# A CLINICAL LECTURE
ON
# SILVER AND SYPHILIS.

*Delivered at the National Hospital for the Paralysed and Epileptic, Queen Square, Bloomsbury.*

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GENTLEMEN,—It is the duty of a clinical teacher to bring out of his treasury things old and new. He is constantly under some temptation to present ideas that are new because they possess more intrinsic interest, although it is generally more useful to give those that are old. Truth and novelty are by no means necessarily associated, although a familiar phrase which suggests mutual exclusiveness goes too far. Nevertheless, it is well for a teacher, if he can, to resist the attraction of the new, and it is always unwise for him to hesitate to inculcate that which is old merely because it is old. Years ago, when I was engaged in giving the elements of clinical instruction to students who were beginning their practical studies, I used to paraphrase the saying of Demosthenes regarding oratory and maintain that the first thing in learning is repetition, the second repetition, and the third repetition. A teacher should remember that to neglect to repeat is his unpardonable sin. There is an unpardonable sin of the student; in fact, it was the remembrance of this that made me use the term. It is not my own idea. Years ago Sir William Jenner used to say to us (and whatever Sir William Jenner said may be *a priori* regarded as certain) that the unpardonable sin of the student was to say "Yes" when he ought to say "No,"—to say that he heard a thing, that he felt a thing, that he understood a thing—when he did not.

For you this lesson is probably useless, because you have all passed the stage at which it is needed. But it may not be too late to remember that it is a mistake to shrink from unattractive repetition. Consider—what are we all learning, or should be learning, on this point, from our great Teacher? What does Disease impress on us? Disease is for ever repeating to us the same things. All the more important laws and rules are ever being pressed upon us, in varied tone, by varied emphasis, or in varying language, but ever repeated. No repetition should be, or will be, useless to us if we take the requisite pains, and are not deterred by weariness from striving to discern the lessons that seem the same, but present always an important difference; and no repetition of fact to us by disease is ever superfluous, unless we will to make it so.

Yet the teacher is, as a rule, compelled to take as a subject for his instruction some fresh illustration of disease. It is seldom that he has an opportunity of taking that which is old. When he can it is well for him to do so, for more than one reason. All old cases afford an opportunity of observing the changes in disease, and the effects that time permits. The question which ever presses on us when we meet with disease in its active stage is this: What will be the future; what will be the result? We can only learn to look forward by taking every opportunity of looking back. We cannot combine in our observation the future and the present. Our own experience and that of others may enable us to guess something of the future from the present; but every personal observation that increases our ability to forecast, everything that helps to make the guess more than a guess, is important. This help can only be obtained when the future has changed to the present. We can realise the past, and discern its relation to the future when that which was future has come. We cannot, with the same confidence, realise the unknown future and apply it to the present. Some of you may remember the lines which come to my mind—lines by a poet who is almost forgotten now, although his name should live in the memory of medical readers. Sidney Dobell wrote in one of his sonnets, in which sadness touches softly:

> And when the now is then,
> And when the then is now.

If the "then" is in the past, we can, by fancy, make it "now," the "then" is clearly seen, for it is now a fact, and we can thus gain some secure experience in prognosis.

I show you to-day a patient who presents an opportunity of illustrating the present by the past, and by a past in itself most instructive, such as seldom occurs to a teacher. I imagine it is almost a unique opportunity for a teacher to be able to take, as a subject, a patient who has been under his observation for a quarter of a century. That is the case here, and I am glad to have so unusual an opportunity in beginning again the series of our Wednesday lectures. It is nearly, if not quite, twenty-five years since this patient was under treatment in the wards of the hospital in the acute stage of his affection.

Did you notice him as he came into the room? If you did not, you should have done so. One of the habits to be acquired, and never omitted, is to observe a patient as he enters the room; to note his aspect and his gait. If you did so, you would have seen that he seemed lame, and you may have been struck by that which must strike you now—an unusual tint of his face. Those two things are important. They are, indeed, connected, but in a way that is rather curious than instructive. It is, indeed, so curious that I cannot resist the temptation of telling you the story it involves.

