SOCIETIES' PROCEEDINGS

ROYAL SOCIETY OF MEDICINE—SECTION OF OTOLOGY.

December 6, 1931.

President—Mr. NORMAN PATTERSON.

Cholesteatoma in Aural Suppuration: Its Relation to Prognosis—T. B. JOBSON.—The paper is published in the *Journal of Laryngology and Otology*, Vol. xlvii., p. 380, June 1932.

DISCUSSION.

Mr. HERBERT TILLEY (after showing two drawings of the anatomy of the tympanic attic, for which he was indebted to Professor Schmiegelow of Copenhagen) said: "These drawings will, I think, go far towards explaining two of the three questions which Dr. Jobson has asked: Why are 'attic' cases so resistant to conservative treatment? In the light of the anatomical conditions which had been demonstrated, surely the resistance must be due to defective drainage, not only between the small areolar cavities in the anterior part of the attic, but also between these cavities as a whole and the freer, undivided portion of the attic posteriorly.

When for these reasons suppuration becomes prolonged, chronic osteitis of the immediately neighbouring bony structures sets in, and hence one finds absorption of the heads of the malleus and incus and of the external attic wall (inner and upper wall of bony meatus), with formation of granulation tissue or polypus formation. At the same time, or later on, the infective process finds its way into the freer posterior region of the attic, thence to the mastoid antrum, and so completes the clinico-pathological picture with which we are all familiar.

It is needless to add that serious complications may arise when the osteitis attacks the roof of the middle-ear cleft or the labyrinthine structures, or lays bare the facial nerve in the aqueduct of Fallopius. Such conditions have their counterpart in chronic suppuration coupled with defective drainage when they occur in the nasal accessory air cavities and particularly so in the case of the ethmoid labyrinth.

Dr. Jobson's third question is, I think, also explained by the anatomical conditions. The perforation is commonest in the upper region of the membrana flaccida because here it is thinnest, while a band of comparatively thick connective tissue separates the larger upper region of the attic (Kretschman's cavity) from Prussak's space below, where one might otherwise expect to find the perforation. I am on less sure ground in an attempt to answer his second question with regard to the close connection between attic suppuration and cholesteatomata. My difficulty would be to explain why we never find true cholesteatomata in chronic suppuration of the

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nasal accessory air cavities, where defective drainage is so pre-eminently present and provision for its freedom forms the basis of all treatment.

May not this disparity be due to some developmental characteristics of the attic epithelium not present in that of the nasal cavities? Virchow rather favoured this view and pointed out that the mucous membrane of the attic is the remains of an epidermal 'bud' from the external layer of the blastoderm which is buried at the level of the first branchial arch and governs the development of the auditory apparatus, of which the aqueductus vestibuli is a normal structure. If Virchow's theory be correct it would not be difficult to understand that infection of the attic would cause irritation and proliferation of its epithelium, which would take the form of the areolar spaces which it lines, ultimately destroy their boundaries and so form the characteristic 'cholesteatomatous mass.'

I cannot see eye to eye with Dr. Jobson in his theory that cholesteatoma is a primary condition, because there are evidences that it may be caused by an initial tubo-tympanic infection of which I have seen a few instances, and many in which it has followed a primary involvement of the tympanum. Schmiegelow has recorded three cases of primary infection of the attic, one as a complication of influenza, the second following operation for adenoids, and a third resulting from a nasal douche employed during an acute nasal catarrh. Furthermore, Schmiegelow, Bezold and Morpurgo have described cases in which the 'bruit de perforation' was heard in purely attic cases; so that it is obvious that infection by way of the Eustachian tube is not so exceptional as one might think.

Nor do I entirely agree with the statement that 'one invariably finds the membrane adherent to the inner wall to a greater or lesser extent.' Otologists must often have been struck with the apparently normal appearance of the 'membrana propria,' especially in comparatively recent cases, and also with the relatively good hearing which may be present—an embarrassing condition when one has had to explain to the patient that if a dangerous lesion in the ear is to be removed he must be prepared to find the hearing of the affected ear considerably damaged.

May I suggest that when the tympanic membrane is much indrawn, this may be a result of a primary infection of the tympanum which cleared up, but only after it had infected the attic?

