

of his individuality, might make his case read:—A. A. B B c d e f g, etc., and this would obviously be called A B. Is there any ground for calling A B and C H different diseases? None whatever. The name given is that particular one suggested by the most prominent feature of the case.

In early days the picturesque, if slightly personal, method of naming men was from their individual characteristics; two brothers might resemble one the other, in a hundred characteristics, yet the one might be named from his large nose, the other from his red hair, and their names would fail to suggest their relationship.

To give a concrete example, it may be said that we speak of *fibrinous*, *catarrhal*, *fibroid*, *cheesy* and *purulent* pneumonia, laying stress upon the differences and failing to insist upon the resemblances of one to the other. We speak of syphilitic gummata and tubercles, as if they were two widely different forms of disease instead of two perfectly parallel processes. We had thought at one time that the existence of the giant cell in tuberculosis was characteristic of that disease. Since giant cells exist in syphilis and in chronic inflammation of a simple character, are we to suppose that Nature has deprived us of a useful aid in the diagnosis of tuberculosis? On the contrary, she has rather indicated the very close parallelism which exists between these three.

Before attempting, then, to understand or teach the pathology of tuberculosis and syphilis as separate diseases, it is absolutely essential that we remember that these are separate examples of the same process, that the features of one are reproduced in the other, that both, in short, are inflammations of a chronic type. One may go further, by making comparison between an acute abscess and a caseated tubercle, for he will find that the old formula of inflammation with its sequences holds in both; in the acute abscess it will be a b c d, etc., and all may be complete in two days, whereas in the tubercle it will be a a b b b b b b c c c d d d d, etc., and the process may require two months. Why the former is rapid and the latter slow may depend upon many circumstances, but the most easily grasped idea, is that the toxin in the former may be of strength A 10, and in the latter M 1.

A workable idea of toxin-power is the following: There are different degrees of toxin or irritant: First there are those which kill the cell as soon as they touch it, which "strike it dead"—formula A 50; second, those which do not kill at once, but send the cell into "a decline," which we describe as the sequence of events, roughly indicated one after the other by the conditions of cloudy swelling, granular swelling (or granular degeneration), disintegration, and final death; this second-grade of toxin may be designated M 1; thirdly, the weakest toxins,—formula S. 5, have apparently no power to injure the cell, but rather