THE LETTSOMIAN LECTURES ON SYPHILIS AND THE NERVOUS SYSTEM.

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LECTURE II.

MR. PRESIDENT AND GENTLEMEN,—From the questions of pathology and causal evidence that occupied us in the last lecture, we pass to-night to the subjects of more immediate practical interest—those of symptoms and diagnosis. The transition may, perhaps, bring with it a sense of relief, but, unfortunately, it brings with it no escape from the difficulties in which the subject is involved, and which surround, not less closely and scarcely less consciously, its practical aspects.

First, however, I should supply one omission in the last lecture. To the long list of maladies, the certain or supposed consequences of syphilis, should be added some of the functional diseases of the nervous system. By some authorities it is thought that such diseases as epilepsy, hysteria, insanity, and neuralgia, in their purely functional forms, are frequent consequences of the influence of the virus on the nerve-elements during the period of its greatest activity. Time fails me for any discussion of the subject, but there are two points that are especially important in any examination of the subject. The first is that if any special form of functional disease seems to be exclusively related to syphilis, it is essential that the positive evidence from the side of syphilis should be supplemented and confirmed by negative evidence that these forms do not occur apart from syphilis. Secondly, if the forms associated with syphilis are not special, it is needful to consider most carefully the question of indirect causation. The causes of these diseases are many and various, and they are widely prevalent. Some of the most potent are involved in the conditions and influences inseparable from constitutional syphilis and its treatment—in which physical depression and mental annoyance are sometimes combined with a reaction from a life of intense excitement of body and mind. It is also necessary to ascertain and allow for every traceable predisposition, and likewise to see that the therapeutic evidence is secured from fallacy by a rigid observance of the conditions that were mentioned as essential in the last lecture. I suspect that these measures would alter considerably the aspect of much of the evidence on which the opinions rest.

In the study of symptoms, not less than in the regions that we have already traversed, we still feel the pressure of the antagonism I mentioned at the outset. The success, incomplete though it be, with which we are able to treat syphilitic diseases of the nervous system, renders it impossible to obtain a knowledge of their symptoms as extensive or as certain as we have gained in the case of most other maladies. We can attain certainty, or at least the high probability which in medicine has generally to do duty for certainty, only by confining our attention to cases in which *post-mortem* evidence places the nature of the disease beyond doubt. But if we thus restrict our attention to these certain facts, and our inference to the sure conclusions we can draw from those facts, our knowledge will be at once small in extent and imperfect in character. It will be based only, or chiefly, on cases of great severity, and the effects of slighter forms and degrees of disease will, to a large extent, escape us. If we include the latter in our investigation, we are upon ground which is always insecure and often treacherous, for an uncertainty, varying with the knowledge and care of the observer, will attach to a large number of our conclusions. This uncertainty must, it seems to me, be frankly admitted. If we recognise its existence, if we employ the probable conclusions as such, and do not impute to them the certainty they lack, we may use them, or many of them, without much risk of being misled. To obtain the greatest practical power we must, on the one hand, be often content with probabilities that are not high; but, on the other hand, we must use these probabilities as such, and trust them only so far as their nature warrants.

In the practical diagnosis of these diseases, just as in their scientific investigation, it is necessary to weigh carefully each step in the reasoning, and to do so far more carefully than in the case of many other diseases. The amount of conscious and deliberate attention to the process of inference, which it is desirable to give, varies much in different kinds of disease. It should always be reduced to the least amount that is consistent with safety. Did we weigh with care each step in every diagnostic act, the limits of patience and of life would be reached before a quarter of the work had been done that might otherwise be accomplished. Here, as so often, our work has to be based on a compromise between the ideal and the adequate. Unconscious inference enters, of course, largely into the simplest so-called "observations," and into such diagnosis as seems to consist of pure observation. Still larger is its share in the recognition of diseases that are inaccessible to direct observation, and yet yield "physical signs" of their presence. Yet, in the latter case, we can allow it to remain unconscious without much risk of error, and we habitually do so, and even speak of "finding" a pneumonia or a pleurisy without a suspicion of error in our language, although we really infer the existence of the disease by a series of mental processes that would be of considerable length if the steps were distinguished. Our short cuts, in such cases, involve little danger to an observer who has been properly trained. But the lesions of the nervous system are wholly beyond the range of observation, however aided, and with all our looseness in the use of words, we do not speak of "finding" a cerebral hæmorrhage, or even a cerebral tumour, during life. In purely inferential diagnosis, in which the disease manifests itself only by effects that are indirect, and, indeed, often doubly indirect—the effects of effects of the lesion—we cannot dispense with the careful consideration of each step we take. This statement is eminently true of the diseases we are now considering, because in them we have to deal not only with the character of the lesion, but also with its nature.

