ELECTROTHERAPY OF SPASTIC MUSCLES IN HEMIPLEGIA

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Abstract

After a short review of existing data about the influence of the therapeutic electrical stimulation on spasticity an experimental set-up for measurement and treatment of spasticity is described. The degree of spasticity is detected through the measurement of resistive torque due to the passive movement of ankle joint. During the therapy procedure the ankle is passively moved with the help of electrohydraulic servo system while the muscle, which is momentarily stretched, is also tetanically stimulated. The data obtained experimentally are evaluated on the basis of calculation of the muscle force response. For the calculation of the muscle forces a linear mathematical ankle joint model is used. Some interesting phenomena and future plans are discussed at the end of the paper.

Introduction

The use of electrical stimulation to relieve spasticity can be traced back as far as 1871 when Duchenne /1/ employed stimulation of the antagonist to relieve spasticity, although at that time he could not be aware of the rationale of such an approach. Unfortunately, more than one hundred years after Duchenne, there still does not seem to be a well documented rationale for stimulating spastic extremities. The lack of solid neurophysiological foundations for electrotherapy has brought the field in disrepute. A typical example reflecting the status of electrical stimulation in spasticity is a recent review of spasticity /3/ which does not even mention electrical stimulation as a possible therapeutical agent.

Still, there are several clinical reports about beneficial effects after stimulating the spastic limb.

Lee et al. /3/ stimulated 27 spinal cord injury patients. They stimulated the spastic quadriceps with continuous faradic (60 - 100 Hz) or sinusoidal (60 - 350 Hz) currents for 15 minutes and observed relaxation for 0 to 20 hours thereafter. In a series of 19 patients who obtained only faradic stimulation all 19 patients showed relaxation of spasms after some or all the treatments. However, they had two patients in whom in several occasions no relaxation was observed following treatment and two patients reported an increase in spasm immediately following one or more treatments. They also noted a "spread of current to muscle groups other than those being treated". In 11 of the 19 patients an obvious spread of the impulses to the contralateral side was observed and after treating one leg it was not uncommon to find some relaxation in the other leg. In seven patients who retained some voluntary muscle control, coordination of grass movements was improved. No patient suffered

regression of voluntary movement, while in several, improvement was noted. Some side effects were reported. Voiding was observed in one patient and penile errections in three other patients. Comparisons between treatments with faradic and sinusoidal currents produced more relaxation and of a longer duration than faradic currents did. Levine et al. /4/ were stimulating the antagonists of spastic muscle in hemiplegic, paraplegic and MS patients with favorable results but no quantitative data were published.

Vogel et al. /5/ stimulated 20 patients with spinal cord lesion with a combination of direct and alternating current for 20 minutes daily. Ten patients with complete paraplegia experienced relief from spasticity for periods ranging from 3 to 24 hours following the treatment. All reported that they could carry on their activities of daily living with much greater ease and comfort. Five patients with incomplete paraplegia could not tolerate the high current intensities required for strong tetanic contractions. Still, they reported relief from spasticity for periods up to six hous. Five patients with MS could not tolerate the current and therefore the results were unsatisfactory. Similar to the observations by Lee, Vogel also found relief from spasticity on the contralateral limb in five of the ten cases of complete traumatic paraplegia. Hufschmidt /6/ published a report on 600 patients who were treated for spasticity by electrical stimulation. Spasticity was of a spinal or cerebral origin, the time after onset of lesion ranging from months to decades. Stimulation was either applied to the spastic muscle alone, or two channels of stimulation was alternately applied to the agonist and antagonists. Treatment durations were between 15 and 20 minutes. He observed relaxation of spasticity, inhibition of pathological flexor reflexes, activation of paretic antagonistic muscles and pain reduction. The therapeutic effects lasted 24 - 48 hours after the first session and up to several weeks when sessions were repeated. He claimed treatment with stimulation should not be administered daily but only every two to three days since the effects of treatment sometimes were first observable two to three days after stimulation. He also tried stimulation in cases of athetosis and ataxia again with encouraging results. In Hufschmidt's opinion, rythmic and coordinated electrical stimulation produced therapeutic results which were superior to results obtained with pharmacotherapy or general physiotherapy.

Other reports with mostly favorable results have been published by McNeal et al. /7/, Dimitrijević /8,9/ and Gračanin/10/ McNeal obtained relaxation of spasticity with implanted electrodes thus by passing skin receptors. Gračanin showed that not only static but also rhytmic forms of spasticity - like clonus - could be compensated by electrical stimulation.

