stand questions and to frame answers. In some of the fits observed, the arms and head moved, but not the legs. He bit his tongue, but did not pass urine or faeces. The duration of the fits was from two to five minutes. In one fit, on December 22nd, movements of both legs took place. At a visit on December 23rd, while saying a few words about his case, I laid my finger lightly on the scar over the pulsating gap, on which he immediately turned his head and eyes to the right, becoming instantly unconscious, twitching his right lower lip and his fingers, but not otherwise moving either arm or leg. On December 24th at 9.30 a.m., he had a fit, in which he foamed at the mouth and bit his tongue. Other fits occurred every ten or fifteen minutes during the rest of the morning. The same morning, while his head was being shaved and otherwise prepared for operation, a gurgling cry prolonged into a moan accompanied the fits then and afterwards.

Operation.

Operation was undertaken the same afternoon with a semi-circular flap including the old scar, which was found adherent directly to the brain and dissected off it. The dura mater was found turned out along the margins of the bone aperture, adhering to it and to the outer surface by scar tissue. This was all stripped with a raspatory, and also freed from the inner table for some distance all round the aperture, and laid down on the exposed brain, leaving a central portion uncovered. The hole in the bone was oval, measuring 1 in. or less in one direction and more in the other. On the exposed brain and dura mater, passing between the latter and the skull, were laid pieces of stiffish goldfoil, cut from Ash's No. 60 (a piece about 4 in. square that weighed $\frac{1}{2}$ oz.). It was found impossible to pass in a single oval piece a little larger than the bone aperture with anything approaching smoothness or neatness, so one smaller piece that broke off was laid on as well as could be done, followed by two more pieces, all by this time thoroughly crumpled. The skin flap was closed down by continuous mattress suture, without attempt at ligature of the numerous vessels, that now bled again on removing the compression forceps which had been applied to the cut edges. Finally, all was covered up under cyanide gauze and capelline bandage.

After-History.

He slept well the next night, but had a fit early the next morning. During the day he was at first depressed, then excited, and towards night became delirious and hysterical, and did not sleep. On December 26th he was very excited, and persisted in sitting up in bed, but slept well at night. On December 27th he was also excited, and only partially rational. Some diet which had been withheld was supplied to him this day, as he was hungry. The dressings also were loose, and so were changed, and the wound was found adhering throughout, and free from redness or swelling at the edges. On December 28th he had had another very good night, and was perfectly quiet and rational. On December 30th he was sitting up in bed reading and taking an intelligent interest in what went on around him. The stitches were removed on January 1st, 1902, and the wound was found firmly healed throughout. He now said that he "never felt better in his life." No pulsation could be noticed over the gap in the skull, either now or at any time since this operation. The clearness of his recollections, and the quickness and certainty of his perceptions and replies, were quite noteworthy. In every way he appeared in excellent health and spirits, and no fit has occurred since the one noticed the day after operation.

Remarks.

On the day of his last admission this patient looked like one stupefied by alcohol, and his dull, hesitating manner led me to make inquiries about his habits. His wife was with him, and supported his own statement that, as a matter of fact, he did not drink alcohol. I made further inquiries as to whether or no he was out of work, in view of some possible motive other than the necessity of illness, and found that he had a good and regular occupation, which was only interrupted by the fits. It was evident that he was genuinely in need of assistance, as the history shows. But at his previous stay in this hospital I was not so fully persuaded of the genuineness of his case. The history of previous recent fits had then a certain vagueness about it, and the one fit reported

after his admission was insufficiently corroborated for my own satisfaction. The operation then performed was not undertaken on account of the alleged fits, but simply to protect him against injurious consequences, in case the thin protruding scar should give way and cerebral fluid escape in the absence of immediate surgical attention. If it had not been for the cystic distension of the scar, I should not have touched him; and I concluded that Mr. Horsley had refrained from interference for a similar reason—namely, that there seemed no occasion to do so for the convulsions' sake. When, therefore, the man left hospital shortly afterwards, to all appearance perfectly well and free from the repetition of any sort of fit, the impression left upon my mind was that before the operation he had probably exaggerated his own condition and requirements; had even been actuated by indolence, or perhaps malingering. I had not then sufficiently informed myself of the absence of all possible motive on his part, as the pathological evidences, partly second-hand, were both scanty and transitory. This time the condition was more marked, and the analysis of his symptoms and motives placed their genuineness beyond all question for both occasions. The time, in fact, had arrived when interference was really called for.

It is possible that the operation undertaken in 1895 may have unexpectedly served the purpose of removing his susceptibility to the fits, and of postponing for more than six years the necessity for interference so obvious on the last occasion.

