inert when inoculated into the subcutaneous tissues of an animal such as the mouse. Silica and certain silicates, on the other hand, are active irritants, and produce recognizable tissue reactions. Furthermore, the intravenous inoculation of tubercle bacilli into animals prepared by previous subcutaneous injections of dusts results in the settlement and proliferation of bacilli in the active dust lesions to a demonstrably greater degree than occurs in the deposits of the inert dusts. So far as I can see, this method provides a reliable means of determining whether or not any particular substance has the power of enhancing the growth of tubercle bacilli in the body. It should also, I think, enable us to form some idea whether a dust is itself capable of producing lesions in the lung. So far as our knowledge goes at present, inhaled dusts are dangerous only when they become soluble in the body and act as tissue poisons; large amounts of inert dusts cause no pulmonary disability because they are incapable of producing anything more than a minor tissue reaction. It may very well be that the action of a dust on the tissues is considerably modified by the way in which it is presented to them. Dust which is slowly picked up from the surface of the lung by phagocytosis may act very differently from dust which is injected directly into the tissues; nevertheless, its final and fundamental action must be the same. And it seems reasonable to suppose that a substance which produces an active reaction in the subcutaneous tissues will be capable of causing lesions in the lung itself.

This simple method, however, will not solve some of

the more complicated problems with which we are faced. It will not, for example, help us in the study of the action of combined dusts in the lung, nor will it help us to determine the exact degree to which infection enters into the production of pneumoconiosis. For these and other problems we must revert to direct observation on the lung itself. It does not necessarily follow, however, that our investigations should be bound by the limitations of the dusting chamber; there are other ways in which particulate matter may be presented to the lung than in the inspired air. R. Hilton and I⁸ have recently described a method of introducing dust into the lung by direct intratracheal injection. It is not necessary to recapitulate the advantages of this method over the inhalation technique, but so far our results have been so promising as to suggest that we have now a means of investigating experimentally in any ordinary laboratory many of the problems of pneumoconiosis which so urgently demand solution.

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RADIOLOGY IN DISEASES OF THE LUNGS*

BY

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It has always appeared to me that the great value of these Annual Meetings lies in the fact that they afford an opportunity to review the work of the past and to see if recent work has anything of value for us. With this thought in my mind I feel that the advantage of a combined discussion between clinician and radiologist is inestimable. It ought to show us team work at its best; and what is good for us is, I would suggest, good also for our patient. For example, the radiologist is asked to give an opinion on the x -ray appearances, as indicating or contraindicating the production of artificial pneumothorax. This is a question that he is perfectly qualified to answer, but the matter does not end here. The clinician is the sole judge of the clinical fitness of the patient.

The value of radiology in the diagnosis of lung disease has now taken its place as one of supreme importance; indeed, it is difficult to imagine any clinician giving a lecture without illustrating it with radiograms. I know when we have a class at Brompton, and it comes to the turn of myself or my co-workers in the x -ray department to give a lecture, it is an extremely difficult matter to find suitable pictures, our clinical colleagues having taken them all. A. Salusbury MacNalty, in his recent illuminating report, well presents the attitude of to-day in relation to radiography of the chest. He says: "From a former tendency to disparage chest radiograms, the pendulum has swung too far in the other direction. Some enthusiasts diagnose pulmonary tuberculosis solely on x -ray evidence, and ignore the results of orthodox physical examination. This attitude is as mistaken as would be that of the clinician of to-day who, when diagnosing pulmonary disease, neglected to take into account the x -ray picture."

* Read in opening a discussion in the combined Sections of Radiology and Medicine at the Centenary Meeting of the British Medical Association, London, 1932.

PULMONARY TUBERCULOSIS

With regard to the value of x -ray examination in the diagnosis of pulmonary tuberculosis, I think I am not overstating the case in putting it as follows:

1. Definite infiltration can be demonstrated upon an x -ray film at quite an early stage of the disease, and frequently before definite physical signs are present.

2. Consistently negative x -ray evidence, extending over a period of some months, may be taken as conclusive proof of the non-existence of pulmonary tuberculosis.

3. In cases in which physical signs are present it may be assumed that the initial stage has passed, and, in such cases, the x -ray picture will as a rule show much more extensive disease than can be demonstrated by physical examination.

As to the incidence of pulmonary tuberculosis, I think there is still some cleavage in our ranks, more especially in relation to the part played by the mediastinal glands. Leaving out the epoch-making researches of Stanley Griffith on the question of bovine tuberculosis, I should like to find some agreement among us on the important question of lung tuberculosis in the infant and in the adult. I should like to make confession of what a long period of work at Brompton Hospital has led me to believe. I believe that the tubercle bacillus gains an entrance into the lung by "aspiration" in the vast majority of cases, and it is also my belief that, in the adult, any further invasion is of the nature of a "reinfection" (that is, exogenous). Against this view is that of many deep thinkers, past and present, who regard pulmonary tuberculosis much as they regard another of the infective granulomata, syphilis—namely, that one stage of the disease follows another in regular sequence, and that, even after a prolonged period of apparent inactivity, further manifestations may reveal themselves. To take this view is, I suggest, to disregard much of the work that has been done of late years upon the allergic state—the acquired reaction peculiar to the previously infected person.

