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THE ASSOCIATION OF TRAUMA WITH PROGRESSIVE MUSCULAR ATROPHY

BY

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We know little indeed about the aetiology of progressive muscular atrophy, but its association with trauma in a small percentage of cases has been described by eminent Continental neurologists during the past decade. Thus Alajouanine, Gopcevitich, and Meillaud (1930) gave an account of an interesting case of post-traumatic muscular atrophy, with an illuminating discussion on the subject. These authors divide such atrophies into different groups.

The Muscular Atrophy Groups

In the first group the muscular atrophy is localized to the region of the muscles in the neighbourhood of the injured joint. They state that to this group belong some of the amyotrophies of the periscapular region which may follow injuries to the shoulder-joint, and suggest that such amyotrophy is of reflex origin, as is indicated by the electrical changes and by the fact that after progressing for several months cases remain stationary or even retrogress. They define this type as "localized post-traumatic reflex amyotrophy."

In the second group the amyotrophy ensues rapidly upon the injury, and it develops on a paralytic background. Its distribution is generally radicular, and it is accompanied by sensory changes of a dissociated character which suggest that this type may follow minor traumatic fissures of the spinal cord, with resulting frustrated haematomyelia. In such cases the wasting, at first progressive, becomes stationary after a time. They describe it as spinal amyotrophy secondary to frustrated haematomyelia.

In a third group of cases the wasting develops more slowly. It has not been preceded by any notable paralytic phenomena and is not associated with sensory troubles or reflex abnormalities. The atrophy in such cases may be localized or diffuse and non-systematized. The electrical reactions are not myelopathic in type, but conform closely to the changes found in the myopathies. This group progresses for a time, but may later display a tendency to regression.

Descriptive Cases

Alajouanine and his colleagues describe the case of a man, aged 34, who was seen at the Salpêtrière for a diffuse muscular atrophy which had slowly progressed over a period of twelve years. The condition had begun two months after an aeroplane accident which had rendered him unconscious, and in addition to contusions had resulted in a split fracture of the head of the radius.

Another case of post-traumatic muscular atrophy is described by Horst Geyer (1934). This patient sustained a fractured tibia and fibula of the right leg in 1905. Four years later wasting was observed in the right thigh. Clubbing of the right foot was also present as a result of acute anterior poliomyelitis which had occurred in infancy. When examined by Horst Geyer in 1932, in addition to the typical claw hands, wasting and fibrillary tremors were widespread. The ankle and knee reflexes were practically absent, and there was no evidence of any pyramidal tract lesion. The small muscles of the hands and feet showed no response to galvanism or faradism.

The next case described by Horst Geyer is that of a male, aged 52, who in 1921 was struck in the small of the back by a branch of a tree. As a result of this he was unable to work for several weeks, but subsequently recovered. Eight years later he fractured his left leg in a motor-cycle accident. When examined at the clinic on May 9, 1930, the left leg showed marked generalized wasting. No abnormalities were observed in the rest of the body. When seen again three years later there were definite signs of progressive muscular atrophy, wasting and fibrillary tremors being present in all the limbs, though they were more marked on the left side than on the right.

Predisposing Factors

Geyer remarks that these two cases of his have in common the onset of progressive muscular atrophy some years after the original injury. In discussing the frequency and pathogenesis of the condition, he says that of the twenty-one cases which appeared at the clinic between the years 1919 and 1933 only one could be shown clearly to be due to trauma. He quotes Kure, who stated in 1931 that the innervation of muscles is fourfold—cerebral, spinal, sympathetic, and parasympathetic—and disturbance of any of these can lead to muscular atrophy. He points out, further, that many eminent neurologists are of the opinion that the main feature is a congenital predisposition, and that trauma has merely "pulled the trigger" in such cases. This theory, he says, is held by Strümpell, Probst, Oppenheim, and others. Marburg asserts that this is merely a possible theory, and that there is no anatomical evidence in support of it. Other authors maintain that one has to consider the question of an acquired disposition. Rose divides the predisposing factors into five groups: one is the simple type previously mentioned; the other four are acquired—namely, toxic; infective; those occurring after cerebrospinal diseases, such as anterior poliomyelitis; and, finally, the traumatic group, such as those associated with fractures. Other authors also stress the association with anterior poliomyelitis in some cases. Thiern considers that there occurs in the traumatic cases a traumatic neuritis which spreads up towards the spinal cord, and eventually by association with the anterior horn cells produces progressive muscular atrophy. Another theory propounded is that, acting on the congenital or acquired predisposition, there occurs in the traumatic cases a shaking or

