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Mucociliary function in chronic infections

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My contribution to this symposium will be some short remarks on the significance of mucociliary function during the development and course of chronic rhinitis and sinusitis. This task is at the same time easy and complicated. Easy because we have an exclusive knowledge from patients with congenital defect cilia headed under the description "immotile cilia syndrome", which always results in chronic infections but also difficult because in usual acquired chronic infections, there are many possible etiological factors to take into consideration.

CHRONIC RHINITIS

Most cases of chronic rhinitis are due to anatomical defects, toxic injuries, degenerative changes of mucous membranes or inadequate healing after acute infections.

A suitable aerodynamic shape of the nose is necessary for an effective airconditioning of inspired air. The cilia are most active at body temperature but tolerate well minor temperature changes. On the other hand they are extraordinary sensible for reduced relative humidity.

Mucosal injuries due to industrial pollutants, cigarette smoke and excessive use of locally applicated drugs or inhalatants may deteriorate the ciliary function and facilitate secondary infections. This type of etiological factors should be easy to handle prophylactically.

Inadequate healing of acute infections are often the background for chronic rhinitis and needs therefore to be commented more closely.

Unlike some types of bacteria - viruses are not routinely present in the nose of healthy individuals but it is well known that some types of common cold viruses influence the ciliary function. A common cold is often accompanied by loss of ciliated cells in the nose and probably further down in the deeper airways. A defect ciliary defence mechanism makes it easier for bacteria to colonize on the mucosa. The nose is normally a reservoir for some types of bacteria. Diphteroids and staphylococcus aureus are found in 40-50% in the vestibulum of healthy indi-

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viduals. Streptococcus pneumoniae and haemophilus influenzae are often colonizing the posterior part of the nose especially in children.
The coaccidental role of air pollutants in combination with bacterial infections is not yet fully known. However, the mucociliary function is reduced by tobacco-smoke and sulfur dioxide as seen in inocculation experiments on volunteers.

CHRONIC SINUSITIS

The etiology of for example maxillary sinusitis is often a blocked ostium due to local infection, trauma or swelling of the nasal part of the mucosa. In the closed cavity oxygen is absorbed tending to a pressure reduction. This facilitates transudation and release of secretions. Then a secondary viral or bacterial invasion can take place. Ciliary movements are in such cases prevented by heavy amounts of thick tenacious mucus. The previous opinion was that the cilia very often were intoxicated and eventually destroyed. I would like to describe this phenomenon as a type of "Immobile cilia syndrome" in contrast to the previously mentioned congenital "immotile cilia syndrome", where the cilia are morphologically defect at birth. Here they are only accidentally immobilized by the mucus. The practical consequences of this would be firstly that the maxillary sinusitis should be drained more frequently than many of us do today and preferably not later than after a failure of the initial antibiotic course instead of prescribing a new one. Secondary, the Caldwell Luc-operations for chronic infections should be done as conservatively as possible without total resection of the mucosa.

FACTORS CAPABLE TO REDUCE THE MUCOCILIARY ACTIVITY

Oxygen deprivation. Cilia need a continous oxygen supply. The oxygen source can be either from the ambient air via the secretion layer or from the capillary bed. In closed cavities, like sinuses and middle ears blocked by secretions the capillary bed is the only oxygen source available.

Enzymes. Leucocytes approaching a center of infection are disaggregated and enzymes like esterase and collagenase are released. They have, however, not only an effect on bacteria but also on the host tissues if not balanced by adequate amounts of specific enzyme inhibitors. When cilia are exposed to pure elastase or collagenase the activity is reduced and the cells more or less destroyed.

Bacterial toxins. The effect of different bacteria and their split products on mucous membranes are of great interest. Our knowledge in this field is still limited. Haemolytic streptococci and Haemophilus influenzae have moderate effects on the cilia. Colitoxins even in high concentrations have no ciliestatic effect. Some strains of Pseudomonas aeroginosa are ciliestatic but other strains have no effect.

Such differences are of special interest for example for evaluation of therapeutic effects.

Air pollutants. It is known that cadmium, nickel, cromats, copper etc. are cilietoxic. The mechanism is supposed to be a binding between the metal ions and ATPase, reducing the ATP available for energy supply to the ciliary movements. Irritant gases from industrial districts such as SO_2, NO_2, NH_3 reduce tracheal clearance. Cigarette smoke is of special interest. Pure nicotine intra arterially have been shown to stimulate the mucociliary activity but several other components in cigarette smoke are cilietoxic. In my opinion the ENT-doctor generally gives their patients no or insufficient advice regarding the effect of smoking in cases with allergy and chronic airway infections. I think we have too long ignored the relevance of such informations.

PRACTICAL CONSEQUENCES REGARDING PATIENTS WITH CHRONIC AIRWAY INFECTIONS

1. Avoid smoke and dust pollution.
2. Avoid the nasal approach for medical treatments.
3. Treat acute infection effectively.
4. Ventilate closed cavities as fast as possible.
5. Judge the physiological consequencies before nasal operations.
6. Investigate the mucociliary function more frequently in chronic cases.

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