Encouraging reports are also obtained from spinal cord stimulation /11,12/ where a diffuse barrage of stimuli applied to the dorsal column of the spinal cord produces various beneficial effects to motor disorders including relaxation of spasticity. The most recent work in this area has been done by Bowman and Bajd /13/. Their results on ten SCI patients do not reflect so much optimistic outlook as other investigations. Similar results were obtained in our preliminary experiments

where muscle tibialis anterior was stimulated to reduce the spasticity in soleus muscle. Tetanic electrical stimulation with sequences 1 s on and 1 s off were applied for 20 minutes, while patients were in sitting position. Resistive torque due to passive movements of ankle joint together with EMG of tibialis anterior and soleus muscles was recorded before and after the stimulation. The resistive torque was measured by means of air contained ballon connected to pressure transducer and inserted between foot and external electrohydraulically powered system which was able to produce passive movements. The long-term therapeutic effect of such a stimulation was noticed after a week long treatment. Data were collected from 5 patients all hemiplegic with the main spasticity in soleus muscles. We found out in these experiments that the spasticity can be reduced after 20 minutes of tetanic stimulation to one half of resistive torque, but not in all patients. This effect was more notieeable in the patients who had not been supplied with any electrical stimulation during the regulary rehabilitation process. The therapeutic effects were also not consistent. Already reduced spasticity could become greater in the following week. On the basis of these experiments we have concluded that the measurements and experimental procedure must be performed very carefuly and precisely. Since our existing electrohydraulic system and the torque meter did not have adequate performances, it was decided to construct a new electrohydraulic actuator were the torque is measured directly with a strain-gauge bridge.

Experimental set-up

In the measurement of spasticity represented by the resistive joint torque it is very important to design a measurement system with precise positioning and adequate frequency capabitilies. The new electrohidraulic servosystem shown in Fig. 1 is able to produce ankle joint movements of any shape of joint angle in frequency range from 0 - 10 Hz. The position of ankle is measured by means of a precise potenciometer and can be determined with accurancy of ± 0.5 degree. In the system built torque transducer is able to measure torque with accurancy of ± 0.05 Nm.

Such a kind of system for spasticity measurements is not new as similar systems were widely used before /14,15/. Unfortunately they did not overcome the experimental phase and are not conventionally available, although Gottlieb /14/ has stated in his paper that"such a system would be extremely simple to build and operate and would provide well-quantified reproducible information about patient status". In Ljubljana Rehabilitation Engineering Center such an electrohydraulic system has also been used for physical therapy of ankle joint of hemiplegic patients for more than two years. In comparison with the Gottlieb's method where he used constant torque system, we have constant - displacement although we can switch our system to constant torque operation.

As an additional observation of muscle activities during passive movements EMG recording of tibialis and soleus muscles is provided. The EMG activity is detected by surface silver-silver chloride disc electrodes (see Fig. 1). All signals