concussion of the spinal cord, which produces a molecular disturbance of the grey substance, and in this way leads to progressive muscular atrophy.

Influence of Injury

The question is further discussed, with the description of another case, by Brodin, Lhermitte, and Lehmann (1931). They state that for some years they have been greatly interested in the subject of the influence of injury on the production of maladies of the central nervous system, on account of the frequency with which certain associated complications appeared which apparently resulted from a "shaking" of the spinal cord. These authors state that they were able to produce progressive muscular atrophy in dogs as a result of trauma, and that pathological examination subsequently revealed that the grey substance of the cord showed degeneration, particularly the anterior horn cells. They were greatly interested in the latent intervals which separated the date of the injury from the first manifestation of the wasting, and describe the case of a patient aged 59 whose past history was entirely uneventful.

In 1925 he was caught between a heavy plank and a ladder after having fallen down several steps, and sustained severe contusions in the region of the dorsal spine. Some months after the accident there appeared very gradually a slight weakness of the right arm. Two years later he sustained a further injury when he fell from a height on to an obstacle and was rendered unconscious.

When seen by the authors in 1931 wasting and fibrillary tremors were well marked in the arm and shoulders, particularly on the right side. There were no abnormalities of sensation, but while the cutaneous reflexes were normal the tendon reflexes were completely abolished in all four limbs. The electrical reactions were correspondingly diminished. There was no sphincter trouble, and no sensory changes were present. The vertebral column did not show any sign of injury or other abnormality.

The authors point out that the latent interval, as manifested in this case, is quite the rule and should not detract from the diagnosis of progressive muscular atrophy, for, as they have shown, it is also borne out by animal experiment.

I shall now describe the history of a patient whom I saw myself on May 22, 1936.

The patient, a man aged 62, stated that for a year and a half there had been increasing weakness in the right arm, together with occasional twitching, as well as slight numbness of the fingers. The past history revealed that forty-two years ago he had sustained a very severe depressed fracture of the skull as the result of a jack falling on his head. Some years later traumatic epilepsy ensued, and this was relieved twelve years after the injury by Sir John Lynn-Thomas, who removed a piece of bone which was pressing on the motor area. The patient stated that he had remained quite well from that time until eight years ago, when the right leg began to swell and was painful. He found, however, that he was able to compensate for this by wearing an elastic stocking.

On examination the first thing to which one's attention was drawn was a very long and deep depression involving the left parietal, temporal, and frontal regions. Then one noticed that fibrillation was very marked on both arms and on both sides of the face. Wasting of muscles was pronounced in the region of the right shoulder and the right arm. The extensor muscles of the right arm were so impaired that he was unable to dorsiflex the wrist. There was the usual wasting, resulting in a typical claw hand. The right arm was flaccid and flail-like, and all reflexes were diminished.

A thorough examination of the central nervous system failed to reveal any other abnormality. The disks were clear

and the fundus oculi was normal. The cranial nerves were normal; the pupils reacted to light and accommodation, and there was no "intention tremor" and no "Rombergism." The sense of position was not impaired, and sensation to heat, cold, pin-prick, and cotton-wool was normal. Speech was also unimpaired. The plantar reflexes were flexor, but the right knee-jerk was rather greater than the left knee-jerk. Muscular power, however, was unimpaired in the legs. The blood pressure was 130/80, and other systems of the body were quite normal.

