TWO CASES OF ARSENICAL PERIPHERAL NEURITIS.

By SAMUEL BARTON, M.D.,

HON. PHYSICIAN TO THE NORFOLK AND NORWICH HOSPITAL.

CASE 1.—Mrs. A. G.—, aged twenty-three, mother of two children (one alive, the other died three hours after birth), was admitted under my care to the Norfolk and Norwich Hospital on Nov. 23rd, 1889. She was conveyed on a stretcher to the ward, as she was totally unable to walk or even stand. She had enjoyed good health, with the exception of suppurating tonsillitis, which occurred for the second time three years ago, when an attendant at the county asylum. There was nothing in the family history bearing on her state. The present attack began on Sept. 28th; the only cause that she could suggest was that she had recently been much frightened by a thunderstorm. She was at this time in her seventh month of pregnancy. The attack began with a severe headache referred to the occipital region. This initial symptom lasted forty-eight hours. On the third day on getting out of bed green vomiting set in, followed by copious watery diarrhoea. This state continued until the sixth day (Oct. 3rd), when she was prematurely confined, the child only surviving three hours. Within a few days the patient noticed that the fore-fingers of both hands were numb; this numbness soon extended to the other fingers, and when she got up on the tenth day after confinement both hands and wrists were numb and tingled. Within the following week the same sensations were felt in both big toes, which gradually extended as far as the knees. She said she distinctly noticed the numbness for some days before the tingling and burning pain began. She soon after began to lose power in the hands and legs, and within three weeks of the beginning of attack could not hold anything in her hands, nor could she stand even with assistance, but could sit erect all day, and had perfect control over sphincters. Briefly her state was as follows on admission: loss of power to extend the hand, great weakness of flexors of the wrist. Dynamometer registered, right grasp 5, left grasp 10; loss of power of ankle dorsoflexion, slight power to extend foot at ankle involving much pain in the calf muscles. Very little power of quadriceps to extend the knee; hamstrings were in a state of semi-contraction. No muscles supplied by the cranial nerves affected, nor muscles of trunk; sphincters normal; great hyperæsthesia of forearm, thigh, and calf muscles, the slightest pressure in these regions gave intense pain, the forcible extension of the knee-joint caused much pain in hamstring muscles; there was also spontaneous pain in these regions, which kept the patient awake at night. She always lay with thighs and knees semi-flexed. No loss of sensation of the skin to touch or prick of a pin, feet always felt cold but were bathed in perspiration. There was absolute loss of muscle reflex in upper and lower limbs, but plantars were normal.

Six weeks after admission motor symptoms about the same; if anything she had more power in the hands. Muscular hyperæsthesia gone, except in the calf muscles; the tingling pains were confined to the feet, but there was marked wasting of forearms and legs, the latter measured two inches less round the calf than on admission. About this time it was noticed that a number of brown maculæ like ordinary freckles, but larger, appeared on the patient's face and legs; at first they were reddish, but soon became brown. She said she never had anything of the sort before. The affected muscles did not react to a strong faradaic current, nor could I, strange to say, get any reaction with a fairly strong constant current.—Feb. 20th (three months after admission): For the last fortnight her muscular power has markedly increased. She can knit, but, owing to the numbness in the end of the fingers, she is unable to work with an ordinary sewing needle. She cannot button any part of her dress unless she can see it. She can stand for a few minutes, leaning with both hands on the back of a chair.

Now the diagnosis of peripheral neuritis in this case from the first could admit of no doubt, but I must honestly admit that the cause was a mystery to me until Jan. 25th, when her husband came under my care in the same hospital, suffering exactly as his wife had done on admission. The

symptoms were those of alcoholic peripheral neuritis without any history of alcoholism. They were too acute for lead poisoning, and there was no Burton's blue line. The sensory symptoms were more marked than is seen in diphtheritic paralysis. She was in no way connected with indiarubber works, to suggest bisulphide of carbon as a toxic cause. She knew of no exposure to arsenic or copper poisoning. The case was too acute for rheumatic neuritis.

CASE 2.—William G.—, aged twenty-three (husband of previous case), a naturalist's assistant, very healthy until two years ago when he had a bite on the forefinger of the right hand from a viper. The hand was much swollen, but he was able to resume his work in three weeks. His work consists chiefly in rubbing a mixture consisting of four parts powdered arsenic to three parts of plaster of Paris into skins of birds and animals (2 lb. of this mixture would be about sufficient for a bird the size of a goose). He takes some of his work home to a room set apart for that purpose. This room his wife has been in the habit of cleaning out twice a week. He says the powder deposits on his eyebrows, eyelashes, and moustache. He has carried on this work for the last seven years. About one month before admission to the hospital he began to suffer from severe frontal and occipital headache, with vomiting of green fluid, followed by diarrhoea and anorexia. Says his eyes are not sore, but his conjunctivæ are much injected. Five days after these symptoms he noticed tingling in the soles of both feet, and soon after in his hands. He endeavoured to work for three weeks, but then had to give up owing to the weakness of his legs, and the pain in his calves. State on admission (Jan. 25th, 1890): He can flex and extend the wrists feebly. Right grasp 12, left 20. On endeavouring to stand the knees collapse owing to weakness of quadriceps. When seated on a chair, with feet on the ground, he cannot raise his toes off the ground, but lying in bed he can slowly flex the knees and ankles. No weakness of trunk muscles or those supplied by the cranial nerves; full control over sphincters. Calves and hamstrings painful; he cannot bear any pressure on them. Lies in bed with knees semi-flexed. Sensation in patches is impaired; conduction is slow. He cannot distinguish two points applied to the skin of legs four inches apart. His feet are always cold, but moist. Patellar reflex slight in both legs; plantars normal; leg muscles flabby, but not markedly wasted. I could not obtain any voltaic irritability of the leg muscles; the right tibialis anticus responded to a strong faradaic current, which did not affect the left. March 1st: No power of flexion or extension at ankles. No true knee-jerk, but, owing to tenderness of ligamentum patellæ, he flinches voluntarily when you strike. Plantar reflexes absent. Sensation much impaired in both legs; in several regions he does not feel a pin-point. Yesterday had much pain in right knee; there is some effusion into the joint. Temperature normal. Muscles still very tender. Patient drew attention to a couple of brown patches on his thigh. As soon as I heard the history I had twenty-four hours urine of this patient saved; the city analyst evaporated it down to a small quantity when arsenic could be distinctly detected. On March 1st I put this patient on two grains of oxide of silver three times a day, in the hope of forming the insoluble arseniate of silver. He rapidly improved from that date, and within three weeks he could walk up and down the ward without assistance. He was discharged on April 5th; the first case on March 22nd. They both walked to the hospital from their home, a distance of one mile, to see me on April 21st. Mrs. A. G.— has slight ataxic symptoms; cannot stand with feet together, and eyes closed; staggers a little when turning round; knee-jerks still absent. All the brown freckles have disappeared. W. G.— feels very tired after the walk; front part of foot comes down with a jerk; knee reflexes normal; sensation in feet to skin stimulation delayed, but present. The close analogy of these cases to alcoholic and other toxic paralysis would leave little doubt that the pathology is the same—namely, an inflammatory state of the sheath, and probably the parenchyma of the peripheral nerves. The moral to be drawn from these cases of peripheral neuritis of doubtful origin is to carefully examine the urine at once for the poisons that are known to produce this state. If I had done this when my first case came under my care, the probability is I should early have arrived at the cause.

Norwich.