observers, that increased elimination of phosphoric acid occurs chiefly in depressed conditions of the nervous system, and that there is evidence of lowered vitality generally, points, to my mind, rather to defective than to increased metabolism, that the tissues are unable to utilise the phosphorus brought to them for nutrition, and that consequently a greater amount has to pass through the system daily. I am therefore inclined to classify the first two groups as *cases of excessive elimination of phosphoric acid dependent upon defective nutrition*.

With regard to the last two groups, the one relating to the connexion between sugar and phosphoric acid in diabetes mellitus is the more interesting. Many explanations have also here been suggested. One, that phosphoric acid replaces the sugar in consequence of some occult change in the nervous system, may be dismissed as visionary and vague. The others that occur to me as being most likely are (a) that instead of the sugar passing into the urine, it becomes partially oxidised in the system into a series of acids—oxy-butyric, crotonic, glycollic, lactic, &c.,—which, circulating through the tissues, dissolves out the phosphates, especially the earthy salts. This explanation receives support from recently ascertained facts respecting the nature of the “acid intoxication” that so frequently marks the course of diabetes. The other explanation is that in diabetes mellitus, as is now a well-recognised fact, there is a considerable elimination of phosphorus in an unoxidised form, as lecithin, or glycerine phosphoric acid. Now, should improved oxidation take place in the body, this unoxidised phosphorus would probably undergo oxidation and appear as phosphoric acid. This view is supported by the fact that in the two cases in which sugar had been observed, but had disappeared and been replaced by an excessive excretion of phosphoric acid, the tendency was towards recovery. At first, with defective oxidation there was sugar and probably unoxidised phosphorus; with improved oxidation the sugar disappeared, and the lecithin or glycerine phosphoric acid was oxidised into phosphoric acid; and a further improvement led to the disappearance of the excessive excretion of phosphoric acid and ultimate restoration of health. No doubt both conditions—namely, the imperfect oxidation of sugar into intermediate acids and the oxidation of unoxidised phosphorus into phosphoric acid—are at work in these cases. With regard to Group 4, which ran a distinct course without association with any special form of disease, as I have already said they seem to correspond, as far as I have been able to judge, with those cases of oxaluria attended with increased excretion of phosphoric acid described by Beneke, who considered them to be due to the excessive formation of lactic acid in the intestines and its absorption into the body and dissolution of the phosphates out of the tissues. These cases, then, probably depend on a similar condition, such as that which causes the appearance in excess of phosphates in saccharine diabetes. I think, therefore, we may classify these two groups as *cases of excessive elimination of phosphoric acid dependent on abnormal formation of acid within the body*.

With regard to the prognosis in these cases, those arising from defective nutrition are the most unsatisfactory. With rest—or, better still, the employment of massage—they regain weight, and the excessive elimination of phosphoric acid is somewhat diminished, never completely; then when allowed to get about they fall back again, till, owing to the general exhaustion, they become victims to some acute disease, or else drift on into phthisis, or, as Teissier says, sometimes into saccharine diabetes.

With respect to the second division, the prognosis is more favourable. Of the three cases of saccharine diabetes associated with considerable excretion of phosphoric acid, two got quite well; the first step being the replacement of the sugar by phosphoric acid in excess. The second case has remained a mild case of diabetes mellitus for over seven years.

Concerning the cases resembling oxaluria with increased phosphoric acid excretion, they usually get much better under appropriate treatment, but are troublesome from their frequent relapses.

Lastly, in respect to the term “phosphatic diabetes,” as will have been gathered from the preceding remarks, excessive elimination of phosphoric acid is symptomatic rather than a disease *per se*. It would be wrong, therefore, to use such a distinctive appellation without strictly limiting the conditions of its use. And this would be to employ it as a distinguishing term from “phosphaturia.” This latter

admirably describes that condition in which phosphates are precipitated from alkaline urine, whether fixed or volatile; but as it would be awkward to speak of phosphaturia due to deposition, and of phosphaturia due to excessive elimination, I would propose to use the term “phosphatic diabetes” to distinguish the latter, and retain phosphaturia to designate the former, condition.

Queen Anne-street, W.

CASE OF

TREPHINING OVER THE LEFT BRACHIAL CENTRE FOR PARALYSIS OF THE RIGHT ARM DUE TO A BLOW A WEEK PREVIOUSLY; CURE OF PATIENT.

By A. W. MAYO ROBSON, F.R.C.S.,

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THE following case, the notes of which have been furnished by my house-surgeon, Mr. Ashton Street, presents so many points of interest that I venture to think a somewhat detailed description may interest the readers of THE LANCET, especially at a time when cerebral localisation, as applied to brain surgery, is engaging the attention of the profession.