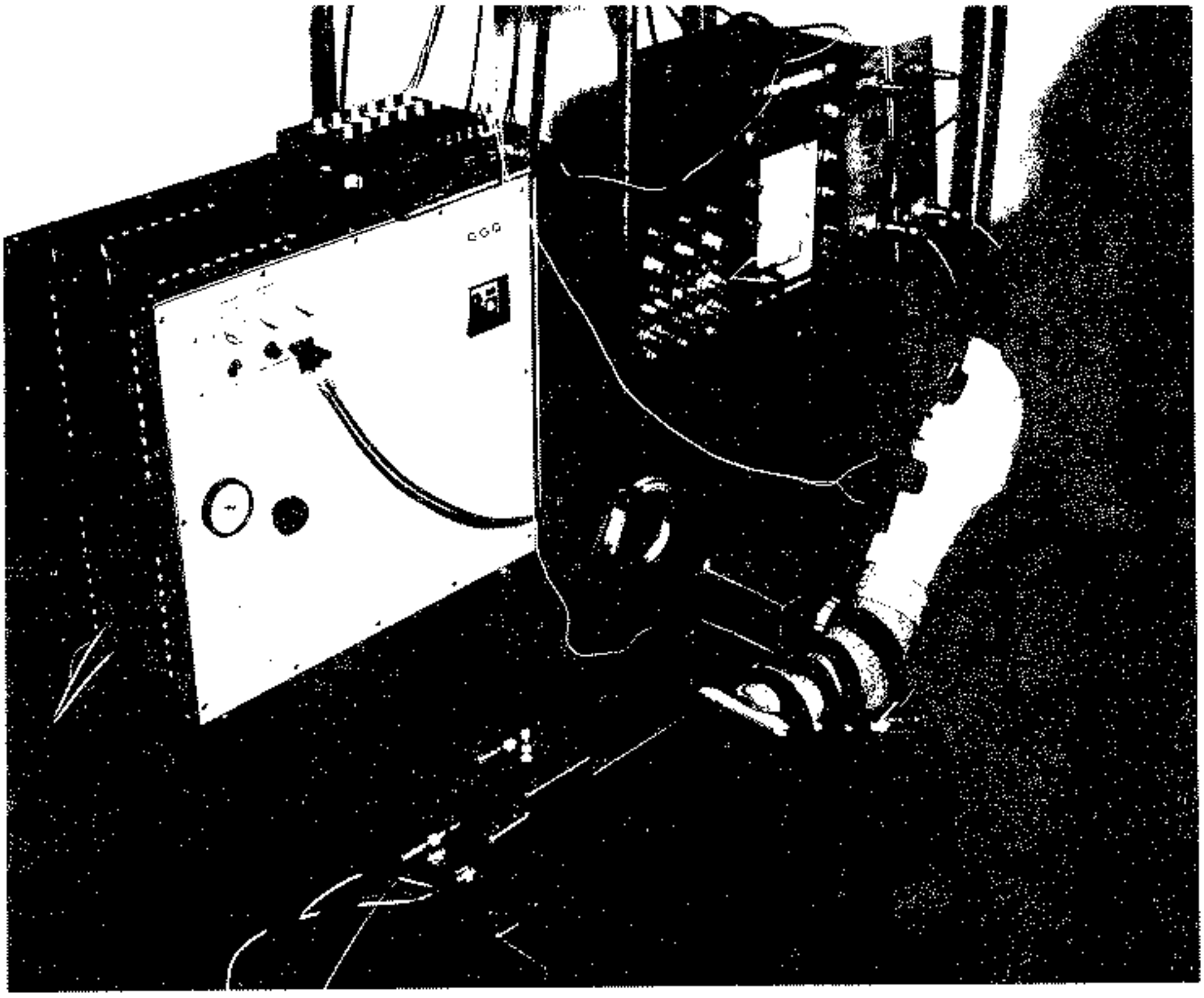

Fig. 1. Electrohydraulic servosystem for physical therapy of ankle joint and the measurement of resistive torque

measured i.e. position of ankle joint, resistive torque and 2 channels of EMG are recorded on four channel Brush recorder. In Fig. 2 typical measurement record is shown.

For tetanic electrical stimulation we use two channel stimulator with amplitude modulation. The stimulation voltage is alternately switched every half of movement period from agonist to antagonist muscle group as it is schematically shown in Fig. Both, the hydraulic servo system and the stimulator are controlled by HP function generator with 360° adjustable phase shift, so it is possible to stimulate the selected muscle group in any time relative to the ankle joint movement.

Measurements in healthy subjects

Before starting with explanation of the experimental measuring procedure and the results in patients the findings in normal subjects have to be described. Seven normal subjects were included in the measurements of resistive torque due to the passive sinusoidal ankle oscillations. The amplitude of oscillations was biased with different DC levels. Such a motion can be described by the following equation:

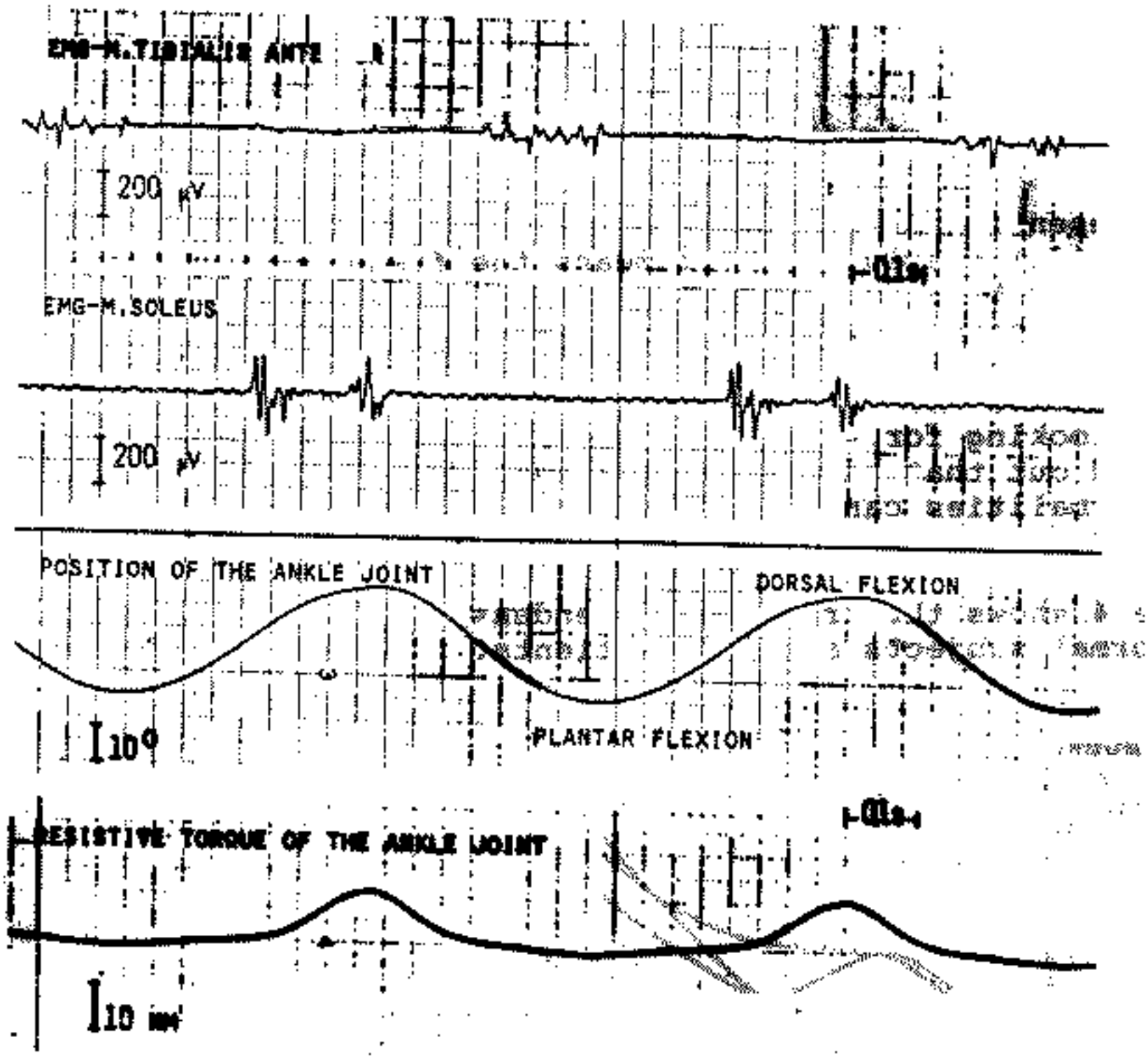

Fig. 2. TYpical record of resistive torque due to sinusoidal movement of ankle and EMG activity in spastic patient

