PRE-EPIDERMOSIS

By A. TUMARKIN (Liverpool)

THE word Cholesteatoma is deeply embedded in Otological literature and is generally regarded as signifying a definite clinical entity. Unfortunately that is not strictly correct. Not only is there controversy as to its ætiology but there is ambiguity with regard to its histological characteristics.

Two main points call for discussion:

(a) Is it possible to demonstrate true stratified epithelium?

(b) Is the presence of cholesterin crystals an essential feature?

With regard to (a) informed opinion today is unanimous that stratified squamous epithelium *can be* demonstrated. There is, however, great controversy not only as to the *origin* of this stratified squamous epithelium, but also the means whereby it enters the middle ear.

As to (b) it cannot be too strongly emphasized that Cholesterin has nothing whatsoever to do with Cholesteatoma. Fig. 1 shows the classical picture of Cholesteatoma—the amorphous debris, the keratinized layers and the stratified prickle cells. Fig. 2 shows an entirely different entity. The outlines of the Cholesterin crystals stand out clearly and in addition giant cells, granulation tissue and blood pigments can be seen.

These are entirely different histological entities, and yet the word Cholesteatoma is regularly used to denote either or both of them. Friedmann 1959, has drawn attention to this confusion and has proposed to clarify matters by denoting the first "Epidermoid Cholesteatoma" and the second Cholesterol Granuloma.

The term "Cholesterol Granuloma" is scientifically accurate and highly evocative. It denotes a condition characterized by cholesterin, granulation tissue and giant cells. The term "Epidermoid Cholesteatoma" is, however, open to criticism since the word "Cholesteatoma" carries with it all the vague suggestions of Cholesterin and of neoplasm which we are anxious to eliminate. In the writer's opinion the word "Epidermosis" is preferable denoting as it does a "process" rather than a preformed condition.

Definition. Epidermosis of the tympano-mastoid cavity is a pathological process in which squamous epithelium of *epidermic origin* grows into that cavity.

By this definition we exclude two other theories of origin, viz.:

(1) The epiblastic theory which postulates congenital inclusions of epiblast, i.e. dermoid (or epidermoid) tumours. That such congenital

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inclusions are possible cannot be denied but they are rare curiosities. In most cases, Epidermosis develops post-partum by an invasion of epidermis.

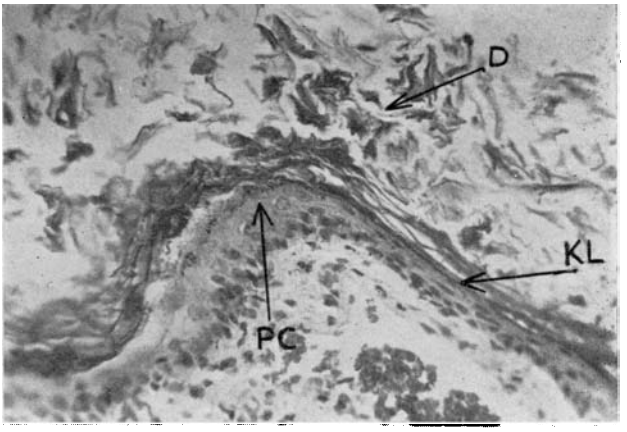


FIG. 1.
EPIDERMOSIS.
Showing prickle cells (PC), Keratinizing layers (KL), Debris (D).

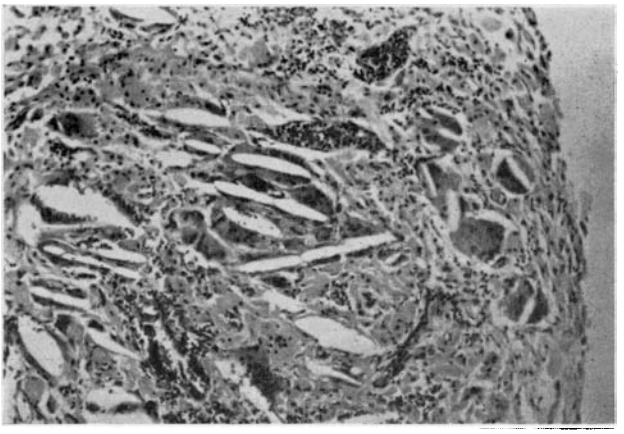


FIG. 2.
CHOLESTEROL GRANULOMA.
Showing typical clefts, giant cells, pigments and granulation tissue.