With regard to the treatment, it has been my experience that if from the history and clinical aspects of the case we can be fairly sure that the condition is of recent origin, it may be possible, by means of Hartmann's cannula, to cure a few cases by daily irrigation and the injection of such solutions as peroxide of hydrogen, argyrol or some of the iodine derivatives. Many more may be cured by ossiculectomy, which provides free drainage and access to the attic cavity for the gentle use of a curette and the application of suitable antiseptics.

In 1891 Ludwig (of Halle) published a record of twenty-eight ossiculectomies. In nine there was no modification of the hearing, in sixteen there was an improvement, and in only three was it made worse. I mention these facts because during recent years there has been a tendency to discount this operation, which has a very real and practical value in carefully selected cases, but there will be a still larger residue of cases in which symptoms and clinical findings will lead the experienced otologist to advise an immediate

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radical operation in order to expose, cleanse and provide for free and permanent drainage from the tympanic attic and mastoid regions of the middle-ear cleft."

Mr. F. H. DIGGLE said that three years ago he had suggested to a clinical assistant the view that these cases were primary, and the rejoinder had been: "That is a very interesting theory; have you written about it? Does anybody believe it?" He had felt it needed someone with a riper experience to put it forward. In the last three years he had had two interesting cases which, he thought, helped to confirm it. Mr. Hamilton would remember the case of a youth in the army who, during the latter part of his career, suffered from vertigo. He had a gun-shot through-and-through wound which was below the mastoid region. The man had been regarded as a malingerer and was returned to duty. At the Board the speaker saw that the man had a definite attack of vertigo. Nothing was to be found in the ear, hearing was perfect, and the membrane was normal. He asked the man to continue to attend at the out-patient department, and one day a white spot developed over the posterior part of the membrane, and in due course he saw it perforate. A limited cholesteatomatous disease was seen at operation, with a perforation into the external semicircular canal.

The next case had been diagnosed as a simple Bell's palsy, probably of rheumatic origin. One day a small bleb developed on the posterior part of the membrane, in the posterior part of the attic region, and, with the experience of the previous case in mind, he (the speaker) cut down and found a limited cholesteatoma, with necrosis, leading into the Fallopian canal. These cases confirmed his opinion that most cases of cholesteatomatous disease were primary invasions of the attic region.

Dr. Jobson had pointed out the importance of diagnosing these cases early; but he, the speaker, did not know how this could be done; the patients had no pain, no loss of hearing and no discharge at so early a date. It was only by a recognition of the fact that vertigo or even facial palsy, which could not be otherwise accounted for, may on occasion be due to an unsuspected limited cholesteatomatous patch in the mastoid bone. Otologists knew the difficulty there was in getting rid of cholesteatomatous material in a mastoid cavity. His chief, the late Sir William Milligan, always made a point in these cases of swabbing out the cavity at the completion of the operation with pure carbolic, with very good results.

Dr. J. S. FRASER said that Dr. McKenzie and Dr. Jobson apparently held that cholesteatoma was a form of congenital tumour due to fœtal inclusion of epiblast in the middle ear. If that was so, it was surely the most frequent form of congenital abnormality which existed in the human race! Cleft palate and dermoid cysts were not nearly so common. He (Dr. Fraser) was convinced that in the majority of cases cholesteatoma was not of congenital origin but was caused by metaplasia. In ozæna, which was the advanced stage of chronic purulent rhinitis, the ciliated columnar epithelium reverted to the squamous type. In chronic bronchitis and bronchiectasis the lining membrane of the bronchiectatic cavity consisted, not of ciliated epithelium, but of squamous cells. Cholesteatoma had been produced experimentally in the uterus and even in the stomach, and in all these regions the squamous cells could not have come from the skin or

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from fœtal inclusion. Cholesteatoma was a reversion from the more highly developed ciliated columnar epithelium to the lower protective or squamous type.

It was a common condition, and if all cases were due to the fœtal inclusion of epiblast in the attic and antrum one would expect that observers like Cheatle and Mouret, who had examined many hundreds of mastoids, would have found a considerable number of specimens showing congenital cholesteatoma formation. So far as he knew this had not been the case.