The patient came here in 1870, with symptoms of a cerebral tumour, of rather rapid onset for such a morbid process. The symptoms had reached to a considerable degree in about two months. The patient presented indications of a subchronic local cerebral lesion, with headache and optic neuritis. These two general cerebral symptoms with the onset indicate that the local process is a growth. Moreover, there was a history of active syphilis; and we know that whenever we have evidence of a local growth of rather rapid course in the subject of syphilis the probability is very great that the growth is syphilitic. They are much less if the growth is very chronic, and this point is important.

The patient was treated according to the diagnosis. He was not under my care, although he was under my constant observation; I was then Medical Registrar to the Hospital, and he was under the care of Dr. Hughlings Jackson, from whom it was my privilege to learn many lessons of ever increasing value, and not the least important were connected with this case. After the patient's discharge from the hospital he was under my care as an out-patient, of late years only seen occasionally, chiefly for the benefit he is always ready to give to others as an illustration.

When he first came to the hospital he was lame, as he is now, and he presented the complexion aspect you see, but in a greater degree. An inquiry into his history showed that two years previously he had been an in-patient at a general hospital, under the care of a physician then well known, who has now been dead many years. The symptoms he then presented were those of a small syphilitic growth pressing on one side of the spinal cord, and causing effects that we now know to be very characteristic. For those symptoms he was treated with nitrate of silver, and his skin acquired the aspect which it has never lost. When he came here he had improved, and I think, for several reasons: it is exceedingly likely that after the nitrate of silver had been given for a considerable time, without other result, mercury was substituted. At any rate, the affection of the leg ceased to increase, improved somewhat, and then became stationary, and when he came to this hospital its state was much as it is now, with due allowance for the effect of the fresh trouble which brought him to us.

In connection with his case there is a little story which I cannot resist the temptation of telling you, especially since the patient here can correct me if I am wrong. Perhaps it is a little beyond the proper subject of a lecture, but I dare say, gentlemen, you will not be strict. The patient suffered from severe headache, optic neuritis, and signs of a local cerebral lesion in one cerebral hemisphere of subacute onset. It was certainly a quickly growing tumour, and almost certainly syphilitic. Thus the case was most instructive. In 1870, as you know, not quite so much was known of optic neuritis as is now known, and he was shown to a good many visitors. The interest, too, was not lessened by the indications of argyria—the staining of the face from nitrate of silver for a morbid process in the

spinal cord similar to that which in the brain lessened with extreme rapidity under iodide of potassium.

Great care and caution were taken in all that was said in his presence. I can even now remember the scrupulous circumlocutory care adopted to guard against any perception, on his part, of what was thought about his previous treatment. But the man possesses a considerable amount of intelligence, and he picked up too much information, although he gave us no indication of the fact. The symptoms rapidly subsided. On the morning after his discharge he paid a visit to the physician under whose care he had been, and from whom he had received the silver. He obtained an interview with the doctor. The result of that visit was, I am certain, to improve the therapeutical knowledge of the physician, and I have also no doubt that the result was very much to the advantage of any other patient who subsequently came under the care of that physician for a similar affection. But the immediate result was a considerable disturbance of equanimity. The patient was wise enough to content himself with thus conveying instruction. He might, I think, have gone further; but I doubt whether even a speculative lawyer would have induced him to do so for he is, after all, a reasonable fellow. I think one cannot find very much fault with the lesson, or even, considering all things, with the way in which it was given.

[Dr Gowers here turned to the patient and asked if the account given was substantially correct. The reply was: "Yes, Sir; it 's all right. I *jacketted* him."]

I imagine that it is very likely that some of you have never seen the tint of argyria. It is less commonly met with now than formerly, because nitrate of silver is less frequently given, and when it is given, it is given with more care. It will therefore be wise for you to note very carefully the aspect of this patient. The tint is rather less than it was, but it persists, and it will persist as long as he lives. There is not now a black line at the edge of the gums; I think it was there formerly. We have been unable to find the old notes of the case, but all the essential facts are adequately impressed upon my memory. I have only myself seen about four cases of staining from nitrate of silver. This is one. Two were in cases of epileptics, for which it was, as you know, once a reputed specific. It was held in very high esteem by some persons in what have been called the "prebromidic days." Both the patients I saw, who had been stained with nitrate of silver for epilepsy, were still patients here for the persisting disease, and therefore my own observation did not lead me to entertain a very high opinion of its value.

