ABSTRACT OF TWO

Clinical Lectures

ON

INTRA-CRANIAL SYPHILIS.

Delivered in the Western Infirmary of Glasgow,

By DR. MCCALL ANDERSON,

PROFESSOR OF CLINICAL MEDICINE IN THE UNIVERSITY OF GLASGOW.

LECTURE I.

GENTLEMEN,—We have at present in the wards three illustrations of intra-cranial disease which, while they present certain features in common, differ considerably from one another in respect to their symptomatology; and yet, as we shall see, they are but branches of one and the same parent stem.

But first of all let me refer to a case of a similar nature which some of the senior students had the opportunity of seeing, the patient having been admitted into Ward 9 on March 30th, 1882. This man—who was twenty-nine years of age, and a grinder by occupation—was then suffering from left-sided hemiplegia. He informed us that for six months he had been troubled with severe and steadily increasing headache, which was limited to the left side. Four days before he came to us he was, in addition, quite suddenly seized with paralysis of the left arm and leg, so that he was unable to walk, and the day following it was observed that he could not converse, but could only answer “Eh!” to every question that was put to him. The day before admission, however, this symptom had in great measure disappeared, although when he came into the infirmary he had a dazed and stupid look, and answered questions in few words or sometimes not at all, while the information which he vouchsafed was found afterwards to be quite erroneous. For a day or two also before admission the paralysis, which was quite complete at first, except as regards the side of the face which was only partially paralysed, became much less pronounced, so that he could move the arm a little and the leg with considerable freedom. On the 8th of April, nine days after he came under observation, he became insensible, the loss of consciousness lasting for about a quarter of an hour, during which time there were convulsive movements of the arm and leg. On recovering consciousness it was found that the paralysis was greatly aggravated, he being unable to move either the arm or leg. The sensation, too, on the same side of the body was rather defective as regards touch, pain, and temperature. Thus, when slightly pricked with a needle, he thought he was touched with the hand, and when hot and cold sponges were applied he could discriminate cold, but not heat.

The symptoms present in this case showed that the lesion was situated on the right side of the brain, so as to involve the motor, and to a less extent the sensory tract. As regards the nature of the lesion, embolism as a cause of the hemiplegia was very unlikely, because it is then generally right-sided; because of the absence of any evidence of deposits on the mitral or aortic valves such as most commonly yield emboli; and because there were no signs of embolism of other organs. The age of the patient, and the absence of symptoms of kidney disease or of degeneration of vessels, rendered a cerebral hæmorrhage improbable. On the other hand, the intense and long-continued hemicrania led to the suspicion that we had to deal with a cerebral tumour, and that it was of a syphilitic nature we concluded from the following facts. The hemicrania was markedly nocturnal in character, as we so often find in syphilitic cases; the skin had a dirty, earthy, pallid appearance; the inguinal and arm glands were distinctly enlarged; and a large white scar about the size of a sixpence, with dark edges, was discovered about the root of the penis, which we were told on inquiry was the result of a chancre which he had five years before. This view was further confirmed by the result of treatment, which consisted in the rubbing of a drachm of strong mercurial ointment into some soft part of the skin every day. It was commenced on April 2nd, omitted by mistake from the 10th to the 20th, No. 3226.

and finally stopped on June 2nd. On June 23rd, when he left the hospital, he was a very different man. The intelligence, which was at first so defective, was perfect in every respect, the nocturnal hemicrania had long since disappeared, the paralysis of the arm was very slight, as he could move it freely in all directions, and the dynamometer, on which at first the left hand made no impression, registered thirty-five kilogrammes. The paralysis of the leg was likewise slight, although rather more pronounced than that of the arm, and he walked a little lame and rather stiffly. This last symptom, taken along with the existence of some tremor when the limb was manipulated, and exaggeration of the patellar-tendon reflex (the ankle clonus was not present), rendered it probable that secondary sclerosis of the lateral column of the cord had to a certain extent occurred. As this lesion, although secondary to the lesion in the brain, was of a non-syphilitic character, it could not be expected to be benefited by anti-syphilitic treatment.