Invagination of Shrapnell's membrane might produce a small cavity lined by squamous epithelium. Acute suppuration of the attic due to influenza might become chronic and this also would account for some cases of attic cholesteatoma.

Mr. ALEX. R. TWEEDIE said that he wondered if there was really such a mystery about the condition as Dr. Jobson had suggested. Surely the initial basis of cholesteatoma was, as Dr. Fraser had just indicated, always some infection from the naso-pharyngeal tract. Indeed, the cases shown that day by Dr. Jobson confirmed this view. He (Mr. Tweedie) would submit that the condition was dependent on some primary infection, from the nose and throat, of the attic or antrum, to which the "owner" offered considerable resistance but only with partial success, so that there continued some development of a low form of sepsis, and therewith the mucous membrane concerned underwent an abnormal alteration.

Neumann, whom he had quoted in this connection last year, had pointed out that the lining cells of the mastoid cavity were a peculiar form of muco-periosteum, and he (the speaker) suggested that it was the irregular desquamation of this infected epithelium, retained by the lack of sufficient means of exit, which was the main factor in the production of cholesteatoma.

With regard to the cholesterin content of the blood, Professor Alex. Rejtö, of Budapest, had already made considerable investigations, as recently reported by himself at a meeting of the "Collegium" in Bordeaux, and also by one of his assistants, in the *Monatsschrift für Ohrenheilkunde.* In this latter communication it was stated that the cholesterin content of the blood had been found to be fairly constant in normal people; was increased in cases with cholesteatoma, but returned to normal when the latter condition had been successfully treated; that there was no causative relation between the cholesterin content of the blood and the *formation* of cholesteatoma, but that the presence of cholesteatoma, by reabsorption of cholesterin, tended to increase the cholesterin content of the blood. As a further interesting clinical application of these statements, it was then suggested that the degree of the cholesterin content of the blood might form a valuable indication as to the urgency of a case of suspected cholesteatoma and constitute a reason for operation. As regards palliative measures, he submitted that oily solutions would be found more efficacious than those with either a watery or spiritous medium.

Dr. ROBERT R. SIMPSON showed the skiagram of a mastoid case. The patient came to the out-patient department complaining of frontal headache, with a foul discharge from the ear. All the membranes and ossicles had disappeared, and the labyrinth was dead. Because of the headache an

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X-ray examination was made. There was an unusually large cholesteatoma, which was one which had effected a maximum amount of damage without having given much evidence of its presence. There was to be seen an erosion of the middle and posterior fossae, and on removing the cholesteatoma from the sinus and cerebellar dura some cerebrospinal fluid escaped. Because of that he, the speaker, thought it wise to delay the completion of the radical operation for forty-eight hours. The external canal was eroded, the facial nerve exposed and the whole promontory gone. He had not suspected any of the complications. There was an extension of the cholesteatoma through the tip deep to the attachment of the sternomastoid. After the radical mastoid operation the patient made an uninterrupted recovery. The ear was now, after a year and a half, beginning to discharge again.

Dr. DAN MCKENZIE: "The fertilising nature of the idea that cholesteatoma is always primary is to be seen in Dr. Jobson's paper, for by it the attention of the surgeon is directed first and foremost, though not by any means exclusively, to the epidermal sheet that is responsible. The grasping of this truth—as I believe it to be—was the most important practical point in my recent paper. The further discussion on the origin of the cholesteatomatous lining is more or less academic; nevertheless, it is of great interest.

There is, as I said a year ago, much to be said for the metaplasia theory; on the other hand, there is also much to be said against it. To begin with, how does it come about that only a fraction of those who suffer from suppuration of the middle ear develop it? And yet, compared with cholesteatoma in the nose and its sinuses, in the gall-bladder, in the uterus and so on, cholesteatoma in the ear is extremely common. It can scarcely be due simply to metaplasia in an enclosed space, or we should find it common also in sinusitis of the frontal and ethmoid bones. As to the relationship of ozæna with cholesteatoma, no one can deny that, in both, the essential pathological condition is the presence of stratified keratinised epithelium where it has no business to be. But the change in ozæna is general, involving the whole of the respiratory tract, down to and including the larynx and sometimes even the trachea. On the other hand, as I pointed out in my former paper, aural cholesteatoma is curiously localised and circumscribed; it presents a nidus. Further, we seldom, very seldom, find aural cholesteatoma associated with ozæna. When the ear goes wrong in ozæna, it is usually dry otosclerosis, or a tubo-tympanic suppuration.