Since I first saw the patient fibrillation of muscle has become widespread, but though wasting is more marked in the right arm it is as yet not present elsewhere.

I have quoted these cases together with the subsequent discussion at some length because, while British neurologists recognize that syphilis and acute anterior poliomyelitis are occasional aetiological factors in progressive muscular atrophy, the majority deny the association with trauma. For many years, however, eminent neurologists on the Continent have recognized that trauma is a definite causal factor in a small proportion of cases.

During the period 1925 to 1934, inclusive, thirty-one cases of progressive muscular atrophy were admitted to the Bristol Royal Infirmary, and in only three of these do the case notes include a history of injury. It is probable that this number would have been higher but for the fact that many physicians do not inquire about a previous history of injury in such cases because the importance of this is not recognized.

Commentary

There is really no evidence in favour of the idea that trauma merely "pulls the trigger" in cases where there is a congenital predisposition, nor is there any practical support for the suggestion put forward by Thiern that an ascending neuritis occurs which ultimately involves the anterior horn cells and so produces the muscular atrophy. In the first place we do not generally find such changes with neuritis, and, secondly, symptoms of neuritis are conspicuous by their absence in the history of practically all the cases of post-traumatic progressive muscular atrophy. Localized reflex wasting after injuries of bones and joints is an entirely different condition. We are faced here with the problem of widespread progressive muscular atrophy developing in some cases years after a severe injury. It seems to me that, as some of the French authors have suggested, we are bound to assume that a molecular disturbance of brain or cord is occasioned by the injury. Is this followed by slow degeneration, or does it render the cells susceptible to the subsequent attack of an unknown virus or toxin? With regard to this it will be recalled that a most mysterious feature of the disease is the lack of correspondence between the pathological process and the symptomatology. It is possible, therefore, that degeneration of the Betz cells in the cortex and of the anterior horn cells in the cord may be occurring for some years after the injury before clinical signs become manifest.

In the present state of our knowledge, however, we can only speculate on the way in which the atrophy is brought about, and the purpose of this paper is primarily to put forward evidence that trauma is a definite aetiological factor in some cases of progressive muscular atrophy. From the medico-legal point of view alone it will be realized that this association is of very great importance.

Summary

1. Cases are described which show that trauma is a definite aetiological factor in some cases of progressive muscular atrophy.

2. The wasting of muscle is frequently not manifest until many years have elapsed since the original injury.

3. The problem of the way in which the atrophy is brought about is discussed.

4. From the medico-legal point of view alone the subject is of very great importance.

I am indebted to Dr. M. E. Disney, medical registrar, for assistance with the case notes at the Bristol Royal Infirmary.

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AETIOLOGY OF ACUTE APPENDICITIS*

BY

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I. The History of Acute Appendicitis

Although appendicitis is usually regarded as a disease of modern civilization a small number of Egyptian mummies have been found to possess unmistakable signs of chronic inflammation of the organ, and in one case, that of a young royal princess, an acutely inflamed and perforated appendix was beautifully preserved.

The first recorded case in modern Europe was in a patient of Mestiver (Germany) in 1759, but the disease does not appear to have been recognized in England until 1812, when Parkinson diagnosed it in London. For some years the aetiology was obscured by the use of the term perityphlitis; but after 1820 appendicitis was increasingly recognized. Between 1820 and 1840 there were thirty-three recorded cases, and during the twenty years 1840-60 the number had risen to 102. In 1918 the deaths from the disease totalled 2,416. The figures showing the incidence of the disease at the Bristol Royal Infirmary are of interest. In 1880 out of 2,591 patients there were four cases of appendicitis. By 1905 the proportion had risen to sixty-four cases out of 3,762 patients, and by 1918 it had reached 113 appendicectomies in 4,021 patients.

