SOCIETIES' PROCEEDINGS

ROYAL SOCIETY OF MEDICINE—SECTION OF OTOLOGY.

March 4, 1932.

President—Mr. NORMAN PATTERSON, F.R.C.S.

Notes on a Case of Acusticus Tumour in which both Auditory Nerves were involved by Separate Growths—ADRIAN DE KLEJN, Utrecht, and ALBERT A. GRAY, London.—The paper is published in the *Journal of Larnygology and Otology*, Vol. xlvii., p. 589, September 1932.

DISCUSSION.

Dr. A. DE KLEJN: "It must be remembered that in the condition described the whole central nervous system may be involved in the tumour formation. In another case of von Recklinghausen's disease the patient came into the neurological clinic deaf and blind, and with symptoms due to lesions in the medulla oblongata. An X-ray examination of the skull was made, and showed no typical disturbances. In that case no skiagrams were taken of the petrous bone. At the post-mortem examination two large acoustic tumours were found. In all cases of central nerve deafness skiagrams should be taken, not only of the whole skull, but also—and especially—of the petrous bone."

Dr. ALBERT A. GRAY: "An interesting point is the extension of the growth into the middle ear; this has not been recorded before. The case also confirms Wittmaack's experiments on the degeneration of the cochlear nerve with loss of ganglion cells. When acusticus tumour occurs as part of von Recklinghausen's disease, the tumours are relatively frequently bilateral. Apart from von Recklinghausen's disease the tumour is practically never bilateral.

"Another interesting feature is the curious deposit of bone in the upper whorl of the cochlea. It looks very much like the newly formed bone of otosclerosis."

THE PRESIDENT said that, with regard to the bilateral tumours in von Recklinghausen's disease, in Henschen's statistics he recorded 245 unilateral tumours, 24 bilateral; Harvey Cushing, however, had remarked that bilateral tumours were more likely to be recorded than unilateral ones, and the proportion that authority gave was about 1 in 100.

He asked whether there was any difference in the involvement of the temporal bone in the bilateral as opposed to the unilateral cases, because in an advanced case it would be impossible to remove the tumour surgically, unless the whole petrous bone was included in the operation. In some cases Cushing apparently carried out an intracapsular enucleation, making no attempt to remove the whole of the tumour. A second operation might be necessary.

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SIR CHARLES BALLANCE said that the first case of auditory nerve tumour that he had seen was in 1887, under the care of Dr. Seymour Sharkey, in the wards of St. Thomas's Hospital, where he (the speaker) had then been aural surgeon for two years.

The patient was a man, aged 42, whom he (Sir Charles) was asked to see on account of headache and optic neuritis. The optic neuritis was bilateral, and the patient had been suffering for over a year from tinnitus and slight deafness. He also had attacks of giddiness, and some loss of hearing on the left side. He (Sir Charles) had diagnosed the case as one of tumour of the auditory nerve, but his diagnosis had been ridiculed. The headache and optic neuritis increased and there was partial loss of vision. The patient also had vomiting and other symptoms and died about six months after admission to hospital. An autopsy was performed and the brain was removed. The late Professor Shattock made a beautiful drawing of the tumour, published in the *Transactions of the Pathological Society*. Since that time he (Sir Charles) had seen a considerable number of such cases, in all of which the tumour had been one-sided. The President's remark that tumours occurred on both sides in only 1 in 100 cases was probably correct; but there was no reason why they should not occur on both sides in von Recklinghausen's disease.

A unilateral tumour in an early stage could be easily and successfully removed by free exposure and by the use of the Bovey-Cushing instrument which cut the tissues without burning them, coagulated them, and prevented hæmorrhage.

Mr. SYDNEY SCOTT asked whether at any stage the patient gave a response to the galvanic test. Also, was there any diminution of the corneal reflex? And was the seventh nerve involved? He might remind Dr. Gray of a section of the labyrinth which he (the speaker) had shown at a previous meeting which bore out Dr. Gray's findings, an auditory nerve tumour having invaded the labyrinth.

Mr. SOMERVILLE HASTINGS said that the organ of Corti was destroyed in the lower coils of the cochlea rather than the upper coils, and asked whether that was usual in cases of pressure on, or destruction of, the auditory nerve. Had Wittmaack recorded this as the result of his experiments?

