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Histomorphological data on human auditory tube dysfunction

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SINCE Toynbee's (1860) observation, we have known that the tympanic membrane moves during deglutition. From Politzer we learned tubal insufflation. He was the first to describe, in 1862, serous otitis media as a consequence of tubal catarrh. Zöllner in 1942 published a very important book about the pathology of the Eustachian tube and recently, in 1977, an International Symposium on the Physiology and Pathophysiology of the Eustachian tube and middle-ear was held in Freiburg. As clinicians we know that the weakest point of the whole air-filled middle ear system is the drum membrane. Like a window it shows all the changes within the tympanic cavity: pressure differences, by protrusion or retraction; loss of details, in cases of inflammation, scars or perforation; as signs of defective healing. The most difficult part to diagnose and treat, the key to otitis media, is the auditory tube. Tympanometry enables us to demonstrate the function of the tube. My effort now shall be to correlate morphological findings with dysfunction. Our material is all human, from our temporal bone collection originally started by O. Mayer. One has to keep in mind the one great limitation of morphology: the histological picture is like a snapshot; only a lot of pathohistological data allow conclusions.

1. *Pressure equilibration*

Optimum function within the air-conducting sound-transmission system is provided by equal pressures within the tympanic cleft and the external ear canal. According to Sadé *et al.* (1976), a tubal block very soon creates a partial vacuum of 10–200 mm. H_2O. If not released soon, transudate flows in. Politzer himself spoke about *hydrops ex vacuo.* The middle-ear effusion resembles a blood ultrafiltrate or an extracellular tissue fluid. The extreme situation is seen clinically in aerotitis after barotrauma.

2. *Ventilation*

Respiratory mucosa absorbs oxygen. A tubal block brings the partial pressure of CO_2 (Inglestedt, 1976) from the normal pressure of 17 mm. Hg. to one of 58 mm. Hg. Moscona (1964) and Sadé *et al.* (1976) have shown epithelial differentiation under the influence of a high CO_2 concentration. This leads to a metaplasia from flat epithelial cells to

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ciliated and secreting cells. The middle-ear effusion contains a mucoid component: clinically this is a sero-mucoid otitis media.

3. *Drainage*

Mucus is an essential part of ciliary transportation, which depends on the relation of ciliated to secreting cells. Within the auditory tube we have to calculate also the condition of the adjacent salivary glands. The middle ear effusion becomes a challenge to the mucosa, if drainage *via* the auditory tube or *via* an ear drum perforation does not take place. Clinically we now have adhesive otitis media.

4. *Mechanical and biochemical barriers against ascending infection*

Otitis media develops mostly from an ascending infection from a contaminated nasopharynx. The cilia beat in a direction towards the nasal orifice of the tube. Immunocompetent cell aggregations, e.g. the tubal tonsil, support local defence mechanisms; secretory elements provide immunoglobulins. The breakdown can be seen in hypervirulent infections such as scarlet fever, or in bacterial superinfection after viral diseases. Nowadays we see quite the opposite: under the influence of chemotherapy the infection seems to be arrested within the tube and causes a functional block. Serous otitis media starts; what Politzer described as a rare instance of serous tubotympanic catarrh, Zöllner more recently spoke about as a closed form of otitis media, Siirala of 'sterile otitis'.

Ventilation and pressure equilibration require opening of the tube. One of the factors involved is the surface tension-lowering substance in the mucus, as observed by Hagan (1977). Pathologic changes in the mucus, such as alterations in viscosity, and hyper- or hypo-secretion, may also influence the opening mechanism.

Morphologically we have looked at the adjacent *salivary glands* and found:

1. Ectatic excretory ducts, dyschylic secretory retention (Fig. 1), and sometimes microliths.
2. Lymphoid cellular infiltration around the ducts and between the lobules.
3. Interstitial fibrosis, parenchymal atrophy, lipomatosis and strands of scar tissue.
4. Formation of cysts, both oncocytic and degenerative.

We have never seen in our slides complete obliteration of the lumen of the tube, either by pressure from outside (tumour or cyst) or by scar or granulation tissue inside.