(2) The metaplasia theory. This in effect says that the stratified squamous epithelium is *not epidermic*, being derived from a transformation of the endothelial lining of the middle ear cleft.

Here again it must be conceded that metaplasia is a theoretical possibility.

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The normal mastoid is lined by a single layer of flattened cells. In acute mastoiditis these change into ciliated columnar cells. This itself is metaplasia and there is no reason to deny that a further transformation from ciliated columnar into squamous epithelium might occur just as it does in the nose, the bronchi and elsewhere. The possibility of this sort of two stage Metaplasia has been supported in one form or another by many workers (including the writer) ever since it was originally proposed by Wendt, nevertheless, no unequivocal evidence of its existence has so far been adduced and it is now generally agreed that the stratified squamous epithelium is derived from the skin of the meatus and tympanic membrane by a process of immigration.

The concept of immigration is, of course, one of the classical theories but on closer analysis it turns out to be far from simple. There are at least five different ways in which immigration is believed to occur and the distinction between them is no mere academic exercise—it has profound practical implications.

Immigration Theories

(a) *Mechanical.* Seventy years ago Bezold (1891) (and later Wittmaack (1918)) suggested that as a result of chronic tubal catarrh the drum collapses and forms a deep blind sac in which products of desquamation tend to accumulate, finally invading the middle ear cleft.

(b) *Inflammatory.* About the same time Habermann (1888) suggested that an acute necrotising otitis media could destroy the drum right down to the annulus after which the meatal epidermis could invade the middle ear. Even in those days, however, it was recognized that this could not explain the more common case characterized by an insidious course and a small marginal perforation.

(c) *Tissue Tension.* During the last decade Eigler (1949) has drawn attention to the peculiar histological features of the skin of the deep meatus—not only a tendency to hyperkeratosis of the stratum corneum, but also a tendency of the basal cells to form solid cones which proliferate inwards. This "Wachstum potenz" theory has been taken up vigorously by many workers. Schroer (1956) claims that it is quite capable of producing a cholesteatoma in the absence of any inflammation whatsoever. Rüedi (1957) modifies this extreme attitude by suggesting that the invasion is more likely to occur if the epitympanal spaces are incompletely pneumatized and that it is usually triggered off by an acute otitis media in infancy. Both these authors point out that this proliferation of basal cells could theoretically produce Cholesteatoma *behind an intact tympanic membrane.* Rüedi, however, adds (*loc cit.* p. 301), "It is unfortunate we do not yet have histological specimens of acute otitis media in which proliferated cones of basal cells have penetrated the connective tissue and

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formed a cholesteatoma. Histological proof of an inflammatory development of a cholesteatoma within an intact Shrapnell's membrane is therefore still lacking."

(d) *Keratosis Obturans*. McGuckin has recently suggested that in many cases the cholesteatoma is a direct result of the accumulation of Keratosis Obturans, a process which Berberich attempted to produce experimentally many years ago.

(e) *Trauma*. Lastly for completeness we note traumatic cholesteatoma which arises following fracture of the temporal bone in which skin is included in the re-united fracture line.

In the writer's opinion the above theories are all open to criticisms even though they all embody the accepted immigration principle. For example acute necrotising cholesteatoma is rarely encountered today and primary enclosed cholesteatoma is almost unknown. Thus these pathological processes can at most only account for a small proportion of the whole. Simple mechanical collapse of the drum is well known, but that of itself is not sufficient to cause epidermosis, whilst the idea of invasion because of unbalanced tissue tension leads us nowhere unless we can explain why the imbalance occurs.

Similarly the theory of accumulating Keratosis leads us nowhere unless we can explain *why* the Keratotic accumulation occurs.

Nevertheless all these ideas are fruitful and in combination yield a plausible theory which not only explains the essential pathological features but leads to positive lines of treatment.

A Synthetic Theory of Epidermosis

We start from the mechanical theory of Bezold. In the course of the upper respiratory infections which are so common in childhood, Eustachian tube obstruction occurs, causing intratympanic vacuum and collapse of the drum head, either in the post superior quadrant, or in Shrapnell's membrane. Dry collapse of the post superior quadrant is a well recognized clinical entity. It is perhaps not so widely recognized that dry collapse of Shrapnell's membrane is *also extremely common*. These two conditions may persist throughout life causing little or no inconvenience. Such ears, however, are vulnerable since under stress they can break down and fall into Epidermosis.

