A CASE OF PUNCTURE OF THE BASE OF THE BRAIN BY THE SPOUT OF AN OIL CAN, IN WHICH THERE WAS LOSS OF MEMORY OF PREVIOUS EVENTS.¹

HORACE M. ABEL, & W. S. COLMAN,
B.A., M.R.C.S., L.R.C.P., M.D., M.R.C.P.,
House Surgeon to the Peterborough Registrar to the National Hospital
Infirmary. for the Paralysed and Epileptic,
etc.

THE following case presents features of such interest from a surgical, medical, and psychological point of view, that it seems desirable to place it on record. We have to thank Dr. Kirkwood, under whose care the case was admitted, for permission to make use of the hospital notes.

The patient, G. T., a sober well-conducted railway fireman, aged 36, was brought to the Peterborough Infirmary at 4 A.M. on October 28th, 1893, with the broken end of an oil feeder protruding from his right cheek, a little behind the angle of the mouth. It was stated that, in stepping from the tender on to the footplate of his engine with the oil feeder in his hand, he slipped and fell forwards, the spout of the oil can being driven forcibly into his face. He was then quite senseless, but partially recovered consciousness in a few minutes. When he was being placed on the stretcher someone suggested a coat for his head, and he was sensible enough at that time to say that his own coat was on the engine, so that the loss of memory, afterwards so conspicuous, was not then present.

He reached the hospital less than an hour after the occurrence of the accident, and was seen at once by one of us (A.). He was then quite conscious, and tried to answer questions. There were frequent twitching and jerking movements of the limbs, most marked on the left side, the legs being drawn up and the body bent forwards, as if he felt obliged to sit up. There was no hæmorrhage from mouth, nose, or ears. The metal spout of the oil can was firmly fixed in the base of the skull, and required firm traction with forceps to relieve it from the grasp of the bone, and then, owing to its tapering shape, it was easily removed by the fingers. It passed up-

Fig. 1.—Diagram (from a photograph taken a year after the accident,) indicating the probable course of the tin spout. The direction was not vertical but inclined backwards.

wards and towards the middle line, and the concavity of the spout, was directed from the middle line. The end of the oil can was firmly plugged by bone from the base of the skull, so that it was converted into a blunt instrument in its passage through the brain. No hæmorrhage followed its

removal. The wound was cleansed and a simple iodoform dressing applied. The track was entirely outside the cavity of the mouth.

After removal of the tube the violent jerking movements ceased, a few occasional twitchings only remaining. There was now marked paralysis of the left side of the face, with inability to close the left eye completely, complete paralysis of the left arm, while the leg was comparatively little affected. No hemianæsthesia could be made out. He had become drowsy and confused, and unable to give replies to any but the simplest questions.

At 10 A.M. (six hours later) he was still half unconscious, and had passed his urine into the bed during the night. His pulse was 82, and his temperature had risen to 102°. The pupils were contracted, the right being slightly smaller than the left. They both reacted to light. There was still well-marked paralysis of the left face and arm, and the left leg was weaker. No anæsthesia on the left side. On the right side there was complete anæsthesia of the right eyebrow and of both eyelids, and of the right cheek for an uncertain distance below the lower eyelid. (The patient's mental dullness rendered accurate testing of his sensation extremely difficult.) In the course of the following week the conjunctiva of the right eye became congested, and three weeks later a small ulcer formed on the cornea, which healed without much trouble. Taste and smell were normal on both sides.

Further Course Summarised.—After a few days power began to return in the left side, first in the lower limb; afterwards, but to much less extent, in the upper limb. The face showed little paralysis when at rest, but on any emotional disturbance the paralysis of the lower part of the face of the usual "cerebral" type was very pronounced. The anæsthesia of the right side of the face remained unaltered. The temperature ranged between 99° and 101° for the first week, the maximum being touched on the sixth day. After the seventh day it was normal. The highest pulse rate was 84, the lowest 68; it was usually 80 per minute.

