Section of Otology

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The Heritage of British Otology

PRESIDENT'S ADDRESS

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"As Practice is the last and chiefest part of Physick, so is Observation the surest and most demonstrating part of Practice."—*Dr. John Bird* (1657).

OUR heritage in British otology does not merely comprise a list of distinguished names, personalities, and traditions. It must also include some of the problems which have been left to us. Especially must it include something of the problems which have been left as an aftermath of the advent of the era of the sulphonamides and the antibiotics.

First, I must mention Sir William Macewen of Glasgow, the real Father of Modern Aseptic Surgery. It was he who maintained that he was a physician condemned to the practice of surgery. This philosophical approach to surgery was the culmination of a lifetime of research and experience. It explains, too, his advocacy of the reliance we should place on *vis medicatrix naturæ*—though some of that faith may have derived from his experience as a house-surgeon in Lister's wards in the days of "laudable pus". As students we realized that his serene composure was the product of both self-reliance and self-sufficiency. He was in himself his own complete research institution and his own craftsman. His operating table, for example, was made entirely of wood and to his own design. No modern gadget in stainless steel could do more to help a surgeon's needs than could Macewen's wooden table. His instruments were also made to his own design and had to comply with his high and severe standards.

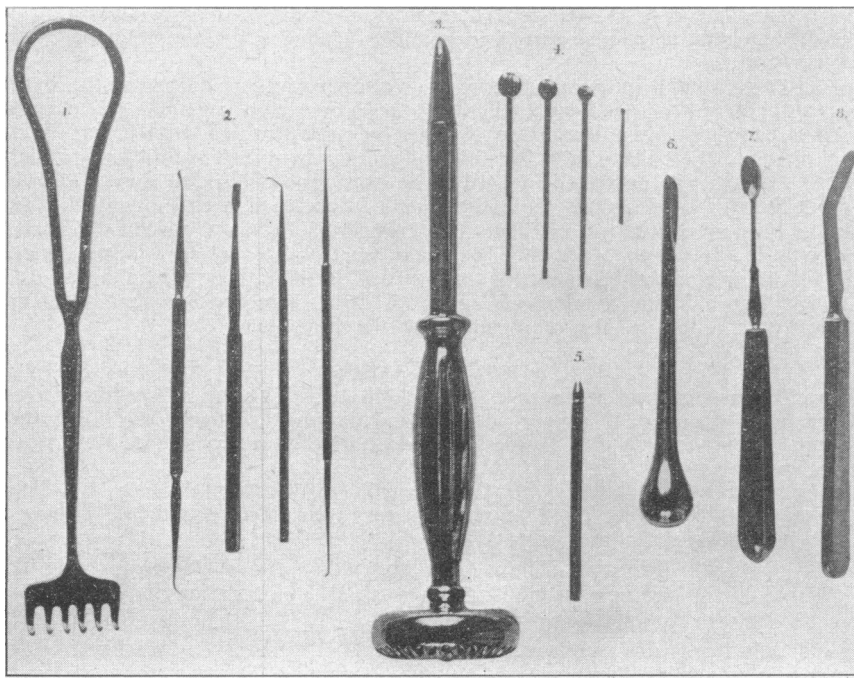


FIG. 1.—Sir William Macewen's instruments for mastoidectomy.

Fig. 1 looks like a tray of instruments set for the modern fenestration operation. It is, in fact, a photograph of the instruments Macewen used for the mastoid operation. It includes a retractor (1); a set of searcher and scoop, chisel, single and double ossicle hooks (2); a bone perforator—I believe Russian in origin (3); a set of rotatory burrs (4); a cerebral exploratory cannula (5); mastoid gouge (6); Volkmann's spoon (7), and a periosteal elevator (8). The photograph was published originally in Macewen's book on "Pyogenic Infective Diseases of the Brain and Spinal Cord" (Glasgow, 1893). It is apt to be forgotten in this era of antibiotics that Macewen's results in the surgery of brain abscess had not only not been surpassed, they had never been approached until the advent of the sulphonamides and penicillin.

The late Dr. Albert Gray's fine collection of the fragile specimens of the actual membranous labyrinth of animals, prepared by himself, is unique and it is still to be seen in the Hunterian Museum at Glasgow University. Members will recall the beautiful and more permanent collection of casts of the cochlea and labyrinth prepared in the family tradition by his son, Dr. Oliver Gray, and shown here a year or so ago. Albert Gray's work on the histopathology of otosclerosis was outstanding, and his "Atlas of Otology" (Glasgow, 1924, 1933) is one of the classics of the specialty. His help and encouragement to younger specialists must be remembered and treasured by many members of this Section.

Albert Gray and J. S. Fraser both taught that in the more advanced stages of otosclerosis some degeneration of the cochlear branch of the VIII nerve occurred. It is possibly too early to ask the question, but I think those of us who are doing the fenestration operation must one day be prepared to say what does happen to the VIII nerve some years after the operation has been done.