Inflammation occuring in such a vulnerable ear will be *characteristically painless* (or comparatively so) since inflammatory products will seep away through the pre-existing fistula or will ulcerate and burst the atrophic drum without causing the distension of the drum which is the cause of Otalgia in acute inflammation of a normal middle ear.

Moreover, this seeping pus will set up an irritative response in the lining of the fistula leading rapidly to superficial osteitis of the annulus

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PATHOGENESIS OF EPIDERMOSIS.

Congenital Epiblastic	Metaplastic	Epidermic Immigration	(1) Tubal Obstruction (2) Necrotising Otitis (3) Wachstum Potenz (4) Keratosis Obturans (5) Trauma

The Epiblastic and the Metaplastic theories are to-day regarded as merely theoretical possibilities. The writer, having in the past supported metaplasia, now admits its unimportance and accepts epidermic immigration. This is regarded as a two stage process, i.e. Pre-Epidermosis and Epidermosis.

THE RELATION OF EPIDERMOSIS TO CHOLESTEROL GRANULOMA.

Diffuse Infection of Upper Respiratory Tract in Childhood

↓

Tubal Obstruction

↓

A PATHOLOGICAL SPECTRUM

Mechanical	Mixed Inflammatory and Mechanical	Inflammatory
↓	↓	↓
Collapse of Drumhead (pre-epidermosis)	Exudative Otitis Media	Acute Otitis Media
↓	↓	↓
EPIDERMOSIS	CHOLESTEROL GRANULOMA	CENTRAL PERFORATION

and the formation of granulations. The next stage is determined by the recognized forces of tissue tension. In the normal process of healing, whenever the skin surface is breached, granulations are thrown up and these are covered by ingrowing of the surrounding epithelium. This re-epithelialization is a primordial defence mechanism and its vital importance is revealed by the fact that it is one of the few examples of tissue regeneration found in higher organisms. It is easy to see how this powerful force would urge the hyperplastic epidermic elements of the fistula over the granulations and so into the tortuous spaces of the middle ear, so setting the stage for the final development of Epidermosis.

The key note of this theory is its emphasis on the preceding collapse as an essential preliminary. I regard this as one of the most important pathological conditions within the middle ear, and because of its importance I propose to speak of it as Pre-Epidermosis.

This theory is, in essence little different from the original theory of Bezold and Wittmaack. It is not incompatible with the tissue tension theories providing these are regarded as secondary contributory factors, which only come into action after the establishment of pre-epidermosis.

The conception of epidermosis as a "misplaced healing effort" was put forward by Habermann and is frequently rejected by modern workers who demonstrate how the epidermic elements seem to burrow *under the*

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mucosa in a pseudo-neoplastic manner. In reply we merely note that the "healing effort" is responsible merely for the initial entry of the epidermic elements—but once inside the middle ear cleft they are in abnormal conditions and it is not surprising that their behaviour is abnormal. Colman (1960) has produced remarkable experimental proof of this assertion in his studies of grafting at the oval window in the cat. When skin was used it produced the characteristic features of epidermosis in a startling fashion. It proliferated widely, burrowed under the mucosa and finally formed an epidermoid cyst which ruptured into the meatus to form a high postero marginal and attic perforation. This author adds: "There is little reason to doubt that typical cholesteatoma would finally have developed in these animals."

Thus the synthetic theory implies a two stage process

(a) A preliminary mechano-inflammatory process leading to Pre-epidermosis.

(b) An activating process leading to true epidermosis. This may be local inflammatory in the sense of an accumulation of squames within the epidermic sac or it may be true otitis media.

