LONG TERM EFFECTS OF FUNCTIONAL ELECTRICAL STIMULATION

L. Vodovnik, F. Gračanin, S. Reberšek, and M. Štefančič

Introduction

Paralysis of extremities in hemiplegic patients are caused by an absence of facilitatory and inhibitory supraspinal control signals which normally activate the spinal motor neurons /1/. The lack of facilitatory signals causes paresis or paralysis - mostly in the extensors, whereas the lack of inhibitory signals causes increased reflex responses, hypertonus, etc. These phenomena are mostly observed in the antagonists of the paralyzed muscle, i.e. the flexors.

In order to improve the organization of motor activity, several methods are used at present. All of them apply mechanical stimuli (including passive movements) which should modify the damaged motor mechanisms at different levels of the CNS /2, 3, 4/.

With the introduction of FESE (Functional electrical stimulation of extremities), in addition to the direct motor response several effects were observed which show that the organisation of the CNS can be influenced also by means of FESE.

The direct motor response (efferent stimulation) leads to a new approach in orthotics and a survey of this area can be found in /5/.

Effects on the CNS which are due to afferent signals have been studied extensively by Gračanin et al, who observed, that due to FESE, a facilitation of voluntary movements can be obtained together with an inhibition in antagonists /6/. The pattern of reciprocal innervation could be restored and improved voluntary control of stimulated and even not stimulated muscles of the extremity was obtained /7/. These and some other phenomena can be explained with the influence of FESE on the motor mechanisms at the apinal level /8/.

Several observations by different workers were made however, which lead to the conclusion that during FESE long-term information processing must have been going on in the higher levels of the CNS. These observations were summarized and some digital models to explain the phenomena were presented by Vodovnik /9/.

The work reported in this paper was supported in part by the Slovene Research Community - Foundation "Boris Kidrič" and by the Department of Health, Education and Welfare, Social and Rehabilitation Service, Washington D.C. under Grant No. 19-P-58415-F-01.

In the present paper we shall describe some experimental results regarding the long-term effects of FESE and attempt to explain them by means of an analogue model.

The Cybernetic Model

The basic assumption for the model construction is that repeated tetanic stimulation of the peroneal nerve with an amplitude sufficient to cause a direct motor response (efferent stimulation) results in a restoration or improvement of voluntary control over the muscles groups which produce dorsiflexion of the ankle. The selection of the ankle joint is, of course, only a specific example within a more general framework which should be offered by any useful model.

The block diagram on Figure 1 shows the basic systems involved in the motor control of man. The signals on the figure are denoted as vectors since each of the signals may have more components. The desire for voluntary movement originates in the cortex and is transmitted as a complex nervous signal $\bar{v}$ through the subcortical structures, the spinal cord, and the peripheral nerve to the muscular skeletal system. An output vector $\bar{x}$ results which can represent biomechanical (force, position) or bioelectrical (EMG) magnitudes. The receptors "measure" various parameters of $\bar{x}$ and the feedback signal $\bar{a}_1$ is sent to the central nervous system.

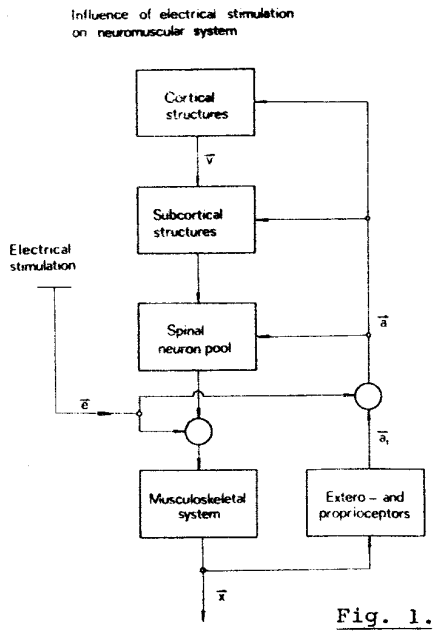


Fig. 1.

