The patient, when seen early in March by Dr. Oliver, seemed in perfect health. Although less than four months since the operation, she had gained upwards of 26 lbs. in weight.

A CASE OF PARALYSIS OF THE SIXTH NERVE FOLLOWED BY DIPLEGIA: RECOVERY.¹

By D. J. WOOD, M.B., C.M. EDIN.,
Cape Town.

ON March 28th, 1896, G. H. came to me complaining of double vision. Diplopia had come on gradually during two days, and he was unable to get about properly. There was convergent squint, absence of rotation outwards, and, when tested with lights, the cause was found to be pure right sixth nerve paralysis.

The history was found entirely negative. He was a healthy, well-built lad of 18, singularly free from disease as a child and since. A year ago he had had a severe headache for two days consequent on exposure to the sun when fishing, but otherwise he had been well. His father was a sufferer from gout, perhaps also from rheumatism which had caused distortion of several joints, especially those of the hands. Syphilis was inquired for in both father and son, and was denied, no doubt truthfully.

The diagnosis seemed fairly certain that it was a case of so-called rheumatic paralysis in a case of inherited gout. Iodide of potassium 30 grains daily and milk diet were prescribed.

I saw him next on April 3rd. The day after seeing me he noticed a feeling of numbness in the toes of the left foot, which increased until the leg fell "asleep." Next day it was worse, and the face and hand participated, and by April 2nd there was left hemiplegia from the lower part of the face down and complete in quality.

The orbicularis palpebrarum and frontalis were normal in action: voluntary motions were abolished in the lower left side of the face, but an involuntary smile was fairly symmetrical. The knee reflex (left) was slightly increased; there was no anaesthesia, and I take it that the numbness alluded to was an expression of motor weakness, as sensation was perfect.

As on the first examination, the vision was normal (acuity and fields), the eyes were healthy, and the heart and arteries apparently sound.

The condition seemed now much more serious and to point to a lesion in the upper part of the pons on the right side pressing on the right sixth nerve in its course, and damaging the motor tract and facial fibres before decussation (to the left). Judging from the onset there seemed a chance that the damage was caused from below the pons, but there were no signs of a gross lesion, and repeated inquiries only strengthened the negative history. Temperature was normal. After five days the arm, leg, and face began to grow stronger, and the improvement continued.

On April 15th fresh symptoms appeared, the hemiplegia was nearly gone, but there was some difficulty in speaking and swallowing. The left side of the palate drooped, and as it apparently did not properly close the upper part of the pharynx, the patient could not prevent his breath from escaping through the nose, and a pseudo-dyspnoea resulted. The iodide was increased to 60 grains daily, and liq. hyd. perch. 3ss added.

Next day the articulation was worse, and a paralysis appeared on the right side of the face, similar to that which had disappeared from the left side. The movements of the tongue were impaired in all directions, but specially forwards and to the right. There was still no anaesthesia, pain, sense of illness, nor any trace of optic neuritis. There was, however, some mental weakness of which the patient was aware; he laughed continually when spoken to or looked at, and though conscious of the absurdity, could not control himself.

On April 19th the original hemiplegia had entirely gone,

¹ Read at a meeting of the Cape Town Branch of the British Medical Association.

but speech was so bad as to be scarcely intelligible, and the patient preferred to write. Consonants "t" and "d" were spoken "nee," "k" and "q" as if "h" and "wh," labials "m" and "n" were imperfectly finished off, "r" was produced fairly well. Owing to the paralysis of the lips the mouth remained open, and fluids escaped from the right side during drinking. The voice also was rather low pitched and husky.

From this time general amelioration set in, in less than a week the face was largely under control, speech more intelligible, and the mouth kept shut.

On May 21st, eight weeks from the commencement, there remained only slight weakness of the right face, some huskiness of voice, and the sixth nerve paralysis which ushered in the condition. Unfortunately the larynx was not examined, but I am inclined to think there was some paresis or other abnormality present.

REMARKS.

It will be at once evident that we have here an unusual and atypical form of disease. We may begin by excluding entirely the suggestion of hysteria as the cause. At no period did any of the symptoms afford any ground for such a diagnosis.

The case resembled an acute or subacute disturbance of motor nerve structures in or near the lower part of the brain. From ordinary bulbar paralysis we separate it by its greater acuteness, the higher nerve centres involved, and the recovery of the patient. Pseudo-bulbar paralysis is even more improbable.