J. S. Fraser will be remembered for his work on the histopathology of the labyrinth, which justly brought to his Edinburgh clinic visiting otologists from all parts of the world. His knowledge of congenital deaf-mutism was encyclopædic, as was also his insight into the breeding habits of the white bull-terriers which were the material for much of his work on the subject. His performance of the radical mastoid operation was a work of art and called for just as much concentration. His demands on his willing staff were equally exacting. I think I am right in saying that he shares with G. J. Jenkins the honour of being among the first to think of and to try the fenestration operation. That was in the days long before penicillin, and so their efforts were almost doomed to failure. The conception of the possibility was there, however, and they both deserve our remembrance.

I recall Fraser's long discussions on the types of labyrinthitis which could occur in chronic otitis media, the finer points in the diagnosis of each type of labyrinthitis, the indications for the Hinsberg operation and for the translabyrinthine drainage of West and Scott. There were, too, his discussions on Gradenigo's syndrome and petrositis. I doubt if anyone sees these operations done now, nor are they indicated to-day. It is even questionable, as Donald Watson pointed out in his Presidential Address (1948, *Proc. R. Soc. Med.*, **41**, 155) whether the labyrinth, in an early labyrinthitis associated with chronic otitis media, should be disturbed at all by having either the cold caloric test or the rotation test performed.

For Dan McKenzie I had a special regard: he was not only a mighty debater but also a persuasive teacher and was prepared to spend much time and trouble to help beginners. The great example of his method of exposition was, of course, his paper on cholesteatoma (McKenzie, 1931).

In 1939 my old chief and colleague, T. Ritchie Rodger, was President of this Section. His original paper on noise deafness was published in 1915. The hearing-tests he used were done with tuning-forks and monochord, for, of course, the audiometer had not even been thought of. Nevertheless his observations have stood the test of time. His Presidential Address was on deafness caused by syphilis. He reported 500 cases of syphilis of the ear, nose and throat seen in his clinic in the ten years, 1925–1934, an average of 50 a year for that period. By 1938, the yearly average of these cases had fallen to less than half that number, i.e. under 25. As I succeeded Ritchie Rodger in the same clinic, the follow-up can be considered comparable for the same area.

Syphilitic Deafness

Since the war I have observed in the seven years 1946–1952, 71 cases of syphilis of the ear, nose and throat, i.e. an average of 10 a year—a most encouraging heritage when compared with the figures of twenty years ago. Of the 71 cases, all had positive Wassermann and Kahn reactions. In 30 cases the ear was involved.

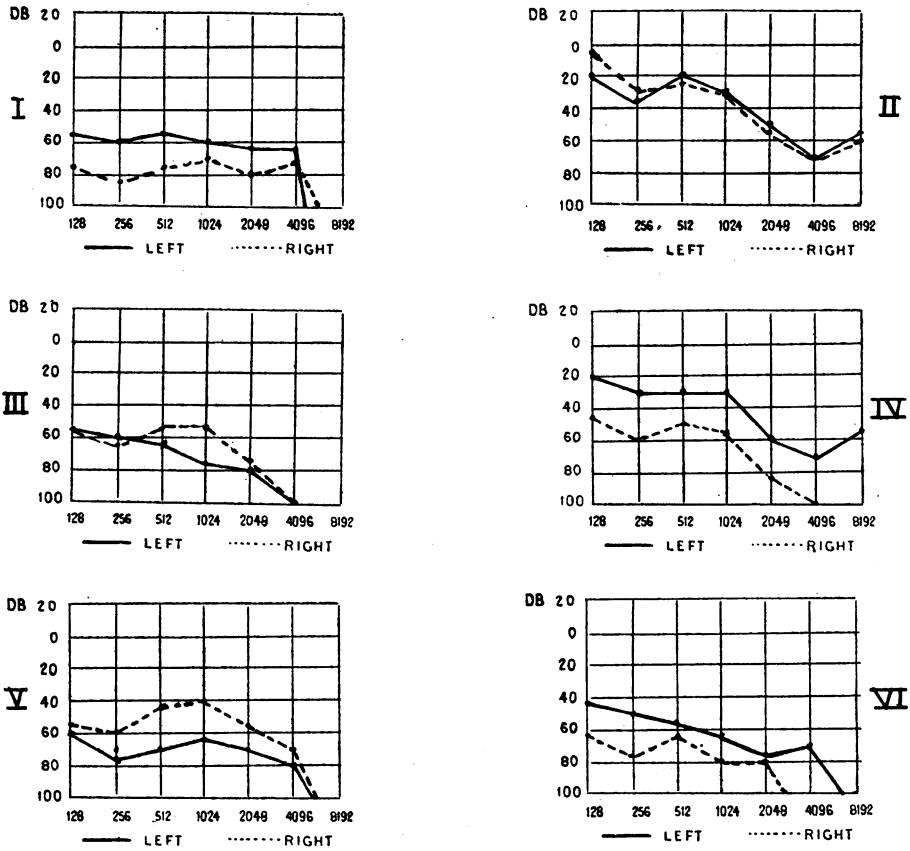
The incidence as between the sexes was about equal, 16 males and 14 females. There were 7 congenital cases and 23 acquired. In 25 cases both ears were affected and in 5 only one ear was involved. The presenting symptoms were:

(*a*) Deafness .. .. 18 cases
(*b*) Deafness, vertigo and tinnitus .. .. 7 cases
(*c*) Otorrhœa.. .. .. 5 cases

Of the acquired cases it was possible to record audiograms in 37 ears. On looking over these audiograms, it occurred to me that they showed features in common. Usually the audiogram shows a decline to the right with a tendency to a sharp loss of the frequencies above 2048. Another feature

which impressed me was that the lower tones showed a gradual decline up to the frequency 256 but at the frequency 512 the loss was not so marked; beyond that frequency the decline continued up to the sharp dip or absolute loss for the upper tones. Six illustrative audiograms are shown.

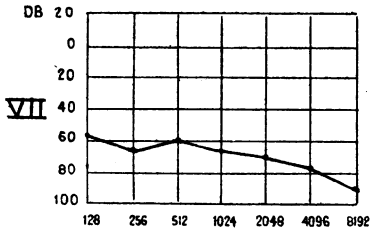
AUDIOGRAMS I–VI



I. Female, aged 37. Deafness.
II. Male, aged 73. Deafness.
III. Male, aged 69. Deafness and C.S.O.M.
IV. Male, aged 67. Deafness.
V. Male, aged 72. Deafness.
VI. Female, aged 57. Deafness.

I took the average of all 37 audiograms at the various frequencies and plotted an average (Audiogram VII). This audiogram confirms the impressions I have mentioned.

AUDIOGRAM VII



VII. Average of 37 audiograms of syphilitic deafness—acquired.

The variation in the decline shown in this average audiogram may be of some significance. I would not say that such an audiogram is diagnostic; but I feel that if, in an unusual case of deafness, we come across an audiogram which shows something of that pattern, syphilis as a cause of the deafness should be suspected.

Perhaps I have said enough about the distinguished members of the Section of the past who have come within my own personal experience to illustrate the fact that we have inherited a record of very considerable achievement in British otology. I turn now from the personalities to the problems which may be regarded as part of the heritage of British otology. It will not be possible to refer to all of them but I shall discuss some of those which are of particular interest to me.

Chronic Suppurative Otitis Media

Despite penicillin and the sulphonamides, chronic otitis media is still met with every day by the otologist. It used to be regarded as a reproach to otology. Is this allegation still true? I have considered some of my figures of the condition in the pre-penicillin and the post-penicillin years. As a pilot scheme I have compared a short series of cases where the percentage and not the actual numbers was important—I wanted the impression rather than the statistics.

I have compared a series of 121 cases of chronic otitis media from the year 1933 with 89 of another series which I saw in 1946. In 1933, 56 of the 121 cases resisted all forms of conservative treatment and came to the radical mastoid operation, i.e. 46% of the series. In 1946, 24 of the 89 cases in the series resisted all forms of conservative treatment and came to operation, i.e. 27%. Of the 56 mastoid operations in the 1933 series, cholesteatoma was found in 25, i.e. in 44·6% of the mastoid operations for chronic otitis media. In 1946, of the 24 mastoid operations, cholesteatoma was found in 21, i.e. in 87%.

It is interesting—and salutary—to keep a constant check on one's accuracy in observation. In these two series of cases I wanted also to find out how often I had recorded in my Out-Patient Department notes whether I thought, on first examining the patient, cholesteatoma was present or not. This exercise does impose a very desirable and fairly strict discipline on one's handling of a new patient. Such a regimen is necessary in these days of unlimited aids to a diagnosis which could possibly be made on observation alone.

In 1933 I noted that I suspected cholesteatoma in 21 of the 121 cases. Of these 14 came to mastoid operation and in 11 of these cholesteatoma was present. That is, my observation was confirmed in 78·5% of the cases where confirmation was possible. In 1946, I noted my suspicion of cholesteatoma in 29 cases. Of these, 19 came to the mastoid operation and in 18 cholesteatoma was found. That is, the observation was confirmed where confirmation was possible in 94·7% of the cases.