As to pulsation,¹ when the skull is opened the brain pulsates at the orifice, unless its own substance or the dura mater be resistant enough to prevent it. For instance, a cerebral tumour which I removed in 1889 showed no pulsation when exposed through a large trephine hole.² The growth was a gumma, adhering inseparably to the dura mater, and firm enough to resist the vascular expansion of the brain at each beat of the heart. But the moment the tumour was removed the exposed brain pulsated, and pulsation has continued to the present time through the unossified hole in the skull, the movement being imparted to the integument (that forms the only covering), to a visible extent, but without raising the parts appreciably from the level of the bone. When the patient coughs, however, the encephalon balloons the skin out, raising it about half an inch from the cranial level.³ In other cases of cranial operation, whether for compound fracture, or in trephining for other purpose, pulsation occurs on removal of portions of the bony case, and continues as long as the coverings remain unossified, even when the bone fragments are replaced. The pulsation gets fainter as ossification proceeds, and usually ceases when the latter is completed.

In the present instance the gold foil appears stiff enough to resist the pulsation that naturally occurs where continuity of the bony case may be interrupted.

The accompanying photographs show the seat of injury as it appeared before my first operation and after the last.

NOTES AND REFERENCE.

¹ Written April 6th on preparing this report for the JOURNAL. ² BRITISH MEDICAL JOURNAL, November 30th, 1889. ³ Strange to say, this patient called upon me the following morning. I had not seen him since December, 1890, but found him in good health, and with clear recollection of past events.

A CASE OF COMPLETE AND TEMPORARY PARALYSIS OF THE LIMBS IN A CHILD,

PROBABLY A CASE OF RECOVERY FROM THE INITIAL STAGE OF
ACUTE ANTERIOR POLIOMYELITIS.

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In a few cases of infantile paralysis the part of the limb which is permanently paralysed is the only part that was affected at the onset of the disease, but as a rule such a correspondence is lacking, the initial loss of power being much greater in extent than the permanent paralysis. Thus at first both legs may be affected, while the final paralysis is limited to a group of muscles in one leg; or permanent atrophy of the shoulder muscles of one arm has been preceded by com-

plete paralysis of both arms, with weakness of some of the neck muscles. Indeed, in some cases where the final paralysis is limited to two or three muscles, the original paralysis was almost universal, affecting all the limbs and many of the neck and trunk muscles.

This want of correspondence in the distribution of motor weakness at different stages of the disease is one of the striking features of infantile paralysis. A consideration of the complete return of motor power in parts that were profoundly paralysed would lead one to assume that in some cases the wave of recovery would spread to every part that was initially affected, yet as a matter of fact this rarely occurs. In nearly every case of acute anterior poliomyelitis some muscles are left permanently, even if only slightly, weak.

As an apparent exception to this rule, the following case is worthy of record:

A girl, aged 9 years, was admitted under my care at the Manchester Royal Infirmary on January 20th this year. Her mother told me that on January 13th the child was "shaky and cold," that on the 15th she was shivering, and her skin was very hot. On the 16th it was noticed that her legs seemed to fail her. On the 17th she was unable to get out of bed; at breakfast time she could use her hands, but a few hours later she was unable to move them. On the 19th she was very feverish, and on the 20th, the day of admission, her temperature was 103° F. On this day I saw her for the first time. She was lying in bed, unable to move any part of her body except the head; this she could raise from the pillow, and move from side to side. She was unable to raise either side of her body from the bed, nor could she move any part of her arms or legs; her limbs were completely and universally paralysed. She had no pain, and no alteration in cutaneous sensibility could be detected. None of the cranial nerves were affected. The knee-jerks were absent, and no wrist-jerk could be obtained. The action of the bladder seemed to be normal.

The possibility of diphtheria being the cause of the paralysis was considered, but there was no history of sore throat, nor were any cases of infectious disease known to exist in the neighbourhood of the child's home. The fauces had a healthy appearance, and the urine was free from albumen.

On the 21st, the day after admission to hospital, the temperature was normal and the child could feebly draw up her legs in bed and could feebly move her arms; she could also very slightly raise her body from the bed. The improvement progressed rapidly, so that on the 27th, a week after admission, the paralysis of the arms had almost entirely disappeared; the legs, too, were much stronger, and when her body was supported the patient could walk a few steps. She was also just able to sit up in bed. A few days later she could walk without assistance, the flexors of the ankles being, however, still weak. She left the hospital on February 9th quite recovered from the paralysis, and the knee-jerks, which were feebly obtained a few days previously, were quite normal.

