REPORT ON AN EXPERIMENTAL INVESTIGATION OF THE NERVE ROOTS WHICH ENTER INTO THE FORMATION OF THE BRACHIAL PLEXUS OF THE DOG.¹

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DETAILS of the research are contained in a paper read before the Royal Society, March 3rd, 1892. In it allusion was made to the attempts of anatomists to determine the functional relationships between the nerve roots and groups of muscles they supply, in which connection the works of Krause, Schwalbe, Herringham, and Paterson were cited. A brief reference was made to the observations of Erb, Duchenne, Knie, and Thorburn, and to the experimental work done in this field by Müller and Van Deen, Kronenberg, Panizza, Peyer, Krause, Ferrier and Yeo, Bert, Maracchi, and Fargue.

The anatomical accounts of the brachial plexus of the dog, as given by Ellenberger and Baum, Chauveau and Arloing, and Fargue, were quoted, the discrepancies which exist between these different accounts pointed out, and my own experiences in this connection, differing in some points while agreeing in others with the descriptions given by these observers, detailed. My methods of experimentation consisted in:

1. Observation of the compound movements in the fore limb of the dog by electrical excitation of the peripheral end of the whole of a cervico-brachial nerve root which had been previously exposed and divided.

2. Minute differentiation obtained by electrical excitation of the individual bundles composing such a nerve root.

3. Direct observation (after dissection) of the muscles thrown into action by electrical excitation of the separate nerve roots. As a corollary to this, the question as to whether or no a single bundle of fibres, representing a single simple movement in a nerve root ever remains distinct in its course to the muscles it supplies, without inosculating with other nerve fibres, was dealt with. A further point determined was whether, when a muscle receives nerve fibres from more than one cervico-brachial nerve root, both nerve roots supply fibres to one and the same muscle fibre or not.

4. Alteration in the action of the fore limb in progression or in standing, evoked by section of a nerve root or roots.

5. Influence of section of a root or roots in excluding part of a generalised epileptic spasm induced in the limb by cortical excitation.

6. Differentiation of parts of the nerve roots by the degeneration method, in which connection an allusion is made to certain results which I obtained which do not accord with the observations of Waller with regard to the condition of the posterior root between the intervertebral ganglion and the spinal cord, but which are in accord with the experiments of Joseph.

From the results of these various methods of experimentation I draw the following conclusions:—

I.—Stimulation Experiments.

1. The compound movement obtained by stimulation of a whole nerve root is a well co-ordinated one, depending on the action of a group of muscles in synergic combination, as Ferrier and Yeo showed to be the case in the monkey.

2. This compound effect may be resolved into its component factors, when it is found that movements diametrically opposed to each other may be represented in the same nerve roots—for example, flexion and extension.

3. Such single simple movements bear a constant relation to the nerve root, and thus such movements always bear the same relation to the spinal level; for example, flexion of the elbow is always represented one root higher than extension of the same joint.

4. Fibres representing a certain movement always preserve

the same position in a given nerve root; for example, extension of the wrist is represented by a bundle of fibres in the upper part of the circumference, while flexion is represented by a bundle of fibres in the lower part of the same root.

5. Each bundle of nerve fibres representing a single simple movement in a nerve root remains distinct in its course to the muscle or muscles producing such a movement, without inosculating with other motor nerve fibres.

6. The group of muscles supplied by any given nerve root occupy both the anterior and posterior surfaces of the limb. In other words, muscles whose unimpeded action would produce one movement are represented in the same root as others whose action would produce a movement diametrically opposite.

7. In such combinations certain muscles are always more extensively represented than others, so that, with a current sufficiently strong to stimulate all the fibres of a nerve root equally, certain muscles predominate in their action over others.

8. The muscles whose action predominates in one root always predominate in that root.

9. If the muscles producing flexion of a certain joint predominate in their action in one root, those producing extension predominate in another.

10. It is possible, by stimulation of a single bundle of fibres in a nerve root, to produce contraction of a single muscle, and it alone.

11. The same muscle is always represented in more than one nerve root, usually two, and to an unequal extent in these.

12. When the same muscle is represented in two nerve roots the muscle fibres innervated by one root are not innervated by the other.

II.—Ablation Experiments.

