SOCIETIES' PROCEEDINGS

ROYAL SOCIETY OF MEDICINE—SECTION OF OTOLOGY

May 7, 1927.

President—DAN MCKENZIE, M.D.

THE Meeting of the Section of Otology, May 1927, was held in the North-eastern Fever Hospital (by permission of the Metropolitan Asylums Board).

The following Demonstrations were given in the wards and laboratories:—

Dr J. C. Blake: "The Schultze-Charlton Reaction in the Diagnosis of Scarlet Fever, and the Schick Test for Susceptibility to Diphtheria." Dr F. Asker: "The Dick Test for Susceptibility to Scarlet Fever." Dr Elsie J. Mann: "Radiography of the Mastoid in Children." Dr Nancy G. Howell: "Treatment by Ultra-Violet Light with special reference to Ear Disease." Dr A. J. Mair: "Diphtheria and Diphtheroid Organisms from the External Auditory Meatus."

The treatment by heliotherapy of children suffering from aural discharge during scarlet fever was demonstrated in the grounds of the hospital.

The following cases were shown:—

Three Cases illustrating Indications for Wilde's Incision.—(1) Child, aged 4, was admitted on 4th January 1927, with a mild attack of scarlet fever.

8th January.—Pain in left ear; temperature 100° F.; slight redness of drumhead; no bulging. On 9th, slight double adenitis. 10th, left drumhead bulging; paracentesis; blood-stained purulent discharge; right drumhead normal.

14th February.—Slight redness behind the left pinna; auricle displaced. On 16th, Wilde's incision; bare bone was felt. From 17th February to 21st March, double cervical adenitis; slight left otorrhœa and discharge from wound. On 23rd, wound swollen. On 29th, sinus discharging. 9th April, left ear dry; small perforation; wound healed.

The general rule in such cases is to do the mastoid operation if (*a*) the temperature has not subsided in two days, (*b*) the wound has not healed in two weeks, or (*c*) the ear is not dry in two months.

(2) Child, aged $3\frac{7}{12}$, was admitted on 19th April 1927, a late case of scarlet fever of septic type; left otorrhœa on admission; cervical

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glands enlarged and a very severely ulcerated throat. 20th, œdema above ear and tenderness over mastoid. Wilde's incision; no pus. 23rd, swelling around wound. Question of anæsthetic discussed and decided against because of condition of throat. Elevator put under periosteum in a forward position, pus found, and tube inserted.

(3) Child, aged 3, was admitted on 23rd March 1927, with rhinorrhœa, Klebs-Löffler bacillus and right otorrhœa. 15th April, swelling behind right ear before rise of temperature to 99.6° F. Wilde's incision after freezing; no pus; bone felt rough.

Necrosis of External Meatal Wall following Mastoid Operation during Scarlet Fever.—Child, aged 5, was admitted on 5th January 1927 with a moderately severe attack of scarlet fever.

On admission, right otorrhœa and tenderness over right mastoid. On 6th, swelling; Wilde's incision. On 7th, facial paralysis; mastoid opened; pus found. 10th February, small sequestrum removed; since then we have been waiting for the sequestrum of the posterior meatal wall to separate. Facial paralysis has disappeared. 20th April, some sequestra removed; others remain for removal, for which an anæsthetic will be necessary.

SUMMER MEETING.

June 10, 11, 1927.

President—DAN MCKENZIE, M.D.

A Preliminary Study of Chronic Middle-Ear Deafness—GEORGE SCOTT-WILLIAMSON, M.D., and E. HORACE RICHARDS, F.R.C.S.E.—The Paper is published in the *Journal of Laryngology and Otology*, Vol. xlii., p. 793, December 1927.

What is Progressive Middle-Ear Deafness?—DAN MCKENZIE, M.D.—The Paper is published in the *Journal of Laryngology and Otology*, Vol. xlii., p. 801, December 1927.

DISCUSSION ON PROGRESSIVE MIDDLE-EAR DEAFNESS.

Mr G. J. JENKINS, said that when he was asked to take part in this discussion he replied that he had not been able to recognise, pathologically or clinically, the condition known as "progressive middle-ear deafness," sometimes described as "chronic middle-ear deafness," or even termed "dry middle-ear catarrh": the answer was: "Say *that*"! He had not heard these terms applied to any form of middle-ear disease in the aural department under the care of Mr Arthur Cheatle at King's College Hospital, and from his own observations had come to the conclusion that the terminology was wrong and misleading.

We were all familiar with the text-book description of the disease under this name, with the detailed accounts of the active local treatment

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and general management of the patient. The pathology was in most books obviously speculative. The symptoms and signs were usually roughly and indefinitely indicated, and so far as they went they were the same as an imperfectly described otosclerosis syndrome. He believed that if a complete modern examination had been made in these cases on which this terminology had been founded, the diagnosis would have been one of otosclerosis.

In his opinion pure middle-ear changes, including fixation of the stapes, would give only pure obstruction signs, and if any symptoms such as internal ear deafness, or those especially associated with otosclerosis, were present, we should then regard the labyrinth as affected. He had not known middle-ear deafness to be progressive, in the sense implied in the title of this discussion, that could be attributed to middle-ear changes only.

From the foregoing statements it would be deduced that he had very little to say about treatment, but if there had not been that persistent description of this class of case in text-books, many patients would have been saved the effects of disappointed hopes.

Mr H. NORMAN BARNETT said that in his opinion this condition might be divided into two classes: (1) Those following long-antecedent middle-ear suppuration, or those secondary to infection from septic tonsils and to obstructed airway caused by deflection of the nasal septum; (2) those arising without these conditions being present, but almost certainly due to infection through the Eustachian tube from some other cause.

In cases following old middle-ear suppuration, the condition of things, then present, led to uncured deposits being left behind. In the other types, the same thing occurred from a different source and gradually the material formed adhesions between the ossicles and sometimes between the ossicles and the tympanic membrane. As time proceeded this material became more organised and the ossicles became more and more incapable of movement till finally no oscillation took place. This process was usually very gradual and the result was that the onset of this type of deafness might occur long before the patient was seen. The sound waves were unable to traverse the tympanum, and thus complete or almost total deafness resulted although there was no lack of perception by the nerve and no otosclerosis, though in some cases tinnitus was a marked symptom.