Mental Condition.—During the first fortnight he was drowsy, and slept heavily, but could be roused to answer questions. His answers were often ludicrously incorrect, but he evidently understood the questions. He was very apathetic, and for many days passed his urine in bed. He could not recognise his wife or his old comrades, and he had also difficulty in recognising common objects and their uses. Thus on one occasion he drank his own urine from a vessel, under the impression it was water, and several times he got pieces of ice out of his ice cup, and declared they were sweatmeats. This failure to recognise, that is, to recall previous mental images for the purpose of comparison with present ones, was strikingly shown by his failure for weeks to recognise a fellow workman who met with an accident, and was brought into hospital and placed in the bed next to his. But what was most remarkable was that the whole of his life for twenty years before the accident was wiped out from his memory. He asserted that he had never worked on the railway, and that he was a farm labourer. He had, however, been on the railway for nearly twenty years, and previous to that had worked on a farm. All memory of the accident was gone, and has never returned. After he left the hospital some previous events did return, but, as will be seen later, after the lapse of a year there were still five years of which he could remember nothing.

He was able to leave his bed at the end of eight weeks, and at the end of three months was able to return home with very fair return of power in the leg, but little in the arm. He was kept under observation from time to time. No change developed except some increase in weakness of mind and in irritability of temper.

Nearly a year after the accident one of us (C.) again carefully examined him, and found his condition to be as follows: There was hemiplegia on the left side affecting the arm chiefly, but there was also distinct loss of power in the face and leg. He could walk several miles, but required to take frequent rests. The arm was kept a little flexed at the elbow, and the fingers were semiflexed into the palm. From time to time slow rhythmical simple movements of flexion were noticeable in the fingers. These lasted for several minutes at a time. They consisted in a repetition of the same simple movements. There were no complex co-ordi-

nated movements such as are seen in athetosis. They were not associated with any unusual sensation such as giddiness, and they did not annoy the patient. No loss of power could be detected after the twitching. There was no anaesthesia. The slightest touches were felt, and localisation was perfect. Sensation to pain, heat, and cold was quite normal on this side, and his muscular sense was accurate.

On the right side there was no motor paralysis, but there was definite loss of sensation to all forms of stimuli on the right upper eyelid, forehead, and anterior part of the scalp, corresponding with the distribution of the supraorbital and nasal nerves (see charts). There was also complete anaesthesia

Fig. 2.

Fig. 3.—G. T., eleven months after injury.

of the cornea. On the right cheek about $1\frac{1}{2}$ inch external to the angle of the nose was a small patch with ill-defined borders where sensation was blunted to both tactile and painful impressions. There was no anaesthesia in the district supplied by the third division of the fifth nerve, and the muscles of mastication acted as powerfully on this side as on the other. Taste and smell were unaffected. Vision was normal. On ophthalmoscopic examination the fundi were found to be quite natural. Perimetric charts showed some general contraction of the fields of vision for white (and proportionally for red, green, and blue), but no hemianopic defect. In all other physical respects the patient was quite healthy, except that he had become very fat. His mental condition was extremely interesting. On the whole he was somewhat indolent and apathetic, and there was very little control over his emotions. He laughed or cried at the slightest provocation, and was as likely to do one as the other at any remark which was made, and the emotional disturbance did not seem to be the expression of any mental pain or pleasure. His irritability of temper was said to have increased, and he was often hasty in his language, although not violent.

Condition of Memory.—There still remained a gap in his memory, although a smaller one than that which existed when he was in hospital; then, it will be remembered, there was entire oblivion of all events for twenty years prior to the brain injury. At this later period, however, he was able to recall the more striking events of his life up to five years before the accident—after which all is perfectly blank until about two months afterwards—when he is able to recollect some incidents of his life in the hospital, and he could recall his life in general from then to the time of examination, although he could not trust his memory on small matters. We were unable to trace the development of this partial

return of memory, but it seems to have been in part due to the habit of his "mates" of coming in and talking to him of the past and continually reminding him of occurrences which

were likely to have made an impression on him. There was still extreme difficulty in retaining in his memory any passing events. If he went out for a walk by himself he was unable to find his way back, and often failed to recognise his own house when he was outside, and there was also frequent failure to recognise common objects and their uses, although nothing now occurred so marked as his not recognising the nature of urine and of pieces of ice referred to in an earlier note. There is thus present in a modified degree the condition described by Hughlings Jackson as "imperception," and by Munk and others as mind blindness. There was no aphasia at any time, and no difficulty in expressing himself; it seemed to be the ideas that were wanting, and not the words in which to express them. His reasoning processes were fairly orderly, but as owing to the blanks in his memory he often argues from false premisses, he arrives at ludicrously incorrect conclusions. For example, he occupies one of the houses built by the railway company for their servants, and, as he has no recollection of working for them for five or six years, he argued that he had no right to be there, and insisted with unnecessary warmth that his wife should pack up and leave the house before they got into trouble for being there. This mental condition is increasing latterly. At first there was merely failure to remember and recognise, now he takes action rather impulsively on the strength of false or imperfect memories.