J. S—, aged thirty-eight, was admitted into the General Infirmary, Leeds, on August 9th, under the care of Mr. Mayo Robson. He gave the following account. Whilst walking along the street six days previously he was struck from behind over the head by a buckhorn-handled stick. He does not remember the blow, and was totally unconscious for ten hours. Just before recovering consciousness there was some vomiting. He noticed as soon as he regained his senses that he had lost the use of his right hand. He was dizzy and confused for some days, and had impairment of memory, as he remembered some events in the police-court, but not all. He was so far bright and intelligent, however, as to give evidence in court the day after the accident. He was apparently so little injured that a surgeon who saw him thought his arm was powerless from simple bruising due to the fall, and his head so little damaged as not to require dressing. He noticed that his right fingers and hand began to twitch twenty-four hours before admission.

On admission there was a lacerated wound about $\frac{1}{2}$ in. long, too small even to admit the tip of the little finger, situated over the left side of the skull, $5\frac{3}{4}$ in. from the root of the nose, 5 in. from the left external angular process of the frontal bone, $4\frac{3}{4}$ in. from the root of the zygoma, $6\frac{1}{2}$ in. from the external occipital protuberance, and $1\frac{1}{2}$ in. to the left of the middle line. On digital examination through the integuments very little could be made out, as there was some inflammatory swelling; but on probing the wound bare bone could be plainly felt, and at the posterior part the probe could be felt to drop suddenly for about $\frac{1}{2}$ in. The whole of the right forearm and hand were markedly paralysed, the extensors and supinators being entirely powerless, whilst with the flexors he could barely make his grasp felt. The muscles of the upper arm were only half as strong as those of the opposite side, but the shoulder muscles were equally powerful. Sensation was much impaired, considerable pressure being needed to make him feel; but when he did he recognised the kind of touch and noticed the temperature. There were constant rhythmic twitchings of the fingers, and occasionally of the hand and arm, those of the fingers being about 40 per minute. The hands were of the same temperature, but he had a peculiar numbness in the right. He was able to walk, the right leg seeming to be as powerful as the left; but the cremasteric and knee reflexes were both exaggerated on the right side. He was able to whistle, but not nearly so loud as before the accident, and could only screw his eyes up with difficulty. His pupils were of medium size and reacted quickly to light. There was well-marked cedema of the left optic disc; his sight, however, was good. Hearing was slightly deficient. He complained of pain at the seat of the wound and all over the frontal region, but more on the left side. He was perfectly conscious and answered questions with rapidity. His

breathing was easy and tranquil; his pulse 85, full and soft; his temperature in the axilla 98.4° . He could swallow well, and passed urine normally.

Mr. Mayo Robson at once decided to operate. The patient having been put under ether, the head was shaved and cleaned, first with turpentine and afterwards with perchloride of mercury solution. Under the spray, and with full antiseptic precautions, a crucial incision was made, the arms being $1\frac{1}{2}$ in. long. The flaps so formed were reflected, exposing the wound in the bone, which was found to be a trifle over $\frac{3}{8}$ in. in diameter. The whole piece of bone was depressed, the posterior part being more so than the anterior. The margins of the surrounding bone were remarkably clean cut and circular, almost as if a piece had been punched out. The periosteum from the neighbouring bone was not stripped. A trephine $\frac{3}{8}$ in. in diameter was applied, but as the centre pin could not be used rather more bone was taken from the posterior edge than in front. The outer table and diploë were then raised in one piece by the elevator, but the inner table was found to be splintered in all directions, one piece of the size of a finger-nail depressing the membranes very considerably, just over the ascending frontal convolution. These fragments were carefully picked out, but without using much force, as the dura mater was stripped from them. The dura mater, which bulged slightly into the wound after the bone had been raised, was markedly pulsating; it was not perforated, and the surface seemed perfectly healthy. The wound was syringed out with perchloride lotion (1 in 500), and the bleeding vessels in the flaps, which had been temporarily stopped by clips, were ligatured. The flaps were then stitched with catgut, and a small drainage-tube put in the centre, so that the bottom of it was just free of the dura mater. The wound was again syringed and dressed with a layer of wet carbolic gauze, iodoform, and salicylic silk. The patient took ether very well, but his pupils dilated and remained so during the operation. After the elevation, and whilst the patient was still under ether, it was noticed that the twitchings had ceased, but whether this was due to the anæsthetic or to the operation is not known, as, unfortunately, the condition of the hand was not noticed before the elevation. The whole operation only lasted twenty-five minutes. He soon recovered from the anæsthetic, and for the first hour had great pain in his head, but the twitchings in the hand had ceased; they returned, however, in the second hour with greater severity. At 6 P.M.—i.e., four hours after the operation—he was much easier. Temperature 98.6° ; pulse 80. At 12 P.M. the patient slept, but was restless; he had no pain except slight frontal headache. The hand was much stiller. Pulse 72. The twitchings did not stop during sleep.

