cover net, the marginal network, Hensen's stripe, and Hardesty's membrane, is chemically identical to the ground substance of the main fibrous layer. The patient did not suffer from systemic disease of mucopolysaccharides such as in Hurler's and Hunter's diseases.

It seems strange to see the changes of the tectorial membrane found in this patient if it occurred 31 years ago. Did this change found in the tectorial membrane really occur 31 years ago? Was it a postmortem

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TABLE I

SUMMARY OF SUDDEN DEAFNESS CASES APPEARING IN THE LITERATURE.
A. History and clinical records.
B. Histopathological findings.

A

Case	Age of onset	Sex	R or L	Associated disorders	Hearing loss	Vertigo	Cause of death	Deaf to Autopsy	Test to Autopsy	Death to Autopsy	Author
1	39	♀	R	Headcold	++	–	Terminal diffuse carcinomatosis	20 yrs.	3wks.	10 hrs.	Schuknecht *et al.*
2	43	♀	L	Acute pharyngitis	+++	+	Ulcerative colitis	11 mths.	6 mths.	15 hrs.	,,
3	63	♂	R	Naso-pharyngitis, acute respiratory distress	++++	–	Leukaemia respiratory insufficiency	15 mths.	14 mths.	20 hrs.	,,
4	52	♀	L	?	++++	?		33 yrs.	20 yrs.	Unknown	,,
5	65	♀	R	Headache	++++	+		20 yrs.	20 yrs.	Unknown	,,
6	58	♂	R	Headache, femoral and splenic thrombosis	+++	–	Basilar broncho-pneumonia	9 days	6 days	6 hrs.	,,
7	63	♂	L	?	++++	+++	Gastro-intestinal bleeding Laennec's cirrhosis, cardiac failure	4 yrs.	4 yrs.	13 hrs.	,,

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8	68	♂	L	Viral, pneumonia, headache	+++	−	Anaemia, duodenal ulcer, complication of hemigastrectomy, cachexia	19 yrs.	19 yrs.	8 hrs.	,,
9	36	♀	R	Cold	++	−	Peritonitis		2 yrs.	2 hrs.	Beal *et al.*
10	34	♀	L		+++		Resuscitation	6 yrs.	6 yrs.	13 hrs.	,,
11	59	♂	R	Acute nephritis	+++	−	Dissecting aneurysm	21 yrs.	19 yrs.	0·5 hrs.	Takahara *et al.*
12	46	♀	R	Emotional episode	+++	++	Suffocation	31 yrs.	4 yrs.	9·5 hrs.	The present case
13	46	♀	L	Emotional episode	+++		Suffocation	31 yrs.	4 yrs.	9·5 hrs.	,,

B

Case	Organ of Corti		Tect. memb.	Reissner's memb.	Stria	Cochlear neurons	Saccule	Utricle cristae	Other changes
	Hair cells	Supp. cells							
1		+++	+++ shrunken	Normal	+		Ruptured collapsed 90% < HC loss	Normal	Otosclerosis
2	+ Atrophied	+		Normal			Normal		
3	+++ OC severe atrophy	+++ collapse of pillars, distortion of H's and Ds' cells		+++ Collapse in basal turn	Collapsed		Normal		Otosclerosis

Case	Organ of Corti		Tect. memb.	Reissner's memb.	Stria	Cochlear neurons	Saccule	Utricle cristae	Other changes
	Hair cells	Supp. cells							
4	+++ OC missing in basal half	+ Well preserved in apical half	Normal	Normal	+++ Severe atrophy	+ 90% loss in basal 20 mm., 40–70% in apical region	Normal	Normal	Saccular Enlargement large coch. aqueduct
5	+++ Missing in parts of basal turn	+	Normal		Patchy atrophy in apical half	90% loss in basal 8 mm., 70% in 8–17 mm., 40% in the remainder	Normal	Normal	Saccular enlargement large coch. aqueduct
	50% present in the remainder	Well preserved							
6	Severe shrinkage, in 10–16 mm., HC identified	+	+++ Small homogen. sphere in basal 15 mm., missing in the remainder		+ Severe shrinkage		Normal	Normal	
7	+++ Missing or flattened in the basal 13 m.,	++	Normal	Normal	+++ 90 % miss. in balsa 4 mm., 50 % in 4–21 mm., 80% in 21 to apex		Normal	Normal	Large coch. aqueduct
	50% EHC missing in 13 mm. to apex	Present 13 mm. to apex							