The treatment he (the speaker) carried out in these cases was designed with a view to causing absorption of the deposited material and breaking down the adhesions, by means of oto-massage of the tympanic membrane, ionisation over the mastoid or in the meatal passage—preferably the former—the passage of iodine, camphor and carbolic acid vapour through the Eustachian catheter to the middle ear. The duration of the treatment varied from three weeks to three months, according to the nature of the case and the state and organisation of the deposit.

The three cases to be shown to-morrow were illustrative of: (1) A cure which may be regarded as complete for all practical purposes—the patient had not been seen for two years, and he was able to perform his professional duties with ease; (2) a case now under treatment and rapidly improving so that the patient is able to do his work without any hindrance; (3) a case

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also under treatment but not so far advanced and of a slower type in responding to treatment.

In his opinion many cases of deafness were turned down and the statement made that nothing could be done, because the value of the above recorded type of treatment was not recognised and in some cases it was thought that otosclerosis was present. He believed otosclerosis to be a comparatively rare condition; many so-called cases were really those of chronic or very advanced types of progressive middle-ear deafness.

Combined with the above treatment a most important factor was the re-education of the hearing centre, for after all it must be remembered that we, so to speak, heard with our brain rather than with our ears, which were a conducting and selecting apparatus. In the deaf, especially those who had been so for a long period, the centre became dormant owing to the fact that sounds had not been reaching it; and even when, after treatment had been begun, the sounds began again to travel through the middle ear and were conducted by the auditory nerve to the centre, there was little or no response and the patient had to be warned to respond to any stimulus of sound that they heard, whether they were wrong in its interpretation or not. Some persons, according to their temperament, were much more difficult to re-educate than others. The best re-educative medium was the patient's own voice, conveyed to the centre through a long speaking-tube.

Dr J. S. FRASER said that in spite of the papers which had been contributed, the subject was still enveloped in a fog. He found considerable difficulty in knowing what kind of case Dr Scott-Williamson had discussed. Dr Williamson spoke of "idiopathic" deafness—a term often employed when little was known about a subject—and added that he had excluded cases of congenital deafness. If Dr Williamson referred to otosclerosis, the proportion of cases in which the deafness was inherited was over 50 per cent. Therefore he must have excluded many cases of otosclerosis from his statistics, and yet he spoke about the facies of the patients, etc. It was in regard to otosclerosis that some had said—he thought with truth—that one could almost "spot" the case when the patient entered the room. It would have been better if Dr Williamson had had his cases classified by an otologist before he examined them.

He (Dr Fraser) said he thought the best classification of these cases of chronic deafness was the following: (1) Those with a normal, and (2) those with an abnormal, tympanic membrane. If one had to deal with chronic deafness in which the patient had a normal drumhead and yet gave the usual tuning-fork results of obstructive deafness, one could be sure it was otosclerosis, whether or not there was a flamingo tinge in the promontory. If the latter was present it only clinched the diagnosis. Further, he wondered whether a considerable number of cases which gave the tuning-fork tests of nerve deafness, in young or middle-aged people, were not really instances of otosclerosis. Shambaugh of Chicago pointed out that many cases of nerve-deafness really had "atypical otosclerosis." These patients had a well-marked family history of deafness, but, on functional examination, one found that bone conduction was diminished and Rinné's test was positive. Shambaugh stated that some of these cases had been confirmed by microscopic examination after death.

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Shambaugh's view was that in these cases the "osteitis vasculosa" affected part of the cochlear capsule but did not involve the region of the oval window. If the latter were affected, one got the test results which one expected in otosclerosis.

He (Dr Fraser) agreed with Mr Jenkins that in most cases with well-marked abnormality of the drumheads the deafness was not progressive. These people had suffered from otitis media in childhood, and otoscopy showed the presence of retracted scars in the drumhead, chalk patches, etc. Yet these patients, as a rule, heard very well until they reached the age when the changes characteristic of senile nerve deafness might be expected. When there was a really abnormal drumhead the recognition of otosclerosis was more difficult than when the tympanic membrane was normal, but if tinnitus was a marked feature in the case, if there was a hereditary history of deafness, if the catheter gave no improvement in hearing and if the deafness was progressive, one was justified in regarding the case as one of secondary otosclerosis.

He (the speaker) was surprised at the President's statistics, which gave a high proportion of chronic catarrhal otitis media in comparison with otosclerosis. In most of the books now being published, chronic catarrhal otitis media was receding into the background, while otosclerosis was markedly advancing. His own view as to the proportion of the two affections would be very different from that shown by the President's figures.

Sir JAMES DUNDAS-GRANT said he could not produce the material which he thought ought to be brought before the Section, namely, *post-mortem* specimens to illustrate the actual condition found in these cases. He still looked to some of the younger workers in the specialty to produce this kind of material, which was much wanted.

Many large and small questions arose for discussion. For instance, what was the influence of pure obstruction of the Eustachian tube? His (the speaker's) view was that many cases of chronic progressive middle-ear deafness were secondary to the narrowing of the walls of the Eustachian tube. He was inspired by a case shown by Mr Tilley and by one he had seen himself, to quote a paper of Krainz's on that subject.* The case recorded was one in which, from compression of the Eustachian tube by a sarcoma in the nasopharynx, an exudative serous catarrh and thickening of the lining membrane of the mastoid cells had followed, apparently as the result of the mechanical obstruction. Every effort should therefore be made to improve the condition and patency of the Eustachian tube. He did not think pure cases of otosclerosis were common in comparison with those of chronic catarrhal change.

With regard to bone conduction, it must be remembered that a negative Rinné might be due to unilateral nerve deafness, as when testing bone conduction on one side, it was impossible to exclude stimulation of the nerve on the opposite side. In many cases of obstruction by cerumen there was not the increased bone conduction which was present in simple Eustachian catarrh—one reason being that there was no increased tension in the middle ear.

* *Journ. Larnyg. and Otol.*, vol. xli., p. 732, November 1926.

https://doi.org/10.1017/S0022215100031327 Published online by Cambridge University Press

Professor Manasse had written an important paper in which he said that many cases regarded as chronic progressive deafness were due to changes in the cochlea. The difficulties met with in analysing these were sometimes enormous.

Mr SOMERVILLE HASTINGS said he would briefly discuss the question of auditory intelligence. Some years ago he and Major Tucker had brought before the Section an apparatus for testing patients' hearing power—an audiometer. He (the speaker) had used it for several years but had not brought his results before the Section, as they were so indefinite; the question of auditory intelligence entered so much into the problem. He agreed that there was need of research on this question.

