# HYPERKERATOSIS VERSUS EPIDERMOSIS

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WITHIN recent years a considerable measure of agreement has been achieved with regard to the histopathology of "Cholesteatoma". Two distinct histological entities have been recognized, viz: Cholesterol Granuloma (characterized by granulations, cholesterin crystals, giant cells and blood pigment) and Epidermosis (characterized by true epidermic elements i.e., prickle cells as well as keratinized squames and amorphous debris). The term epidermosis has been coined by the writer to emphasize that in this condition there is true living epidermis within the mastoid cavity. It is synonymous with "epidermoid cholesteatoma" as used by Friedman.

Unfortunately there is as yet no general agreement with regard to the ætiology of these two entities, although most workers regard them as truly pathological. The writer's views may be stated as follows.

In the course of the diffuse infection of the upper respiratory tract of childhood the eustachian tube is liable to become blocked. This leads to exudative otitis media and this in its turn can lead to one of two conditions. The exudate may set up an irritated smouldering reaction within the air cells leading to the formation of cholesterol granuloma, or it may slowly absorb whilst simultaneously sucking on the tympanic membrane so that the latter collapses either in Shrapnell's membrane or in the post-superior quadrant. For this latter condition I coined the title pre-epidermosis, believing it to be the essential precursor of epidermosis. The concept of a primary eustachian block is of course in no wise original. It was, for example, put forward by Bezold to account for the entry of epidermis into the middle-ear cleft, and it figures prominently in Wittmaack's theorizing on the pathogenesis of the hypocellular mastoid.

In my opinion no theory of middle-ear disease can be regarded as adequate unless it accounts for these five pathological entities (viz. exudative otitis media, cholesterol granuloma, hypocellularity, pre-epidermosis and epidermosis).

On the continent, however, a totally different theory has recently emerged to the effect that epidermosis is in a class by itself, being due to some sort of inborn tendency (Wachstum-potenz) of the skin of the deep external auditory canal to invade the middle-ear cleft.

A similar viewpoint has recently been adopted by McGuckin who asserts that about 20 per cent. of normal people seem to have a tendency



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to hyperkeratosis in the deep meatus. On the basis of this single assumption he has erected a new theory to account for what he calls "destructive disease of the middle ear".

According to this worker, hyperkeratosis may occur in the outer ear in the absence of any other known pathology either in the outer or middle ear. Amorphous keratinized debris accumulates, perforates the tympanic membrane, enters the middle ear and so sets up destructive bone disease. This theory might be plausible if in fact the material within the middle ear merely consisted of keratinized squames and amorphous debris, but numerous workers have shown that in addition to this dead material the living elements of the epidermis, i.e. prickle cells, are present —the condition is epidermosis and not simple keratosis. Thus the keratosis theory is inadequate. We need a hypothesis to explain the presence of *living epidermis*. To that extent the "Wachstum potenz" theory at least has the virtue of accounting for epidermis by its assumption of a natural urge on the part of that epidermis to march into the middle ear. Both these theories however (i.e. Wachstum potenz, and hyperkeratosis), suffer from major defects inasmuch as they take no account whatsoever of the above-listed pathological conditions.

The issue therefore seems to be quite simple. We have on the one hand epidermosis (alias epidermoid cholesteatoma, alias cholesteatosis, alias Cholesteatoma), a frankly pathological condition, leading to destructive disease of the middle ear. We have on the other hand four other pathological conditions (exudative otitis media, cholesterol granuloma, hypocellularity and pre-epidermosis). In the writer's opinion these four are all intimately connected with epidermosis whereas according to McGuckin they have no relation whatsoever to that condition, the sole pathogenesis being an innate tendency to "hyperkeratosis".

