AUDITORY NERVE TUMOURS

By PHILIP SCOTT (London)*

AUDITORY NERVE TUMOUR, although a comparatively rare disease, presents so many problems both from the point of view of diagnosis and treatment, that it is hoped an account of the clinical history and post-mortem pathological findings in the temporal bones in three cases, will be of interest.

During the last three and a half years I have obtained twenty-six temporal bones from patients who suffered from auditory nerve tumours, many of whom I had the opportunity of examining during life. Of these twenty-six bones, there are twelve still in the process of decalcification. I have chosen six bones, from the fourteen already cut in serial sections, upon which to base these observations. I take this opportunity of saying that I am preparing a more complete survey of all these cases from clinical, surgical, and pathological points of view.

Owing to the co-operation necessary to obtain material and the labour involved in cutting serial sections of the temporal bones, it is perhaps natural that many of the papers on this subject are directed towards the clinical aspect. I am hoping to make out a good case for the routine pathological examination of both temporal bones, whenever VIIIth-nerve tumour has been suspected during life.

The late Mr. Theodore Just read an interesting paper[1] before the Section in 1930 upon the diagnosis of acoustic nerve tumours, based upon thirty-three unilateral cases examined by him in six years at the National Hospital. At the same meeting the late J. S. Fraser[2] and Mr. W. T. Gardiner reported upon thirteen cases, one of which was bilateral, seen at the Royal Infirmary, Edinburgh, during eleven years. In March, 1932, Professor de Klejn[3] and the late Dr. Albert Gray read a paper describing a case of acusticus tumour involving both auditory nerves in von Recklinghausen's disease. In December, 1932, Mr. F. C. Ormerod[4] described a similar case in a patient aged 14.

* From the Ferens Institute of Otology, Middlesex Hospital.

https://doi.org/10.1017/S0022215100004102 Published online by Cambridge University Press

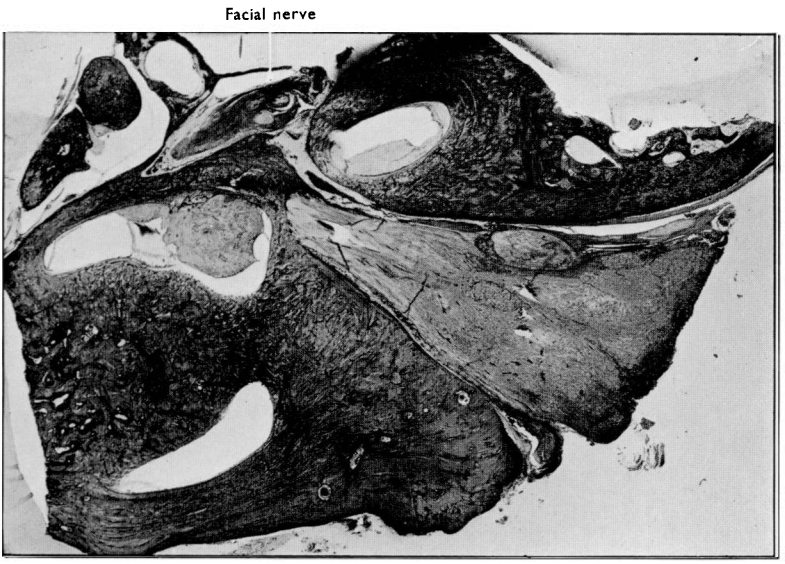


FIG. 1 (Case I, No. 158).—Left/180. Neurofibroma filling the internal auditory meatus and involving the vestibule. There is some growth in the facial nerve.

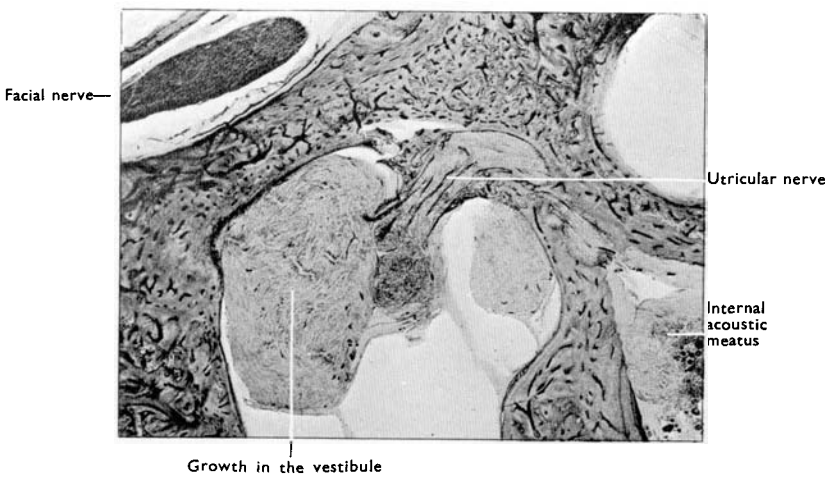


FIG. 2 (Case I, No. 158).—Left/220: The growth has followed the utricular nerve and destroyed the utricular macula.

[face p. 772

https://doi.org/10.1017/S0022215100004102 Published online by Cambridge University Press

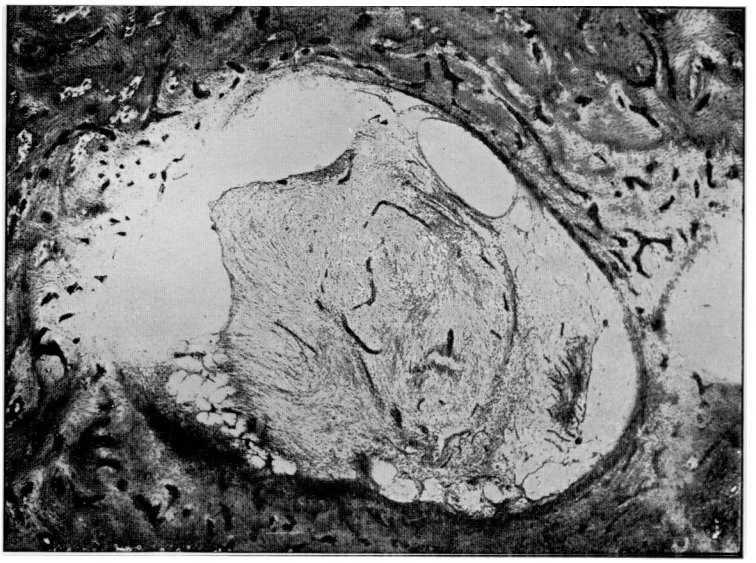


FIG. 3 (Case I, No. 158).—Left/160; Superior semicircular canal filled with growth.

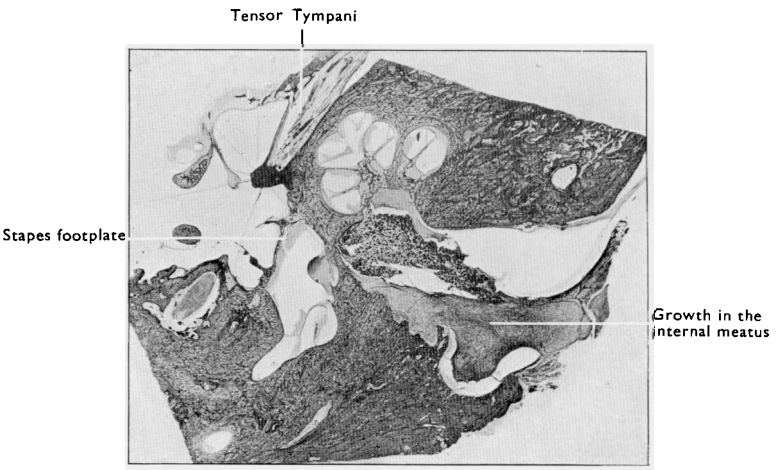


FIG. 4 (Case I, No. 158).—Left/270: Pressure expansion of the meatus by growth. Modiolus of cochlea and footplate of stapes free.

https://doi.org/10.1017/S0022215100004102 Published online by Cambridge University Press

The pathological report on the left bone only was given by Dr. Gray[5] in 1933, together with that of another bilateral acusticus tumour in von Recklinghausen's disease.