He also was struck by the proportions given by the President between the chronic catarrhal and otosclerotic types of chronic deafness. He (the speaker) was a heretic in this matter, as, in his experience, otosclerosis was very rare. He could only remember two cases in which with the ordinary signs of middle-ear deafness there was a normal drum-head, nose and nasopharynx, and nothing to suggest a history of infection in these regions. He did not doubt the accuracy of the description of the pathological changes, and admitted that the cases ordinarily called otosclerosis might run in families. But he suggested that they nearly all began with sepsis in some region of the nose and throat at some period of life.

It was of interest that Dr Scott-Williamson was able to date the onset of these troubles, on the average, from about the time of puberty. His own view was that in many diseases it began in quite early life. Those who examined the ears of young children found otitis media was much commoner than they had suspected, and, in a large proportion, changes in the ears could be found. It was very difficult to distinguish the cases in which the process persisted, whatever was done, from those in which it could be arrested. He thought the right course was to make a very careful examination for a possible source of sepsis, and to treat it when found. It would be found present in a sufficient number of cases to render this course well worth while.

Mr J. F. O'MALLEY said that it was only natural there should be differences of opinion among members on such a subject. If Politzer's text-book, published twenty-five years ago, was consulted on the subject of otosclerosis, a reference would be found to the Bezold triad: (*a*) A normal-looking tympanic membrane, with free Eustachian tube, (*b*) a middle-ear type of deafness, and (*c*) negative Rinné. Cases of that type were still encountered, and they showed a certain familial tendency, *i.e.*, apparently there was a hereditary factor. Most otologists would probably classify them as otosclerosis.

That there was a definite middle-ear catarrhal trouble which led to deafness must also be conceded, because patients complained of deafness following a cold, and after treatment, when the cold passed away, the hearing was restored. These were two distinct types. In the former there could also be superadded a middle-ear catarrhal infection. If this did not clear up and an otosclerotic change was progressing simultaneously in the labyrinth, the resultant clinical picture differed from that which either

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condition separately would present. Shambaugh, in a recent communication, stated that one-third of the cases coming to his clinic on account of deafness had otosclerosis. He (Dr Shambaugh) thought otosclerosis was now more common than it had hitherto been the custom to regard it, and that was his (Mr O'Malley's) view also.

Like Sir James Dundas-Grant, he had hoped to hear some encouraging contribution to this subject, and he still trusted that as a result of the discussion help would be forthcoming in elucidation of this difficult problem.

Mr J. A. GIBB said that if a patient who was practically stone-deaf, except to bone conduction, was willing to have anything done which would improve his hearing, a hole made in the postero-inferior quadrant of the drumhead would enable him to hear fairly well. How could that be explained? The case on examination was clearly otosclerosis, yet on perforation of the drumhead opposite the oval window hearing was obtained. He had such a case, and the patient now heard as he had not been able to hear for the past eight years. It should be borne in mind that in primary otosclerosis there was an osteoporosis of the round window (an extreme case).

Mr BROUGHTON BARNES said that he was surprised to hear Mr Jenkins deny the existence of progressive catarrhal middle-ear deafness. One could not do anything for the otosclerotic, whereas if the condition was catarrhal one could hope to arrest the process, except possibly for the underlying progressive factor referred to by the President. He did not think Mr Jenkins would say that a case with thickened drumheads was one of otosclerosis, yet surely these were cases of progressive middle-ear deafness. There were cases in which the drumheads were greatly thickened, often they were retracted and rather dull. Sometimes in the better ear the postero-superior angle had escaped the thickening, and there might be some mobility ; on inflation the drumheads returned to the position in which they were before. Should that be called otosclerosis? A year ago he had had a case which he thought was otosclerosis, the patient, a woman, being aged 23. She had a thin, bright drumhead, of normal mobility, and he could see a pink glow. She had marked middle-ear deafness, and there was a history of family deafness. In this patient there had been three years of progressively increasing deafness. She had a cold, and he gave a nose wash, and on coming back a month later she could hear perfectly. That increased his caution in diagnosing otosclerosis. As an incurable condition, it should only be diagnosed when tympanic causes had been excluded. He asked how the last speaker (Mr Gibb) kept open the perforation mentioned by him.

Mr HERBERT TILLEY remarked that the President spoke of tinnitus being a cochlear symptom. If that was always so, how did he explain those desperate cases in which suicide was contemplated because of the perpetual noises and for which the auditory nerve had been divided within the skull, and yet the tinnitus continued? He took it that in such cases the tinnitus was of cerebral origin. Did the President contend that a primary cochlear lesion had set up the central change which persisted after the operation?

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Mr A. R. TWEEDIE said that, like Dr Fraser, he could not quite understand the character of the cases described by Dr Scott-Williamson and Mr Richards. On the one hand, some of them suggested otosclerosis, as the patients had heard better when their general health improved—possibly a peculiar characteristic of otosclerosis. On the other hand, fifteen of the cases were definitely associated with a narrow face and ill-developed alæ nasi, which intimated that here there was a definite intranasal reason for the deafness. He suggested that with a detailed description of the objective examination and results of the functional tests, the value of Dr Scott-Williamson's paper would be much enhanced.

He agreed with Mr Jenkins' remarks and objected to the term "catarrh"—urging that it would be of assistance both in diagnosis and treatment if this terminology were dropped. If catarrh meant anything, it was only a symptom and could only indicate an excess of the normal secretion. If this description were acceptable, it was obviously quite impossible to talk of "dry catarrh." He did not think there was really any such condition as progressive middle-ear deafness (that is, quite apart from any question of otosclerosis), but he considered that such cases really represented some old past lesion, combined with the normal depreciation of hearing in accordance with years. The latter was accentuated and came into evidence sooner because of the past middle-ear lesion.

He would also lay stress on the extreme importance of the functional integrity of the inner tympanic wall, to which allusion had been made by previous speakers—whilst, as regards the tympanic membrane, so long as it was normally mobile, he did not consider its opacity constituted any rcognisable factor in loss of hearing, and certainly it was unscientific to describe such opacity as "thickening"—a description which presupposed measurement.

He was very glad that this discussion had been introduced, and hoped that it would lead to the establishment of some standard method of examination and standard test, by means of which our observations could be better compared and studied.