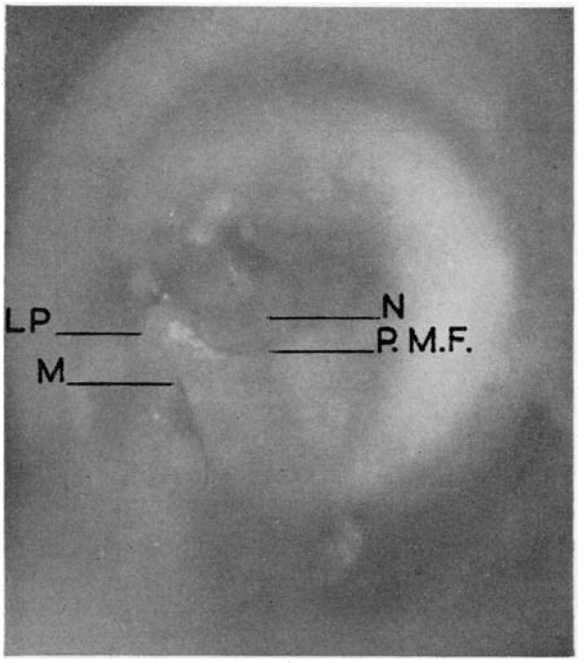
Evidence for the Synthetic Theory of Epidermosis

The most cogent evidence for this theory is in fact the widespread occurrence of "pre-epidermosis". This can be regularly demonstrated in patients who are entirely unaware of its existence. For example, in almost every case of active unilateral epidermosis the opposite ear will show latent pre-epidermosis. Moreover, cases in this latent condition, if carefully observed over a prolonged period, will be found occasionally to become activated. Conversely, if an active case of epidermosis is encountered at a sufficiently early stage and is treated correctly, the violent response can be cured and the condition returns to quiescent pre-epidermosis.

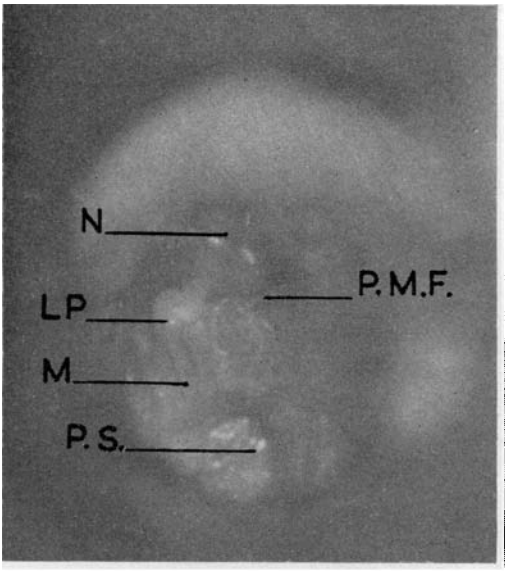
Again, this theory explains the enigma of the patient who presents with a fully fledged sentinel polyp in Shrapnell's membrane and established attic epidermosis who insists that his trouble only began a few weeks earlier with minimal discomfort and insidious discharge. Clearly this is no acute otitis in the ordinary sense of the word and the Otologist tends to discount the patient's story and to insist that chronic infection must have been present for very much longer than stated. We now see that the truth lies midway. The Otologist is correct in insisting that *some* pathology must have been present for very much longer, i.e. pre-epidermosis laid down probably in childhood. Whilst the patient is correct that the active process only started a few week earlier, i.e. epidermosis.

Lastly, the theory explains how an acute inflammation can *apparently* produce a marginal perforation and a collapsed drum. It says in effect that the collapse preceded the inflammation.

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A.



B.

FIG. 3.

PRE-EPIDERMOSIS. THE VULNERABLE EAR.

A shows the characteristic recession of Shrapnell's membrane. The posterior fold of the malleus stands out rigidly. The neck of the malleus can be seen receding into the attic.

B shows pre-epidermosis in *both* sites of election.

LP : Lateral process of malleus.
PMF : Posterior fold of malleus.
M : Manubrium.
PS : Post superior quadrant.

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All our understanding of the mechanics of the middle ear indicates that if the drum is normal an acute otitis media must *bulge* the pars tensa and cause a *central* perforation. The post superior collapsed perforation calls for the entirely different explanation outlined above.