We now pass on to the consideration of the cases at present in the wards. The first to be admitted (Feb. 10th, 1885) was a blacksmith, thirty-eight years of age. He complained chiefly of severe pain in the forehead and upper part of the head, which had been present for about four months. The family history was good. Twelve years ago he suffered from an attack of gonorrhœa, which lasted about three or four months, and did not appear to have left any bad results. About eight years ago he had what he calls rheumatism, describing it as constant pains in the bones and joints, becoming very bad on movement and markedly worst at night. Three years ago he had a painful affection of the throat lasting for about a year. With the above exceptions he had always been a healthy man previous to the onset of the present illness. About four months ago he was suddenly seized, while sitting at the fire, with a violent pain in the forehead above the left eye. It lasted several hours, and he was then free of it for about a fortnight. After that he had repeated attacks, and noticed that the intervals between them gradually became shorter, and since the beginning of the year they had been almost constantly present, although with occasional great exacerbations. It was worse at night. The pain above described was often very intense, so much so as altogether to prevent sleep, and frequently to produce delirium. About the beginning of January his eyesight became somewhat impaired, so that he was unable to read as well as he used to do. On looking at anything steadily for a time, his vision became dim, as if a “scum” was before his eyes, and sometimes there was a feeling of pressure in them, especially on attempting to read. He professed never to have had any eruption on the skin or falling out of the hair. During the three months previous to admission he had lost flesh very much, having become, as he thought, about three stones lighter than formerly. A few scars were observed on the legs, which he said were due to the application of croton oil. The scar on the right leg, a little above the outer malleolus, was distinctly coppery in tint. There was a rounded and depressed cicatrix on the glans penis a little above and to the left of the frænum. This he attributed to a burn from a piece of live coal. The foreskin was adherent to the glans at the sulcus. There were no scars at the angles of the mouth. There was no paralysis of the muscles of the face or eyelids, save a slight drooping of the left upper lid. The eyeballs were slightly prominent, especially the right, but this condition, he said, had been present since childhood. The tongue, on being protruded, appeared to be slightly drawn to the left side. On tapping all over the head two spots were found about two inches and a half above the outer angle of each eye, which were distinctly tender. He could hear the ticking of a watch better when placed opposite the ear than on the temple. The greatest distance he could hear the watch with the left ear was eighteen inches, with the right twenty-four. The smallest type he could read on admission was a double small pica, whilst before the illness he could read the ordinary type of a newspaper, which is only half the size of the above (see Dr. Reid’s report¹).

¹ “Aided by eight-inch convex, he is able to see the smallest print at twelve inches with either eye. The area of the field of vision is complete in both eyes, there being no defects to be found in the lateral parts of the field. The power of distinguishing colour is perfect, with the exception of deep purple, which he mistakes for black. Ophthalmoscopic examination:—Hypermetropia of $\frac{1}{2}$ found (left pupil only dilated). The left optic nerve seems rather prominent, tissue consolidated, and slight signs of effusion along the course of the vessels as they leave the optic

Smell and taste seemed to be normal, and the dynamometer registered 105 kilogrammes with each hand.

A consideration of the history and symptoms present in this case led to the conclusion that we had to deal with intra-cranial syphilis. Before his admission into the infirmary he had been under the care of half a dozen medical men in succession, and appears to have been treated for the most part by means of quinine and other anti-neuralgic remedies. His last medical adviser, however, evidently suspected the syphilitic nature of his complaint, and gave him iodide of potassium. He took it at first in doses of ten grains three times a day, but for a week before his admission the dose was increased to thirty grains. This had not the slightest effect upon the pain or other symptoms. Accordingly, on the 12th of February, he was ordered to rub into the skin daily one drachm of Shoemaker's mercurious oleate ointment, while thirty minims of paraldehyde were prescribed at night to procure sleep. In a few days the pain had completely disappeared, and up to the present date (April 1st) he has remained perfectly well. The mercurial treatment is, however, being continued as a precautionary measure.²

WHY DOES SALICYLIC ACID CURE RHEUMATISM?

By P. W. LATHAM, M.A., M.D., F.R.C.P.,

DOWNING PROFESSOR OF MEDICINE, CAMBRIDGE; SENIOR PHYSICIAN
TO ADDENBROOKE'S HOSPITAL.

(Concluded from p. 1121.)

IF, then, as I assert, uric acid or its antecedent is the *materies morbi* which develops rheumatic fever, and if, as I have previously advocated, it is according to the accepted opinion the cause of gout, why should it in some individuals produce this disorder and in others rheumatism?

Let us in the first place consider what, under the normal state of things, is the effect of cold and heat applied to the surface of the body. The effect, as you know, is very different as regards cold-blooded and warm-blooded animals. External cold diminishes and heat increases the metabolic activity of the cold-blooded animal, acting like a mixture of dead substances in a chemical retort. But in a warm-blooded animal, within certain limits, cold increases and heat diminishes the bodily metabolism, as shown by the increased or diminished consumption of oxygen and production of carbonic acid as the temperature falls or rises. There is obviously a mechanism of some kind counteracting and indeed overcoming those more direct effects which alone obtain in cold-blooded animals. But under the influence of urari, which paralyses the end-organs of the muscular nerves, a warm-blooded animal behaves as regards its bodily metabolism under the influence of external cold and heat like a cold-blooded animal. "We can best explain these results by supposing that under normal conditions the muscles which, as we have seen, contribute so largely to the total heat of the body are placed, by means of these motor nerves and the central nervous system, in some special connexion with the skin, so that a lowering of the temperature of the skin leads to an increase, while a heightening of the temperature leads to a decrease, of the muscular metabolism. Further, though the matter has not been fully worked out, the centre of this thermo-taxis reflex mechanism appears to be placed above the medulla oblongata, possibly in the region of the pons Varolii. When urari is given the reflex chain is broken at its muscular end, when the spinal cord is divided the break is nearer the centre."¹