Irritation of the tube is also indicated by dysarrangement in the relation of ciliated cells to goblet cells. Hypersecretion and changed viscosity within the tubal mucus blanket may result. Mucus is a very important factor for clearance of the middle ear because of its role in ciliary transportation. Therefore a disordered relation of ciliated to

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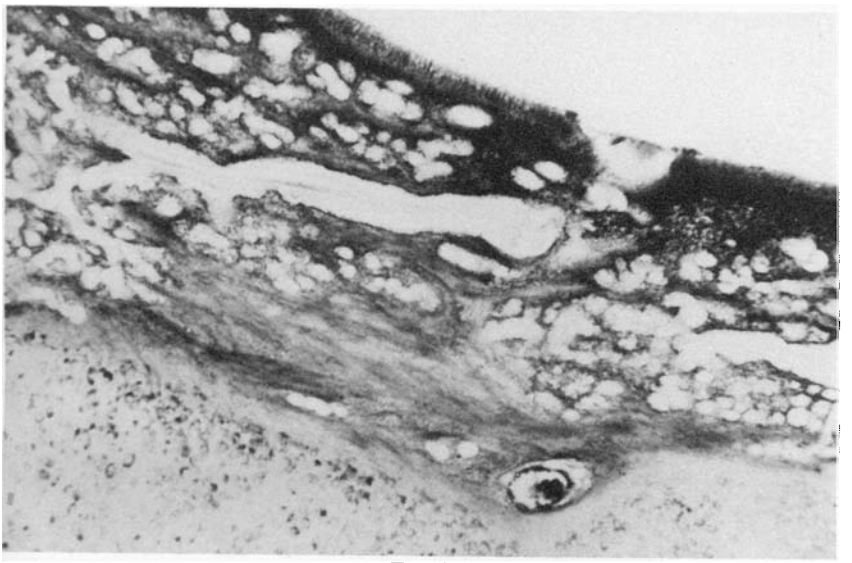


FIG. 1

Auditory tube, cartilaginous part; dyschylia within the salivary glands.

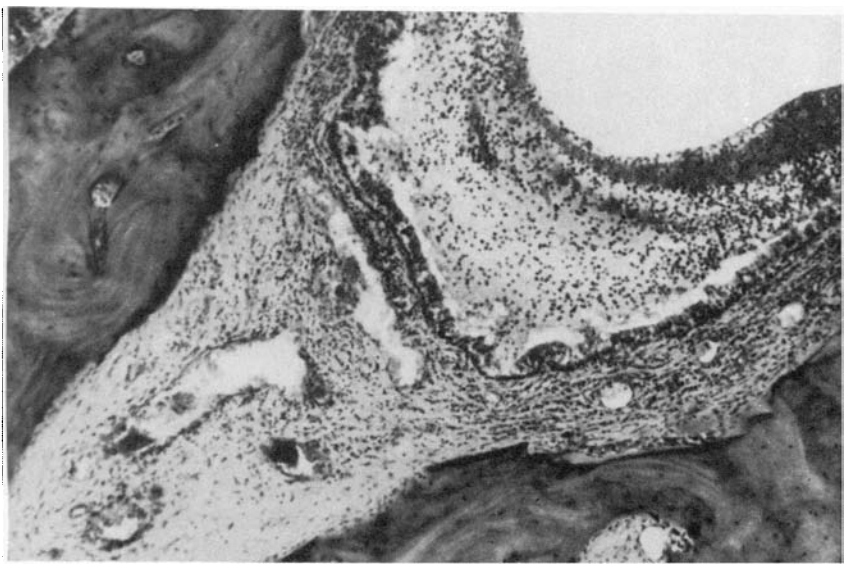


FIG. 2

Auditory tube, osseous part; effusion undergoing organization.

secreting cells can be an obstacle. A consequence of dysfunction in mucociliary transportation is the deposition of middle ear discharge at the tubal opening. The secretion produces a mucosal reaction which ends in organization (Fig. 2), and the formation of granulation tissue. In cholesteatoma typical cysts may be observed (Fig. 3).