Dr. Gray[5] said that apart from von Recklinghausen's disease acusticus tumours were practically never bilateral, and Mr. Norman Patterson quoted Henschen's statistics (245 to 24) and Harvey Cushing's (100 to 1).

Dr. Gray stressed the following points :

(1) All three were cases of von Recklinghausen's disease.

(2) In all the cases the tumour was bilateral.

(3) New bone, similar to otosclerosis, was found in the labyrinthine capsule in two cases, and new bone in the apex of the cochlea in one case.

(4) Vacuolization of the cells of the stria vascularis in the first case.

In Mr. Ormerod's case the hearing in the left ear was normal two months before death, but histological examination showed a small deposit of tumour in the lowest whorl of the cochlea.

My particular interest in this subject was aroused by these papers and by the opportunity of examining several cases for Mr. Just at the National Hospital.

Of the three cases, which I shall now describe, one was examined by Mr. Just and the bone was given to me by Dr. Gray at the Ferens Institute, where I have had the privilege of preparing all my specimens ; the other two patients were examined by me when I was acting for Mr. Just.

CASE I (No. 158).—The patient, a woman aged 65, had had some degree of deafness in the left ear for twelve years, following otitis media, and for two years had had deafness in the right ear, also following otitis media.

She had had failing vision for two years and weakness of the right lower extremity for one year. She had had no vertigo, and no tinnitus.

Physical signs.—No facial asymmetry when in repose, but slight right facial weakness or ? left facial spasm. Eyes: 3 to 4 D papill-œdema in each eye ; vision : $\frac{6}{18}$; $\frac{6}{24}$. Nystagmus rapid and coarse on lateral deviation in both directions. Outward movement of the right eye slightly impaired. Left corneal reflex diminished.

Report on hearing (15.1.34) : " Deafness in left ear ; cannot hear by bone conduction (left), with noise-box in the right ear. Cold caloric test left : no added nystagmus, giddiness, or falling reactions.

https://doi.org/10.1017/S0022215100004102 Published online by Cambridge University Press

Left VIIIth nerve apparently not functioning in either division." Unfortunately there is no specific reference to the right ear.

I have deliberately excluded details of reflexes as being beyond the scope of this paper, but a diagnosis of probable left acusticus tumour was made.

No details are available of the operation performed on 1.2.35, but the patient died on the following day.

Report on post-mortem examination (Dr. J. G. Greenfield) : When the brain was removed, " a soft acoustic neurofibroma was found attached to the left VIIIth nerve and entering the left internal meatus which was much eroded and enlarged, so that a piece of tumour the size of a bean was present under the dura mater. This was surrounded antero-externally by a cystic collection of yellowish fluid."

There is no mention of any other tumour masses or fibromata elsewhere in the body, but fortunately both temporal bones were preserved, and after decalcification I cut them in serial sections in the horizontal plane.

I wish to emphasize the fact that up to 24.1.35—one week before death—the clinical history was apparently taken by word of mouth. The house-physician records that the hearing then was the same as on 15.11.34, when the first report was made. The patient's husband, to whom I wrote in June, 1938, confirms the fact that there was considerable hearing capacity in the right ear.

The sections from the left bone show neurofibroma filling the internal auditory meatus and extending outwards along the line of least resistance ; the tumour has followed the nerve to the macula utriculi and expanded into the vestibule (Figs. 1 and 2). Earlier sections show the upward extension of the tumour mass in the vestibule, involving the ampullae of the superior and horizontal semi-circular canals (Fig. 3). Sections lower down, passing through the stapes footplate and the modiolus of the cochlea (Fig. 4) show this part of the vestibule and the saccule to be free from growth. There is no sign of otosclerosis in this bone. The cochlea at this level (Fig. 6) shows almost complete degeneration of the spiral ganglion cells. There is also considerable change in Corti's organ ; this latter is probably post-mortem disintegration (Fig. 8). Sections below the centre of the modiolus show growth filling the spaces of the modiolus and spreading outwards into the scala tympani. This growth is in direct continuity with that in the meatus (Fig. 7).

One striking feature of this tumour is the presence of large numbers of psammoma degeneration bodies (Fig. 5) usually found in meningiomata, but the typical palisade formation and whorling fibrous structure of true neurofibromata, seen well in Fig. 2, establish beyond question the diagnosis in this case.

The right bone was examined for comparison, but in spite of the

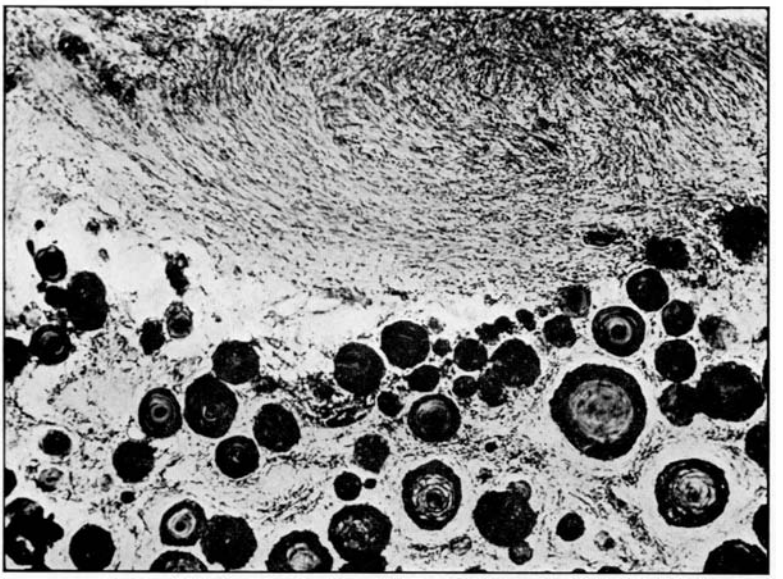


Fig. 5 (Case I, No. 158.)—Left : Growth and psammoma bodies.

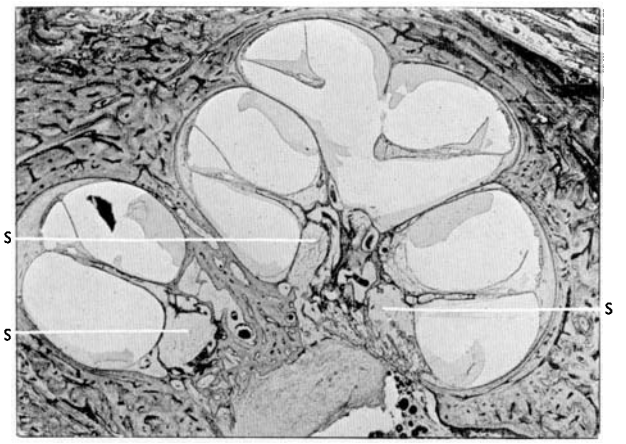


Fig. 6 (Case I, No. 158).—Left/270 : Modiolus of cochlea. S = Absence of spiral ganglion cells. Albuminous coagulum in cochlea.

[face p. 774

https://doi.org/10.1017/S0022215100004102 Published online by Cambridge University Press

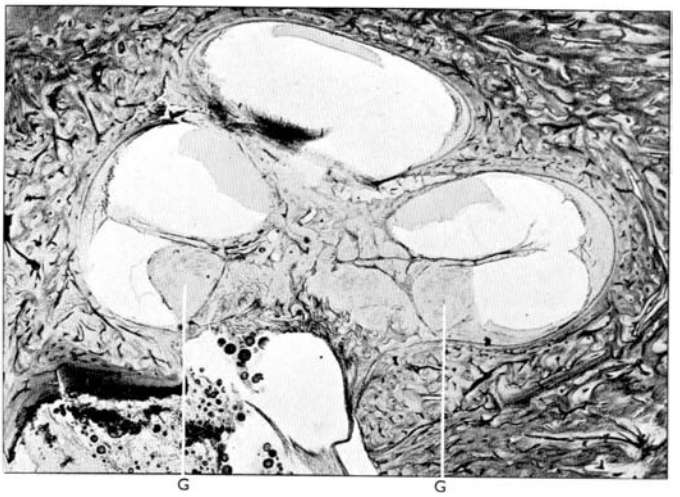


FIG. 7 (Case I, No. 158).—Left/250 : Cochlea at a higher level. G=Growth spreading up the modiolus into the scala tympani.

