

PHYSIOLOGICAL STUDY OF TES BY EXACT ACTIVE-ASSISTIVE SYSTEM

Y. Muraoka, Y. Tomita, T. Fujiwara*, Y. Masakado**

Institute of Biomedical Engineering, Keio University

*Keio Tukigase Rehabilitation Center

**Department of Rehabilitation Medicine, Keio University School of Medicine

3-14-1 Hiyoshi, Kohoku-ku Yokohama, 223-8522, Japan

ABSTRACT

We developed a system that gave electrical stimulation with intensity in proportion to the voluntary EMG through the EMG recording electrodes, and used it for hemiplegic training. The training effect was observed in long (a month of the training) and short (ten minutes after the training) period. We investigated this training effect of this system physiologically.

We carried out two experiments;

1. Observation of disynaptic Ia inhibition between tibial anterior muscle (TA) and soleus muscle (SOL) in hemiplegic patients before and after therapeutic electrical stimulation (TES) to TA.
2. Observation of the convergence onto spinal Ia inhibitory interneurons from Ia afferent and from fast conducting corticospinal axons using a peroneal nerve electrical stimulation and a magnetic cortical stimulation.

The inhibition from peroneal nerve to SOL did not change, while the inhibition from tibial nerve to TA decreased. TES changed the synaptic transmission efficiency of mutual inhibition. The signals from corticospinal axons and antagonistic muscle nerve converged onto human spinal Ia inhibitory interneurons. We suggested that (1) short-period effect after TES was caused by the descending signals passing the efficiency-changed synapse, (2) the efficiency was also changed by the signal's from cortex, and (3) the reorganization in the brain by the biofeedback training is necessary to obtain and keep the long period effect.

Keywords: hemiplegic patients, electrical stimulation, magnetic stimulation, biofeedback training

INTRODUCTION

We have developed a system that gave electrical stimulation with intensity in proportion to the voluntary EMG. The stimulation was applied to a muscle with the EMG recording electrodes.[1] Thus the system can exactly assist and amplify a weak muscle contraction by adding electrical stimulation (Exact Active-Assistive System).

We used the system for the hemiplegic training. The training effects of muscle strength, range of motion, and coordination of ankle joints were observed in short (ten minutes after the fifteen minutes training) and long period (a month of the training). The training by the system was more effective than the ordinary TES.[2]

The system seems to have several advantages superior to the ordinary TES. The system can train patients to transmit a command signal from cortex to the target muscle by using the EMG feedback. The exact contraction can be produced from transmitted command, because the stimulation intensity is proportional to the transmitted signal and the same electrodes are used for recording and stimulation. The patient can recognize the transmitted command with muscle contraction. The sensory receptor such as tendon and muscle spindle can transmit afferent signals generated by the muscle contraction to the spinal cord and the brain. The patients learn movements by repeating this process, and the ordinary TES does not have it. In this paper we suggested the necessity of this process adding to ordinary TES by two experiments.

METHODS

Experiment 1. Disynaptic Ia inhibition between TA and SOL in five hemiplegic patients before and after TES to TA [3] (TES: the duration of 0.3 ms, the frequency of 20 Hz, monophasic rectangular wave, repeating 5 seconds stimulation and 15 seconds rest for 15 minutes)

H reflex at SOL (H_{SOL}) and at TA (H_{TA}) could be induced in all 5 subjects and in 3 subjects, respectively. Excitability of the motorneuron pool was assessed by the size of the H_{SOL} and H_{TA} .

Subjects were seated comfortably and paired electrodes were placed over bellies of the soleus and tibial anterior muscles for recording EMG and the level of the head of the fibula to stimulate the common peroneal nerve to elicit H_{TA} . The posterior tibial nerve was stimulated through a monopolar stimulus electrode placed in the popliteal fossa to elicit H_{SOL} . The anode was placed over the anterior part of the patella. The duration of the test stimulus of both the tibial and the peroneal nerve was 1 ms. The test H_{SOL} and H_{TA} were kept between 20 and 40 %, and between 5 and 10% of M-max, respectively.