Returning to the metaplasia theory, we must remember that whatever theory we adopt it will have to explain the predilection shown by cholesteatoma for the attic-antrum region; and the metaplasia theory is weak in this respect. True, in my former paper I suggested that, as the lining of the tympanum consisted mainly of cubical cells at this part, it was conceivable that their transformation would be more easily affected than if they were cylindrical. But this is pure hypothesis. We know that cylindrical epithelium changes into stratified epithelium readily enough. Why then should the metaplasia from suppuration be confined to the attic and antrum? Why does it never appear in tubo-tympanic suppuration? And if cholesteatoma were due to simple metaplasia in a confined space, why do we not see it as an everyday occurrence, as often as scar tissue

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forms in a suppurating ear? There is no answer to these questions, and until there is we must regard the metaplasia theory as dubious.

Let us turn now to the possibility of congenital implantation. By way of interest I show here a photograph of the lining of an ordinary implantation cyst, for permission to use which I am indebted to Professor Muir. Its resemblance both to aural cholesteatoma and to the cholesteatomatous brain tumour leaps to the eye.

I do not propose to recapitulate what was said in my former paper on this subject. I will merely add a few points in its favour that have since occurred to me. When we examine the course of development of the external auditory meatus and tympanic cavity, the likelihood of congenital implantation through the detachment of epidermal cells cannot be denied.



Implantation cyst. Source not stated. They occur on cutaneous integument and conjunctiva. (From Professor R. Muir's *Textbook of Pathology*. With grateful acknowledgments.)

For we are looking at a peculiar, a unique structure here. Nowhere else in the body does the subcutaneous tissue form the periosteum as it does in the roof and posterior wall of the deep meatus. That is to say, nowhere are skin and bone so closely, so intimately, connected. You might even call them contiguous. Again, in Shrapnell's membrane you have this fine skin-layer unsupported by bone, in contact with tympanic epithelial lining.

Now this arrangement is due to the invasion during fœtal life of the mesenchyme by a solid plate of ectodermal cells, sprouted off from the primary external auditory meatus, which pushes in through solid mesenchyme to form the deep meatus, hairless and devoid of glands; to form also, with the mesenchyme and the endoderm, the tympanic membrane and finally, though not actually until after birth, Shrapnell's membrane. During this invasion, be it remembered, the only hard structures the meatal plate encounters are the handle of the malleus and the tympanic ring, both of them cartilaginous. Obviously, nothing is more probable than that in its penetration and expansion to form the deep meatus the meatal plate should shed a cell here and there, which, sequestered and enclosed by the later developing bone of the outer attic wall and of the bony meatus, would form the starting point of an epidermoid—a congenital implantation cyst.

But, it may be objected, if this were to occur, surely evidence of such epidermal rests ought to be found in these regions. This objection can be met. Such rests have been found and reported under the picturesque name of epidermal 'splinters,' and their significance relative to the development of primary cholesteatoma has been recognised.*

* Precěchtěl, *Acta O.-L.* xiv., Fasc. 1-2.

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Let me remind you also of the cholesteatomatous cyst of the membrane I depicted in my paper. Finally, I should like to say that this theory explains also the small dry puckerings, depressions and even tunnels or fistulae so frequently seen in Shrapnell's membrane.

We are therefore entitled to suppose that the congenital implantation cyst is probably quite a common event.

Before concluding, I should say that my attention has been drawn to the report of aural cholesteatoma by invasion being successfully and artificially produced in animals. Unfortunately, the reports available have been so scantily detailed that I am unable to express any opinion upon them, whether critical or otherwise. They certainly seem to have been accepted by well-known authorities. But authorities occasionally err, as Politzer did over Kirchner's Haversian invasion.