Aug. 10th.—Patient felt much better and had no pain; pulse 72.—11th: Slept well in the night, and during sleep the movements of the hand stopped. Wound was dressed for the first time, the spray being used. The tube was removed, as the wound looked well and there was no pus. Movements of fingers were much less frequent.—12th: Patient was so well that he wanted to get up. Temperature and pulse normal. His diet having been confined to fluids since admission, he felt hungry, and asked for and was ordered solid food. Power in the hand had very slightly returned. Twitchings were rather less frequent. Had slight soreness in the concatenate glands. Temperature normal.—15th: Patient dressed, as the glands were rather swollen and painful. There was a little œdema of the scalp, but no suppuration. The centre of the wound where the blow was given gaped a little, as the tissues had been bruised and lacerated. Pulse 70.—17th: Swelling of glands was subsiding. Dressed under the spray, and the wound found healed, except the centre of the cross, which was still open. Twitchings had completely gone, and since the 15th he had only had one or two, when he attempted to raise the arm. Patient could hold his hand straight up without the fingers falling. Slight increase of power when the hand was held horizontally.—19th: Grasp of right hand could be plainly felt in any position, equal to the grasp of thumb and forefinger of the left hand. The œdema of the optic disc had disappeared, the outline being clear and normal.—21st: Grasp of dynamometer with right hand 35 lb., with left 95 lb.—25th: Grasp of right hand 50 lb., left 95 lb.—28th: Patient got up, but felt a little dizzy.—Sept. 3rd: Wound quite healed. Grasp of right hand 70 lb.—11th: Patient discharged cured. Both hands and arms equally strong. No headache or dizziness. Sensation perfect. The temperature throughout had been normal.

Remarks.—The man had evidently only been seen casually by a surgeon on account of the arm, which the patient thought had been bruised by the fall, the head injury appearing trivial; but the case illustrates well the great importance of a very careful examination in all, even trifling, head injuries, as out of this misapprehension arose a medico-legal difficulty, the assailant having been sent for a week to prison for ordinary assault, when it might have been a case of manslaughter. Further points of interest in the case are—(a) The recovery of consciousness and intelligence within a few hours of the accident, although the local paralysis continued; concussion apparently accounting for the former and bruising and pressure for the latter. (b) The exact correspondence as proved by measurement of the brachial centre and the wound. (c) The threatened extension of paralysis to the facial, which adjoins the brachial centre. (d) The congestion of the optic disc on the injured side, which congestion disappeared within a few days of the operation. (e) The exaggerated cremasteric and knee reflexes on the right side. (f) The localised rhythmic epileptiform movements of the paralysed hand and arm, which began on the fifth day after the accident, were forty to the minute at the time of operation, ceased for an hour afterwards, then returned as frequently as ever, to gradually lessen in violence and number until they disappeared on the sixth day after operation. (g) In the success of the measures taken to render the wound aseptic, although it was thoroughly septic at the time of admission, and had been exposed for six days previously. (h) Lastly, in the complete and perfect recovery of the patient.

Leeds.

PYREXIA AND HYPERPYREXIA.

By T. J. MACLAGAN, M.D., M.R.C.P.,

PHYSICIAN IN ORDINARY TO THEIR ROYAL HIGHNESSES PRINCE AND PRINCESS CHRISTIAN OF SCHLESWIG HOLSTEIN.

(Continued from page 416.)

It is evident that the view that the nervous symptoms of hyperpyrexia are caused by the high temperature lacks the clinical support which would commend it to our reason. Experimental evidence seems to afford to it more support. The chief evidence of this kind is that adduced by Wood.¹ He placed rabbits, cats, and dogs in a box, and raised the temperature of this 120° to 130° , and found that in animals so exposed the temperature rapidly rose, and that by the time it reached eight or ten degrees above the normal the animals died. The experiments were evidently performed with great care, and the results very accurately noted. From these Dr. Wood draws the conclusion that it may be regarded as proven that "external heat applied to the body of normal animals, including man, so as to elevate the internal temperature, produces derangements of the functions of innervation, of respiration, of circulation, &c., precisely similar to those seen in natural fever, the intensity of the disturbance being directly proportionate to the rise in temperature." He further adds, no doubt in explanation of his including man in this generalisation, that "the brain of a man is much more highly organised, and no doubt correspondingly more sensitive, than that of a cat; and if a temperature of 113° F. be fatal to the brain of a cat, whose normal temperature is 102.5° , it seems very certain that the temperature of some cases of insolation (113°) is sufficient in itself to cause death in man, whose normal temperature is 99° . Much as one admires the patience and skill with which Dr. Wood made his observations, one cannot fail to see that they do not warrant these inferences, and that Dr. Wood has failed to grasp the correct interpretation and bearing of his own facts.

In the first place, there are no grounds for "including man" in the general inference which he draws. No observations were made on man, and in this matter the results of experiments made on cats and dogs cannot be accepted as applicable to him. Man naturally eliminates through his skin an enormous amount of heat, and possesses a power of accommodating himself to high temperatures, which is not possessed by the animals experimented on by Dr. Wood.

¹ Fever: a Study in Morbid and Normal Physiology. By B. C. Wood, M.A., M.D. Philadelphia, 1880.