Dr. GRAY (in reply) said that in consequence of the erosion of the temporal bone it would have been impossible to get the tumour away completely. It was not Wittmaack who discovered the degeneration of the organ of Corti in acusticus tumour cases, but Panse. In some cases the organ of Corti underwent complete degeneration throughout the cochlea, and in others it remained undamaged. But, as Mr. Somerville Hastings pointed out, the curious feature about this case was that the organ of Corti disappeared in the lowest wall, not the upper. If the patient had lived longer the upper part might also have disappeared.

Pathological Changes in the Auditory Nerve in Otosclerosis and their significance clinically, especially with regard to Paracusis Willisii—ALBERT A. GRAY, London.—The paper is published in the *Journal of Laryngology and Otology*, Vol. xlvii., p. 598, September 1932.

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DISCUSSION.

Sir JAMES DUNDAS-GRANT suggested that many cases of paracusis might be due not to the action of the noise on the patient, but to the raising of the voice on the part of the person to whom the deaf person was listening. On one occasion he had had a young woman patient suffering from paracusis and in an advanced stage of otosclerosis, but without adhesions. When he put the noise machine on the patient's head she heard worse, but when he put it on her mother's head and she (the mother) began to speak, the girl said, "I hear you; you are speaking through a telephone." When the accompanying person and the patient were together, the noise, usually of a deep pitch, deafened the person with good hearing, while not intensifying the deafness of the patient who was deaf mainly to the deep tones as in otosclerosis. That was the explanation of many cases of paracusis. A number of years ago a French writer on the subject said that the lesion which was at the bottom of otosclerosis was in the medulla lying between the nucleus of the 5th and that of the 8th nerve.

Mr. SOMERVILLE HASTINGS said that he had been particularly interested in Dr. Gray's ideas on paracusis. The explanation suggested was the only intelligible one which he (the speaker) had heard. Reference had been made to the work which Gordon Scarff and himself had done in 1926, using Tucker's apparatus, whereby an oscillating wireless valve produced a pure note. One of 520 vibrations was used for the most part, but it could be varied. They did not use a tuning-fork as the source of sound; as the disturbing sound they used an unclamped or unweighted tuning-fork of 72 vibrations. This sound could be put into the same ear as the test sound or into the opposite ear. On their telephones were thick pads of felt so that when, for instance, the disturbing sound was turned on to the left ear and the right was being tested, it was impossible for the vibrations to have been carried through the skull and so have shaken up the ossicles on the right side, as predicated by the ordinary explanation of paracusis. The disturbing sound was tried on the opposite ear, as a sort of fool's experiment, and in 12 cases they had found that there was definite improvement in the hearing of the one ear by making a noise in the other ear. That was done many times, and in some cases marked improvement was established. In one case the hearing capacity in the right ear was doubled by the presence of a disturbing sound in the left ear. They recorded the facts, but did not suggest any explanation, and what Dr. Gray now said explained matters.

By means of such accurate records of otosclerotic cases a knowledge of this disease ought to be built up. It was now time that this Section adopted some definite classification — based, it might be, on clinical symptoms—of otosclerosis, so that when otosclerosis was spoken of the speaker and the hearer might have in mind a definite picture of what was meant.

Mr. E. WATSON-WILLIAMS asked whether these were definitely cases of otosclerosis? The characteristic feature of otosclerosis was ear deafness, whereas one would expect a patient with such extensive degeneration of the nerve as that shown to have the signs of nerve deafness. Did these patients suffer from paracusis? *A priori*, one would expect that if nerve impulses overflowed from one nerve fibre to another, say from a fibre conducting

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a continuous noise-sound into one conducting conversation-sound, it would be more difficult, rather than less so, to distinguish the finer modulations on which depended the appreciation of speech. He would like to ask whether there was any regional distribution of the degeneration in the first case, for example was it central? (Dr. Gray: "Yes.") This was interesting, as he had often observed that the loss of hearing in otosclerosis was absolute for very low tones, that is, it was a nerve deafness.

Mr. A. LOWNDES YATES said that Dr. Gray had placed an entirely new view of paracusis before the Section, and so brought into line what had previously been divergent views. There was the possibility in many cases of a false paracusis, *i.e.*, the patient did not hear the noise. There was also a type which could be called a "flush" paracusis. Any noise caused congestion of the vessels of the tympanum; one did not know if it also caused congestion of the vessels of the internal ear, though this was possible.