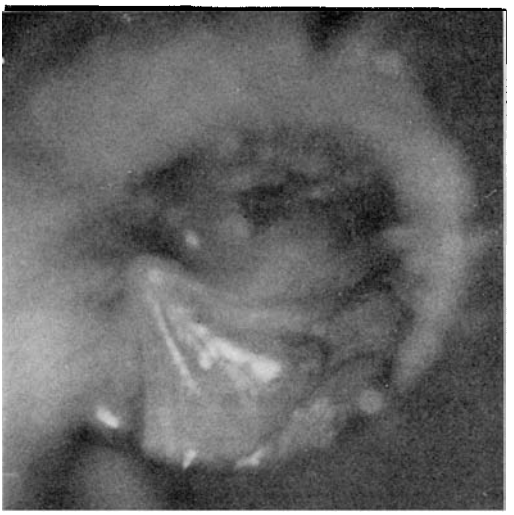
Epidermosis and Hypocellularity

I have shown elsewhere (Tumarkin, 1957) that hypocellularity is a stigma of the upper respiratory diseases of childhood and that it arises as a result of Eustachian obstruction and intra tympanic vacuum. We now see that Epidermosis arises in the same way and we naturally expect the two pathological conditions to occur together. In fact they do so quite commonly, nevertheless it would be wrong to expect that association to be invariable. Pneumatization starts at birth and may be well advanced within a few years. Respiratory infections are commonly delayed till the age of five or thereabouts. Thus a child may easily have developed a large pneumatic system before he develops pre-epidermosis. Indeed our recent investigations suggest that in childhood some degree of pneumatization is almost invariable. Where pneumatization is lacking and the mastoid is truly diploetic we deduce that pneumatization must have been frustrated at birth as postulated originally by Wittmaack.

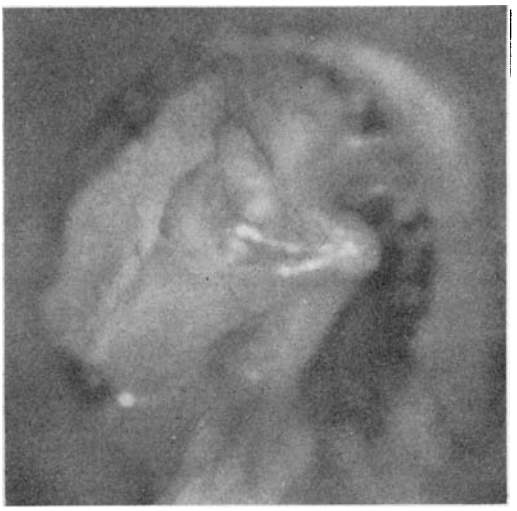
On the Nature of Cholesterol Granuloma

I have said that this condition shows no histological resemblance to Epidermosis. The fact remains that Cholesterol Granuloma and Epidermosis are frequently encountered side by side in the same ear, especially in children, and it is natural to inquire whether they may not share some common ætiological factor. In the writer's opinion that common factor is to be found in the familiar syndrome of the diffuse infection of the upper respiratory tract in childhood. In the course of that syndrome the Eustachian tube is very liable to become obstructed. The obstruction may be a purely mechanical phenomenon from adenoid hyperplasia, but more commonly there is an element of salpingitis as well. The final effect on the ear will be determined by the relative magnitude of the two factors, i.e. (1) Mechanical; (2) Inflammatory. When infection is violent acute otitis media develops. When mechanical factors predominate, the middle ear remains dry and the drum collapses. These, however, are merely the extremes of a pathological spectrum. Between them can be found all those varieties of Inflammatory reaction which go to make up the ill defined but vastly important clinical complex "enclosed tympano-mastoiditis". Under that heading I include the various manifestations of exudative otitis media. This condition may sometimes be limited to the middle ear but in most cases it is an exudative salpingo-tympano-mastoiditis. The mastoid air cells become more or less filled with a sullen exudate

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A.



B.

FIG. 4.
PRE-EPIDERMOSIS. THE VULNERABLE EAR.

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which defies the natural process of absorption and healing, because the obstructed tube will not allow air to re-enter and replace the exudate.

Thus the natural processes of absorption tend to reproduce the original vacuum, the sodden lining of the air cells tends to become vascular, to throw out granulations and to bleed. Thus in most cases of exudative otitis media subjected to mastoid exploration some degree of Cholesterol Granuloma will be found.

Surgical Implications of this Theory

Certain important principles emerge from this analysis.