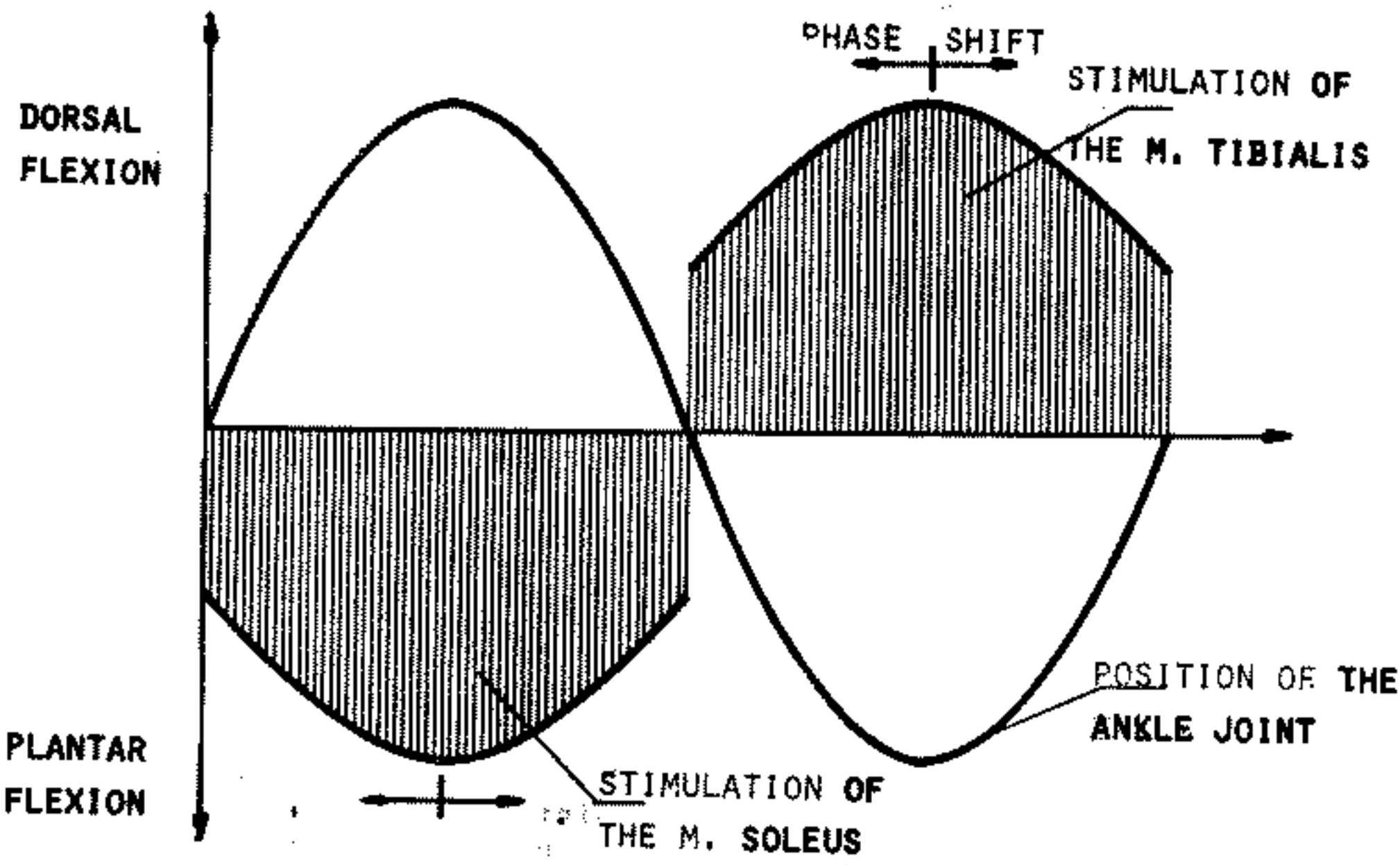

Fig. 3. Schematic description of stimulation sequences in the relation to the ankle movement

$$\phi = \phi_o \pm \phi \pm \phi_s \sin (2\pi ft) \qquad (1)$$

where ϕ_o represents a DC bias component, ϕ_s is the peak value of applied sinusoidal displacement, f means the frequency of oscillation in Hz, and t is time. In the measurements four discrete frequencies were chosen: 0.5, 1, 1.5 and 2 Hz. Lower frequencies are not important as the resistive torque at 0.1 Hz is practically the same as that at 0.5 Hz. On the contrary to Gottlieb's method where he was looking for the resonant frequency at approximatelly 6 Hz, we found out that enough information for recognition of patients abnormalities can be found from the frequency range from 0.5 - 2 Hz.

