

Prevention of Muscle Atrophy by Functional Electrical Stimulation

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Location: Room 304C

Category: Laryngology

Authors

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Abstract

Problem Addressed: After denervation of recurrent laryngeal nerves, the intrinsic laryngeal muscles are atrophied. Functional electrical stimulation (FES) to the denervated thyroarytenoid muscle induces vocal cord movements, and also prevents muscle atrophy. However, the mechanisms of preventing skeletal muscle atrophy by FES are unknown. Valosin-containing protein (VCP) plays an important role in transportation of misfolded proteins from endoplasmic reticulum (ER) to cytosol, and is associated with the ubiquitin-proteasome degradation pathway. Storage of misfolded proteins in ER induces apoptosis, which may relate to the muscle atrophy after denervation. We studied whether the FES delivered to the denervated skeletal muscles effects on VCP expression and apoptosis.

Methods and Measures: The soleus muscle of rats was employed. We prepared the muscles in four conditions, that is, denervated muscle without FES, denervated muscle with FES, non-denervated muscle with FES, and non-denervated muscle without FES (normal). Sciatic nerve was surgically resected under general anesthesia. FES, with 2 mA rectangular pulses of 0.4 ms duration at 2 Hz lasting for 1 hour, was delivered percutaneously once every 2 days. After 4 weeks, soleus muscles were removed under general deep anesthesia. cDNA array, immunohistochemistry staining and Western blotting were performed.

Results: VCP expression highly increased in the denervated muscle with FES compared to that in the muscle without FES. The staining of ssDNA and activated caspase-12 in the denervated muscles was seen stronger than those in other conditions.

Conclusions: These results suggest that the FES elevates VCP expression, and that enhanced VCP might prevent apoptosis and muscle atrophy.

Clinical Significance of Study: FES can prevent the atrophy of intrinsic laryngeal muscles after denervation of recurrent laryngeal nerves.

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