The fourth case is instructive because it was due to the use of nitrate of silver for the good it can unquestionably do in gastric affections, especially when pain occurs before meals, that is, when it coincides with the absence of food. Although in cocaine we have an agent which seldom fails under these conditions, it is not unlikely that this use of silver will again increase. It may be then that the lesson this case gives may be again needed. A doctor, an esteemed practitioner in a suburb of London, gave his brother, who suffered much gastric pain, a "dinner pill," to be taken before food every day; it contained some oxide of silver. A year or two after, when shooting together in Scotland, the doctor become uneasy at his brother's cyanotic aspect. He watched him closely, and at last asked him: "Do not you get short of breath as you go uphill?" But there was no shortness of breath, and the doctor did not think anything more about it. Six months later, the tint had increased, and it suddenly flashed across the doctor's mind, "Why, it must be silver; there is that pill!" He turned to his brother and said, "Have you been taking those pills ever since I first gave them?" "Yes," was the answer, "I have been taking them ever since, and am still." By this time his face had become deeply tinted. The line on the gums was most distinct. Although they were at once stopped, other remarkable troubles came on. There were no signs of lead poisoning, no colic, and no conceivable source of saturnism, but the patient developed wrist-drop, just like the wrist-drop of lead poisoning, and also developed the gout that is so often due to lead, and he developed the albuminuria associated with it. Silver does cause palsy in animals; we know how many metals may cause the symmetrical extensor palsy, and I think there can be no doubt that in this case the palsy of the extensors of the arms was due to the silver, although the case as far, as I know, in that respect, is unique. The sequel of the symptom, I may add incidentally, was most illogical. The patient died two years later, but he died from cancer. With adequate mischief to terminate life by intelligible effects, he died from something altogether different, which is an illustration of the limits there are to our power of inference and forecast.

Let us turn to the patient before us. He left the hospital with some symptoms remaining from the disease in the brain; these had become stationary, and they have persisted ever since. I said he had symptoms of a local cerebral lesion. Those symptoms were slight left-sided weakness, left hemianæsthesia to all forms of sensation up to the middle line, head, limbs, and trunk; considerable diminution of the special senses on that side—taste and hearing, while vision was affected as hemianopia. Of smell I am not sure. If impaired, the defect did not persist, and his present recollection is that it was normal. The hemianopia was at first complete.

Leaving smell out of consideration for the moment, you know that left side hemianæsthesia, involving the skin and the special sensories, is the characteristic of what is called hysterical hemianæsthesia, a functional condition of the existence and reality of which there can be no doubt. Nor can there be any doubt of its practical independence of the patient's will. There was the difference, however, in this condition from the hysterical form—that in the latter the affection of vision is a diminution in the whole field of vision. There is a considerable general diminution in the field of the opposite eye, and a slighter similar diminution in the field on the other side—that is, the side of the central hemisphere involved. In this case, however, there was hemianopia on the opposite side. That is the great difference between this form of hemianæsthesia, that which is due to organic disease, and that due to a functional affection. Nevertheless, there is evidence to show that "crossed amblyopia," dimness of vision, with general restriction of the field, greater on the opposite or "crossed" side to the affected hemisphere, may occur from organic disease as it does from hysteria. But the reason why an organic lesion generally causes hemianopia is this. The lesion is generally at one place, at the region which Charcot has called the "sensory crossway," at the posterior extremity of the internal capsule between the optic thalamus and the end of the lenticular nucleus.

