MODELLING OF SPASTICITY AND ITS COMPENSATION BY ELECTRICAL STIMULATION

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Summary

After a short discussion on spasticity and on some conventional ways for its treatment, two control loop models are proposed. The first assumes no feedback from the receptor fibers to the gamma motoneurone whereas the second supposes that such a relation might exist.

Transfer functions of a spastic extremity are developed and it is shown that the control-theory approach supports the contention that increased sensitivity of the muscles spindle is responsible for spasticity.

Electrical stimulation as a possible agent to reduce spasticity is examined and a hypothesis regarding the therapeutic effects of stimulation is forwarded.

Recommendations for further work are given and it is suggested that stimulation to relieve spasticity be incorporated into advanced orthotic procedures.

Introduction

Spasticity is one of the most common disorders of tone seen after lesions of the central nervous system in man. Clinically, spasticity is characterized by a resistance to passive movement which builds up in intensity as a muscle is stretched until it reaches a high tension; the muscle then relaxes rather abruptly in the socalled "clasp-knife" reaction. This resistance to passive movement is sensitive to rate of displacement as well as position and will build up faster when the limb is stretched more rapidly.

With the exception of certain cerebellar disorders, the mechanism of spasticity is probably quite similar in various neurological disorders, i.e. hyperactivity of the gamma motor system, supplying the muscles' length recorders, the muscle spindles [1, 2, 3, 4]. The pattern of muscles affected and the postures assumed are determined by the areas of the nervous system that are spared by the lesion [5, 6].

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Animal experiments have shown that lesions in the corticospinal tract allow remaining subcortical systems, e.g. the vestibular system, to cause an increased discharge of the fusimotor fibers (gamma) to the muscle spindles [7]. This increased bias on the muscle length recorders makes them more sensitive to static and dynamic stretch.

The muscle spindle afferents increase their discharge onto the alpha motoneurones and cause them, in turn, to increase their firing rates. These increased efferent impulses keep the muscle in a state of contraction and increased sensitivity to stretch that we call spasticity. Thus loss of voluntary control over muscles is over-compensated for by heightened reflex activity. This excessive tone can interfere with the reorganization of muscle movements into effective, functional movements as voluntary control returns. Several approaches have been attempted to prevent the development of spasticity or to reduce it once it has been established.

Neurosurgical and orthopedic procedures relieve spasticity by cutting nerves to prevent impulses from reaching muscles or by cutting the tendons of spastic muscles so that they can no longer exert force at their insertions. Such approaches achieve limited results by increasing the patient's deficit rather than effecting a physiological improvement.

The use of various drugs such as Valium has been of some help but beneficial effects often require doses that depress the patient's sensorium. Preferential block of gamma fibers with dilute procaine, while effective experimentally, is very difficult to control in the human and the effects are transient [8, 9]. Phenol injections into the spinal canal or motor endplate are not always selective in their destructive effects on nerves [10, 11].

While mechanical vibration of limbs [12, 13, 14] is more physiological in concept it is difficult to vibrate specific muscles preferentially since movement is transmitted to all of the muscles of the limb.

Since certain afferent fibers in nerves facilitate the motoneurone pool and others inhibit it, it would seem logical that appropriate stimulation of certain of these fibers might be used to reduce the imbalance found in spasticity. Since electrical stimulation offers the advantage of selectively activating facilitatory or inhibitory circuits by appropriate muscle (and/or nerve) stimulation it would appear to be an ideal approach in any study oriented toward reducing spasticity.

Our approach will be to offer an engineering model of spasticity based on neurophysiological principles and to show why we feel electrical stimulation should be considered as a feasible area of investigation in efforts to relieve spasticity in human neurological disorders.

Engineering Models of Spasticity

Mechanisms of Spasticity

Spasticity is caused by an excessive gamma motoneurone discharge. This increased discharge can occur from a control-theory viewpoint in two basically different ways:

1) As an increase in gamma activity which is independent of muscle length or limb position.

2) As an increase in gamma activity dependent upon afferent fiber discharge.

