

Motor Unit Conduction Velocity during Sustained Contraction after Eccentric Exercise

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ABSTRACT

HEDAYATPOUR, N., D. FALLA, L. ARENDT-NIELSEN, C. VILA-CHÃ, and D. FARINA. Motor Unit Conduction Velocity during Sustained Contraction after Eccentric Exercise. *Med. Sci. Sports Exerc.*, Vol. 41, No. 10, pp. 1927–1933, 2009. **Background:** Eccentric contractions induce muscle fiber damage that is associated with a decreased capacity to generate voluntary force and increased fiber membrane permeability. Changes in fiber membrane permeability results in cell depolarization that is expected to have an effect on the action potential propagation velocity of the muscle fibers. **Purpose:** The aim of the study was to investigate the action potential propagation velocity in individual motor units before and 24 and 48 h after eccentric exercise. **Methods:** Multichannel surface and fine-wire intramuscular EMG signals were concurrently recorded from two locations of the right vastus medialis muscle of 10 healthy men during 60-s isometric contractions at 10% and 30% of the maximal force. **Results:** The maximal force decreased by $26.1 \pm 16.1\%$ ($P < 0.0001$) at 24 h and remained reduced by $23.6 \pm 14.5\%$ ($P < 0.0001$) 48 h after exercise with respect to baseline. With respect to baseline, motor unit conduction velocity decreased ($P < 0.05$) by (average over 24 and 48 h after exercise) $7.7 \pm 2.7\%$ (10% maximal voluntary contraction (MVC), proximal), $7.2 \pm 2.8\%$ (10% MVC, distal), $8.6 \pm 3.8\%$ (30% MVC, proximal), and $6.2 \pm 1.5\%$ (30% MVC, distal). Moreover, motor unit conduction velocity decreased over time during the sustained contractions at faster rates when assessed 24 and 48 h after exercise with respect to baseline for both contraction forces and locations ($P < 0.05$). **Conclusions:** These results indicate that the electrophysiological membrane properties of muscle fibers are altered by exercise-induced muscle fiber damage. **Key Words:** DOMS, MUSCLE DAMAGE, MUSCLE FIBER, INTRAMUSCULAR EMG

Eccentric contractions induce muscle fiber injury that is associated with a decreased ability of the muscle to generate force (29). Damage to sarcomeres (27) and failure of excitation–contraction (E–C) coupling are two prominent signs of damage in skeletal muscles after eccentric exercise (16,31,39). Thus, the cause of a force deficit after an eccentric task has been commonly attributed to a disturbance in the mechanisms involved in generating force within the skeletal muscle (1) and action potential conduction in the E–C coupling pathways (39). Decreased

neural drive to the muscle after eccentric exercise has also been demonstrated by an increase in force production with stimulation to the motor cortex or motor nerve with respect to the force obtained during a maximal voluntary contraction (MVC) (31).

The sarcolemma, which conducts the action potential, is subjected to substantial tears during eccentric contractions (26). Increased sarcolemmal membrane permeability has been indicated as one of the features of the damaged muscle fiber, as assessed by loss of soluble intracellular proteins (e.g., creatine kinase, myoglobin) and uptake of membrane-impermeant dyes by damaged cells (26). An abnormal sarcolemmal membrane permeability would also depolarize the fiber membrane because of increased intracellular sodium [Na^+] and calcium [Ca^{2+}] and extracellular potassium [K^+] (23,24).

Changes in membrane depolarization are expected to have an effect on the action potential propagation velocity of the muscle fibers. A recent animal study has shown that eccentric contractions did not affect muscle fiber conduction velocity measured during evoked twitch contractions via electrical

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