ON THE NATURE OF THE SPINAL LESION IN POLIO-MYELITIS ANTERIOR ACUTA, OR INFANTILE PARALYSIS.

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It is the opinion of well-nigh all pathologists that the paralysis and atrophy so typical of this disease are the result of destruction of the ganglionic cells in the anterior grey cornua. This is the position taken up by nearly all clinical and pathological teachers, without any fear of contradiction; and, indeed, numerous post-mortem examinations, made in cases where death has resulted from some independent affection, some time after the onset of the lesion, have proved its truth. It has, however, fallen to the lot of very few to examine the cord of a child very soon after it has become affected, in what might be called the acute or inflammatory stage, for, as is well known, it is a lesion that very rarely proves fatal; consequently the exact nature of the essential and primary lesion is little more than a matter of conjecture. Such a chance has recently occurred to me—for which opportunity I am indebted to my friend Dr. Pope, of South Shields. The child whose spinal lesion I am about to describe was a girl aged five. At breakfast-time of the day of her death she was in her usual health, but shortly after her meal she felt sick and vomited. She was then put to bed, when she slept for several hours. Early in the afternoon it was observed that she was feverish and evidently in a dying condition, an observation that was soon consummated, as her death took place six or seven hours after the commencement of her illness—the result, as was afterwards surmised, of respiratory paralysis. At Dr. Pope's request I made a post-mortem
examination. The body was well nourished. The heart was healthy. The lungs were somewhat congested, and in the left was found a small nodule of catarrhal pneumonia, about the size of a walnut. The abdominal cavity and its organs were normal. The brain appeared to be healthy in every respect. Failing to account for death by the changes noted in the lungs, which, so far, were the only morbid conditions detected, I proceeded to examine the upper part of the spinal cord, when the usual transverse sections at various levels from the medulla oblongata downwards brought to light an altered condition of the anterior grey horns between the third and fourth cervical nerves, the appearances at the level of the latter suggesting red softening. This portion of the cord was hardened in a solution of bichromate of ammonium, cut with an ice-freezing microtome, and stained with carmine.

When examined by the low power of the microscope (half inch), the extreme vascularity of the anterior horns was at once apparent. In some of the sections, in addition to the unusual display of capillaries, minute patches of yellow figured prominently upon the carmine groundwork. The ganglionic cells were seen to be numerous and distinct, though apparently swollen. With the high power (1/6 inch, see figure above), the
blood-vessels, distended with corpuscles, formed a striking feature, not only of the anterior horns, but of the anterior white columns and middle and anterior portions of the posterior horns as well, for the inflammatory mischief was by no means confined to the first-named region. Following the tracks of the arteries (internal and external anterior root arteries, and the antero-lateral branch) as they made their way from the front of the periphery of the cord, through the white matters to enter the anterior horns, minute haemorrhages were scattered here and there, whilst congestion, with free leucocytes, was apparent. In places the neuroglia was swollen and so altered as to obliterate in part the meshy character of the structure, and thus conceal the cut ends of the axis cylinders. In this way the bulk of the anterior columns formed a marked contrast to the beautiful and distinct arrangement of neuroglial network, with the nerve fibres seen as central dots, which marked the postero-lateral columns. As the anterior grey horn was approached, the signs of inflammation increased, attaining a maximum as the swollen and somewhat ill-defined mass of grey matter was reached. Here the dilated vessels were crammed full of corpuscles, whilst the capillary network was rendered most distinct, each vessel containing its single or double row of cells. The minute haemorrhages, which were seen as collections of yellow-coloured blood-cells, formed the most striking feature of each field that was examined. These extended as far back as the distribution of the posterior branches of the central artery on each side, that is, quite into the posterior horns, but they were most numerous in the anterior cornu, about the ganglionic cells. The large cells formed prominent objects, though they were apparently swollen, granular, and rather ill-defined; and it was observed that the majority had lost their nuclei, though here and there a ganglionic cell with a nucleus could be distinguished. Their processes were no longer visible, and they appeared to fill completely the spaces in which they lay. The cells that presented the most obvious changes were more or less surrounded with dilated and blocked capillaries and minute haemorrhages; some, indeed, appeared to be imbedded in blood-corpuscles.
In most of the sections it was apparent that one horn had suffered more than its fellow, though in most of those examined both horns showed inflammatory changes. The grey fibres which form the bulk of the anterior horns were swollen and obscure, the whole presenting an indefinite and somewhat granular appearance, which contrasted strangely with the uninjured portions of the posterior horns and roots.

The pronounced changes above described were only observed in a very limited portion of the spinal cord, for as the sections gradually ascended or descended from the affected area, a return to the normal state was brought to light, the signs of inflammation diminishing in intensity, until a few isolated hemorrhages with distended capillaries alone were found.

Although the accident of the situation of the lesion, attacking as it did the region of the origin of the phrenic nerve, prevented the little patient from living long enough after the onset of the inflammation to admit of an exact clinical diagnosis, I do not imagine that any one will be found to question the identity of her case with *polio-myelitis anterior acuta*, and I make no apology for the assumption that they are the same. I think, therefore, that the case is one of considerable importance, as demonstrating the early changes that lead up to the later degenerations which all are so familiar with in old-standing cases of infantile paralysis.