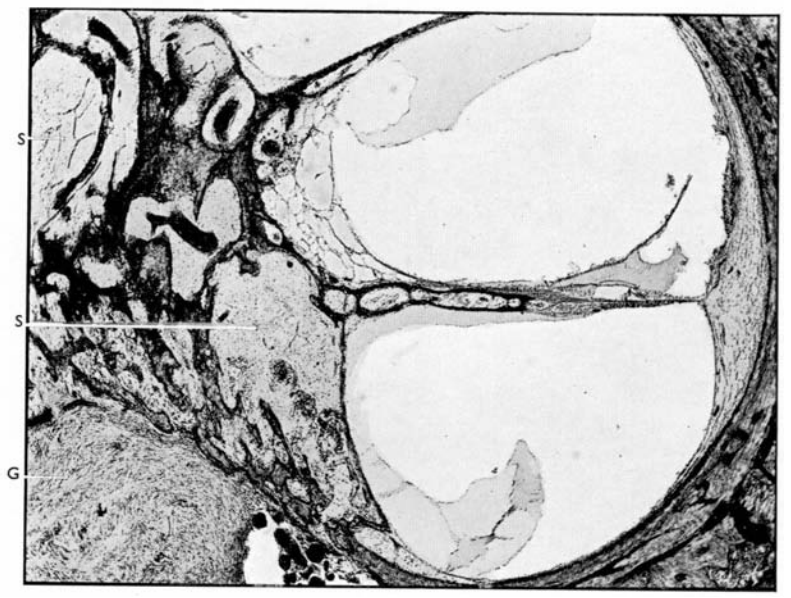


FIG. 8 (Case I, No. 158).—Left/270. S=Spiral ganglion degenerate. G=Growth in modiolus of cochlea.

https://doi.org/10.1017/S0022215100004102 Published online by Cambridge University Press

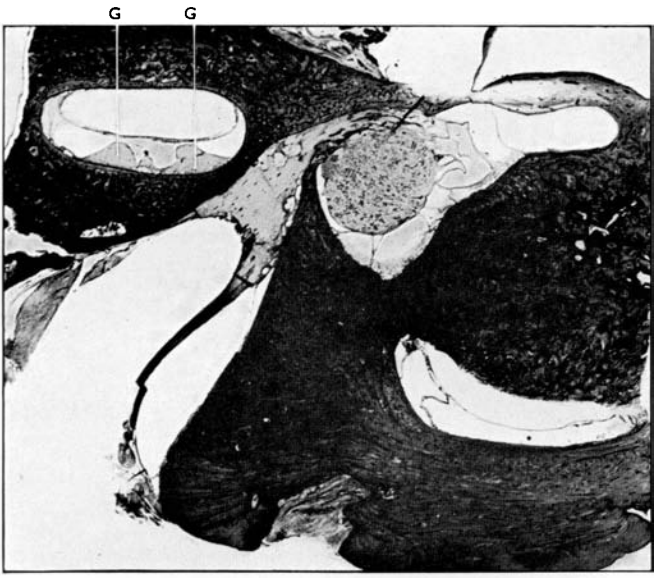


FIG. 9 (Case I, No. 158).—Right/160. Neurofibroma expanding in the vestibule. Note nodules of growth in the cochlea G.

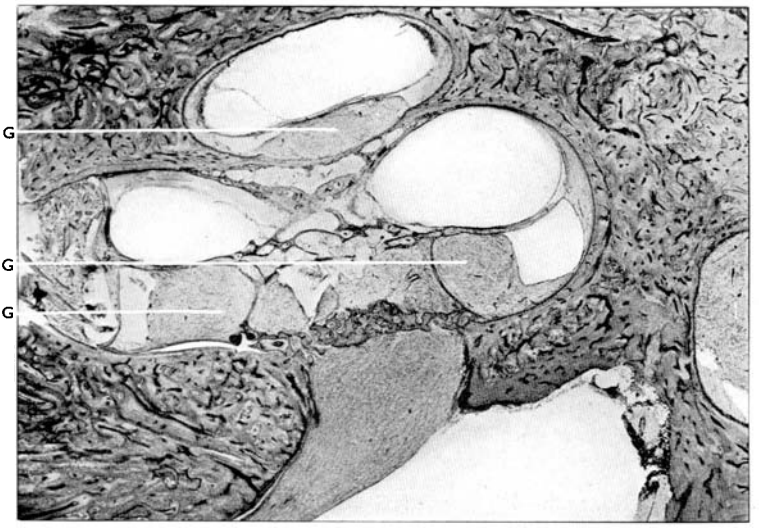


FIG. 10 (Case I, No. 158).—Right/240: G=Growth passing up the modiolus of the cochlea and spreading into the scala tympani.

https://doi.org/10.1017/S0022215100004102 Published online by Cambridge University Press

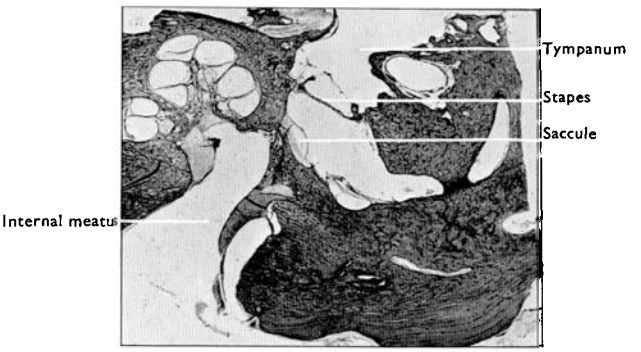


FIG. 11 (Case I, No. 158).—Right/230 : Growth in the lower branches of the vestibular nerve. Stapes free. No otosclerosis.

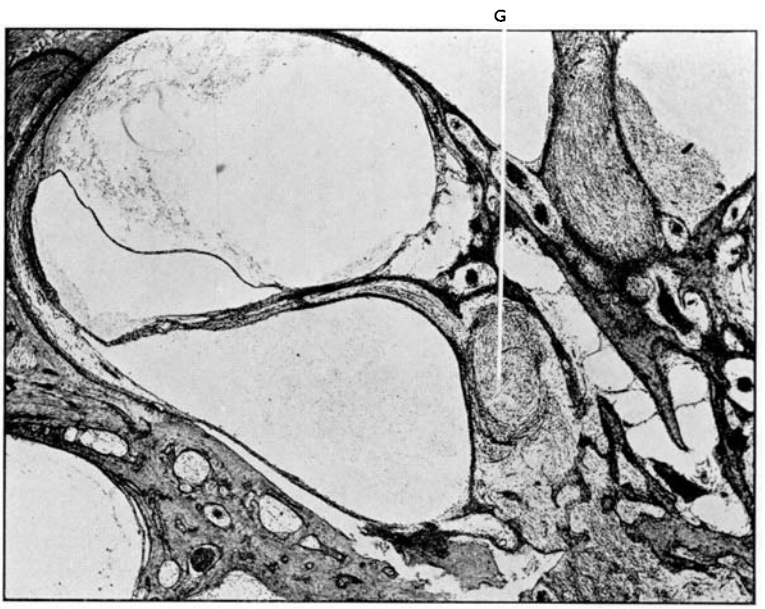


FIG. 12 (Case I, No. 158).—Right/230 : G = Growth replacing ganglion spirale.

https://doi.org/10.1017/S0022215100004102 Published online by Cambridge University Press

apparent ability of the patient to hear the spoken voice in the ear one week before death, this bone shows involvement by neurofibroma strikingly similar in distribution and structure to that found in the left (Fig. 9). The growth is involving the vestibule and distorting the crista of the horizontal canal, of which only a remnant is visible, and there is also growth in the upper part of the cochlea.

