ATROPHIC RHINITIS AND OZAENA

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The clinical picture of atrophic rhinitis, marked by dryness, crusts and foetor has been well known to every rhinologist. Conversely, its pathologic physiology is considerably obscure. Consequently, there does not exist a unique causative treatment and therapy is wavering in a wide range from utmost conservatism to the most radical operative interferences. In this report, a series of viewpoints on pathologic physiological occurences of the nasal mucosa are to be elucidated, in the scope of which the place of the clinical picture termed in everyday life rhinitis atrophica resp. ozaena is to be determined. Moreover, we must recall that it was only at the threshold of this century that functional observation of vital processes outweighed previous entirely morphological perceptions. Hence the observation of inflammatory processes on the respiratory mucosa has entered a new phase.

Initial hyperaemia and raised production first only of goblet cells, further on of submucous glands as well, should stimulate in the first stage, increased metabolism and enlarged production of mucus, elements necessary for abating successfully the action of the injurious agent. If the effect of that agent continues with unchanged intensity and the mucous membrane with its raised metabolism and enlarged production of mucus fails to neutralize this effect with success, hypertrophy of these elements occurs, whereby the reinforced interstitial tissue presents a more solid base for the enlargement of specific elements. Such a condition subsists either as long as the mucosa with its increased function does not suppress the infecting organism or as long as it is capable, along with the undiminished infecting organism to neutralize its effect. In case the mucosa succeeds by its proper function to get the infecting organism under, the mucous membrane with the disappearance of the noxious agent returns gradually to its normal limits as well. But in case of further increased intensity of the infecting organism, a fact which surpasses the capacity of the mucosa to neutralize this organism and if the mucous membrane fails to intensify its further resistance, the subsisting glands start to deteriorate owing to exhaustion. The producton of new series of goblet cells becomes reduced, atrophy of specific elements of the mucous membrane occurs and they are being replaced by connective tissue. The disappearance of specific elements produces an irreversible condition of complete atrophy and there arises a mucous membrane deprived of all functions.

That is the given scope within which the response of the respiratory mucosa to outer noxae occurs. But within this scope there are also certain variations depending on the mutual play of individual constituents and their potential power. It is just this potential power of each of these constituents of the respiratory mucosa, being the congenital factor which the organism brings into the world by inheritance, that produces various patterns developed by the nasal mucosa during the course of inflammation. In addition, if we take into consideration, that these elements differ not only by their morphological

structure but also by their different origin — being partly of ecto-resp. endodermal - partly of mesenchymal origin as well — and therefore also that owing to separate genetic potential particularities, there may supervene the possibility of their being either exposed to the effect of various noxae on that heterogeneous developmental way or of their different response to a certain stimulus concerning their specific developmental way, then we may better comprehend the wealth and variegation of the forms in which the effect of the inflammatory noxae on the mucosa is demonstrated as an entity, though they must necessarily occur within a definite scope, conditioned by the morphological composition of the mucous membrane itself. If in a given case, there is a prevailing pattern either of hypertrophic or atrophic inflammation, then it depends as well as its duraton first of all on the potential power of the mesenchymal elements of the lamina propria and the intensity resp. duration of the inflammatory noxa. If the mesenchyma possesses considerable potential power, the lamina propria will abate successfully the noxious effect of the inflammatory noxa by swelling of its connective tissue constituents and protect with its enlarged mass the remaining highly differentiated constituents of the mucous membrane from complete destruction. Thus it makes their restitution after surmounted danger possible. On the contrary, the fibroblasts as well succumb to the effect of the injurious noxa. Not only highly differentiated elements, as ciliated cells and glandular epithelium, but also the basal cubic cells undergo metaplasia. The matrix, only from which complete restitution may arise, disappears. The elements of the reticulo-endothelial apparatus as well as those of the terminal nervous processes decrease. Complete atrophy of the mucous membrane is brought about, having become a thin connective tissue coating deprived of all functions.

From the above we may conclude that inflammatory reaction depends likewise on the morphological structures as well as on the function of the affected tissue. Thus this dependence exists, regardless of the specificity of the infecting organism which may perhaps affect only selectively rather ecto-resp.-endodermal or mesenchymal elements of the affected region. The intensity of the response to the inflammatory noxa does not exclusively depend on peristasis, but also on the biological value of each of the elements of the mucosa. This biological value is not only based on the potential of each element of the mucous membrane, but on the entire complex of interdependence not only of separate units of a single tissue variety or of a single organ mutually, but also of various systems within a biological unit, all of which together form but the unity of a multicellular organism.

Hence inflammatory processes depend first on the potential power of their separate constituents whereby highly differentiated elements have first of all to give way in that struggle to those developmentally more advanced ones and thus more resistant to pathological changes. Starting from this point of view, we hence cannot speak of a specific hypertrophic resp. atrophic inflammation which does not at all exist as a biological unit. We may only take for granted that there is a hypertrophic resp. atrophic stage in the otherwise uniform process of the inflammatory reaction of the nasal mucous membrane.

LA RHINITE ATROPHIQUE ET L'OZENE

Les phénomènes physiopathologiques de la rhinite atrophique ne sont pas encore entièrement expliqués. La réaction inflammatoire de la muqueuse nasale dépend de la structure morphologique aussi bien que de la fonction du tissu atteint. Elle est donc conditionnée sans égard au caractère du facteur étiologique qui peut attaquer sélectivement une fois les éléments de l'ectoderme ou de l'endoderme, une autre fois les éléments du mésenchyme. L'intensité de la réaction ne dépend pas uniquement de la péristase, mais également de la valeur biologique de chacun des éléments de la muqueuse. Elle n'est pas uniquement fondée sur la résistance de chaque élément de la muqueuse, mais elle dépend aussi de l'interdépendance de tout un ensemble: non seulement de certaines unités d'une même sorte de tissu ou d'un organe mais également de différents systèmes à l'intérieur d'un milieu biologique, en d'autres termes, de l'unité d'un organisme multicellulaire. Dans cette lutte succomberont d'abord les éléments plus différenciés, contrairement à ceux qui se sont développés plus tard-qui sont moins vieux-donc aussi plus résistants aux événements pathologiques.

Partant de ce point de vue, nous ne pouvons pas parler d'une inflammation hypertrophique respectivement atrophique spécifique. Nous pouvons seulement supposer qu'il existe un stade d'hypertrophie respectivement d'atrophie dans un seul et unique processus de la réaction inflammatoire de la muqueuse nasale.

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