constitutes the hyaline basement membrane everywhere underlying the surface epithelium in the normal state, but which in this disease disappears.

With regard to their parasitic nature my investigations at present do not permit a decided expression of opinion, although several friends, whose biological experience is greater than mine, have expressed themselves in favor of that view.

Pigment masses are not constant in their appearance: they occur for the most part in irregularly-shaped clusters, sometimes enclosed in branched connective tissue cells, at others blocking the lumen of the capillaries, and distributed both superficially and deeply.

Changes in the Glands. The gland changes vary in degree, from a simple cloudy swelling of the secretory cells, with blocking of the lumen, to a complete disorganization of the acini by ingrowth of small cell inflammatory tissue. The duct epithelium apparently resists these changes until very late, excepting in those instances in which the ducts were distended by plugs of laminated keratin masses. Most writers refer to these cell changes as being fatty in nature; whilst confirming this in a few instances, careful examination showed that mucoid and keratinoid degenerations occurred much more frequently. The plugging of the ducts bore a strong resemblance to the comedones of sebaceous glands.

Changes in the Lymphoid Tissue and Vessels.—In every specimen the lymphoid tissue gave distinct evidence of change. In early stages the corpuscles were numerically increased, whilst in later stages they diminished in numbers but increased in size with absorption of the reticulum—in fact, presenting the appearance of granulation tissue, such as occurs in lupus, and, like it, invading other structures and undergoing subsequent sclerosis.

The capillaries, which normally present long loops reaching to the hyaline membrane, became entirely obliterated. The cavernous spaces became less distended, and finally atrophied, due to diminished blood supply, induced by a general insterstitial fibrosis, and in some cases a process of slow endarteritis obliterans in their afferent vessels. I could not observe any decided active changes in the arterial walls; they seemed to be undergoing a process of atrophic stenosis.

This vascular atrophy and perversion of gland function are greatly responsible for the altered secretions, but a most significant feature is the disappearance of the lymphoid tissue.

Changes in the Bones.—I have entirely failed to demonstrate any histological changes which might be considered specific. The walls of the bony cancelli in advanced cases were decidedly attenuated, even more so than what would be considered normal to the patient's age, and the osteoblasts were few and flattened. Osteoclastic absorption was well shown in early cases, but not excessively. When the disease occurs in early life it must obviously interfere with the proper growth of the turbinal bones; it is, therefore, not surprising to find them smaller than natural, but this diminution must not be attributed at any time to rarefying osteitis, nor must rarefying osteitis be considered necessary to atrophic rhinitis.

Too much stress has been laid upon the simple presence of osteoclasts as indicative of a particular morbid process. These periosteal and endosteal changes are simply part and parcel of a normal osteo-porosis or cancellation, a process essential to the development of these and other bones. It is only when the osteoclastic changes become excessive that they justify a morbid attribute.

Many writers explain the bone atrophy as the result of pressure from the drying crusts, like a collodion film, whilst it has been suggested by Zaufal* that it is the result of a congenital defect, and has an important causal relation to the disease in question.

Considering the nature of the changes occurring in the soft tissues, it would be surprising if the bones did not give indications of a diminished blood supply; but this atrophy presents the features of a passive rather than an active process, occasionally producing patches of bare bone.

Relation to Lupus.—Spencer Watson† has advanced the view that there is a very close analogy between atrophic rhinitis and lupus non exedens, and that they may both be due to a common bacillus. That they probably possess a few features in common may be correct, but the suggestion of a common origin in a particular bacillus requires

* "Aerzte corresp. für Böhm," 1874, Nos. 23 and 24.

† "Diseases of the Nose," 1887, p. 85.