Mr C. HAMBLEN THOMAS said he was anxious to find out, as a result of this meeting, whether a patient who was progressively getting deaf was becoming so because of catarrh, or whether the deafness was due to some bony change. If there had been some damage to the middle ear through catarrh, an early form of inflammation, it might proceed to cause greater damage later, through the fibrous tissue formed. Might not there be such a change in advancing years? And if an ear was damaged, might it not share in any general lowering of the bodily resistance and become more deaf in consequence? Did the otosclerosis mentioned as the cause of increasing deafness by previous speakers, primarily start in the bone, or might any bony changes be secondary to some affection of the mucous lining of the middle ear?

Mr C. A. S. RIDOUT said he felt that at the root of all these troubles there was some septic infection of the throat or nose at some time in life. And the personal factor came in: in some patients it progressed, in others it stood still. Once he (the speaker) had had severe suppurative

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rhinitis, and during the illness his hearing diminished considerably, but did not progress. He began to suffer from tinnitus thirteen years ago, and he had it still; he did not get colds in the head, and he had no nasal trouble. Some people contracted continuous colds and the deafness progressed—whether they showed signs of otosclerosis or chronic catarrh (terms which could well be abolished). That the condition under discussion should be called by some name all were agreed upon.

Dr W. S. SYME said he had been hoping that some light would be thrown on this problem, especially on the question of treatment, but all he heard was that otosclerotic cases could be sent to the radiologist. He did not think "chronic progressive deafness" was a scientific term, any more than was the term "chronic cough." Cases should be described in terms of pathological changes. He believed there was such an entity as chronic progressive changes in the middle ear.

There was a common type of case in which there was thickening of the lining membrane of the Eustachian tube and middle ear, probably due to infection by an organism of low virulence, and the movements which took place caused a re-infection of the parts which was perpetually in progress; this resulted in a chronic adhesive process, which took years to become permanent. Adhesions formed afterwards, and there was a definite condition of the middle-ear which constituted what were called chronic middle-ear changes. He did not think such changes put out of consideration the question of otosclerosis, as an associated condition brought about possibly by the nervous tension under which these patients suffered. The right treatment was to discover the origin of the infection. He believed it was always in the nose and nasopharynx.

He did not think very much reliance could be placed on the immediate effect of catheterisation in these cases. If there were immediate results from that, so much the better, but he would not regard a case as outside the possibility of improvement because catheterisation did not at once make the hearing better. When there were changes in the nose and nasopharynx, they should be treated.

Dr SCOTT-WILLIAMSON (in reply) said he was not responsible for the nature of the cases which were sent to him. Fifty out of the fifty-one cases presented a picture which made it plain to him that the cases were of a type, and he congratulated the clinicians at the Central London Ear Hospital on having clearly defined a condition of progressive middle-ear deafness.

Mr G. J. JENKINS (in reply) said that nothing had been said in the discussion to cause him to alter the statement he had made in the opening remarks. He could not recognise a distinct clinical entity "Progressive Middle-Ear Deafness." A middle-ear infection might progress to a cicatricial condition and the clinical symptoms and signs would be the result of this effect only, and could not, in his opinion, be progressive middle-ear deafness.

Otosclerosis was sometimes found concurrent with mild infection of the middle-ear tract, or even with chronic suppurative middle-ear disease, but this association did not necessarily imply a causative relationship.

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Dr DAN MCKENZIE (President) said he supposed Mr Jenkins meant by otosclerosis, bone changes. He (the speaker) did not feel convinced, however, that the change was in the bone. There was a change beyond the catarrhal or suppurative process when a new element was introduced, and then the deafness progressed independently. But he did not think one was in a position to call that new change otosclerosis, meaning thereby insular osteoporosis of the labyrinth capsule.

The terminology had been criticised, but those in the specialty knew what was meant. The feeling of despair at not doing anything for otosclerosis should not be encouraged. To-day's discussion would have been amply justified if it led to research along effective lines.

Demonstration of Radiograms of the Normal and Abnormal Labyrinth—H. GRAHAM HODGSON, M.B., B.S.—The Paper will be published in the *Journal of Laryngology and Otology*, vol. xliii., 1928.

The Saccus Endolymphaticus and an Operation for Draining the same for the Relief of Vertigo—Professor GEORGES PORTMANN.—The Paper is published in the *Journal of Laryngology and Otology*, vol. xlii., p. 809, December 1927.

The Formation of a Circumscribed Interdural Abscess at the Site of the Saccus Endolymphaticus—JOBSON HORNE, M.D.—The Paper is published in the *Journal of Laryngology and Otology*, vol. xlii., p. 818, December 1927.

DISCUSSION ON PAPERS BY PROFESSOR GEORGES PORTMANN AND DR JOBSON HORNE.

Dr DAN MCKENZIE (President) commended the beauty and graphic clearness of the demonstration Dr Portmann had given, and he thanked that gentleman, on behalf of the Section, for the admirable contribution. Many members of the Section would look back on this meeting as marking a distinct advance in regard to the possibilities of aural surgery generally. He had been wondering whether the method might not be adopted experimentally, not only in vertigo, but perhaps in some cases of nerve deafness.

Sir CHARLES BALLANCE said that Professor Portmann's demonstration was based on just that kind of investigation to which great scientific problems lent themselves, and which, when carried out in this way, generally led to some practical result. Without doubt the anatomical and physiological investigations of Professor Portmann had borne fruit in the discovery and description of an operation which might prove of the greatest service to mankind.

Fortunately the region in which this remedial operation had to be performed was well known to surgeons and otologists. The region, the

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outer part of the posterior surface of the petrous, had been often exposed by many ; he had often done so for the purpose of draining an abscess on the posterior surface of the petrous bone. Only a few months ago he had watched the President perform a splendid operation in a condition in which the saccus endolymphaticus was involved, and he watched the procedure with the greatest interest, because Dr McKenzie followed, through the petrous, a track of disease through very hard bone, leading on to the posterior surface of the petrous bone in the region of the saccus endolymphaticus.

The whole address had been of extreme interest, as it opened with researches upon the lower animals, presenting a continuous picture from the Selachiæ right up to man. That was the foundation of what could be looked forward to in the treatment of tinnitus and vertigo, and—he hoped with the President—of deafness as well.

In this operation Professor Portmann was probably on the verge of further discoveries, and he hoped that at some future date Professor Portmann would be in a position to indicate exactly the cases in which the operation could be performed.

Mr G. J. JENKINS said that the term "glaucoma" had been used by him in 1913 in this connection. He had opened the semicircular canals to relieve vertigo, but had not felt sure of his ground. Mr Mollison also had published three or four cases in which he opened the external semicircular canal for vertigo. He found accumulations of cerebrospinal fluid in front of the cerebellum in some cases of vertigo, and it had been suggested that these might be cases of dilated saccus endolymphaticus. The difficulty would be in deciding in which kind of case of vertigo the operation suggested should be done.