Localisation of the Injury.—From the facts that the mouth was uninjured and that the fifth nerve was injured behind the sphenoidal fissure, it would seem that the oil can must have passed up from the scar under the zygoma to the base of the skull, perforating the great wing of the sphenoid bone. It will be remembered that the concavity of the spout was directed outward, so that the point, after passing through the bone, would pass at first almost at right angles to its previous direction, and then the whole instrument would be pushed bodily some distance into the centrum ovale (following the same kind of movement as that of a sound being passed round the curve of the urethra and then pushed bodily into the bladder). From its length it is certain that it could not have reached the cortex by this route. The

Fig. 4.—Exact outline (natural size) of feeder spout after removal. The dotted line represents the position of the cheek wound; from A to B measures $6\frac{1}{2}$ inches.

passed round the curve of the urethra and then pushed bodily into the bladder). From its length it is certain that it could not have reached the cortex by this route. The

symptoms also point to an injury in the region of the internal capsule. The paralysis, at first almost confined to the face and arm, would indicate, according to Horsley and Beevor's experiments on the monkey, that the anterior fibres of the motor tract were injured, and the absence of hemianopia and of any left-sided sensory symptoms show that the posterior end of the capsule was uninjured. On taking a skull and passing the original feeder spout in the direction above described, it was found to plunge into the brain and to injure the capsule near the genu, as we suppose it to have done in the present case.

Note on Mental Condition.—The loss of memory for events occurring hours or days before an accident involving injury to the head has been frequently observed, and two extremely interesting papers have been published by Mr. Joseph Bell¹ and Sir William Savory.² We are unaware, however, of any case in which there has been so prolonged a period sponged out from recollection as in this case. The symptom does not appear to have any localising value.

With regard to the other mental symptoms, the inability to fix in his memory and to recall visual images of things presented to him now, and his impulsive acting on this imperfect representation of facts would suggest some defect in an interruption of free communication between the higher sensory and motor centres of the man's brain. In the absence of a necropsy to confirm our localisation of the lesion, and to exclude other unsuspected ones, it is impossible to make more than a guess at the physical cause for the psychical condition. The injury we have seen reason to believe was about at the genu of the internal capsule, and would doubtless become more extensive from inflammatory softening during the first few days, so as to encroach on the anterior limb of the internal capsule, of whose functions little is known. Its size increases in animals as we ascend the scale of cerebral development, and it has been proved by Flechsig and others to contain fibres uniting the frontal lobe with the opposite cerebellar hemisphere. Many of the other fibres have been traced back to the grey matter of the pons, but their ultimate destination is unknown. This case would seem to suggest that they form a connection between the anterior and posterior parts of the brain in which motor and sensory processes are probably predominantly represented. The failure to recognise objects (imperfect perception) has for twenty years been held by Dr. Hughlings Jackson to be specially characteristic of lesions in the right occipital lobe; may not the symptoms in our case be due to a cutting off of this connection between this sensory storehouse and the centres for initiating volitional motor which are almost certainly in the anterior part of the brain?

A CASE OF IDIOPATHIC PERNICIOUS ANÆMIA IN WHICH ARSENICAL PALSY HAD BEEN INDUCED TREATED BY BONE MARROW.

By ALFRED G. BARRS, M.D. EDIN., F.R.C.P. LOND.,
Physician to the General Infirmary at Leeds; Professor of Materia
Medica and Therapeutics in the Yorkshire College,
Victoria University.

THE following case of pernicious anæmia, although the only one in which bone marrow has been given under my personal observation, seems to me to be worthy of record at the present moment because it illustrates amongst other things how, in pernicious anæmia, arsenic, though given till arsenical palsy is induced, may fail entirely to affect the blood state, and how the blood state may be entirely removed and perchance cured by the administration of a hæmopoietic tissue, namely, bone marrow.

