TUBERCULOUS PHTHISIS.*

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AMID the various opinions upon phthisis, it seems absolutely necessary to give up the idea of Laennec and Louis, that "tubercles in the lungs should be recognised as the anatomical character of phthisis". Further research and more accurate observation enable us now to class under the head of phthisis several tolerably well-known diseases, and especially those forms of chronic pneumonia which Dr. Addison described and figured beautifully long before Niemeyer published anything on the subject. But the questions which I would venture to propose for our consideration, are—How far are we to go in this direction? To what extent are these new views in accordance with the clinical experience of many of us? The data for discussion are contained in Niemeyer's monograph upon Phthisis, and would include the following propositions.

1. Chronic catarrhal pneumonia is one of the very commonest diseases, and is not particularly dangerous, under appropriate treatment.

2. The greatest danger to most phthisical patients (*i.e.*, those suffering from chronic catarrhal pneumonia) is the development of tubercles.

3. If there be only a few tubercles, they are almost exclusively found in the immediate neighbourhood of cavities or cheesy deposits; and they seldom, if ever, exist without there being found somewhere or other in the body cheesy deposits.

4. Tuberculosis is in most cases a secondary disease, originating in some way unknown to us in the action of cheesy morbid products on the organism.

5. The connection is probably a local influence, in which perhaps the lymphatics take a prominent part, rather than an infection of the blood.

Niemeyer's views seem to me to be too much confined to the lungs. Although I am very far from allowing Virchow's dictum to be true—viz., that the lungs are the organs least often affected with tubercle—yet in the consideration of this subject it is necessary to look at the disease as it affects the whole system. General tuberculosis, as seen in young children, may affect almost every internal organ of the body. We see it complicated with, and perhaps dependent on, chronic disease of the ear; we see it often enough associated with cheesy bronchial or mesenteric glands; but I confidently appeal to the experience of all pathological observers, when I say that it is not unusual to meet with general tuberculosis without any glandular or inflammatory lesion that can have preceded the tuberculous growth in point of time. Even Niemeyer himself confesses that miliary tubercles are found without any pre-existing cheesy deposit or cavity, though in his opinion this occurs but seldom. I know that the growth of tubercle in many organs after the injection of various substances into the cellular tissue of guinea-pigs, as seen in the experiments of Dr. Wilson Fox and others, will be cited against the blood-view of this disease; but none of these experiments exclude the idea of septic influence, and thus they rather support the opposite of Niemeyer's dictum.

But if this condition of general tuberculosis can exist, say even once in a hundred times, without pre-existing cheesy deposit or other degenerative result of previous inflammatory action, there must be some other factor not recognised by the author of these new views, which may possibly at least influence the development of tubercle in all cases. Do we not learn something of this by studying the mode of death in these victims of tubercle? I do not speak of cases in which the lungs are enormously implicated, and much of the pulmonary substance rendered useless, or of those in which exhausting diarrhoea with intestinal ulcerations may have hastened the fatal event. But what do we constantly see? a patient ill for two or three weeks with symptoms simulating enteric fever or acute pneumonia, and no *post mortem* appearance, save lungs studded with miliary tubercle, but otherwise healthy. It is not from interference with the pulmonary function that such a patient dies. Collect all the tubercles together, and they would not take up the space of half a lobe of one lung—and we know with what amount of lung-spoiling a man may live for years. It is not even from the high fever (bad as that is) that death ensues—for in these cases the temperature often manifests the most startling remissions; it is from the condition of the blood, hopelessly unfit for any of the functions of the body. The man dies, just as one dies in the first week of typhus or variola, before any lesion of the internal organs can be recognised. Take, again, tubercular meningitis. The lungs may be scarcely affected; the lesion of the

meninges so slight that it requires a practised eye to realise it: nay, even in some cases death ensues with headache, convulsions, strabismus, that terrible specific cry, etc., and without any lymph being poured out even over the base of the brain, and without any tubercle on the vessels. To what does such a case succumb? Clearly not to pressure on the brain; clearly not to lesion of any organ. Death must depend on the condition of the blood itself. Similar facts occur to us in connection with tubercle on the peritoneum and pleura.