Conditioning stimulus with the duration of 1 ms were applied to the peroneal nerve for testing Ia disynaptic inhibition of H_{SOL} or to the tibial nerve for inhibition of H_{TA} , using the same electrodes to elicit test H reflexes. For conditioning stimuli to the peroneal nerve, intensity was always kept just at motor threshold (1.0MT) for TA. For the stimulation to the tibial nerve, intensities around 0.7MT for the H_{SOL} were applied.

Conditioning and test stimulation interval (C-T interval) were set ranging from 0 to 10 in the peroneal nerve and ranging from -5 to 5 ms in the tibial nerve. One round was defined as each once in every C-T interval and unconditioned stimulus (H_{test}) have been randomly generated every 5 seconds, and ten rounds were repeated. The inhibition was demonstrated by plotting the amplitude of the conditioned H-reflexes (H_{cond}) as a percentage of H_{test} . The size of H-reflex of disynaptic Ia inhibition was denoted H_{Ia} . H_{Ia} of post-TES were standardized by the mean of H_{Ia} of pre-TES, and the values of all subjects were summarized. These were compared between pre-TES and post-TES by paired t-test.

Experiment 2. Convergence onto Ia inhibitory interneuron from corticospinal axons and antagonistic muscle nerve in five intact man

Before second main experiment two preliminary experiments were carried out.

(1)The above-mentioned experiment (the inhibition of H_{SOL} experiment) was carried out to investigate arrival time to Ia inhibitory interneuron from the peroneal nerve stimulus in each subject.

(2)After setting stimulus and recording electrodes at the same positions with Experiment 1, the 8-shaped coil of a magnetic stimulator was held flat on the scalp. Its optimal position was determined by slight displacements until motor evoked potential (MEP) of the highest amplitude was recorded from TA. The stimulus intensity was tuned at threshold of the MEP. The transcranial magnetic stimulation (TMS) was performed as a conditioning stimulus in order to investigate arrival time to Ia inhibitory interneuron from the cortex in each subject.

C-T intervals were set ranging from -10 to 10 ms. One round was defined as each once in every C-T interval and H_{test} have been randomly generated every 5 seconds, and ten rounds were repeated. The inhibition to H_{SOL} by TMS was demonstrated by plotting H_{cond}/H_{test} .

Their arrival time after antagonistic stimulus and TMS by the preliminary experiments were set and we observed the inhibition to H_{SOL} in the conditioning stimulus of three patterns; (a) electrical stimulation to peroneal nerve, H_{Ia} [n] (n: round number), (b) a magnetic cortical stimulation that induced motor evoked potential at TA, H_{TMS} [n], (c) (a) and (b), H_{Ia+TMS} [n].

One round was defined as once in every condition patterns and unconditioned stimulus (Htest[n]) have been randomly generated every 5 seconds, and ten rounds were repeated. The inhibition to H_{SOL} by conditioning stimulus of four patterns was demonstrated by plotting H_{cond}/H_{test}.

The values of H_{TMS}[n]-H_{Ia+TMS}[n] were standardized those of H_{test}[n]-H_{Ia}[n], and were summarized in all subjects. They were compared by paired t-test.

RESULTS

Experiment 1 Fig.1 (a) and (b) show the representative results of a hemiplegic subject, and (c) shows the summarized result of all subjects. C-T intervals of the disynaptic Ia inhibition of H_{TA} and H_{SOL} were 0 and 3 ms, respectively as shown in Fig1.(a),(b). The inhibition from peroneal nerve to H_{SOL} did not change and the inhibition from tibial nerve to H_{TA} decreased by TES as shown in Fig.1 (c).

Experiment.2 Fig. 2 (a)-(d) were the summarized results of all subjects. C-T interval in the peroneal nerve experiment was 2 ms and the interval in TMS was -1 ms as shown in Fig2. (a), (b). The H_{SOL}/H_{test} of TMS in figure 2 (c) was more than 1.0, since not only IPSP but also EPSP were generated to anterior horn cell by TMS that could not stimulate selectively. The mean of H_{SOL} was smallest in the condition of both stimulations Ia+TMS. The amount of disynaptic Ia inhibition with the effect of the descending impulse (Ia+TMS - TMS) was significantly larger than that without the effect of the descending impulse (Ia-test) as shown in Fig. 2 (d).

