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CURRENT EVALUATION OF NASOPHARYNGEAL DEFENSE MECHANISMS

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Most otolaryngologists have been trained to an anatomic attitude respecting the defense mechanisms of the upper respiratory tract. Aside from purely mechanical factors, it was for years not considered feasible for most patients to receive treatment based on physiologic lines, until the advent of vaccines and more recently of antibiotics.

While Harris Peyton Mosher was at first intrigued by the anatomy of the ethmoidal labyrinth and the other accessory sinuses, he began the study of regeneration of antral epithelium and (using vital stains) of excised tissue, as long ago as 1931. Proetz and Hilding were already at work on ciliated epithelium; and Larsell and I since 1928 had begun our work on mucosal protection. We made, thereafter, a thorough study of various factors, tending to the belief that the main element in mucosal defense consists in the carpet of mucosal cells lining the nose, nasal cavities, nasopharynx and upper respiratory tract. Herewith, somewhat dogmatically stated, are our conclusions down to the present:

The first line of defense is the covering of mucous secretion on the epithelium, as produced by the goblet cells and glands of the mucous membrane. Inert dust particles and bacteria are removed with the mucus, by ciliary action, helped occasionally by sneezing. Mucous glands and goblet cells are found in the walls of the nasopharynx and also in the lungs.

The content of lysozyme in this mucous coat is variable but constant under normal conditions; and the pH of the nasal secretion is changed materially with the onset of local disease.

Cilia in the nasopharynx and accessory sinuses carry on purposeful activity, keeping a fresh sheet of protective mucous secretion over the epithelial surface. Increased mucous secretion to cope with irritation or infection is an attempt to strengthen the first line of defense, but may produce a blockade in the nasal passages or filling of the accessory sinuses.

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Virus infestation of bacteria, notably in cases where response to antibiotic medication has been slowed down or lost, may produce severe reaction to bacterial invasion, in which case local use of antibiotics is worthless.

Allergic reaction of respiratory epithelium, with production of local edema and obstructive phenomena due to secondary infection, is controlled by specific drugs acting upon the sympathetic innervation. But local changes due to secondary infection may overshadow the original allergic etiology—and permit the survival of virus strains which will renew their attack upon the cells when bodily resistance is lowered by cold, exposure or unusual mental or physical strain.

The epithelium is thus the actual first defense, but will change with hyperventilation, loss of cilia and trauma, to become stratified squamous in type, with ingrowth of fibrous tissue, which may thicken greatly.

In the paranasal sinuses epithelium is normally simple, thin ciliated columnar, with but few goblet cells and glands. In response to inflammation or blockade, this epithelium swells enormously to a stage which may present in the nasal airway as polypoid protrusions.

The respiratory membranes, resting on a layer of connective tissue, are attached to rigid walls, without muscular movement to force lymph-containing toxic substances along lymphatic channels to help remove noxious elements from the membranes. This slow removal tends to permit accumulation of toxic products, and lymphocytes and neutrophile leukocytes come in by migration from the blood stream. This produces edema and a greatly thickened membrane filled with cells, which begin to phagocytise the bacteria found in the depth of such swollen tissues.

The second line of defense consists of neutrophiles and of "scavenger" cells, the histiocytes, which proliferate or migrate and increase rapidly in number in the early stages of sinus membrane infection. These histiocytes engulf bacteria, supplementing the neutrophile efforts, and may migrate through the epithelium to escape into the sinus cavity. But swelling may close sinus ostia and aggravate the sinus situation; and after a month the histiocytes and leukocytes disappear and are substituted for by plasma cells.

These latter, often in large numbers in "chronic" sinus membranes, are not phagocytic, but are assisted in the task of healing by the histiocytes, which later become fibrocytes and then fibroblasts.

Swelling of sinus membranes, aggravating acute or residual infection, may be produced by vasomotor changes due to reflexes carried

by the autonomic nervous system, often arising from abdominal or pelvic congestion and calling for relief elsewhere than in the nose.

Lymphatic drainage, first to the retropharyngeal and superior cervical lymph nodes, has been traced by vital stains to show that these glandular structures form the third and major line of defense of the body against sinus infection. Localization about such structures may produce abscess; but if the deep superior cervical nodes are overwhelmed, drainage into the cervical lymph ducts may occur, with eventual drainage into the superior vena cava. Such drainage may travel into the right heart and the pulmonary capillary bed—and thus may eventually reach the perivascular and peribronchial lymphatics, which constitute a fourth barrier to systemic invasion by upper respiratory infection.

Fifty years ago Henry Wagner of San Francisco suggested that the natural immunity of the mucous membranes of the respiratory tract rested not on the mechanical protection of mucin, but upon the protoplasmic activity of living body cells, and Harris P. Mosher, artist-anatomist, brilliant surgeon and teacher, dissatisfied with the results of operative procedures, turned to the work of his laboratory for explanations of the healing effects of the cellular linings of the accessory sinuses. May his example not be forgotten!

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With Ralph Fenton, 1939.

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