Nager in 1925, in a large series of cases, found that 14·5% of ear, nose and throat out-patients were cases of chronic suppurative otitis media, and that of these one-third showed cholesteatoma. I wanted to compare that with the figures of the Edinburgh Royal Infirmary of about the same period and so I referred to a statistical survey in which I shared in 1927. To my surprise, there was no mention of cholesteatoma in the report. In a recent survey of 3,397 cases of chronic suppurative otitis media, Scott Stevenson (1949) found that only 106 required the radical mastoid operation, and of these only 37 had cholesteatoma, i.e. 35% of the radical mastoid operations; his figures for the incidence of cholesteatoma at operation are, therefore, the same as Nager's. In a still more recent series of cases, Hughes and McKenzie (1953) reported 28 cases of cholesteatoma in 41 mastoid operations for chronic otorrhœa, i.e. 68·3%. I felt certain that the figures of the pilot study I have mentioned represented fairly accurately my post-war experience. But to obtain the actual position I have reviewed 250 consecutive mastoid operations performed by me since 1946 for chronic suppurative otitis media which had resisted all kinds of conservative treatment, an average of just under 34 such operations per annum. At operation I found 218 mastoids showed cholesteatoma to be present, i.e. 87·2%. In theory that is what we should expect for, with the help of the antibiotics and the sulphonamides, we should be able to eliminate the majority of the straightforward infections of the middle ear and mastoid, provided cholesteatoma is not present.

As long ago as 1881, an American, Randall, asserted that "the cause of chronic or recurrent otorrhœa is cholesteatoma—a rule having few exceptions" (Randall, 1929). I am certain that no British otologist at that time, nor indeed even up to 1945, would have agreed with him. But when it is found that cholesteatoma is present in 9 out of every 10 mastoidectomies for chronic otitis, I confess that I am now bound to agree with that elderly American.

Cholesteatoma

Since cholesteatoma figures so largely as a cause of resistant chronic otitis media and as the nature of the condition is still a mystery, some of the details in the series of 250 radical mastoids are of interest as a record of what I think is the present position. I shall deal with a few of the practical

points in diagnosis, in an attempt to assess their relative clinical value, and then I shall discuss my conception of the theory of the origin of cholesteatoma.

TABLE I

250 *Mastoid Operations for Chronic Suppurative Otitis Media*

Type of mastoid. *Acellular*	218 (87%)
Cholesteatoma found in	197
(i.e. in 90·4% of the acellular mastoids)	
Type of mastoid. *Cellular*	32
Cholesteatoma present	21
(i.e. in 65·6% of the cellular mastoids)	

In 116 cases the right mastoid was involved and in 134 the left.

While the advent of the antibiotics and the sulphonamides has relieved us of a great deal of mastoid surgery, it has left us one curious legacy. It is something of a paradox to realize that the mastoid surgery which we now do is technically more difficult. George Seed made some reference to this in 1950, when he drew attention to the small number of cortical mastoidectomies now done and the resulting loss of training opportunities for our registrars and house surgeons. The main type of mastoid operation now undertaken is for chronic otitis media. It is my experience since the war that of 250 mastoids opened because of chronic otitis media, 218 were acellular in type, i.e. 87%. When we remember that in the acellular mastoid the bone is densely sclerotic, the sinus is far forward, the dura is low, the antrum is deeply placed, the bone over the facial nerve is brittle and fractures easily, and that we cannot expect to find the mastoid with lots of elbow room for the beginner, we must surely feel a great sympathy for the trainee. But when we add to all these frightening thoughts the difficulty, for the beginner, of the endaural approach, then the prospect is truly alarming. There is little wonder that McGuckin emphasizes the absolute necessity for excellent lighting and adequate magnification in endaural surgery. Those of us who trained in the days of multiple emergency acute mastoids of the spacious type must surely conceive it our duty to see to it that the trainees of to-day have all the care and the time that we can find spent on our own personal supervision of their training.

Radiological findings.—The radiologists committed themselves to an opinion on the presence or absence of cholesteatoma in 203 instances. In 72 cases (35·5% of the total) they said there was radiological evidence of cholesteatoma and in 62 of these cases, cholesteatoma was found at operation, i.e. they were right in their positive findings in 86%. In 131 cases (64·5% of the total) where they found no radiological evidence of cholesteatoma, cholesteatoma was found at operation in 114, i.e. in 87% of their negative findings they were wrong.

Clinical findings.—Clinical examination showed the presence of cholesteatoma in 185 cases (74%) and in 174 of these, cholesteatoma was found at operation, i.e. the clinical observation was correct and was confirmed in 94%. Of the 65 cases where cholesteatoma was not noted clinically, 44 showed cholesteatoma at operation.

TABLE II.—SITE OF PERFORATION AND INCIDENCE OF CHOLESTEATOMA

1. *Attic perforation.* 85 cases (34% of total). Cholesteatoma present in 80.
   (94% of the attic perforations which came to operation)
2. *Polypi or granulations filling the meatus.* 61 cases (24·4% of total). Cholesteatoma was present in 45.
   (73·8% of this type which came to operation)
3. *Postero-superior quadrant.* 50 cases (20% of total). Cholesteatoma present in 49.
   (98% of this type which came to operation)
4. *Posterior half of membrane.* 36 cases (14·4% of total). Cholesteatoma present in 29.
   (80·55% of this type which came to operation)
5. *Attic and aditus erosions combined.* 18 cases (7·2% of total). Cholesteatoma present in 15.
   (83·3% of combined erosions which came to operation)

The percentage incidence of cholesteatoma given in Table II is, of course, in relation to the site of the perforation in those cases which came to the radical mastoid operation and not in relation to

the site of the perforation in chronic otitis media in general. It does probably bear some relation to the more general incidence; certainly the figures show clearly the high incidence of cholesteatoma in association with the postero-superior perforation (98%) and the attic perforation (94%).

