HISTOLOGY AND HISTOPATHOLOGY OF THE NASAL MUCOSA

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The respiratory membrane of the nose is similar in structure to that of the trachea and bronchial tree and shows a pseudostratified columnar epithelium, generally ciliated. But the cilia are often lacking, and the epithelium becomes stratified; the nuclei of the cells are situated on several planes. Under normal conditions the anterior ends of the lower and middle turbinate may represent areas of hyperplastic columnar epithelium or even of squamous metaplasia **(ASH** and **RAUM).** These can be considered physiologic rather than actual pathologic changes. There is considerable individual variation to the exact position of the transition from the stratified squamous epithelium of the vestibule to that of the respiratory epithelium of the nose. The term "pseudo-stratified ciliated epithelium" is generally applied to the nasal epithelium as well as to the epithelium of the lower respiratory tract. This is due to the various depths in the columnar cells of their nuclei which gives a false appearance of stratification (**Eggston** and **Wolff**).

In the various portions of the nasal cavity the nasal mucosa varies in thickness from 40 to 90 microns. The thickness of the mucous membrane has something to do with the amount of tunica propria and the contained cavernous blood spaces and vessels. Under pathologic conditions the character of the surface epithelium may undergo considerable alteration or metaplasia. The respiratory epithelium rests upon a cribriform connective tissue membrane, the membrana propria or basement membrane **(Eggston** and **Wolff).**

Between the columnar cells there are cells filled with mucin which are greatly increased under irritating stimuli. They assume the shape of a goblet and are therefore called goblet cells. This is one source of the protecting mucus coating of the nasal epithelium. Mucus, likewise, is secreted by the sero-mucinous glands of the entire nasal cavity. The goblet cells are probably identical with the ciliated columnar cells but when irritated by chemical or bacterial toxin are greatly increased in number, actually due to mutations of the ciliated columnar cells. After the goblet cell has disgorged itself, it collapses and the nucleus migrates to the central zone of the cell cytoplasm. The cytoplasm retracts towards the nucleus after the cell has disgorged itself and again becomes granular in character.

Stockinger has applied electron microscopy to the study of the ciliated

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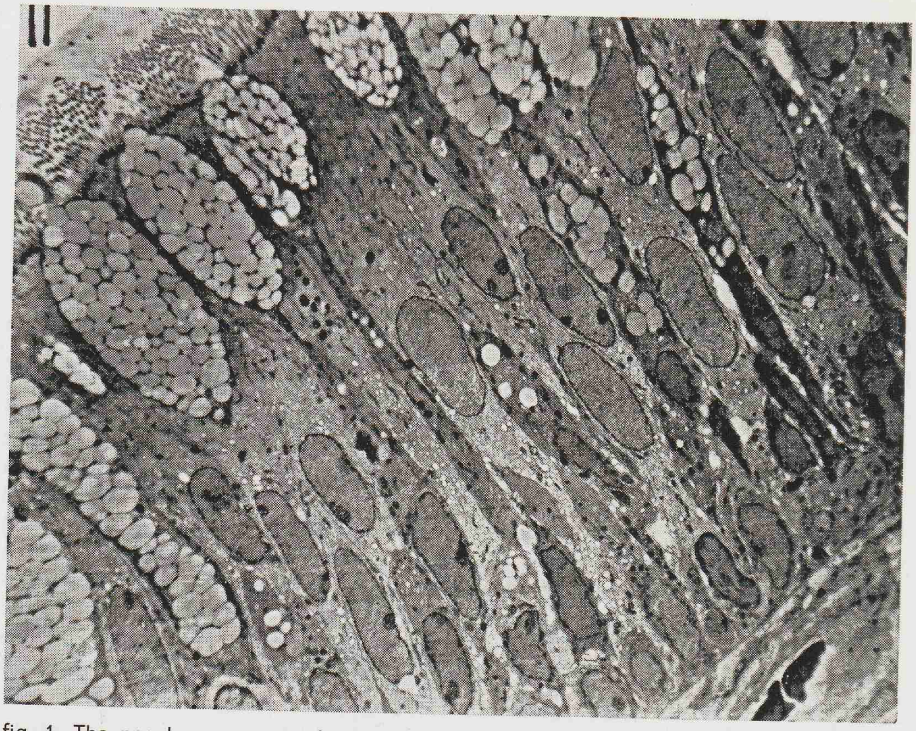

fig. 1. The nasal mucous membrane of the rat: ciliated pluristratisfied epithelium with caliciform cells and basal (replacement) cells.

