MALARIAL NEURITIS AND NEURO-RETINITIS.

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THE following cases are of interest because they appear to throw light on the nature of the changes which sometimes occur in various nerves among persons suffering from malaria; instances of this description are perhaps more common in this country than is generally supposed, and in places where malaria prevails cases of hemiparesis, sciatica, and loss of sight in connection with intermittent fever are not unfrequently met with.

CASE I.—Mr. M. had been employed as a tea planter in Assam and Cachar for thirteen years, and during that period had frequently had intermittent fever, after more than one of these attacks his eyesight was affected so as to prevent him from reading or writing for ten days or a fortnight; the impairment of vision had then cleared away. Mr. M. returned to England in April, 1889. On May 19th he got wet through in a shower of rain; the same afternoon he had a severe fit of ague. The following morning I found that his sight, which had been perfect twenty-four hours previously, was so far affected that with the right eye he could only decipher the letters of Snellen 2.25, and with his left eye he was unable to do more than dimly count fingers held up between his eye and the light. There was no pain in the eyes, photophobia, or conjunctival congestion, the pupils were somewhat dilated, and acted imperfectly to light; the optic discs were completely obscured by effusion which extended into the retina; the retinal veins were tortuous and congested. The patient took 10 grains of quinine at 11 A.M. and again at 3 P.M., but the fever returned at 5 o'clock. On the 21st the vision in the right eye had recovered so as to be about equal to that of the left eye on the previous day. Mr. M. had no pain in the head, vomiting, or symptom of intracranial trouble; his urine contained neither albumen or sugar; he had some enlargement of the spleen, but was not anæmic; there was no evidence of either syphilis or rheumatism in this case. Forty grains of quinine were administered on the 21st; the fever did not return. Subsequently the patient took arsenic and strychnine, and by avoiding exposure to chill and removal to a bracing seaside locality he remained free of intermittent fever; his optic papillæ gradually cleared, but it was not until two months after the attack that his vision so far improved as to enable him to read Snellen 1.25. I saw Mr. M. in November, 1889, his optic discs were white, but he could read Snellen 0.5, and his vision was $\frac{1}{2}$ in his right eye, and $\frac{1}{4}$ in the left; he had had no return of fever, and was about to join his appointment in India.

CASE II.—This officer was referred to me by Surgeon-Major Maxwell, who was asked to see him the day after he complained of impairment of sight. Lieutenant — had been in India for five years, and during that period suffered frequently from intermittent fever. While on the march with his regiment he suddenly found his eyesight growing dim. He was obliged to go on the sick list, and was therefore sent forward to Meerut, and so came under the care of Dr. Maxwell, who found that he was suffering from neuro-retinitis in both eyes. "The optic disc in the right eye was indistinct and congested, the vessels engorged and tortuous, bending over the swollen portion of the retina. The vessels were in some places concealed by the swelling of the retina. The right eye presented signs of disease similar to the left. The patient was quite blind; his pupils were dilated and inactive." There was no suspicion of syphilis, no albumen in the urine; the patient had not suffered from rheumatism; his spleen and liver were much enlarged. This gentleman was without delay ordered home, and arrived in England within three months after the affection of his eyes had commenced; since leaving Meerut he had been free of fever. In the left eye the optic papilla was hazy, and there were several white patches over the retina with filiform hæmorrhages near the yellow spot; with this eye the patient could count fingers; the left eye was in much the same condition, the vision, if anything, being worse than in the right eye. The patient's liver was of normal size, but his spleen was still enlarged. Full doses of arsenic combined with strychnine was ordered, and he was sent to a bracing climate; the symptoms of neuro-retinitis gradually passed away, the patient's vision became normal, and he returned to duty twelve months after his arrival in England.

CASE III.—This patient was a lad aged 10, residing in India; he had suffered from intermittent fever on and off for some time before I saw him; during an attack of this kind his sight became dim, and in the course of a few days he was completely blind; it was then that he came under my care. I found his spleen enlarged, there was a trace of albumen in his urine with fine casts covered with leucocytes. The optic papillæ in both eyes were obscured by effusion, the retinal veins were distended, the serous effusion extended into the retina. This lad was ordered sulphate of quinine and subsequently arsenic and strychnine, by means of which further attacks of ague were prevented, and within a month of his coming under observation the symptoms of neuritis had passed away, and the boy's vision appeared to be almost normal; he was unable to read English, and I could not therefore test his vision by means of Snellen's types.

CASE IV.—This patient, aged 27, was a native of Bengal, who, after an attack of ague, first began to experience a certain amount of dimness of vision; the fever returned every day, and the sight became worse until the patient was almost totally blind; at the same time he experienced much difficulty in swallowing, fluid returning through his nose; the muscles supplied by the left ulnar nerve were paralysed. Under treatment similar to that referred to in the above cases, the patient, within a period of three months regained complete power over the various nerves and of the muscles whose functions had been temporarily lost.