Serum cholesterol.—Attempts have been made in the past to diagnose the presence of cholesteatoma by chemical tests on the aural discharge. There has been no recent report in the literature on these tests and so far as I am aware they have been abandoned. The biochemistry of cholesterol is as yet imperfectly understood, but it has been assumed that there is some relationship between the high serum cholesterol and the incidence of cholesteatoma. In this series of 250 mastoidectomies, the serum cholesterol was estimated in 81 cases.

TABLE III

1. In 65 cases the serum cholesterol was high (above 220 mg./100 ml.). In 60, cholesteatoma was found at operation (92%)
2. In 13 cases the serum cholesterol was normal (180–220 mg./100 ml.). In 12 cholesteatoma was found at operation (92%)
3. In 3 cases the serum cholesterol was low (below 180 mg./100 ml.). In all 3 cases cholesteatoma was found at operation.

I conclude, therefore, that while a high serum cholesterol may be of some value as confirmatory evidence of the presence of cholesteatoma, it is no certain indication of its presence; nor is it, in my experience, of any value at all as an indication either of the size or the extent of the cholesteatoma. My conclusions are at variance with those published by Bernovits in 1931.

Now I should like to discuss my own concept of the origin of cholesteatoma.

"BLACK" CHOLESTEATOMA

It is conceivable that the increased cholesterol content of the blood or serum is a pathogenic factor in the production of cholesteatoma. This brings me to the consideration of another aspect of cholesteatoma, and one which I have not heard discussed. It is, from its appearance at operation, what I have called "black" cholesteatoma. In the series of 250 mastoids, this condition was present in 6; in 1 patient it was bilateral. In all 6 cases the mastoids were cellular. In only 1 case was there

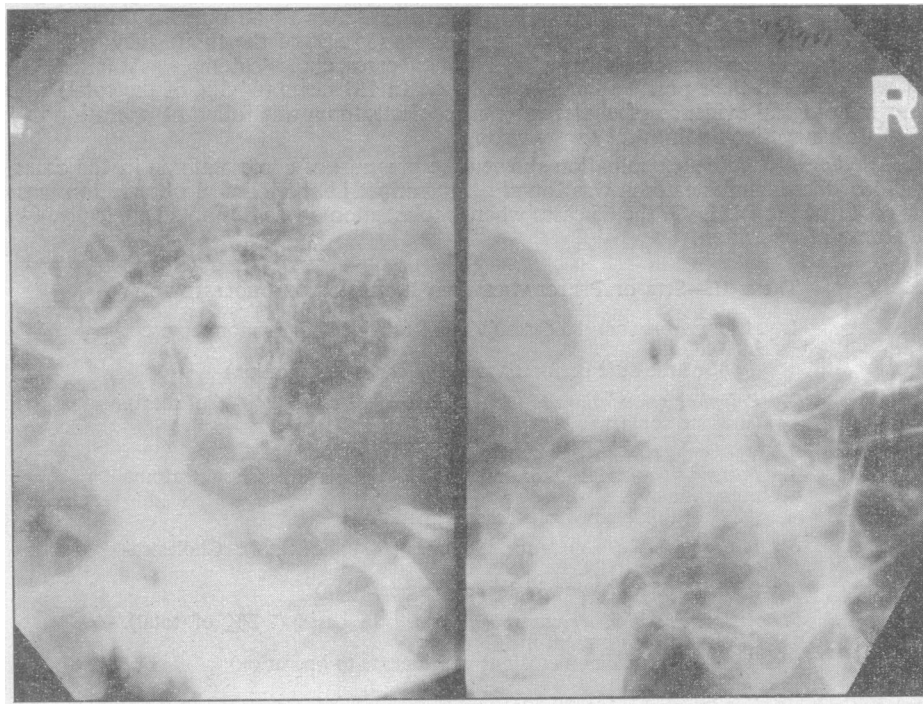


FIG. 2.—Extensive pneumatization of left mastoid. Right mastoid appears to be much less cellular, but at operation it was found to be very cellular.

any indication in the middle ear that such a condition might be present in the mastoid. In this case the middle ear was filled with what appeared to be—seen through the membrane—a blue-black fluid, the "blue" drum, which was thought to be an old hæmorrhage. Douglas Ranger in a recent paper on "idiopathic hæmotympanum" has discussed two similar cases. The details of my own case are:

Male, aged 27.—Recurring chronic otitis media (right) which began after swimming fourteen years ago. Present recurrence began with earache three days previously. There was a slight blood-stained discharge. Examination of the right ear showed what appeared to be the remains of a hæmorrhagic bulla over Shrapnell's membrane and a blood-stained effusion in the middle ear. The membrane was not bulging.