Peripheral neuritis seems more likely, but the absence of any toxic cause, and the fewness of the nerves effected, do not warrant the supposition. To account for the right sixth nerve paralysis, followed by the left hemiplegia, one must practically admit the disease to be in the right side of the pons, where also one must look for the cause of the facial paralysis. The locality is further narrowed by the fact that the sixth nerve is probably damaged below the point of decussation (nucleus or below), while the facial fibres (for the left face) were involved before decussating, and this could only be if the lesion were in the upper part of the right side of the pons. The latter right facial paralysis would be accounted for by the disease spreading down the right side of the pons till it reached the right facial nucleus or nerve. Extension of the disease from the right sixth nucleus to the right seventh nucleus would be easy on account of their close relation.

The connection between the nerve cells supplying the lower facial muscles and the hypoglossal nucleus affords a possible explanation of the paralysis of the tensor palati from the close connection between the hypoglossal and accessory nuclei. The point may be noted that the only infranuclear lesion, that of the sixth nerve, remained long after the others had vanished. Paralysis of the right sixth nerve had not entirely disappeared before the end of July; and diplopia to the right side, slight in degree and due probably to contraction of internal rectus, still (September 8th, 1896) persists.

The nature of the disease is even more obscure than its locality. Syphilis and other infective diseases seem entirely excluded, but I am not so certain that with a rheumatic history, even in a heart apparently healthy, we can exclude the chance of an embolic lesion with subsequent degeneration of the nerve substance.

The nearest case seems one related by Gowers of an acute poliomyelitis of the pontine and medullary nuclei and nerves, but in our case there was no symptoms of inflammation.

A CASE OF WANDERING OEDEMA, PROBABLY OF SEPTIC ORIGIN.

By E. W. G. MASTERMAN, F.R.C.S.,
Damascus.

THE following case recently under my care in this city seems to me to present features of a sufficiently unusual kind to be worth recording.

J. T., a Jew, aged 8, was on December 30th, 1896, suddenly seized with pain in the left leg. Shortly after cedema of the leg was noticed gradually ascending from the foot to the

knee. The next day (December 31st) the left arm began to be painful, and to swell rapidly, and towards evening some swelling about upper eyelids was noticed. On January 2nd, 1897, I was called to see him for the first time, and these are my notes:

"The boy presented an extraordinary appearance. Both upper eyelids are tensely oedematous, and also the area between the eyes and over both temples. The lower eyelids are not at all affected, so that the eyes can be seen (with some difficulty) by drawing down the lower lids. The oedema is tense and does not pit. It is very tender. There appears to be no oedema in other parts of the head, but the great enlargement of the temples, etc., gives the appearance of a very large head. The left arm is very swollen and tender. There are a number of irregular raised purpuric patches about the extensor surface of the forearm. The left leg is slightly oedematous (much less, according to the friends, than it was). On the left heel is a small abrasion, said to be since commencement of the illness. There is no oedema elsewhere, and except for the purpura the oedema is pale and not inflammatory. Pulse slightly quickened, and temperature a little over 100° F. Urine examined, no albumen.

January 3rd. To-day both upper and lower eyelids excessively swollen, also temples, behind ears and the occipital regions on both sides. No oedema to be detected on top of head. Eyes absolutely closed.

On January 4th the oedema of the face had very much improved, and he could open his eyes. The swelling in the left arm was better. The purpuric spots were now brownish, raised, and the largest of the size of a threepenny piece. The oedema everywhere pitted on pressure, but was still tender. There was very slight fever, and the patient felt much better.

On January 5th the right arm had begun to swell, and purpuric spots like those on the left arm had appeared. There was a large purpuric spot around the wrist, and many smaller ones on the extensor surface of the forearm. The oedema of the face and left arm and leg had much improved.

On January 7th the face was almost normal, but oedema with great tenderness had appeared on the back. Scarcely any oedema of the left limbs remained. The temperature was 101.2°.

On the evening of January 9th he was seen in extreme pain. The right arm had suddenly become tensely swollen from the elbow downwards, and fresh purpuric spots had appeared, and also a couple of vesicles. There was also considerable abdominal pain and tenderness of the back. The oedema elsewhere had practically disappeared. The temperature was about 101°.

From this time onward there was steady improvement. Oedema gradually disappeared and fever quite ceased. The contents of the vesicles became purulent, and then they dried up.