1. Division of any given root produces paresis of the group of muscles supplied by it.

2. This paresis is only temporary, and soon passes off almost completely.

3. Such division of a nerve root does not result in inco-ordination of the remaining muscular combinations represented in other nerve roots.

III.—Exclusion of a certain Root or Roots during an Epileptic Spasm in the Limb (the root being divided at the time, and not some time previously.)

1. Division of one or more nerve roots produces alteration of the position of a limb during an epileptic spasm, which altered position depends on the particular muscular combinations that have been thus thrown out of action.

2. No inco-ordination is produced in the action of the remaining muscular combinations.

3. There is no evidence of overflow of the impulses which ought to travel down the divided root into other channels through the spinal centres, so as to reach the muscles by new paths.

IV.—Degeneration Method.

1. These experiments confirm the anatomical facts that had been previously ascertained by dissection, as to which nerve roots supply any given nerve with fibres.

2. The degeneration which results in the nerves is not a scattered one, but is localised to distinct bundles of nerve fibres occupying a certain position in the transverse section of the nerve.

3. Waller's observation—that when a nerve root is divided on the distal side by the intervertebral ganglion no degeneration of fibres is found in the posterior root between the ganglion and the spinal cord—is found to be erroneous; for such degeneration does exist, and points to the probability that there are certain nerve fibres which do not depend on the ganglion for their trophic supply, but derive the same from elsewhere, either the spinal cord at another level, or the periphery.

In conclusion, I call special attention to the value of the method of excluding one or more nerve roots during an epileptic spasm, as affording a means of confirming the facts that have been previously observed from stimulation of the nerve roots, and also of ascertaining new facts with regard to them and the plexuses which they form. I further go on to point

¹ Part of the expenses of this investigation have been defrayed by a grant from the Scientific Grants Committee of the British Medical Association.

out that it supplies a valuable means of studying the manner in which conduction of impulses from the cortex through the nerve roots and plexuses to the muscles takes place; and that it is capable of still wider extension, as if, instead of producing general epilepsy, less powerful stimuli be applied to the centres for different movements, as represented in the motor cortex, it will afford a means of connecting such centres, or parts of these, with the nerve roots to which fibres proceed from these cortical motor centres.

CLINICAL NOTES ON PARALYSIS OF THE DIAPHRAGM.

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PARALYSIS of the diaphragm is a rare affection, but in my opinion it is frequently overlooked, though it may be easily detected if searched for. During the past year I have met with six cases. The first case, which I saw with Mr. Stanley, of Small Heath, was that of a young gentleman who had strained his neck over a horizontal bar in an athletic display. Within an hour he complained of numbness and weakness of his legs, and when I saw him these symptoms had increased. He had difficulty in swallowing, and scarcely any power of phonation. I found paralysis of the right half of the diaphragm, the left half acting but feebly. By keeping the patient absolutely at rest, and by careful feeding, with frequent application of a faradic current to the right phrenic nerve, the patient was kept alive. The diaphragm gradually recovered power, and in six or seven weeks he was quite well. The lesion in this case was probably hæmorrhage around the cord above the origin of the phrenic nerve, the pressure being greater on the right side.

The second case, which I saw with Mr. Hall-Edwards, was that of a young lady who was suffering from influenza, and I was called in on account of severe neuralgic pain in the right lower extremity. The pain was paroxysmal, and of such severity that morphine injections had to be given constantly. On my second visit I found the right half of the diaphragm paralysed. There was no dyspnoea and no alteration of the voice. We decided to apply a faradic current to the phrenic nerves, one pole being placed at the lower end of the anterior triangle in the neck, the other over the hypocondrium. The patient improved with this treatment for a day or two, and then died quite suddenly. This was in my opinion a case of acute multiple neuritis, and the paralysis of the right half of the diaphragm was due to neuritis of the right phrenic nerve, the implication of the left nerve being the probable cause of sudden death.