In (Fig. 10) the cochlea is seen to be widely invaded by growth and sparsity of spiral ganglion cells is also apparent.

The stapes and the lower vestibule (Fig. 11) show freedom from growth. In none of the sections described was any sign of otosclerosis or new bone-formation found, and later I shall refer to a possible explanation of Dr. Gray's[3] findings in this connection.

CASE II (No. 183).—The sections next to be described were made from the temporal bones of a woman aged 50. The left bone was cut in the horizontal plane, and the right bone in the vertical plane, in the line of the superior semi-circular canal. This patient had a right-sided meningeal endothelioma, mostly in the middle fossa, so that many of the physical signs, although of extreme interest neurologically, are scarcely relevant to this discussion, but my reason for including the case is that a large tumour in the right ponto-cerebellar angle was noticed post mortem. This proved to be a neurofibroma, but after cutting sections of both bones, I found a much more extensive tumour in the left bone, although there was no evidence of this growth visible at the autopsy.

History.—Diplopia, two years; deafness, three months. The first symptoms were referable to paresis of the left external rectus oculi, and later paresis of the right external rectus. Three months before admission the patient complained of deafness in both ears for the first time, and this progressed so rapidly that in from four to six weeks she became stone deaf, and all communication with her had to be made in writing. She had no headaches or vertigo, and only occasional slight tinnitus in the head—not referred to either ear.

Physical signs.—Eyes: 3 to 4 D papillœdema in each eye. Right, marked internal strabismus, with no movement outwards beyond the mid-line. Marked proptosis.

Left, movement now full. On deviation to the left there was a fine horizontal nystagmus with a rotary element. No facial weakness. Co-ordination of limbs good: no dysdiadokokinesia. Cerebrospinal fluid: no cells; protein 0·08 per cent. X-rays showed bone destruction near the right optic foramen.

Report on ears (13.8.36): "Tympanic membranes normal." Hearing tests: "She apparently hears no sound by air or bone conduction. Vibration of some forks felt, but not heard. Caloric and rotation tests do not give rise to any labyrinthine response.

Galvanic reactions will be carried out." Conclusion: "Both labyrinths are defunct."

Operation (30.8.36).—A right-sided middle-fossa tumour was discovered. It was too extensive to be removed by one operation, and a second attempt was made on 25.9.36.

Dr. Greenfield reported as follows upon the tissue removed at the operation:

"The tissue consists of irregular bundles and large whorls of fairly fleshy, fusiform cells with fairly large, oval, clear nuclei. Only a few small whorls are seen. There is much collagen, both as rounded knots and thick trabeculae running irregularly through the tumour. Mitoses are very rare, but a few definite figures are seen. Diagnosis: The tumour is an arachnoidal endothelioma (exothelioma).

Unfortunately the patient died soon after the second operation.

Post-mortem findings (14.10.36).—"A large mass of tumour adherent to the bone in the region of the right cavernous sinus. The bone in the region of the tumour showed superficial osteitis. A firm whitish tumour the size of a hen's egg lay in the *right* ponto-cerebellar angle, where it protruded as an easily detached tongue into the right internal auditory meatus. This tumour proved to be an acoustic neurofibroma. Two smaller, rounded sessile masses probably true fibromata, were found on the dura of the vertex." No other neurofibromata were found.

The sections obtained from Case II show on the right side—in which a tumour was noticed post mortem—a large mass of neurofibroma filling the fundus of the meatus, but although it is in close relationship with the facial nerve in this situation, there is no apparent involvement.

Fig. 13 illustrates a section of the cochlea cut in the vertical plane through the modiolus, and a higher magnification shows that the hair-cells are fairly well preserved, but there is a marked absence of spiral ganglion cells. There is no invasion by growth of the perilymphatic space. On this side the growth lay in the ponto-cerebellar angle and did not spread widely in the temporal bone, although a tongue of growth can be traced following the course of the utricular nerve, and expanding beneath the macula utriculi (Fig. 14).

The routine examination of the unsuspected bone from the left side exposed the fact that a neurofibroma of much greater size filled the meatus and spread into the vestibule which, at one point, was completely obliterated (Fig. 15). From here the growth extended upwards to occupy the ampullae of the superior and horizontal canals (Fig. 16) where the crista are lifted up and compressed, the growth lying between the bony wall and the endosteum.

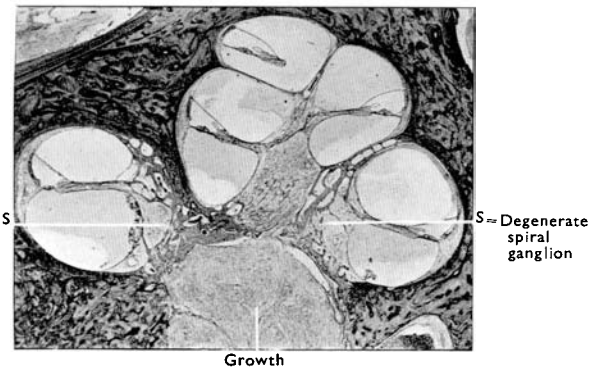


FIG. 13 (Case II, No. 183).—Right/310: Corti's organ well preserved. Few spiral ganglion cells. No spread of growth into the cochlea.

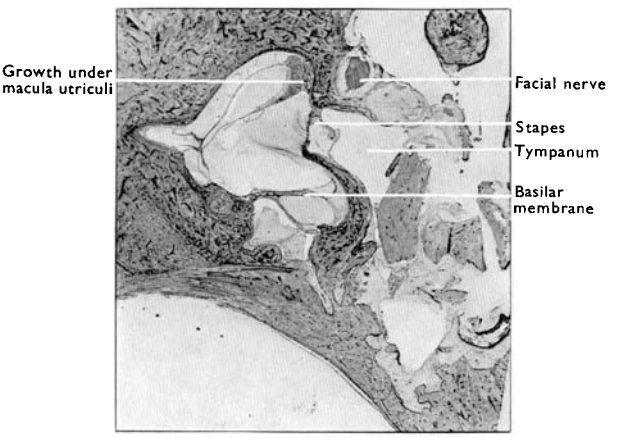


FIG. 14 (Case II, No. 183).—Right/510: Vertical section passing through macula utriculi, invaded by growth. Stapes free. Some coagulum in the vestibule.

[face p. 776

https://doi.org/10.1017/S0022215100004102 Published online by Cambridge University Press

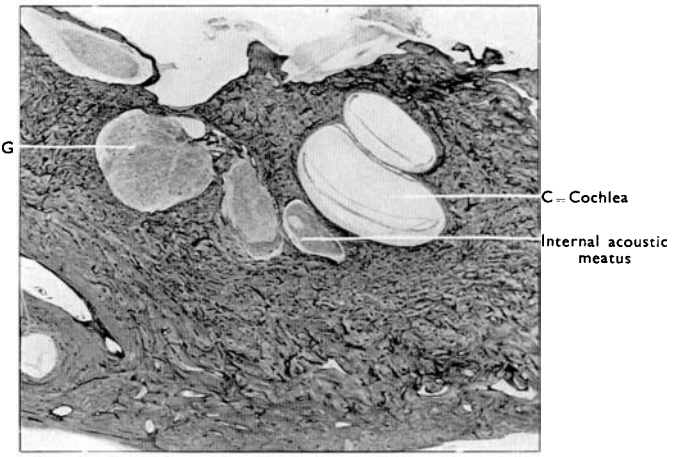


FIG. 15 (Case II, No. 183).—Left/150 : Left temporal bone (horizontal section). G=Growth completely filling the vestibule. No sign of the macula utriculi. Facial nerve free.