It is useless to talk to us of special vulnerability of such patients if there be no wound of tissue or organ to be found; but inherent tendency to tubercle, irrespective of all preceding inflammation, is intelligible, and, I believe, in accordance with daily experience.

Let us turn for a moment to chronic bronchopneumonia. It is idle to object to this disease being a true pneumonia because of its occurrence at the apices of the lungs, or because its primary symptoms differ from what we meet with in acute inflammation of the parenchyma of the lung, or because the physical signs are not the same. Bronchopneumonia is much more rich in cell-growth than acute parenchymatous pneumonia. In the latter, the air-cells are filled with an exudation much more rich in fibrine. Acute bronchopneumonia is just as likely to affect the apices of the lungs as the bases, as is often seen in croup and whooping-cough. But between ordinary acute bronchopneumonia and that chronic form that results in phthisis, there are what seem to me specific differences, apart from the question of duration of the disease. The acute form is singularly amenable to treatment; the chronic can be by treatment rendered inert for years, but is scarcely ever cured. The acute form is generally unilateral; the chronic, in a large number of cases, bilateral; the acute form never, so far as is known, hereditary; the chronic not seldom hereditary. These points go some way to show that the latter disease is not merely accidental, but that it depends on some constitutional condition. And when, in addition to this, we find after death miliary tubercle profusely scattered over the portions of both lungs that are unaffected by the original disease, or attacking the pleura or the peritoneum, it is more reasonable to believe that it is a manifestation of constitutional disease rather than a consequence of the previous inflammation.

The very incurability of the disease points, I think, to its constitutional origin. How often do we see the consolidated spots in one and often in both lungs remain in the same condition for many years. Now, under Niemeyer's view, there surely is no need that a large surface of the lung should be so consolidated for infection of the system to take place. The smallest spot of disease would act equally well. But if the disease be of small compass it will remain inert, under suitable treatment, for a whole life. I have lately seen a lung *post mortem*, in which, from the history of the case, we were sure that the small portion of lung affected had become consolidated thirty-five years before. Certainly, in the face of facts like these, if Niemeyer be right at all, that tuberculosis originates in the action of morbid products on the organism, it is indeed, as he says, in some way unknown to us.

Take another instance. There are instances—unhappily too frequent—in which every member of a family becomes phthisical at a certain age. It is not unusual to see large families with one or both parents phthisical, and almost every member of the family will die of phthisis before twenty-one. In a case lately under my care, and in which no tubercle could be detected, although the right lung was everywhere riddled with cavities, there was a strong family history of phthisis, four sisters having died of it.

Niemeyer says that pneumonia, resulting in cheesy infiltration, occurs chiefly in delicate badly nourished persons. This experience is partly founded on the great vulnerability of such persons, and partly on the fact that the inflammatory nutritive changes occurring in them show a tendency to an abundant production of cells, and thereby to a cheesy metamorphosis of the inflammatory products; and that pneumonia of this character does not usually occur, even in delicate and vulnerable persons, before the age at which pulmonary diseases generally become more frequent, and it then takes the place of those inflammatory diseases of other organs which have prevailed during the preceding period of life: and he would also say that, although it is not proved that tuberculosis is an inheritable disease, yet an inherited disposition to pulmonary phthisis may exist; *i.e.*, a weakness or vulnerability of constitution, which in the parents has already been either the cause of pulmonary phthisis, or has only been developed in them by the disease. Now this seems to me to be forcing facts to suit his theory. Clinical experience demands an explanation of the close relation between scrofula and phthisis, considering how large a number of phthisical persons have suffered from glandular swellings in early youth, from which they have entirely recovered, and after death we find in them no caseous degeneration of either cervical, bronchial, or mesenteric glands. So our author falls back upon this delicacy of constitution imparting

