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Neuromuscular diseases: revisiting the "overtraining"

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Neuromuscular diseases (NMD) are a heterogeneous group of disorders of the anterior spinal cord, peripheral nerves, neuromuscular junction and skeletal striated muscles. It is a consensus that exercise programs may optimize the motor and cardiovascular functions, in addition to preventing atrophy by disuse and deconditioning in people with NMD. The literature suggests an individualized, submaximal approach, adapted to the peculiarities of each disorder, with solid and often reanalyzed treatment goals, in order to avoid overtraining.

There is the belief that the motor training in patients with NMD may be deleterious and cause the overtraining syndrome, characterized by the installation of symptoms which reflect a non-ideal relationship between effort stress and the tolerance to it, being externalized with the decreased of physical performance, increased muscle injuries and even immunosuppression, increasing the susceptibility to infections. Many NMD of chronic and progressive nature, and often relentless, need to be addressed with a focus on the management of muscle weakness, and not the increase in strength, rule which also applies to the muscles of swallowing and breathing.

The grueling training with intense cardio accelerates the degradation of motor performance and death in animal models of Amyotrophic Lateral Sclerosis; on the other hand, low-intensity exercises increase their survival rate. The type of exercise also appears to influence neuronal death. Swimming causes less death of motoneurons than treadmill exercises, especially the medium-sized ones which innervate phasic muscle fibers, preserving the number of astrocytes and oligodendrocytes of the anterior spinal cord and increases survival in animal experiments. It should be noted that there are few randomized controlled trials in humans with NMD verifying the effects of therapeutic resistance exercise, which makes them inconclusive. In patients with post-polio syndrome, the submaximal exercise therapy may contribute to a better control of muscle weakness, cardiorespiratory aptitude and walking pattern, and should be avoided activities which cause muscle fatigue or joint pain.

This principle is also valid for some dystrophinopathies, in which repeated and exhaustive muscle activation increases both muscle oxygen and nitrogen, affecting the contractile function. In the mitochondrial myopathies, moderate aerobic exercise was effective in improving muscle performance. Aerobic exercises on a cycle ergometer were also beneficial in order to increase VO_2 max and muscle strength in the lower limbs of patients with Becker muscular dystrophy, without causing increased levels of the creatine kinase enzyme (CK) and changes in muscle tissue or at the echocardiography. If prescribed properly and with

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caution, there is evidence that neuromuscular electrical stimulation focusing on the slow twitch fibers and the low intensity resistance exercises are also beneficial for improving the strength and functionality of dystrophic patients. However, if overtraining of the motor units occurs, functional damage will most likely occur.

As a conclusion, we may face the motor unit as a large electric generator which requires stimuli to trigger energy. A reduction of stimuli presumably causes an ineffective activity; however, too many stimuli in an already ailing system will overload it, further impairing their function. There are no protocols designed specifically for groups of NMD, nor standardized assessments. Programs should be developed based on the clinical findings of the patients and in accordance with the natural history of the disease addressed. The exchange of knowledge among professionals, the use of supportive and protective equipment, as well as psychological support, should be part of the proposed rehabilitation.