How does this bear upon rheumatism? An individual is exposed to a cold draught or gets wet; the surface is chilled; the cutaneous vascular areas are constricted; by reflex action through the vaso-motor system the splanchnic vascular areas and the vessels of the muscular areas are dilated; the vaso-motor nerves distributed to these parts are para-

lysed, so to speak; with this paralysis of the vaso-motor nerves of the parts there is not only a dilatation of the vessels in the part, but there is also a weakening of the power by which the albuminous molecules are held together, and from both causes heat is developed. The normal change of the cyan-alcohols into cyanamides, and amido-acids with the ultimate oxidation of the latter, is modified.

The molecules $\text{CH}_2 \begin{Bmatrix} \text{OH} \\ \text{CN} \end{Bmatrix}$ and $\text{C}_2\text{H}_4 \begin{Bmatrix} \text{OH} \\ \text{CN} \end{Bmatrix}$ become detached from the benzene nucleus, and more or less from each other, and by hydration (by which heat is developed) form substances—glycollic acid and lactic acid—which are more easily oxidised than the amido-bodies. The oxygen, though conveyed in larger quantities than normal by the increased blood-supply to the tissue, is completely used up in oxidising the glycollic and lactic acids which have been formed; the excess of lactic acid and the glycocine (which has also been formed) are unoxidised and pass into the circulation. Now, in a normal or healthy state of things, the irritating or stimulating cause acting on the skin being removed, reaction would be set up there, the cutaneous vascular area would dilate, and consequently the vessels of the muscular area would contract, and this latter contraction would be increased by the stimulating effect of the glycocine or resulting uric acid, which, being a morbid product, would stimulate, as in gout, that portion of the nervous system which controls the nutrition of the joints just in the same way as carbonic acid in the blood stimulates the respiratory centre. In this way less blood would go to the muscular area, and the albuminous molecules would again be held more firmly together and undergo the normal changes, the uric acid being meanwhile excreted by the kidneys and the lactic acid by the skin. I have here drawn you a picture of an ordinary feverish cold—the primary chill, the ensuing febrile condition, the elimination of lithates with the subsidence of the attack.

But suppose the individual who is exposed to damp or cold has been previously reduced in strength, is tired out or exhausted—that is to say, his vaso-motor system is in a weak state, the weakness either developed from the above causes, or it may be inherited. Then following the chill there would be the same effects produced as I have above described, but the vaso-motor nerves regulating the vessels of the muscular area would be more completely paralysed, and when reaction set in on the surface of the skin there would be less power in the muscular nerves to recover from that paralysed condition; and more than this, the central portion being exhausted, the stimulus of glycocine or uric acid produces not stimulation, but exhaustion.

Let me illustrate what I mean by reference to the sciatic nerve.

"Division of the sciatic nerve of a mammal causes dilatation of the small arteries of the foot and leg. Where the condition of the circulation can be readily examined, as, for instance, in the hairless balls of the toes, especially when these are not pigmented, the vessels are seen to be dilated and injected, and a thermometer placed between the toes shows a rise of temperature amounting, it may be, to several degrees.² But the dilatation so caused after a few days disappears; the foot on the side on which the nerve was divided becomes not only as cool and pale, but frequently cooler and paler than the foot on the sound side. If the peripheral portion of the divided nerve be stimulated with an interrupted current immediately or very shortly after division, the dilatation due to the division gives place to constriction; the sciatic nerve acts then quite like the cervical sympathetic, except perhaps that this artificial constriction cannot be maintained for so long a time, and is very apt to be followed by increased dilatation. If, however, the stimulation be deferred for some days, until the dilatation has given place to a returning constriction, the effect is not constriction, but dilatation; the nerve then acts, so far as its vaso-motor fibres are concerned, like a muscular nerve, and not like the cervical sympathetic.³ So also with regard to the mylohyoid muscle. Section of the nerve produces dilatation, but the dilatation is transient. The vessels speedily return to their former calibre, and then it is found that stimulation, of whatever strength, of the peripheral portion of the divided nerve brings about not constriction, but dilatation. If then the vaso-motor fibres of the muscular nerves have been weakened, the result would be that the normal stimulating effect of uric acid on the motor nerves generally

disc; but no distinct evidence of there having been choked disc or engorged optic nerve, the disturbance not being more than is sometimes found in a high degree of hypermetropia. The defect of vision which he complains of is to be referred to deficient power of accommodation rather than to actual loss of accommodation. This deficient power of accommodation is sometimes seen to follow a shock to the nervous system."

² This patient was seen on April 27th, and was found to be quite as well as at the beginning of the month.

¹ Foster's Physiology, 4th edition, p. 467.

² Ibid., 4th ed., p. 199.

³ Ibid., p. 209.