The condition was thought to be an influenzal type of acute otitis media and he was given a full course of penicillin by injection and sulphatriad by mouth. He was kept under observation but after a month the appearance of the membrane and the middle ear had not altered materially, except that a small pin-point perforation was seen in the posterior half of the membrane. The otorrhœa was always slight and there was never enough effusion in the middle ear to make me feel justified in either aspirating or incising.

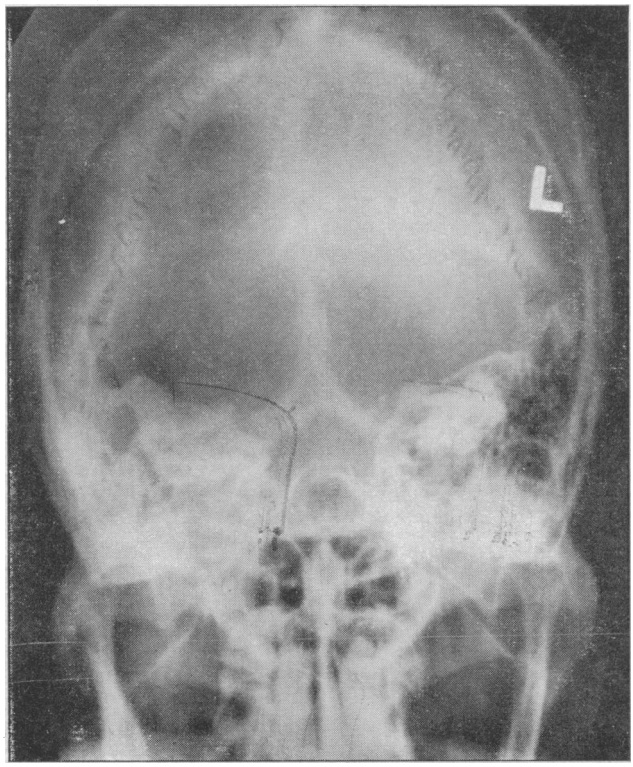


FIG. 3.—Deep extension of cells into petrous shown on left side. Similar extension of cells into petrous in right side found at operation.

At this time the radiologist's report on the mastoids was:

Right mastoid: Air cells are hazy and sclerosed and there appears to have been complete destruction of the attic.

Left mastoid: Air cells well pneumatized (*see* Figs. 2 and 3).

Four months after the onset, as there was no change in the condition of the middle ear or mastoid, he consented to operation.

At operation the cortex was found to be sclerotic but after removing this a very cellular mastoid structure was revealed. On this point I would make the same comment as Ranger, "it was a more cellular mastoid than appeared from the X-rays and there was no evidence of any infection". All the cells were filled with "black" cholesteatomatous material. There was a deep petrous extension of cells and they were filled with similar material. The attic was completely eroded and filled by the same material. On opening the middle ear black tarry fluid exuded. The ossicles were buried in this

material but showed no macroscopic change. Similar material was found in the eustachian tube end. Areas of the cell structure showed breaking of the intercellular trabeculæ but there was no eburnation of bone. The material was easily scooped out of the cells and, like the usual cholesteatoma, showed a pearly sheen where it had been attached to the bone. The blood in the mastoid cavity during the operation showed the characteristic oily appearance on the surface that one associates with cholesteatoma.

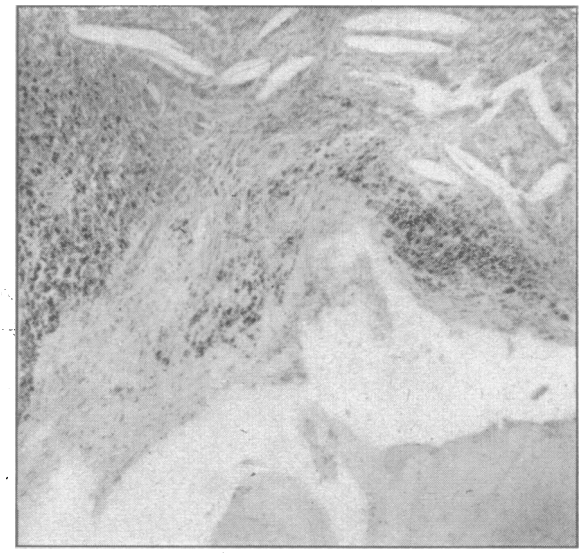


FIG. 4.—For description *see* text.

Pathological report (*No.* 1).—The specimen consists of a fragment of bone with some granulation tissue on its surface. The granulation tissue contains numerous cholesterol clefts and a fair amount of blood pigment which stains positively for iron. The granulation tissue is covered with cuboidal epithelium which does not show any tendency to squamous metaplasia (Fig. 4).