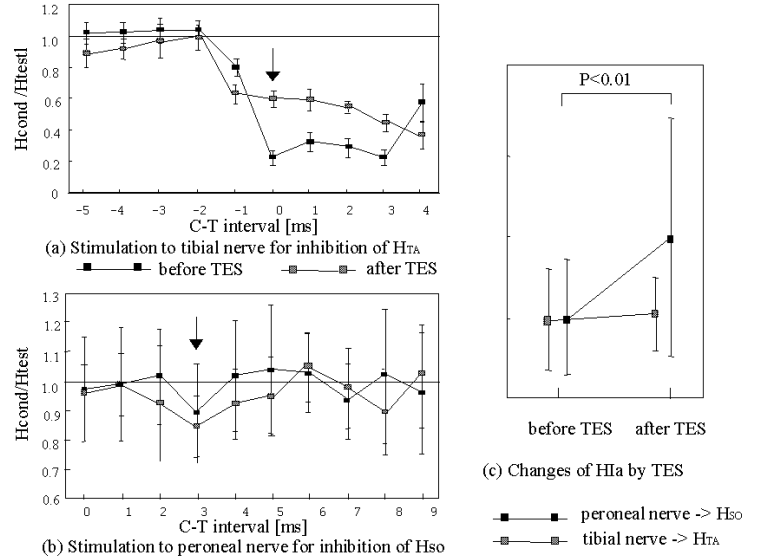


Fig. 1 Disynaptic Ia inhibition changes between before and after TES

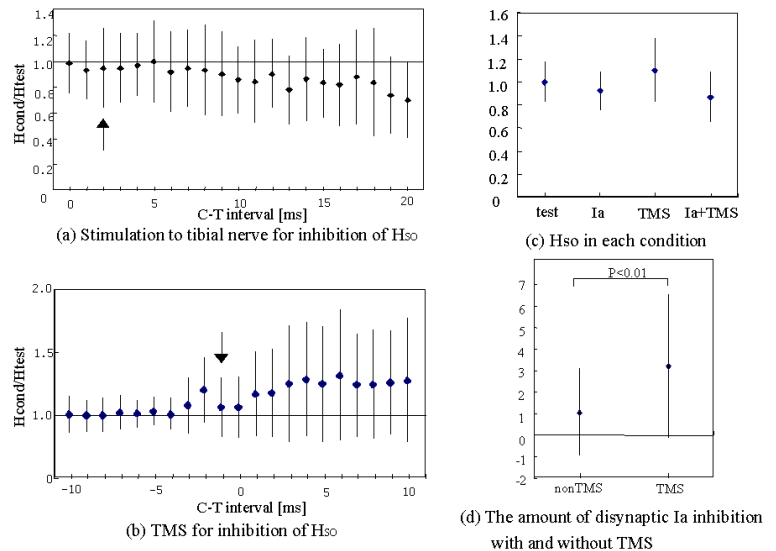


Fig. 2 Convergence onto Ia inhibitory interneuron from peroneal nerve and corticospinal axons

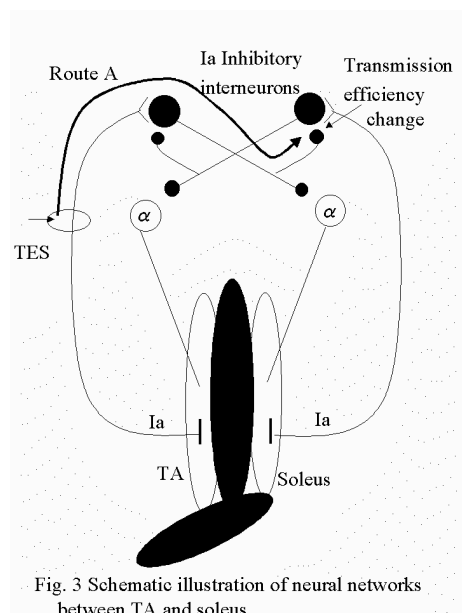


Fig. 3 Schematic illustration of neural networks between TA and soleus

DISCUSSION

The strong disynaptic Ia inhibition of H_{TA} from tibial nerve was observed in hemiplegic subjects before TES and this inhibition significantly decreased after TES. We suppose that it is an effect of transmission efficiency change of the mutual inhibitory synapse from flexors inhibitory interneuron to extensors inhibitory interneuron as shown in Fig.3. TES from peroneal nerve activated the route A of Fig. 3 and kept it after TES for a few hours. The synapse was also activated because it

