effect, an effect peculiar to the extracts of these organs. The poison present in the adrenals is more like an organic acid or a "ptomain" in its action; it paralyzes the spinal cord, etc., and causes death by paralyzing the "bulbar centre," and especially the respiratory centre. Is this, then, the product of proteid disintegration which it is the province of the adrenals to pick out of the circulation and metabolise into a harmless substance, and which, when these organs are diseased, accumulates in the blood, and produces nervous symptoms and hyperthermia; for Foa and Pellacani found that it also produced increase of temperature?

We know that the poisonous alkaloids of animal origin, the ptomaines and leucomaines,²³ are products of "proteid disintegration." The poison of the adrenals is, however, probably neither the one nor the other, but a nitrogenous, non-crystallisable substance akin to Panum's septic poison; such bodies produce hyperthermia, whereas the ptomaines and leucomaines produce hypothermia. The bodies which are nearer proteids, or themselves of a proteid nature, appear to be even more poisonous than their lower metabolites. As is well known, Wooldridge²⁴ obtained a proteid poison from serum which prevents shed blood from coagulating for several hours; and another proteid poison²⁵ from the testis and thymus of the calf, which, injected into the blood vessels of an animal, causes instant death, due to wide-spread intravascular clotting. Wolfenden,²⁶ too, has shown that the poison of the Indian cobra (*Naja tripudians*) and the Indian viper are of a proteid nature.

The presence of urohæmatoporphyrin in the urine of Addison's disease led me to hope that I should find this or some other metabolite of hæmoglobin in the skin of patients sufficiently bronzed for the examination, but in this I have been disappointed. Professor Victor Horsley and Dr. Dingley kindly procured some skin, and the latter some slate-coloured mucous membrane from the mouth, from patients suffering from Addison's disease; but although I detected traces of iron, I could not see any bands. But this is not surprising, seeing how quickly blood becomes altered in those situations.

Dr. Riehl²⁷ and Dr. Ernest Kummer²⁸ have made some observations on the distribution of pigment in the skin in these cases. Dr. Riehl concludes that the pigment is extracted from the blood by the cutis cells, and that it is not produced *in situ* by metabolic processes in the cells of the rete; he also found thrombi in the blood vessels of the skin. Kummer confirmed these results, and thinks that we may assume a disease of the blood as a cause of Addison's disease. But it seems to me that the pigment primarily deposited in the skin is different in its origin from that deposited later, when the blood itself has become deteriorated from the presence in it of the products of incomplete metamorphosis. It is interesting to compare with Dr. Riehl's statement that of Wiedersheim²⁹ on the pigmentation of the skin of lower vertebrates; he shows that the derma is permeated by leucocytes, especially in fishes. These leucocytes penetrate to the superficial layer of the epidermis, and carry pigment granules. Here they take on amoeboid movements, and break up into numerous small, pigment-containing particles, which are taken up by the epithelial cells.

It would appear, then, that comparative anatomy and comparative embryology, physiological chemistry, the evidence supplied by the spectroscope and by pathology, all point to the same conclusion.

Just as there has been a progressive development of vertebrata in time, the fishes appearing in the upper silurian, the reptiles in the carboniferous, the birds in the triassic, and mammals in the jurassic, or even in the triassic systems,³⁰ so has there been a progressive development in their organs. As the animal body became more complex, and a greater abundance of respiratory pigments had become necessary for internal and ordinary respiration, certain organs had to be set apart, or had to take on a new function, in the increased "division of labour." This function in the case of the adrenals was the removal from the circulation of useless and worn-out pigments and their accompanying proteids.³¹

When the adrenals are diseased, these effete pigments and effete proteids circulate in the blood; the former, or their incomplete metabolites, producing pigmentation of skin and mucous membrane, and appearing often in the urine as urohæmatoporphyrin; the latter producing toxic effects, and leading to further deterioration of the blood with its consequences.

NOTES ON A CASE OF TRAUMATIC EPILEPSY SUCCESSFULLY TREATED BY TREPHINING.¹

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Two years and a half ago I admitted A. T., a boy aged 16 years, into the Newcastle-upon-Tyne Infirmary, suffering from fits. He was a healthy looking and well nourished lad, but somewhat of a reserved disposition. It appears that until four months before I saw him he had never had a day's illness, and the history I got was as follows. On a certain day he joined some boys of his own age in the game of tug-of-war. When pulling with all their might, some of the boys suddenly let go; and, as a consequence, the patient fell violently upon the side of his head upon the ground. For a few moments he was stunned, but he soon regained consciousness without vomiting, and was able to go home apparently none the worse for the injury. For ten days he was quite well. One morning, after getting out of bed and whilst in the act of dressing himself, he had his first convulsive seizure. It was extremely sudden in its onset, without any prodromata, and was accompanied by unconsciousness. He was put to bed, and on regaining consciousness complained of pain in the right side of his head. From that day he suffered from several fits daily—each series more severe than the preceding—and followed by an alteration in his disposition; moroseness and sullenness replacing a character previously noticed for its liveliness.