The fact indicated in the last word is the first important consideration in regard to the nature of the diagnostic process. The process is, and must be, always a double one; and of these two parts one should always be taken before the other. We should always ask what is the nature of the morbid process before we attempt to decide whether or not it is specific and due to syphilis. In greater detail the essential steps are these: 1. What are the seat of the lesion as indicated by the symptoms, and its nature as shown by their course? 2. Is the process thus indicated one of those that may be specific? 3. Has the patient had syphilis? 4. Can any other cause of such a morbid process be traced? 5. Lastly, and subsequently, we have to see whether the result of treatment confirms our conclusion. This last element is different from the rest in character and separate in time. Its consideration involves questions that will come under our notice in the next lecture, and are moreover essentially those we examined in the scientific use of the therapeutic test. They need not therefore detain us to-day. But the other steps we must consider in the abstract, because the only mode of dealing with these cases, of avoiding error, and reaching as sure a conclusion as the conditions permit, is to treat each case as a new problem, irrespective of other cases or generic types, and to work it out by taking each step separately, and comparing them only at the end of the process.

One of these questions—whether any other cause can be traced—may seem superfluous when we are dealing with processes that have no other cause than syphilis. But this statement is true only of the pathological lesion. Most syphilitic processes have their analogues in other processes that are not syphilitic. The effects of the two on the nerve-elements may be the same, and the symptoms due to those effects may be the same. A gumma of the cortex and a glioma in the same part may produce identical symptoms. The consequences of the occlusion of an artery by syphilitic disease and by embolism may absolutely correspond. The element of specificity, therefore, is wholly absent during life. There are no symptoms of which we can say, as we can of *post-mortem* aspect: "This proves a specific lesion." There are no symptoms and no combinations of symptoms produced by syphilis that are not also produced by other diseases.

While all specific processes have their simple analogues, there are many simple processes that have no specific analogues, and many of these processes are expressed by characteristic symptoms. Some of these simple processes may be due to syphilis, as we have seen, but even then they are not specific; so far as we can discern, they are simple. If they are due to syphilis, they are cer-

tainly not influenced by treatment as the specific lesions are. Thus, while there are no symptoms that prove a specific lesion, there are many symptoms that prove that a lesion is non-specific. Hence arises the paramount importance of considering, in the first instance, what kind of lesion the symptoms indicate. To neglect this question—to conclude, because a patient has a disease of the nervous system, and has had syphilis, that the disease is specific—is a proceeding precisely the same as if we assume that a subcutaneous swelling in a syphilitic patient is a gumma without feeling its consistence or observing its characters, or as if we assume that a sore throat is specific without looking at it. If a syphilitic person has an attack of hæmatemesis, it would not be considered quite logical to assume at once that the hæmatemesis was of syphilitic origin, and yet observed facts actually afford more justification for this opinion than for the conclusion that paraplegia of absolutely sudden onset is due to syphilis. We know that syphilis has caused hæmorrhage into the stomach, but we do not know that it has caused hæmorrhage into the spinal cord.

This consideration of the kind of lesion that must exist to cause the symptoms may enable us to say one of two things—either that the process is one that may be specific, or that it is one that cannot be specific. To deal with these indications in detail would be to enter into the whole region of the diagnosis of diseases of the nervous system. There are, however, two conditions to which almost all specific lesions conform—one of time, the other of place. First, true specific lesions are generally subacute or subchronic. An acute onset—acute, not actually sudden—practically means acute inflammation, and we saw in the first lecture that the occurrence of acute syphilitic inflammation must be considered as still *sub judice*. As a matter of fact, the symptoms of certainly specific lesions very seldom develop to a considerable degree in less than a week. They are also very seldom actually chronic—seldom occupy more than three months in development. An actually sudden onset, however, is not at all rare; it is the common onset of the symptoms due to vascular occlusion. With these exceptions, when symptoms develop in a few days or many months, they are not likely to be due to a true specific lesion. The degenerative sequelæ are of course chronic in onset, but they are not truly specific. Moreover, the chronic interstitial inflammations, doubtfully specific, are also chronic.

