THE EVOLUTION OF TUBERCULOSIS IN MAN.*

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It is sometimes necessary to apologise for dealing with this disease, for the old opinion that tuberculosis is far too frequent to be interesting is still occasionally expressed. On the other hand, tuberculosis has lost much of its horrors; it is a declining disease at least in civilised countries, although sudden outbreaks in turbulent periods are not out of the question—as the example of the Great War has shewn. Yet tuberculosis has still retained immense interest for the medical profession, especially in general pathology. It illustrates well the borderline between normal life and disease, between infection and infectious disease, between predisposition and resistance. What does this mean? It is a simple and well-known fact that many, probably the majority of us, harbour in our bodies the living infectious agent, the tubercle bacillus, i.e., are infected, but do not develop pathological signs. We live our normal life, yet as bearers of viable tubercle bacilli we might develop disease at any moment; we are infected, yet we do not suffer from infectious disease; some of us are predisposed to succumb to tuberculosis, some of us are resistant. Tuberculosis therefore offers an excellent field for studying the borderline between what we call "normal life" and the "pathological" condition.

What converts a simple infection into a "morbus," into disease? A quiescent, even uncalcified, lesion may be an interesting pathological phenomenon, but it usually does not affect the normal life of its bearer at all. What causes such lesions to flare up, to become active and progressive, or, in morbid anatomical language, why does it liquefy and produce a cavity, the centre of danger, the source of disease and consumption? I shall not specially deal with this question here, but with another problem of the pathology of tuberculosis which deserves a great deal of general attention.

It is quite true that a tuberculosis specimen as such is far too common to attract the attention of the pathologist. Every student knows fairly well of what elements a tubercle or a caseous focus consists. But it is no longer the single focus which offers the points of interest; it is the type of the total of lesions

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appearing in various periods of life to which we should pay attention. What I mean by "total of lesions at various periods of life" can be shortly explained by saying that these typical lesions are:

1. The primary complex.
2. Disseminated or generalised tuberculosis; and
3. Isolated pulmonary or bronchogenic tuberculosis.

These are the fundamental types in which tuberculosis appears in man. They appear no matter what are the individual circumstances, such as race, constitution, age, fate of the individual: irrespective of whether disease develops or not, whether healing or death occur. The primary complex, i.e., the lesion at the portal of entry of the bacillus combined with a homologous focus in the regional lymph gland, is the anatomical change which invariably develops in an individual who is being primarily infected. This often occurs in youth and often heals by calcification; but a fresh primary complex appearing in adult age or the primary lesion producing progressive disease is characterised by the same fundamental features as the primary one, as the healing or childhood complex. A post-primary lesion does not show the typical combination of a focus in the organ and in the regional gland. Disseminated hæmatogenous tuberculosis may lead to death from acute miliary dissemination; it may also produce a small spread of calcified nodules in various organs—the fundamental feature of hæmic dissemination over more than one organ is common to both of these conditions, so different in prospect and outlook. And, finally, although one person may die from consumption and the other may develop a quickly-healing lesion limited to the apex of the lung, yet both of them develop the same type of tuberculosis, namely, an isolated pulmonary lesion. Primary complex, hæmic dissemination and isolated pulmonary tuberculosis are therefore the fundamental forms in which tuberculosis might appear in individuals of every age, race, constitution and fate.

What I particularly want to discuss is the question of how these fundamental forms are connected with each other. Everybody infected with the tubercle bacillus will first of all develop the primary complex. There is no doubt about that. But what is its relation to the post-primary forms? How often and when does dissemination occur? Does the latter directly follow the primary complex? What is the relation between disseminated and isolated pulmonary tuberculosis? Does disseminated tuberculosis precede the latter, and if so, how often and in what form? What is the source of the isolated pulmonary tuberculosis? Is it a re-infection from without (exogenous re-infection) or a hæmic metastasis from within (endogenous