I have notes of a considerable number of cases of this kind but selected these because the patients came under observation, we may suppose, in an early stage of the affection of the optic nerve although it is quite possible the neuritis may have existed for some time before the sight became impaired. I referred to cases of this description as far back as 1866, in the *Indian Medical Gazette*, and again in 1872. In the former of these communications intermittent hemiparesis with neuritis in connection with malaria was discussed; and in the second, neuro-retinitis, the result of malarial toxæmia. My idea was that these affections of nerves in cases of malarial fever were the effect of peripheral neuritis, an idea referred to by Dr. Todd, who was under the impression that periodical neuralgic affections were frequently due to the determination of some poison to a particular nerve, as the paludal poison, or some matter generated in the system, gouty or rheumatic. Dr. Todd thought that "there was no reason why such morbid matters should not affect a motor nerve as they affect a sensitive nerve, causing paralysis in the one case and neuralgia in the other." He adds, "Mr. Bowman has met with several cases of distinctly rheumatic paralysis of the portio dura, and also of some of the nerves of the orbit, at the Ophthalmic Hospital, Moorfields."

Among the patients I have referred to above it will be noticed there was an entire absence of symptoms indicating either meningeal or cerebral disease; they all regained complete power of vision; they were not suffering from marked anæmia although they had enlargement of the spleen; there was no indication of serious renal mischief, nor was there any evidence of syphilis or of rheumatism among them. Each of these patients was under the influence of malaria when they developed symptoms of neuritis affecting the distribution of the optic nerve. They all recovered their sight, but this is by no means the ordinary course of malarial neuritis or of neuro-retinitis, for if the fever continues, atrophy of the optic papilla and total blindness too often occurs. In the cases to which I have referred, it was possible to mitigate the power of the poison from which the patients suffered in an early stage of the disease; and it is to this cause I attribute the successful termination of the morbid process so far as the eyes were concerned. The neuritis passed off gradually, it is true, but effectually, because we were able to administer drugs which destroyed for the time the influences which produced the fever. I may observe, however, that the malaria was during this time in the bodies of these patients: they ate, drank, and slept well, were in no pain or weakness beyond the condition of impaired vision, but if exposed to sudden damp and cold, any one of them would probably immediately have suffered from an attack of ague. The poison was in their system; we could see its persistent action on the structures comprising the optic papillæ, it took two months before it left this spot, and its departure was probably hastened by the action of certain drugs.

After carefully watching cases of this kind, I incline to the idea that the inflamed state of the optic papilla is due to something of the nature of a microbe which becomes planted in the affected tissues, and growing there produces ptomaines, which in their

turn cause irritation of the tissues, engorgement of the vessels, and the transudation of serum and leucocytes into the retina and optic papilla. This effusion probably increases the already impeded circulation through the retinal veins by pressure exercised upon them as they pass outwards through the discs. I may repeat that, whatever the direct cause of the changes observed in the optic papilla and retina, it had an intimate connection with the cause inducing the fever from which these patients suffered, for unless the fever could have been checked, the neuritis would have gone on to produce atrophy of the disc. In one of the cases above referred to, other nerves besides the optic and retina were implicated, probably through analogous changes to those which had been observed in the distribution of the second nerve.

The idea I have referred to regarding the pathology of malarial neuritis as it affects the optic nerve is strengthened by the observations of Dr. Kipp on "Malarial Keratitis," published in the *American Ophthalmological Society's Report for 1889*. Dr. Kipp has had 120 cases of this kind under his care, and he states that when the affected eye is carefully examined within a few hours of the commencement of the pain, photophobia, and circumcorneal congestion, that "not far from the margin of the cornea, a number of small greyish opaque elevations may be seen extending in a line towards the centre of the cornea. The following day the elevations give place to a narrow seriginous ulcer, which extends superficially from without inwards," thrusting out branches in its progress, and reminding one of the fungoid growth which erodes the surface of our microscope lenses in India.

Dr. Kipp states that the duration of these ulcers of the cornea depend on that of the malarial fever which is always present in cases of this kind. He remarks that "until the fever is cured, the affection of the cornea continues in spite of treatment." In fact, as in certain questionable syphilitic cases, we may be able to clear up the diagnosis by watching the effects of anti-syphilitic remedies on the disease, so in these malarial cases the beneficial action of quinine, arsenic, and strychnine are indicative of the nature of the affection from which the patient is suffering. In the optic nerve, and also in the distribution of the nerves of the cornea, it would appear that whatever the nature of malaria may be, it is something capable of fixing itself on these structures, and, by producing irritation in the part may lead to destructive inflammation of these delicate tissues.

MEMORANDA:

MEDICAL, SURGICAL, OBSTETRICAL, THERAPEUTICAL, PATHOLOGICAL, ETC.

ON RIGIDITY WITH EXAGGERATED TENDON REACTIONS, AND CEREBELLAR INFLUX.

