advanced stage of tuberculosis, and are very marked in the acute form. They are of spheroidal shape, and somewhat irregular outline. Great stress has been laid on them as significant of tubercle, but they have been met with in various tissues of the body, in scrofula, in syphilis, and in merely hyperplasic lymph-glands of those perfectly free from tubercle.* As they grow they send out long, branched processes. With Klein, I believe them to be excessively developed or fused epithelial cells.

The structure in which all these cellular elements are found, especially, perhaps, the last described, is *a fine net-work* like the fine trabecular net-work in the interior of lymphatic glands; and this led to the belief entertained until lately by Rindfleisch, that tubercle is a lymphoid growth. But this is not stating the whole of the manner and arrangement of the cells in tubercle. They are found in the lungs filling the alveoli and infiltrating—generally as small round cells—the alveolar walls, and leading to very considerable thickening of the latter.

To sum up, cell-growths by themselves, not peculiar, but representing different grades of development, — some still rapidly growing, others shrivelling and full of dense matter; all capable of being washed out of a fine reticulum, or accumulating in masses both within and in the walls of air-vesicles,—this structure, this grouping, may be regarded as tubercle. Then there are certain secondary alterations that take place in the tubercle formation and the invaded tissue which must also be mentioned, and which bespeak a retrograde change and low vitality. The main of these changes is a degeneration of the cell-growths, an accumulation of granules and fatty material, and an occlusion of the pulmonary capillaries, probably from pressure, and here and there a fibroid transformation of the giant cells.

Now, what causes all this? Some still maintain a specific non-inflammatory deposit; some say an inflammatory process of slight intensity, others a specific inflammation. I pick up a recent journal, and see that malaria is at the bottom of all this cell-growth and rapid decay. I turn to one of this month, and I find in the front of the periodical an article proving that tubercle has its origin in disorders in the trophic centres, and in the middle pages another, showing that it is an accident, the result of the capillary interference, due to altered condition of the blood from the presence of yeast. It is almost needless to say that the bacteria are made to explain the peculiar formations, for how could these patient little beings that are bearing so quietly being made the scapegoats of the pathologists of the last half of the nineteenth century escape having charged to them this additional sin? I turn with eagerness to discussions of the subject replete with learning in societies similar to ours, and there is little but negation. It is not this, and not that, say men who are known wherever medicine is cultivated; and you begin to doubt if there is such a thing as tubercle at all, until the first clinic-room you go into—and see the familiar face, hear the cough, and recognize the well-known signs—confronts you with the stern reality of the awful disease. With all these doubts and gropings after the truth, I may be pardoned if I hold fast to the belief that the process, whatever it be, is something special, though something of which we do not hold the key.

To return from this digression to one part of the subject around which much of what is positive in our knowledge has clustered, and which is of most obvious applicability,—the relation of these mysterious tubercular formations to inflammation.

Now, we all know how the relation of tubercle to inflammation has engaged the attention of the present generation of pathologists. Yet the consideration of the question long antedated them; and the much-neglected observations of that sagacious thinker, Addison, are really the key-note to many of the views now brought forward under other names. But this is a historical issue, with which we cannot further concern ourselves here. The active discussion of the matter started with the observation of Virchow that the caseous matter previously regarded as infiltrated tubercle might originate from the fatty degeneration of diverse morbid products, and was non-tubercular; indeed, that the gray granulations alone were tubercular and non-inflammatory. Niemeyer expanded this thought, and engrafting on it the doctrine

* Weiss, Virchow's Archiv, lxviii.