For arsenic in efficient doses to fail to influence, to some extent at least, the course of pernicious idiopathic anæmia is, in my experience, quite exceptional. As a rule, its administration results in marked improvement if not actual cure for the time being, to be followed very soon by relapse, in which, strange to say, the drug frequently fails to be of

the smallest benefit, and the patient ultimately succumbs to his disorder. This curious behaviour of arsenic seems to me to be quite characteristic of the disease; its failure in relapses is frequently as signal as its success in the first period of the disorder, in which, as I have said, it seems to effect a complete cure.

The patient whose case is narrated below was, when admitted to the infirmary, to all appearances at the point of death. It was consequently thought desirable to push the administration of arsenic, in spite of the constant vomiting which was such a marked feature of the case in its early stages, until large doses had been reached. This was done, until in a month, although the anæmia had not advanced, well-marked palsy was induced. Nervous manifestations are known to occur in pernicious anæmia.¹ I have myself seen a case in which the knee-jerk was absent, and returned under treatment by arsenic, the patient making a complete recovery, only to relapse and die in a few months. There can be no doubt, I think, that the distribution and character of the palsy in the present case point clearly to the arsenic administered as its cause.

This case is the second I have seen in which arsenic given in medicinal doses has produced profound and long-standing palsy. The first will be found very briefly recorded in the BRITISH MEDICAL JOURNAL for 1893, vol. i, p. 239. The boy whose case is there given has not yet recovered the proper use of his limbs, and it is now fully two years since the arsenic was given which produced his paralysis. Such disastrous results, although occurring with very great infrequency, must give us pause, and make us ever watchful for the earliest signs of arsenicism in cases in which large and increasing doses of the drug are being used.

In my own cases no warning of impending danger was given by the occurrence of gastro-intestinal irritation. The arsenic seemed to go direct to the nerves without affecting the stomach or intestine, much in the same way as alcohol will get to them without affecting the liver. I have now made it a rule to watch carefully the knee-jerk in all cases in which arsenic is being administered, as I believe the failure of this reflex to be a sure index of its deleterious effects upon the nervous system.

The administration of bone marrow in cases of anæmia was first suggested in this country by Dr. Dixon Mann.² He used a glycerine extract of marrow, and gave it (1) to a boy suffering from hæmophilia, (2) a girl suffering from long-standing anæmia (chlorosis), and (3) a man the subject of profuse hæmatemesis, and in all with good results. But it was not till Professor Fraser published his case in June of last year,³ that any example of idiopathic pernicious anæmia successfully treated by bone marrow had been recorded.

In what manner bone marrow or its extract after undergoing the process of digestion proceeds to effect the marked changes in the blood which are shown to have been effected in the cases recorded by Professor Fraser and myself, I am at a loss to explain, unless it be by assisting in the effort which the marrow, according to some recent observations of Dr. Muir,⁴ seem to make in pernicious anæmia to supply the deficiency of hæmocytes. I ought to mention that in October last I saw, with Mr. Kershaw, of Pudsey, a case of pernicious anæmia in which bone marrow failed to do good; and in November last Dr. Honeyburne, of Idle, asked me to see with him a patient suffering from profound idiopathic anæmia in which the marrow could not be taken. In the latter case the nature of the anæmia was not at all clear, the case being much complicated by a great enlargement of the liver and some enlargement of the spleen without leucocytosis.

W. R., aged 43, a miner, was admitted on April 21st, 1894, and is still (November 6th, 1894) in the infirmary. He was admitted with extreme pallor, feebleness, restlessness, insomnia, and at times much delirium. He presented no malarial or syphilitic taint, and there was no evidence of plumbism or other acquired toxæmia. During the twelve months before admission he had been unable to work through increasing feebleness, great shortness of breath on even quite trivial exertion, and vomiting. He stated that he had lost flesh, though he was still fairly stout, even to corpulency. He had never lost any blood. He was a short thick-set man, well nourished, the subcutaneous fat being abundant.

¹ On the Association of Disease of the Spinal Cord with Pernicious Anæmia, by H. M. Bowman, M.D., *Brain*, Part lxxvi, 1894, p. 198.

² *Lancet*, March 10th, 1894, p. 599.

³ BRITISH MEDICAL JOURNAL, June 2nd, 1894, p. 1172.

⁴ *Journal of Pathology and Bacteriology*, February, 1894, p. 354.

¹ *Edin. Med. Journ.*, 1883.

² *Lancet*, vol. ii, 1882.