In the report of remarks I made in the discussion following the reading of Dr. Charlton Bastian's paper at the Royal Medical and Chirurgical Society¹ occurs: "In cases of exaggeration of the knee-jerks and rigidity of the limbs it was not essential that there should remain a connection of the cerebellum with the cord below the lesion." This gives what I was obliged to admit² before the important researches of Dr. Bastian. Now I can, as I said in the discussion, withdraw that admission, and adhere to the hypothesis of "cerebellar influx" as I stated it.³ Dr. Bastian's conclusions and mine as to the concern of the cerebellum with exaggeration of the knee-jerks and rigidity in cases of hemiplegia are, as he mentioned in his paper, fundamentally the same. He has advanced proofs where I only speculated. Mr. Bowlby's cases give strong support to Dr. Bastian's position. The counter-evidence adduced by Dr. Buzzard will receive respectful consideration. As the subject is one both of practical and scientific importance, and as Dr. Bastian's paper will not appear in the *Transactions of the Medical and Chirurgical Society* for many months, I refer those interested to his book, *Paralyses: Cerebral, Bulbar, and Spinal*, 2nd edition, pp. 216 *et seq.*

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THE DYNAMOMETER AS AN INSTRUMENT OF DIAGNOSIS.

THE dynamometer is gradually making its way for its obvious purpose—namely, to measure accurately the strength of paralysed muscles of the upper extremities, and to determine the slightest difference in the muscular power of the two hands. Only flexile power is measured; the capacities and ranges of other movements are not even guessed at.

My object in this brief note is to remind those who use the dynamometer of its latent potentiality in making a diagnosis. At Bath, as at other spas where a number of rheumatic and gouty people crowd for relief, we have continually to bear in mind the discrimination of gout and rheumatism from their frequent counterfeit, osteoarthritis. Three specific types of hand are ever before us: 1, The gouty hand; 2, the rheumatic hand; 3, the osteoarthritic hand. The outward pathological anatomy of the three hands is often singularly alike. To state positively what that morbid anatomy is, merely from inspection and comparison, sometimes eludes the ripest judgment; and the collateral evidence of medical history is not always an infallible guide. For a hand, crippled by an original rheumatism or gout, may be gliding into another arthritic condition, more destructive and therefore more incurable.

Here the dynamometer steps in, and tells us with scientific promptness and decision that osteoarthritis is not merely the disablement of a mechanical apparatus, but a vital atrophy and paralysis as well. The struggling hand scarcely moves the pin on the dial; and I have seen a large masculine hand, not impotent to outward vision, unable to make the pin do more than a shuddering quiver. It is not pretended that this weakness proves the existence of osteoarthritis. Arthritis is assumed to be the postulate of the case; the question for our diagnosis being, What is the nature of that arthritis? Pure gout and rheumatism never produce this wasting and inertia in an early stage; but osteoarthritis often strikes its blow and works its havoc in a period which may be measured by weeks rather than by months.

The dynamometer is useful in showing improvement or further deterioration, and should be frequently employed during our treatment with the Bath thermal waters and their medicinal allies.

The instrument I use is made by Messrs. Krohne and Sesemann. Some explanation of the figures might be engraved on the dial.

Bath.

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HEREDITY AS TO TRIPLETS.

THE following case is not without interest, showing how very hereditary triplets are in some families.

I was consulted by a Mrs. M. B., a Wiltshire woman, aged 44, who gave me this history of her family: Great-grandmother, maternal side, had nine children at three births, triplets each time. Grandmother had seven children, triplets once, twins twice. Her mother had twelve children, once triplets, twice twins, five single births. Her mother's sister had seven children, triplets once, four single births. Her mother and her aunt married two brothers. Her mother had two brothers who married; neither had children. Mrs. M. B. has had sixteen children, triplets twice, ten single births. She has seven girls living. Eldest daughter, aged 25, married, has four children, one triplet and one single birth. Second daughter married September, 1889; pregnant. She states she herself was one of a twin, and her mother also. Her family seems to be well known in the village whence she comes as the "triplet and twin family." Her maternal great-aunt, aged 90, single, is still living, who declares that her grandmother told her that her grandmother informed her triplets were in the family as far back as any record could be obtained.

I do not suppose this case possesses any exceptional interest, and doubtless there are many families where this heredity is still more marked.

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CEDEMA OF THE LARYNX A SEQUEL OF INFLUENZA.

H. W., a girl, aged 23, by occupation a barmaid, came to me on January 14th complaining of great hoarseness. A pale bladder-like projection of the right ary-epiglottic fold was seen with the laryngoscope, hiding the posterior third of that vocal cord, and occupying the interarytenoid region, so as to hide from view quite a third of the glottic aperture. The vocal cords underneath were

¹ JOURNAL, March 1st, 1890.

² Med. Times and Gazette, February 12th, 1881.

³ Med. Examiner, April 5th, 1877, and March 29th, 1878.