Dr. Mason Warren has also (*Boston Medical and Surgical Journal*, 1866) written upon the treatment of cysts of the jaws, and strongly recommends a milder and even more conservative practice than that followed by Mr. Butcher, which he thus summarises: "The treatment consisted in the puncture of the sac within the mouth, evacuating its contents, and at the same time obliterating its cavity by crushing in its walls; and lastly, in keeping up, by injections, etc., a sufficient degree of irritation to favour the deposition of new bone."

I have now treated a considerable number of simple and multilocular cysts by Mr. Butcher's method, and, as has been noted, with recurrence in at least two of the latter. Mr. Butcher does not appear to have met with further trouble in his cases, and this may depend upon his "carrying out the gouging fearlessly and far wide of the disease." I should in future be guided by the age of the patient and the amount of solid material found in the cysts. In young persons with cysts having fluid contents and little growth in the bone, I should be still inclined to adopt palliative measures and to gouge very freely, carefully watching the case with a view to a more radical proceeding, should further development take place. In cases of much solid deposit in connection with multilocular cysts, and still more in cases of solid tumour with one or more large cysts, there should, I think, be no doubt as to the removal of one-half or more of the lower jaw, or of any portion of the upper jaw involved.

CLINICAL LECTURE ON HEMIPLEGIA IN CHILDREN.

Delivered at the Hospital for Sick Children, Great Ormond Street, June 1st, 1897.

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FORMERLY all forms of paralysis in children were grouped under the head of infantile or essential paralysis, and were believed to be of the same nature. Dr. Goodhart and Professor Henoch, of modern writers, are those who have most clearly recognised that a very considerable number of cases of paralysis in children own a cerebral cause; the latter writer (*Diseases of Children*, American translation, p. 100), indeed, goes so far as to say that this paralysis is much more frequently due to cerebral than to spinal disease. I suspect that this statement is nearer the truth than many might be disposed at first to believe. The causes of hemiplegia in the adult are hæmorrhage, arterial obstruction, whether from embolism or thrombosis, and tumour, and all these causes may be found in operation in childhood. Cerebral hæmorrhage in infants must be exceedingly rare, and is very uncommon in children; almost the only known causes are purpura hæmorrhagica, the hæmorrhagic diathesis, or aneurysm of one of the cerebral arteries as a result of pre-existing heart-disease, and in a very few instances a highly vascular sarcoma has given rise to a large extravasation of blood into the brain. It is conceivable, too, that massive hæmorrhage might take place in whooping-cough, but I do not know of any instance of its occurrence. Meningeal and capillary or punctiform hæmorrhages will require separate consideration.

In regard to arterial obstruction, inasmuch as heart-disease is common in early life, it is not surprising that embolism of one of the cerebral arteries should occasionally be met with; indeed, as Dr. Goodhart (*Diseases of Children*, 2nd edition, p. 471) remarks, it is a little strange that embolism from heart-disease is not more common. It is unnecessary to say more about this variety, as it presents no distinctive characters apart from those seen in adults. Thrombosis of an artery must be the result of previous disease of the vessel, and, just as in adults, syphilis probably causes hemiplegia in this way; the point will be referred to again.

Tumours are not uncommon in children, and there is no reason why they should not occur sometimes in the motor area of the cortex, or somewhere in the motor path between this and the base; they are more commonly found, however, in the pons or crus. The same symptoms are produced in the child as in the adult, and no further reference need be made to them.

It is just worth mentioning here, as the point will not be again referred to, that cases of tubercular meningitis are not infrequently first betrayed by one-sided paralysis.

We have now run through the ordinary causes of hemiplegia in the adult, but I have only accounted for a very few of the cases, some fifty in number, of which I have notes. The remaining cases may be conveniently divided into two groups, those which succeeded one of the acute infectious diseases, and a much larger group where no such cause could be assigned.