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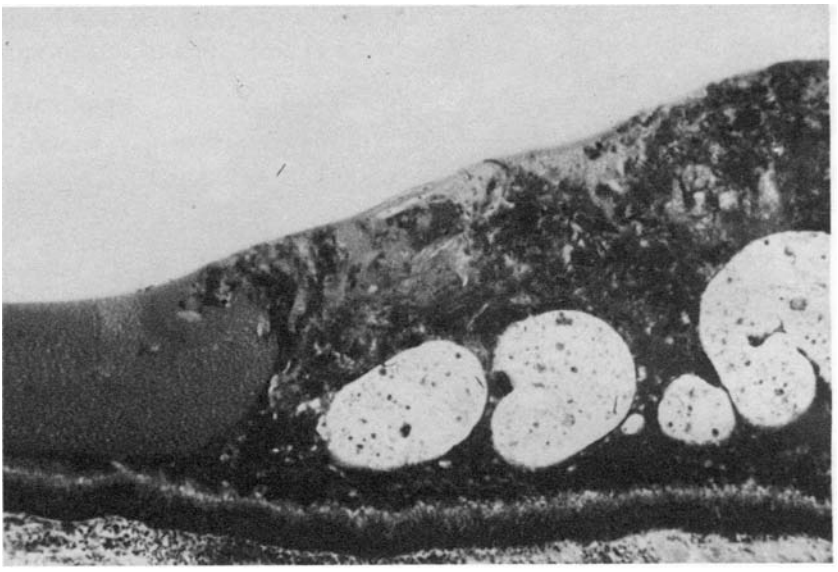


FIG. 3

Auditory tube close to the tympanic orifice; cyst-like formation within effusion in a case of cholesteatoma.

The ciliated cells and their mucus transportation are mechanical barriers against ascending infection. As a sign of *local defence mechanisms* we have to deal with two morphological findings:

1. The tubal tonsil: in all chronic ears immunocompetent cells aggregate around vessels, not only resembling lymph follicles but also forming a real layer of lymphoid cells (Fig. 4). The tubal tonsil is the immunological response to noxious infection. In normal tubes one cannot see anything like this.

2. The condition of the lamina basalis of the mucosa: very often the lamina basalis is thickened close to the tubal tonsil but interrupted in places by granulation tissue proliferation. The lamina basalis is a very sensitive indicator of aggressive infection, as we have been able to show within the middle-ear mucosa. Morphological changes in tubal dysfunction are not limited to the tube itself, and the demonstration of distant effects is seen in the so called 'otitis media chronica adhesiva'. Hence, in the closed type of otitis, there is a phase-like progression from serous effusion initially, to proliferative granulation formation and finally to the adhesive process. Those mucosal reactions which are observed within the tube may also be observed, perhaps at a different time, within the tympanum.

1. *Serous otitis media*

The mucosa is thickened by edema, and the vessels are wide, often surrounded by lymphoid infiltration (Fig. 5). The lamina basilaris is

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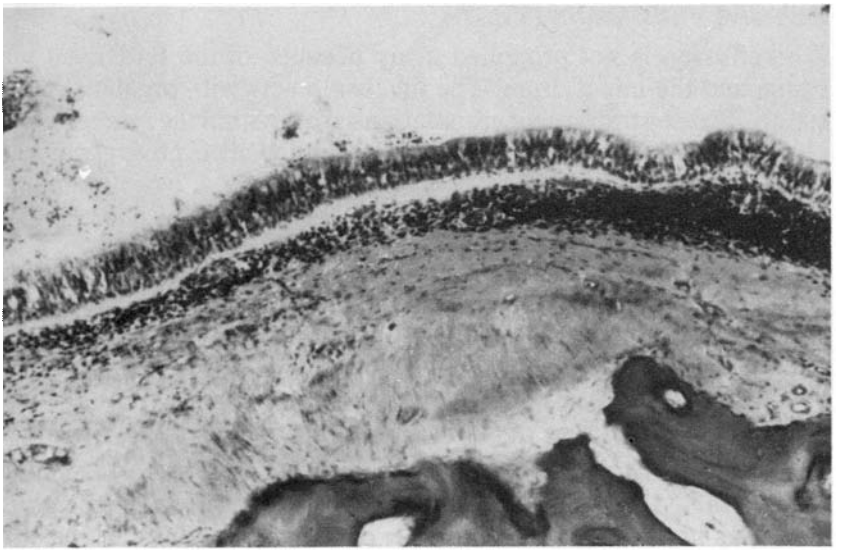


FIG. 4

Auditory tube, with so-called tubal tonsil below a markedly thickened lamina propria.