Let us suppose now that electrical stimulation $\bar{e}$ is applied to the peripheral nerve. If antidromic effects are neglected, the influence of $\bar{e}$ is threefold. First, there is a direct effect on the muscle through the α-nerve fibers (efferent stimulation). Secondly the sensory fibers can also be directly excited by $\bar{e}$. And finally since $\bar{e}$ influences $\bar{x}$, the afferent output from the receptors $\bar{a}_1$ is modified by $\bar{e}$. Therefore $\bar{a}$ is changed by $\bar{e}$ in a rather complex way and the interesting phenomena we observed during our experiments are at least partly due to this interplay of afferent signals and signals due to electrical stimulation.

It is supposed, that any long-term effects of FES are due to changes in the subcortical and spinal structures. Therefore in our model the most important signals are $\bar{v}$ and $\bar{a}$ where $\bar{a}$ is closely related to $\bar{e}$.

Factors which are thus crucial in the changes of $\bar{x}$ after brain haemmorage or trauma are:

- intensity of voluntary effort $\bar{v}$
- intensity of electrical stimulation $\bar{e}$ and its correlate $\bar{a}$
- state of the CNS
- state of the musculo-skeletal system.

The state of the CNS is influenced by $\bar{a}$ in two ways. Due to electrical stimulation some facilitation of the volitional pathways occurs and since these effects are of a duration which exceeds the stimulation time, we speak of learning and long-term effects. On the other hand stimulation can produce also some inhibitory effects which might be identified as central fatigue, habituation, etc.

These two antagonistic effects influence $\bar{v}$ in a rather complex way and the phenomena which were observed should be interpreted also in terms of facilitation in inhibiting activities in the CNS.

The state of the musculo-skeletal system depends on its biomechanical properties which are changed through repeated contractions of the muscle fibers. The most evident effect due to contractions is muscle fatigue.

Based on these assumptions, Figure 2 was developed. For reasons of simplicity all vector signs are dropped from the signals.

The volitional desire for movement is repersented by the signal v. The block W symbolized the neural network which is partially damaged. Modifications of the "conductivity" of W are achieved by the signal m. The effects of learning and central fatigue are represented in blocks G_1 and G_2. The outputs of these blocks are g_1 and g_2. Let us now assume that linear systems theory may be applied to our pro-

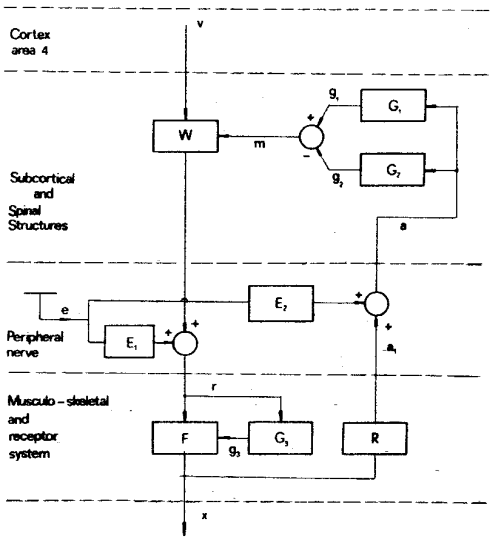


Fig. 2. Block diagram indicating possible influence of FES on subcortical and spinal structures.

blem. We are fully aware of the crude simplification such an assumption is introducing, but for an initial investigation and in order to get an idea about the required mathematical tools, such a simplified approach seems justified.

It is essential that $g_1(t)$ and $g_2(t)$ are not only functions of $a(t)$, but also of the history of a, since just this history is responsible for the learning and fatigue effects which were observed during experiments. Therefore the signals g_1 and g_2 may be expressed in terms of the convolution integral

$$g_1(t) = g_{10} \int_0^t h_1(t-\tau)a(\tau)d\tau \qquad (1)$$

$$g_2(t) = g_{20} \int_0^t h_2(t-\tau)a(\tau)d\tau \qquad (2)$$

In the above integrals g_{10} and g_{20} are constants, where as h_1 and h_2 are pulse responses of the systems G_1 and G_2.

In an analogous way the input signal to the muscle r modifies the musculo-skeletal system F. Since the effects we are interested in have time scales in the orders of tens of seconds, minutes, and more, the dynamics of F may be neglected. Thus F may be described as

$$F = F_o - ng_3 \tag{3}$$

Here F_o means the initial transfer function of the muscle, n is a constant and g_3 represents the effects of muscle fatigue. Therefore

$$g_3(t) = g_{30} \int_0^t h_3(t-\tau)r(\tau)d\tau \tag{4}$$

The blocks E_1 and E_2 represent the transformation of stimulation magnitude to the equivalent signals of nerve.