Mr SYDNEY SCOTT said that Professor Portmann's demonstration would encourage otologists to pay more attention to the saccus endolymphaticus.

As a practical outcome of Dr Jobson Horne's demonstration in cases of labyrinthitis, should we be content with opening and draining the labyrinth or should we also expose the dura mater, in the way Professor Portmann had described, and open up the saccus endolymphaticus? In the great majority of cases of vertigo it was quite unnecessary to open the labyrinth or the saccus. In the operations designed to give relief in specially selected cases the middle ear was opened to obtain access to the labyrinth.

When Professor Portmann performed his operation on the saccus, he (Mr Scott) suggested he should try to do so without opening mastoid cells, and so avoid possibly fallacious conclusions that it was only drainage of the saccus that caused the beneficial results.

Dr J. S. FRASER said that he regretted that Dr Albert Gray was not present, as he had worked along much the same lines as Professor Portmann, *e.g.*, post-mortem injection of indian ink into the saccus endolymphaticus, causing the ink to circulate throughout the entire membranous labyrinth.

How did Professor Portmann explain the good hearing of patients

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suffering from meningitis? Professor Portmann had shown the relation of the saccus endolymphaticus to the posterior fossa of the skull, and had explained that changes of pressure in the posterior fossa affected the labyrinthine pressure. The pressure was, of course, greatly raised in meningitis and yet patients suffering from meningitis heard remarkably well.

With regard to "glaucoma of the labyrinth," he had felt there must be some disease in the inner ear corresponding to glaucoma in the eye. Here we had to do with the secretion and circulation of the endolymph. The endolymph was probably secreted by the cells of the stria vascularis. Any blockage of the endolymphatic duct or sac would be likely to cause "glaucoma of the labyrinth." As to his own (the speaker's) case, which Professor Portmann mentioned, of congenital syphilis affecting the middle and internal ear, it was true that the ductus endolymphaticus was blocked up, and that the utricle, saccule, and cochlear canal were dilated, but it must be remembered that one met with dilatation of the membranous labyrinth not only in cases of blocking of the ductus endolymphaticus but in many cases of spontaneous cure of labyrinthitis, in which no blockage of the endolymphatic drainage could be found. There must be some other causes of dilatation of the membranous labyrinth than mere blockage of the ductus and saccus.

With regard to empyema of the saccus endolymphaticus, he had microscopically examined over 100 cases in which the patient died as a result of the intracranial complications of middle-ear suppuration. In thirty-five of these the labyrinth was involved, but in only one case was there empyema of the saccus endolymphaticus. That was in opposition to what Mr Sydney Scott had said. Inflammation in the perilymph spaces was common, and in a smaller number of cases there was inflammation of both endo- and peri-lymph spaces, but affection of the endolymph spaces alone was very rare if not unknown.

Concerning experimental work on the labyrinth, he was sure members would recall Professor Biehl's work which was published in the *Journal of Laryngology and Otology* a short time ago (vol. xli., p. 637). Biehl made an incision in the tympanic membrane in the lower posterior part, and then inserted an angled needle round the niche of the round window, and tapped the perilymph space at the beginning of the cochlear canal. Biehl was doing this operation for the same class of case as that described by Dr Portmann. The great difficulty was to know how one could be sure that the vertigo was due to hypertension in the inner ear.

The operation proposed by Dr Portmann was not unlike that which was described for exposure of the jugular bulb from the mastoid wound. He (the speaker) admitted that in this operation there should be no injury to the posterior semicircular canal, but it might be a delicate matter to avoid such injury.

Mr H. BANKS-DAVIS said he gathered that Professor Portmann closed the wound completely. He (the speaker) did not understand what was the purpose of the operation if its result was to be only temporary. Did a fistula remain in the saccus, or was it merely opened and allowed to close? Also, was drainage subsequently carried out?

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Mr A. R. TWEEDIE said that when he first heard Professor Portmann's demonstration last October, he then expressed the opinion that it would provide a true explanation of the cases, to which Bárány drew attention, of suspected cerebellar lesions, but in which the posterior cranial fossa was opened and nothing abnormal was found, whilst the patients subsequently recovered.

Bárány did not think that he (the speaker) was right, but he (Mr Tweedie) maintained that this operation did re-establish the secretory connection between the saccus endolymphaticus and the dural spaces, which he submitted was the essential objective of the operation. Dr Albert Gray had been present at the former demonstration and made some remarks on it.

He (the speaker) was glad to hear stress laid upon the need of a careful selection of cases for the operation in question. It was, of course, important to try other methods first, reserving operation as a last resort. A possible indication, he thought, for this operation might be a hyposensitive response to the galvanic and caloric tests, which might be regarded as the result possibly of hypertension in the labyrinth.

He was very glad that Dr Jobson Horne brought his specimen forward, as one of his earliest otological recollections was hearing a demonstration by Sir Frederick Andrews, at St Bartholomew's Hospital, in which he drew attention to the saccus endolymphaticus, and the part it could play in the production of meningitis.

Professor PORTMANN (in reply) said he was interested in hearing that the term "glaucoma" had already been used in this connection; it was often the case in medicine and surgery to find that one's supposed originality had been anticipated. Bárány had said it was not necessary to open the saccus endolymphaticus, and that he had had good results from exposure of the dura mater. But in that case Bárány performed the same operation as himself (Professor Portmann), because he detached the dura mater from the petrous bone and thus ruptured the saccus endolymphaticus where this organ penetrated into the aqueductus vestibuli. He agreed as to the importance of what had been said relating to the posterior semicircular canals, but with care in the operation they could be avoided. He much preferred the route of entry which he had described.

When a new surgical procedure was initiated it could not be expected to be perfect at once. It was temporary, but it might be the means of making a way for a new communication. If closure occurred and a year or two afterwards there was a return of the symptoms, the operation could be repeated. It was not yet possible to know whether troubles were set up by pressure irregularities, but when a case appeared to resist all medical treatment, this operation might well be tried.

Dr JOBSON HORNE (in reply) emphasised the extreme rarity with which pus in the saccus was met with in the human adult. The aqueduct itself might remain patent, but even so, the pus tracked down and there would form an intradural abscess of the sac itself.

https://doi.org/10.1017/S0022215100031327 Published online by Cambridge University Press

Royal Society of Medicine

Clinical Cases shown on 11th June.