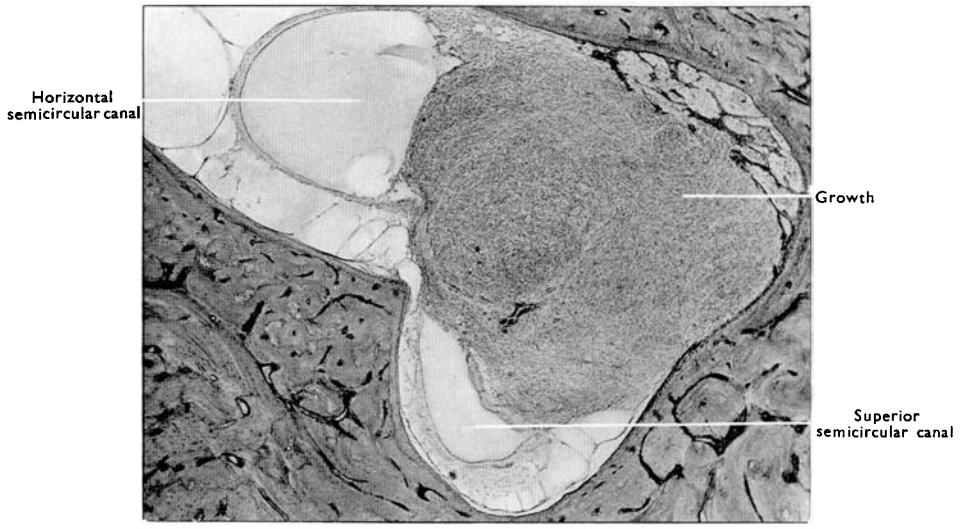
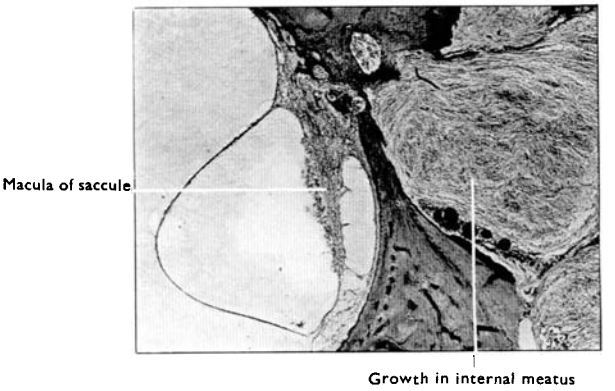
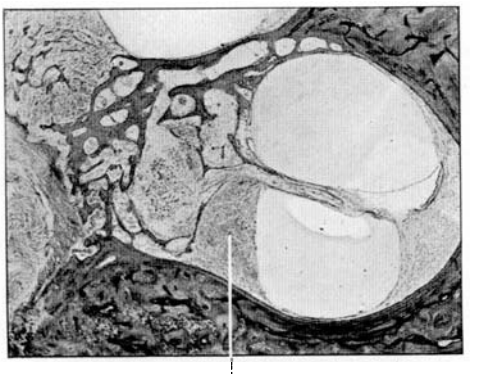


FIG. 16 (Case II, No. 183).—Left/100 : Ampullae of the superior and horizontal canals distorted by growth which almost fills the space.

https://doi.org/10.1017/S0022215100004102 Published online by Cambridge University Press



(A)



(B)

FIG. 17 (Case II, No. 183).—Left: (A) Macula sacculi with growth replacing the saccular nerve. (B) Growth in scala tympani. Note absence of spiral ganglion. Acellular coagulum in scala vestibuli.

https://doi.org/10.1017/S0022215100004102 Published online by Cambridge University Press

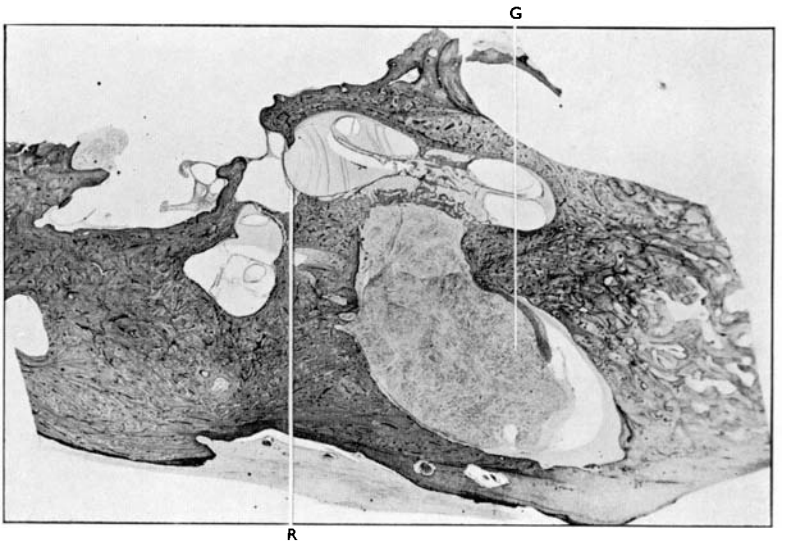


FIG. 18 (Case II, No. 183).—Left/300. R=Round window membrane. G=Growth.

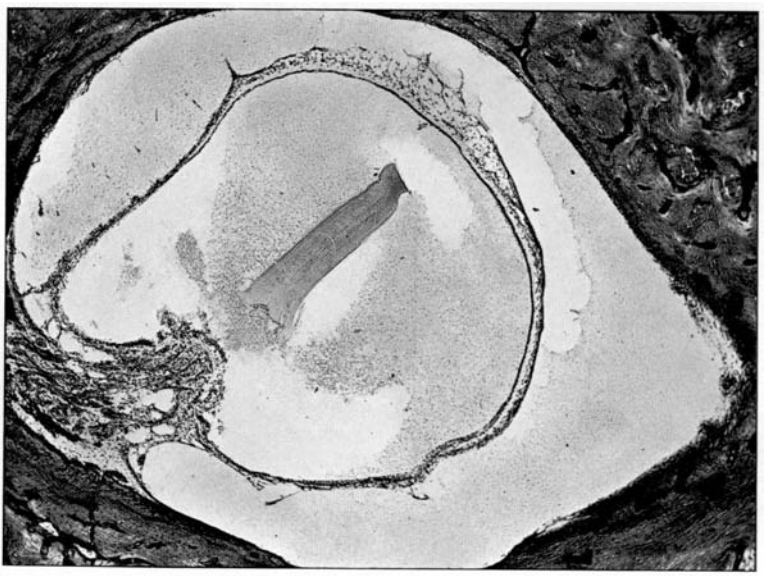


FIG. 19 (Case II, No. 183).—Left/340. Ampulla of posterior semicircular canal filled with albuminous coagulum.

https://doi.org/10.1017/S0022215100004102 Published online by Cambridge University Press

Unfortunately, owing to a slight error of orientation of the block before cutting, none of the sections passes completely in the plane of the modiolus, but I have examined the whole series in this neighbourhood, and can find very few spiral ganglion cells, although the organ of Corti is fairly well preserved. The stapes footplate is free and, as in the other bones described, there is no otosclerotic process.

As in the preceding case, the lower vestibular cavity and saccule are free, although the cochlea at this level shows invasion of the scala tympani (Fig. 17B). The region of the fossula rotunda and secondary tympanic membrane is normal.

CASE III (male, aged 60. No. 184).—The histological differentiation between acoustic neurofibroma and meningeal endothelioma is sometimes extremely difficult. In order to establish more certainly the nature of the labyrinthine tumours in the two cases described, I am also showing slides of sections from a third case—in the left temporal bone of which there is a tumour that is almost certainly an endothelioma. Sections from the right bone are also illustrated, as relatively normal, for comparison.

History.—Deafness: slow onset for two years. Staggering to left, and giddiness, for ten months. Impairment of speech, taste, and swallowing, for eight months. Headaches for five months. The onset of the deafness, two years before admission to hospital, was gradual for twelve months, by which time the patient was completely deaf in the left ear, but at this time he had no vertigo or tinnitus.

Signs.—Unable to sit or stand upright without support; falls or staggers to the left. No papillœdema; ocula movements full.

Nystagmus: Fine rotatory, on deviation to right; also on upward movement to right. Some wandering of eyeballs on looking to his left (? inattention; ? fatigue). No true coarse nystagmus. Left corneal reflex diminished. No facial weakness.

Hearing tests:

Tympanic membranes natural.

