IN VIVO EVALUATION OF EFFECTS OF ELECTRICAL STIMULATION WITH VASCULAR OCCLUSION ON DISUSE ATROPHIED MUSCLE WITH SPECIAL REFERENCE TO INTRAMUSCULAR ENERGY METABOLISM.

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Purpose

Various countermeasures to prevent disuse atrophy of skeletal muscle have been investigated. Recently, muscle exercise under ischemic conditions has been reported to be effective for rehabilitation after knee surgery to prevent post-surgical muscle atrophy (4). Its efficacy has been supported by another experimental study (5). Thus, it is suggested that training with vascular occlusion may be efficient to prevent the development of disuse atrophy as well. Intramuscular energy metabolism in working muscles is one of the important indicators of skeletal muscle function and it can be monitored in vivo using 31P-MRS (3, 7). The purpose of this study was to evaluate the effects of muscle electrical stimulation training with vascular occlusion as a countermeasure to prevent disuse atrophy of skeletal muscle, with reference to energy metabolism in the working muscle as assessed by 31P-MRS.

Methods

Materials

Twenty-four Sprague-Dawley rats were used and divided into four groups: control group (C), hind-limb suspended (2) for 7 days (HS), HS plus muscle electrical stimulation training with vascular occlusion (VO), and HS plus muscle electrical stimulation training without vascular occlusion (ES). The electric stimulation for training of the gastrocnemius-plantaris-soleus (GPS) muscles was attained at 100Hz for 5 min a day from the second to 6th day during the suspension period. For the VO group, the stimulation was conducted under ischemic conditions with the thigh wrapped with tourniquet especially designed for this purpose. During electrical stimulation, the pressure of the tourniquet was maintained at approximately 300mmHg, so that vessels were occluded. The GPS muscles in each group was subjected to the following measurements, and the data were compared among the groups.

Measurement

After a 2-min rest, contraction of the GPS muscles group was induced by electrical stimulation of the sciatic nerve at 0.25Hz for 10 min, then the frequency was increased to 0.5 and 1.0Hz every 10 min. During the stimulation, twitch forces were recorded by a strain gauge and 31P-MRS was simultaneously measured. On each spectrum, the peaks of phosphocreatine (PCr), and inorganic phosphate (Pi) were observed. The ratio of the area of both peaks (PCr/(Pi+PCr)) and intracellular pH, determined by the chemical shift between both peaks, were calculated as indicators of energy metabolism. The PCr/(Pi+PCr) and force were measured at a steady-state in each frequency, and the relationship between the PCr/(Pi+PCr) and peak twitch force times rate (force*rate) was examined to evaluate oxidative capacity of the muscles. Maximum tension was measured at the muscle contraction induced by 0.25Hz; the wet weight of the whole GPS muscle was also measured.

Results

Muscle weight

The weight of the whole GPS muscle in HS, VO and ES group decreased significantly (p<0.05) compared with C group (2.66, 2.34, 2.04, and 1.97 (g) in C, HS, VO and ES, respectively).

Tension

The maximum tension was 512, 441, 494 and 346 (gw) for C, HS, VO and ES, respectively. In the VO group only, tension was maintained at the control level.

MRS Measurements

Intracellular pH did not decrease below 7.0 during muscle contractions, indicating the muscle exercise was aerobic. Significant (p<0.05) linear relationships between PCr/(Pi+PCr) and force*rate were found in all groups during muscle contraction. The slope in C group was gentler than that in HS and ES (p<0.05), but no difference was observed between C and VO (Fig. 1). During aerobic exercise the slope indicates muscle oxidative capacity. Therefore, the oxidative capacity of muscles was shown to be maintained for seven days when electrical stimulation exercise was attained under ischemic conditions.

Discussion & Conclusion

Skeletal muscle atrophy, as evaluated by measuring the muscle’s wet weight, worsened by the hindlimb suspension, suggesting that HS could induce muscle atrophy of the gastrocnemius-plantaris-soleus muscle. In the ES and VO groups, the weight was also significantly smaller than C group, and was almost same value as the HS group. These data suggested that the tetanic electrical stimulation for 5 min a day could not be an effective countermeasure to develop the disuse induced muscle volume reduction, nor the ES with vascular occlusion could not.

The muscle oxidative capacity was evaluated in the working muscle in vivo using 31P-MRS. Although the oxidative capacity decreased during hind-limb suspension as shown in the HS group, it could be maintained at control levels by electrical stimulation with vascular occlusion. Only the electrical stimulation could not maintain the muscular oxidative capacity, therefore, the ischemic condition produced by vascular occlusion may have some additional advantage effects. The previous in vitro studies demonstrated that muscle training under ischemic condition could increase the activity of citrate synthase that is an marker of oxidative capacity (1). The present study is good agreement with the in vitro study. The training with local leg ischemia in human was also reported to increase type I fiber in the trained muscle (6). Type I fiber is demonstrated to reduce by disuse, therefore, the training under the ischemic condition is considered to be effective as a countermeasure that facilitates aerobic metabolism in the disuse atrophied muscle.

The electrical stimulation with vascular occlusion was shown to be effective for reduce the degradation in the muscle energy metabolism, and not for the development of muscle volume reduction in this study. Thus, it is indicated that to prevent volumetric and functional deterioration in the disuse atrophied muscle we need to further investigate optimal electrical stimulation along with the protocol used in the present study.

Fig.1 The relationship between the PCr/(Pi+PCr) and peak twitch force times rate

References

4) Takarada Y. et al.: J Appl Physiol, in press
5) Takarada Y. et al.: J Appl Physiol, in press

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