(a) Exudative tympano-mastoiditis cannot be cured unless tubal function can be restored. Where the condition has gone on to Cholesterol granuloma it becomes necessary—unfortunately—to eradicate the pneumatic system in the mastoid and this calls for a meticulous cortical exenteration. It must be emphasized, however, that the most meticulous mastoid surgery will fail unless tubal function can somehow be restored. Thus, if we divide the auditory cleft into its tubal, tympanic and mastoid segments, each with its individual anatomical, functional and pathological characteristics we see that treatment of Cholesterol Granuloma calls for an attack on the tubal and mastoid segments whilst the tympanic segment should be spared as much as possible. It is true that myringotomy suction is an accepted and valuable line of treatment in exudative otitis media but this is not strictly an attack on the tympanic segment. Its success depends on breaking a vicious circle by removing "glue" and enabling the tube to resume its function, and it must fail if the mastoid segment contains Cholesterol granuloma.

(b) Epidermosis calls for a totally different approach, since attention must be concentrated on the tympanic segment. In the earlier stages it is easy to suck away the tiny sentinel polyp in Shapnell's membrane thus exposing the epidermotic fistula to the desiccation of insufflated Boric iodine powder. In later stages the epidermosis has advanced further into the attic and can only be laid bare by the removal of the outer attic wall by atticotomy.

Provided the mastoid segment is healthy this limited operation can yield gratifying results. For example when, as happens occasionally in adults, the mastoid is "ivory" the simple uncapping of the attic and antrum can yield a tiny healthy cavity within a few weeks (Tumarkin, 1947-1950).

Many writers have maintained (Walsh, 1951) that it is essential to remove the epidermic lining in all cases. This is far from correct. In advanced epidermosis radical surgery is often unavoidable. In the early stages of epidermosis, however, it is not wise to remove the matrix since the process of healing is only too likely to be accompanied by a regrowth of epidermosis into the exposed cavity.

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Pre-Epidermosis

Simple Marsupialization and subsequent desiccation will yield satisfactory results but at the price of perpetual vigilance. Patients who have undergone this form of surgery must be supervized regularly for fear of relapse. This is particularly so when the ossicles are thought to be viable and are preserved. The incus is particularly vulnerable and must be closely watched. There is no alternative. Pre-epidermosis can always flare up into Epidermosis.

(c) The combination of Epidermosis and Cholesterol Granuloma is of course the most challenging and implacable of all since all three segments are involved.

Unfortunately this combination is particularly liable to occur in children and the following considerations must be borne in mind.

(1) The disease is not infrequently bilateral.
(2) Even if the opposite ear does not exhibit active pathology it will almost certainly show pre-epidermosis with some loss of hearing.
(3) The infected ear may have reasonably good hearing.
(4) Unless surgery is planned carefully and carried out meticulously it is likely to result in further loss of hearing.

It would seem at first sight that nothing short of radical surgery could suffice and indeed that approach is still extensively used. The functional results of radical surgery are of course notoriously poor, and within recent years, high hopes have been entertained that better results might be achieved by means of the newer techniques of tympanoplasty.

Unfortunately those hopes have to a large extent proved illusory. However successful tympanoplasty may be in other types of tympano-mastoid disease it has little to offer in the more florid infections of childhood (Bosatra, 1958).

If there is active disease in the mastoid segment free skin grafts have little chance to survive. Moreover it seems curiously illogical to dissect out laboriously every speck of epidermis only to replace it by means of a free skin graft.

Within recent years, therefore, the author has utilized a combination of

(1) Exenteration of Cholesterol granuloma, plus.
(2) Marsupialization for the epidermosis.

Where the epidermosis has advanced into the antrum destroying the ossicles this combined operation turns out to be nothing other than a classical radical. In earlier cases, however, the posterior meatal wall can be preserved almost intact except in so far as it must be taken down to permit of marsupialization. Providing the cortical exenteration is adequate, the ossicles are viable and tubal function can be restored, it is possible to obtain gratifying results, but the mere statement of these provisos will indicate the enormity of the technical problem, once pathology is well established, and our thoughts naturally turn to the possibilities of prophylaxis. If we cannot *cure* Epidermosis should we not direct more of

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our energy towards *preventing* it. In that respect the concept of pre-epidermosis is of critical importance, leading as it does to certain definite lines of action. Our ultimate object is to prevent the development of Pre-epidermosis. This calls for an all out attack on the whole problem of the recurring infections of the childs upper respiratory tract, a problem of the utmost complexity with ramifications into the realms of virology, immunology, climatology, dietetics and so on. The Otologist has the humdrum—but vitally important and deeply satisfying role—of dealing with the ingravescent pathology as it manifests itself in these children often in their earliest years. This work—only too often cursorily carried out in minor ailment clinics is, in the writer's opinion, worthy of far more attention that it has so far received.