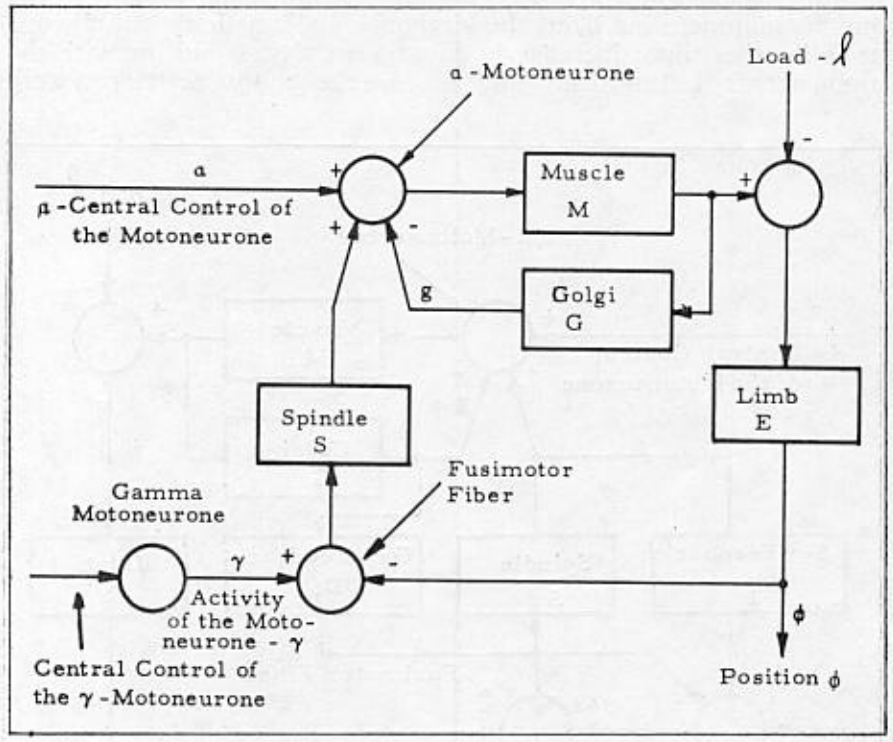

Fig. 1.

Figure 1 shows a simplified block diagram of the main neuromuscular loops. If the symbols within the blocks represent transfer functions, the limb position Φ depends on the supraspinal alpha

innervation, the supraspinal gamma innervation, and the load. Using the symbols from Figure 1, it can be seen that:

$$\Phi = \alpha \frac{ME}{1+GM+SME} + \gamma \frac{SME}{1+GM+SME} - l \frac{E(1+GM)}{1+GM+SME} \quad (1)$$

which simply tells us, that if for whatever reason the gamma activity increases, the output angle Φ will increase as well, assuming that no changes in load or supraspinal alpha-activation occur.

This model thus describes spasticity as a result of increased gamma discharge acting as an independent input signal to the neural control system.

A different viewpoint on spasticity can be taken however, if the assumption is made that spasticity is the result of an unstable non-linear feedback loop. Such a model would require that a connection exists from the output of the neuromuscular system to the gamma motoneurone. Thus the mechanism of spasticity would work as follows: a slight increase in gamma activity would increase the alpha activity but in turn would increase the gamma activity as well,

