SOME CLINICAL CHARACTERS OF TUBERCLE

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THERE are certain clinical characters of tuberculous disease which do not get enough attention ; they are not brought out collectively, a proceeding possible in nearly every form of the disease, and one which seems to result in a strengthening of the outlines of our general impression of tuberculosis. Such characters are: the existence of a predisposition or diathesis, the ætiological influence of trauma, the phenomena of muscular spasm and atrophy, the vicissitudinous course, and, finally, the strong tendency to recidivism. These five are taken as being at present inadequately recognised, which is the same as saying that otherwise one would not be justified in troubling the reader with them. But to deal here with all would be impossible ; we shall have plenty on our plate with the last four only.

Trauma as a contributory cause of tubercle is fully acknowledged in some varieties of the disease. The common antecedent, in bone and joint lesions, of a knock or sprain is mentioned in all general text-books ; and so is the common history of a rather bad fall preceding meningitis. But only in the fullest of them does one read that direct violence often precedes tuberculous peritonitis ; and even the writers of the latter have not made up their minds whether injury to the thorax favours the evolution of phthisis. This is strange, for specialists have long been clear on that point. Brehmer [1], writing in 1885, quotes earlier monographs in which surprise is expressed at any doubt about it. The phthisis, starting perhaps from a localised contusion pneumonia, mostly supervenes on the injured side, a useful indication in medico-legal work. In adult tuberculous testicle, too, the lesion often dates from an injury to the part, while trauma is mentioned in the ætiology of Addison's disease.

Similarly, muscular atrophy and spasm are classical signs in surgical tuberculosis. Witness the flexor (psoas) spasm, or the "boarding" of a tuberculous spine ; likewise the wasted buttock in hip disease. Unfortunately for patients, they are not yet classical signs in phthisis. Yet the "Schulter-Phänomen" in consumptives, and the drooping on the affected side of the scapula, point of shoulder and nipple, as noted by Krönig [2] (which is generally accompanied by distinct wasting of the trapezius), are known to those abreast of the literature of tuberculosis. So are Fischer's description of the increased irritability of the thoracic muscles overlying tuberculous foci, and Pottenger's description [3] of spasm and degeneration of the muscles of neck and thorax in pulmonary tubercle. In the case of phthisis it is surely allowable to connect all these signs, to say that a tuberculous lung apex is often accompanied by fibrillary contraction, or by atrophy, of the neighbouring muscles, and that whether or not the "degeneration" and "pulpy change" that Pottenger describes occur in those muscles, certainly atrophy often does — which atrophy seems a likely cause of the characteristic local deformity seen so often when its appearance is known, for it is plausible that the wasted trapezius allows the shoulder to sag.

Nervous influence—a reflex irritation from the chronically inflamed lung tissue, in which the sympathetic, the spinal cord segment, and the motor nerves to the muscles are concerned—may indeed be responsible for these changes, since pain in the shoulder often occurs, and a diminution of pigment in the areola of the corresponding nipple, which, by the way, is sometimes smaller than its fellow. As for the muscular spasm of surgical tuberculosis, what is usually advanced as its cause is that it is protective, being Nature's way of securing rest of the joint affected. There are several objections to this theory. Spasm may be seen, so it is said, in the neighbourhood of tuberculous cervical glands. Again, protective immobilisation is no way of accounting for muscular atrophy, which would make a joint looser rather than firmer. Further, such an explanation as "Nature's way of securing rest," is teleological, not pathological. Certain processes of inflammation may by natural selection have been evolved to therapeutic ends, but we need a closer explanation than that. All good clinicians agree that the wasting of the neighbouring muscles in *morbus coxæ* cannot be primarily traced to disuse, as it is so early a physical sign. It is certainly an early one in phthisis, and moreover can arise fairly quickly. In three cases in which I have had opportunity of timing its appearance, in all it occurred in under four months, and in one instance within ten weeks. This speedy occurrence is against the atrophy being a sequela of continuous spasm in the way that atrophy of any tissue ensues upon continuous pressure. On the whole the nervous influence theory of Pottenger applies best to the "Schulter-Phänomen" (to take that as a useful collective term)—and it may also apply largely to the muscular phenomena of surgical tubercle. For the point I would urge, not having seen it elsewhere raised, is this: that these two things are one; *that the muscular spasm and atrophy with a tuberculous joint are, in all probability, much the same as those with a tuberculous lung apex*; although at the same time one would not deny weight to the idea of protective immobilisation, seeing that muscular spasm is much more in evidence round a diseased joint than round non-articular bone lesions, or indeed round an affected apex.

One cannot leave this subject without mentioning its high diagnostic significance.

