Counter-irritation sometimes lessens the pain, and the actual cautery is certainly the most effective form. A local measure that often does good is the deep hypodermic injection of cocaine, repeated daily for two or three weeks. It unquestionably promotes the subsidence of the sensitiveness, provided the ease obtained is not abused by increased exertion. Probably eucaine would be equally effective.

At present we are without any direct evidence of the real nature of these affections, such as can only be furnished by the microscope and modern methods of discerning that which it reveals. We cannot wonder at our ignorance, still less complain of it, for it is only quite recently that the minute structure of the sensory elements of muscle and tendon has been clearly perceived, and much of the normal structure still remains obscure. It is one of the departments of pathology in which the recognition of the changes that attend disease has to come long after the full discernment of the normal structure. We must therefore be content to wait, and content also meanwhile to rely on the apparent meaning of the symptoms of disease, as far as that meaning can be made out. Opportunities for pathological research are rare. Possibly it would be more true to say that they are rarely seized. They are not uncommon, but need to be watched for to be found. These diseases have little gravity, so far as concerns the mechanisms by which life is ended. The processes that cause death absorb attention, and minor maladies which coexist are naturally disregarded. I shall be indeed glad if what I have said to-day should lead you to look for and to take advantage of opportunities which others may use, if you cannot do so yourselves.

NOTE.—The recent investigations of Cattaneo, Sherrington, Ruffini, Batten, Horsley, and others have added much to our knowledge of the structure of the muscle spindles, but leave their physiology still in the realm of conjecture. Still more is this true of what may be called their clinical pathology, the attempt to discern the part they play in disease. The suggestions made above are, I think, as much as is at present justified—that their normal function may be described as that of "muscle meters." One other remarkable fact may be referred to. It has been definitely ascertained that when all the fibres of a muscle perish in consequence of division of its nerve, or of atrophic disease due to changes in the spinal cord, those that are contained in the muscle spindle remain undegenerated. It is so, even when the nerves within the spindle perish in consequence of their separation from the posterior ganglia. The intrafusal muscle fibres (as those within the spindle are termed) clearly possess some independent vitality. We can only associate this with the remarkable mass of nuclei which occupy the fibres in the middle ("equatorial") region of the spindle. There, for a short space, they seem to replace the transverse striation, which reappears beyond them. Wherever we can trace the element on which vitality depends, it is a nucleus with protoplasm around it. The only reason we can conceive for the presence of these nuclei in the muscular protoplasm, is to secure independent vitality, and it is the only mechanism we can at present perceive by which their nutrition is maintained when all other muscular fibres perish. Far as the retina is removed from these structures, we seem to have in it a peripheral vital centre. Its ganglion cells are believed to control the nutrition of its elements and of the fibres of the optic nerve. But a ganglion cell is simply a nucleus surrounded by circumscribed protoplasm, and a vital influence may be equally exerted by nuclei whose individual protoplasm is not thus limited. The most important researches regarding the muscle spindles are contained in *Brain*, vol. xxvii, 1898, and to Dr. Batten's valuable paper, references to earlier writings are appended.

THE MEDICAL PROFESSION IN AUSTRIA.—According to the Austrian *Medizinalkalender* for 1904 the total number of medical practitioners in the Austrian Empire at present is 11,689. The number last year was 11,339. They are distributed in the several provinces as follows: Lower Austria, 3,454; Upper Austria, 396; Salzburg, 133; Styria, 692; Carinthia, 151; Carniola, 109; Coastland, 352; Tyrol and Vorarlberg, 598; Bohemia, 2,905; Moravia, 955; Silesia, 240; Galicia, 1,413; Bukowina, 146; Dalmatia, 145. In Bosnia and the Herzegovina there are 108 practitioners. In the principal cities they are distributed as follows: Vienna, 2,721; Linz, 63; Salzburg, 44; Graz, 274; Klagenfurt, 41; Laibach, 41; Trieste, 188; Innsbruck, 70; Prague, 580; Brünn, 186; Troppau, 39; Lemberg, 312; Czernowitz, 60; Zara, 20.

CHRONIC CYANOTIC POLYCYTHAEMIA WITH ENLARGED SPLEEN.

By WILLIAM OSLER, M.D., F.R.S.,
Professor of Medicine, Johns Hopkins University, Baltimore.