Right ear: Hearing good. From 16 d.v. to C 5 (4,096 d.v.)—Bezold tuning-forks. Rinne positive. Bone conduction, using 256 d.v., equals that of observer. Whisper heard at 2 to 3 feet.

Left ear: Questionable if tuning-fork heard at all by air conduction (with noise box). Bone conduction: ? Heard 256 d.v. Rinne negative (probably pseudo-negative). Weber to right. Loud shout at 6 inches from pinna is not heard.

I reported as follows: " The left ear is very deaf; it is doubtful

if any sounds are heard in this ear." One week later I confirmed this opinion.

Vestibular tests :

Cold caloric.—Right ear : Nystagmus to left obtained in one and a half minutes, rapid and rotatory. Diminution of spontaneous nystagmus to right ; this disappeared altogether about sixty seconds after ceasing the cold-water syringing. Vertigo present. Forced movements ; patient almost fell out of the chair. One minute later, nystagmus to left and right ; symmetrical. Five minutes later, returned to previous condition, i.e. fine nystagmus to right.

Left ear : No response. Nystagmus unchanged. No vertigo.

Rotation test.—Clockwise : Nystagmus to right almost obliterated. Definite nystagmus to left lasting five seconds only. No vertigo. Anti-clockwise : Nystagmus increased in rate and range to right ; none to left. Vertigo present.

Conclusion : The right labyrinth is normal ; the left is defunct, or nearly so.

I cut sections from the temporal bones, the left in the horizontal plane, and the right in the vertical plane. The tumour in this case is of strikingly different histological structure from that in Cases I and II (Fig. 20.) The well-defined large, oval, cellular structure of the meningioma, with relatively little fibrous support, contrasts with the whorling fibres, small cells, and palisade formation, of the neurofibromata in the other cases. The difference is also displayed in the behaviour of the meningioma, which has not passed into the labyrinth and is confined within the meatus, except in the upper part, where it was found to have spread into the marrow spaces (Fig. 21).

The stapes, vestibular cavity, and round window are free (Fig. 22), but high magnification of the cochlea, besides showing the usual deficiency in spiral ganglion cells compared with the normal—taken from the right temporal bone of the same patient (Fig. 24)—shows an alteration in staining reaction of the organ of Corti which is interesting. To appreciate this difference it is necessary to compare the organs of Corti on both sides, which have been treated exactly alike, as to the time interval after death before fixation, and the details of decalcification and staining.

Fig. 25A shows two such sections, and even in the monochrome photograph some differences are apparent. In the upper figure, taken from the left or abnormal side, there is a maintenance of normal architecture, but a loss of differentiation of

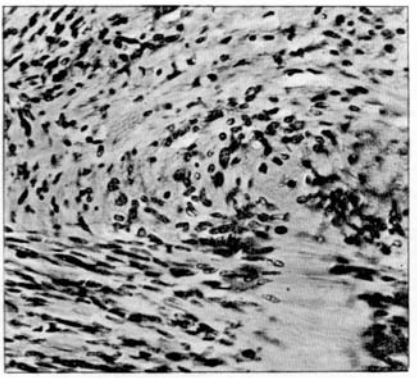


Fig. 20A. (Case I, No. 158.) Left.

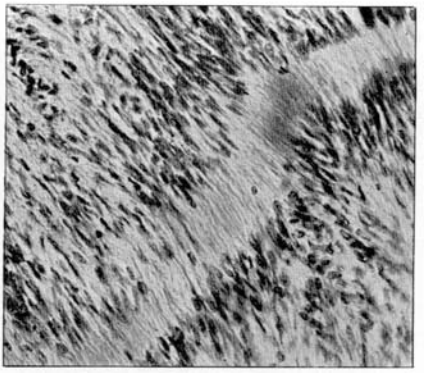


Fig. 20B. (Case I, No. 158.) Right.

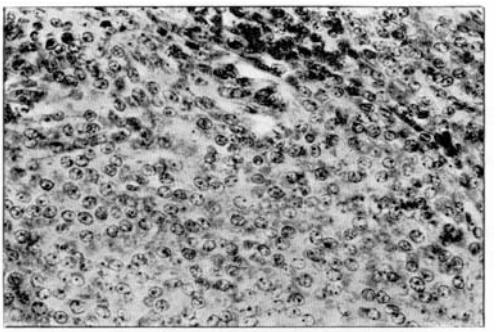


Fig. 20C. (Case III, No. 184.) Left.

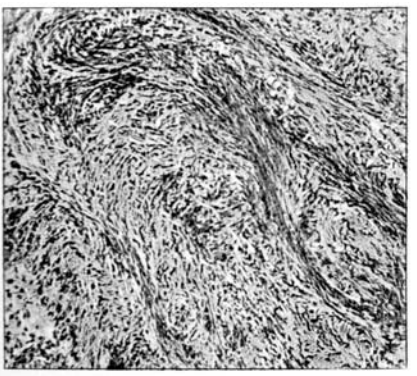


Fig. 20D. (Case II, No. 183.) Left.

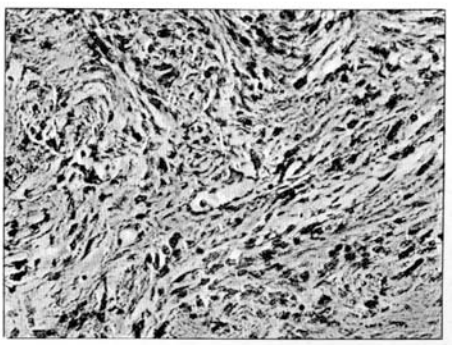


Fig. 20E. (Case II, No. 183.) Right.

Fig. 20.—Comparison of the five tumours.

[*face p.* 778

https://doi.org/10.1017/S0022215100004102 Published online by Cambridge University Press

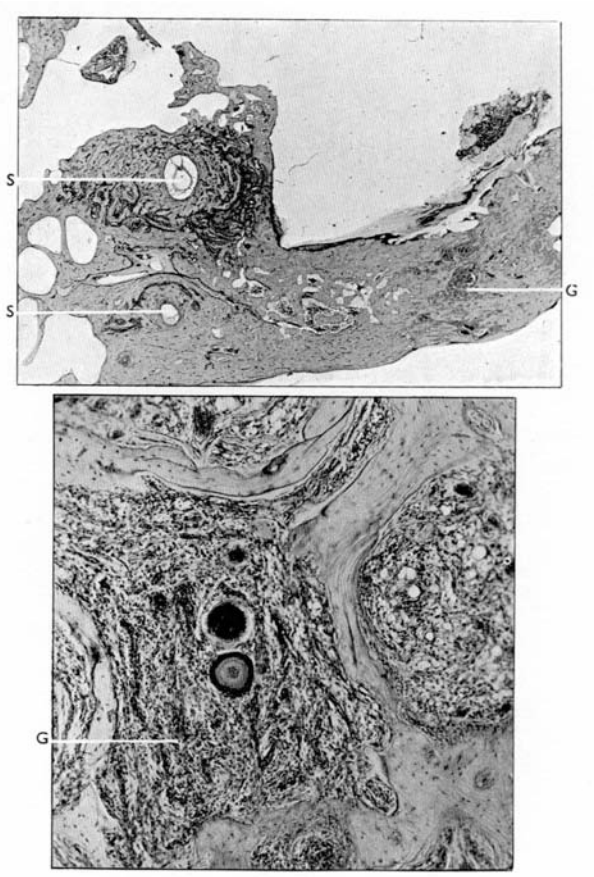


FIG. 21 (Case III, No. 184).—Left/100 : A section cutting across the superior vertical semi-circular canal, S. The apex of the petrous is invaded by endothelioma, G, seen under higher magnification in the marrow in the lower figure. S=Superior semi-circular canal. G=Growth filling marrow spaces.