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	+ OC missing or small mound in basal 13 mm. Normal HC popul. in remainder	+++ Pillar's, Ds' missing	Atrophied throughout	Normal	+++		Small disruption of sensory epithel. Normal
9	+ HC identified		+++ Atrophy distortion cellular encasement	Collapsed in lower basal and middle turn	+++ All areas except very apex		Collapsed normal
10	++ OC collapsed		+++ Atrophy distortion cellular encasement	Normal			Normal
11	Present		Normal	Collapsed	Segmental atrophy	Missing in lower basal turn	Distended saccular wall and duct. reuriens
12	Missing, atrophy and degeneration		Degeneration	Normal	+++ severe atrophy	Complete loss in the upper middle and basal turns, less toward apex	saccular wall showed a perforation Moderate loss of sensory epithelium
13	Atrophy		Degeneration	Normal	+++ Severe atrophy	Complete loss 0–17 mm. area, less apicalward	Moderate loss of sensory epithelium

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change? Did the change occur independently of the sudden deafness shortly before death? As the tectorial membrane seldom showed pathology, we thought it occurred at the time of sudden deafness. The tissue does not show this type of morphology in postmortem changes. A possible explanation for this is that the tectorial membrane changed its physicochemical property by either viral infection or unknown causes. A sol to gel change might occur. The tissue does not have a cellular element, indicating that there is no lysosome in it. Therefore, infected and/or altered tissue holds its shape without being digested for many years.

A spherical tectorial membrane is known to occur in viral labyrinthitis. Schuknecht found spherical distortion of the tectorial membrane in the specimens (cases No. 6 and 8) from patients in which the onset of the disease to autopsy was 9 days and 19 years (Schuknecht *et al.*, 1973). The change was noted also in temporal bones of the trisomy syndrome (Lindsay, 1973; Saito *et al.*, 1974), Mondini type anomaly (Lindsay, 1973) congenital deafness (Makishima, 1972; Altmann, 1950), measles and influenza (Lindsay, 1971). Similar findings of the tectorial membrane were observed in cats deafened experimentally (Schuknecht *et al.*, 1951). According to Lurie (1948), the tectorial membrane lies under the vestibular lip in the internal sulcus in a deaf dalmatian dog. The tectorial membrane appeared swollen and ballooned out, filling the internal sulcus, and it had strands of tissue binding it down. Also he found that in a deaf collie dog, the tectorial membrane appeared to have been solidified as if made up of a coagulum. In cross-section it has the appearance of a shelf projecting into the scala media.

Reviewing temporal bone reports of sudden deafness (Schuknecht *et al.*, 1962, 1973; Beal *et al.*, 1967; Takahara *et al.*, 1974) 5 ears out of 11 showed pathology of the tectorial membrane. As the present case showed changes of the tectorial membrane bilaterally, 7 ears out of 13 had pathology of the membrane (Table).

Of particular interest in the present case is disintegration of the tectorial membrane. A large droplet observed in the tectorial membrane of the left cochlea is not identical with those reported in previous papers on sudden deafness. The dot formations on the surface of the tectorial membrane of the right cochlea were not reported previously. The droplet, thick mass on the limbis, and the dots are the same substance as far as histochemical study is concerned. These are stained by alcian blue, toluidine blue and P.A.S. It is red in Azan specimens.

As the tectorial membrane shows weak or no staining for the staining procedures, it is suggested that the membrane has altered its chemical property and an altered substance comes out of the tissue. The substance must be hard, like keratin. The change (coagulation necrosis) occurred primarily in the limbal portion.