At the meeting of the Association of American Physicians in May, 1903,¹ I called attention to a group of cases characterized by chronic cyanosis, polycythaemia, and enlargement of the spleen. The chief symptoms were constipation, headache, vertigo, and weakness; in some cases the patients were in very good health, in spite of an intense cyanosis. The 3 patients which I have seen present a fairly well-defined clinical entity. I was able to collect from the literature 6 additional cases; of the 9, 6 were males and 3 females. The youngest was 35 years of age. The cyanosis was the most remarkable feature, without, as a rule, any respiratory distress, and without any obvious cause. Advanced emphysema and chronic heart disease could be excluded in all the cases. The blood presented a greatly increased viscosity, an unusually dark colour, and in 8 of the cases the red blood corpuscles were above nine millions per cubic centimetre. The haemoglobin ranged from 120 to 150; the leucocytes showed no special changes.

In 7 of the 9 cases the spleen was enlarged, in 4 reaching nearly to the navel. Pigmentation of the skin was present in 5 cases. The symptoms were chiefly sensations of fullness in the head, headache, vertigo, weakness, and prostration. Constipation was present in 4 cases, attacks of nausea and vomiting in 2. There were necropsies in 3 of the cases. In Case iv (Stockton) the heart was normal. There was moderate emphysema of the lungs, with cyanosis and moderate enlargement of the spleen. In Case vi (Cabot) the patient died comatose, with cerebral haemorrhage. There was passive congestion of all the organs. In Case ix (Satndby and Russell) there was hypertrophy of the left ventricle and great enlargement of the spleen.

The following additional cases may be mentioned: Dr. J. N. Hall² of Denver, reports a very characteristic case in a woman with cyanosis of an extreme grade, dyspnoea, and a blood count of nearly ten millions of red blood corpuscles per cubic centimetre. Türk presented to the Medical Society in Vienna in January, 1902, a woman, aged 40, who had had enlargement of the spleen for about ten years with remarkable cyanosis, and a polycythaemia of nearly ten millions; haemoglobin, 180 per cent. Dr. Joseph Collins reports a case³ of an Irish girl, aged 24, with an extreme degree of cyanosis of the face and extremities, marked constipation, pain in the side, and a blood count of 9,800,000. The spleen was greatly enlarged. In a recent number of the *Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie* (Bd. xi, Heft 4) Dr. Rosengart discusses the question of splenic tumour and hyperglobulism. His patient was a man, aged 41, who complained of stomach troubles, vertigo, and loss of weight. The skin of the face was mottled with yellowish and violet shades, but was lighter than in the ordinary cyanotic discoloration. The hands were of a purple-red colour. The mucous membranes were very dark. The patient had marked dyspnoea. The red blood corpuscles were ten millions, and the haemoglobin up to 190. The spleen was enlarged. There was slight albuminuria. The case is similar to those reported in my paper, but, not aware of this group, Rosengart leans to the belief, based on certain cases reported in the literature, that the condition is one of tuberculosis of the spleen.

Scharold⁴ in 1883 reported a case of miliary tuberculosis of the spleen without infection of other organs. There was no blood count, but the colour of the face is reported to have been between red and deep blue. Rendu and Vidal⁵ report the case of a policeman who had an attack of vomiting without apparent cause, with dyspnoea. The temperature was normal. Red blood corpuscles, 6,200,000; leucocytes, 6,000. This count gradually diminished. On examination, skin sub-icteric, cyanosis of face and hands marked, to a less degree, all over the body. A tumour, evidently the spleen, reaching from diaphragm to iliac crest. Eventually ulcers developed on tongue, and the liver became enlarged. Necropsy: Spleen adherent to diaphragm, fibrous on section, filled with caseous masses.

Collet and Gallavardin⁶ report a case of a man, aged 60, with an enlarged, not painful, spleen of two years' duration, without any cachexia and without any leukaemia or fever. There was also enlargement of the liver. The face was very congested (*très colorée*). The blood from the finger was deeply

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.

REFERENCES.

- ¹ *American Journal of the Medical Sciences*, August, 1903. ² *Medical News*, New York, 1903. ³ *Ibid.* ⁴ *Aertzt. Intelligenzbl.*, München, 1883, No. 32. ⁵ *Bull. et Mém. Soc. Méd. des Hôpitaux*, 1899, 3 S xvi, 528. ⁶ *Arch. de Méd. Exp. et d'Anat. Path.*, March, 1901, No. 2. ⁷ *Trans. Assoc. Amer. Physicians*, vol. xvi. ⁸ *University of Pennsylvania Medical Bulletin*, February, 1903.

CHANGES IN THE SPINAL CORD IN DIABETES MELLITUS.

By R. T. WILLIAMSON, M.D. LOND., F.R.C.P.,
Physician, Ancoats Hospital; Assistant Lecturer on Medicine, Owens College, Manchester.

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