As mentioned before, block W is representative for the conduction capabilities of v to the lower motor neuron. If N_n is the number of normal neurons and N_d the number of distroyed ones, then

$$W = K \frac{N_n - N_d}{N_n} \tag{5}$$

For $N_d = 0$ we deal with a normal subject whereas $N_d = N_n$ would mean total paralysis. It is expected that with "a" (which is strongly correlated to electrical stimulation) mostly the destroyed cells can be influenced and it is expected that their number can be reduced with increased m. This would mean that electrical stimulation could help to restore the damaged cells or their effect - perhaps by activating new, and unused synaptic connections or new neurons.

Let us suppose that the number of damaged cells can be reduced with increased m according to the relation

$$N_d = N_{do} - km \tag{6}$$

where N_{do} is the number of damaged cells when no electrical stimulation was applied. If Equation 6 is introduced into Equation 5 the following expression is obtained,

$$W = K\left[1 - \frac{N_{do}}{N_n} + \frac{km}{N_n}\right] \tag{7}$$

The conductivity of the subcortical and spinal neural nets increases with increased m. From Figure 2 it is evident, that

$$m = g_1(t) - g_2(t) \tag{8}$$

Therefore

$$W = K\left[1 - \frac{N_{do}}{N_n} + \frac{kg_1(t)}{N_n} - \frac{kg_2(t)}{N_n}\right] \tag{9}$$

With these equations and Figure 2 we might attempt to write the relation between v, e and x.

$$(vW + E_1 e)F = x \tag{10}$$

Introducing Equations 1, 2, 3, 4, and 9 into Equation 10 the relation between output x, stimulation e, and voluntary control v is obtained

$$x = \{M_o - ng_{30} \int_0^t h_3(t-\tau)r(\tau)d\tau\} \cdot \{E_1 e + vK\left(1 - \frac{N_{do}}{N_n}\right) +$$

$$+ v \frac{Kk}{N_n} \left(g_{10} \int_0^t h_1(t-\tau)a(\tau)d\tau - g_{20} \int_0^t h_2(t-\tau)a(\tau)d\tau\right)\} \tag{11}$$

The first bracket is an analytical expression for muscle fatigue. The first term in the second bracket represents the immediate effect of electrical stimulation on x (efferent stimulation). The second term shows to what extent there exists voluntary control over the paretic muscle. This term can increase (with maximal v) only by decreasing N_{do} - for example, by means of spontaneous recovery. The third and fourth terms show the effect of stimulation upon the voluntary control. The third term causes an improvement of voluntary control, whereas the last term decreases that effect due to central fatigue and habituation. In the following section specific experimental data will be presented and attempts will be made to fit the results of measurements with the hypothesis proposed in the foregoing paragraphs.

Patients and Methodology

The study included six patients with hemiplegia after cerebrovascular diceases. All patients were hemiplegics for at least one year. Their muscle force for M. tibialis anterior was reduced from normally 5 to levels between -2 and -4. The Ross test modified by Gračanin /8/ of motoric functions for the lower extremity gave scores between 10 and 16 (of maximally 25). There was an increased heel-tendon reflex with tendencies to clonus.

The expected long-term effects were measured on the dorsal flexors of the affected foot. The patients chosen for measurements had stopped using electrical stimulation a week before in order to eliminate the eventual long-term effect of former electrical stimulation. Every experiment included four phases of measurement. First we measured the voluntary moment before the stimulation. After that the pa-