(1) **Deafness due to Occlusion of the External Auditory Meatuses caused by External Otitis: Operation to Restore Patency of Left Meatus resulting in Greatly Improved Hearing**—T. B. Jobson, M.D.—Male, aged 31, a railway fireman, was seen on 1st March 1923, with external otitis, right and left, and the walls of meatuses uniformly thickened: he was treated with various lotions and drops without benefit. Seen again in 1926, he was treated with vaccines and local treatment. No improvement followed.

18th January 1927.—Operation: mastoid incision; pinna reflected forward; cartilage of meatus separated; strip of cartilage removed from posterior wall; bony meatus enlarged by removal of some of the posterior wall; rubber tube 1 cm. diameter was left in for ten days.

(It is important to keep the rubber tube in for about ten to fourteen days, as there is a great tendency for the meatus to contract as it heals.)

Hearing for watch, which before operation was only "contact" right and left, is now 6 in. on left. His hearing for conversation is now good. Before operation it was only 2 ft.

Sir James Dundas-Grant said such cases of occlusion could sometimes be successfully treated by means of the laminaria tangle tent. In one case in which the removal of hyperostosis of the meatus by another surgeon was followed by complete occlusion, Sir James forced the point of a probe through the fibrous tissue and dilated the minute opening by means of a fine tangle tent whittled down to a point. He then introduced a minute rubber drainage tube, and eventually, after numerous dilatations and the introduction of rubber tubes of greater and greater size, the passage became so large that there was scarcely any narrowing. In contractions following the radical mastoid operation he had effected considerable widening in a similar way. In addition he had plugged the outer part of the cavity with cotton wool soaked in Burow's solution of acetate of lead and alumina acetate, and the result was excellent.

Mr A. D. Sharp said he had obtained excellent results in such cases by a plug of 10 per cent. ichthyol in glycerine. Even the very narrow type responded excellently.

Aural Electrodes for Use in the Treatment of Chronic Otorrhœa by Zinc Ionization and Zinc Electrolysis—A. R. Friel, M.D.—Dr A. R. Friel exhibited some electrodes for the ear which he used in the treatment of chronic otorrhœa.

(1) An electrode for the treatment of sepsis in an accessible position.

(2) An electrode consisting of two zinc wires fastened to a light wooden handle for the destruction of polypi or large granulations; and also for the treatment of an annular stricture of the meatus.

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(3) A monopolar zinc electrode for the incus area; and a bipolar electrode for the same area. Both of these are used to destroy tissue in order to make an inaccessible area more accessible.

(4) A bipolar electrode for the destruction of part of the outer wall of the attic.

Dr NORRIE (of Calcutta), introduced by Dr Friel, exhibited two portable rheostats and electrodes for the treatment of suppuration in the ethmoid cells; and nine needles for the treatment of hypertrophy of the superior turbinals as well as the electrode he used in the treatment of chronic otorrhœa. He showed an ingenious arrangement for retaining an electrode in the position in which he had placed it.

Cases of Deafness—H. NORMAN BARNETT, F.R.C.S.—*Case I.*—Female, aged 44, a physician's wife.

History.—No deafness in the family. One brother had tuberculosis. Patient had measles and scarlet fever as a child but no history of ear trouble. She has been deaf since 1914, getting steadily worse since then, with occasional tinnitus of a mild character.

Examination.—She has septic tonsils, deflection of septum, retracted tympanic membranes.

Hearing.—Right: B. C. – 2; R. – ; T. F. – 30; C. 1 ft.; V. 6 in.; W. 2/160. Left B. C., N.; R. – ; T. F. – 40; C. 2 in.; V. 1 in.; W. P./160.

Enucleation of tonsils, submucous resection of septum and submucous fracture of posterior ends of turbinate bones were performed. Ionisation over mastoid and vaporisation through Eustachian catheter with hot iodine, camphor and carbolic vapour, and oto-massage were carried out.

This case is still under treatment and is improving, but improvement much slower than in the other case reported as still under treatment. She can now hold conversation with one person at a 3 ft. radius. The case will require a considerable time to complete.

Case II.—Male, aged 35, inspector of police.

History.—In 1916 was blown up by shell which exploded. Part of shell hit steel helmet on left side, indenting it badly. Was unconscious for twelve hours. When consciousness returned patient noticed he was deaf on left side. This partly passed off later but hearing was never normal. Two years ago deafness got worse and gradually increased. Two months ago had influenza and since then the hearing in the right has been affected and has got steadily worse.

Examination.—He had septic tonsils and deflection of nasal septum. Tympanic membranes appeared normal.

Hearing.—Right: B. C. – 1; R. – ; T. F. – 30; C. 6 ft.; V. 2 ft.; W. 6/160. Left: B. C., N.; R. – ; T. F. – 40; C. 2 ft.; V. 1 ft.; W. 4/160.

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Enucleation of tonsils, submucous resection of septum, submucous fracture of posterior ends of turbinate bones, ionisation over mastoid and vaporisation through Eustachian catheter with hot iodine, camphor and carbolic vapour, and oto-massage were carried out.

The patient is now under treatment and steadily improving; he is able to perform his duties without difficulty. The case will probably require another fortnight or three weeks to complete the cure.

Case III.—Male, aged 50, a physician.

History.—General health poor and general family health good; no other member of the family was deaf.

Query as to whether middle-ear disease followed measles at four. At thirty, while starting a motor cycle, he suddenly got a "stuffy" feeling in ears which lasted for about eight years. While serving in Aden the ears seemed to be better but not normal. He had malaria badly in 1915, and has had twelve relapses—blood normal for past few years and no quinine during that time; also had dysentery in 1915; he was deafer two months after this.

History of Deafness.—Has never heard as well as other people, but noticed deafness particularly as a student when using stethoscope. Deafness became worse about eighteen years ago. Two years ago he began to get steadily worse and had great difficulty in using stethoscope. Four years ago saw a specialist, who suggested operation on turbinates, which was carried out with improvement for a time. In September 1925, the left ear became completely deaf. On this occasion patient had some inflation treatment with little or no result. Was advised to give up practice as nothing could be done for his hearing.

Examination.—First seen 22nd December 1925. Examination showed a marked deflection of the nasal septum with an adherent remnant of the right middle turbinate. He has buried septic tonsils and retracted and inflamed tympanic membranes. Antra clear on transillumination.

