friends and attendants that one or more of the limbs are limp and powerless. It is also interesting to find that a child may suffer from a similar attack and get well again without any paralysis taking place; then a few weeks later perhaps he is seized again with similar symptoms, which this time are followed by paralysis. In some cases reported the primary attack has been slight or indeed absent; in cases in which the lesion has not been extensive it is easy to understand the symptoms may not be marked and be readily forgotten or overlooked by the friends. With regard to the actual lesion, it is perfectly clear that there is an acute inflammatory lesion involving the anterior horns and more or less of the grey matter of the cord, more especially the lumbar and cervical enlargements. There is an inflammatory engorgement of the arteries which pass from the anterior spinal arteries of the cord to the anterior horns, accompanied by exudation of liquor sanguinis into the nerve substance, and apparently also in some instances by actual rupture of the capillaries and consequent minute hæmorrhages. The evidence points in the more severe cases at least to an engorgement of the cord, the most vascular parts, namely, the anterior horns being the spots where the greatest damage occurs; part of the damage done to the nervous mechanism is recoverable as time goes on by absorption of the effused inflammatory material, and part of the damage is irretrievable, on account of the breach made, so to speak, in the grey matter of the cord. That the anterior horns which contain such important groups of nerve cells, and consequently are exceedingly vascular, should bear the brunt of the damage is not surprising, still there is evidence to show that the rest of the cord does not entirely escape, for in one of my own cases there was some loss of sensation during life, and distinct evidence of slight inflammatory lesion of the vessels supplying the rest of the cord. It is important to bear in mind that evidences of inflammatory action in the shape of gorged vessels surrounded by leucocytes may be found six weeks or two months after the onset of the paralysis, this would point to the inflammatory action being subacute, the inflammatory stage lasting long after the symptoms of the primary attack have passed away. This must be remembered in any line of treatment we adopt.

Peripheral Paralysis.—Of these it is not necessary for me to speak at any length; as far as we know, they do not play an important part in the paralyses of early life. Injury to the brachial cords is apt to take place during labour by traction on the arm by an unskilled attendant, and a troublesome paralysis result. In congenital syphilis we occasionally see a peripheral paralysis due to some nerve lesion, though it need not be said we must be careful not to confuse the inability to use a limb in consequence of epiphysitis or periostitis with paralysis. In rickets periosteal tenderness and muscular weakness give rise to paresis, though even here we are still uncertain if the muscular weakness may not deserve the name of a paresis due to a nerve lesion or the effects of a poison. Of diphtheritic paralysis, and similar paralyses due to the effect of toxic matters in the blood, it is unnecessary for me to speak.

TWO CASES OF CONGENITAL AND INFANTILE CEREBRAL PALSY.¹

By E. MANSEL SYMPSON, M.B.CANTAB.,
Lincoln.

THE patient, H. E., aged 19, was the third child of his mother, who states that she had a long and hard labour with him, but that no instruments were used. Ever since birth he has been palsied on his right side, the right leg getting fairly strong and useful, while the right arm has always been a good deal weaker than the left. About three years ago he had a sharp attack of rheumatic fever, with probably some dilatation of the heart, for there was then a mitral murmur, which I am glad to say has now vanished. For several years he has been subject to sick headaches, scarcely worthy, perhaps, to be dignified with the title of migraine, though the attacks were paroxysmal, of very severe headache, with vomiting, cold extremities, but with no affection of sight. Also about three years ago, while bathing directly after breakfast, he was suddenly seized with a fit of struggling, vomited freely, and was unconscious for some time. The next

fit took place eighteen months afterwards, the last about four months ago; there have been about five fits altogether. There is no aura or warning; he is unconscious for from three to five minutes, in apparently clonic general spasms, eyes turned upwards and rolling, face purple, no involuntary evacuations, petechiæ on face, and on several occasions he has injured himself severely.

Present Condition.—The head is obviously larger on the right side; from the vertex to the top of the left ear is more than half an inch smaller than from the vertex to the right ear; eye movements normal; heart slightly hypertrophied.

Right forearm an inch and a quarter smaller in circumference and half an inch shorter than the left. Its muscles react well to faradism. The chief wasting or want of development seem to have taken place in those arising from the inner condyle of the humerus. The interossei are very poor. The forearm is generally somewhat flexed on the elbow, the hand flexed on the wrist, the fingers overextended; supinator, biceps, and triceps reflexes are easily obtainable; the hand and arm are colder than on the left side, while the skin of the hand is markedly smooth and almost glossy; sensation seems unimpaired; grasp, though considerably weaker than that of the left hand, is fair, but he can do nothing demanding delicate touch or manœuvring.

Right leg is also half an inch shorter and three-quarters of an inch smaller than the left; knee-jerk and ankle clonus are evident; he walks with a slight limp.

The treatment latterly has been, of course, only directed to these epileptoid fits and the attacks of sick headache. I began with potassium bromide, 20 grains twice a day, afterwards small doses of tinctura belladonnæ and tinctura digitalis; finally, he is now taking potassium bromide and tinctura digitalis. For four months he has had no fit of any kind, and he has improved in every way, probably from their absence. For the sick headache I have found nothing so satisfactory in this, as in other cases, as the effervescent citrate of caffeine.