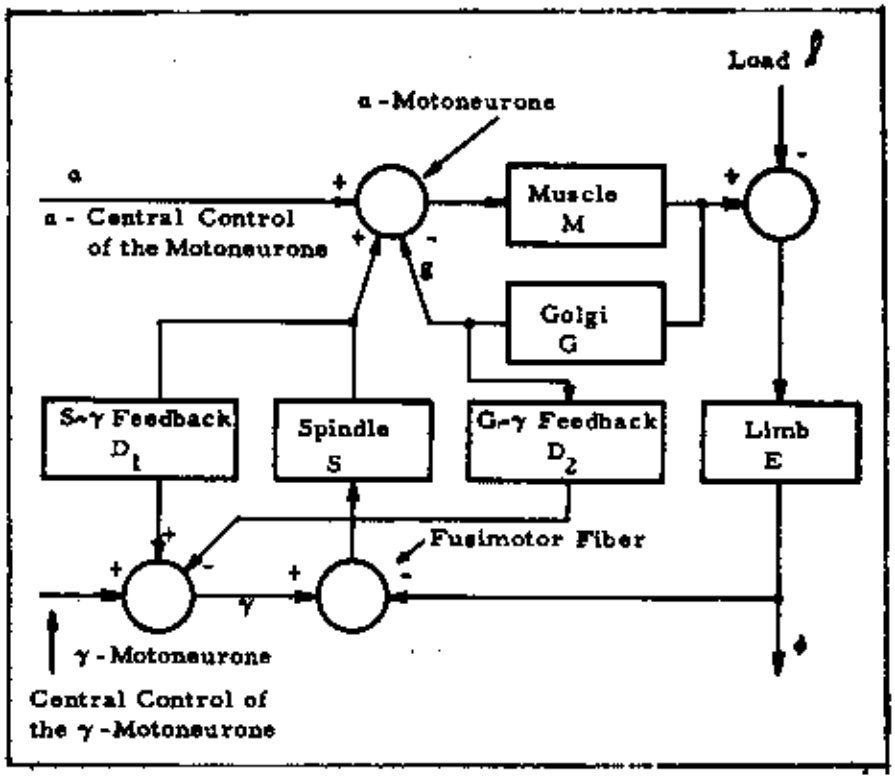

Fig. 2.

which would further increase spindle activity, etc. Although most workers question such autogenetic effects on the gamma motor system, some neurophysiological support for such an assumption can be found in Voorhove and Van Kanten [15]. Figure 2 shows a block diagram based on such a feedback system from afferent fibers to the gamma motoneuron.

Since spasticity is due to an increase in spindle afferent discharge, the facilitatory input (D_1) must predominate over the inhibitory (D_2). The latter may be important in the inhibition of organs (inhibitory) fire at higher thresholds of tension.

Transfer Function of a Spastic Extremity

In the following section we shall try to develop the relation between output angle and load or external force from two viewpoints. First, use will be made of the fact that clinical observation of a spastic extremity shows an increased resistance to stretch. This resistance is proportional to position and velocity [16, 17, 18].

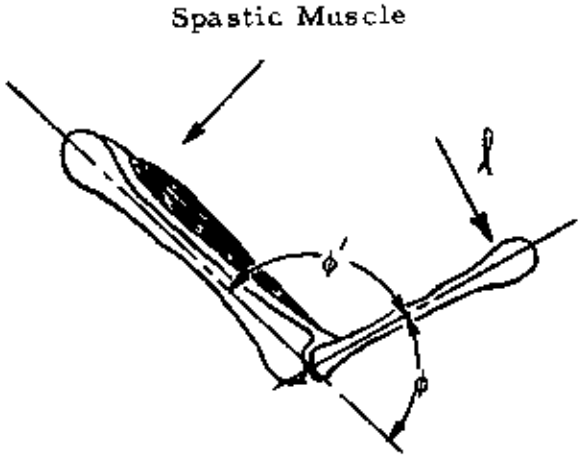

Fig. 3.

Since the extremity has a mass or moment of inertia, the force will be proportional to acceleration as well, (Fig. 3). Therefore,

$$I = K_1 \Phi' + K_2 \frac{d\Phi'}{dt} + K_3 \frac{d\Phi'}{dt^2} \tag{2}$$

On the other hand, from Equation 1 a relation between I and Φ can be derived.

The following questions can now be asked: if in a spastic extremity the resistance to movement increases with position and velocity, what changes in the transfer function (Equation 1) should be expected to account for the difference in resistance to movement compared with the normal extremity?

Suppose, that during the manoeuvre of passive extremity movements, the alpha and gamma central signals do not change. The position would then (according to Equation 1) depend only on the external load,

$$\Phi = -l\,\frac{E(1+GM)}{1+GM+SME} \tag{3}$$

Approximations for transfer functions shown in the blocks of Figure 1 are as follows [19]:

$$M = M_0 = \text{Constant} \tag{4}$$

$$G = G_0 = \text{Constant} \tag{5}$$

$$E = \frac{E_0}{s\,(as+b)} \tag{6}$$

$$S = \frac{cs+d}{es+f} \tag{7}$$

Since Equation 2 is valid in a range of movement where the high-threshold Golgi organs do not fire (before the "clasp-knife" region), the influence of the tendon organs is neglected. This changes Equation 3 to:

$$-\frac{l}{\Phi} = \frac{1+SME}{E} \tag{8}$$

On the other hand, if the Laplace transform of Equation 2 is taken, we get,

$$l(s) = -K_1\,\Phi(s) - K_2\,s\,\Phi(s) - K_3\,s^2\,\Phi(s) \tag{9}$$

and after rearranging

$$-\frac{l}{\Phi} = K_1 + s\,K_2 + s^2\,K_3 \tag{10}$$

Thus, the parameters for the transfer function of a spastic extremity can be determined by equating Equations 8 and 10 i.e., comparing expressions from the neurological control system and from clinical findings:

$$\frac{1+SME}{E} = K_1 + s\,K_2 + s^2\,K_3 \tag{11}$$

If Equations 4, 5, 6, and 7 are inserted into Equation 11 we have:

$$\frac{s_0 ea + s^2(eb + af) + s(bf + cM_0E_0) + dM_0E_0}{E_0 f} = K_1 + K_2 s + K_3 s^2 \quad (12)$$