Figure 4 shows the frequency dependent resistive torque for three normal subjects and four patients.

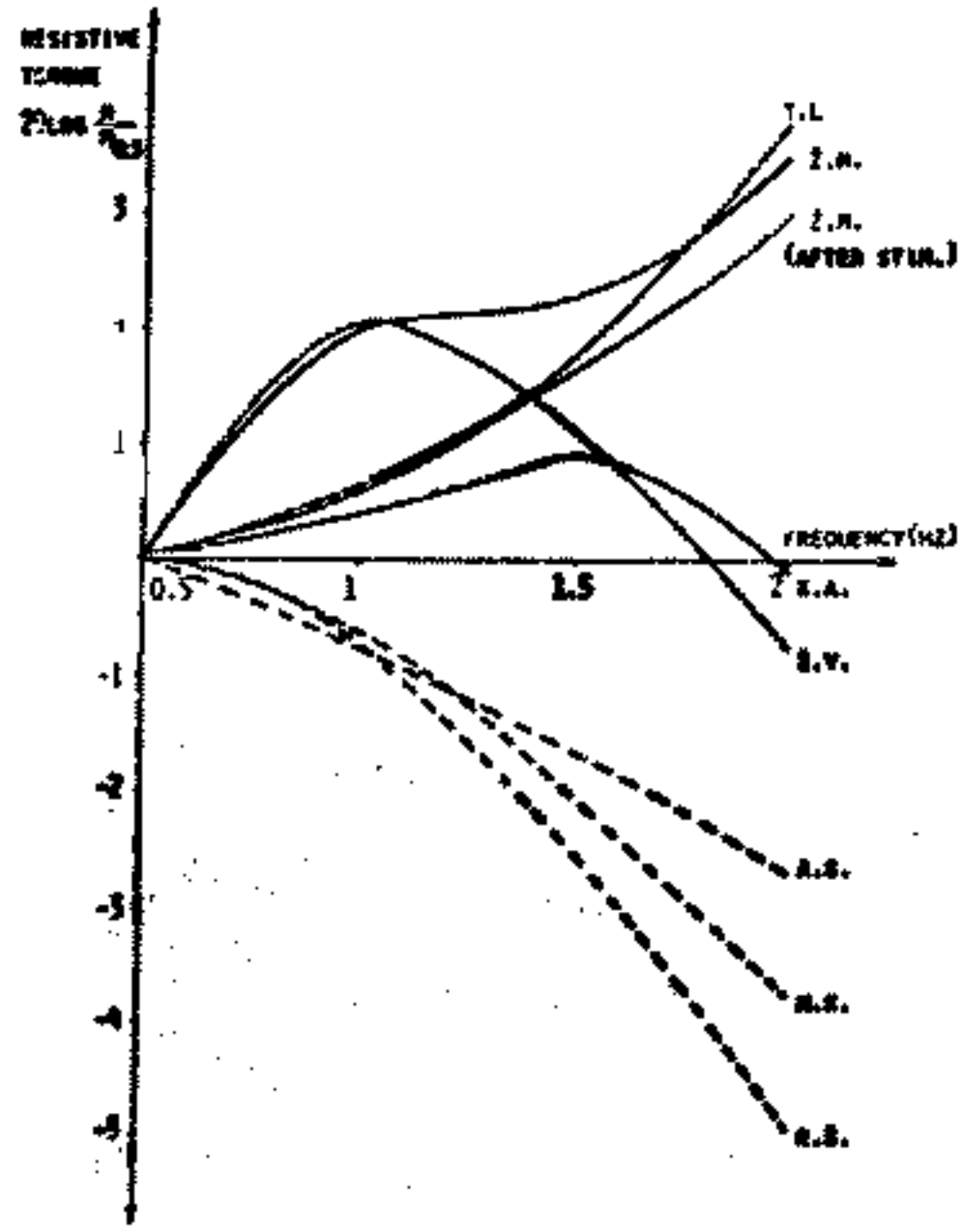

Fig. 4. Frequency characteristics of resistive torque for normal subjects and hemiplegic patients

The amplitude ϕ was 15^o in all the measurements. The biasing angle measured between tibial and foot surface varied from 0 - 15^o.

A very significant difference between normal subject and patients is evident from the slope of the frequency response. While in all the normal subjects the resistive torque is decreasing rapidly with higher frequencies, in the patients it is increasing. Sometimes first slight increasing and than decreasing is noticed being always slower than the response in normal

subjects. In the Fig.4 the resistive torque is normalized to the value obtained at 0.5 Hz so that zero line can be treated as a limit between normal subjects and patients. It is interesting that the resistive torque due to sinusoidal ankle oscillations is not sinusoidal not even when the amplitude of the oscillations is very small (few degrees only). Two possible reasons exist; first, the mechanical part of ankle joint is nonlinear and second, both muscle groups are active during the movement. The first hypothesis was tested with measurement of resistive torque peak value at different bias and oscillation angles. From these measurements it was concluded that within the limits of $\pm$ 20° around the neutral point of ankle angle do not exist significant nonlinearities, which could produce the torque curve measured. As a support to this conclusion we found out some EMG activity in both muscle groups although the measured subject was asked to be relaxed as much as possible. With the calculations of muscle forces from the linear mathematical model, which are described at the end of the paper, it is possible to show, that the nonlinearities are present at the highest velocity and at the highest displacement of ankle movement. With other words this means that proprioceptive feedback has an influence on pasive movements also in normal subjects.