tients walked for 20 minutes using the Ljubljana FEPB. Immediately after the stimulation the voluntary moment was measured again the same measurements were repeated half an hour and one hour after stimulation. Simultaneously with the measurements of the moment the EMG activity of the M. tibialis was recorded. Every measurement of the moment consisted of 10 - 15 contractions, their duration being 3 s, and the rest interval, 3 s. The order for both the contraction and the pause was given by use of a light controlled by a pulse generator. When this light was turned on the patient was told to contract the dorsal flexors as much as he could, and when the light was off, to relax them. All the measurements were done with the patient in a sitting position. The affected leg was fastened into an ankle brace and the isometric moment measured by means of strain gauges. The patient could watch the moment's value, which was shown by an instrument on the same display panel where the light for the contraction order was located. The whole measuring equipment is shown in Figure 3. At the beginning of every measurement the experimentor turned on the switch V_1 - V_2. Therefore the lamp V_o started to light periodically and the muscle contraction resulted. At the same time contact V_2 activated the marker channel of the recorder and thus indication was obtained of the command for voluntary contraction. The moment and EMG activity were recorded on two separate recording channels. The instrument on the display panel indicated the obtained moment to the patient and provided him with visual feedback to check his performance. In the rest periods between measurements the patients remained in a sitting position.

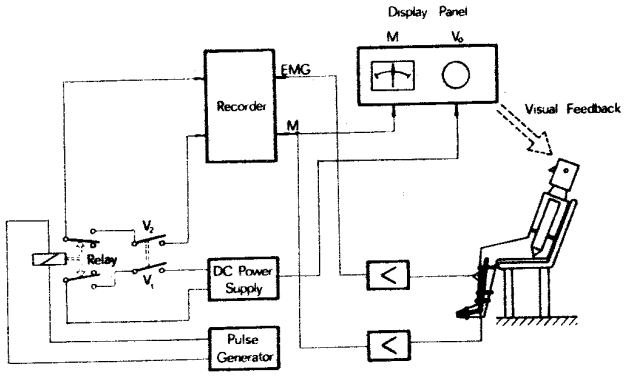


Fig. 3.

Preliminary Results and Discussion

A survey of the obtained results is given in Table 1. The mark "x" indicates that due to failure in equipment, the measurement was not carried out.

Table 1.

Patient:	Isometric moment of ankle joint [Nm]			
	before Stimulation	Immediately after Stimulation	Half an hour after Stimulation	one hour after Stimulation
Š. F.	5,5	4,9	7,1	7,3
I. T.	3,4	7,9	x	11,3
G. I.	3,7	6,8	8,3	9,7
R. I.	1,2	2,1	2,5	2,7
K. A.	4,1	4,6	6,8	7,6
K. I.	0,5	1	1,3	1,7

Figures 4 and 5 show the response of the voluntary moment measured at t = 20, 50, and 80 min for two patients. Each moment curve was integrated with a planimeter and the result divided by the time in which the moment was recorded. This result was multiplied by the appropriate coefficient which give the average values of the measurement. Two typical recordings are shown in Figure 6, namely the first two measurements, i.e. before and after the stimulation. Since the scales for the EMG and the moment are the same for both records the difference in moments and the EMG activities before and after stimulation is evident.

Let us try to discuss the measured data. Table 1 and Figures 4 and 5 clearly show the main characteristics of the results as follows:

1) With time the voluntary moment increases after stimulation. This phenomenon is rather unexpected but can be explained using the model proposed in the former section.
2) The voluntary moment is larger just after stimulation than before except in one case. This increase was expected and fits well with our basic assumption that FESE improves the voluntary control of paretic muscles. In the exceptional case the decrease of the moment immediately after stimulation to a smaller value than before stimulation and its la-

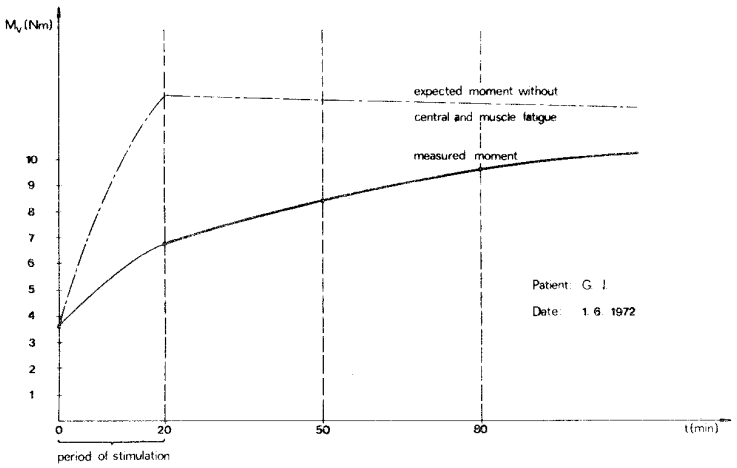

M_v (Nm)
expected moment without
central and muscle fatigue
measured moment
Patient: G. J.
Date: 1. 6. 1972
10
9
8
7
6
5
4
3
2
1
0
20
50
80
t(min)
period of stimulation

Fig. 4.