Let us now introduce another simplification. According to some authors [20] the constant e in Equation 7 may be neglected. This simplifies Equation 12 to:

$$s^2 \frac{a}{E_0} + s\left(\frac{b}{E_0} + \frac{cM_0}{f}\right) + \frac{dM_0}{f} = K_3 s^2 + K_2 s + K_1 \quad (13)$$

Comparing coefficients we obtain:

$$\frac{a}{E_0} = K_3 \quad (14)$$

$$\frac{b}{E_0} + \frac{cM_0}{f} = K_2 \quad (15)$$

$$\frac{dM_0}{f} = K_1 \quad (16)$$

The constants K_1, K_2 and K_3 can be obtained by measurement and by using the above equations, a better insight into the influence of different parameters within the loop can be obtained. In a normal case the muscle resistance to passive movement is negligible compared to a spastic extremity. In spasticity therefore, K_1 and K_2 are increased. This means, looking at Equations 15 and 16, that d/f and c f have to increase. Thus both the static and dynamic component of the spindle transfer function have to increase during spasticity, which is in agreement with the increased gamma activity in a spastic neuromuscular loop. From Equation 15 it can be seen that for an increased K_2 the viscous component b in the extremity could be responsible. But spasticity usually develops too quickly in a limb to be due to organic changes in the tissue with alterations in its viscous properties. Contractures do occur as later events if the limb is not mobilized. Therefore we maintain, that increased K_1 and K_2 are mainly due to increased spindle sensitivity.

Another proof for increased d/f and c/f in spasticity is the equation which shows the response due to a tendon tap. [34] Without going into details at this point, it can be shown, that the amplitude in the myotatic response is proportional to:

$$af\sqrt{\left(\frac{b}{a} + \frac{cM_0E_0}{af}\right)^2 - \frac{4dM_0E_0}{af}} \quad (17)$$

which shows that both spindle constants cause an increased reflex response, but that the static component d/f probably has a greater influence. This is in keeping with the clinical observation that hyperactive reflexes and clonus have an optimal position at which they are most easily elicited. Equation 17 indicates that this optimum would be the maximal length, but we must not forget that the influence of tendon organs has been neglected in our mathematical derivations. In fact, however, at maximal muscle length the Golgi organs play an important role in the neuromuscular loops and Equation 17 therefore looses its validity.

Compensation of Spasticity by Electrical Stimulation

In spite of the fact that many physiatrists would even today consider electrical stimulation of a spastic limb as contraindicated or at least controversial, the first attempts to use electricity as a therapeutic agent in spastic conditions can be traced backed to Duchenne [21].

Since then, many workers have treated spacticity with electrical currents either by stimulating the spastic muscle or its antagonist with different types of currents. [22, 29] All authors obtained some degree of relaxation irrespective of their specific method. Not much knowledge exists at present regarding the basic mechanisms which cause the therapeutic effects. Therefore a hypothesis is forwarded regarding treatment of spasticity with electrical stimulation.

To understand the process of therapeutic stimulation, the two following questions should be answered:

1) Why do electrical currents relax a spastic muscle?

2) What is the mechanism of long-term effects of therapeutic stimulation?

On the second question speculations can be made using different concepts ranging from post-tetanic potentiation to learning, conditioning, and memory [30, 31, 32]. In this paper we shall concentrate on the first question which deals with the short-term effect stimulation, leaving the discussion of the second question to another report.

Our hypothesis, dealing with the short-term effects of stimulation, requires two assumptions:

a) A nonlinear characteristic exists between input and output frequency (or other signal-parameter of interneurones and receptors).

b) Excitation and/or inhibition can occur at the alpha motoneuron pool depending upon the intensity, duration, frequency and site of stimulation .

