RAPID SERIAL SARCOMERE LOSS CAUSED BY ELECTRICAL STIMULATION IN RABBIT TRICEPS SURAE MUSCLES

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INTRODUCTION

Muscle spasticity, as occurs in patients with cerebral palsy or people who have suffered from a stroke, are involuntary muscle contractions that usually occur due to lesions in the brain. Muscle spasticity causes muscles and fascicles to become short and stiff, thereby limiting the range of motion of joints and affecting everyday function. It has been suggested that stiffness in spastic muscles is caused by a decrease in the number of serially arranged sarcomeres, but the reason for this loss in sarcomeres remains a matter of scientific debate. In 1981, Tabary and Tardieu [1] showed that 12 hours of continuous low-level stimulation of the soleus muscles in guinea pigs caused a 25% decrease in the number of serial sarcomeres. Patients with brain lesions in the motor cortex also have low level, involuntary stimulation of their “spastic” muscles, thus it seems possible that the low-level stimulation may cause a loss of serial sarcomeres. Therefore, the purpose of this study was to test if after 10 hours of continuous electrical stimulation of the tibial nerve in New Zealand White rabbits, there would be a substantial decrease in the number of serial sarcomeres in the Soleus, Plantaris and Medial Gastrocnemius (MG) muscles.

METHODS

The MG, Plantaris, and Soleus muscles were stimulated via the tibial nerve in New Zealand White rabbits (n=3) for 10 hours in one of the legs (20 Hz at three times the α-motoneuron threshold). The tibial nerve in the contralateral control leg was transected to ensure that no cross-over training effects occurred. An additional four animals were tested without stimulation, but with the ankle joint fully plantar-flexed for ten hours so as to shorten the target muscles as much as possible. After the experimental period, the animals were sacrificed and the hind limbs removed. The hind limbs were then placed in a 10% formalin solution at carefully controlled knee and ankle angles, for at least a week. Four to six tissue samples were harvested from each muscle from precisely defined locations, and were placed in 30% nitric acid for approximately 50 hours to digest the connective tissues. The acid digested samples were then placed in 100% glycerol. Following the digestion process, five individual fascicles were teased out from each of the tissue samples and mounted on prepared slides. These slides were then analyzed for fascicle length by using a camera system with specialized software. Sarcomere lengths were determined at five points along the entire fascicle length using a laser diffraction method [2]. The number of serial sarcomeres in the muscles was then calculated by dividing the fascicle length by the average sarcomere length.

RESULTS

The serial sarcomere number in the stimulated experimental limbs decreased by 23% (± 4.4%) in the MG, decreased by 25% (± 2.9%) in the Plantaris, and decreased by 29% (± 7.0%) in the Soleus. The decrease in sarcomeres in the Soleus was significantly greater than that in the MG (r=0.0353). The serial sarcomere number in the experimental (non stimulated but fully plantar-flexed hind limbs) did not change. The sarcomere loss observed here is similar to the 25% loss reported by Tabary and Tardieu in guinea pigs.

DISCUSSION & CONCLUSIONS

The results of this study suggest that chronic, low-level muscle stimulation is a potent regulator of serial sarcomere number, whereas muscle shortening alone is not; at least within the short time frame observed here. We propose that the massive shortening of spastic muscles, as observed in children with cerebral palsy, and the associated over-stretching of sarcomeres [3] may be caused by the involuntary and persistent activation of spastic muscles in these children. Now that this was seen in our laboratory, many questions go unanswered: What is the mechanism causing this rapid sarcomere number loss? Can this sarcomere loss be prevented by inhibiting the overstimulation in patients with brain lesions? Will stretching during contraction phases prevent or reduce this effect? Before applying this finding clinically, future studies will need to focus on how this sarcomere loss may be limited or stopped in patients with chronic, involuntary stimulation of muscles caused by brain lesions.

REFERENCES


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