smooth surface, deficient in blood supply and clinging closely to the irregularities of the spongy bones.

About the hyaloid bodies, their distinguishing feature is the fact that, although the entire turbinated cushions shrink continually in direct relation to the progress of the disease, these hyaloid bodies increase in numbers as the disease advances. Wingrave describes them as small, round, refractive, homogenous masses embedded in the interlobular tissues of the glands, in the adjacent lymphoid tissue and sometimes in the stratified epithelium. Their size varies from the 2000th to the 800th of an inch. Latterly they break up into minute refractile bodies resembling spores. The question of the nature of these is still *sub judice*. Some pathologists believe them to be the parasites of atrophic rhinitis.

As I said before, the rest of the changes in the mucous membrane and turbinated bones are those of constant contraction, permanent shrinkage. The adenoid or hyaloid membrane which, in hypertrophic rhinitis remains intact or increases in dimensions, in this disease shrinks gradually out of existence.

The gland follicles become crowded with lymph corpuscles of a low inflammatory type, blocking their lumen, often producing complete disorganization of the acini without ever developing into true hyperplasia. The capillary blood-vessels disappear. The cavernous sinuses become less and less distended. A general fibrosis contracts the vessels, and finally they, too, shrink away, involving the underlying spongy bones in the common atrophy.

Incidental pathological changes occur in a majority of cases. Out of sixty recorded, the faucial and pharyngeal tonsils had in fifty-six entirely disappeared; while in the remaining four they were small, thus indicating a peculiar relationship between the surrounding lymphoid structures and the atrophic disease.

Perforation of the cartilaginous septum is also of not very infrequent occurrence. It is, however, generally believed to be not so much the direct result of the atrophic disease as of digital picking, the region being so easily accessible to the finger. In my own experience I do not remember a single case of perforated septum co-existant with atrophy, in which I could not trace the origin of the perforation to the period of childhood. When it comes under the notice of the physician the margin of the perforation will usually be found coated with tenacious mucus, overlying a layer of proliferated epithelium. Very rarely will there be any indication of recent ulceration.

Etiology.—Perhaps there are few subjects in medical science upon which there exist so many differences of opinion, as upon the origin of atrophic rhinitis. Bosworth, as is well-known, makes the statement that purulent rhinitis, "he believes to be in every case a cause of, or the primary stage of, the atrophic form." There are few, if any, other writers who support him in this extreme view, while many, if not all, believe that it may sometimes be the cause. The more general opinion among the profession is that it usually occurs as a sequel to hypertrophic disease. J. N. Mackenzie goes so far as to affirm that: "Atrophy appears to be always a secondary affection . . . resulting from previous inflammatory thickening." Seiler while acknowledging that it frequently results from hypertrophy, also says that: "It may be of the atrophic variety from the start." Drake claims chronic purulent inflammation of the accessory sinuses as the cause. Gottstein holds that defective development of the turbinated bones is responsible for the disease. And Sajous, while accepting some of the causes already named, very justly says that an unusually dry atmosphere, the inhalation of tobacco smoke, or an abnormal patency of one or both nasal chambers, may any of them be the proximate cause of atrophic disease. Added to all these, as well as the parasitic origin already mentioned, Mayo Collier has recently thrown out the plausible suggestion that it may yet be discovered: "That the initial disease was degeneration of the nerve ganglion and nerve fibres supplying the parts.

Personally I have seen a number of cases which I could trace back to purulent rhinitis of childhood; others, where I had reason to believe that the atrophy was a sequel to hypertrophy; several, in which a certain amount of hypertrophy in one nostril accompanied atrophy in the other; and more than one in which the breathing of a dry atmosphere was the presumptive cause.

Usually when patients have presented themselves for treatment, they have already been