Measles, scarlet fever, whooping-cough, and diphtheria may all be followed by hemiplegia. Four times I have known it succeed measles; in three cases the children were under 3 years of age; in the fourth the child was 5; the onset took place on the ninth day in one of the cases, in the others at about the end of a fortnight; but inasmuch as the patients often do not come under observation until some years after the onset, we must not expect too great accuracy as to dates. I have only seen one case of scarlet fever origin, and although there was no evidence of heart-disease then, one would have to bear in mind the fact that endocarditis is not uncommon during and after scarlet fever, and that it would afford a ready explanation of the paralysis, by affording facilities for the formation of an embolus. Of whooping-cough as a cause, I have also notes of only one case: a little boy just over 2 years old, was brought with well-marked hemiplegia, dating from an attack of convulsions of thirty-six hours' duration during whooping-cough about a year previously.

Diphtheria, if I may judge by my own observation alone, is a more common cause than any of the preceding, but it is often very difficult to get the necessary amount of evidence respecting the primary ailment. Thus, in one case, a little girl had been under my care for some months with hemiplegia, and all I could learn from the mother was that it had been preceded by "low fever," when one day the mother brought me another child, and told me he had never been well since he had the diphtheria, just before the child whom I was treating had been taken with her so-called "low fever." The following case is worth quoting in some detail, as I had the opportunity of watching the whole course of the affection to a fatal issue, and of making a *post-mortem* examination. G. W. (*Medical Times and Gazette*, September 23rd, 1882), aged 6, was under treatment for diphtheria, when on the fifteenth day from the onset, in the afternoon, he was seized with a convulsion which was general, and after it his mouth was drawn to the right, and he complained of right frontal headache. On the following morning he was again convulsed for twenty minutes, but on the right side only; when I saw him a few minutes afterwards there was complete left hemiplegia. He died eleven days later, no change in the paralysis having taken place. The right middle cerebral artery was found plugged by an adherent thrombus. Infarctions were found in the spleen and kidneys. There was no heart-disease, and no source for an embolus was discovered.

Of the remaining cases there was evidence of congenital syphilis in four, and there was good reason to attribute the paralysis to this cause. In three the paralysis dated from quite early infancy, having as usual been ushered in by fits. In the fourth case I made a *post-mortem* examination, and may give you the facts briefly. T. H. (*St. Bartholomew's Hospital Reports*, xvi, p. 35) came under observation at the age of 10 years 9 months. There was conclusive evidence of syphilis. At the age of 6, whilst walking in some fields, she had suddenly fallen down, and was then found to be paralysed on the right side, and that side had been weak ever since. On *post-mortem* examination a small old adherent thrombus was found in the middle of the longitudinal sinus. There was atrophy of the left hemisphere, the cortex and central parts both sharing in the atrophy; the meninges were thickened and adherent, especially over the left side and the convolutions (chiefly the ascending frontal and frontal convolutions) were sclerosed. The middle cerebral arteries were diseased.

Syphilis may have had a share in some of the other cases also, as two of the children in whom the paralysis followed measles were certainly syphilitic, and consequently may have had disease of their vessels, whilst it was a possible factor in other cases to which I shall shortly allude.

The remaining cases would seem to fall into three groups, namely, the traumatic, the congenital, and those which are neither traumatic nor congenital, and yet own none of the causes which we have been discussing. I have only seen one case where the paralysis could with reasonable probability be set down to a traumatic cause, though in some cases there can be no doubt that injury done during labour, especially when forceps have been used, has been the cause. In the case I refer to, a child at the age of 9 months, fell on to his head, causing a bruise over the right parietal bone; he was very ill for the next six months, and four months after the accident it was noticed that the left side was paralysed; when I saw him two years later there was marked left hemiplegia with rigidity.

I have seen only one case in which the paralysis was actually

noticed directly after birth, and in that case there was no evidence of injury at the time of birth; but in three other cases the paralysis was observed so soon after birth that it is a fair assumption to suppose that it was due to some disease or abnormal condition which must have existed at the time of birth. One of these is worth mentioning, as it appears to be a case in which the leg only is paralysed. A boy who was brought to me when about 5 years old, had been liable to fits since he was 6 weeks old. It had been noticed from quite early times that he did not use his left leg, in fact the mother could not say whether he had ever been able to use it; no paralysis of his arm had been observed. There was no obvious paralysis of his left arm, but his gait was quite hemiplegic in character, and the left leg was stiff at both knee and ankle-joints, and there was a very exaggerated knee-jerk on this side. It is a curious fact that in these three cases in which the hemiplegia had shown itself at the earliest possible age, there was strong reason to suspect syphilis in all, but as the evidence was not absolutely conclusive I have refrained from classifying them with the undoubtedly syphilitic cases.