Let us therefore pass in review the relevant evidence. First consider the relation between pre-epidermosis and exudative otitis media. In the course of the past ten years we have had under close observation over 300 children with middle-ear exudate (proven by myringotomy and aspiration). We have had the mortification of seeing many of these little ones resist all our efforts and under our eyes, develop the characteristic collapsed drum of pre-epidermosis. It is impossible to resist the conclusion that these two are causally connected. Next consider the relation between pre-epidermosis and epidermosis. Every otologist is familiar with the type of patient who gives a history of a quite recent painless, scanty, offensive otorrhœa who is found to have a small granulating marginal perforation. Many of these cases can be rapidly cured by purely local treatment, *but* they never show a normal drum after the cure is complete, they always show the characteristic collapse of pre-epidermosis. Moreover almost every case of unilateral epidermosis is found to have pre-epidermosis in the opposite ear. No one studying the tortuous pre-epidermotic



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sacs in these apparently quiescent ears can fail to be impressed by their manifest vulnerability. The conscientious clinicians will be familiar with the poor blood supply of this collapsed drum with its tendency to form small crusts and granulations at the annulus and with the tendency for hyperkeratotic debris to accumulate in niches around the skeletonized ossicles. These cases exemplify the transition from pre-epidermosis to epidermosis whilst the healing of the marginal granulating perforation exemplifies the opposite transition from epidermosis to pre-epidermosis. The connection between these two entities and the essential pathological nature of them both seems to be established beyond all reasonable doubt. It is impossible to understand why in the "hyperkeratosis" theory this obvious pathological process is so completely ignored in favour of a mysterious *natural* tendency for which no supporting evidence has ever been produced. Let us however proceed to consider some implications of the hyperkeratosis theory. Every otologist is familiar with those cases of widespread "cholesteatoma" in which there is only a minute perforation in Shrapnell's membrane and no sign of massive hyperkeratosis in the outer ear. Are we to believe that *dead squames* first accumulated in the outer ear, then drilled a minute perforation through Shrapnell's membrane, entered the attic, and underwent a resurrection into living prickle cells, whilst the original hyperkeratosis more or less disappeared? This surely is an accumulation of improbabilities.

Again we ask why, if hyperkeratosis is a normal function of so many people, we only encounter pathological changes in comparatively few. What is the *extra factor* that damns the few and spares the rest.

Every otologist is familiar with the *elderly* patient who comes with an epidermosis of *recent* origin. The writer's oldest patient started at the age of 72. Are we to believe that a normal hyperkeratotic tendency can be latent for 70 years and then break out for no good reason?

Lastly we consider the familiar condition of "Keratosis obturans" (characteristically, although not invariably associated with sinusitis and bronchiectasis). Here certainly we have a condition in which massive keratosis accumulates against the tympanic membrane under circumstances which must really produce "epidermosis" if the hyperkeratosis theory had any foundation in fact. It does indeed happen that in grossly neglected cases—especially those associated with secondary suppuration, the drum may be eroded and the middle ear invaded. These complications are however, *rare*, and in the majority of cases even when the outer ear is occluded by the hyperkeratotic mass the drum remains intact. In most cases the mass can be cleanly dissected away frequently revealing on its under surface an exact negative mould of the intact drum. In other words, just as we have already seen that epidermosis is not associated with hyperkeratosis of the outer ear, so we now see that true hyperkeratosis of the outer ear is not associated with epidermosis. How then can we



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accept hyperkeratosis as an explanation of "destructive disease of the middle ear"?

To sum up:

1. The connection between tubal obstruction and exudative otitis media can be asserted with confidence.

2. The connection between exudative otitis media and

(*a*) Cholesterol granuloma
(*b*) Hypocellularity
(*c*) Pre-epidermosis

} is equally plausible.

3. The connection between pre-epidermosis and epidermosis is clearly demonstrable.

4. In none of those conditions is there any evidence that any ætiological rôle is played by hyperkeratosis of the outer ear.

5. Hyperkeratosis of the outer ear is only demonstrable in one condition, namely Keratosis obturans. This quite definitely bears no relation whatsoever to epidermosis. Moreover it is clearly a pathological process and so cannot be identified with McGuckin's "natural hyperkeratosis". The concept of a "natural tendency "to hyperkeratosis adds nothing to our understanding of these familiar pathological processes. It leads to intolerable paradoxes and is at variance with the daily experience of every clinician. As yet no valid evidence of the existence of such a "natural" tendency has been adduced and until that evidence is forthcoming it is illogical to base theories of Otopathology on such a nebulous concept.

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