MUSCLE SPASM IN POLIOMYELITIS

ITS TREATMENT AND SUGGESTED ETIOLOGY

A PRELIMINARY REPORT

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Spasm *in poliomyelitis* is still regarded as a reflex tonic contraction, based on Sherrington's Law of Reciprocal Innervation[1] or the reversal of this law as suggested by Bouman and Schwartz.[2]

Our opinion regarding the relationship of Sherrington's Law to the etiology of spasm, is that it only applies when actual movement is taking place. At rest there is no overaction of the antagonist of a paralysed muscle. For instance, if the radial nerve be divided there is no immediate contraction of the flexors of the wrist and fingers resulting in deformity of the hand. Wrist-drop, when it occurs, is due solely to the effect of gravity. If the hand is placed midway between pronation and supination, resting on the ulnar border (to eliminate gravity) no deformity ensues. These facts suggest that the spasm and deformity occurring in poliomyelitis, in the early stages, whilst the patient is at rest, is not due to the overaction of unopposed antagonists.

We suggest an alternative explanation for the mechanism of spasm. Our premise is that it is due to sympathetic overactivity resulting in heightened muscle tone; i.e., the clinical phenomenon known as spasm. Treatment directed towards diminishing or abolishing the influence of the sympathetic nervous system results in dramatic disappearance of muscle spasm. Furthermore, other concomitant features of sympathetic overactivity which may occur with spasm, *namely coldness, sweating and pain, disappear pari passu* with the abolition of spasm.

INVESTIGATION OF SPASM

On the above premise we treated 25 cases of poliomyelitis with varying degrees of spasm with etamon, a drug which temporarily blocks the transmission of impulses, through autonomic ganglia. As far as we know, this is the first time that a drug with this particular action has been employed in the therapy of one specific component of poliomyelitis, viz. spasm.

Administration of Etamon. The drug can be given intravenously or intramuscularly. The maximum dosages recommended are (intravenously) 7 mg. per kg. body weight and (intramuscularly) 20 mg. It is advisable not to exceed the recommended dose initially, so that the reaction of the patient may be assessed. We found that this amount was inadequate in the majority of cases and, therefore, increased it slowly in subsequent injections without any ill effects. All injections were given intravenously because of limited supplies. The common effects of intravenous administration are :—

1. Bitter taste in the mouth.
2. Tingling and paraesthesiaee all over the body.
3. Shivering and coldness.
4. Ptosis and blurring of the vision.
5. Tachycardia in some cases.
6. Drop in blood pressure resulting in postural hypotension.

The latter which occurred in only two cases, can be counteracted with prostigmine or adrenaline. We, however, did not use these antidotes on any occasion.

The above side effects disappear within a very short time. After the injection most patients feel tired but are still able to co-operate.

The number of injections required depends upon the response of the patient. Whilst spasm is present, it is advisable to administer the drug three times a day rather than space the injections at one- to two-day intervals. The treatment should be continued whilst spasm and pain persist. After each injection intensive passive movements of the spastic muscles should be performed. This is considered to be an important adjuvant. To test the efficacy of etamon, its effect in a few cases was compared with paravertebral lumbar blocks. In no instance was the effect of the block superior to that of etamon.

RESULTS OF THERAPY

1. Subjective Improvement. Improvement commences within 10 to 15 minutes of the injection and is maximal within 2-3 hours. Once the disagreeable symptoms associated with the injection have disappeared, all the patients treated had marked relief of pain. The majority stated that they were able to sleep comfortably for the first time. The feeling of tightness in muscles disappeared, resulting in increased mobility. Despondency, due to the stiffness, pain and deformity, gave way to serenity and optimism.

2. Objective Improvement :

i. Disappearance of Spasm. This was demonstrated by an increase in the range of passive and active movement of the muscles involved. For example, prior to the injection some patients had poker-stiff backs and complained of severe pain and discomfort when flexion of the spine was attempted. Soon after the treatment, however, passive and active spinal flexion was carried out comfortably and not uncommonly the patient was able to appose the forehead to the knees. Extension at the knee with the hip flexed, which, prior to therapy, did not reach 90 degrees, was now noticeably increased.

ii. Disappearance of Pain. Pain is a distressing and constant source of irritation to the patient with poliomyelitis. He can neither sleep nor move around in bed without aggravating this disability. There are several types of pain, namely :—

(a) Pain at rest, which is constant, nonsegmental and diffuse.
(b) Pain on pressure of the spastic muscles.
(c) Pain produced by active and passive movements of involved muscles.
(d) Pain due to hyperaesthesia of the skin.

