numerous in the cervical region, scanty in the dorsal and lumbar regions (see Figs. 1 and 2). A very few scattered degenerated fibres were seen in Burdach's columns. Degenerated fibres (indicated by black dots) were also seen in the intramedullary course of the posterior nerve roots, between the posterior surface of the cord and the posterior horn of grey matter, and to the median side of the posterior horn (see Fig. 3). This degeneration of the intramedullary portion of the posterior root fibres was best seen in the lumbar and cervical regions. The posterior roots, external to the spinal cord, generally presented no degenerated fibres in sections stained according to Marchi's method. In a few sections, however, degenerated fibres (stained black) were seen in the posterior roots for a very short distance, just outside the pia mater; but this degeneration ended very abruptly close to the cord.

The spinal changes here described are probably secondary, and due to the altered blood condition in diabetes mellitus. At the region where the posterior root fibres pass through the pia mater, on the posterior surface of the cord, they lose their myelin sheaths for a very short distance; and it appears probable that at this point the fibres are specially liable to suffer from the action of toxic substances in the blood.³ The changes in the intramedullary portion of the posterior root fibres could be traced to this point in the case here recorded.

Similar changes in the intramedullary fibres of the posterior root may be detected in tabes.

The knee-jerks and tendo-Achillis reflexes were absent in the case just recorded. These signs have been attributed to changes in the peripheral nerves, and no doubt this is the explanation in many cases. But I have found the peripheral nerves (in diabetes) of normal appearance on microscopical examination, when the knee-jerks have been absent. It is possible that the loss of knee-jerks and tendo-Achillis reflexes may sometimes be due to changes in the intramedullary fibres of the posterior nerve roots. In the case here recorded there were no symptoms of peripheral neuritis beyond the loss of the reflexes just mentioned, but the peripheral nerves were not examined pathologically. The changes in the intramedullary fibres of the posterior roots were, however, sufficient to account for the absence of the reflexes.

REFERENCES.

¹ BRITISH MEDICAL JOURNAL, February 24th, 1894. ² References to other cases recorded are given in this article and in the writer's book on *Diabetes Mellitus and its Treatment* (London, 1898). ³ D. Orr, *Rev. of Neurology*, vol. i, No. 10, p. 658, 1903.

A NOTE ON THE TENDON REFLEXES IN URAEMIA.

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ALTHOUGH uraemia in its various clinical manifestations is so common, I am not aware that the condition of the reflexes in this affection has attracted particular attention, and on looking through the literature of the subject I find it noted that in acute uraemia there is an increase of muscular irritability with frequent exaggeration of the knee-jerks, but there is no reference to the diagnostic significance of the excess of tendon-reflex excitability which is undoubtedly present in some cases to a most remarkable degree.

Since uraemia may occur in persons apparently healthy and may reveal itself by such varied symptoms, it is of importance to recognize any sign which may prove of help in the differential diagnosis of this affection from others which it may more or less closely simulate.

When we consider that convulsions, twitchings, and restlessness are common symptoms in uraemia, it seems probable *a priori* that an exaggeration of reflex activity would frequently be present, and that such is the fact I have satisfied myself from the examination of a considerable number of cases; I find, too, that the condition may show itself not only by an exaggeration of the knee-jerks, but that in severe cases such phenomena as ankle-clonus, knee-clonus, wrist-jerk, and elbow-jerk may all be present and well marked.

My attention was first directed to this subject about a year ago by seeing a patient suffering from well-marked uraemic coma, in whom I was surprised to find that beside exaggerated knee-jerks, ankle-clonus, knee-clonus, wrist-jerk, and elbow-jerk were all present.

The next two cases of acute uraemia which came under my

observation presented exactly similar phenomena, one being a case of coma due to cirrhotic kidneys and the other one of chronic parenchymatous nephritis with oedema, vomiting, and headache, ending a few hours afterwards in coma.

In addition to these cases I have noticed the same signs in other acute cases to a greater or less degree.

Although I do not wish to assert that the reflexes are constantly excessive in cases of uraemia, nevertheless, I hold that this symptom is common, and I will now point out some of the circumstances in which it may prove of diagnostic value.

(1) *In Comatose States.*

In many cases it is very difficult to come to a conclusion respecting the cause of coma, especially when nothing is known as to the previous history of the patient or mode of onset of this condition; specially is it of importance to decide whether the condition be due to a cerebral lesion such as haemorrhage, or to a toxic condition of the blood, such as uraemia.

In many cases, of course, the distinction is comparatively easy from a consideration of the history and accompanying symptoms, for frequently uraemic coma is of gradual onset, and accompanied or preceded by convulsions, twitchings, or restlessness, and is very rarely associated with hemiplegia.

Great difficulties, however, may sometimes arise, for cerebral haemorrhage may begin with a convulsion, it may not be very profound, and may be unassociated with any evident paralysis; and, on the other hand, uraemic coma may supervene suddenly in an apparently healthy subject: may be profound and unaccompanied by convulsions; and, in rare cases hemiplegic symptoms (not referable to an organic lesion) may be present.

