temperature, and towards the end of the illness the heart showed distinct signs of weakness, the first sound becoming faint and signs of dilatation being present. The patient became much thinner and lost over 14 lb. in weight. The bowels throughout were constipated; there was no enlargement of liver or spleen. Urine showed no albumen but often deposit of urates. No late rash was noted.

Convalescence was very slow and he suffered much from headache. He was not able to get about much for six months after the bite, but up to the present time no distinctive manifestations have occurred, though he still occasionally complains of giddiness and headache.

Treatment by various drugs—*aspirin*, *phenacetin*, *quinine*, *sodium salicylate*, *sodium sulpho-carbolate*, etc.—did not seem to influence the course of the disease. The two first named were useful when headache was severe. Later on tonics containing *strychnine* proved useful and the condition of the heart began to improve.

Unfortunately, at the time the finger was amputated the diagnosis of the disease had not been made and a thorough blood examination and culture were not instituted. Later on the patient refused to allow this examination.

For some time the diagnosis was difficult. When Mr. E. C. Bevers had seen the case we concluded that the rat bite was undoubtedly the cause of the condition, and he was kind enough to look up the subject and found Dr. T. J. Horder's article. From the description of those cases and from the after-history of this case the condition became evident.

A short time before the patient was bitten he had been using a virus for the extermination of rats, but whether this could in any way be associated with the disease I cannot say.

A very complete article on this disease is published in the *International Clinics*, Series XXI, vol. iv.

Since writing the above I find a case of rat-bite fever was described at a meeting of the Royal Academy of Medicine in Ireland on December 15th, 1911, by Dr. O'Carroll.⁶

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A RULING HABIT STRONG IN DEATH.

BY

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THE case here reported presents, I think, many points of interest.

X. Y., aged 63, the senior partner of a firm of engineers largely interested in Government and other contracts, with works in South London, an old school friend of mine, had for some years been failing in health. Five years ago he had rheumatic fever with heart affection, and I saw him with his medical attendant, Dr. Matthews, of New Cross Road. This illness was followed by Bright's disease, but under Dr. Matthews's care he got about again much as usual. Three years ago he had a large carbuncle, which laid him up for two

about 10 p.m., and was laughing and joking with his wife and son. About 11.30 he was seized with a very violent attack of breathlessness, and got out of bed. His wife found him lying on the floor. With the help of her son he was got on to the bed; it was observed that his left side was useless. He asked for the asthma medicine and various things, and talked sensibly, though his voice was somewhat peculiar. Dr. Richardson, of Boscombe, who had been sent for, gave full instructions, but made a serious prognosis.

The patient talked incessantly until daylight, when his speech became more and more unintelligible, his neck began to swell, and he grew more unconscious. At 10 a.m. on July 8th he appeared quite unconscious, but he went through the motion of taking out his eyeglasses, putting them on, taking his fountain pen from his pocket, and then writing a letter on the bedclothes. During this morning he tried at times to throw off the bedclothes to get out of bed to pass water; and his wife thought, when she took his hand and asked him to kiss her, that he pressed her hand and pursed up his lips.

months. He got about his work again, but the albuminuria continued. He could not be induced to take proper care, but exposed himself in all weathers and took long cycling rides with his son. He kept on his work as usual until May, 1912, though his disabilities increased. He then became dangerously ill, the symptoms being those of heart and kidney disease accompanied by cardiac asthma. Dr. Matthews impressed on him the necessity of giving up his work, but he was unwilling to do so, and asked me to see him. I visited him on June 9th. He was then improving as to the acute symptoms, but was clearly unfit for work owing to advanced heart and kidney disease and arterial degeneration. I pressed him to resign himself to the inevitable, and, with Dr. Matthews's consent, undertook to arrange a consultation.