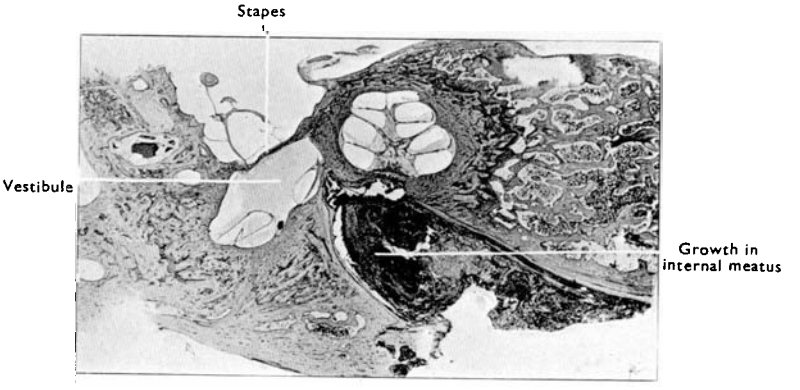
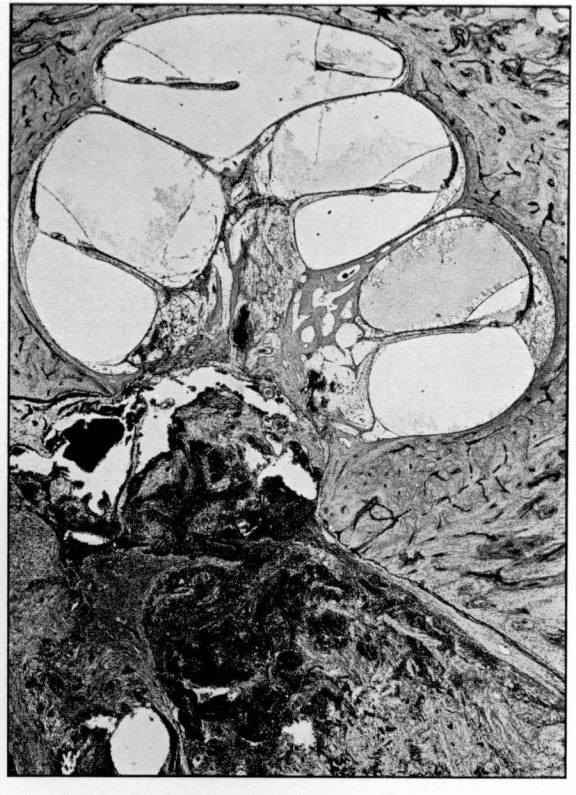
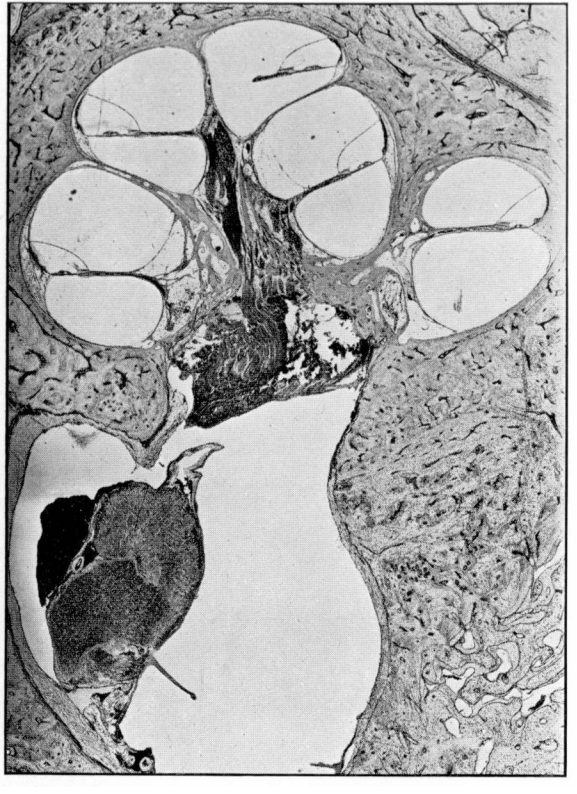


FIG. 22 (Case III, No. 184).—Left/270 : Modiolus of the cochlea ; footplate of the stapes. Vestibule contains coagulum. No spread of growth beyond the meatus. No otosclerosis.

https://doi.org/10.1017/S0022215100004102 Published online by Cambridge University Press

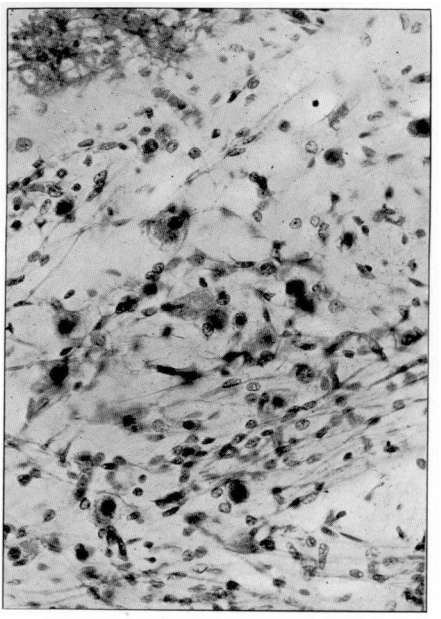


Case III, No. 184.—Left/290.

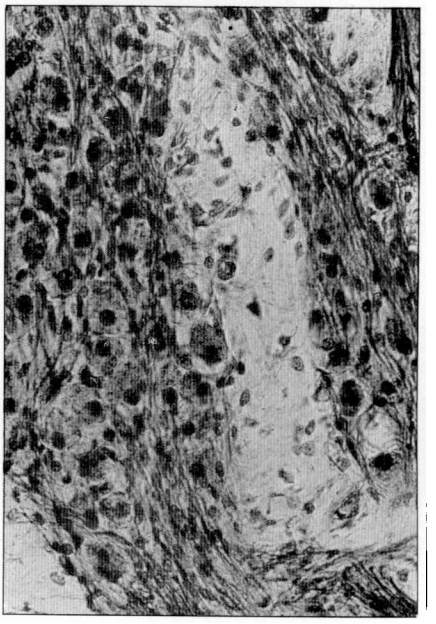


Case III, No. 184.—Right/310.

FIG. 23.—Cochlea from left and right side compared. That from the right side shows only post-mortem degeneration.



(A) Case III, No. 184, left.



(B) Case III, No. 184, right.

FIG. 24.—The spiral ganglion from the left and right sides compared. Note sparsity of cells and remnants of nerve-fibres in the left figure.

https://doi.org/10.1017/S0022215100004102 Published online by Cambridge University Press

staining, the nuclei and cytoplasm being stained darkish blue. The lower figure maintains the usual differentiation between nucleus and cytoplasm. An almost identical change is found in the stria vasculosa from these cochlea. (Fig. 25B.)

I showed these slides to Dr. C. S. Hallpike, who pointed out a similar change in a case of Ménière's syndrome at a recent meeting of the Section of Otology[6]. It is always a matter of considerable difficulty to say with certainty whether a given alteration from the normal is due to ante-mortem, or to post-mortem, degeneration.

The factor in which the two bones in Case III differ is the presence of a tumour on the left side. Dr. Hallpike's case showed a similar change without a tumour, but there was almost certainly increased intra-labyrinthine pressure, which would interfere with the blood supply to the stria vascularis and Corti's organ. In Case III it seems possible that a tumour of this size filling the meatus will have severely affected the blood supply to the same structures, while upon the right side in this case presumably only post-mortem degeneration is present.

It is difficult and unwise to draw conclusions from so little material, but it would be of practical interest if sections of the cochlea in suitable cases were examined from this point of view. A considerable step forward in the interpretation of temporal-bone pathology would be made if post-mortem degeneration could be more accurately defined.

The subject of interference with the vascular supply of the cochlea recalls the question of the causation of new bone-formation within the cochlea described by Dr. Gray in 1933,[3] and attributed by him to otosclerosis. Recent investigations by Ashcroft, Hallpike and Rawdon-Smith[7,8] make possible an alternative explanation. In these it was found that division of the internal auditory artery in the cat constantly induced the formation within the cochlea of masses of new bone the appearance of which in the published photomicrographs closely resembles that found by Dr. Gray. This, it seems likely, may have arisen in a similar manner from occlusion of the vascular supply by the tumour mass.