La muqueuse nasale du rat: Epithélium cilié pluristratifié avec des cellules caliciformes et basales (de remplacement). 1500 x (Stockinger).

epithelium (Fig. 1) of the rat. Between the ciliated cells there are goblet cells and near the basement membrane basal (replacement) cells.

In allergy and also after slight experimental irritation in rats (fig. 1) (**Stockinger** and **Burian)** there is a strong secretory reaction in many goblet cells between the columnar cells. After a brief active injury to the respiratory epithelium of the rat damage of the ciliated cells with broken filaments and swollen mitochondria results. Three days after such injury to the epithelium we can distinguish between the normal ciliated cells and empty goblet cells (disgorged). The important role of the connective tissue is described by **T. F. Dougherty.**

In allergic rhinopathy we found a multi-layer ciliated epithelium and abundant goblet cells, a mucous degeneration of the epithelium with increased secretion of mucus, sometimes also epithelial metaplasia. The basement membrane varies greatly in thickness, is usually thickened and shows hyaline changes. There is subepithelial oedema due to increased capillar permeability and eosinophil infiltration.

Hlavacek and **Loyda** have summarised their investigations into mast cells in the mucous membrane of the upper respiratory tract under normal and pathological conditions as follows:

The mast cells are a normal part of the mucous membrane of the respiratory

tract. Their number varies greatly according to circumstances. During chronic processes they are greatly increased in number. During acute inflammation they are disrupted and their number is diminished or they disappear altogether. During the acute stage of allergic reaction they found a decrease or eventual disappearance of mast cells. On the other hand, an increased number of eosinophils can be observed.

The mechanism of the degranulation of mast cells is probably similar to the anaphylactic reaction in the sensitized animal. In this condition degranulation develops during contact of antigen-antibody and some substances, chiefly histamine, contained in the mast cells, are liberated. The liberation of histamine causes such well known effects as congestion and immigration of eosinophils.

The following schematical table, compiled by **Terracol** and **Chevance**, intends to enumerate the principal histo- pathological manifestations of allergy and infection of the nasal sinuses.

Structures involved	Allergy	Infection
mucus	non-purulent hypersecretion decreased viscosity abnormal isoton city	purulent hypersecretion variable viscosity and isotonicity
vibratory cilia	decreased or arrested movements, disappearance of the cilia in the zones affected by the allergy. Loss of efficacy of the cilia in case of chronic allergy	remarkable resistance for a very long time. Fragmentary destruction which only becomes total in case of severe or protracted infection
superficial epithelium	cellular structures dissociated and regressive; at the worst, epithelial structures undifferentiated, but possibility of regeneration persists for a long time	metaplasia more progressive and less reversible than in allergy; rapid destruction in severe cases
glandular structures	hyperactivity and multiplication of these structures, which are the least affected of all tissues involved for a long time	glandular multiplication, followed by atrophy in case of protracted infection
membrane basement	remains normal for a long time, then infiltrated by eosinophiles, accompanied by thickening	rapid thickening and destruction

tunica propria	enormous oedema and infiltration by wandering cells; high percentage of eosinophiles.	oedema less pronounced infiltration of polymorphonuclear inflammatory cells; sclerosis, developing more rapidly as infection is severe
vascular structures	vasodilatation, disturbed permeability. Exudate without large parietal lesions for a long time	exudate often haemorrhagic. Early parietal lesions and fibrogenetic thrombotic manifestations and micronecrotic factors

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