CHRONIC ADHESIVE OTITIS*

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CHRONIC adhesive otitis is an elusive condition which is by no means as common as a study of the former literature suggests. There is by no means general agreement about what can be included under this heading, but if words mean anything, then chronic adhesive otitis indicates a long-standing adhesive process as the result of inflammation of the ear; and it is usually assumed that the middle ear, its contents and its boundary walls are the parts affected. Some authorities exclude cases of former suppuration, but if I were to do this, then the number I have seen would fall from 22 to 5 and I do not think that it is practicable to exclude cases with adhesions as the result of former inflammation on the grounds that there was suppuration as well.

The diagnosis is established by the history of former ear trouble and conductive deafness often from an early age, combined with abnormalities of but not deficiencies in the tympanic membrane. These abnormalities include scars, opacities, chalk deposits and perhaps most important of all impaired mobility. Although an adhesive process may sometimes be present in the middle ear, when there is a perforation of the ear drum, be it dry or moist, I have not included such cases in the adhesive group. Nor have I included cases without any obvious hearing loss though I have been surprised how often even gross abnormalities of the tympanic membrane can exist without any noticeable deafness, and sometimes without any history of previous ear trouble.

Now that you have heard my criteria for a diagnosis of chronic adhesive otitis I would like to consider in more detail some of its features and I will start with the incidence, as to me this is one of the most surprising aspects of the disease.

Incidence

For many years now I have kept a note of the diagnosis of every new patient seen and I have been particularly on the look out for cases of chronic adhesive otitis for some time now. During the three years 1953-1955 I saw 1,541 patients whose hearing in both ears was impaired, and of these only 22 were noted as being cases of chronic adhesive otitis.

These figures include all patients seen at hospital and in private practice, and I would add that 10 of the 22 cases of adhesive otitis were seen in

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private practice. With regard to the hospital cases, it may be wondered whether this small number is due to an unduly high proportion of adhesive cases being diverted away from me; but my associates will bear me out that for some years now we have been talking about adhesive otitis and how little we see of it, and I have asked if I might see as many cases as possible. I think, therefore, it is unlikely that many cases in my clinics have gone unnoted; though I am the first to admit that some may have been allowed to creep into the otosclerotic group. With this in mind I have reviewed all the fenestrations done by my associates and myself in the past three years. These total 635 and in three the cause of the deafness appeared at operation to be due to adhesive otitis, and on reviewing the notes there were in all three changes in the appearance of the tympanic membranes.

I should say again that I have not included under the heading of adhesive otitis any case of active suppuration, or with an obvious perforation. Nor have certain cases with minor blemishes such as small scars or chalk patches in a mobile tympanic membrane been included in the adhesive group particularly when they present other features suggesting otosclerosis. Finally only cases with deafness have been included.

Another feature about the incidence is that it does not appear to be on the increase. I have no figures to support this impression as until a few years ago I used to group these cases under the heading of "former otitis media", but it would be interesting to know if others have the same impression. If, as Mr. MacNaughtan suggests adhesive otitis is predisposed to by injudicious management of acute otitis media, then either the General Practitioners are managing it better than we used to, or there is a great deal less acute otitis media. On the other hand it may prove to be that adhesive otitis is due to other causes than acute otitis media as it now occurs and behaves.

The Hearing in Chronic Adhesive Otitis

Polvogt (1936) in an interesting paper on histological variations in the middle and inner ears of patients with normal hearing found gross adhesive changes in the tympanum of 38 subjects with normal hearing. He also found that bands of embryonic or fibrous tissue in the niche of the round window and even in the fossa of the oval window could be present without any loss of hearing. Perlman (1943) confirmed Polvogt's observation, adding that given an intact and mobile ossicular chain, occlusion of the round window niche to a large extent by fibrous tissue was compatible with normal hearing.

I am able to confirm all these findings, as I have seen from time to time tympanic membranes with gross abnormalities including adhesion to the promontory and normal hearing. Also, for the past three years I have at operation been inspecting the tympanum and particularly the round win-

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dow niche in cases of otosclerosis. Small bands of tissue are frequently seen in the round window niche and they do not seem to play any part in the cause of the deafness.

Wever and Lawrence (1949) in their interesting papers on sound conduction have also stated that gross adhesive changes in the tympanum are compatible with only very slight loss of hearing acuity.

When the hearing is affected the deafness is primarily conductive and as a rule the loss is not severe. In my series there was an average loss of 45 decibels for pure tones within the range of speech and in only five cases out of twenty-five was the loss greater than 50 decibels.

The Tympanic Membrane in Chronic Adhesive Otitis

Lumio (1951) of Finland in a valuable contribution to the knowledge of chronic adhesive otitis studied 185 cases all with intact tympanic membranes. In nearly half there was a history of former ear trouble in the shape of earache or discharge.

The commonest abnormality was impaired mobility of the drum in 75 per cent.; then came generalized opacity in 63 per cent., adherent drum in 55 per cent., scar in 38 per cent. and calcium deposit in 17 per cent.

I have yet to recognize a case in which the tympanic membrane is normal, and in those few in which an original diagnosis of otosclerosis had to be altered at operation, there were always variations from the normal in the appearance of the tympanic membrane. I think that perhaps the most difficult cases are those in which the tympanic membrane is uniformly opaque, the so-called "milky drum".

In addition the drum is usually immobile when there is an underlying adhesive otitis. May I emphasize the point that in a bilateral case the hearing may be better in the ear with the more obviously affected tympanic membrane, though as a rule mobility rather than appearance of the ear drum is the most important clue.