The remaining cases only permit of one subdivision, namely, into those which occurred before the age of 2 years, and those in which the paralysis did not show itself until after that date. Of the former class I have notes of fourteen cases, and since these are essentially the cases of infantile cerebral paralysis, it will be worth while to spend a little time over them. The following cases illustrate the mode of onset. Edith A., brought at the age of 2 years, with well-marked left hemiplegia; when 10 months old she had a fit, lasting about a quarter of an hour; both sides were convulsed. Three weeks later, when apparently well, she was again seized with convulsions, which lasted a whole week; the left side was almost exclusively affected, and from that time she was paralysed on this side. Another case was that of Winifred H., aged 2, whom I first saw in January of this year; I learnt that six weeks previously she had been taken with a fit which lasted seven hours; for the first hour it was general, but after that was confined to the left arm and leg, and left side of the face. She had no subsequent fit, but next day the left arm and leg kept on being slowly drawn up and down; after that for a week she could not move her arm at all, and the leg not much; since then she has been steadily improving, but she throws the left foot very high when she walks. When I first saw her there was very distinct loss of power on the left side, but she has been gaining strength on that side ever since, and now walks really well. I once saw a case in which a child appeared to have a second attack of hemiplegia. The first occurred when she was 10 months old, and she lost the use of the left arm and leg; she improved much, but did not get quite well. When about 3 years old, as the mother said, she had a fresh stroke, that is, she was unconscious for several hours, and again lost the use of her left side; she remained hemiplegic after this attack, and epileptic.

The paralysis is not often permanently complete, and rigidity is seldom wholly absent; sometimes the rigidity is much increased, or only recognisable by manipulation of the paralysed limbs. Sometimes the paralysis is very slight, but the affected limbs are the seat of movements, a condition known as athetosis when the movements are slow, or post-hemiplegic hemichorea, or mobile spasm (Gowers) when the movements are more rapid.

Of the fourteen cases ten were girls, a much larger proportion than can be the consequence of mere accident; and, moreover, it is in corroboration of the fact already noticed by Dr. Gowers (*Epilepsy*, p. 127), that in children under 5, hemiplegia is twice as common in girls as in boys. In several of the cases the intellect was seriously below par, some of the children being absolutely idiotic; only a very few presented no evidence of mental deficiency. But it must be remembered that by reason of their paralysis these children had been unable to mix with others, and their education had almost always been neglected, so that their backwardness may in part have been due to want of opportunity. The fact, however, remains, that the intellect is much more liable to be affected in the hemiplegia of children than in that of adults.

In all cases improvement was much more marked in the leg than in the arm. Arrest of development of the paralysed limbs was noticed in some of the cases, the arrest falling on the bones chiefly. No obvious muscular wasting was found, and there was a good layer of subcutaneous fat even on the paralysed side. In a large majority no evidence of neurotic tendency could be elicited, but in one of the cases it was stated that all the children in the family (and they were many) had cut their teeth with fits; in two instances there was a suspicion of syphilis. Only four cases fall into my last category, that is, children in whom the paralysis was not developed until after 2 years of age. In two the paralysis succeeded epilepsy, the third was remarkable not only because the arm was much more affected than

the leg, but because aphasia was the chief symptom throughout, and persisted after the paralysis had passed off. In the fourth the question of hysteria cannot be wholly excluded. The patient is a little girl, aged 8½ years, who was frightened three years ago, and has had weakness of the left arm ever since, and movements have been gradually coming on. She is said to have had hysterical fits, though I cannot say exactly what is meant by that expression. She has choreiform movements of the left hand, neither slow nor rapid, and is only able to use that hand clumsily and feebly; she drags the left leg a very little.