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a vulnerability; *i.e.*, increasing the chances of having pneumonia, leaving out of sight—in fact, denying—what seems to be a much more reasonable explanation of the phenomena—viz., that the phthisis is only another manifestation of the same constitutional diseased condition; for we question that the great tendency to pulmonary disease exists more specially at puberty than during childhood. The age at which pulmonary phthisis is most prevalent and most fatal is certainly not the age at which patients are particularly liable to other morbid conditions of the lungs. It would be more in accordance with medical experience to say that childhood showed a greater liability to diseases of the lungs; and, if so, then, according to Niemeyer, the age most liable to pulmonary disease shows tuberculous disease in other parts of the body more frequently than in the lungs.

Take the case of gout. It is a fairly recognised fact that we may inherit gout—and not only so, but that we may inherit it from a grand-parent, although the parents may have shown no manifestation of it. But at what age does gout manifest itself? Certainly not, as a rule, at all ages of life; much more frequently at or after middle life. We do not say that such a person inherits a vulnerability; we say that he inherits a constitutional taint, that manifests itself most usually at late periods of life, probably because then the system is weaker, repair more difficult, the mode of life more conducive to that partial and insufficient metamorphosis which seems to act as the exciting cause of this disease. Is not the fact that phthisis often does not attack its victim until the age of puberty to be explained in a similar way? It is a matter of common observation that childhood is the age for general tuberculosis, tuberculous meningitis, and tubercle of the peritoneum; that puberty is the period most liable to tubercle associated with pneumonia; and that acute miliary tubercle of the lungs is about equally to be found at puberty and between the ages of twenty and forty. But that a constitutional taint should be inherited and manifest no special phenomena until the age of seventeen, and then attack the patient with all possible virulence, is only analogous to what we see in other diseases universally recognised as transmissible from parents to children.

Does clinical observation throw any light on this subject? Certainly the stethoscope does not help us. The physical signs, as gathered by auscultation and percussion, are pretty much the same in tuberculous and non-tuberculous phthisis; or at least, excluding acute tubercle (in which disease the physical signs are mainly negative), the breaking down of lung associated with tubercle, and a similar condition without tubercle, cannot be distinguished by means of the stethoscope. The flattening of the chest may exist with both; the emaciation, the rapid pulse, the hurried breathing, the perspiration, the curved nails, the dyspepsia, and the diarrhoea. Albuminuria, which is often found in acute tubercle, is absent in these other conditions, unless the patients have renal disease unconnected with the pulmonary mischief. The chlorides are never, or scarcely ever, diminished in either disease; indeed, although constantly examining this point, I never but once found any diminution of the chlorides in the urine, though possibly this experience may be due to the fact that the majority of cases come under notice long after their commencement.

Is there any help to be got from the thermometer? Wunderlich says that it is impossible by means of the thermometer to distinguish tuberculous from non-tuberculous phthisis, and it is not easy to express a different opinion, considering his long and careful observations upon temperature. Still, in the progress of these cases, there does seem to me to be a difference in temperature. In the chronic pneumonia of the non-tuberculous subject, the temperature will stand between 100 deg. and 103 deg. Fahr., not necessarily higher in the evening than the morning—indeed, not unfrequently higher in the morning than the evening, but keeping regularly about these points without sudden and irregular remissions. It may also, under suitable treatment, reach the normal point and remain quiet for a considerable time, or can be entirely recovered from any recurrence of the progress of the disease, however, being manifested by an exacerbation of temperature. But the case is somewhat different in tuberculous phthisis. Niemeyer says, "We have not yet arrived at definite conclusions in our investigation of the hectic fever of phthisical patients, especially concerning the causes by which its regular courses are disturbed. But we can already say this much, that in tubercular phthisis in the restricted sense, and when tuberculosis complicates a destructive pneumonia, the differences between morning and evening temperatures are as a rule much smaller. We give, therefore, *ceteris paribus*, a more favourable prognosis as long as the pyrexia has the character of a febris remittens, with almost intermittent type, than when it approaches to that of a febris continua."