Secondly, as to place. The specific processes are, as we have seen, outside the nerve-elements, and they have, therefore, no special relation to nerve-function. Hence their effects are random in distribution. They are related to a special function only when that function is subserved by one region, and this is so rare as to be of little practical importance. As a general rule, if we find certain structures of common function picked out for isolated impairment from among others of different function, we may be sure that we have not to deal with a true specific process. It may be a post-syphilitic degeneration, but it is not a specific lesion. These two indications are thus chiefly of importance as enabling us to exclude processes that are not syphilitic.

Assuming that we have evidence of a process that *may* be specific, we can often go a step further and say that, from the symptoms, it is *likely* to be specific. But the next important step is to ascertain whether, in the case before us, this possible cause can be traced. Syphilis may be a cause of the lesion; is this particular cause an actual antecedent in the case before us? In every department the fact that a certain cause can be traced may always justly be allowed weight in diagnosis. It must never take precedence over more direct evidence of the nature of the lesion, but in due subordination it is an indication of great value. Hence the next question is, Has the patient had syphilis? At least this is the form in which the question is commonly put, and in many cases this form is sufficient; but we must not be content with a negative answer. We have already seen that some of those who have had syphilis do not know it. We have seen the necessity of recognising this fact in the scientific investigation, and it is equally important in respect to practical diagnosis. We cannot absolutely exclude syphilis unless we can exclude the possibility of infection. The disease is so seldom contracted except in one way that we need not take other possibilities into consideration. Hence, if there has been any chance of exposure to infection, the disease cannot be considered to be out of the question. The truth and importance of this will be doubted by no one who has seen much of the late effects of syphilis, but to those under whose notice such effects seldom come the statement may seem scarcely credible.

In connection with this point, the question arises what influence the uncertainty as to the fact of syphilis should have on the diagnosis. How far should the probability of the syphilitic nature of the lesion be diminished by the improbability of past syphilis, and *vice versa*? I think that the common error is to allow too much weight to this element of uncertainty, but it is an error that is not easily avoided. The symptoms, in a given case, are of such a character that their cause may be a syphilitic process; if the patient is known to have had syphilis, the syphilitic nature of the process seems more probable than if his syphilis is doubtful. It is probably right to attach some weight to the point, but it should be attached to the element in the diagnosis to which it properly belongs—the element of probability. It must not be allowed to influence any distinct indications of either a positive or negative character. If, for instance, the symptoms are such as distinctly indicate a syphilitic lesion, a lesion scarcely ever due to any other cause, it is evident that this indication is not really influenced by the fact that the evidence of syphilis is inconclusive, provided the disease is possible. On the other hand, if the symptoms are such as to make it most unlikely that the process is syphilitic, this improbability is not materially lessened, even by a certainty that the patient has had the disease. If, however, the definite indications are equally balanced, the improbability of syphilis may be allowed weight.

If the lesion is one that may be syphilitic, if previous syphilis is certain or possible, the question arises, Can any other cause of such a morbid process be traced? If such other cause exists, the difficulty is greatly increased. The diagnosis then depends on a comparison of the precise character—first, of the lesions produced by the two causes with that indicated in the present case, secondly, of the symptoms by which the lesions are manifested with those observed in the patient.

This abstract outline of the process of diagnosis may seem to you needlessly complex. You may think that the considerations are purely theoretical, but they actually represent, in the simplest form, the essential processes adopted in actual diagnosis. I give them in the abstract because their application varies in each case, and for their effective use familiarity with their principles is essential. It must, however, be frankly admitted that exact diagnosis of the nature of these specific lesions is sometimes impossible. The symptoms are often equivocal; they may be such as might be produced by more than one syphilitic process. But we can generally reduce the possible lesions to two, and see some balance of the evidence in favour of one or the other.