DISTRIBUTION OF THE DISEASE IN THE COMMUNITY

Both sexes are equally prone to the disease, and the age of greatest liability is from 10 to 30 years. While it does occur in infants it is rare to find the disease before 3 years of age. It is also rare to find it at the other extreme of life, but it has been known to occur at 84 years. The disease is more serious at the extremes of life than it is during the years of its maximum incidence. As regards the influence of social position, the disease is definitely more common among the well-to-do; poorer people, especially those living in institutions, enjoy a relative immunity. For instance, there was only one case in ten years at Portland Prison. At Clifton College, Bristol, twenty cases occurred among 500 boys in five years, while at Müller's Orphanage, also in Bristol, there were only four cases among 950 children in the same period.

NATIONAL DISTRIBUTION

The disease is commonest in the United States of America, where its incidence is nearly twice what it is in Great Britain. Holland and Sweden have an incidence

equal to our own, but the disease is less common in Denmark, Italy, Spain, and Greece. In the West Indies it is extremely rare among the natives but relatively common among the white population. In the villages of India and China the disease is unknown, but it is seen in the towns, both among the white population and those natives who adopt a European diet. This in fact is true all over the world. Native peoples, among whom the disease has been previously unknown, become very prone to it as soon as they live on a European diet. In seeking a cause of appendicitis the following summary of historical facts will be found helpful.

1. The disease was present, but relatively rare, in this and other countries until the end of the nineteenth century, since when it has become increasingly common in most countries. The rise in England began in 1890, and was pronounced between 1895 and 1905. From 1905 onwards it has been fairly stationary.

2. The rise was at first most marked in the towns; later the rural districts were affected, until at present the incidence is the same for town and country.

3. Inmates of institutions, living on a plain diet, are less prone to the disease than are more well-to-do people.

4. The privations of the war did not affect the frequency of the disease.

5. Appendicitis is unknown in wild animals, while it is common among animals in captivity.

THEORIES OF CAUSATION OF APPENDICITIS

As with all diseases of unknown causation, theories of the cause of appendicitis abound. One of the earliest was that it was due to the replacement, by the water-closet, of the squatting position in the act of defaecation. Another was that the fault lay with the substitution of stone flour rollers by steel ones, but no pieces of steel have ever been found in the appendix.

Of greater interest is the theory that the disease is due to an alteration in the food habits of the people, resulting in a marked reduction of the cellulose intake. During the last forty years English diet has changed very considerably, and it is of interest to examine these changes to see whether they have any bearing on the causation of appendicitis.

(1) Since 1890 there has been a tremendous increase in the consumption of cocoa and chocolate; but these articles can be dismissed at once, since animals in captivity which develop appendicitis do not eat them.

(2) *Bananas*.—West Indian natives eat large quantities of bananas, but they do not develop the disease.

(3) *Butter*.—This commodity has always been plentiful.

(4) *Meat*.—There is a very close resemblance between the increase in appendicitis and the rise in imported meat in the period 1895-1905. During these years our meat imports doubled, both beef and mutton being affected. There is no doubt but that the average consumption of meat per head of the population increased parallel to the rise in appendicitis, but if we examine the problem further we find that large sections of the community were heavy meat-eaters before 1895. In 1889, for instance, Pearce's dining-rooms in London supplied 30,000 carmen daily with a lunch containing 5 oz. of meat, and each soldier in the British Army was allowed 12 oz. of meat a day, yet the incidence of appendicitis among them was very low.

(5) *The Reduction in Cellulose Intake*.—Between the years 1890 and 1915 the consumption of preserved meats, butter, tea, cocoa, chocolate, rice, bananas, etc., increased considerably, as has been mentioned, so that the older, coarser, cellulose-containing foods, such as swedes, turnips,

* The successful thesis entered for the Martyn Memorial Scholarship in Pathology in the University of Bristol.