If a patient heard better in a noise and had true paracusis, that capacity to hear better continued after the noise had ceased for a varying time, which might be short or might be much prolonged. If one placed a strongly sounding apparatus near an ear which was the seat of paracusis, one could experimentally cause such a patient to feel that he was hearing better. Such improvement lasted for a time, and then the hearing relapsed to its former standard. Many reports of benefit to hearing from what were in effect noise machines, were due apparently to this temporary improvement—the result of "flush" paracusis. Flush paracusis appeared, however, to be present in other conditions besides otosclerosis. In the case under discussion the diagnosis of otosclerosis was established by microscopy.

During life a certain number of cases which were clinically similar showed signs of former inflammatory changes in the middle ear. Was it not possible that if there was a lesion of the membranous periotic capsule, a slight inflammatory change might cause a good deal of mischief in the region of the round window or the oval window, leading to the type of deafness known clinically as otosclerosis? There must be many conditions of deafness clinically classified as otosclerosis in which one had no proof that there was any lesion of the bony periotic capsule until the patient died and the ear had been submitted to microscopic examination. In some of these, nerve changes might not yet have produced the lesions of the bony periotic capsule. Dr. Gray's observation formed a definite and important addition to knowledge of the ætiology of otosclerosis.

Dr. ALBERT GRAY (in reply) said that Sir James Dundas-Grant was right when he referred to the raising of the voice in order to be heard. Some were not true cases of paracusis at all; other sounds did not disturb them so much as they disturbed normal people. But in cases such as those mentioned by Mr. Somerville Hastings, and most others in which that factor did not enter, it seemed that there was a relative increase.

Otosclerosis was a manifestation of changes in which there was a certain general uniformity in the clinical and pathological pictures. But cases of otosclerosis differed from each other a good deal in minor details, and these differences depended on the extent to which the symptoms were due

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on the one hand to changes in the capsule of the labyrinth with fixation of that part, and on the other hand to changes in the nerve. All the changes, he contended, were degenerative and never inflammatory. If the results affected the oval window the ordinary otosclerotic type ensued, but in other cases types were found which were like the nerve type which Manassé described. In the latter case the patient was examined clinically, and after death the bones were examined. At the examination fixation of the stapes on one side was found, while on the other it was a pure nerve deafness. A common but unknown origin was therefore present. He (the speaker) considered that the cause was related to the nutrition of the tissues.

With regard to Mr. Watson-Williams' remarks, the nerve impulses were added to by the disturbing noise. He reminded Mr. Watson-Williams that the axis-cylinders were still functioning, and if the disturbing noise was a loud one the nerve-current was at a higher tension, and the axis-cylinder which had the lower tension would receive nerve impulses from that of higher tension. The changes first appeared in the central part of the nerve. The fibres from the upper part of the cochlea came down in the core of the modiolus, and the other fibres were wound round like cotton on a spool.

In answer to Mr. Lowndes Yates' remark as to paracusis lasting after the sound was removed, he (Dr. Gray) thought that was due to the stimulating effect of the vibrations, such as occurred in the use of Noebel's apparatus. Active congestion in the vessels of the labyrinth certainly helped the hearing, and one was familiar with the effect of nitrite of amyl. He did not see how changes in the oval window and round window would affect the paracusis.

Cases.

Two Cases of Endothelioma of the Middle Ear.—Lionel Colledge, F.R.C.S.

(1) A man, aged 43, was admitted to St. George's Hospital on 9th March 1931, under the care of Mr. Ivor Back. Below the right ear was a tender swelling, which was incised by the house-surgeon and pus was evacuated. Examination later showed a foul purulent discharge from the meatus and granulations obscuring all view of the tympanic membrane. A probe was passed from the opening in the neck upwards through the floor of the meatus. There was deafness of the middle-ear type. The patient did not wish anything further done and was discharged. He was re-admitted on 11th October 1931, in exactly the same condition. A radical mastoid operation was performed on 14th October. The granulation tissue appeared to be of an unusual consistence, tough and gritty, so was kept for examination and the post-aural wound left wide open. The tissue appeared also to have invaded the inner end of the skin of the meatus, and the skin in this region was excised. Dr. John Taylor reported that the tissue was microscopically myxo-endothelioma and identical with a parotid

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tumour. On 16th November, in view of the report, the cavity was widened still further, curetted, and covered with a Thiersch graft. No communication with the parotid gland was found. There has been no sign of further growth, and at no time has there been any facial palsy, nor has there been any evidence of a parotid tumour.