We may now consider the symptoms of one or two of these specific processes, and first those of syphilitic growths. A gumma causes, for the most part, symptoms like those of any other intracranial tumour of rapid growth—general cerebral symptoms, and focal symptoms due to the local influence of the growth. Their special features are never more than suggestive. They depend on the course, and also on the fact that gummata are rather more frequent in certain parts than in others. The course, like that of other specific lesions, is subacute or subchronic; the seat is generally superficial, in the cortex or at the base, sometimes within the pons, seldom in the cerebellum, occasionally in the thalamic region, growing in from the side of the crus. Hence symptoms of cortical irritation are relatively more frequent than in other tumours; convulsions are common, and give most important information from their local distribution or commencement, or from the aura that equally indicates the spot at which the discharge begins. But all these indications, even when combined, furnish no absolute or even approximate ground of distinction. They may be, and indeed often are, produced by growths of other nature, especially by gliomata of rapid growth.

The indication afforded by the course of the symptoms is chiefly useful for its negative significance; a very chronic growth is not likely to be syphilitic. On this point we may often gain instructive information from the optic neuritis that is so often present. Syphiloma always causes an acute form of optic neuritis, and becomes intense. A rapid growth never causes a chronic form of neuritis, although now and then a slow growth may cause an acute form. Hence, while acuteness of the neuritis is of little diagnostic value, chronicity—a neuritis that remains for a long time moderate or slight in degree—is distinctly opposed to the diagnosis of a syphilitic growth, and adds considerable weight to the similar indication afforded by great chronicity of other symptoms. This indication is especially valuable when the early symptoms are equivocal, and we find it difficult to say how long the tumour has existed. These growths are among the specific

lesions in which the effect of treatment is of most diagnostic value. It usually causes a prompt diminution in the symptoms, but, to be of significance, this effect should be considerable, and should involve both sets of symptoms, the local and general. It is a remarkable but certain fact that a considerable diminution in the symptoms of other kinds of tumours, and especially in the general symptoms, sometimes follows the administration of iodide. The effect is most frequent in gliomata—the growths that give rise to most diagnostic difficulty. Sometimes even the optic neuritis may lessen, but more often this is unchanged, and should put us on our guard.

In estimating the significance of the subsidence of the neuritis, we must remember that every intense inflammation, when it has reached its height, tends to subside into a cicatricial atrophy, although the tumour that has caused it continues to grow. We must not mistake this natural subsidence for the result of our treatment. But this difficulty can often only be met by waiting. If any of the symptoms return or increase, if any symptom persists that would be readily influenced were the growth syphilitic, we must assume that this nature is not probable. The delay involved cannot be avoided, but it is a grave disadvantage, since the progress of surgery has so greatly increased the need not only for exact but for prompt diagnosis.

The chronic local meningitis of syphilis causes distinctive symptoms chiefly when it affects the base of the brain and damages the cranial nerves, or when, at the convexity, it implicates the motor region. The symptoms are such as indicate a surface lesion, and the absence of the signs of any considerable loss of function of the subjacent tissue, such as considerable compression would cause, is the chief distinction from a gumma in the same situation. Especially when the symptoms indicate a wide area of irritation with merely superficial damage, the diagnosis can be made with considerable probability. This focal meningitis is probably less frequent at the convexity than is a growth, and it is certainly less frequent there than it is at the base of the brain. In the latter situation the most important symptoms are due to the damage to the cranial nerves, and the most important diagnostic points are well known. The therapeutic test is available only in recent cases of meningitis. When the development of the new tissue into fibres has set in, this seems to go on in spite of treatment, and the inevitable cicatricial contraction may perpetuate the damage produced in the more active stage. In a case of focal meningitis that has lasted for some months, although it may afterwards be proved to be syphilitic in nature, the symptoms are generally influenced but little, and occasionally not at all, by anti-syphilitic treatment, however energetic. The other forms of meningitis, interesting and important as they are, I am compelled to pass over.

(To be continued.)

ON SOME OF THE RELATIONS OF NEUROLOGY TO SURGERY AND DERMATOLOGY.