The sensory fibres from the skin, from the head, and limbs run in the posterior third of the hinder limb of the capsule. The optic tract conveys impressions to that region, the fibres from the same-named half of each retina conducting impressions from the opposite half of each field of vision pass to it. Thus the impressions passing to this hemisphere are those from the side on which the motor processes act on the limb. Each half of the brain receives impressions from the side on which it moves the limbs.

The fibres from the optic tract that subserve vision pass into the white substance of the occipital lobe. They probably have an intermediate station in the posterior extremity of the optic thalamus. But I need not now dwell on this. Whether they pass from the thalamus or directly from the tract they must pass close to the extremity of the capsule, close to the sensory fibres from the skin. Thus disease here causes hemianopia and cutaneous anæsthesia. But it also causes loss of taste and hearing. This is so well established that we are sure that the paths for these sensory impressions pass by this region. Moreover, there are cases on record of an affection of smell from disease of this region, but the point needs further evidence, and as this case has no definite bearing on it, I will pass it by. But I must emphasise the fact that cases of organic disease have been met with which cause symptoms resembling closely the hysterical form of hemianæsthesia. The latter we must ascribe to an inhibition of the sensory structures in one hemisphere. In the cases of organic disease that cause similar symptoms, extensive disease has existed on the convexity of the hemisphere, so extensive that we cannot infer more than that it is in the convexity of each hemisphere that the impres-

sions are represented which come from all the special senses of the opposite side, including smell. In these cases there is "crossed amblyopia." But, beyond recognising the fact, you need not now consider the condition. Our subject is the sensory effect of disease that causes vision to suffer as "hemianopia." Half-sight is lost with the other senses if the disease is in the tract, the sensory crossway, or the half-vision centre in the occipital lobe. This was the combination the patient presented. The association of hemianopia and impairment of the general and special senses on the same side proved that the disease was situated where all the paths are in contiguity—that is, at the place I have mentioned. Beyond this the paths diverge, so that the combination can be produced only by disease invading the whole cortex of the convex surface, including the occipital lobe, and then there is the crossed amblyopia as well as the hemianopia. There is restriction of the field as well as half loss. We may now turn to the symptoms the patient still presents, the permanent residue of those caused by the active disease. Persisting as it has for the past quarter of a century, we may actually expect it to persist for the next quarter of a century, which is probably about as long as the patient may be expected to live. A slight defect of taste and of hearing is still distinct. In the limbs there is still imperfect sensation.

The defect in vision that still persists is particularly instructive. You can easily verify it for yourselves. In one pair of charts before you, you see first the fields of vision, as they were nine and a-half years ago—that is to say, about fifteen years after he came for treatment—and the others present the condition that exists at present. The two are practically identical. In each there is a loss of the left lower quadrant. At first the whole half field was lost, up to the middle line; but the upper part recovered slowly, so as to leave the loss confined to the area you see. In it there are points that it will be instructive to you to note. The loss stops short of the fixing point. That, as you may know, is a characteristic of all forms of hemianopia, at any rate of lateral hemianopia. I show you another diagram, in which the loss is of the whole half field, in which the feature is well shown. The blind area reaches the middle line, above and also below; but the dividing line between sight and blindness curves round the fixing point, so as to leave an area of vision. This is the rule—I believe invariable. Around the fixing point there is vision on the blind as well as the seeing side. I believe that apparent exceptions are due to imperfect observation—imperfect almost of necessity, because the area of sight around the fixing point varies, and special means are needed to ascertain it when it is small in extent. The explanation of it is that from the region just around the macula lutea fibres pass by each optic tract to each hemisphere. Hence, disease of either optic tract, while causing hemianopia, does not cause loss in that small region from which the fibres go to each tract. It may have occurred to you that if fibres pass to each optic tract, disease of either should lessen the function of the whole of the region, although causing no absolute loss on either side. It is so. If you test minutely the central area of vision of a patient with hemianopia you will find that, although the region round about the fixing point is spared, the acuity of vision in it is definitely reduced.