The next three cases were all due to diphtheria, and were all fatal. I believe that paralysis of the diaphragm is the cause of the great majority of sudden deaths after diphtheria, and that only a few can be attributed to syncope. I believe also that in many cases the diaphragmatic paralysis is not recognised. Of the three cases one was a man and the other two children. In all the cases paralysis of the legs was present, and there was no difficulty in recognising the diphtheritic origin of the mischief, though in one case the sore throat had been very slight. While at rest in bed there was no dyspnoea, but phonation was very feeble and defæcation and micturition impeded. On examination of the abdomen the diagnosis was readily made. The hypocondrium on the affected side became depressed on inspiration instead of being propelled forwards, and by placing the hand under the ribs the non-descent of the diaphragm could be easily ascertained. There was compensatory overaction of the lower intercostal muscles and great enfeeblement of the breath sounds at the base of the lung on the affected side. In each case the right half of the diaphragm was chiefly affected, but I believe this is due to the presence of the liver on this side and to the inability of the weakened muscle to push the organ down, while on the left side the muscle can descend until quite paralysed.

In all three cases the paralysis was recognised a day or two before death, and special precautions taken, but in each case death occurred quite suddenly. One little boy was brought

to my consulting room; observing the feeble cough and phonation, I had him stripped and examined the diaphragm; I found it paralysed. This enabled me to caution the parents of his grave danger, and I heard afterwards that he died suddenly the day after seeing me. If both halves of the diaphragm become paralysed, death ensues from asphyxia, and the fatality of diaphragmatic paralysis after diphtheria can easily be understood when we remember the severity of the neuritis which frequently follows diphtheria, some patients being paralysed for a year or more.

As to treatment I would recommend that every case of diphtheritic paralysis be kept in bed from the first, and that plenty of nutritious food be given. Iron and strychnine should be administered in large doses. If weakness of the diaphragm is observed the patient should be raised in bed with pillows, so that the diaphragm may act more easily. A gentle faradic current should be used three or four times a day, and blistering fluid painted over the course of the phrenic nerve in the neck. Stimulants should be given freely.

The prognosis of paralysis of the diaphragm after diphtheria is very grave, and its onset may possibly be prevented by keeping patients in bed and at rest whenever any signs of paralysis are present. The early recognition of paralysis of the diaphragm is very important from a prognostic point of view.

The sixth case was that of a woman, aged 37, who was admitted for a second attack of alcoholic paralysis. The hands and feet were dropped, and the usual symptoms were present in a typical manner. The diaphragm was observed to be paralysed, and the patient died suddenly a day or two after this observation. I am not aware that paralysis of the diaphragm has been previously observed in alcoholic paralysis, or that it has been noted as a cause of sudden death, most of such deaths being attributed to cardiac paralysis. This case emphasises the great similarity that exists between alcoholic and diphtheritic paralysis, and as alcoholic paralysis is recognised as being due to multiple neuritis, the paralysis of the diaphragm being also due to neuritis of the phrenic nerves, we may reasonably infer that paralysis of the diaphragm after diphtheria is also due to neuritis of the phrenic nerves. In none of the above cases could a *post-mortem* examination be obtained.

A CASE OF RAPIDLY FATAL DIABETES MELLITUS IN A BOY AGED 10.

By WILFRED WATKINS-PITCHFORD, M.R.C.S., L.R.C.P.

J. A. C., aged 9 years and 9 months, was first seen on April 23rd. For about a fortnight the child had been complaining of dryness of the throat and mouth, of being very thirsty, troubled with cough, and of passing more water than natural. He was also, though eating heartily, growing thin. He was a thin boy, with flushed, anxious-looking face and widely dilated pupils. The skin everywhere felt dry and harsh. There was no congestion of the fauces. The tongue was clean and of a bright red raw colour. No abnormal physical signs could be detected in the thoracic or abdominal organs. The liver dulness was normal in extent. The urine, of a pale greenish tinge, was of acid reaction, specific gravity 1035, and free from albumen, but gave immediate evidence of abundance of sugar with the liquor potassæ and sulphate of copper test. The pulse was strong, 80, and the temperature normal.

Upon these symptoms a diagnosis of diabetes mellitus was founded and a correspondingly serious prognosis given.

The subsequent history of the case was as follows:—The patient was at once put on a diabetic diet, and for a day or two partook freely of milk and water, eggs, green vegetables, and diabetic biscuits. He was ordered 5 minims of the liquor morphinæ hydrochloratis every three hours. On the following day the tongue became furred down the middle with white fur; the breath was noticed to smell like that of a milking cow. During the twenty-four hours ending 6 p.m. he passed 6 quarts of urine, presenting the same characteristics as that examined on the previous day.

During the next twenty-four hours 5 quarts of urine