Here is a case in which right-sided trapezius atrophy and shoulder drooping constituted almost the only physical signs; there were in addition very slight prolonging of expiration just below the right clavicle, and a trifling narrowing of the right band of apical resonance, of course highly equivocal findings. On the strength of this deformity I sent the patient several years ago to the sanatorium, where the resident made out the right lung to be clear, and marked something out the left apex. She relapsed not long after her return from there, and a year later nothing seemed to be present on the left side, but over the right upper lobe there was loud bronchial breathing with râles.

The naturally up-and-down course of tuberculosis, independent of treatment, and its tendency to relapse, are the most characteristic clinical features of all. They hold good in skin tubercle as well as in scrofulous, osseous and pulmonary lesions, which the two characters first described do not. In fact, they probably apply to almost every form, miliary infection in children not excepted. "In later childhood there is this to be reckoned with, that as in the case of adults occasionally complete healing of a miliary attack or of localised lung disease occurs, in other cases recovery with sub-

sequent recrudescence of the disease." ¹ To this characteristic is due the fact that tuberculosis has made the fortunes of so many quacks and semi-quacks, and has deluded honest men until there is "no drink or stink" which has not been vaunted as a remedy for it. The fallacy of *post hoc, propter hoc* is nowhere so potent as in phthisio-therapeutics. Recidivism has long been recognised in surgical tubercle, but only lately in phthisis, for the good reason that until lately practically no consumptive recovered, so that practically none could relapse. The advent, some twenty years ago, of the hygienic-dietetic treatment, in other words, the sanatorium, arrested lung tubercle in many persons; and when some of these, as might have been predicted, lost their health again from a lighting-up of the disease, the sanatorium came in, rather unfairly, for a chorus of

blame. But the results in surgical tubercle were also marred by recidivism. Not so much so; for the consumptive, who is the commonest tuberculous patient, is unluckily also the one most prone to backslide. The main reason for the irregular course, either upward or downward, of tuberculosis is of course the exactness of its inverse variation with the subject's general health, receding when that is improved, and *vice versa*. But it is not the only reason. An exacerbation of scrofula will occur at times with rosy cheeks. A tuberculous larynx will get worse when the lung is mending and the patient putting on weight, or improve under opposite conditions; lupus will heal in one dying of consumption or hip-

¹ *Handbuch der Therapie der chronischen Lungenschwindsucht*, Schröder u. Blumenfeld, Leipzig, 1904, p. 960.

disease. The explanation of these last instances is, however, probably purely bacteriological.

These four clinical phenomena—Trauma, Muscular Signs, Vicissitude, Recidivism—to what do they point? Probably to high natural immunity to the disease. I should not be surprised if they were rare in tuberculous lunatics or in patients of some non-Western races, like negroes or Polynesians. It would also be interesting to hear from a syphilologist or leprologist of anything analogous to be found in the other infective granulomata.

REFERENCES.

- [1] BREHMER. "Die Aetiologie der chronischen Lungenschwindsucht," 1885, 401.
- [2] KRÖNIG. *Die deutsche Klinik am Eingange des XX Jahrhunderts*, 11, 581.
- [3] POTTENGER. *Beiträge zur Klinik der Tuberkulose*, Bd. 20, H. 1.

SHORTER COMMUNICATIONS.

THE "PRETUBERCULOUS" CHILD.

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"PRETUBERCULOUS" is a somewhat indefinite term, hard to define clearly and capable of a wide interpretation; there are indeed a few noted medical men who deny that such a condition exists and maintain that there is no half-way house to tuberculosis. In my own experience the weight of evidence has been sufficiently overwhelming to convince me of the existence of such a condition, of its prevalence amongst children, and of its vital importance.

DEFINITION.

It is of course well known that if from any cause a child's vitality remains materially lowered, not only tuberculosis but any other illness is especially prone to occur, though from this reason alone no child should be considered pretuberculous. But if in addition some or all of the following conditions persist, viz., frequent rise of temperature from some indefinite cause, enlarged lymphatic glands, loss of weight or failure to put on weight, chronic bronchial catarrh, loss of appetite, carious teeth with stomatitis, or general strumous condition, then such a child might well be termed pretuberculous; more especially if with the foregoing is associated a family history of tuberculosis, or "contact" with a tuberculous person. Though incomplete, the above statement will serve as a rough guide as to what is usually meant by a pretuberculous child.

PROGNOSIS AND TREATMENT.

Not all such cases develop tuberculosis, as a certain percentage of them have a high degree of resistance and readily respond to ordinary treatment and care. What however is practically sure is that the majority of such