Therapeutic procedure

The described measurement method was used to select patients for the proposed therapy. We have decided to test with the described measurement method all available hemiplegic patients and then select the most spastic ones for the therapy. More than 20 patients were tested and four of them are presented in Fig. 4 as typical examples. Two has constantly increasing frequency dependence of resistive torque and the other two have first increasing and then decreasing response.

The patients selected were measured two or three times in time intervals of one week to determine their status of resistance to passive movements and EMG of both muscle groups during such movements. During this time period patients have not participated in any electrical stimulation. In the third or fourth experiment both muscles, i.e. tibialis and soleus were stimulated for 20 minutes so as to activate the contraction in the streched muscle. When the foot was passively moved in direction of dorsal flexion the soleus muscle was stimulated (see Fig. 3) and V.V. The effect of such a stimulation was directly measured with torque meter while the hydraulic system was activated at frequency of 1 Hz. Immediately after the stimulation was switched off the measurement of resistive torque was repeated for all frequencies. The joint torque produced with the stimulation only was compared to the value measured at the beginning of the stimulation session. The measurement of resistive torque due to passive movements was repeated two more times. First after 10 minutes and second after 20 minutes. After first therapy treatment with the described stimulation the same procedure was performed every working day for 20 minutes without measurements. The control measurements were done only once a week. The duration of the therapy was decided to be as long as a decrease in spasticity can be observed.

RESULTS AND EVALUATION

The measured resistive torque doesn't tell much about time responses of both muscle groups during passive movements, because the dynamics of the mechanical part of the ankle can hide some important time responses. To overcome this problem somewhat simpler model of ankle joint as one by Trnkoczy et al. was used /16/. This mathematical tool was intended for the calculation of muscle force. For this calculations linear model was used being shown in Fig. 5 where B_1, B_1' represent viscous damping at agonist and antagonist muscle, K_1, K_1', K_2, K_2' represent muscle elasticity, J is the ankle moment of inertia, K_3, K_3' represent elasticity of ankle joint, B_2, B_2' represent viscous damping of ankle joint T_1, T_2 are agonist and antagonist muscle torque generators respectively, ϕ_1, ϕ_2 are angles which represent muscle displacement and ϕ is the ankle angle. Such a mechanical system can be described by three differential equations. Supposing equal properties of both muscle groups we obtain:

$$-B_1 \dot{\phi}_1 - K_2\phi_1 - K_1 (\phi_1-\phi) + T_1 = 0 \qquad (2)$$

$$-K_1 (\phi-\phi_1) - 2 K_3\phi - 2 B_3\dot{\phi} + M - K_1 (\phi-\phi_2) = J\ddot{\phi} \qquad (3)$$

