of the tubercular and fibrous forms will enter into the discussion as is consistent with a complete understanding of the subject. A great deal of loose and indefinite material has been thrown around the subject of phthisis, which has very much interfered with a true conception of its relations, and, in order to avoid a similar difficulty, and to make an intelligent discrimination between the different varieties and their true etiology, we will, in the first place, devote a few thoughts to the elementary structure of the pulmonary organs. The parts of the respiratory organs which are principally affected in pulmonary phthisis are the alveoli or air cells. The walls of the air cells are composed of fibrous connective tissue, which is completely ramified by capillary blood vessels and lymphatics. On their external surface, or the surface which is in contact with the atmosphere, they are lined with a flat or pavement epithelium, and these are the elementary bodies which are principally involved in catarrhal phthisis. Between these epithelial cells there are stomata, or true orifices, which communicate freely with the lymphatic vessels in the alveolar wall, and it is through these openings that carbon particles and other foreign materials in a fine state of subdivision gain access into the lymphatic circulation, and produce the well-known discoloration of the lungs. The lymphatic vessels are distributed, in their course, around the blood vessels and the bronchi; those which wind around the blood vessels are called the peri-vascular, and those which wind around the bronchi are called the peri-bronchial lymphatics. These are the structures which play such a pronounced part in the production of true tuberculosis, and their importance must not be lost sight of. We have, then, presented for consideration, in this connection, the alveolar walls, covered on their outside with epithelium and ramified internally with blood vessels and lymphatics.

Now, catarrhal phthisis is generally an extension of chronic bronchitis into the alveoli, or is the product of acute catarrhal pneumonia. In either case it implies a catarrhal affection of the alveolar epithelium. The blood vessels become engorged, and the epithelial cells multiply and accumulate and clog up the alveoli with their products. The filling up of the alveoli with these catarrhal aggregates produces small bodies which partake of the shape of these cavities. In this way one alveolus fills up after another, until a whole group or cluster of them is involved, giving rise to roundish nodular bodies which are so frequently mistaken for true tubercles. They are not tubercles in the technical meaning of that term, but are merely *accumulated inflammatory or catarrhal products.* This train of pathological changes is due to a disturbance of the relationship existing between the production and expulsion of epithelial products, *i. e.*, the expectoration did not keep pace with the proliferation. If such a relationship were preserved, or could by any means be restored, it is evident that the disease would be at once called into a state of abeyance. But the continued accumulation of excretory products exerts a pressure on the capillaries in the walls of the alveoli and in the interlobular septa, and in due course of time these infiltrated spots, thus cut off from their source of nourishment, will give rise to changes of a different pathological character, which will be discussed after we have disposed of another question which has an important bearing on our subject.

It has already been stated that catarrhal phthisis is evolved from catarrhal pneumonia, and the question arises here, why only from catarrhal, and not from croupous, pneumonia? As well as the other question, when does catarrhal pneumonia become catarrhal phthisis? In regard to the first question, it can be answered that croupous pneumonia seldom, if ever, passes into catarrhal phthisis, because its etiology and pathology rest on an entirely different basis from that of catarrhal pneumonia, as the following comparison of their chief characteristics will show: In croupous pneumonia the blood pressure is suddenly elevated, the blood vessels become intensely turgid and injected, the heart-beats become vigorous and powerful, fibrin leucocytes and red corpuscles exude from the more porous arterial walls into the alveolar cavities, where the whole assumes a semi-solid infiltration, undergoes a retrograde, fatty metamorphosis, becomes resolved and expunged in a short time, after which the disease comes to an abrupt termination. Catarrhal pneumonia pursues a different course. The disease comes on gradually and does not pass through the well-defined stages which mark the course of the croupous form; the tone of the circulation is reduced, and the whole constitution is in rather an adynamic condition; there is, as a rule, no exudation of fibrin, but instead the alveoli becomes filled with cast-off epithelium, leucocytes and some red corpuscles. These products have a strong tendency to undergo cheesy degeneration, and owing to its undecided progress and course it is very apt to become chronic, *i. e.*, to leave a vestige of disturbance here and there throughout the lungs, which, upon the slightest provocation, is fanned into freshness again. Again, it is important to observe the respective portions of the respiratory organs which are attacked by the two diseases. It is but rare that pure croupous pneumonia attacks an apex, unless it involves a whole lung, but it always shows a preference for the basic portions of the lungs, and involves either a whole or two lobes. On the other hand, catarrhal pneumonia shows a disposition to attack small portions of lung, such as one or two lobuli, and if it shows a decided preference for any locality, it is the middle or upper portions of the lung. This is particularly true of its chronic form.

Whether the difference in the nature of the pathological products in the two diseases—the one being a fibrinous exudation, and the other a catarrhal secretion—has any influence in determin-