In regard to the help in diagnosis given by the examination of the heart, pulse, and urine, it is to be remembered that cerebral haemorrhage is not infrequent in cases of granular kidney, in which affection uraemic symptoms are common, and that albuminuria is occasionally the result of cerebral haemorrhage; absence, however, of albuminuria does not of necessity disprove the existence of granular kidney, for in this affection the urine may be free from albumen for days together.

When all these possible pitfalls are taken into account, it would seem impossible in some cases to make a differential diagnosis between uraemic coma and cerebral haemorrhage, for very little stress can be laid upon the "hissing breathing" which sometimes characterizes the uraemic condition, nor on the state of the pupils, which is subject to wide variations, and indeed the difficulties may be so great that cases are quoted in hospital reports of patients being admitted in an unconscious state, and of death occurring before a definite diagnosis could be made.

It is in such cases as these that I think an examination of the deep reflexes might often prove of much assistance; for if one found exaggerated knee-jerks and ankle-clonus, with, perhaps, other signs of great myotonic irritability, a diagnosis of uraemia might be made; though I am not prepared to assert the converse—namely, that the absence of such signs would negative that affection.

In this connexion reference may be made to the statement by Sir William Gowers, that in rare cases of cerebral haemorrhage there may be an exaggerated knee-jerk with ankle-clonus on one side even from the first. Being unilateral, however, this rare occurrence would not be misleading.

Obviously the diagnosis of uraemic coma from apoplexy is of the greatest importance, since the treatment in the former affection should be active, while in the other non-intervention is indicated.

(2) *In Convulsive Affections.*

The epileptiform convulsions of uraemia may closely simulate epilepsy and other conditions. Although in such cases the diagnosis of uraemia may be easy from the presence of other symptoms, nevertheless the exaggeration of the deep reflexes may be helpful.

It may here be noted that Beevor has pointed out that after epileptic fits the deep reflexes may be exaggerated and ankle-clonus be present, but that this condition only lasts for a few minutes.

(3) In other cases, especially those of granular kidney, where certain symptoms may be present without attracting attention to the state of the urine, such as vomiting, headache, *malaise*, dyspnoea, sleeplessness, etc., an exaggerated reflex excitability might lead us to suspect that uraemia was impending, and make us cautious in giving morphine for the relief of such symptoms.

It has been stated that the presence of true ankle-clonus shows almost invariably an organic lesion (lateral sclerosis) of the spinal cord, but as already mentioned, Beevor has shown that it is not uncommon for a few minutes after an epileptic fit. Dudley Buxton has proved that it may be present from the administration of nitrous oxide gas, and Angel Money has demonstrated its existence in some cases of nervous debility, and also, though rarely, in wasting illness such as cancer.

Since I am not aware that the presence of ankle-clonus and other signs of exaggerated reflex excitability have previously been noted in cases of uraemia my object is to draw attention to the fact and to point out its clinical significance from a diagnostic standpoint.

I may say that I do not wish to assert that the above-mentioned exaggeration of the reflexes is present in all, or even in most, cases of uraemia, for the investigations which I have made upon this point do not bear out such an assumption; indeed, when we consider how complex is the uraemic condition, and how variable are its manifestations, it would be surprising to find any symptom common to all cases. There is no doubt, however, that exaggerated reflex action is a symptom which is present in some cases, and that its "presence" may be of considerable diagnostic import. I will not dwell upon a theoretical explanation of the phenomena of increased reflex excitability in uraemia, for it is obvious that renal inactivity may lead to the presence in the system of some toxic material, whose effects are comparable to those of strychnine or the toxin of tetanus; indeed, all the symptoms of uraemia may be explained by the action of toxic substances on the nervous system, and the varied clinical phenomena observed may reasonably be referred to the preponderating influence of one or other of such toxins.

A NOTE ON "BRACHIAL NEURALGIA."

By HOWARD DISTIN, M.B.LOND.,
Enfield.

DR. SIMON's short article on the above subject in the BRITISH MEDICAL JOURNAL of July 11th last was of the greatest interest to me, as I have had two cases under my care quite recently which coincide in almost every particular with those quoted by him.

CASE I.—A retired army officer, aged about 65, who is an enthusiastic gardener, and quite accustomed to using the spade, became conscious last winter of some loss of power in the right arm, together with pain in the shoulder radiating down the limb to the fingers. The cause of these symptoms was quite clear. He was digging for hours one day whilst suffering from synovitis in the knee, and his efforts to keep the leg straight threw an unusual amount of strain on the arm. In a short time the paresis had so increased that he was unable to give an appreciable grip with the hand. The pain also became so intolerable, especially at night, that he was able to get only occasional snatches of sleep, and he told me he could have cried with the agony. He was accustomed to take a long walk every morning before breakfast, and, the weather at the time being cold and very damp, he found the pain much aggravated thereby, and he used to come home utterly exhausted. No treatment seemed to have the slightest effect, and the pain gradually wore itself out, but even now there is some loss of power. While the trouble was at its worst the patient was unable to shave himself, not so much from inability to raise the razor to the cheek, but from the intense pain caused in the effort to do so. In this case there was fortunately no great deterioration in the patient's general condition, considering the bad time through which he passed.