$$-B_1\dot{\phi}_2 - K_2\phi_2 - T_2 - K_1(\phi_2-\phi) = 0 \qquad (4)$$

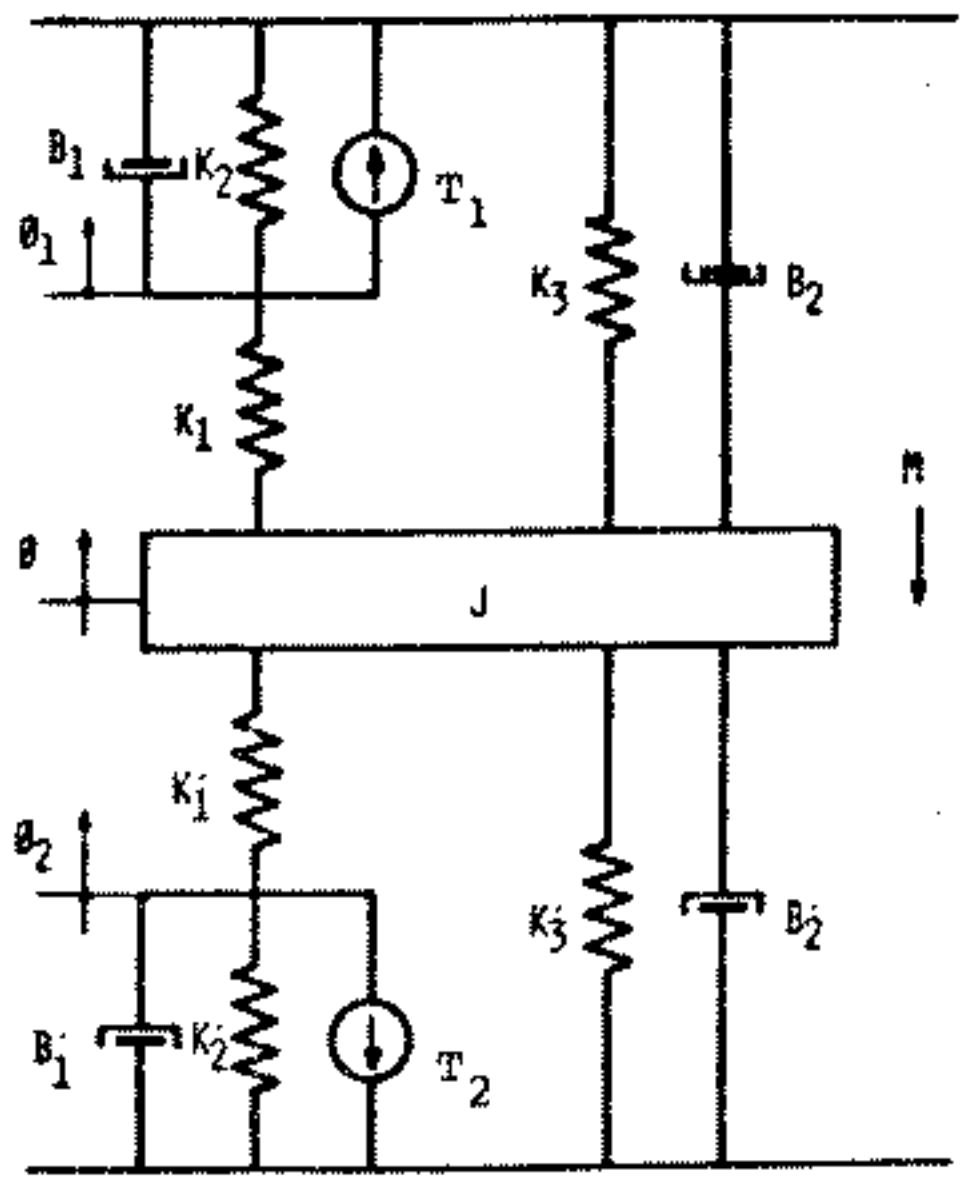

Fig. 5. Mathematical model of the ankle joint

From the equation (3) ϕ_2 can be expressed and its derivative $\dot{\phi}_2$ can be also obtained. After inserting ϕ_2 and $\dot{\phi}_2$ into the equation (4) we obtain:

$$(K_1+K_2)\ \phi_1 + B_1\ \dot{\phi}_1 - \frac{JB_1}{K_1}\dddot{\phi} - \ddot{\phi}\ (\frac{2B_1B_3}{K_1} + J\ \frac{(K_1+K_2)}{K_1}) -$$

$$- \dot{\phi}\ \frac{2B_1(K_3+K_1}{K_1} - \frac{2B_3(K_1+K_2)}{K_1} -$$

$$- \phi(\frac{2(K_1+K_2)(K_1+K_3)}{K_1} - K_1) - \frac{B_1}{K_1}\dot{M} - \frac{K_1+K_2}{K_1}M - T_2 = 0 \qquad (5)$$

The equation (2) can be rewritten as:

$$-(K_1+K_2)\phi_1 - B_1\ \dot{\phi}_1 + T_1 + K_1\ \phi = 0 \qquad (6)$$

Finally we make a sum of equations 5 and 6 what gives the difference of equivalent muscle torque generators:

$$T_1-T_2 = \dddot{\phi}\ \frac{JB_1}{K1} + \ddot{\phi}\ (\frac{2B_1\ B_3 + J(K_1+K_2)}{K_1}) +$$

$$+ \dot{\phi}\ (\frac{2B_1(K_3+K_1) + 2B_3(K_1+K_2)}{K_1}) +$$

$$+ \phi\ (\frac{2(K_1+K_2)\ (K_1+K_3)}{K_1} - 2K_1) - \frac{B_1}{K_1}\dot{M} - \frac{K_1+K_2}{K_1}M$$

At sinusoidal oscillations of ankle (ϕ) first, second and third derivative can be easily analitically obtained. The only problem is the first derivative of the measured resistive torque M, which can be satisfactory calculated using central difference numerical method. The difference T_1-T_2 can be than arithmetically determined.

Five patients were treated with the described stimulation program. In only one the therapy is already finished, while the others are still in the therapeutic program. In more than ten initial experiments with these five patients only once the resistive torque M was increased for about 100 %. This specific experiment will be discussed later. In all other experiments a significant decrease of resistive torque was noticed after twenty minutes of therapy which remained on the same value for additional 20 minutes, until the measurement was completed. In Fig. 6 the short term effect of therapy is shown for one patient The curves represented in Fig. 6 are typical for all five patients. On the top of the figure the decrease of resistive torque M is evidently seen, while at the bottom we can see the correction of muscle activity due to therapy. The muscle activity T_1-T_2 after stimulation is much more similar to that obtained by normal subjects.