To sum up briefly the symptoms of this form of paralysis in children, we may say that it usually attacks children under 1 year of age, commencing with a prolonged convulsion, lasting for several hours, and sometimes even for some days, the convulsion being frequently limited to the side subsequently found to be paralysed, though often at first general. When the fit passes off the child is found to be paralysed on one side, and may have lost his speech, or his reason, or both. Sometimes the child comes out of the first fit unharmed, but is left paralysed by a second or subsequent seizure. A little improvement, especially in the leg, may be looked for early, but complete recovery is not common; arrest of growth of the paralysed limbs, a certain amount of rigidity, and clumsy, irregular movements are frequently met with. Sensation in the paralysed limbs is rarely affected. The face is occasionally paralysed on the same side as the limbs. Unilateral atrophy of the skull on the side opposite to the paralysis is sometimes very marked. The paralysis may come on after one of the acute infectious diseases, or as a result of syphilis, but in a considerable number of cases no cause can be found.

If we now turn to inquire what is the pathological condition underlying these cases, we find that, after a considerable lapse of time at any rate, there is a singular uniformity in the appearances; atrophy and sclerosis of the convolutions in the motor area of the hemisphere opposite the paralysed side is the almost invariable result, with thickening and opacity of the meninges, and changes in the vessels. The opportunity for making an examination in the early stages is so exceptional that great doubt has arisen as to what is the actual condition of things at first, and there is much diversity of opinion, even amongst those who have had the largest experience. For instance, according to one authority, an acute inflammation of the multipolar cells in the cortical grey matter is the initial change, a lesion exactly analogous to that in the spinal cord in the ordinary infantile paralysis. According to another, capillary hæmorrhage into the brain would explain all the cases, while another finds that hæmorrhage into the meninges and consecutive meningo-encephalitis is the first change. Another regards thrombosis of the veins and sinuses as the primary lesion, whilst others express their belief in embolism or thrombosis of the arteries. These diverse and diverging views are held by men of the greatest eminence, and as they cannot all be right—and it seems impossible that they should all be wrong—the only legitimate conclusion is that several different causes may terminate in the same result.

Let us now take these various theories separately; and, first, with regard to the polio-encephalitis. This is the view taken by Professor Strümpell (*Deutsch. Med. Woch.*, 1884, No. xlv); but it appears to be based on theory rather than on actual observation. He says that in old necropsies one constantly finds scars in the region of the motor centres of the brain which cannot be confounded with congenital atrophy, and which certainly result from acute inflammation of the grey substance, and he holds that clinical observation permits of a parallel being drawn between the cerebral and the spinal forms. A recent writer has tabulated 160 cases collected from various sources, and in no single instance was inflammation found in the grey substance as an independent lesion. The cause is a possible one, but we must have some further proof of it than has yet been adduced.

We may pass on, then, to hæmorrhage. I have already pointed out how exceptionally rare gross hæmorrhage must be; but capillary hæmorrhage is quite another thing, and would account very well for the atrophic and sclerotic changes ultimately found. Dr. Eustace Smith (*Disease in Children*, p. 328) is the chief advocate of this condition. The hæmorrhage, he says, generally occurs in minute scattered points. The first symptom is an attack of convulsions, from which the child will recover, with more or less of paralysis. If repeated convulsions set in, and continue with intervals of stupor, and especially if any contractions and rigidity are noticed in the hands and feet, the temperature remaining low, we should suspect hæmorrhage. He is conversant with instances of meningeal hæmorrhage, but says this is seldom a cause of paralysis. Professor Hensch (*Op. cit.*, p. 102) says that small capillary hæmorrhages often occur as the result of tubercle of the brain, tubercular meningitis, or throm-

bosis of the sinuses; and Dr. Barlow (BRITISH MEDICAL JOURNAL, Path. Soc. Rep., p. 1098) has lately described how small hæmorrhages so caused may coalesce to form larger extravasations of blood.