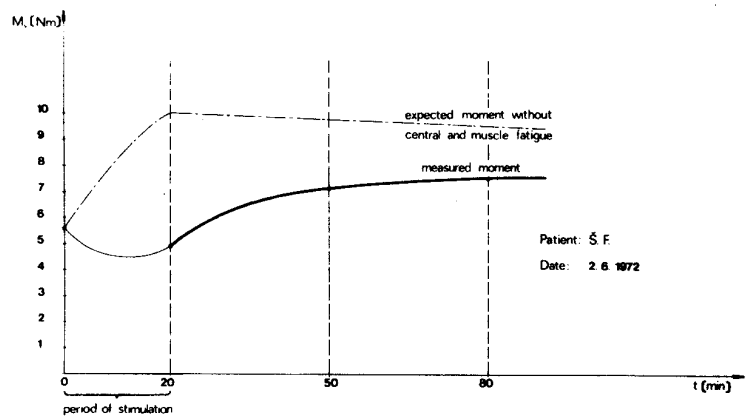
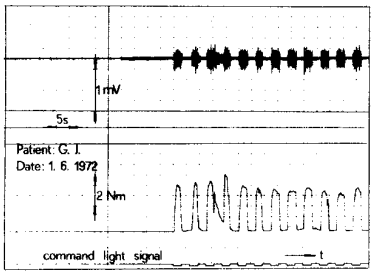

M_v (Nm)
expected moment without
central and muscle fatigue
measured moment
Patient: Š. F.
Date: 2. 6. 1972
10
9
8
7
6
5
4
3
2
1
0
20
50
80
t (min)
period of stimulation

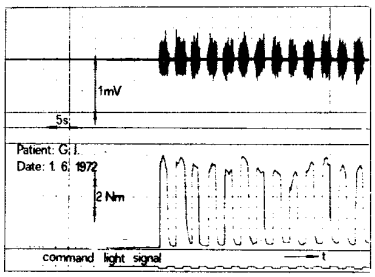
Fig. 5.

ter increase to values higher than at t = 0 and t = 20 min. seems to be more peculiar (Fig. 4). We shall show however, that all these results might be explained with the same model.

Let us now attempt to fit the obtained data to the proposed model. Looking at the curves fitted to the data on Figures 4 and 5, and knowing that many biological phenomena behave according to exponential curves, we suppose that solutions of the convolution integrals are exponential functions. We are fully aware of the shortcomings of our measurements since not enough data were collected. This happened mostly since we did not know exactly what would be the essential time scales for the various phenomena. Still it seems that our approximations with exponential functions are quite useful.



Voluntary moment and EMG activity before stimulation



Voluntary moment and EMG activity immediately after stimulation

Fig. 6.

We suppose therefore that the solutions of the convolution integrals are functions of the type

$$y = Y_o \, e^{-\frac{t}{T}} \tag{12}$$

where t = 0 begins at the end of the 20 minute stimulation, i.e. t = 0 in Equation 12 is equivalent to t = 20 min. in Figures 4 and 5.

Instead of the general output x in Figures 1 and 2 in our experiments specifically the moment M has been measured. Since the measurements where performed only when no stimulation e was applied, Equation 11 can be written for the post-stimulation period in the following form:

$$M = v\left[F_o - A\, e^{-\frac{t}{T}}\right]\left[B + C\, e^{-\frac{t}{T}} - D\, e^{-\frac{t}{T}}\right] \tag{13}$$