Hearing.—Right: B. C., N.; R. –; T. F. – 20; H. N. – 10; C. 20 in.; V. 20 in.; W.P./60. Left: B. C., N.; R. –; T. F. – 15; H. N. – 15; C. 18 in.; V. 15 in.; W.P./60.

Submucous resection of cartilage and bone of nasal septum which was found to be fractured; removal of diseased and adherent turbinates; administration of ultra-violet rays, general and localised for throat. Subsequently oto-massage with ionisation over mastoid and vaporisation through the Eustachian catheter and the tympanum with hot iodine, camphor and carbolic vapour.

On testing on 17th February 1926, and again on 1st March 1926, hearing was found to be much improved for all tests; while on 19th March 1926, hearing for conversation and whispered voice was found to be practically normal, and treatment was stopped. Patient has not

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since been tested but reports further improvement and is able to carry out all examinations in his profession with ease.

Mr A. R. TWEEDIE asked what was the result of catheterisation on the power of hearing before the operation, what was the response to the Gellé test, and did Mr Barnett try the effect of plain catheterisation afterwards without any "medication" to see if that alone would assist the hearing?

Sir JAMES DUNDAS-GRANT congratulated Mr Barnett on having been able to induce his patients to undergo thorough treatment. Most otologists knew definitely what was best for patients, but it was often difficult to get the latter to accept the treatment which was best for them.

Mr H. BANKS-DAVIS asked what Mr Barnett meant by the term "submucous fracture"; did it mean fracture of the bone after elevating or stripping the mucous membrane?

Mr A. D. SHARP said he considered that the good results in these cases were mainly due to the submucous resection of the septum, the tonsil operation, and the treatment of defect in the turbinals. He did not follow the rationale of ionisation over the mastoid. In his opinion thorough nasal treatment, in such cases, was to be commended and not condemned.

Mr R. SCOTT STEVENSON said the encouraging point was that Mr Barnett did make a determined effort to do such patients good. Many otologists, when they met with a case of chronic otitis media or otosclerosis, were apt to fold their arms and say they could do nothing for the patient. Even if simple catheterisation was carried out, to mention no other treatment, and the case was persevered with, some improvement often ensued in chronic otitis media. Sometimes it was found that one's patients had not previously had the catheter properly passed into the tube. He criticised the so-called "honest" attitude which led aurists to tell such patients that nothing could be done for them.

Dr J. S. FRASER said he took a line which was opposed to that of Mr Scott Stevenson; he was of opinion that many useless operations were performed on patients who were suffering from otosclerosis. The name "chronic catarrhal otitis media" was applied to these cases, but the bulk of them were suffering from otosclerosis. He was speaking especially of cases having drumheads which were normal in curvature but were slightly opaque. French women had facial skin which was somewhat more opaque than the facial skin of British women, hence the latter showed more pink in the cheeks than their French sisters. Similar differences applied to individual tympanic membranes; one was more opaque than another, yet both were normal. Such operations as enucleation of tonsils, submucous resection of the septum and turbinotomy, were frequently performed on these patients, without making them any better. Sometimes in cases in which only one ear was affected, the patient was told that the septum should be resected to prevent the deafness spreading to the "other" ear. He (Dr Fraser) did not think this was fair to the patient. Tonsils might require enucleation in a case of otosclerosis, but only because of tonsil disease—not with any hope of improving the hearing.

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If the drumhead was normal in curvature, even if opaque, no good could be done by operation on the nose. Otosclerosis was a deeper-seated disease than could be cured or even improved by submucous resection and the other procedures mentioned.

Dr DAN MCKENZIE (President) said that as one became older one found more and more cases which had been operated upon for dry middle-ear deafness, but the results, in his experience, were disappointing; the deafness continued to increase, whatever was done. Yet, despite that finding and despite what Dr Fraser had just said, medicine was an experimental science, and not until the experience of many men over a long period had been accumulated could one say how much could or could not be done for these cases. Each one's experience gave him a particular point of view, and he held that people who were active in their operating for these states were at the present moment justified. Even if their experiment proved finally that on the whole success was not to be obtained from surgical measures, they were nevertheless doing good work in making the attempt. But there might be a percentage of cases which would benefit.

Mr NORMAN BARNETT (in reply) said that he always operated, when operation was necessary, before beginning treatment of the middle ear. The operations were only performed when they were demanded on general surgical principles, although he believed the presence of septic tonsils and deflected septum were common predisposing causes of middle-ear deafness. Eustachian catheterisation with inflation by air without medicated vapour was of no use, except for a very temporary period.

Submucous fracture of the turbinates was performed by a special instrument which had been made for him. This necessitated no stripping of mucous membrane; the instrument was passed between the turbinates and the septum into the nasopharynx, and gentle pressure exercised when the bone fractured, one part overlapping the other and becoming fixed in that position: it was a preferable alternative to removing the posterior ends of the turbinate bones.

In Case III., as in all of the cases, history meant the account given by the patient of his disability, and not his (the speaker's) observations; examination referred to his (Mr Norman Barnett's) observation of the case when first seen. "Completely deaf" referred to the patient's statement; "practically normal" referred to Mr Norman Barnett's final examination. In the account of the operation "turbinates" should read "tonsils."

The three cases shown were examples of one cured, one approaching cure, and one better though still very deaf. They were all cases of progressive middle-ear deafness, and he considered the results were due to suitable treatment—such as referred to yesterday, and in the history of the cases—having been carried out. They were not people who had been slightly deaf, but very deaf indeed. One patient, a physician present at the meeting who would speak if allowed to do so, could tell of his inability to perform his examinations, and that now his hearing was perfectly normal. Another case was that of a schoolmaster who was dismissed by the Board of Education but reinstated after treatment and was carrying on satisfactorily. There were several of these schoolmaster

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cases. Another case amongst those shown was that of a police inspector who had to have his name removed from the "short list" for a chief constableship because he was unable to hear, and he would probably have had to leave the Force. He was now carrying on normally and his name could be reinstated on the list.

He (Mr Barnett) divided the case into three classes: those whom he never saw again but heard that their hearing was continuing satisfactory; those who came at intervals of three or four years for "tuning up"; those who wished to come oftener but were found not to have gone back very far.