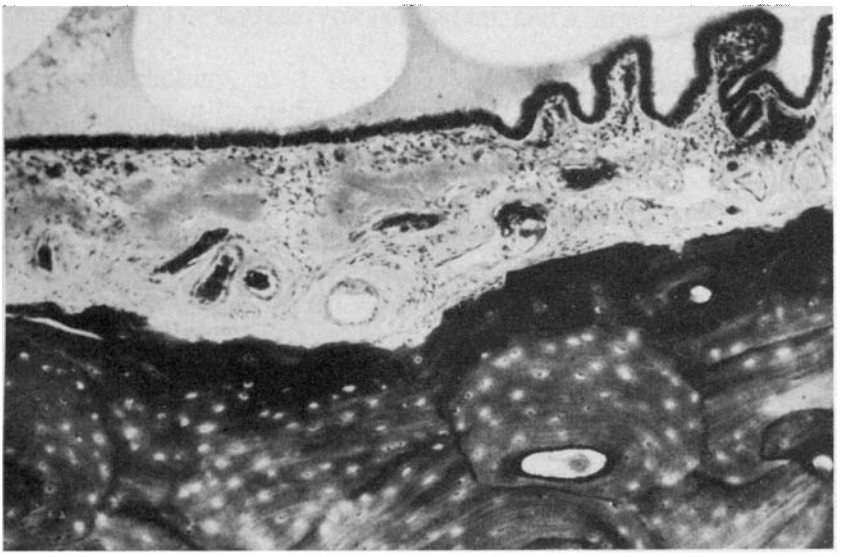


FIG. 5

Middle-ear mucosa in serous otitis media.

clearly seen. Finger-like mucosal folds plunge into the effusion devoid of cells. The epithelial layer undergoes metaplasia: ciliated and goblet cells cover the surface, not only at the bottom of crypts: and the histomorphological data are seen when there is a lack of tubal function. Ventilation and pressure exchange in the tympanic cleft are malfunctioning.

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2. *Adhesive otitis media* (Fig. 6)

The effusion is not propelled away because of the functional tubal blockage and the intact drum. The mucosa reacts with organization and digestion. Tissue strands and granulations grow within the tympanic cleft. Cholesterol granulomas occur as a sign of limited absorptive capacity for effusion remnants.

3. *Adhesive process*

Atelectatic tympanum, tympanofibrosis. As a result of defective healing after delayed restoration of tubal function, the tympanic cleft becomes obliterated, more or less. Calcification, fatty degeneration and scar tissue prevent movement of the drum and ossicles.

4. *Retraction pocket and atypical cholesteatoma* (Fig. 7)

Scar tissue and vacuum within the remnants of the tympanic cleft create a retracted drum in places where the fibrous layer of the membrane is absent, as in Shrapnell's membrane, or in the pars tensa if it has been destroyed by inflammation. Atypical cholesteatoma (Fig. 8) starts because of the formation of a bottle neck or niche without a real drum perforation, and the cholesteatoma shuffles the distended membrane along.

5. *Chronic mastoiditis*

Granulation tissue, bone apposition and poor pneumatization, all sequelae of malfunction of the tube, destroy an important safety device for

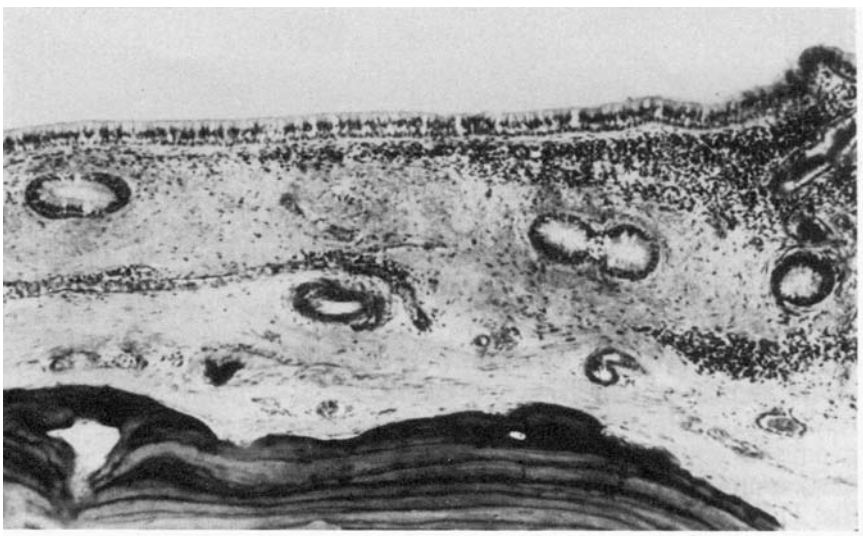


FIG. 6

Middle-ear mucosa in chronic adhesive otitis media. Note gland-like formations, immuno-competent cell aggregations and cellular metaplasia within the middle-ear lining.