where A, B, C and D are constants,

$$A = n g_{30}$$

$$B = K\left[1 - \frac{N_{do}}{N_n}\right]$$

$$C = \frac{Kk}{N_n} g_{10}$$

$$D = \frac{Kk}{N_n} g_{20}$$

The developed torque M is thus proportional to the voluntary effort v, the biomechanical and biochemical properties of the muscle, and to a modifying factor in the CNS. Since practically none of the above magnitudes are amenable to direct measurements we are rather free in choosing our constants. A few guidelines however can be obtained from the experimental results. The initial moment before stimulation is $v\, F_o B$ and the same level has to be reached for $t \to \infty$. Equation 13 satisfies this condition. The curves on Figures 4 and 5, however, do not return within 1 hour to the initial value of the torque, which only means that the measurements were not taken over enough time intervals. Former experiments on muscle fatigue show that T_1 has its order of magnitude in minutes, whereas T_2 and T_3 are much longer since the long term-effects were observed to last over periods exceeding one hour. The slow increase of M after stimulation is thus interpreted as a result of an intricate interplay of three phenomena, each with its own time constant and its own magnitude. Now it is not sur-

prising any more that after stimulation M increases. Such a phenomenon is the result of a relatively fast decrease of both "fatigue" terms compared to the decrease of the learning term. The fact that M was just after stimulation smaller than before stimulation (Fig. 5) could be explained with a large amount of muscle fatigue. Since $T_1 \ll T_2, T_3$ there is a fast muscle recovery and the torque starts to increase and even exceeds the M before stimulation.

By a proper combination of the three exponential functions in Equation 13 practically every experimental curve can be fitted adequately. Further work will be directed to a more detailed separation and quantative identification of the three main phenomena which influence M.

Conclusion

Former work has shown that FESE can influence the organisation of the CNS on a short term basis.

From clinical observations it was known that after FESE the voluntary force is improved for longer periods of time (hours and more). Therefore an attempt has been made to verify and explain the long-term effects of FESE on a more quantitative basis.

Experiments have shown that this effect was present in all six hemiplegic patients which were included in the study. It was found that the moment increases after 20 minutes of FESE for at least one hour.

A mathematical model was proposed which is capable of explaining the results. It consists basically of three exponential functions which represent muscle fatigue, learning in the CNS and "fatigue" in the CNS.

The long-term effects are of such an order of time-magnitude that they can not be ascribed to changes in the known mechanism of the spinal cord level but must occur at supraspinal levels.

The results obtained have important clinical implications. They lead to the conclusion that FESE is not only a modern approach in orthotics but that it will also develop into a useful therapeutic method for the treatment of CVA patients.

References

/1/ Ruch, C.T., "The Cerebral Cortex: Its Structure and Motor Functions", *Neurophysiology* (Ruch, C.T. et al.), ed. W.B. Saunders and Co., Philadelphia, Sec.ed.repr. 1968, pp. 274-276.

/2/ Knott, M., and Voss, D.E., *Proprioceptive Neuromuscular Facilitation*, sec.ed. Harper et Row, Publ. New York, 1969.

/3/ Rood, M.S., The Use of Sensory Receptors to Activate, Facilitate and Inhibit Motor Response. Automatic and Somatic in Developmental Sequence. Approaches to the Treatment of Patients with Neuro muscular Dysfunction, W.C. Brown Co. Dubuüe, Iowa, 1962, pp. 26-37.

/4/ Bobath, K. and Bobath, B., "Control of Motor Function in the Treatment of Cerebral Palsy", *Physiotherapy*, Vol. 43, 1957. pp. 295-303.

/5/ Vodovnik, L., "Functional Electrical Stimulation of Extremities", *Advances in Electronics and Electron Physics*, Vol. 30, 1971, pp. 283-297.

/6/ Gračanin, F., Dimitrijević, M.R., "Analysis of Spinal Reflex Activity During and After Tetanic Atimulation of the Peroneal Nerve in Hemiplegic Patients", 5th International Congress of Physical Medicine, Montreal, August 25-31, 1968.

/7/ Štefančič, M., Gračanin, F., "Changes in the Kinematics of Walking of Patients with Hemiplegia After Conditioning of Spinal Reflex Mechanisms" (in Slovene), *1st Congress for Physical Medicine and Rehabilitation of Yugoslavia*, Belgrade, June 4-6, 1972.

/8/ Gračanin, F., "Study of the Influence of Functional Stimulation on the Reflex Motor Activity in Patients with Hemiplegia" (in Slovene), D. Sc. Dissertation, University of Ljubljana, 1971.

/9/ Vodovnik, L., "Information Processing in the Central Nervous System During Functional Electrical Stimulation", *Med. & Biol. Engng.*, Vol. 9, 1971, pp. 675-682.