In the year 1870, Dr. Clifford Allbutt recorded in the *Lancet* a case which has been frequently quoted by writers on infantile paralysis (Ross and others), and I think erroneously, as an example of the disease. In this case an examination of the cord, soon after the occurrence of the lesion, revealed two hemorrhages in the cervical region, "one a small one in the left posterior horn, and the other larger, in the right posterior horn, breaking into the lateral column."

I do not think this case can be fairly regarded as a true example of *infantile spinal palsy*, as Dr. Allbutt points out that the paralysis, which affected all four extremities, came on a few minutes after the child, whose age was six months, had been lifted sharply, when its head fell heavily forward. Obviously there is here an element of traumatism foreign to most cases of acute anterior polio-

myelitis of infants; and, further, the hæmorrhages appeared to be confined to the posterior half of the cord, a region which cannot be looked upon as the real seat of the disease, though affected in the case I have described, and also in one to which I am about to refer, recorded by Dr. Angel Money. I am therefore disposed, with due deference to Dr. Allbutt and those who, in quoting the record, have shared his opinion, to think that the hæmorrhages in the case were the result of injury rather than inflammation.

In the 'Transactions of the Pathological Society of London' for 1884 (just published), page 45, Dr. Angel Money describes under the title, "The Spinal Cord of a recent and old case of Infantile Palsy," the appearances of the cord of a child, aged two years, who died of pulmonary disease sixteen weeks after the onset of an attack of acute anterior polio-myelitis. The changes found in the lumbar enlargement were "(1) great distension and thrombosis of vessels, especially in the anterior cornua; (2) infiltration of the cornua with abundant leucocytes; (3) absence of large multipolar or other nerve cells." Dr. Angel Money goes on to state as follows:—"Further, it was seen that the disease was not confined to the anterior horns, but spread forwards, outwards, and backwards; though the principal focus of the mischief was certainly the centre of the anterior horn." This observation, published some time after I had written the above short account of the spinal cord in Dr. Pope's case, confirms my own, in so far as it shows that the inflammation is not by any means confined to the large ganglionic cells, or even to the anterior horns; but the fact that it was made sixteen weeks subsequent to the attack, is sufficient to explain the apparent slight differences between Dr. Money's and my own description of the primary lesion.

Althaus states in his essay, 1 and I believe him to be correct, that there were at that date no post-mortem records where the child had not been paralysed for at least two months, nor am I aware that any more recent examinations have been made since he wrote. The case I have recorded would appear to set at rest the question discussed by Charcot, Roth, Dujardin, and others, namely, whether the inflammation is

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1 'On Infantile Paralysis.' London, Longmans, 1878.
parenchymatous or interstitial, for it is obvious that the several parts affected—the neuroglia, the nerve fibres, and the ganglionic cells—must take part, though they may not all share alike, in the destructive processes induced by the inflammation. Indeed, the brunt would appear to fall on the large cells, a fact revealed alike by the examinations of old-standing cases, and the clinical features of the disease.

Reference has already been made to the extension of the inflammation beyond the limits prescribed by the various writers on the subject, for it will be recollected that though the fresh changes were found most abundantly in the anterior horns, they, nevertheless, affected the antero-lateral white columns, and part of the posterior horns as well; in fact, the anterior cornua were only the centres of inflamed areas, which reached considerably beyond their limits. It will, therefore, become a question whether the name proposed by Kussmaul, and adopted by the majority of writers—anterior polio-myelitis—is really an appropriate one, though usage in the nomenclature of disease, as in other matters, will be found to resent interference.

In conclusion, I would like to point out two ways in which the recognition of the real nature of the primary lesion of infantile paralysis is of importance. In the first place, it throws light upon the attacks of temporary spinal paralysis of infants—seizures, if I may be allowed the expression, which I confess have puzzled me not a little. An example, briefly related, may not be out of place. A little boy, aged 16 months, whilst cutting his eye-teeth, was observed one day to be unusually peevish when being dressed to go into the garden in his perambulator. As he continued to fret, his nurse attempted to amuse him by offering him a ball, when she discovered that he could not raise the left arm to seize it. He was at once taken into the house and undressed, in order to discover if the arm had been injured, when it was found that he was feverish, and that the left arm was very hot and painful (hyperæsthetic). There was no bruising or other evidence of injury, but the limb hung down completely paralysed. When asked to clap his hands, an accomplishment which he had acquired some time before, he contented
himself with slapping his right hand against his leg, ignoring the left entirely. The paralysis lasted for 24 hours, and recovery of motion, which was apparently accelerated by the application of cold to the limb, was first observed in the fingers.

It will not be difficult to explain such an attack by the supposition that a limited area of the left anterior horn in the cervical enlargement had suddenly become congested, the lesion stopping short of actual inflammation.

In the next place, the necessity for early energetic antiphlogistic treatment is emphasised, in order to overcome the inflammation, or reduce it as much as possible, by rest, cold to the spine and affected limb, leeches, or, as recommended by Althaus, the hypodermic injection of ergotin; and the fact that some attacks are only temporary, whilst in others, and indeed nearly all, there is a tendency to recovery shown by some of the muscles at first paralysed, should stimulate us to endeavour to recognise the condition as early as possible, and to carry out thoroughly the curative means at our disposal.