Pathological report (*No.* 2).—The section shows a fibroblastic granulation tissue in which there are numerous cholesterol clefts and scattered chronic inflammatory cells. Large collections of brown pigment, much of it intracellular within histiocytes, are also present. In the lower right-hand corner there is a fragment of bone showing osteoclasis (Fig. 4).

These reports correspond also to the findings in the two cases reported by Ranger.

There are two points in such a case which require some elucidation. The first is the possible source of the blood and the second is the source of the cholesterin crystals. It is possible that the correct explanation of both mysteries may help towards the explanation of the origin of cholesteatoma. Ranger found no satisfactory explanation for the origin of the blood in either of his cases and it is his opinion that the amount of blood was the result of repeated hæmorrhages. There was no obvious source in my case and I would agree that the amount present and the state of breaking down of the blood found suggests that more than one hæmorrhage had occurred, and over a long period. But how did these hæmorrhages arise? Going back to the history in my case, I noted that the patient sought advice because of earache occurring in an ear which had been discharging on and off for fourteen years. My first impression on examining the ear was that I was dealing with an influenzal type of acute otitis media with the remains of a hæmorrhagic bulla over Shrapnell's membrane and a blood-stained effusion in the middle ear. It is a common experience that *Hæmophilus influenzæ* causes mucosal hæmorrhages. They may recur during the acute phase and then cease. The initial hæmorrhage in my case may therefore have been due to this cause. But why did they keep on recurring over a period of weeks, and long after the acute influenzal phase had passed? Here I think the presence of the cholesterol crystals may come in. It is well known that the separation out of cholesterin crystals may occur in an enclosed hæmorrhage. If we imagine cholesterin crystals present, forming in a very confined space such as the attic and in the cells of a very cellular mastoid, we can conceive the effect of the tissue reaction, as shown in Figs. 4 and 5, on the cell mucosa and on the intercellular bone trabeculæ. The result we can well imagine would be small and recurring hæmorrhages from the mucosa and even from the intercellular trabeculæ, due to bone absorption. This happened to the cellular bone of the attic and to the cells of the mastoid in the case I have described.

As to the source of the cholesterin crystals, Ranger, in discussing this problem, concludes that they arise from the fluid filling the middle-ear cleft. This was shown to be altered blood, and it is established that cholesterin may crystallize out in this type of hæmorrhage. He also mentions a case of secretory otitis media on which a mastoid operation was done. Histological examination in this case showed cholesterin crystals in the mastoid but the amount was less than in the two cases of hæmotympanum. He makes the comment that this is in keeping with the fact that the cholesterol in serum is in a more soluble form than that in red corpuscles and is, therefore, less likely to crystallize out from the serum than from the blood.

But if the concentration of the cholesterol in the serum is greatly increased, it is conceivable that there would be a greater tendency for the cholesterol to crystallize out in circumstances such as I have postulated. In my own series of investigations, I find that in 45 cases where the serum cholesterol was considered to be unusually high, i.e. over 300 mg./100 ml., cholesteatoma was present in every case. There is the possibility, therefore, that in a patient who has a high serum cholesterol, deposits of cholesterin crystals could arise in any small serous effusion. Such effusions, as we know to our cost, can and do occur in the attic and the middle ear and the mastoid cells from various causes. It is thus possible that in such circumstances we have the beginnings of cholesteatoma. If this conception should prove correct, "cholesteatosis", as suggested by Gavin Young, would be a more accurate description, because the subsequent chain of events can be regarded as a tissue reaction to the presence of an endogenous foreign body—the cholesterin crystals. Such a tissue response would be a continuing one so long as the high level of the serum cholesterol persisted, because the serous effusion evoked by the tissue reaction would tend to produce still more cholesterin crystals. If, however, the serum cholesterol returned to normal, as we know it can do within a short period of time, the tissue reaction would be limited. An example would be those encysted attic cholesteatomas which have been in the past classified as "primary". Fig. 5 illustrates this point.

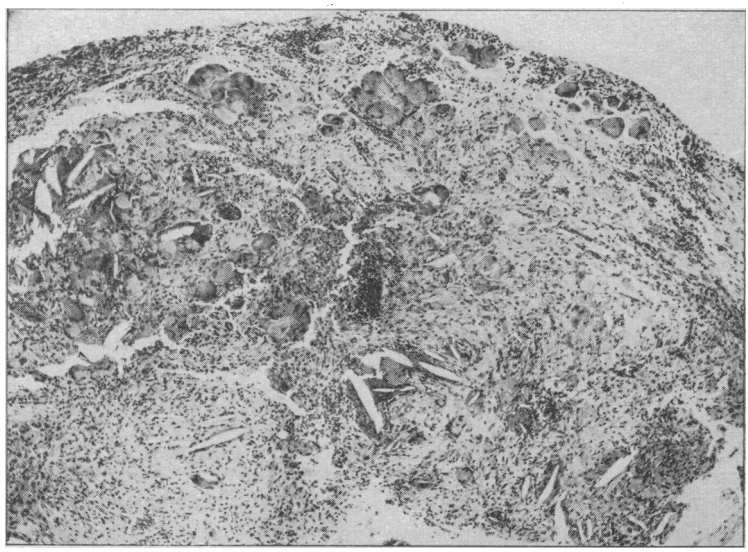


FIG. 5.—Section of small attic polypus showing cholesterin clefts with giant-cell activity very marked—the picture of the extrusion of a foreign body. (Section kindly lent by Mr. Guy Thompson.)