Note also another point of significance. There is but little restriction in the general field of vision. The very slight diminution in the general area of the peripheral parts of the fields is not greater than can be accounted for by a dark day, or even by an individual variation; yet note how different is the extent of the remaining half fields in the other chart I show you. Notice the remarkable restriction of field, and that it is much greater in the eye on the side of the half loss—that is, the right half of each field being lost, the remaining part of the right field is much smaller than the left. I must not, however, allude to this to-day beyond asking you to remember it in connection with "crossed amblyopia."

And now, gentlemen, in conclusion, I have to impress upon you one practical lesson which this patient gives us, and which he gives as an old patient. It is the persistence, in some degree, of the effects of syphilitic disease, and that in spite of the fact that the patient was treated thoroughly, a few weeks after the development of the symptoms—which is as promptly as most patients are treated. It was manifestly adequate, for the urgent general symptoms, headache and optic neuritis, began to lessen within a week, and in the course of a few weeks all the acute symptoms had passed away, and the local symptoms were rapidly improving, the loss of power had become trifling, the hemianæsthesia gradually became partial instead of complete. Yet some of these symptoms have never quite passed away.

The optic neuritis when he came in was considerable but not extreme. The whole disc was obscured by a swelling of moderate prominence and considerable vascularity, yet the acuity of vision was perfect. The inflammation was not sufficient in itself to have involved the nerve fibres so as to impair sight by the process of inflammation in them, nor was it sufficient to produce new inflammatory products sufficient to damage the fibres by their cicatricial contraction. Those are the two ways in which sight suffers from the neuritis. In those days ophthalmic surgeons generally refused to believe that considerable neuritis could exist with perfect vision. The scepticism was chiefly dispelled by Dr. Hughlings Jackson, and I think that this patient was one of the cases by which the dissipation was effected. Fortunately the treatment was early enough to remove the neuritis before grave damage was done to the fibres. His sight has remained good so far as acuity of vision is concerned, and his optic discs now present such a perfectly normal aspect that you would not suspect they had ever been inflamed. I should advise you to examine them carefully, because it is very seldom that you have an opportunity of seeing optic discs which are known to have been the seat of inflammation, and which now present no indication of it, and especially discs that were inflamed long ago.

The practical lesson that I mentioned, and which I should like you to take away, is this: The idea is not yet extinct that all syphilitic disease will yield to treatment, and that if only the symptoms are certainly due to syphilis they can be cured. Probably you know well as regards the nervous system how erroneous that idea is. You know that a syphilitic ulcer of the skin will, and must, leave a scar which nothing can remove. It destroys the tissue of the skin, and that tissue of the skin being destroyed is never replaced by structure which is like the old skin in aspect and function. So it is with the brain. If there is absolute destruction of tissue by a syphilitic process, that tissue cannot be renewed, and is not renewed; the symptoms dependent on its destruction will not pass away unless other parts of the brain can compensate for their loss. Remember that, as I have often said, the symptoms of a local lesion are never due directly to the syphilitic process. In true syphilitic affections, those which can be removed by iodide of potassium and by mercury, the syphilitic process is altogether outside the nerve elements themselves. These suffer secondarily, as they would from any other process of a similar general character. They suffer from compression by such a syphilitic gumma as this patient had, as they would from any other tumour. They suffer somewhat from the inflammation adjacent to any rapid growth, but this is usually simple inflammation with no necessary specific element. Through these processes they undergo damage and destruction, and no removal of the growth can do more than permit the recovery of those structures which are so little damaged that their recovery is possible. In the patient before you the damaged structures recovered. Destruction, of necessity, persisted, and the effects of the loss of tissue remain, where no compensation could be effected. That is the case with the half-vision centre and the fibres that carry impulses to it. Destruction of these causes destruction of the function and lasting loss. We have in the quadrantic loss of the fields of vision a proof of this fact. We have in it also evidence of the equally important fact that the effects of syphilis are often far more than that to which the term "syphilitic" can be applied. If you learn from this to look at a process with the imagination that is as important in practice as in science; if you learn to discern the elements on which symptoms depend, and to be cautious in your prognosis in such cases of syphilitic disease; if you learn also the lessons the patient teaches regarding silver as well as syphilis, you will not have wasted your hour here to-day.