CASE II.—A gentleman, aged 73, long since retired from an occupation which entailed a large amount of manual work, began to suffer from pain in the region of the shoulder-joint. He had a few days previously assisted to lift his wife, who had had a serious illness, from her bed to a couch, and in doing so he admitted that it had cost him an unusual amount of exertion, which he noticed at the time. In a few days the pain had extended down the arm, especially along the course of the ulnar nerve, to the finger tips. I particularly observed that the most painful spots were over the acromion and along the course of the short head of the biceps, thus peculiarly corresponding to Dr. Simon's first case. Muscular tenderness, especially in the upper arm, was very marked, the myositis being evidently secondary to the perineuritis. There was progressive loss of power, again caused by the extreme pain produced by any effort to raise the arm. Brushing the hair became impossible except by lowering the head to the level of the hand, and the greatest inconvenience was experienced in writing, a prolonged rest being imperative after every few lines. But this patient also complained chiefly of the distress caused by the pain in the arm at night. Continuous sleep was out of the question, and he spent several hours every night sitting up in an armchair getting what little rest he could. This went on for months, and, of course, it told considerably on his general health. In this case also no treatment seemed to produce any improvement in spite of the patient consulting two neurologists of great experience, both of whom warned him that his recovery could

only be very gradual. Poultices, liniments, ointments, and blisters were tried locally, and internally sodium salicylate, potassium iodide, arsenic, colchicum, phenacetin, antipyrin, etc., were each tried in turn without success. Considering the age of the patient, it was surprising to me that he did not become more seriously ill owing to prolonged loss of sleep.

In each of the above cases the patient had always been most regular and abstemious in his habits. In both, strain, otherwise traumatism, was undoubtedly the exciting cause, although this only clearly followed upon a depressed condition of health in Case II. In neither case was muscular atrophy very marked. The distressing pain at night seemed to be quite characteristic. It was not brought on by the patient becoming heated, or by the limb being exposed. In each case it came on shortly after the first sleep (in other words, after the limb had been still for an hour or two). Consequently, after midnight very little further rest in bed was obtainable owing apparently to the impossibility of placing the limb in a comfortable posture. What to my mind is the most important point of all, and here I am sure I am echoing the opinions of the patients, is the fact that all forms of treatment were equally unsatisfactory with regard to the relief of pain.

In neither case did I employ morphine, as in my opinion its use causes the last state of the patient to be worse than the first.

In conclusion, any one who reads Dr. Simon's article in conjunction with the description of the above cases must be struck with the many points of similarity.

Since writing the above, there has been a very gradual improvement in the condition of Case II, effected by bathing the shoulder and upper arm with very hot water thrice daily, a remedy suggested by one of the consultants. But perhaps the result has been rather *post hoc* than *propter hoc*.

RESPIRATORY SPASM FOLLOWED BY CESSATION OF BREATHING IN A RECENTLY-BORN CHILD.

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IN view of the difference of opinion held in regard to the production of infantile spasm or stridor, particulars of the following case that came under my notice may be of interest.

Late in the evening of March 2nd last I was called by Dr. J. Hamilton Campbell, of Partick, to see a child that suffered from difficulty in breathing. The history of the case was as follows:

The baby, a first child, was not quite 3 days old, and although forceps had been used to assist its delivery no real difficulty was met with, and labour was regarded as normal. Both parents were young and healthy, but the father was of a decidedly neurotic type. The child was a plump, well-developed boy, without any traceable mark or abnormality, who, until thirty-six hours after birth, showed no unusual symptoms. About this time he appeared to be irritable, and cried a good deal, and when he cried his face became rather deep in colour. During the night following this he slept well, but in the morning the nurse stated that, on awakening, after uttering one or two cries, he appeared to cease breathing and became livid. She placed him in a hot bath without any effect, and it was only after artificial respiration had been carried on for some time that his normal colour returned. Similar attacks followed with brief intervals, during which his breathing and cry were natural.

Dr. Campbell, having been summoned, found that the child was alive, though its pulse could not be felt at the wrist, and its heart's sounds were so slow and feeble that they could barely be detected. The attacks occurred without any apparent cause, as the child lay breathing naturally, with eyes shut, and sometimes when asleep. They lasted from fifteen to thirty minutes, with intervals of from five to ten minutes, and during these intervals the baby took nourishment readily from a spoon. Breathing appeared to be arrested at the end of inspiration, and it is doubtful how far artificial respiration affected the duration of the attacks, as only a shallow sigh could be heard at long intervals. Ether was injected hypodermically, and used by inhalation without any effect.

State on Examination and Progress.—These attacks had been in progress for over twelve hours before I saw him, and he was then deeply cyanosed, although artificial respiration was being carried on. Supported in the arms of the nurse, he lay limp and motionless as if in the last stage of asphyxia. The artificial respiration was continued for some time without any apparent effect, although it probably kept the child alive, but shortly after oxygen had been brought into use respiration gradually became established, and his normal colour returned. For about five minutes the patient seemed peaceful and comfortable, but