*Being an Abstract of the President's Address delivered at the
Neurological Society of London, January 24th, 1889.*

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MR. HUTCHINSON'S address, after thanking the Society for the honour of electing him President, proceeded to deal with certain subjects in which surgery and dermatology were concerned in diseases of the nervous system. He mentioned a case which had recently come under his care in which a man, after the ordinary operation for piles, suffered from permanent incontinence of urine and faeces. The incontinence was attended by loss of sensation in the skin about the anus and complete paralysis of the sphincter, similar conditions being apparently present in the urethra. The history made it probable that this condition of paralysis had been present from within a day or two of the operation. After discussing the associated innervation of the two sphincters, attention was asked to the very common occurrence of inability to empty

the bladder after the operation for piles, and the question proposed as to how such occurrences were to be explained.

The subject next considered was that of "dry mouth," for which the name "xerostomia" was proposed in substitution for that of "aptyalism." The cases which have recently been under the consideration of the Clinical Society, with some others, were briefly reviewed, and the whole question considered as an important illustration of the influence of the nervous system in controlling and entirely arresting the secretion of glands. It was suggested that what we see demonstrated in the mouth, in cases of "xerostomia," may probably occur in other regions and viscera, and that there may possibly be a neurotic arrest of secretion of the liver, pancreas, testes, etc. Some remarks were made on the converse condition of excessive secretion of fluids into the mouth, or "hydrostomia." It was thought that this was much more frequently a symptom of aggressive disease of the nervous system than the opposite condition of dry mouth appeared to be. Attention was next asked to the nervous phenomena in Raynaud's disease, and to various cognate conditions in which the supply of blood to different parts of the skin was injuriously influenced through the nervous system. The chief subject-matter of the address was, however, the consideration of those diseases of the skin which should be classed as neurotic. Putting aside derangements of nutrition of the skin which might follow upon injuries, it was proposed to class the disorders referred to under three heads: first, those due directly to disease of the nerves themselves, or to some trophic influence exercised by them; secondly, those due to disturbances of the circulation produced through nerves; and, lastly, those in which the nervous system, although not being directly concerned, still took some share in the way of general reduction of tone. In the first, and by far the most definite, group, herpes and morphea or scleroderma were the principal examples, and concerning both of these Mr. Hutchinson entered into considerable detail. Attention was asked, in reference to herpes, to the remarkable fact that while common zoster rarely occurred twice in the same place, there were forms of herpes of which the peculiarity was liability to recurrence. It was asserted that while arsenic very frequently caused zoster, it acted almost as a specific in preventing recurrences of the other forms. The theory was upheld that herpes zoster was always due to peripheral neuritis, which involved the end-organs; that it was, in fact, as had been long ago said, a neuritis attended by an eruption. It was suggested that one great difference between zoster and the recurrent forms consisted in the severity of the neuritis in the former, which disorganised the nerve affected, and thus prevented the possibility of its being again attacked. In dealing with the subject of scleroderma (morphea) Mr. Hutchinson took first the localised form, remarking that it was very different, though perhaps not belonging to a wholly different class, from that in which the affection was symmetrical, diffused, and, in greater or less degree of severity, universal. He repeated an assertion which he had made, he said, many years ago, that all the main facts which were true as regards the development and evolution of zoster were true as regards morphea. He held that it was certainly located, and in all probability produced through nerves. Its remarkable differences from zoster were its slowness of evolution and its long persistence, taken together with the fact that it was not at all uncommon to find it affecting several parts of the body and limbs at the same time. He believed that its earliest manifestation was always in the form of a cluster of spots, and that the large patches often seen at a later stage were always due to a confluence of these. He drew attention to the circumstance that fascia, muscles, and bones were not infrequently affected together with the skin, whilst comparatively little loss of sensation attended the changes. He thought it highly probable that the vasomotor filaments distributed in conjunction with those for motion and sensation were the parts primarily affected. The remarkable defects in the subsequent nutrition and growth of the parts involved were illustrated by photographs and drawings. Respecting the condition known as hemiatrophy of the face, it was contended that it was always the consequence of morphea affecting the fifth nerve, and that it was not strictly an atrophy, but an arrest of development to be explained by the fact that the disease had occurred in early life before the parts had attained their full growth. A number of portraits were shown to illustrate the remarkable similarity of appearance in different examples of this singular condition. Some remarks next followed on the general principles which should guide us in determining whether any given