On January 17th, however, he was suddenly seized with an acute attack of dysentery. Blood and matter was very copious, and tenesmus severe. Under treatment these symptoms rapidly disappeared, and since the 21st convalescence has been interrupted.

As regards treatment, quinine internally, and ext. belladonnæ c. glyc. externally, were used at first; later, lotio plumbi c. opio was found more soothing as an external application. The dysentery was treated in the ordinary way with pulv. ipecac. and mist. ol. ricini.

REMARKS.

The interest of the case lies in the very wandering nature of the oedema, and especially in the sudden swelling of the face and equally sudden disappearance of oedema. The fever, the purpuric spots, and possibly the dysenteric symptoms, point to a septic infection, and possibly it may have been a septic infection from the abrasion of the heel. There were, however, never any red lines visible on the limbs, nor were the lymphatic glands enlarged or painful. The following case, which I had a short time previously, may throw some light on the above. A boy, aged 18, came to me with a small but intensely painful abscess of the axilla, which I opened. Two days later the whole arm on that side became tensely swollen and painful, and the temperature went up to 103°. In two days more the boy was unable to leave his bed, on account of

a similar swelling affecting both legs. The arm and the abscess rapidly improved, but it was two weeks before the fever left him and the condition of the legs improved. They, however, remained oedematous for several weeks more. Here, however, there were distinct red lines in the course of the superficial veins, and I think the diagnosis of a septic phlebitis, due to infection from the axilla, was clear. Although some of the symptoms were different, I am inclined to think, the first-mentioned case was of the same nature, though the *rationale* of the anatomical distribution of the disease is a mystery to me.

TECHNIQUE OF THE TREATMENT OF SYPHILIS BY INTRAVENOUS INJECTION OF MERCURY.

By JAMES B. BYLES, M.B.LOND.,

Late House-Surgeon at the London Male Lock Hospital.

Having read in the BRITISH MEDICAL JOURNAL of December 12th, 1896, Mr. Ernest Lane's able paper on the Treatment of Syphilis by Intravenous Injection of Mercury, I feel it would be of interest to describe in detail the method of carrying out that treatment, together with a few points gained by experience in the wards of the Male Lock Hospital, London, by attention to which the small operation may be successfully performed. That each separate injection be successful is of primary importance, due to the fact that it has to be repeatedly performed while the only veins available are those of the upper arm and forearm, so that if a mistake is made the treatment is probably interrupted owing to the pain and swelling occasioned. The apparatus required is exceedingly simple, and may be narrowed down to a hypodermic syringe with a glass piston, two platino-iridium needles, the solution to be used and a piece of rubber tubing for a tourniquet. The patient has his arm bared to the shoulder, and a couple of turns taken round the upper part with the tourniquet, the ends of which he may be allowed to hold and let go when required. A prominent vein is now chosen for the injection, and as a general rule I should suggest that those actually at the elbow-joint should not be selected, for here, owing to the frequent movements of the part, even the small injury caused by the needle prick may give rise to some slight phlebitis, placing that arm *hors de combat* for the time. Choose rather the cephalic vein or one of the superficial veins of the forearm.

The syringe is now filled with the solution to be used, the needle affixed and sterilised in the flame of a spirit lamp. All air bubbles should be cleared from the syringe, but should any minute ones remain, they will be out of harm's way against the base of the piston, being driven there by holding the syringe at a slightly higher level than the needle whilst injecting. The best form of syringe is one with a glass rod as piston running through a washer of asbestos; it has a capacity of 20 minims, but one holding 40 minims would be better. This syringe may be sterilised for all practical purposes by boiling.

To insert the needle into the vein chosen, hold the syringe almost horizontally, and in the direction of the blood-stream. It is an important point that if the needle has failed to enter the vein, movement of the needle, especially depression, will give rise to dimpling of the skin, whereas if the needle is in the vein, not only is movement of it free, but in many cases blood will pass into the syringe and colour the solution.

The patient is now told to release the tourniquet gently. After this has been done, the least possible quantity of injection is to be thrown into the vein; if the patient feels nothing the vein has been successfully entered and the injection may be completed; if, however, the patient complains immediately of smarting pain withdraw the needle, as the vein has been missed, and the solution is passing into the subcutaneous tissue.

By careful attention to these details the painful bruising arising from a misplaced injection is rendered a matter of impossibility. In a few cases some staining may appear at the site of injection a day or two after, from a few drops of blood from the vein having escaped into the surrounding