coloured. There was no blood count. At the necropsy the spleen was found greatly enlarged, 2 kilos in weight, and had enormous caseous masses scattered through it. There were a few old granular tubercles in the lungs.

In D. D. Stewart's case of acute tuberculosis of the spleen⁷ the patient had an illness lasting in all a little over two months. Typhoid fever was suggested. There was no increase in the number of red blood corpuscles, which were at one count below four millions. The statement is made that there was a tendency to cyanosis and continued blueness of the extremities and "cold water was resorted to to reduce the temperature."

Cyanosis and polycythaemia are met with then in primary tuberculosis of the spleen, but it does not seem at all likely that the cases of chronic cyanotic polycythaemia are due to this cause. In the series which I have collected there were three necropsies, and no mention is made of caseous masses or miliary tubercles in the enlarged spleen, and, moreover, the chronic course—eight or ten years in some cases—is opposed to it.

In studying obscure cyanosis it is well to bear in mind the possibility of chronic poisoning with the coal-tar products. Dr. W. B. Platt sent me the report of a very interesting case of two years' duration without any enlargement of the spleen or hyperglobulism. The patient denied having taken any drugs, but Dr. Platt has recently found out that she had taken for nearly two years a quack medicine which contains acetanilide.

In a case of chronic acetanilide poisoning reported by Stengel and White⁸ the cyanosis was extreme and the spleen could be felt 2 in. below the ribs. The red blood corpuscles were only three millions per cubic centimetre, but the most remarkable feature was the extraordinary increase in the number of nucleated red blood corpuscles, which were on one occasion up to 71,400 per cubic centimetre.

I would suggest to those who meet with cases of chronic cyanosis with polycythaemia to study carefully the condition of the heart and lungs, and in the blood the specific gravity and the viscosity, as the cyanosis probably depends upon the latter and upon the increased difficulty in the flow of the blood through the capillaries.

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CHANGES IN THE SPINAL CORD IN DIABETES MELLITUS.

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A NUMBER of cases are now on record in which degenerative changes have been found in the posterior columns of the spinal cord in diabetes mellitus. In 1894 I recorded¹ two cases in which these changes were present.² Recently I have found similar changes in another case of diabetes mellitus of a very severe form.

The patient was a man, aged 25, who was admitted into the Ancoats Hospital, April 6th, 1903. The diabetic symptoms had been first noticed nine months previously. The disease had developed after a period of mental worry and anxiety caused by loss of employment. The man was much wasted. The urine had a specific gravity of 1032; the daily quantity varied from 150 to 200 ounces; the amount of sugar was usually from 18 to 25 gr. to the ounce; a trace of albumen was present, and a deep brown-red coloration was obtained with perchloride of iron. The knee-jerks and tendo-Achillis reflexes were absent. There were no signs of tuberculosis at first, but a slight dullness at the apex of the left lung was detected about three weeks after admission. On April 28th symptoms of diabetic coma appeared, but passed away after the administration of large doses of sodium bicarbonate, with urotropine and purgatives (castor oil and calomel). After a temporary improvement the weakness increased, and death occurred on May 11th, not from coma, but apparently from asthenia.

The necropsy did not reveal any macroscopic change of importance beyond early tuberculosis of the lungs and the marked general wasting. The pancreas, kidneys, and liver appeared normal on macroscopical examination. The spinal cord appeared normal at the time of the necropsy, but after hardening in Müller's fluid a slight change could be detected, on section, in the columns of Goll in the cervical region. To the naked eye Goll's columns in the cervical region appeared slightly paler than the other parts of the white matter. The change was only seen in the cervical region.

Specimens were embedded in celloidin, and sections stained with aniline blue-black, logwood and eosin, and according to the methods of Weigert, Pal, Van Gieson, Marchi, and Bielschowsky.

Fig. 1.—Diagram showing position of degeneration in section of cervical region of the cord, stained according to Marchi's method. Degenerated fibres represented by black dots.

Fig. 2.—In the centre of the figure, transverse section of Goll's columns of cervical region of spinal cord under high power of microscope; showing numerous degenerated fibres, stained black. (Marchi's method). In Burdach's columns at each side very few degenerated black fibres are seen.

Fig. 3.—Degeneration of intramedullary fibres of posterior nerve roots between the surface of the cord and the posterior horn of grey matter. Degenerated fibres stained as black dots, Marchi's stain. Lumbar region of cord. Extramedullary part of posterior nerve root, seen at upper part of figure, is normal.

Microscopical examination of the spinal cord revealed a slight excess of neuroglia connective tissue in Goll's columns of the cervical region. This was best seen in sections stained with aniline blue-black, and according to Van Gieson's method. Even to the naked eye, in sections stained according to these two methods, Goll's columns appeared of a deeper colour than the rest of the white matter. The nerve fibres of Goll's columns were mostly diminished in size; the myelin sheaths and axis cylinders were usually smaller than in Burdach's columns or in other parts of the white matter. In some parts, chiefly at the posterior third of Goll's columns and close to the posterior median septum, a few nerve fibres with swollen axis cylinders were seen. Also in this region a few fibres with distended myelin sheaths were detected (these were best seen in sections stained according to Weigert's method).

In sections stained according to Marchi's method, degenerated fibres, stained black, were seen in Goll's columns in the cervical, dorsal, and lumbar regions; but they were most

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.

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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.