Three or four years ago he saw Sir James Paget on account of hemiplegia. Massage and galvanism were recommended, with the result that, as I have stated, the muscles are in very fair condition. The diagnosis is, I think, clear; it is a case of congenital cerebral spastic palsy, the birth palsy of Gowers, with epileptoid convulsions coming on rather late in puberty. I called it congenital because apparently it dates from birth, and cerebral because the difference of the two sides of the head, the spastic hemiplegia, the electric reaction of the muscles, and the later developed epileptoid convulsions leave me no room to doubt that the brain is responsible for his condition. I also, for the same reasons, feel pretty sure that the locality of the lesion is in the cortex cerebri, covering the leg and arm centres on the left side, and that lesion now is almost certainly a localised atrophy, the cause of that atrophy being, as Dr. Gowers says on this subject, meningeal hæmorrhage, which occurred probably during parturition. The association of those migraine-like attacks with epileptoid fits is suggestive of the near alliance mentioned by Dr. Hilton Fagge and others between migraine and epilepsy proper.

The second case I am bringing before you is that of a girl, aged 13, suffering from infantile cerebral palsy, and I am much indebted to the kindness of Mr. A. L. Peacock, of Lincoln, for an opportunity of seeing her and making notes of her condition. She seems to have been a fairly healthy child till she was 3½ years old; then, in close connection with an attack of measles—the mother's memory was rather shaky as to the exact relation in time of the two—she was seized with aphasia and right hemiplegia. Since then her speech has never been quite perfect.

Present Condition.—She is an intelligent child; head well shaped and equal on both sides; eye movements normal; sight excellent. Her right arm and leg are much weaker, and the right leg is half an inch less in circumference than on the left side. The right foot is always colder than the left. No deep reflexes are to be gained in right arm or leg, though the left knee-jerk is very obvious. There is no spastic condition of muscles of the palsied limbs. No affection of sensation was evident; micturition and defæcation were quite natural.

In this case again I think that the hemiplegia, with no great atrophy or ill development of muscles, accompanied by aphasia at its first onset, points indisputably to the lesion being cerebral. The association with measles, as occurred in seven of Dr. Gowers's, four of Dr. Abercrombie's, and four also of Dr. Osler's, forms a starting point for several diverse explanations. It is almost

¹ Read in the Section of Diseases of Children at the Annual Meeting of the British Medical Association held in Leeds.

exactly on all fours with Case No. xcvi in Dr. Osler's admirable little work on *The Cerebral Palsies of Children*. Polioencephalitis, cortical venous thrombosis, embolism, meningeal hæmorrhage, capillary hæmorrhage, are all rivals in the field. Elsewhere I have given a few reasons for thinking that all spastic palsies in children are the result of a cortical lesion produced by meningeal hæmorrhage; and I find, on the authority of Nothnagel² that, in thrombosis of cerebral veins, it is not uncommon for rupture of the vessels to occur, causing hæmorrhage into the meninges.

In infantile cerebral palsy the rule is that there be some degree of spasm of the palsied muscles; here, as in three cases of Dr. Osler's, there was none. Whether this depends on a different locality of the lesion, or upon the more perfect recovery of the once damaged brain cells, I do not know. In this case, as in No. iv of mine,³ the right great toe was kept turned upwards and backwards. According to D'Espine, this is due to the unbalanced action of the extensor proprius, the plantar arch falling through the paralysis of the peroneus longus; and I have since noticed the same in apparently a genuine case of Erb's primary spastic paraplegia in both toes. In both these cases, and in No. iv above mentioned, the affected limbs were colder than the sound ones, and the same is noted by Dr. Osler in his cases LXVII and LXX. This is not nearly so marked as in acute anterior poliomyelitis. It simply follows, I suppose, from their being old cases of hemiplegia, as Dr. E. L. Fox points out, that the palsied limb appears colder than the other. Whether this be due to chronic irritation of the cortical centres (of Eulenberg and Landois), which, according to these observers, influence the vascular state of the limbs, to the palsied muscles needing less blood, or to the muscles manufacturing a less amount of heat, is beyond my present purpose to discuss.

HEMIPLEGIA ACCOMPANIED BY LOSS OF SPEECH IN CHILDREN.¹

By THOMAS OLIVER, M.A., M.D., M.R.C.P.,

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APHASIA is always an attractive subject, particularly in children. The diseased condition which gives rise to it occurs in them at a time when development is incomplete; when organisation of certain parts of the brain is taking place, and upon whose healthy functional activity depends the power the individual has of giving expression to intelligent thought.

On August 6th, 1888, I was asked to see in consultation O. H., a pale-faced delicate child of 2½ years of age. She was a twin, and the more delicate of the two, owing to her having suffered from recurrent attacks of cold. Her father and mother are healthy, and there is no history of tubercular disease in the family.