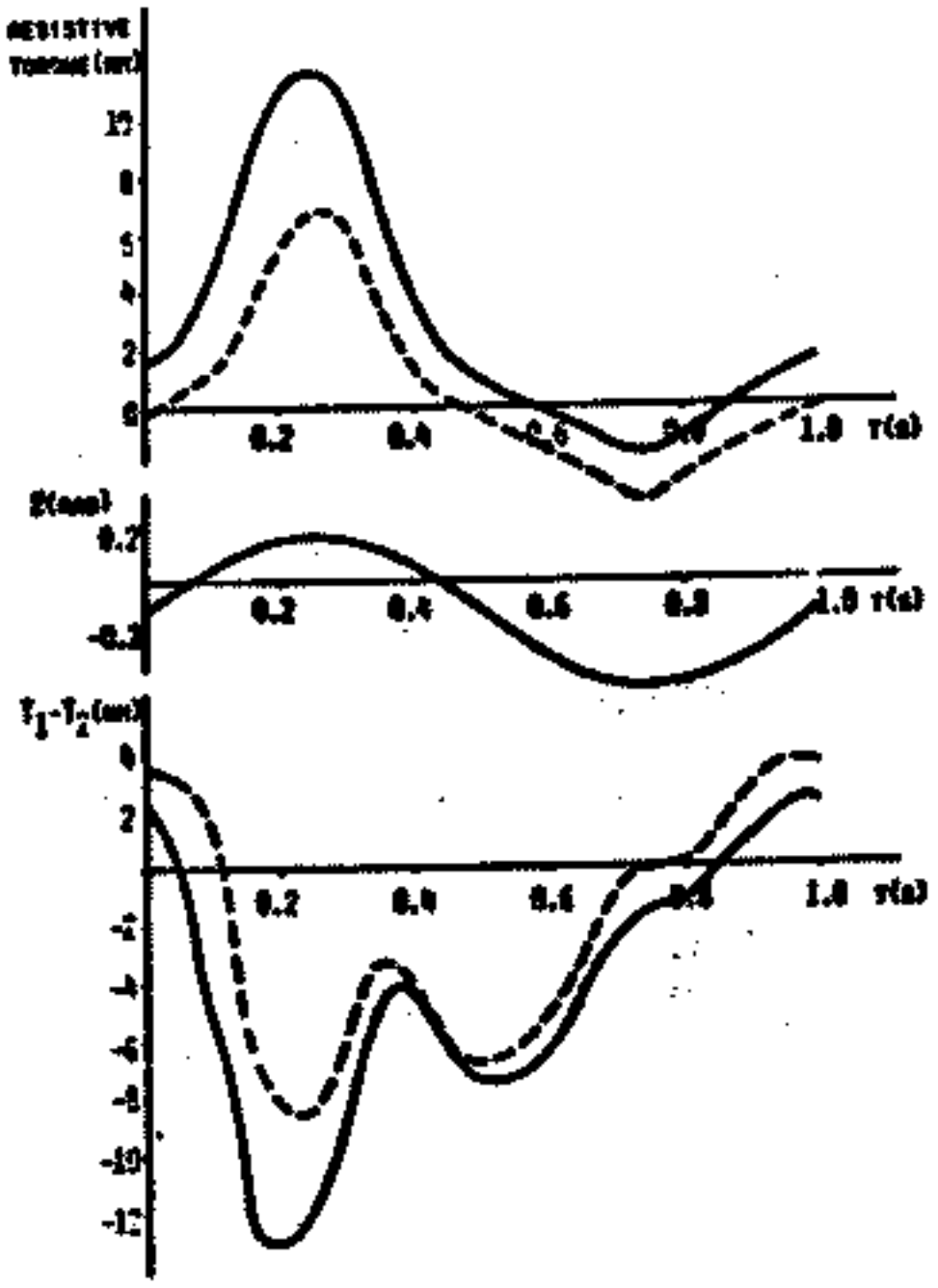

Fig. 6. Resistive torque and muscle activity T_1-T_2 (see text) before and after therapy. Solid lines represent the status before therapy and dashed after therapy. Ankle angle is both times the same.

As mentioned, only in one patient the therapy program has been already completed. It is hard to make conclusions about long term effects on one patient. After the therapy, the peak value **of** resistive torque was reduced in this patient from 17 Nm to 9 Nm at frequency 1 Hz and amplitude of angle 15°. **For comparison to this value, the resistive torque in normal subject in the same conditions** is 6 Nm. The frequency response which was sustainly increasing before therapy was after the therapy slightly **decreasing.** The clonus was induced much more difficult **after therapy** and always stopped after few oscillation **periods.**

Conclusion

First we must **comment the experiment where the resistive torque was** increased. **It** was the **first** experiment with **the stimulation** for that patient. The stimulation threshold value was extremely high, and we had to apply very high stimulation **voltage (about** 100 V) to obtain only 2 Nm of joint torque. Although **an** increase **of** spasticity was discovered in this patient **the** therapy was performed and after one week the resistive torque **was** significantly lower than before first stimulation session. The stimulation threshold was highly reduced. Two possible mechanisms **can be involved in this specific experiment.** First possibility **is activation of exteroceptive afferent pathways and second is**

activation of proprioceptive signals or direct gamma excitation The first explanation seems to be more probable since the stimulation was surface and the patient's skin was extremely dry.

One another interesting measurement is done in our experiments which was not found in any similar experiments before. That is the measurement of the direct effect of stimulation on resistive torque during the FES therapy. With this measurement we have found out that the peak value of resistive torque is the exact sum of the resistive torque without stimulation and torque due to stimulation, but the difference comes out in the time response of the resistive torque. This effect suffers the suggestion, that electrical stimulation has direct influence on phasic stretch reflex, while on the tonic stretch such influence doesn't exist. There is probably also some connection with the clonus. In any case this phenomena must be carefully investigated in the near future. The described eksperiments represent the first step on the way to understanding why some times electrical stimulation decreases spasticity some times increases, or has no influence on it.

Acknowledgement

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