His father is a healthy man, his mother, though the subject of mitral stenosis, enjoys good health; one sister, at the age of 9, suffered from fits which were regarded as epileptic, and which suddenly disappeared after a severe fall, which she had in her twelfth year, after the closure of an extensive scalp wound, the consequence of the fall.

When I saw him, his one complaint, in addition to the fits, was severe pain localised over the right temporal bone, over an area, an inch in diameter, located at a point almost midway between a line drawn from the outer angle of the right orbit to the ear, and about one inch and a half superior to it. He could not bear any pressure applied to this point. Before, and immediately after a fit, the pain over this small localised area was increased in severity, but no fit was ever induced by pressure made upon it. There was nothing to be seen or felt here, the skin was perfectly healthy, it had never been broken at the time of the fall. Whilst the fits were of a purely epileptic character, there are yet one or two points that call for attention. An aura was now felt before nearly all the fits, particularly those that became severe, and were accompanied by unconsciousness. He thought he saw a cat, the picture of which was still present in his mind, even after all his motor disturbance had subsided, and while yet in the transitional stage from coma to complete consciousness. It was noticed that in many of the severer fits the left arm and leg were much more convulsed than the right, and that as regards precedence, they were the first to be affected, the two sides of the face suffering equally. In addition, not only were the left arm and leg frequently the seat of marked paresis, which lasted for several days after the fit; but occasionally they were the subjects of choreiform movements over which he had no control, and which at times passed over to the right arm and leg, but were never of the same extensive range as on the left side where they originated. Frequently, after a severe fit, the stage of stupor was very protracted; for two or three days the patient would lie speechless and deaf, able to take liquid food when brought to him, but never asking for it. On regaining consciousness, and whilst yet deaf, he always at first made known his wants to us in writing. I had been told frequently by the nurses of the great difficulty which they had of keeping him in bed during and immediately after the fits, and happening to be in the wards one night when he was seized with

²³ Cf. A. M. Brown, *A Treatise on the Animal Alkaloids*, 1887; also Sir W. Aitken's little volume on the same subject.

²⁴ *Proc. Roy. Soc.*, xlii, 230-232.

²⁵ *Ibid.*, xl, 134-135.

²⁶ *Journ. Physiol.*, vii, 327-370.

²⁷ *Zeitschr. f. Klin. Med.*, x, 5 and 6.

²⁸ *Corr. Bl. f. Schweiz. Aerzte*, xvi, 15, 16, 1886.

²⁹ *Comp. Anat. Vertebrates*, Eng. trans., p. 16.

³⁰ *Geikie's Textbook of Geology*, p. 664, 2nd ed., 1885.

³¹ *Philos. Trans.*, Part I, 1886.

¹ Read in the Section of Medicine, at the Annual Meeting of the British Medical Association, held in Dublin, August, 1887.

a severe fit, I asked the nurses, after the excessive muscular movements had subsided, to leave him alone so as to see what he would do. Whilst thus unconscious, he made an effort to get out of bed, but in doing so he slightly fell towards his left. With a little difficulty he pulled himself together and began to walk down the ward. It was all he could do to keep on his feet, owing to the drooping and want of power in his left arm and leg, and yet whilst thus unable to move his left leg easily, he at once set off half running, the gait being extremely awkward, from the causes that I have mentioned. He suddenly stopped at the foot of one of the beds in the long ward, knelt down and scanned closely the whole of the floor under the bed. With a little difficulty he got on to his feet again, walked to the other end of the ward, to the table where the wash-hand basin and jug were kept, and attempted to raise the jug to his mouth; the accomplishment of which was prevented by the marked feebleness of the left as compared with the right hand. On coming to himself, though quite aware of having had a fit, he knew nothing of all that I have described. He told me, however, that he had seen a cat, the image of which had lasted a long time in his memory.

On ophthalmoscopic examination, beyond perhaps the slightest hyperæmia, the discs presented nothing unusual; the lungs and heart were healthy; urine was quite normal before, after, and during the intervals between the fits; knee-jerk was generally more marked on the left than on the right side; no ankle-clonus was present, but it was noticed that the push of the left foot and the grasp of the left hand were much enfeebled compared to the same on the opposite side of the body.

Painting with iodine, and blisters were applied locally, bromide and iodide of potassium, liq. hydrarg. bichlor., tinct. bellad. were tried alone and in combination, and in increasing doses without the least benefit.