The reasons for the saturation characteristic can be refractoriness [33], polysynaptic delays, and logarithmic or square root relations in receptors. In general, as long as the relation,

$$\frac{f_{n+1}-f_n}{X_{n+1}-X_n} < \frac{f_n-f_{n-1}}{X_n-X_{n-1}} \tag{18}$$

is valid (Fig. 4) a simplified three-neuron network model (Fig. 5) can explain the resulting inhibition occurring in stimulated spastic extremities.

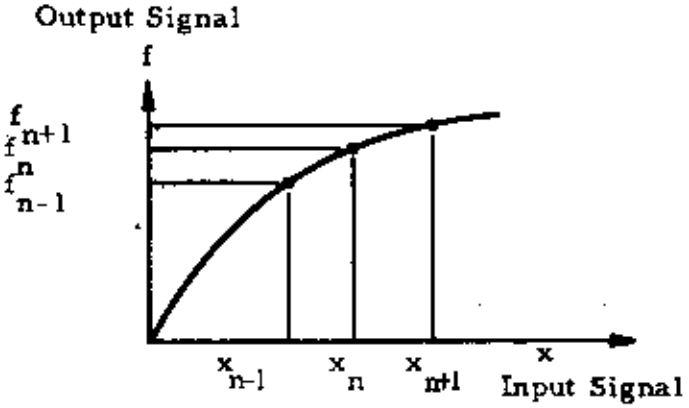

Fig. 4.

In the normal case X_1 and X_2 are relatively low and the neurons operate in the almost linear part of their characteristics (points 1 and 1' on all curves in Figure 5). If spasticity is present, we may assume two possibilities: a) increased excitation or b) diminished inhibition. Let us first discuss the situation where spasticity is due to increased overall excitation of the alpha-motoneuron (N_3 in Figure 5). In this case the excitatory input is rather high whereas the inhibition remains unchanged. Since we assume that the resulting equivalent input frequency to N_3 is the difference between the excitatory and inhibitory input frequency, the output frequency of N_3 is increased (points 2 on curves a_1, a_2 and a_3). If the spastic extremity is stimulated, our assumption requires that excitation and inhibition increase, since stimulation can activate different receptors and nerves on the agonistic and antagonistic side of the extremity. In Figure 5, X_1 and X_2 increase, but f_1 can increase less than f_2 and therefore f_3 decreases (point 3 on curves a_1, a_2 and a_3) and the spastic muscle relaxes.

The same model can be used if the reason for spasticity is decreased inhibition. This situation is presented by curves a_1', a_2'

and a_3'. In the normal case the relations are represented by points 1'. In spasticity X_2 decreases but excitation remains unchanged and an increased alpha-motoneuron frequency f_3 results. With stimul-

