Role of Motoneuron Pulsed Activity in the Regulation of Myosin Composition in the Slow Skeletal Muscle

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The possible role of pulsed motoneuron activity in the regulation of the qualitative composition of the guinea pig slow skeletal muscle myosins is studied by immunohistochemical methods using monoclonal antibodies to the fast myosin heavy chains. Guinea pig intact *musculus soleus* contains only the slow muscle fibers. Blockade of axon transport by colchicine application on the sciatic nerve during intact pulsation leads to the expression of fast myosin. Tenotomy simultaneous with the axon transport blockade prevents the appearance of the fast muscle fibers. Our results indicate that pulsed activity of motoneuron as a factor of neurotrophic control regulates the qualitative composition of myosins, thus supporting the functional activity of the muscle.

Key Words: *slow muscle; myosins; neurotrophic control; tenotomy; immunohistochemistry*

Neurotrophic control of skeletal muscle function is realized via trophic factors produced in motoneuronal perikaryons and transfered to the muscle by axon transport and is determined by the type of pulsed activity [3,4,6]. In order to study the role of axon transport in the realization of neurotrophic control it is blocked by statmokinetics, for example, colchicine [5] which binds the axon microtubule tubulin without impairing pulsed activity. Previously, we showed [4] that the blocking of axon transport by colchicine application on the sciatic nerve, in contrast to denervation, induces the production of fast myosin in a homogeneously slow guinea pig musculus soleus. These results suggest a possible role of pulsed motoneuron activity in the regulation of qualitative composition of slow musce myosins. On the other hand, it is not clear how pulsed activity regulates the qualitative composition of skeletal muscles. At least two variants are probable: pulsation directly affects the muscle fiber (MF) genome inducing the expression of fast myosin or it is a factor preserving the muscle contractility. In order to answer these questions, we examined the guinea pig slow muscle after axon transport blockade and tenotomy by immunohistochemical methods. The muscle looses its functional loading under such conditions, despite intact pulsation.

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MATERIALS AND METHODS

Musculus soleus (a slow muscle) of male guinea pigs weighing 300-400 g was examined. Cryostate sections (8-μ) were stained by the indirect PAP method with monoclonal antibodies to the fast myosin heavy chains (Sigma). To block the axon transport, 5 mM colchicine solution (Merck) was applied for 10 min on the sciatic nerve [1]. Tenotomy was carried out in parallel with colchicine application by crossing the Achilles tendon. The muscles were studied after 3 weeks. The interventions and controls were described in detail previously [2,3].

RESULTS

The guinea pig slow *musculus soleus* is homogeneous and contains no MF stained by antibodies to

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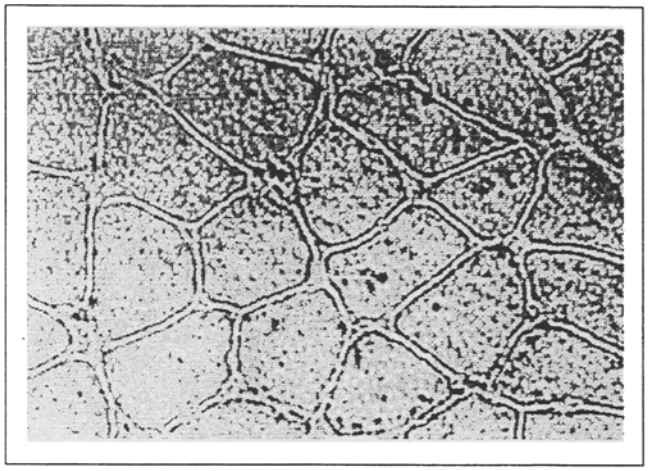

Fig. 1. Intact guinea pig musculus soleus. Here and on Figs. 2 and 3: immunohistochemical staining (PAP method) by monoclonal antibodies to fast myosin heavy chains.

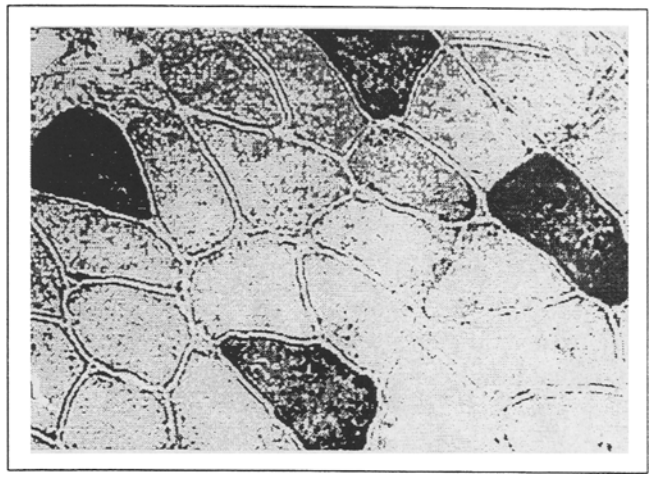

Fig. 2. Guinea pig musculus soleus after colchicine application on the sciatic nerve.

fast myosin (Fig. 1). The blockade of axon transport by colchicine application on the nerve leads to the appearance of the fast dark-stained MF in the muscle (Fig. 2), i. e., induces the production of fast myosin. The blockade of axon transport and subsequent tenotomy do not modify the initial immunohistochemical profile of the slow muscle (Fig. 3), that contains no fast MF. Thus, our results show that tenotomy prevents the expression of fast myosin in the slow muscle after colchicine application on the nerve. This indicates that pulsed motoneuron activity as a factor of neurophysiological control regulates the qualitative composition of the skeletal muscle myosins, thus maintaining the muscle functional activity.

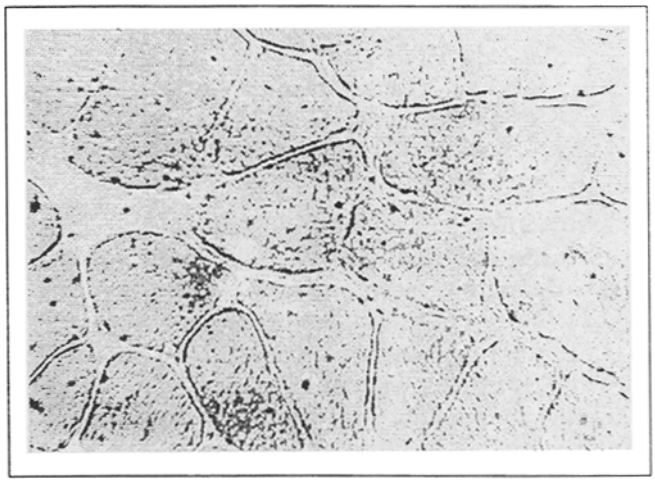

Fig. 3. Guinea pig musculus soleus after colchicine application on the sciatic nerve and tenotomy.

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