Failing thus to relieve the patient, a general survey of the history and symptoms of the case—namely, a healthy lad meeting with an injury to his head, this followed in a fortnight by a convulsive seizure, recurrence of fits, now accompanied by unconsciousness, and associated with an excess of movement on the left as compared with the right side of the body, lingering left-sided paresis and choreiform movements, these attended by a localised painful spot on the right temporal bone, pain at which was said to be more severe just before or after a fit—led me to entertain the belief that the epileptic seizures were due to a localised irritation, possibly in the membranes lying upon the motor areas in the convolutions of the right side of the brain. The absence of any break in the skin dissipated any feeling that I might have had of fracture of the inner table of the skull. I suggested trephining as the only likely method of dealing successfully with the fits. My surgical colleagues saw the case with me, and agreed as to the advisability of the operation, recommending, however, the application of the trephine over the painful spot, rather than upon that part of the skull lying upon those portions of the brain now recognised as concerned with the initiation of the movements of the arms and legs—I mean the ascending frontal and parietal convolutions. The painful spot, as I have already mentioned, lay anterior and somewhat inferior to these two convolutions. The operation was performed by Dr. Hume, under antiseptic precautions. A piece of bone, the size of a shilling, was without difficulty removed. The bone was quite healthy, and was easily lifted from its bed, being in no way adherent to the underlying membranes. The dura mater was healthy, in no wise thickened. As the dura mater was healthy it was debated for a few seconds as to the advisability of opening it. By degrees, however, it was noticed that the dura mater was becoming tense, and shortly after this membrane was shot out through the opening beyond the level of the bone, the tension now being very great. Accordingly a crucial incision was made into it, and in a few seconds there escaped from the wound several teaspoonsful of serum, containing a few flakes of lymph. The arachnoid and pia mater seemed healthy, as did also the surface of the brain and the small blood vessels which lay upon it. A very small drainage-tube was inserted, the cut ends of the dura mater were drawn together with delicate catgut, the skin flaps were gently drawn together, a sponge applied, and the ordinary antiseptic dressings placed over all. That evening the dressings were found to be soaking, and on removal a few teaspoonsful of serous fluid were squeezed out of the sponge. More than two ounces of serous fluid escaped that day. The dressings were renewed, and never afterwards required to be changed on account of the escape of fluid. The patient had one or two minor fits on coming out of the

chloroform, and also for two or three days after the operation, but after this they became separated from each other by the interval of a day, then two days, a week, a fortnight, and so on, until no fit occurred during a period of six months. The wound had healed kindly and quickly, without even the least rise of temperature. The patient having long left the infirmary, feeling quite well, no longer having any localised pain in his head, followed for a time the occupation of a telegraph boy, but latterly became the cash boy in a confined counting house. Here he was much harassed by the push of business, the one monotonous call which was made upon him, and by the long hours spent in a wretched atmosphere, and the result was that after being free of fits for six months, he had again to be admitted into the infirmary, where he was placed under my care. The fits were now quite changed in character, the muscular movements were slight, and were never attended by loss of consciousness; the visual aura of the cat was no longer present. The fits were rather those of hysteria, and with rest in bed, iodide of potassium and iron, his general health soon improved, the fits disappeared, and for several months have ceased to trouble him.

Trephining, as a method of treating epilepsy, is only applicable to those cases where there is the history of an injury to the head followed by localising symptoms or by convulsions, either unilateral or more pronounced on one side of the body than the other, or where an injury to the head has been followed by a depressed fracture or separation of a portion of the inner table of the skull. Macewen, of Glasgow, has operated successfully on cases of epilepsy due to injury of bone, and in one case of Dr. Ralfe's—a patient who had been trephined for depressed fracture, and who years afterwards was found suffering from epilepsy—an absolute cure was only effected by the cicatrix due to the trephining operation being again opened up and allowing of the removal of a small portion of necrosed bone evidently belonging to the inner table. As soon as this was done the fits ceased. Gowers speaks of having seen or heard of 65 cases of epilepsy owing to a traumatic cause, and of trephining as the only line of treatment likely to be successful, and that, be it remarked, as in my own case, where neither disease of bone, membrane, or surface of the brain was found at the time of the operation. Relief comes either from the operation acting as a strong counter-irritant lasting all through the period of healing, or from the reduction of tension consequent upon the escape of pent-up serum.

It is several months since this paper was written and read, but the last account that I had of this patient from his mother was that he was still keeping free from fits.

ON PUERPERAL APHASIA.¹

By F. BATEMAN, M.D., F.R.C.P.,

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APHASIA, as a complication of the puerperal condition, does not find a place in our classical treatises on midwifery, and the literature of the subject is so scant that, in the course of my reading, I have only met with one observation recorded in this country, although it has been made the subject of a most elaborate essay by a continental observer, to which I shall have occasion to refer after the detailing of my own case, which I desire to head as follows:

CASE OF LOSS OF SPEECH OCCURRING AS A PUERPERAL SYMPTOM; LACTEAL DERANGEMENT; RIGHT HEMIPLEGIA; FATAL TERMINATION.

On September 7th, 1886, I was requested to see, in consultation with Dr. Watson, of Norwich, Mrs. S., aged 23, who had given birth to her second child eight days previous to my visit. Upon inquiry I ascertained she had never been the subject of any cardiac or rheumatic affection, that she had never suffered from any nervous disorder, and, in fact, that she had always enjoyed good health during her single life, with the exception of a disposition to occasional headaches. Her mother was insane, and died in the Norwich City Asylum, but beyond this fact there was no neurotic tendency in the family.

She was delivered of her first child in July, 1885, and at the

¹ Read in the Section of Medicine at the Annual Meeting of the British Medical Association held at Dublin, August, 1887.