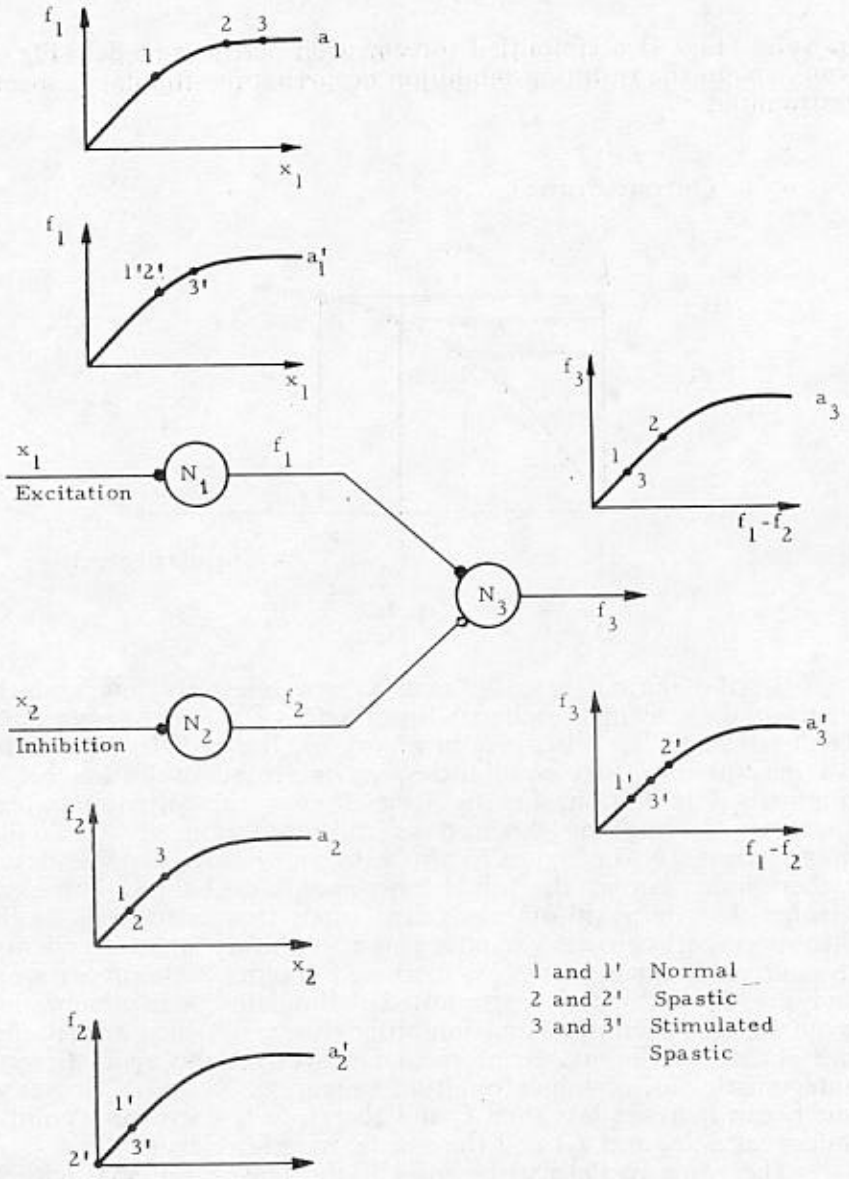

Fig. 5.

ation, X_1 and X_2 increase, but due to the non-linear characteristic the inhibitory frequency f_2 increases more than the excitatory frequency f_1. Thus, f_R decreases and relaxation is obtained.

We do not intend to demonstrate all possible excitatory and inhibitory situations which are implicit in the simplified model described above. Let us therefore examine just two possibilities. Using a block diagram (Fig. 6) obtained as an extension of Figure 1, assume there is spasticity in the flexor and relaxation is obtained by stimulating the extensor, a situation described, for example, by