In the infant the incidence of pulmonary tuberculosis would seem to depend upon the initial dosage and the

degree of resistance. If the dose be great and the resistance low, then we get an acute caseous bronchopneumonia (infantile phthisis), so rapid and so fatal as to be practically hopeless from the onset. If the dose is small and the resistance good, then the usual course is for the infant to get a primary lung focus in the lymphoid tissue found in the region of the smaller bronchi, with resulting response in the tracheo-bronchial glands. The work of Canti and Ghon in relation to this is so well known as to need no repetition. The small focus may be situated in any part of the lung parenchyma, and usually gives little or no clinical evidence of its presence. In due course the lung focus clears up and may entirely disappear, or it is revealed as a small oval or rounded dense opacity—a calcified nodule. The bronchial glands calcify in similar fashion, and the story is ended. On the other hand, the gland may not behave in this way. It may undergo extensive caseation and burst either into a bronchus or into a blood vessel. We may then get an aspiration infection in the case of the bronchus, or a miliary tuberculosis in the case of the blood vessel. In the former case the history is much as we might expect, depending upon the resistance and dosage—truly a “vicious cycle,” as Professor Still has remarked.

Yet there is a school of thought that would ascribe all these changes to the glands themselves. As well might one argue that a thecal abscess was caused by a mass of glands in the axilla and not vice versa. Why should we submit the lung to a different pathology from that obtaining elsewhere?

Finally, just as we get a focal reaction in the region of a tuberculous gland elsewhere in the body, so we get a similar reaction in the area of a tuberculous gland in the mediastinum. Though this condition has been discussed under different names by many clinicians and pathologists from the time of Laennec and Virchow, the reaction is the same. It may be quite evanescent, disappearing within a few days, or it may be persistent or even recurrent. Clinical evidence may be slight, but there is usually some pyrexia and general malaise, and the infant reacts positively to tuberculin. This, I would submit, is evidence of local infection, and not of an active lesion. The x-ray evidence is definite, and shows an area, usually wedge-shaped, extending in any direction from the inflamed gland into the surrounding tissues. After a variable period of time this local area disappears entirely. To call such a condition by the name of pulmonary tuberculosis, with all that such a diagnosis entails, seems to me wrong.

TERMINOLOGY

I should now like to say a little about certain matters of importance, not only to the radiologist, but to the clinician.

I would press for the general adoption of the word “infiltration” as describing a parenchymatous invasion by the tubercle bacillus. I am quite aware that in the strictly pathological sense this may not be accurate, but there is no other term that so aptly expresses what is in the mind of the radiologist. “Mottling” will obviously not suffice, for this word is much more applicable to the appearances seen in miliary tuberculosis, in the early stages of pneumoconiosis, and in the coarse, mottled appearance seen in a miliary carcinoma. To apply the term “infiltration” to every departure from the normal is equally wrong, and increases the already great difficulties of reasonable interpretation.

The term “fibrosis” ought, in my submission, to be used in a wider sense than has recently been the case, as describing a condition of healing in which there is definite deviation of the mediastinal content to the affected side. In a parenchymatous invasion of the lung the first evidence of healing is found radiologically in the somewhat

ill-defined and cloudy appearance becoming more linear in character, and this without any suggestion of deviation of the trachea or mediastinum. Such expressions as “active” or “healed” ought to be used with caution, or never used, seeing that such conditions are clinical rather than radiological. It is reasonable, in the state of our present knowledge, to draw a distinction between a “recent” and an “old” lesion, for the difference to the trained radiologist is very definite, and the value to the clinician is definite also.

STANDARDIZATION

With the advent of modern apparatus and the ease with which we can reduce shadow deformity by radiography at two metres distance, or even less, there has emerged a tendency, which is quite natural, to adopt the upright posture for chest radiography. Failing this, the patient may be laid in the prone position on a couch with the film (cassette) on the front of the chest and the tube at the requisite height above. The advantages are:

1. That the early screening can be done and the tube racked out for long-distance radiography without moving the patient.

2. That fluid levels in a cavity or in pleural effusions can be seen.

The disadvantages, equally definite, are:

1. That it is not possible to centre the tube with such accuracy as to obtain an identical picture months or even years after the original picture has been taken.

2. That it is impossible to deal with the ill patient, say, one who is on absolute rest following haemoptysis.

3. That the female breast is dependent and consequently throws a much more obstructive shadow than would otherwise be the case. It was with regard to these disadvantages that at Brompton Hospital, when we were constructing a new department, we wished to have all the advantages of distance and still be able to deal with the large numbers of stretcher cases. To my colleague, R. L. Rawlinson, I owe a suggestion which rapidly cleared up our difficulties—namely, that of cutting a hole in the floor. We discovered that a hole 1 foot square was amply sufficient, and it is easy to see how simple the matter is, provided only that there is a space available in the room below. As the tube is fixed the necessary shift of couch, which runs on rails and has a movable top, has to be provided for. The centring is done by means of a plumb-bob. There is one disadvantage only—namely, the fact that screening is not possible except for an instant of time.

OTHER PROBLEMS

As I am to be followed by a number of other speakers, I will not anticipate what is going to be said, save upon one or two points. As with the rest of enthusiasts, there are very many questions for which I look for an answer to-day. In lung abscess, for instance, what is the proportion of direct infections as compared with the great number that are of embolic infection, particularly in lung abscess following tonsillectomy? Why does “A,” with marked infiltration of both lungs, resent being invaded, while “B,” with a very slight infiltration of one zone, feels very ill and readily tired? Shall we find agreement on the still vexed question of the most suitable method of administering lipiodol in the diagnosis of lung conditions? These are just a few of the questions, and, knowing as I do the work and reputation of the speakers, I feel confident that the answers will be forthcoming.

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