As to the site of the lesion in this case, the flaccid paralysis and the loss of the deep reflexes point to an affection of the lower neurons for the limbs and trunk. The absence of sensory and of all other symptoms than paralysis indicates that the motor path was alone at fault. Which part of the lower neuron was affected? Was it the nerve cell or its axon? Was the case one of anterior poliomyelitis or one of peripheral neuritis? It is certain that instances of purely motor peripheral neuritis do occur, and in the present case the complete recovery speaks more for a peripheral than a central disorder. Still such great rapidity in the disappearance of symptoms as occurred in this case is probably as rare in neuritis as in poliomyelitis. Then again multiple neuritis in the child, apart from diphtheria, is rare, whereas anterior poliomyelitis is common. In favour of the latter condition, too, may be mentioned the completeness of the palsy.

Just as difficult to settle as the site of the lesion, if not more so, is its nature. Obviously there was a profound, if only a transitory, disturbance of the functions of the parts attacked. The action of some poison seems to be the simplest explanation. Yet such speedy destruction or elimination of the poison as must have occurred in the case related is very remarkable.

Evidence is accumulating that many disorders of the central as well as of the peripheral nervous system are initiated by the presence of a toxin, the product of micro-organisms, which

circulates in the blood and, according to its nature, selects for its attack certain definite parts of the nervous system.

Suggestive in this respect are the characters and the distribution of the early lesions in infantile paralysis, together with the etiology of the disease and its occasional occurrence in large epidemics. Moreover, in a few instances micro-organisms have been found. Thus, Schultze detected the Jäger-Weichselbaum's diplococcus in the cerebro-spinal fluid obtained by lumbar puncture in an early case of infantile paralysis. Chapin, too, in a case which terminated fatally, found diplococci in the anterior horn which presented the characteristic histological changes of acute anterior poliomyelitis. Dercum has also recorded a case of infantile paralysis in which the fluid obtained by lumbar puncture showed diplococci which stained with gentian violet, and also by Gram's method. They resembled, both as regards their structure and their affinity to certain stains, the diplococci of Sternberg.

Experiments on animals have shown that various forms of myelitis can be produced by the injection of bacteria or their toxins into the blood. Thus Marinesco has produced poliomyelitis in animals by injecting micro-organisms into the blood vessels which directly supply the spinal cord. He has also set up changes like those of acute anterior poliomyelitis by the injection of streptococci and of the influenza bacillus within the spinal dura mater. Other investigators, too, have produced in animals spinal lesions resembling those of acute poliomyelitis by the injection of various bacteria; of these may be mentioned the typhoid, influenza, and diphtheria bacilli, also the streptococci of erysipelas, and the bacterium coli.

Hence, it is not unlikely that infantile paralysis may be set up by the action of a toxin which is produced by micro-organisms, and which circulates in the blood. The infective agent may find its way into the system through the gastrointestinal tract, or possibly by the nasal mucous membrane, or even, as suggested by Schultze, through the middle ear.

In the case which I have related no facts were obtained which threw any light either on the nature or on the mode of entry of a supposed infectious agent. If we assume that a toxin did attack the lower neurons of the parts paralysed, the rapid disappearance of the paralysis would suggest that the toxin was speedily destroyed or rendered innocuous, or quickly eliminated from the system.

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CASE OF SPORADIC CRETINISM, IN WHICH A RELAPSE OCCURRED OWING TO OMISSION OF THYROID EXTRACT.*

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THE following case emphasizes the fact that relapses may and do occur in cases of sporadic cretinism, if the treatment by thyroid gland substance be omitted, and that it is of absolute importance that cretins should be kept carefully under observation if the results of treatment are not to be spoiled:

History.—A. R., a boy, aged 15 months, was sent up to me at the Sheffield Royal Hospital in February, 1896, by Dr. J. H. Wilson, of Hillsborough. At birth he was not noticeably different from other children, but when a few months old he was found to have a "goitre." This was treated—how I do not know—and it disappeared when he was six months old. The parents date the change in his condition from that time.

State on Examination.—He was continually crying with a harsh cry, moving his head restlessly, and always pulling faces. Skin dry, with fine branny desquamation on the forehead and eyebrows. Circumference of head 18 in.; hair dark and scanty; complexion pasty; anterior fontanelle widely open, posterior slightly; eyelids purple and oedematous; opening of eyes narrow; pupils large; slight internal strabismus; nose broad, depressed bridge; nostrils wide, snuffles; mouth large, two lower incisors depressed appearing; arms flabby, thick; hands very broad and thick, skin loose and flabby and in folds; abdomen not very prominent; large umbilical hernia; scrotum pendulous; legs very thick, skin loose, and thrown into very loose folds; feet thick and broad; height 26½ in.; weight 18 lb. 10 oz.; temperature 97°.

* Read at the meeting of the Yorkshire Branch of the British Medical Association.