All the above varieties were either abolished or considerably relieved by one or more injections. As a result of this relief, physiotherapy could be started immediately.

iii. Disappearance of Deformities. In four cases an equinovarus deformity of one or both feet disappeared immediately after etamon. The result was dramatic. In two other cases, flexion deformities of the wrist and fingers, with marked adduction at the shoulder joint, resulted in complete functional disability of the arm. Soon after treatment the deformities were corrected with complete return of function.

iv. Increase in Mobility. An important feature in some cases was the restoration of function in muscles which hitherto were regarded as "paralysed." This was due to the fact that the muscular power was disguised by the existing spasm in the muscles themselves or their antagonists. This functional return had a great effect psychologically in that the patients noticing the rapid improvement, lost their depression and apathy. We think that active physiotherapy should be initiated as soon as pain and spasm have been relieved. This still further increases the co-operation of the patient, minimises muscle wasting and effectively restores muscle co-ordination.

v. Disappearance of Vaso-Motor Disturbances. The cold, clammy limbs were replaced by warm, dry ones.

DISCUSSION.

Since Kenny re-emphasised the phenomenon of muscle spasm, attempts have been made to explain its mechanism. Toomey regards spasm as a reflex tonic contraction occurring after paresis or paralysis has established itself. He does not recognise spasm as a separate entity but regards it as a secondary manifestation in antagonists when paralysis has developed in the agonists. Bouman and Schwartz[2] state that spasm is a reflex phenomenon not associated with localised lesions in the muscle itself, thereby differing from Pohl[3] who postulates local muscle pathology.

According to the former, spasticity is due to a reversal of Sherrington's Law. Spasticity and weakening of muscles are two separate phenomena, each dependent upon disturbances of specific functions of the anterior horn cells. Kabat and Knapp,[4] ascribe spasm to localised lesions of the internuncial neurones of the spinal cord.

Our concept of the etiology of spasm differs from the abovementioned in that we regard it as a manifestation of sympathetic overactivity.[5] Our reasons for stating this are as follows: It was noticed during the 1945 epidemic of poliomyelitis in South Africa that a large number of cases with cold, sweaty limbs had muscle spasm. Paravertebral blocks were given primarily to increase the temperature of the limb. Following a successful block, muscle spasm and pain were relieved simultaneously. In the 1948 epidemic these associations, viz.; spasm, vaso-motor and sudomotor disturbances, were again evident and we attributed these phenomena to sympathetic overactivity because of their dramatic alleviation following inhibition of this activity either by the use of etamon or paravertebral block. Both these therapeutic agents act on sympathetic ganglia.

In 25 cases of spasm in poliomyelitis, inhibition of sympathetic tone by means of etamon resulted in complete disappearance of the muscle spasm. In some instances one injection was sufficient to produce a permanent therapeutic response. Since etamon acts primarily on ganglia of the sympathetic nervous system, it is reasonable to postulate that spasm is intimately connected with autonomic function. There is a strong body of opinion which supports direct sympathetic influence on muscle tone.[5] (Boeke, Dusser de Barenne, Kuntz and Kerper, Fridman and Spiegel.)

In poliomyelitis, pathological changes have been described in the sympathetic ganglia of humans and chimpanzees.[6,7] The absence of neurone destruction as emphasised by Bodian and Howe is clinically substantiated by the fact that in a great number of cases of poliomyelitis, no instance of sympathetic paralysis occurred in the limbs.

Our clinical observations demonstrate the presence of sympathetic overactivity which can be explained by the existence of pathological changes in such ganglia as described by the above workers.

The phenomenon of spasm is of variable duration. The reason may be as follows: The neurotropic virus of poliomyelitis is absorbed from the gastro-intestinal tract along sympathetic pathways. Hence the first structures it encounters in its centripetal progress towards the spinal cord are the sympathetic ganglia. The duration and severity of the spasm will depend upon two factors, viz. the intensity of the pathological change induced by the virus and its length of stay in the ganglia.

Our view that spasm in poliomyelitis is due to sympathetic overactivity is borne out by the fact that therapeutic measures abolishing this overactivity, result in disappearance of the spasm. Furthermore, our observations support the principle of direct sympathetic influence on muscle tone.

CONCLUSIONS.

1. A physiological concept of the etiology of spasm in poliomyelitis is presented.

2. This is related to lesions in sympathetic ganglia, producng sympathetic overactivity.

3. Treatment directed towards decreasing or abolishing this abnormal autonomic overactivity was undertaken, using etamon or paravertebral block.

4. The described method of treatment will accomplish the following :—

i. Relieve spasm.
ii. Relieve pain.
iii. Relieve cold, clammy limbs.
iv. Enhance the mobility of affected parts.
v. Diminish deformity.
vi. Shorten hospitalisation.

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