(2) Male, aged 66, came to St. George's Hospital complaining of a painless discharge from the right ear of uncertain duration. The meatus was occupied by a large aural polyp bathed in pus. There was deafness of the middle-ear type.

10*th February* 1932.—Radical operation: some of the mastoid cells contained only mucus, but the ossicles were necrosed. The wound was left wide open behind. The operation has been followed by a partial facial palsy. The polyp and granulation tissue were sent for examination and Dr. John Taylor reported endothelioma. The section exhibited shows a typical endothelioma.

Auditory Nerve Tumour.—T. B. JOBSON.

Mrs. E. G., aged 63, domestic servant. Came to hospital on 17th February 1932. Deafness in right ear noticed twenty years ago. Very marked for three years, now total. Facial paralysis (right) one year. Tinnitus: severe, continuous. Vertigo: daily attacks, falling forwards, no loss of consciousness. Nystagmus: slight spontaneous. Right frontal headache. No vomiting. Pupils equal and react to light. Eye movements normal. Optic discs show ? some blurring of E. and N. edges on right. Knee-jerks normal. No Rombergism. Blood-pressure 210. Tongue and palate move freely. Tympanic membranes are mobile and slightly opaque and show no evidence of suppurative disease.

Hearing: Does not hear a shout in right ear with Bárány control. C. V. left = 2 feet. Weber lateralised to right. C. 256 heard on right mastoid but not C. 128, 64 or 32. Cold caloric test produces no response in $2\frac{1}{2}$ minutes.

A stereoscopic skiagram does not show any erosion of temporal bone. Further radiograms are being made. This case shows the very slow onset and the classical symptoms of deafness, tinnitus, vertigo, ataxia, nystagmus, dead labyrinth and paralysis of 7th and 8th nerves.

I am indebted to Dr. Stanley Whitton and my colleague, Dr. A. F. Treadgold, for permission to show this case.

Herpes Zoster Oticus, with involvement of the Vestibular Nerve.—N. ASHERSON, F.R.C.S.

Patient, a girl aged about 10, complained of severe pain in the left ear, with some tenderness over the mastoid process. The temperature was slightly raised. Five days later complete facial palsy suddenly developed; coincident with this, a serous bleb appeared on

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the tympanic membrane. This bleb rapidly disappeared, leaving the membrane normal again.

Within thirty-six hours of onset of the palsy, giddiness, ataxy and vomiting ensued. Coarse horizontal rotatory nystagmus to right; none to left (the affected ear). Pain no longer present. No pyrexia at any stage. No hyperacusis; hearing practically normal. No disturbance of sensation on side of tongue.

The case was admitted as one of acute mastoiditis. A single line of herpes developed on the face, running from the angle of the mouth towards the ear. This eruption faded two days afterwards. The labyrinthine symptoms subsided first, and the facial palsy had disappeared by the tenth day from its first appearance. The nystagmus could be elicited as before.

Six weeks later: still some slight nystagmus on looking away from the affected ear.

Specimen of a Temporal Bone, with Pus in the Mastoid Antrum, from an Infant dying from Diarrhœa and Vomiting.—N. ASHERSON, F.R.C.S.

The infant had diarrhœa and vomiting for fourteen days, and was referred for aural examination by Dr. D. W. Winnicott. Examination of the right ear showed a slightly sagging tympanic membrane which was little different from the normal membrane. Incision revealed pus. The child took its first feed for ten days without vomiting, after this. Within a few days the symptoms recurred. The other ear, on myringotomy, revealed pus also.

A Mastoid Operation performed through a Meatal Incision.—C. I. GRAHAM, F.R.C.S.

Mr. B. L. had an operation for acute mastoiditis on 3rd February 1932.

The incision consists of two limbs meeting at an obtuse angle at a point one quarter of an inch within the meatus on the posterior wall. One limb is vertical and is carried upwards through the crus helicis, the other limb is directed backwards and slightly downwards in the lower part of the cavum conchae. The incision is carried to the bone and the triangular flap is raised from the bone and retracted backwards. The operation is performed by the usual method, and the flap is allowed to cover the posterior wall of the cavity, after removal of a piece of cartilage, and is retained in place by a single suture passing from the apex of the flap to the skin in the post-auricular groove.

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