This conception has the advantage of being an explanation of the origin of all types of cholesteatoma, whether they are found in the attic, the antrum, the mastoid or even the petrous. It has the advantage, too, of dovetailing with all the points Dan McKenzie made about cholesteatoma. I shall consider only 3:

(1) In his conception of cholesteatoma being a primary condition, he stressed that "cholesteatoma may be found without any membrane whatever". If the conception that cholesteatoma begins as a tissue reaction to cholesterin crystals is accepted, then the question of a membrane being present is a late and a secondary matter.

(2) "The lining is sometimes found when examined microscopically to be devoid of epidermal cells." In the view I have expressed no epidermal cells need be present at all unless as a tissue response and then only as a tissue response of the lining cells. Here would arise the so-called metaplasia, the changing of the simple cuboidal epithelium to the keratinized, resistant, epidermal cells.

(3) "It is noteworthy that the normal tissue, bone and connective tissue, beneath the cholesteatoma in the sections shown, betray no evidence of any inflammatory reaction"—the simple explanation being that in essence it is not an inflammatory reaction. Inflammatory reactions are found in many sections of cholesteatoma but they are due to secondary infections occurring after the initial cholesteatomatous change has started.

He maintained, too, that however large the cholesteatoma, it always presents a nidus, a point of origin, and that a closed cholesteatoma was devoid of fœtor. Thus he came very near to my conception of what happens but because of his preoccupation with the supposed function of the epidermal cells in the production of cholesteatoma, he failed to find a satisfactory explanation. "The epidermal cells", he said, "may, like osteoclasts, exercise a chemically solvent action on the bone upon which they are lying". But he thinks this conception is negatived by the fact that the living epidermal cells are not in immediate contact with bone, the fibroblastic layer lying between them. In my view, it is, of course, the cholesterin and not the epidermal cells which originate this action. Then he goes on to quote Kirchner on a point which I think adds some weight to my conception. Kirchner showed sections in which both epidermal cells and cholesteatomatous detritus were inside the Haversian canals of the bone in the neighbourhood where their action, whether mechanical—as Kirchner held, or biochemical—as I conceive it to be, would certainly lead to bone absorption.

The interesting practice of Sir James Dundas-Grant, of leaving the lining of the cholesteatomatous cavity instead of removing it, raises the question for those who maintain the epidermal metaplasia view, why were some of these cases of his so successful? If the epidermal cells were responsible for the cholesteatoma, they should have continued to produce it. On the other hand, if the epidermal cells are a direct response of the tissues to the presence of cholesterin—a protective response—then removal of the cholesterin should allow the epidermal cells to revert to their original function, and hence they will become true epidermis. This explains, too, the dry cavities seen on occasion when Nature herself has completed the radical mastoid operation.

It may be objected that I have not shown a high serum cholesterol to be present in every case of cholesteatoma. But I do not consider it necessary to do so. Little is known of the biochemistry of cholesterol. It varies within fairly wide limits in the normal person, but in some people it is persistently high. It should only be necessary to show that when the effusion first starts the serum cholesterol is high. But the case may only be seen months or years after cholesteatoma has been established, by which time the serum cholesterol may have returned to normal or even less. Neither pain nor deafness need be present in an attic effusion, and so it is unlikely that the patient will be seen in the earliest initial stage.

It is a line of investigation which will take a considerable time and much patience, but I feel it will be rewarding. If some such theory can eventually be established, then clearly, so far as we can judge at the moment, cholesteatoma cannot be prevented. The high incidence of cholesteatoma found in my series of mastoid operations for chronic otitis media—9 out of every 10 cases—would support the view that we are now able to deal conservatively with almost all cases of chronic otitis media which can be expected to respond to treatment, and that the failures in conservative treatment are due to the presence of cholesteatoma. Operation on these cases is inevitable in our present state of knowledge of the condition.

If it is conceded that in modern otology the failures in the treatment of chronic otitis media are due almost entirely to the presence of cholesteatoma, and if it is agreed that cholesteatoma is not only not preventable, but that when it does occur, operation is inevitable, then I think I have answered the question I posed at the beginning. We may claim that there is no longer any justification for the allegation that chronic otitis media is a reproach to otology. That is part of our heritage in which we can take some pride.

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