THE ORIGIN OF BRONCHOGENIC TUBERCULOSIS IN THE ADULT

PATHOLOGICAL ASPECTS OF "EXOGENOUS AND ENDOGENOUS REINFECTION"

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I still think that work on tuberculosis should be based on morbid-anatomical findings. At any rate, these should be taken into consideration and not simply ignored.

Significance of the Primary Complex

Morbid anatomists agree that a primary complex is found in almost every case, regardless of natural resistance or any other condition except that primary infection has taken place. This applies particularly to recent infection in young adults. In these the actual primary foci are often very small —pin-point or pin-head size. It may take the anatomist a long time to find them, but he will do so if he persists. Such foci obviously do not show up in skiagrams. Wallgren (1938) has shown that the primary focus cannot be seen in the radiograph unless calcified or forming an infiltration. *Nevertheless the primary complex is there,* and it is there regardless of age, race, or constitutional type, provided that the person was primarily infected.

Late Primary Infection and Bronchogenic Tuberculosis

Late primary infection is prone, after a short time, to cause an Assmann focus.* This has been shown by the investigations of Malmros and Hedvall (1938), A. S. Hall (personal communication), Kayne (1942), Simmonds (1942), C. B. Thompson (personal communication), and others. While it is theoretically possible that these Assmann foci are due to a new "exogenous reinfection," their quick succession upon primary infection and the interposition of small disseminated, notably apical nodules (so-called Simon foci), as shown by Malmros and Hedvall, suggest that all these are manifestations of the same infection. The findings of Malmros and Hedvall are in remarkable conformity with the morbid-anatomical observations. The latter (see Pagel, 1942) have shown that a primary complex—old or fresh—precedes bronchogenic tuberculosis in *all* cases, adults as well as children.

The Evidence for "Endogenous Reinfection"

In support of this theory we used to adduce two facts: (1) The pictures of recrudescence of old especially calcified foci. I agree that about 70% of primary foci sterilize themselves after some years. Yet the occurrence of more than one primary complex in one person remains the exception (see figures: Terplan, 1940). Sterilization is less effective, and takes a longer time in calcifying *post*-primary foci—and it is these which are outstandingly incriminated in bronchogenic tuberculosis due to recrudescence. (2) The common occurrence of solitary Assmann foci in cases of disseminated tuberculosis in which all manifestations are blood-borne.

But what about the cases without recrudescence? We now know that many cases are actually *late primary* infections with bronchogenic tuberculosis ensuing within a short period (see above). The quick succession of primary infection and early bronchogenic tuberculosis with interposition of the typically haematogenous Simon foci is no proof, but strong circumstantial evidence in favour, of the three focal changes (primary, apical, and infraclavicular) being manifestations of the same process; in other words, the post-primary changes are affiliated to the primary focus—they are due to haematogenous dissemination from it.

Endogenous reinfection by recrudescence of calcified foci is therefore of less importance than it used to be at a time when the majority of primary infections were acquired in childhood. Endogenous reinfection nowadays does occur from comparatively fresh primary foci.

Some recent investigations (see review of literature, Daniels, 1944) are based on clinical data and the rabbit experiments of Lurie (1941). Their finding is that in adults no primary infection is visible in the skiagram, and the process apparently begins at once as bronchogenic tuberculosis. The absence of a primary complex and disseminated tuberculosis is interpreted as a sign of natural resistance, as observed in certain inbred rabbits and in white patients in contrast to negroes.

It is not unlikely that, among negroes, primary infection is more conspicuous in the skiagrams, and disseminated tuberculosis more common, than in white patients, and that this is due to lack of natural resistance on the part of the former.† But this proves no more than it does in babies, which show severer infection and more fatal dissemination than older children and adults. Any tuberculous infection runs a severer course in the less resistant.

It does not prove, however, that the type of tuberculosis developing (primary, post-primary, disseminated, and bronchogenic) depends upon natural resistance. Experimental, clinical, and morbid-anatomical evidence goes far to show that almost every primary infection is liable to cause dissemination. This is usually mild, and does not lead to appreciable changes. Disseminated tuberculosis, even if fully developed, shows good healing in many cases. On the other hand, bronchogenic tuberculosis is so often fatal. The number and viability of bacilli causing primary infection, the environment, the nature of the infecting source, acquired resistance (allergy, economic conditions, repeated superinfection), etc., will decide the form of post-primary tuberculosis, if any such manifestation develops. Of course, natural resistance has a word to say therein, in that it will modify the influence of the economic make-up, superinfection, and allergic response.

Restriction of the tuberculous process to the lung may be interpreted as an indication of acquired resistance, since it can be reproduced in the experimental animal by vaccination with heat-killed tubercle bacilli or live organisms of attenuated virulence prior to virulent intravenous infection (Pagel, 1936). The fate of the tuberculous person is decided by his inborn resistance.

Conclusion

Contact (exogenous infection) can play an important part in the genesis of bronchogenic tuberculosis. This may be due to its causing late primary infection, soon followed by tuberculosis, developing from within.

Inborn resistance decides the severity of the infection in the individual. It does not decide the type in which tuberculosis manifests itself (primary, post-primary, disseminated, bronchogenic).

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* I.e., a post-primary caseous change, preferably in the infraclavicular area of the upper lobe, which by its tendency to cavitation and spread via the bronchial system causes the common bronchogenic tuberculosis of the adult. This is restricted to the lungs and, in contrast to primary infection, is not followed by caseous changes in the regional lymph glands. In the rare cases in which bronchogenic spread is caused by a cavitating primary focus ("primary cavity") this is recognizable as such by the presence of caseous changes in the corresponding lymph glands.

† It should be borne in mind, however, that the size of the primary changes bears no relation to the severity of ensuing dissemination. A pin-head primary complex in the lung may be rapidly followed by meningitis or miliary tuberculosis.

The Dublin Rheumatism Clinic Association has issued its first annual report, covering the year 1943. The clinic is situated at 34, Upper Mount Street, under the direction of Dr. T. J. O'Reilly. The association was set up by the Minister for Local Government and Public Health and incorporated in 1942, and its immediate work falls into three stages: (1) the establishment in Dublin of a clinic for the treatment of rheumatic diseases; (2) study of the rheumatism problem based on the experience afforded at this clinic; (3) the planning and provision for the country of facilities for such treatment as are available to Dublin. The first stage of the work has been accomplished, and the report records progress made in that respect as well as indicating some progress in the second stage.