I saw recently a statement made by a prominent authority to the effect that cholesteatoma could form in acute suppuration of the middle ear in two or three days even in the scarlatinal form by invasion. This is incredible. Politzer said cholesteatoma, under those circumstances, did not occur—acute sepsis rendering such an event impossible. We know with what difficulty the normal epidermis extends over an inflamed ulcerated surface, and yet we are to believe that in the presence of acute scarlatinal suppuration of the middle ear, in spite of the intensity of the infection, it is possible for the epidermis to invade the tympanic cavity. I find it difficult to believe that at one and the same moment the epidermal covering of the membrana tympani dies, forming the familiar white pellicle found in this condition, and also manifests so much increased vitality that it can make a victorious invasion of the middle ear."

Dr. JOBSON, in reply, expressed his gratitude to members for their reception of his paper.

Sir James Dundas-Grant had referred to the effect of pressure; he (Dr. Jobson) thought there was something operating besides pressure, namely, the absorptive power of cholesteatoma.

One met with cases of pseudo-mastoid cavity, such as Case No. VI., in which there was no pressure at all; there was simply a layer of epithelium which absorbed bone and had produced the cavity. That had gone on year after year and caused the condition. He agreed, however, that pressure had an effect in some cases.

Mr. Tilley accounted for the obstinacy of the cases by the very complex form of the cavity of the attic. True, but that was common to all cases of suppuration; it was as common to cholesteatomatous cases as to those of ordinary tympanic sepsis with a perforation in the lower tympanic membrane. But the difference in behaviour in these two groups of cases was very striking. When one met with a fair-sized perforation in the lower tympanic membrane one could almost guarantee that the ear would clear up and the sepsis would abate. In both cases, however, the sepsis involved the attic, the only difference being that there was a different type of epithelium lining the attic in one case as compared with that in the other, this accounting for the chronicity and the obstinacy.

He was aware that Dr. Fraser's argument was very strong, that if cholesteatoma was called a congenital tumour it was the commonest of all forms; but if one called it by another name, congenital epithelial defect, the hypothesis of Dr. McKenzie became more acceptable.

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Societies' Proceedings

THE FOLLOWING ILLUSTRATIVE CASES WERE SHOWN.

Cholesteatoma exposing the Lateral Sinus—SOMERVILLE HASTINGS, M.S., F.R.C.S.

L. H., a woman, aged 47, was shown at a meeting of this Section in November 1917. She gave a history of chronic discharge from the right ear for eight years, and a pain in that ear of a few weeks' duration. She said that she had never had any operation on the ears. The right ear was filled with pus and epithelial débris, and when this was removed a smooth-walled cavity was exposed, all trace of the membrana tympani and ossicles being lost. The walls of this cavity were ulcerated in places and posteriorly for about half an inch, the lateral sinus was exposed and could be seen pulsating. The hearing in the right ear was then very defective and no tuning-fork could be heard by air conduction, but middle and lower tones were heard by bone conduction, with a noise machine in the left ear. There was much scarring of the posterior wall of the pharynx, which was said to be the result of scarlet fever in childhood.

The patient attended hospital at rare intervals until July 1930, when she was admitted urgently with a history of hæmorrhage, lasting several hours, from the right ear. When blood clot was removed, a tiny ulcer over the site of the lateral sinus was visible, but no hæmorrhage took place while the patient was in hospital. The small ulcer rapidly healed but no pulsation has been visible in the lateral sinus since this incident. Since July 1930 the patient has attended hospital three or four times for removal of epithelial débris from the ear, but there has been no ulceration.

Cholesteatoma and Epithelioma: Specimen — DAN MCKENZIE, M.D.

In a paper read before the Section last year mention was made of the possibility of transformation of cholesteatoma into epithelioma. The present sections are from a case in which the two were present in association.

Healed Marginal Perforation of Posterior Half of Membrana Tympani: Specimen—E. D. D. DAVIS, F.R.C.S.

Cholesteatomatous suppuration of the attic with a fistula of the ampulla of the external semicircular canal.

The patient was admitted with a temperature of 104°, drowsy and too ill for labyrinthine tests. At a mastoid operation a latent lateral sinus thrombosis with an extensive abscess was found.

The patient died from cavernous sinus thrombosis.

Twelve months before death there was a history of severe vertigo.

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