He improved somewhat, however, under treatment, and insisted on trying a few weeks' change at Boscombe; but on July 7th I heard from his wife that he had not benefited by the change, and had made up his mind to return home on July 8th. On July 7th he was pretty well and cheerful; he went up to bed

I saw him late on the evening of July 8th. He was lying on his back in bed quite unconscious. His face and neck were swollen, his eyes half closed, and the eyeballs prominent. Breathing was somewhat stertorous, quick and irregular, the mouth partly open, and the tongue swollen and dry. The pulse was full and hard, he was quite comatose, and apparently in a hopeless condition. On the morning of July 9th he was quiet and seemed quite unconscious; he was breathing heavily and unable to swallow, but at 10 a.m. he again began to go through his habitual procedure when about to write. His wife was inspired with the idea to see if he could write, and got a writing pad with some paper, put his fountain pen in his fingers, and guided his hand to the right place. He began to write a long and evidently business letter, crossing his t's, and when he thought he had got to the bottom of the first sheet of paper, he turned it over with his little finger (a habit of his) and went on writing on the second sheet.

He continued quite unconscious all day, and on the morning of July 10th made no motion or attempt to write, and continued

if you could - but it would be as how what you would want for a letter of mine

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until the following evening, when the breathing became easier, the swelling of the neck went down, and he swallowed a little fluid given him by Dr. Richardson; a trying cough came on, and his chest seemed clogged up and his mouth full of blood. He died on July 12th, having given no sign of consciousness or answer to his wife's frequent appeals to notice her since July 8th. During the illness there was no action of the bowels, and nothing swallowed until the evening of July 11th, but urine was passed regularly up to the last.

I have set down carefully the account given me by his wife, who was constantly with him. Mr. F. was very methodical in his habits, and had little tricks and habits which were quite characteristic. He went to the works every day about 9 a.m. and had a general look round, and sat down in his office at 10 a.m. to look to his correspondence, and it was at this hour that on July 8th and 9th he began to try and write on the bed; evidently it was a business letter. His partner had written to him that an important customer had called at the office respecting a large order, but would wait to see Mr. F. himself on his return, and probably the letter he attempted to write was in reference to this appointment. The writing, although words, as will be seen from the facsimile, are quite clear here and there, is not sufficiently connected to be intelligible, but I think this was due to the insurmountable difficulty of keeping the pad in apposition with the pen; this, indeed, is clear from the writing itself. The fact that no attempt to write was made after July 9th was, I think, due to continuing haemorrhage. Clearly this was a case of ingravescent apoplexy. I am content to record the facts and leave the explanation to others.

EIGHTIETH ANNUAL MEETING

OF THE

British Medical Association.

Held in Liverpool on July 19th, 20th, 22nd, 23rd, 24th, 25th, and 26th.

PROCEEDINGS OF SECTIONS.

SECTION OF NEUROLOGY AND PSYCHOLOGICAL MEDICINE.

LANDEL ROSE OSWALD, M.B., President.

DISCUSSION ON THE DIAGNOSIS AND TREATMENT OF COMPRESSION PARAPLEGIA.

OPENING PAPERS.

I.—ERNEST S. REYNOLDS, M.D.Lond., F.R.C.P.,
Physician to the Manchester Royal Infirmary.

In opening the discussion I am at once confronted by the term "compression paraplegia," which taken literally limits the discussion to a paralysis of both legs and sometimes of both arms, and caused by something actually compressing the cord. If taken thus literally, however, the discussion might not include a considerable number of interesting and important cases, amongst others those of unilateral compression, those of secondary new growths (in which paraplegia may not be present), those very difficult cases of compression paraplegia in the early stages, and also intramedullary tumours.

Causes.

It is necessary, however shortly, to mention the principle causes of compression paraplegia, and it is simplest to work from without inwards.

First, aneurysms, chiefly in the dorsal region and after eroding vertebrae, tumours sending in a tongue of tissue through the intervertebral spaces (naturally causing unilateral symptoms at first), and parasitic cysts, generally hydatids, similarly reaching the cord from subpleural or subperitoneal connective tissue.