The child had been apparently quite well until three weeks before I saw her. She had caught cold; this was followed by bronchitis. The bronchitis had passed away, and she had made, it was thought, a satisfactory recovery. She had got again into the habit of walking about the house when, one day—five or six before I saw her—she was found lying helpless on the scullery floor. Lifted by her mother, and placed upon her feet, she at once fell, and it was then only too apparent that this complete inability to stand was due to paralysis of the right side of the body. She had lost, too, her power of speaking. These symptoms continued. Within the next few days tremor of the right hand developed, which gradually passed into strong convulsive movements, affecting both sides of the face and the body generally. During the week previous to my seeing her she had not spoken; she simply expressed her wishes by pointing with her left hand. The right arm by this time was passing into a state of extreme rigidity with spasm; it had been carried back behind the trunk, was directed outwards, whilst the hand was twisted round and bent upon the wrist. The right leg was held out in a state of extension and rigidity; the big toe all the while bent and pointing upwards. Though the arm and leg were in a condition of rigidity and spasm—almost permanent—yet from time to time the spasms relaxed. Particularly was this the case so far as the fingers and the knee and ankle on the right side were concerned. From a con-

dition of relaxation the limbs would at once pass suddenly into a state of spasm, the act being evidently accompanied by a great amount of pain. There was complete absence of the knee-jerk on either side; the plantar reflex was slightly exaggerated. The pupils were equal. Temperature was normal; it had never risen. Vomiting had been present prior to the seizure, but none since. Headache, if ever present, had never been complained of. Pulse 106. Tongue moist; clean. All over the chest numerous small rhonchi were heard. Heart was healthy. We diagnosed meningitis, affecting principally the motor areas of the left side of brain. On August 31st the child died, never having regained speech or the use of the right arm and leg.

At the *post-mortem* examination considerable difficulty was experienced in removing the calvarium, owing to strong adhesions existing between it and the dura mater. This membrane was found to be thickened, and where it was adherent dense bands of fibrous tissue were seen radiating outwards from either border of the longitudinal sinus. The walls of this sinus were thickened, and a large, firm, dark clot occupied its interior. The vertex of the brain was bright and glistening. A clear gelatinous fluid occupied the subarachnoid space, particularly in the upper part of the so-called motor areas on the right side of the brain and over the whole of the motor area on the left side, where it was most marked over the third anterior inferior frontal convolution. On careful examination it was noticed that this effusion pressed chiefly upon the lower half of the left ascending frontal convolution and the whole of the third anterior inferior frontal convolution. The convolutions, taken as a whole, were softened. At the base of the brain the vessels were healthy, as were also those traced deeply into the fissure of Sylvius. The white substance of the right half of the cerebrum was found on section to be quite firm and healthy to the touch; that on the left was softer and showed many more and much larger puncta hæmorrhagica. The substance of the convolutions of the left motor areas was extremely soft. About 3 or 4 drachms of bloody serum occupied the left lateral ventricle. The left internal capsule was extremely soft compared to the right. The rest of the brain was healthy.

A vertical meningitis, followed by effusion, pressing upon and irritating chiefly the left motor areas and Broca's lobe is, in a word, the pathological condition that existed, the doubtful cause of which was an injury received by the child some months previously to her death.

That the meningitis stood in a causal relationship to the loss of speech in this case there cannot be the least doubt, and it is a good illustration of "irritative inhibition" of the speech centre, the function of which was just as completely destroyed as that of the motor areas, evidence of which was seen in the paralysis of the right arm and leg. Had the child's life been spared, the right arm and leg would have remained paralysed, and their growth would have been retarded, and the lesion would have been regarded as a polioencephalitis. Speech might have been regained through development of the right side of the brain, and the assumption by it of the word-making function. This case is interesting as showing at what an early age "duality of brain" was completely established, the association of right-handedness with left cerebration, and the developmental interaction of these two as regards the function of speech.

2. *Left-sided Convulsions, followed by Hemiplegia and Aphasia, in a Child aged 5½ Years.*—This little patient I saw in consultation with Dr. Russell, of Heaton, to whom I am indebted for the notes of the case. He first saw the child on August 9th. Until the middle of the preceding month her health had been good; she now began to lose her appetite, and became extremely irritable in her temper, was feverish at night and her memory and speech became gradually affected. She was no longer able to walk, staggered much and fell frequently. There was a history given of a fall down stairs a year previously, and of the patient having been rendered unconscious for one day, and at the present time the cicatrix of this bruise is seen over the upper part of the left frontal bone.

On the day previous to that on which Dr. Russell saw her she was very noisy; during the night she had slept badly and was delirious. In the morning severe convulsions set in. The patient, who was a well developed child, was found convulsed, generally and in a severe manner, but the convulsions were much more severe on the left than right side of the body. There was strabismus, also twitching of the muscles of the face, dilated and unequal pupils, the left being the smaller; neither of them responded to light; pulse 52, and irregular; heart healthy; a few mucous rales

² Ziemssen, xii, 13.

³ *Practitioner*, February, 1889.

¹ Read in the Section of Diseases of Children at the Annual Meeting of the British Medical Association held in Leeds.