We are of the opinion that in some cases a site of the disease of sudden

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deafness is the tectorial membrane. Possibly changes in the physico-chemical property of the tectorial membrane occur all of a sudden, caused by either viral infection or other unknown etiology. In this regard, Lindsay thought that malformation of the tectorial membrane and Corti's organ might be the direct cause of deafness following maternal rubella (Lindsay *et al.*, 1953). In specimens of the human temporal bone, the tectorial membrane is usually lifted up from the sensory cells. Therefore, it is hard to tell the premortem state of the tissue. In case the tissue showed morphological changes it is obvious that the tectorial membrane-sensory hairs relationship is pathological. As far as deafness is concerned, either detachment or ultra-structural pathology at the junction of the sensor hairs and the tectorial membrane is significant. The 'rolled up', shrunken, and droplet states of the tectorial membrane are the final stages of a succession of morphological changes, the initial stage of which makes deafness.

Summary

Histopathological studies were performed in the temporal bones from a 77-year-old woman who had bilateral sudden deafness 31 years before her death. The common findings in both cochleas were atrophy of the organ of Corti and stria vascularis, and degeneration of the tectorial membrane. Pathology of the tectorial membrane consisted of atrophy and droplet formation of the middle and marginal zones. In one area, an atrophied tectorial membrane was lifted up by a mass on the limbus.

The saccular wall in the left temporal bone was ruptured. There was no pathology in the vascular system in the labyrinths except for moderate degeneration of sensory epithelium.

The pathogenesis of sudden deafness was discussed in the light of the present findings.

Alteration of the tectorial membrane seemed to be the main factor as a possible cause of sudden deafness.

Histopathological findings were reviewed from the literature.

REFERENCES

Altmann, F. (1950) *Archives of Otolaryngology*, **51**, 852.

Beal, D. D., Hemenway, W. G., and Lindsay, J. R. (1967) *Archives of Otolaryngology*, **85**, 591.

Davis, L. E., Shurin, S., and Johnson, R. T. (1975) *Nature*, **254**, 329.

Hemenway, W. G., and Bergstrom, La V. (1967) In F. McDonnell and P. H. Ward (Eds.): *Deafness in Childhood*, pp. 91–122. Vanderbilt University Press.

Kirikae, I., Nomura, Y., Shitara, T., and Kobayashi, T. (1962) *Archives of Otolaryngology*, **75**, 502.

Lim, D. J. (1972) *Archives of Otolaryngology*, **96**, 199.

Lindsay, J. R., Caruthers, D. G., Hemenway, W. G., and Harrison, S. (1953) *Annals of Otology, Rhinology and Laryngology*, **62**, 1201.

Lindsay, J. R., and Hemenway, W. G. (1954) *Annals of Otology, Rhinology and Laryngology*, **63**, 754.

https://doi.org/10.1017/S0022215100083213 Published online by Cambridge University Press

LINDSAY, J. R. (1971) *Otolaryngologic Clinics of North America*, **4,** 249.
LINDSAY, J. R. (1973) *Annals of Otology, Rhinology and Laryngology*, **82,** Suppl. 5.
LURIE, M. H. (1948) *Laryngoscope*, **58,** 279.
MAKISHIMA, K. (1972) *Audiology Japan*, **15,** 145.
SAITO, H., OKANO, Y., FURUTA, M., ASAMOTO, H., FUJITA, H., and TAKEUCHI, T. (1974) *Archives of Otolaryngology*, **100,** 386.
SAUNDERS, W. H. (1965) In A. B. Graham (Ed.): Sensorineural Hearing Processes and Disorders. pp. 403–410. Little Brown and Co., Boston, Mass.
SCHUKNECHT, H. F., NEFF, W. D., and PERLMAN, H. B. (1951) *Annals of Otology, Rhinology and Laryngology*, **60,** 273.
SCHUKNECHT, H. F., KIMURA, R. S., and NAUFAL, P. M. (1973) *Acta Oto-otolaryngologica*, **76,** 75.
TAKAHARA, S., SAITO, R., KONISHI, S., and IGARASHI, M. (1974) *Japanese Journal of Oto-Rhino-Laryngology*, **77,** 959.

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