Dr. Goodhart (*Op. cit.*, p. 470) is the chief exponent of the meningeal theory. He holds that convulsions are an adequate cause of extensive cortical lesion by producing, first, congestion of the brain, and then meningeal hæmorrhage; or they may initiate chronic changes in the meninges which will thicken them, compressing and causing atrophy of the brain, or a large cyst. He admits all the other causes that have been mentioned as likely, but is very doubtful about Strümpell's hypothesis. It appears to me that the chief objection to these theories of hæmorrhage is that there ought to be no special tendency for them to affect one part of the brain more than another; but I am not aware that the sclerotic changes are found to any great extent elsewhere in the brain than in the motor area. The sclerosis is not necessarily limited to the motor area; but when not so limited, it is generally found in adjoining convolutions, as if it had spread there, and is not scattered about as might fairly be expected on the hæmorrhage hypothesis.

The next theory—that of thrombosis of the veins and sinuses—is supported by Dr. Gowers (*Diagnosis of Diseases of the Brain*, p. 194). This occurs, according to him, in children suffering from debility, exhausting diseases, especially acute specific diseases, or from blows on the head, now and then in very young children without any exciting cause, and it is frequently seen during hot seasons. Thrombosis in arteries, he says, is a very rare lesion in childhood, far more rare than combined thrombosis in sinuses and veins. The initial symptoms closely resemble those that have been observed when a cortical vein has been plugged in adults. After death thrombosis is found in the longitudinal sinus and in the veins leading into it. The circulation is slow, and the conditions are favourable to coagulation.

No doubt Dr. Gowers is right in saying that, if the vessels are healthy, thrombosis will be more likely to take place in the veins than in the arteries; but the same objection cannot be applied to embolism, which, in my belief, is the chief cause of paralysis in children. Dr. Gowers contends that, if embolism were the cause, we ought to find a cavity, and not shrinking and induration; but surely it is a rare result in embolism for the part shut off to become completely liquefied, and it is well known that very often the obstruction is not permanent, and the clot gets washed on, though not until irreparable damage has been done.

Dr. Ross (*Diseases of the Nervous System*, 2nd edition, vol. ii, p. 480), a great authority on diseases of the nervous system, admits softening as one of the precursors of sclerosis, and Dr. Goodhart (*Op. cit.*, p. 471) accepts embolism as the probable cause in the cases coming on after the exanthemata. In this I entirely agree with him, for you will recollect that in the case of G. W., already quoted, the paralysis was proved to have been due to embolism of the middle cerebral artery, and I am quite unable to see anything in the symptoms in the other cases of this group in the least degree incompatible with the theory of embolism. Indeed, the history of the cases is much more in accord with this mode of onset than any other; for in all of them it is a fair assumption to make that the blood was impoverished in quality, and therefore more readily coagulable, and that the heart's action was impaired; and the formation of a clot in its cavities rendered possible; and that this can take place we must assume to be proved by the case of the child just referred to, as there was no valvular disease or other obvious source of embolism, and yet there were infarctions in the spleen and kidneys; a sufficient proof of the nature of the obstruction to the middle cerebral artery. We may take it then that, in all probability, embolism of one of the middle cerebral arteries is the cause of the paralysis when it supervenes during convalescence from one of the acute infectious disorders. I believe that the syphilitic cases, too, may be explained by arterial obstruction, only in them the cause is more likely to be thrombosis than embolism. In the case of T. H., where I made a *post-mortem* examination some four years after the onset of the paralysis, you will remember that besides an extensive cortical sclerosis there was found plugging of some of the cortical veins in the motor area, and extensive disease of the cerebral arteries. The case might be regarded as supporting the view held by Dr. Gowers; but I would rather take it that arterial disease was the first change, and that this was followed by a plugging of the affected vessel, the lumen becoming restored after no great interval. It is now well known that changes in the walls of the blood-vessels do occur in cases of congenital syphilis. Ten years ago Dr. Barlow (*Path. Soc. Trans.*, xxviii) demonstrated this fact at the Pathological Society, and showed that even in very young infants the vessels of the brain may become greatly thickened, and their lumen narrowed, or even obliterated, by

the growth of a nucleated spindle-celled tissue in the intima between the intima and the fenestrated membrane.