The temporal bones I have examined do not confirm Dr. Gray's suggestion of a direct relationship between Auditory Nerve Tumour and Oto-sclerosis.

Summary

Two cases of bilateral auditory nerve tumours are described, and compared with one case of unilateral endothelioma.

In Case I deafness in the right ear was apparently not complete, although the cochlea was invaded by growth. This growth, as also the left-sided tumour in Case II, would never have been suspected if serial sections had not been cut from both bones.

It is possible that the statistics quoting the proportion of bilateral to unilateral growths are misleading, unless both bones from a suspected case have been examined histologically. I have so far examined temporal bones from ten patients with VIIIth nerve tumour, but in only three cases have I obtained both bones. Of these two were the cases of bilateral growth described in the above report.

These cases have not confirmed the findings of: (1) New bone-formation or otosclerosis. (2) Vacuolization of the stria vascularis.

Degeneration of the spiral ganglion cells is noted.

A possible explanation of the new bone-formation in the cochlea described by another observer is given.

There appears to be no direct connection between otosclerosis and acoustic neuroma.

Some observations are made upon the changes in the staining reactions of Corti's organ and the stria vascularis on the side of a tumour in the internal meatus, and compared with those on the normal side.

The Pathological part of this work was carried out at the Ferens Institute of the Middlesex Hospital, which has borne a considerable part of the expense. I should like to thank Mr. F. J. Cleminson, Honorary Director of Research, for these facilities and W. Pilgrim, the Senior Technician and other members of the staff for their assistance in the preparation of the serial sections. I am indebted to Dr. Hallpike for much helpful advice and criticism.

In conclusion I also wish to express my thanks to the Medical Committee of the National Hospital, Queen Square, for permission to refer to case notes.

This paper is part of the work in which I have been engaged as the Geoffrey Duveen Student of London University.

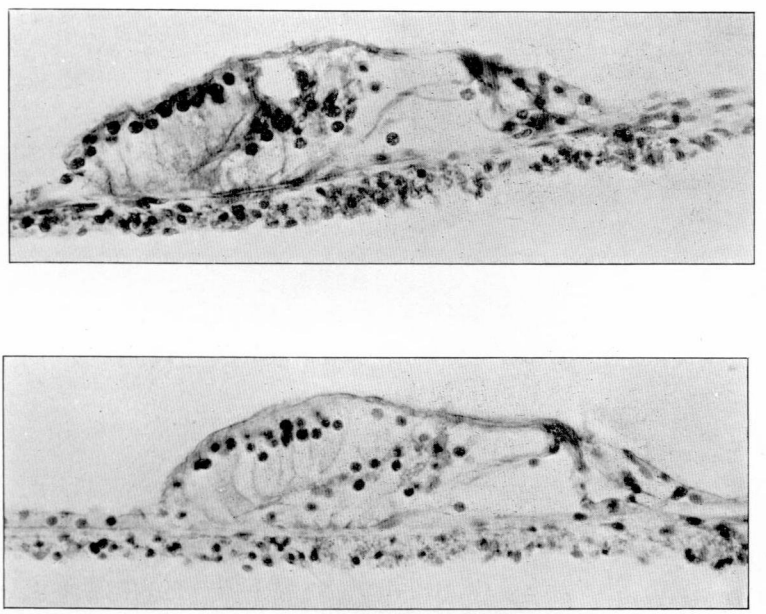


FIG. 25A.—*Above :* Corti's organ from the left side, showing loss of differential staining. *Below :* Corti's organ from the right side, showing post-mortem degeneration.

[*face p.* 780

https://doi.org/10.1017/S0022215100004102 Published online by Cambridge University Press

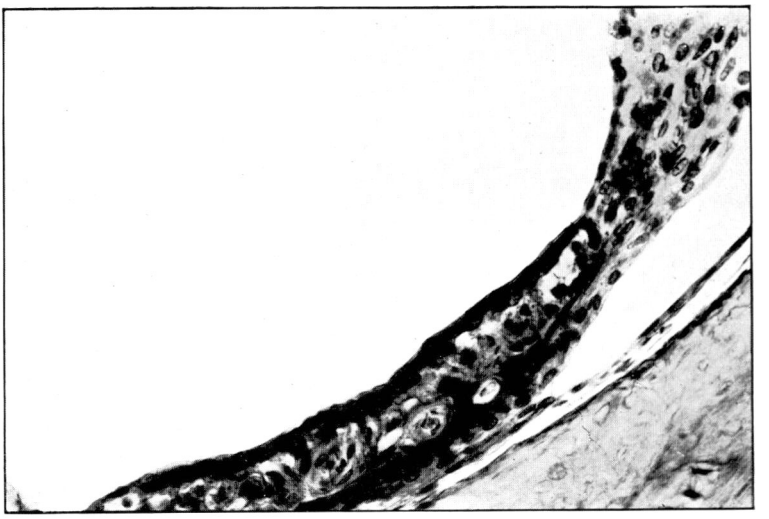


FIG. 25B (Case III).—Stria vascularis, left side. ? Ante-mortem change, showing loss of differential staining.

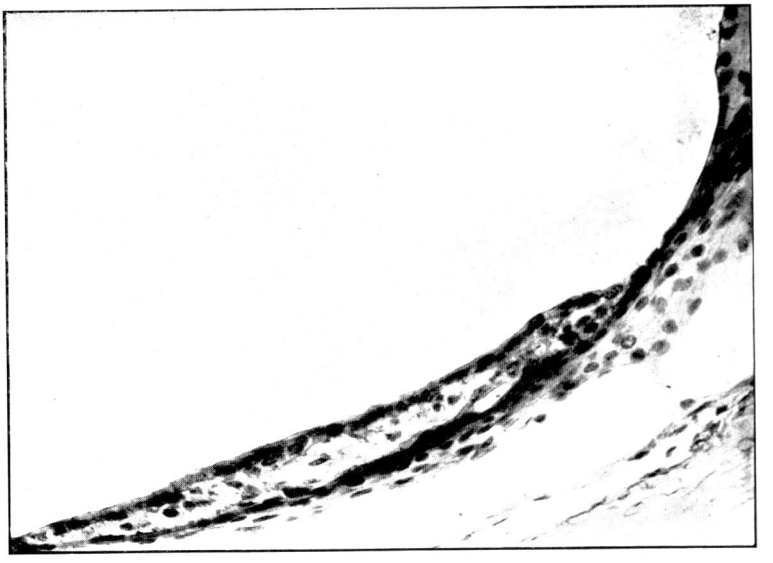


FIG. 25B (Case III).—Right. Stria vascularis, showing post-mortem degeneration.

https://doi.org/10.1017/S0022215100004102 Published online by Cambridge University Press

Auditory Nerve Tumours

REFERENCES

[1] JUST, T. H., *Proc. Roy. Soc. Med.*, 1930, xxiii, 722 (Sect. Otol., 30).

[2] FRASER, J. S., and GARDINER, W. T., *Proc. Roy. Soc. Med.*, 1930, xxiii, 726 (Sect. Otol., 34).

[3] DE KLEJN, A., and GRAY, ALBERT, *Proc. Roy. Soc. Med.*, 1932, xxv, 1273 (Sect. Otol., 85).

[4] ORMEROD, F. C., *Proc. Roy. Soc. Med.*, 1933, xxvi, 566 (Sect. Otol., 18).

[5] GRAY, ALBERT, *Proc. Roy. Soc. Med.*, 1933, xxvi, 1310 (Sect. Otol., 58).

[6] HALLPIKE, C. S., and CAIRNS, H., *Proc. Roy. Soc. Med.*, 1938, xxxi, 1317 (Sect. Otol., 55).

[7] HALLPIKE, C. S. and RAWDON-SMITH, A. F., *Journal of Physiology*, 1934, 83, 243.

[8] ASHCROFT, D. W., HALLPIKE, C. S. and RAWDON-SMITH, A. F., *Proc. Roy. Soc. B.*, 1937, 122, 186.

https://doi.org/10.1017/S0022215100004102 Published online by Cambridge University Press