It is well known that the dramatic reduction in acute pyogenic ear disease that followed the introduction of anti-biotics has not been accompanied by a comparable diminution in these more chronic diseases. Epidermosis continues to challenge and reproach the Otologist, and it is not surprising that the opinion still lingers in certain quarters that it must be some sort of congenital condition beyond human control. This pessimism is, in the writer's opinion, wholly unjustified. Epidermosis will some day disappear—once we have learnt how to protect the Eustachian tube and control the hyperplastic reactions in the epipharynx. Until that time comes we must set our sights rather lower. Since we cannot —as yet—eliminate pre-epidermosis let us concentrate on preventing its transition to Epidermosis. It is axiomatic that the earlier in the transition we see our patients the easier it is to halt the disease. But since Epidermosis is so insidious cases rarely present themselves in the earliest stages, especially in children. Consequently we must systematically search for them. Routine Otoscopy is already practised together with audiometry by certain Education Authorities but attention is usually directed to major pathology—more especially otorrhœa. We must now intensify this examination with a view to detecting Pre-epidermosis. Any child in whom this condition is detected should be reviewed at close intervals throughout its career and hospitalized at any sign of activity. This applies also to exudative otitis media which, as we have seen, is so closely allied both to Cholesterol Granuloma and pre-epidermosis. Moreover, on reaching maturity the child must be made to understand the potentialities of this menacing stigma so that he may report the earliest sign of efflorescence

The practical vindication of this programme obviously calls for painstaking field work over many years. Its theoretical justification stems from the concept herein expounded of Pre-epidermosis.

Summary

The word Cholesteatoma has outlived its usefulness and should be replaced by *two* terms which must denote unequivocally two quite

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different histological entities. Friedmann has suggested Cholesterol Granuloma and Epidermoid Cholesteatoma. The writer proposes Cholesterol Granuloma and Epidermosis.

Cholesterol Granuloma is characterized histologically by Cholesterin crystals, granulation tissue and giant cells. It occurs in advanced exudative otitis media and is common in children as a complication of the diffuse infection of the upper respiratory tract.

Epidermosis is characterized by the presence of true epidermis within the tympano-mastoid cavity.

Various theories of origin are discussed and a synthetic theory is herein proposed embracing them all. This leads to the conception of Pre-epidermosis, viz.: a marginal collapse of the drum either in Shrapnell's membrane or in the post-superior quadrant. This arises—like exudative otitis media—as a result of tubal occlusion and intra tympanic vacuum during the course of the diffuse infection of the upper respiratory tract in childhood. The methods of its transition to active Epidermosis (formerly known as Cholesteatoma) are discussed. Problems of treatment are outlined leading to the following conclusions:

(1) For CHOLESTEROL GRANULOMA—through cortical exenteration plus restoration of tubal function;

(2) For EPIDERMOSIS—marsupialization.

The combination of both conditions presents an extremely difficult problem for which no entirely satisfactory surgical technique has been evolved. At present the author is combining cortical exenteration with Atticotomy. Atticotomy alone is disappointing, so is tympanoplasty. In view of these disappointments the author is now considering the possibility of prophylaxis. True prophylaxis calls for the elimination of the upper respiratory diseases of childhood—a vast problem with no true solution in sight. Meanwhile much can be achieved by the intensive treatment of exudative otitis media. It is suggested that Epidermosis might be controllable if systematic otoscopy of all school children was carried out with a view to compiling a register of Pre-Epidermosis.

All children showing this menacing stigma should be inspected at regular intervals with a view to detecting and arresting the earliest manifestations of Epidermosis. Such a scheme has now been inaugurated in Liverpool and the results will be reported in due course.

The work embodied in this paper has been generously supported by a grant from the Medical Research Council for which grateful acknowledgement is made.

Photographs of the drum head proved far more difficult than anticipated. Fig. 3 is the result of an enormous amount of work by Mr. R. Pracy, F.R.C.S. to whom I record my sincerest thanks. Thanks are due also to Dr. R. W. Rewell who personally prepared Figs. 1 and 2.

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