Now this is only very partially correct. The height to which the temperature ascends is often in excess of what is seen in chronic pneumonia; but, as has often been said, the temperature in tuberculous phthisis is as irregular as in typhoid fever it is regular. Any one who pays at-

tention to this point will find how often and yet how irregularly the temperature will fall from 104 deg. or 105 deg. Fahr.—five, six, and even eight degrees Fahr., to rise again in the course of a few hours. Except in certain examples of pyæmia, the intensity and irregularity of these remissions will be found in no other morbid conditions, and in certain cases of obscurity they will prove of diagnostic value. The marked difference between this irregularity of tubercle and the comparatively regular remissions of non-tuberculous phthisis will prove useful in the determination of the special form of disease we may have before us, and affords an illustration of the truth of an idea, sometimes lost sight of, that nosological distinctions must be drawn not only from minute microscopical alterations of structure, as seen *post mortem*, but from such appearances plus the clinical phenomena during the life of the patient.

We would sum up the basis for discussion thus.

1. There are several forms of disease included under the head of phthisis.
2. Tubercle, whether of the lungs or of other organs, is due to a constitutional disease; and this is shewn by death occurring without *post mortem* results sufficient to have caused it.
3. Tubercle often exists quite independently of caseous deposits or vomicae.
4. This morbid condition is inheritable, but may be developed *de novo* by debilitating influences.
5. Its possible manifestations at all ages, and its frequent non-manifestation until the age of puberty, is only analogous to what is seen in other constitutional and inheritable diseases.
6. Chronic pneumonia found associated with miliary tubercle is not connected with it as cause with effect. The tubercle only becomes developed in cases in which the patient has previously had the tuberculous taint of constitution.
7. Some fatal chronic pneumonias may owe their irremediable properties to the same constitutional weakness, even where no miliary tubercle is discovered after death.
8. In general, non-tuberculous phthisis can be distinguished from tuberculous by clinical phenomena, and especially by the use of the thermometer.

ON PUNCTURES OF THE COLON FOR THE RELIEF OF TYMPANITES.*

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THE case which forms the basis of this paper has already been recorded in the columns of the *Practitioner* for October last. My object in again bringing it forward is to elicit an expression of opinion on the propriety of resorting to puncture in those distressing cases where other means have failed to relieve the tympanites. Having read in the *Practitioner* for February, 1869, the notes of a case of obstruction of the bowels under the care of Dr. Clifford Allbutt of Leeds, in which the distension was so great that Mr. Teale, whose assistance was obtained, punctured the colon, I determined to adopt such a procedure in the first suitable case that presented itself. Such an one came under treatment in a few days. On the 13th of the same month my father, Mr. Wathen, was requested to visit Mrs. G., aged 29, who had, half an hour previously, while engaged in some domestic duties, been suddenly seized with most severe lancinating pain in the lower part of the abdomen. She was cold and collapsed, presenting the appearance of one suffering from severe shock. The patient supposed herself to be about six weeks gone in pregnancy, and stated that in her previous pregnancies she had experienced pain and faintness about this period. Acute peritonites soon declared itself. This, under full and frequent doses of opium, turpentine stupes, etc., was considerably subdued; but the abdomen became intensely tympanitic. We failed to afford any relief, although free fecal evacuations were procured by turpentine and castor-oil enemata, administered by an O'Beirne's tube. The distension increased, causing great distress, accompanied by vomiting. I advocated puncture of the bowel, but could not obtain consent until the evening of the third day, when the patient earnestly requested relief by operation. After consulting with Mr. Brown of Haverfordwest and Mr. Wathen, I first plunged a small hydrocele trocar and cannula into the transverse colon, then into the descending, and lastly over the ascending, colon. As soon as the force of the current of gas lessened, the cannula was withdrawn and reintroduced, being guided by the most prominent portion of the colon. The first two punctures emitted gas with sufficient force to blow a candle out when held near the cannula;

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