I would like to mention two cases each of which I think may properly be regarded as a case of chronic adhesive otitis yet the history and clinical features are dissimilar. The first is the case of a young girl of 17 with a history of progressive deafness for five years. At the age of 10 she had an acute otitis media on the left side for which a cortical mastoid operation was needed. Apart from this there was no history of ear trouble.

She had a conductive deafness in each ear with an approximately even loss of 45 decibels in all, but the highest frequencies where the loss was only 30 decibels. Each tympanic membrane presented a uniform milky opacity and though each eustachian tube was patent, neither tympanic membrane moved freely. She was submitted to a fenestration operation on the right ear, and the tympanic cavity and antrum were lined with a membrane reddish and opaque and so thickened as to hide the usual

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landmarks, though there were no adhesions in the cavity. The stapes did not move freely, but the round window niche was not clearly seen. A fenestra was made in the bony lateral semicircular canal, and though an active fistula sign has been retained ever since she has never gained any improvement in hearing.

The next case is of a man aged 46 who had been very hard of hearing since his later schooldays. He had been subject to repeated bouts of earache and deafness since early childhood and from time to time his ears had discharged for a short period.

He had a severe conductive deafness with a loss between 60 and 75 decibels across the frequency range. Each tympanic membrane was thickened, scarred and immobile. At operation the mucosa was thickened and the tympanic cavity contained several thick bands between the tympanic membrane and the inner tympanic wall, and the incus and head of the malleus were bound down by a dense mass of whitish fibrous tissue. The stapes did not appear to move freely. At that time I was not paying the attention I now do to the round window niche so I cannot say what state it was in. Despite a formal fenestration with subsequent retention of an active fistula sign there was no improvement nor was there any loss of hearing.

Differential Diagnosis

Though it is easier to decide the cause in a case of conductive than in a case of perceptive deafness, it is at times quite difficult to decide between an adhesive process and otosclerosis as the cause of a conductive deafness.

This is because the mechanism of the impedance is similar in both cases, and it may well be that at times both causes operate simultaneously.

Furthermore, it has been established that bony deposits in the labyrinthine windows may be caused by new bone formed as the result of inflammation as well as otosclerosis, so that mechanically as well as pathologically the causes are virtually the same.

The history and appearance of the tympanic membrane are the main features in arriving at a diagnosis and I cannot do better than commend to your notice the table prepared by Lumio of the differential diagnosis between chronic adhesive otitis and otosclerosis.

Prophylaxis

With regard to prophylaxis, I think that we need to study the possible causes more carefully before we can come to any definite conclusions. This means that we must not only enquire carefully into the previous history of each case that we see, but also we must start at the other end and follow up some of the cases of tympanic disease which come under our care when they are still young. At present I have the impression that many

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of the cases we used to see formerly were the result of severe tympanic sepsis following specific fevers; and the reason we see less of this group of cases is that the otitic complications of specific fevers are reduced in severity by the early exhibition of sulphonamides and antibiotics.

The Surgical Treatment of Chronic Adhesive Otitis

Now we come to the treatment of chronic adhesive otitis and so far as the medical management of the established condition is concerned this means a hearing aid. Fortunately most cases are able to hear well with an aid, but the success of the fenestration operation in otosclerosis has raised the hopes of both patient and Otologist as a possible form of treatment, in chronic adhesive otitis.

Some years ago I tried the effect of a fenestration operation on four cases of chronic adhesive otitis and, though I learned quite a lot about the pathology of the condition, all four cases failed to respond and so I lost my enthusiasm for this form of treatment. Since then I and my colleagues have, either by accident or design, operated on a further seven cases, again without result.

These experiences leave me with a feeling that the formal fenestration operation alone is not the proper treatment for chronic adhesive otitis.

It may well be that some form of tympano-plasty is needed; and in any case an inspection of the tympanic cavity by the Rosen approach is likely to be a useful preliminary.

There is one important point always to be borne in mind in the surgical treatment of deafness, and that is to make sure that there is one and *only one* mobile window on the vestibular and another on the tympanic side of the basilar membrane. For instance, if the stapes is freely mobile then an additional mobile window on the vestibular side of the membrane in the lateral semicircular canal is likely to be followed by a most disturbing condition, in which loud sounds cause giddiness or, in other words, the "Tullio Phenomenon".

I have had to treat several patients distressed by this condition and in some the operation has been carried out for the relief of adhesive deafness in which the stapes was mobile. It is for this reason that I believe that a necessary preliminary to the surgical treatment of chronic adhesive otitis should be a test, preferably visual, of the mobility of the oval window.

If the stapes moves freely then a fenestra should not be made in the lateral canal: if it is obviously impeded, either by bone or by dense fibrous tissue, then a fenestration may be tried; but, of course, an unimpeded round window is a necessary condition for success, and I think that we may need to pay more attention to this region.

I wish that I could have added a more positive contribution to our knowledge of chronic adhesive otitis, and I feel that I, at any rate, must

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continue to be on the lookout for further examples of it. We need to know more about its incidence and causes, so that in time we may be able to offer more effective treatment for the established condition.

REFERENCES

LUMIO, J. S. (1951) *Acta otolaryng.* (*Stockh.*), **39,** 196.
OJALA, L. (1953) *Arch. Otolaryng.* (*Chicago*), **57,** 378.
PERLMAN, H. B. (1943) *Ibid.*, **37,** 680.
POLVOGT, L. M. (1936) *Arch. Otolaryng.* (*Chicago*), **23,** 48.

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