Dr TOWNTEY (the patient referred to) said it was true to say his left ear was completely deaf; if he lay on his right side he could hear nothing. Mr Barnett was not the first man he had seen about his deafness. Eustachian inflation without medicated vapour did him no good. Enucleation of tonsils was not performed for his deafness, but because it needed to be done. After three weeks he found that ionisation was producing results, and at the end of six weeks he could hear very much better. Now, fourteen months afterwards, he was able to carry on his work. For three months he could not hear anything with the stethoscope.

Acute Lateral Sinus Thrombosis following Chronic Otorrhœa—J. A. GIBB, M.D.—Patient, aged 13, had otorrhœa of some years' duration.

23rd August 1926.—Admitted for mastoid operation; lateral sinus was opened and plugged.

25th August 1926.—Rigor at 3.30 P.M.; temperature 104° F.; internal jugular vein ligated, tied below facial vein and severed. Lateral sinus further exposed by trephining; septic clot scraped away; clot extended to the neighbourhood of torcular.

31st August 1926.—Slight rigor at 3.15 A.M. 20 c.c. antistreptococcic serum repeated every twelve hours for three days.

15th September 1926.—Delayed serum rash with swelling of knee-joints. Calcium chloride and iron given which soon relieved this.

22nd September 1926.—Tonsils and adenoids removed.

2nd October 1926.—Discharged.

Meningeal Symptoms Developing, Disappearing, and Reappearing. Cerebellar Abscess possibly Leaking into Meninges—E. BROUGHTON BARNES, F.R.C.S.E.—Female, aged 14, was sent to hospital on 12th October 1926, as a case of meningitis.

History of pain in the left ear for three weeks; discharge for four days; headache for forty-eight hours; bowels not open for four days; she vomited twice yesterday; no rigors; in a letter received subsequently the doctor stated that his diagnosis was based on the headache, vomiting, positive Kernig, and general facial expression.

On examination, however, Kernig was negative, Babinski negative. There was no head retraction, the child was not drowsy and appeared

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quite cheerful and normal mentally. There was a considerable amount of œdema of the tympanum. There was no mastoid tenderness or œdema of the posterior bony meatal wall. Temperature 98.8° F.; pulse 72. Diagnosis of acute otitis media was made.

Forty-eight hours later the child became drowsy during the morning and about mid-day developed meningeal cry. There was slight retraction of the head. Temperature was 98.4° F.; pulse 80. Operation, 14th October: superficial mastoid cells appeared to be normal, but there was a small deep perisinus abscess and the sinus wall was exuding pus with each heart-beat. The sinus was opened. It was not possible to get below the thrombosis from the mastoid. The internal jugular was tied and a portion excised at the level of the first tracheal ring. The pulse was imperceptible at the end of the operation. The patient recovered rapidly from the operation, had no further meningeal or intracranial symptoms for a week. There was no nystagmus and no atonia or dysdiadokokinesis. At 3.30 A.M. on the 22nd the patient suddenly started a meningeal cry; her pulse, which had been running between 80 and 100, suddenly fell to 52. She had a deviation of the eyes to the right with occasional flicks of cerebral correction, and rapidly became unconscious. She had a marked positive Kernig. Lumbar puncture: 5 c.c. of clear fluid were withdrawn under very slightly increased pressure. Two hours later, operation through the mastoid incision: cerebellum was explored through the sinus wall, which was adherent to the cerebellum. Cerebellum was obviously compressed, and at a depth of half an inch an extremely stout abscess wall was found. On opening the abscess a slightly turbid fluid in considerable quantity was followed by pus which appeared to be in *loculi* and came in rushes as the forceps were moved in the cavity. The pus was followed by cerebrospinal fluid. The abscess wall was so stout that a thick rubber drainage tube was immediately collapsed by it. A T-incision had to be made in the abscess wall to drain it.

Twelve hours later the patient was unconscious and apparently dying. The pulse was 136 and extremely weak. At half-past six she suddenly sat up and asked for an orange. She had then lost all nystagmus and deviation, but her left side was notably atonic. Lumbar puncture; fluid contained short chain streptococci and Gram-negative bacilli. The streptococci grew on culture, the bacilli did not. Pus from the abscess contained the same organisms. Forty-eight hours later, 28th October, lumbar puncture; fluid under slight pressure contained streptococci which did not grow on culture. Cerebrospinal fluid drained through the tube in the abscess for three days.

29th October.—Lumbar puncture: slightly increased pressure; no organisms in the fluid. The dysdiadokokinesis and atony persisted and became slightly more marked at the beginning of November. On

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16th November radical operation was completed and a skin graft placed which did not take well. Recovery from this point was uneventful. The child did not complain of headache after the end of October. Her tonsils and adenoids were removed on 21st December.

There have since been troublesome granulations from the anterior part of the radical cavity.

I think that the cerebellar abscess was leaking into the meninges at intervals.

Dr DAN MCKENZIE (President) said that last year he had shown specimens of brain and meninges from a similar case. Meningitic symptoms appeared and disappeared three or four times, the child being ill three months. The reason of the death was that there was a double abscess; a deeper satellite abscess had escaped notice. These cases showed that the meninges had great power of resistance against infection. In his case the arachnoid space was occupied by tough fibrous tissue as a result of the sepsis.

Dr J. S. FRASER said Mr Barnes was to be congratulated on his results. What would be of value would be for some members of the Section to give the results of *all* their cases of meningitis. These two cases and their results gave a wholly erroneous impression of the usual percentage of cures in such conditions.

Mr J. A. GIBB said he had a case of the kind in a man aged 25 who had chronic otorrhœa with a fulminant mastoiditis, thrombosis of the lateral sinus and abscess of the temporo-sphenoidal lobe. He was treated and made a complete recovery, the wound healed, and he (the speaker) went away for a holiday. Three days before his return the man had a fit and died. A *post-mortem* examination was not made.

Mr BROUGHTON BARNES (in reply) promised to write and send in a list of his cases, procedure and results.

The two cases shown were actually consecutive, but he did not claim anything like a hundred per cent. successes. His cases were too few at present for statistical purposes.

AUSTRIAN OTOLOGICAL SOCIETY.

MEETING OF FEBRUARY 1927.

(*Monatschrift für Ohrenheilkunde*, April 1927.)

Abstract of Proceedings by ALEX. R. TWEEDIE, F.R.C.S.

Fracture of the Left Temporal Bone and External Acoustic Meatus without Injury to the 8th Nerve: Laceration of the Skin of the Bony Meatus—S. GATSCHER.—As the result of a fainting fit, 19th February 1927, the patient fell on the back of her head at 7 P.M.; she was brought to hospital unconscious,

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