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THE ETIOLOGY OF CHRONIC ATROPHIC RHINITIS.*

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For twenty years I have maintained and still maintain that atrophic rhinitis is a development from the purulent rhinitis of childhood. There is not a single clinical case on record that justified the assertion that atrophic rhinitis ever follows hypertrophy of the mucous membrane. In early life it is the epithelial structures which are especially involved in a tendency to catarrhal inflammation of the mucous membrane. The result is an increase of the secretion, together with an exaggerated proliferation of the epithelial cells. This results in a discharge of mucus, containing young and unripe epithelial cells, constituting a muco-purulent discharge. This process gradually extending into the follicular structures and into the racemose glands, these glands eventually becoming denuded of these epithelial cells, collapse and disappear. The result is that the secreting surface of the mucous membrane is to a certain extent destroyed. The secretion in time becomes inspissated, and dries readily into crusts. These dried crusts interfere markedly with the flow of blood in the mucous membrane. The resulting contraction of the mucous membrane causes an interference with the respiratory function of the nose. Not only is the whole thick-

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ness of the mucous membrane involved, but the turbinated bodies and, sooner or later, the periosteum, and then follows atrophy of the turbinated bones. The muco-purulent secretion of the nose, when dried, is not easily expelled, and its retention and consequent decomposition readily explains the stench observed in such cases. The disease, therefore, is essentially one of childhood, and after six or seven years it advances sufficiently far to cause the formation of crusts and produces a stench. These cases are comparatively rare—about one case for every forty of the hypertrophic variety. He had never seen but one case of fully developed atrophic rhinitis cured, although they sometimes improved spontaneously at about the age of forty.

The sequence of pathological changes in the tissue would seem to be easily comprehended, but the question as to why the initial change should set in is not easily answered. In a certain proportion of cases of the disease which I have seen in its early stages there was notable evidence of mal-nutrition, but it could not be traced in any instance to syphilis, tuberculosis, scrofula, lymphatism or any of the constitutional dyscrasia.