was included the route A. The synapse inhibited the inhibition of TA from SOL, consequently the inhibition of TA decreased. It is reported that the SOL spasticity decreased with TES. We expected the inhibition from the peroneal nerve to the SOL would change by TES, but it did not be observed. Other effect should be considered for finding the mechanism.

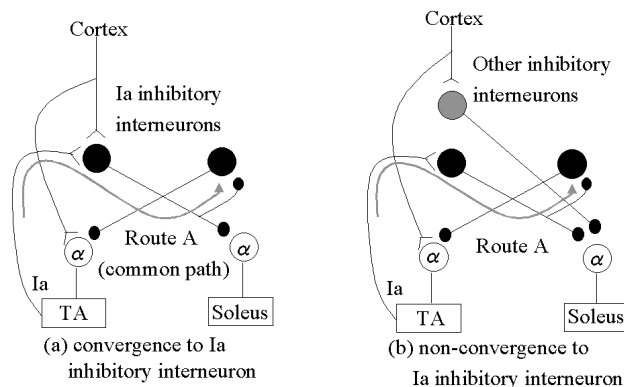


Fig. 4 Schematic illustration of neural networks in case of convergence and non-convergence

The effect of disynaptic Ia inhibition of TA from peroneal nerve significantly increased and was influenced by TMS. It means the corticospinal axons and the peroneal nerve converged onto Ia inhibitory interneurons of TA. Thus the impulses from cortex also pass through the route A and they are especially possible to activate it. (see Fig.4)

Here we suggest the causes of the decline of voluntary contraction at TA after stroke and training effect of the exact active-assistive system. (1) Due to brain stroke, the transmission efficiency of mutual inhibitory synapse from flexors to

extensors decreases, since the frequency of impulses passing through the route A decreases. (2) TES activates the synaptic transmission efficiency and keeps it for a few hours after TES. (3) As long as the efficiency is kept high, the signal from the cortex can transmit and contract the target (carry over effect). (4) The efficiency can not keep for long time since the descending impulse frequency is small in stroke patient. (5) The exact active-assistive system train patients to transmit a command signal from cortex to the target muscle exactly. The training (motion learning in brain) can increase the descending impulse frequency and keep it for long time gradually. Therefore the wrong training (learning) due to wrong feedback by misplacement of electrodes leads the situation of illness worse. Therefore, appropriate feedback information is essential for stroke patient.

We suggest the biofeedback training adding to TES is necessary to acquire long time effect of TES, and the exact feedback information is essential for training in the stroke patients. The exact active-assistive system satisfies these conditions.

REFERENCES

- [1] Y.Muraoka, Y.Tomita, et.al, EMG-controlled hand opening system for paraplegia, 6th Vienna international workshop on functional electrostimulation, Vienna, 1998.9
- [2] R.Shirakawa, Y.Muraoka, et.al, The effect of neuromuscular electrical stimulation with hemiplegia inpatients; comparison of different levels of stimulation, 13th International Congress of The World Confederation for Physical Therapy, Yokohama, 1999.5
- [3] Y.Okuma, R.G.Lee, Reciprocal Inhibition in Hemiplegia: correlation with clinical features and recovery, The Canadian Journal of Neurological Sciences, vol.23, pp.15-23, 1996.

AUTHOR'S ADDRESS

Yoshihiro Muraoka

Institute of Biomedical Engineering

Department of Applied Physics and Physico-informatics, Keio University

3-14-1, Hiyoshi, Kohoku-ku, Yokohama, 223-8522 JAPAN

e-mail:mura@thx.appi.keio.ac.jp