Secondly, affections of the vertebrae. Traumata, such

as fracture dislocations and injuries without apparent breach of continuity (about which I will say something later in considering diagnosis in "compensation" cases); syphilis very rare; tuberculous caries most common of all, principally in the dorsal region, and often causing symptoms only secondarily and sometimes by quite simple and transient cord changes, such as oedema; tumours, generally malignant, sometimes primary, either sarcoma or myeloma, or secondary, carcinoma, sarcoma and lymphosarcoma, often with a very small primary growth in the breast, sometimes overlooked, or removed two or three years ago, or rarely a growth in an old enlarged thyroid gland.

Thirdly, the spinal meninges. Here it is of course possible that the cause, if a tumour, may be in the dura mater only, an important point from a surgical standpoint, but often the pia arachnoid is also affected. The affections are: Haemorrhages seldom causing marked spinal symptoms if extrathecal, but marked symptoms if intrathecal; inflammations, spreading from without, as from bedsores; slow chronic inflammations causing local thickenings either in the dorsal or lumbar regions, or in the cervical region (cervical hypertrophic pachymeningitis), sometimes possibly the result of injury; tubercle and syphilis, the two commonest affections; and tumours, extra-dural chiefly in the dorsal region, sarcoma, fibroma, lipoma, and intra-dural arising from the inner surface of the dura mater, or from the pia arachnoid, such as sarcoma, fibroma, psammoma, myxoma, lipoma, endothelioma, or parasitic cysts. Intramedullary growths may of course cause compression of the cord from within outwards, and if possible, especially for surgical reasons, should be diagnosed from extra-medullary growths; they are more rare than meningeal tumours, and include solitary tubercle, glioma, sarcoma, gumma, and myxoma. Tumours of the cauda equina, of course, must not be forgotten.

Symptoms and Regional Diagnosis.

Obviously in dealing with paraplegia one should first mention the motor symptoms. These may vary from a mere feeling of tiredness and heaviness of the legs to a total loss of power, which may be due to an atrophic paralysis if the cauda equina or lumbo-sacral cord is affected, a spastic paralysis if only some part above this is involved, or a flaccid (not necessarily atrophic) paralysis if a part above the lumbo-sacral cord is completely divided.

If the lesion is in the cauda equina there is an atrophic paralysis of the leg muscles; this is usually limited to those supplied by the sacral plexus, but if the lesion is extensive nerves not higher than the third lumbar may be affected. If in the conus terminalis only (that part of the cord below the origin of the second sacral nerve), the only muscles paralysed would be those of bladder and rectum. In the lumbo-sacral enlargement below the second lumbar root the paralyses would be atrophic with loss of knee-jerks. Anywhere above this there would be, in the parts below, as a rule spastic paralysis; but if the cervical enlargement is involved, an atrophic paralysis of muscles supplied from the area involved by the lesion and, of course, a spastic condition below.

It would take too long to go into the whole question of Bastian's theory, put forward in 1890, of a flaccid paralysis (without marked atrophy of muscles, no great alteration of electrical reactions, and no affection of the grey matter in the lumbar cord), which occurs with a total transverse lesion of the cord in the cervical or lumbar regions. I published a summary of the subject in *Brain*, 1895, and an excellent more recent account of the matter is to be found in the *Medical Chronicle*, vol. xxxvii (1902-03), p. 101, written by Dr. Warrington. This later account on the whole confirms Bastian's view that if the lesion is total, involving, therefore, the destruction not only of the pyramidal tracts but also of the mesencephalic long efferent fibres in the antero-lateral regions of the cord, a flaccid paralysis ensues. It must, however, be remembered that there may be, as I showed some years ago, a total anaesthesia and total paralysis below a dorsal lesion, but with markedly spastic paralysis. The only new contribution I can give to this subject is that of a gentleman who had a flaccid paraplegia due to a small dural tumour at the sixth dorsal level; this flaccidity lasted for many months, and was gradually replaced by a typical spastic condition of the parts below the lesion.