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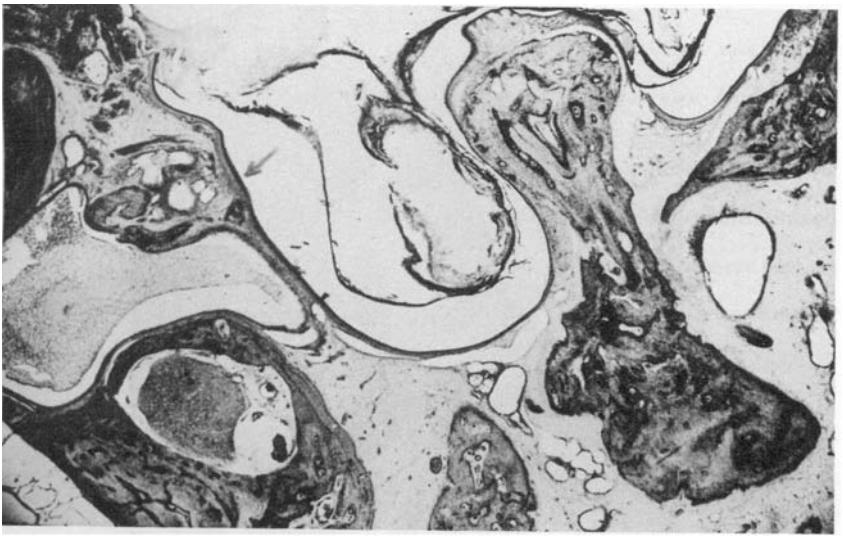


FIG. 7

Atelectatic middle-ear with retraction pocket reaching remnants of the stapes head.

the middle-ear. The air reservoir is filled up, and a large resorption area is blocked.

Histomorphology, as I see it, is a guide line to the functional understanding of otitis media. *Nihil nocere,* as a general rule in therapy, gains a great deal of information from pathology and to-day influences conservative treatment as well as tympanoplasty. In both I believe that the key to otitis media, and to complete eradication of the disease, is to be found in the condition of the auditory tube and in its functional capacity. All our efforts in middle-ear surgery should deal with these factors. Surgery must help nature, and it must assist the healing process within the tympanic cleft; it should be as conservative as possible, and destructive only when this is unavoidable!

Summary

By means of histological observations on human temporal bone slides from our own collection, we have found morphological reasons for dysfunction within the auditory tube:

1. Mucosal lining: Of great importance seems to be the relation of ciliated cells to goblet cells, i.e. the mucociliary transportation system and secretion. Granulation tissue close to the tympanic end, or massive destruction of the mucosa in virulent infections, interfere with clearance of the mucus. The organization of stuck-up exudate leads to the formation of cysts or narrowing.
2. Tubal tonsil: Always a sign of chronic on-going infection, it demonstrates an immunological reaction. Its appearance may be

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FIG. 8
'Early' cholesteatoma, in the site of Shrapnell's membrane.

like an accumulation of immuno-competent cells around some vessels, but it may also be follicle-like, resembling a lymph nodule.

3. Tubal salivary glands: For opening of the tube without resistance, a kind of surface-active substance is necessary, and there must be sufficient moisture within the lumen. Both are provided by the salivary gland system. Cysts, sialoliths, dyschylia, scar tissue and fatty degeneration in the parenchyma are, in our opinion, symptoms of failure in secretion.

Whatever the influence of antibiotic treatment may be, tubal dysfunction seems to be more frequent today. So we have calculated, as distant effects of functional tubal obstruction:

1. Secretory otitis media, leading to the adhesive process.
2. Atelectatic tympanum, with retraction pockets and resulting in atypical cholesteatoma.
3. Chronic proliferative mastoiditis, with bony destruction and apposition.

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