I believe that arterial obstruction will hold good as the cause in the great majority of the other cases also, and my chief reason for this belief is the fact that the mode of onset in the one class is precisely the same as in the other; the child is suddenly seized with convulsions, often more or less limited to one side, and when the fit passes off he is found to be paralysed on the side which has been most convulsed. If we admit that this points to embolism when there is a history of preceding infectious disease, why should we not accept the same explanation when there has been no such precursor?

There is another argument in favour of this view which I have already hinted at, and that is the fact that we do not find similar lesions—namely, atrophy and sclerosis—limited to other parts of the brain. Embolism, as we all know, rarely affects other vessels than the middle cerebrals, and therefore the localisation is the more intelligible on the embolic hypothesis.

I am not unmindful of the fact that, in some form or another, hæmorrhage is sometimes the cause, as crystals of hæmatoidin have been found in the atrophied area of the brain; and in the traumatic cases I should be inclined to think that a meningo-encephalitis, often consecutive to meningeal hæmorrhage, was the cause; whilst this, or capillary hæmorrhage in the motor area, would be an efficient cause, and might conceivably happen in such an affection as whooping-cough, where all the blood-vessels of the head must be subjected to severe strain. I cannot help, however, noting once more that I do not recall any instance which would prove that such hæmorrhages were found, or that meningo-encephalitis was set up, in other parts of the brain than the motor area in these cases.

I need say very little about the congenital cases; the pathological conditions found are often much more extreme than in the acquired forms; complete absence of portions of the brain or a large serous cyst are amongst the most usual forms, and in these there is often a considerable increase in the thickness of the skull in the corresponding region. These changes may be due to injuries received during delivery, followed by hæmorrhage into the meninges, or to intra-uterine injury with a similar result leading to arrested development. There can seldom be any difficulty about the diagnosis. Infantile spinal paralysis is very rarely confined to one arm and leg on the same side; and, if it were, the affected limbs would be flaccid, wasted, and cold. Flaccidity and wasting are not met with in the cerebral form; on the contrary, the general nutrition of the limb always remains good, and there is always more or less tendency to rigidity; sensation is rarely affected, and the electrical reactions are unaltered. Disturbances of speech or intellect are common, and the growth of the affected limbs is often impaired; in cases that date from early infancy, the skull on the side opposite the paralysis may show some atrophy, and the circumference on this side of the head may be smaller than on the other.

The prognosis as regards life is good; children rarely die during the initial convulsion, or, if they do, it is before the nature of the affection has been recognised. The bodily health is speedily restored and maintained, and when the intellect has not been affected the child may grow up to be a useful member of society; the leg usually shows some sign of improvement early, and may recover, the arm is much less likely perfectly to regain its strength; when choroidal movements or athetosis have once developed, as a rule, they persist.

During the fit we might put the child into a bath, using a warm one if the temperature were not raised, or if any fever were present a cool bath proportionate to the degree of fever. In the uncertainty which exists as to the exact pathology, there is little else we could do with safety or advantage; to abstract blood would, in the case of embolism or thrombosis, only tend to make the clot firmer, but might be beneficial in a traumatic case, where presumably there is some meningo-encephalitis going on; a blister behind the ear on the side opposite to the convulsed limbs would do no harm and might do good; and of course any source of reflex irritation—teething, worms, etc.—would be sought for, and, if present, relieved as far as possible. After the convulsion a prolonged course of iodide of potassium and alkalies would be the best line to follow, and it is surprising how well children take the iodide, even when administered in an unsparing manner. I have only once known iodism produced, and that was in a little girl nearly 3 years old, who had been taking three grains of the iodide every six hours for about a fortnight. When there is marked contracture, faradisation of the non-contracted muscles will be found useful.

ERRATUM.—In the coloured illustration of the Bacteria found in Scarlet Fever, published last week, two of the drawings were displaced by the lithographer. The description under Fig. 11 applies to Fig. 3, and that under Fig. 3 to Fig. 11.