

section through a grey miliary tubercle with that of true adenoid tissue, we find in the latter a more regular arrangement of the small round cells of adenoid tissue—an absence of branched leucocytes, the presence in their places of large oval endothelial cells peculiar to adenoid tissue, and lastly, what I regard as the most reliable differentiating test between tubercle and adenoid tissue is that when sections of the latter are shaken with water in a test tube the cells separate, leaving a perfect reticulation of reteform tissue destitute of cells, whilst in the former, although shaken until the tissue breaks down, the cells never leave that fibrous meshwork in which they are held. I do not agree with those who regard tubercle as a modified adenoid tissue, and much less do I agree with those who regard tubercle as being always associated with endothelial cell proliferation. Who can deny the frequency with which tubercle originates within the walls of the alveoli of the lungs altogether removed from any endothelial cells? Many have quoted Klein, (the original discoverer of the endothelial cell) as an advocate of this latter view, but in this he is misquoted. In his treatise "On the Relations of the Lymphatic System to Tubercle," he states, that after the appearance of the tuberculous change in the alveoli, the lymphatic trunks become enlarged in the neighbourhood of the blood vessels, this enlargement being in all probability due to the presence of inflammation. The character of the tubercular deposit in the lung is, that it never leaves the alveolar wall.

Let us contrast with this the exudation in catarrhal pneumonia. In the latter we have a large quantity of fibrine, newly organized, staining in hæmatoxylin solutions of a pale green colour. The fibrinous bands are thick and opaque. In the meshes of the fibrinous framework are leucocytes, large, free from pressure, and usually abundant. This mass of exudation is usually balled up in the centre of the alveolus. How much then do these two products resemble each other in their structural features? A little longer stage of development given to the pneumonic exudation, so that its fibrine might become firmer and more highly organized, its leucocytes more contracted, and their protoplasm more condensed, completes their transformation into the tubercular cell. Given a constitution where the absorbents are less active, so that inflammatory exudations are allowed to re-

main unabsorbed, and you have those conditions required to convert a simple inflammatory exudation into a mass of true tubercle.

I was recently asked to examine, microscopically, a large mass of exudation found upon the parietal peritoneum of the abdomen. The mass was about $1\frac{1}{4}$ inches in thickness at its thickest part, and about 3 inches in width; it extended from a point corresponding to the position of the umbilicus downwards toward the right ilium. Scattered through the intestines were masses of *tubercle*. Tubercles and enlarged mesenteric glands were found in the mesentery, and masses of lymph gluing together the coils of the intestines. I thought the mass in question was a mass of dry lymph, but on examining it microscopically I found it to be composed mainly of lowly *organized fibrine*, abundance of large branching leucocytes and a number of small, round, regularly-shaped nuclei, identical with the tubercle cells. This was not one of those masses of adenoid tissue so frequently met with in the peritoneum, since it was markedly different in its histological characters; neither did it respond to the test as previously mentioned. This I submit as an example of an exudation standing midway between simple inflammatory exudation and a complex tuberculous formation.

From these observations it is plain to my mind the close connection which exists between the products of simple inflammation, which in most constitutions are so ready to undergo absorption and entire removal, and *tubercle* which seldom if ever is absorbed and removed.

Secondly—As to how tubercle is produced? Niemeyer answers the question in reference to the lungs, by saying that "when tubercle appears in the lungs it is always as the changed product of a previous pneumonia." In reference to this point I may be permitted to submit a few experiments made upon rabbits by inoculating them, by injecting into their blood—old inflammatory products. The experiments number eight, and consisted of injecting into the jugular vein of a rabbit about 3ss of caseous lymphatic gland dissolved in saline solution and milk. The solution was slowly injected, the puncture allowed to heal, and the lungs examined after a period varying from two to ten weeks. At the end of these periods inflammatory products were found in the lungs—those of the earliest stages were the products of simple inflam-