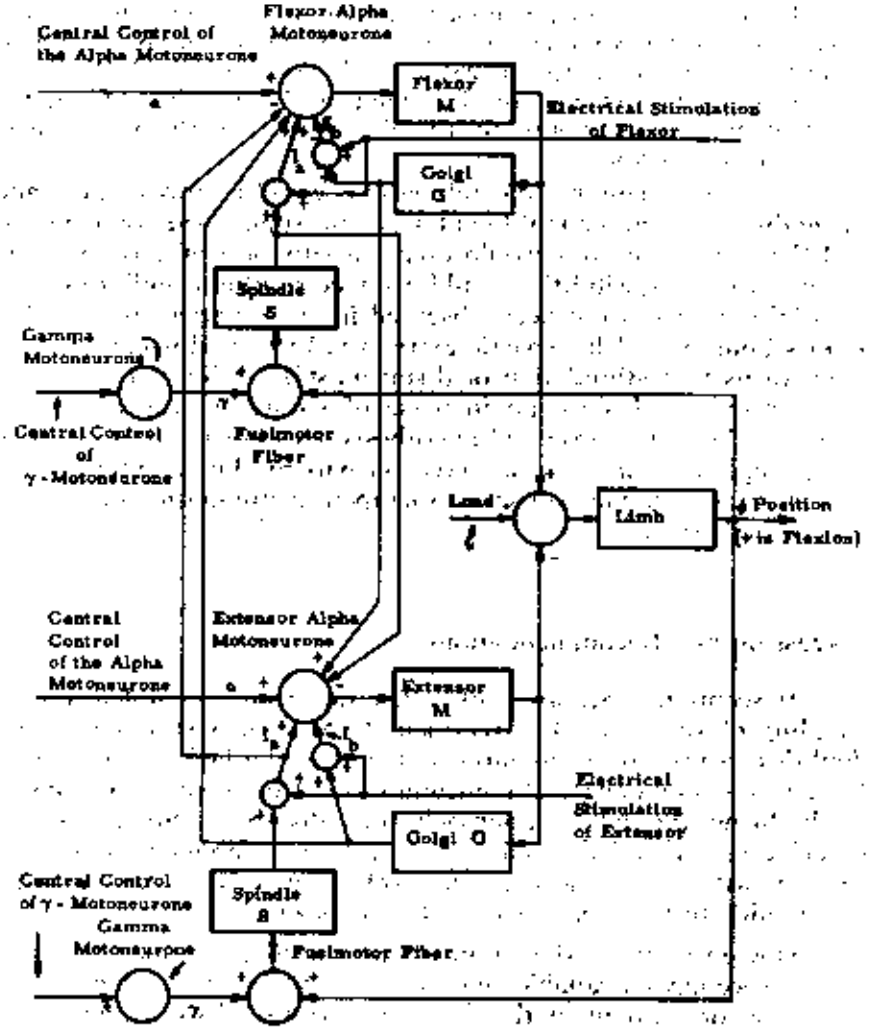

Fig. 6.

Levine et al [24]. Let us suppose that no supraspinal influences are present (spinal transection). Since the lexor spindles are hypersensitive, the flexor Ia fibers are firing at a high rate. When the extensor is stimulated the Ia fiber activity of the flexor might still increase slightly (the flexor is stretched). Now, since the flexor Ia fiber's frequency was close to maximum before, the increase due to stimulation is smaller than the increase in the Ia extensor fibers (due to direct nerve stimulation). Before stimulation this input produced only a negligible inhibition on the flexor motoneurone. Thus the Ia extensor fibers efficiently inhibit the flexor motoneuron and cause relaxation.

If the spastic flexor muscle is stimulated, the therapeutic effect can also be explained. The increase in Ia activity of the flexor is small compared to the increase in Ib autogenic inhibition and due to a possible stretch of the extensor, Ia inhibition from the extensor might contribute to the decrease of alpha-motoneuron activity of the flexor.

Of course many more combinations could be investigated and the addition of supraspinal facilitation and suppression on extensor and flexor motoneurons would create a situation of extreme complexity. Not all possibilities would produce resultant inhibition. But then inhibition has not been observed in every patient who underwent stimulation of his spastic extremity. In fact, Hufschmidt [26] even warns that stimulation of distal muscles in lower extremities is contraindicated since an increase of spasticity was frequently observed. Thus, the proposed hypothesis does not intend to explain every single phenomenon observed on patients but it offers a way to understand the mechanism of therapeutic stimulation on a more generalized basis.

Discussion and Recommendations

It seems that we are still rather far from a complete understanding of spasticity. Its treatment by electrical stimulation has doubtless a promising potential but due to the lack of knowledge there is always a danger of contraindicated treatments, which could easily bring the whole approach into disrepute. Some authors describe very detailed "cook-book" procedures about their way of therapy without any attempt to explain or justify their recipes, which adds to the scepticism of their colleagues.

Obviously then, there is no unique recommendation for therapeutic procedures available and only a few institutions presently treat spastic patient with elecrical currents. Two avenues for further work are possible:

1) Continue basic neurophysiological research until enough progress is made so that thorough understanding of any treatment will be available.

2) Look for a reasonable, rational and cautious compromise where treatment should be attempted even if not all details of it are understood.

We believe, that the second path should be taken but maximum use should be made fo present basic knowledge and the treatment methods modifiel according to recent neurophysiological findings.

Since we do not know what an optimum stimulation current would be and since it is highly probable that large variations exist for different clinical cases a statistical approach to therapeutic stimulation is proposed. Many different stimulation waveforms can be stored on a tape recorder and can be played back through an output unit to the spastic muscle and its antagonist or even more muscles. The "performance" of each stimulation current is monitored as myoelectric activity from the spastic muscle. The data are fed into a computer which makes decisions regarding the next stimulation waveform attempting to minimize the EMG in the spastic muscle. Through trial and error and applying a self-optimizing policy programmed into the computer, the system is expected to coast towards an optimum current for a given patient. With this current the patient could then be treated regularly and the therapeutic effect ascertained over a longer period of time.

There is no evidence that electrical stimulation would relieve spasticity permanently. It is therefore proposed that miniature stimulators with characteristics obtained from the method described above should be given to patients for home use. Patients could thus treat themselves or be treated by their relatives regularly and go to their physician only for regular check-ups. A further extension of these thoughts would be the use of the stimulator on a permanent basis. The patient would normally wear the stimulator with him (perhaps even surgically implanted) and could switch it on according to demands.

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