

THE CONTRIBUTION OF PHYSICAL THERAPEUTIC EXERCISES TO THE IMPROVEMENT OF MUSCLE IMPAIRMENT CAUSED BY MUSCULAR DYSTROPHY

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Abstract: Muscular dystrophies (DM) are neurological conditions of great complexity and severity that involve progressive loss of muscle mass and associated reduction in muscle strength. The causes are genetic and involve mutations in the dystrophin gene that lead to the absence or deficiency of functional dystrophin, a cytoskeletal protein that enables general muscle function. In neuromuscular dystrophies there are direct molecular mechanisms responsible for exercise intolerance and inability to contract muscles. The use of therapeutic physical exercises is a way to slow down the pregression of the disease, to combat the loss of functionality of the muscle fibers as well as to delay the appearance of secondary manifestations of the disease, manifestations that occur with the ability to ambulate and with the installation of sedentarism caused by muscle weakness and degeneration. The effects of therapeutic exercise on patients with muscular dystrophy (including the effects of exceptional or maximal exercise) have been analyzed in the literature, and it has been found that aerobic exercise performed under the guidance of a physical therapist can be effective in improving oxidative capacity and progressive muscle function of the patient. The exercises must be adapted to the individual case, depending on the patient's general condition and availability of effort. Careful supervision and monitoring of patients can achieve the objectives obtained in the form of beneficial effects for the affected muscles. The present dissemination proposes a brief presentation of different types of therapeutic exercises (aerobic, endurance, resistance) and their role in the recovery program of patients with progressive muscular dystrophies.

Key Words: Muscular Dystrophy, Muscle Degeneration, Recovery, Exercise.

INTRODUCTION

The in-depth study of neuromuscular diseases over the last few decades has brought new knowledge based on physical evidence to the scientific community. It has thus been demonstrated that physical exercise has proven to be effective in the therapeutic

management of muscular dystrophies (DM), this theory being relevantly confirmed in recent years [2].

At the same time, studies on neuromuscular diseases have disproved the fact that physical activity based on therapeutic exercises is contraindicated for patients with DM, being harmful to their general condition and responsible for the aggravation of the disease over time [12].

The accumulation of new data and the enrichment of knowledge related to the evolution of neuromuscular diseases have led to a clearer perception of the effectiveness of physical exercise in patients with DM [11]. Therapeutic exercises must be adapted to each individual patient, depending on the stage of the disease, age and general condition, so that they benefit from personalized care aimed at managing the psychosocial needs and easier integration into the social life of each patient, as well as increasing the period of time during which patients maintain their functional independence and especially ambulation [22]. The demonstration of the effectiveness of physical exercises and the access to new treatments, which have appeared during the last years, led to a higher interest in the field of physical therapy and its effects on neuromuscular diseases [20].

Therapeutic exercises have the role of reducing muscle damage, muscle contractions, preventing bone fractures and above all prolonging the patient's independence. There must be a balance between physical activity and muscle relaxation because the physiological and metabolic changes that occur during exercise, can lead to states of asthenia and muscle fatigue.

During physical efforts, the mode of energy supply changes depending on the intensity of the effort.

Maximum intensity efforts can only be sustained anaerobically, based on direct phosphorylation and anaerobic glycolysis, which is why this type of effort is called anaerobic.

Efforts of moderate or low intensity are supported by chemical reactions that occur in the presence of oxygen, the efforts being called aerobic.

In addition to the high intensity, anaerobic efforts are also characterized by a short duration, taking place with the accumulation of an oxygen debt that will be paid when

the activity is stopped and whose size is closely related to the degree of training for the effort.

The production of energy by anaerobic glycolysis in the sarcoplasm of the muscle cell represents the main energy supply when the effort is very intense and the oxygen needs of the muscle cannot be met.

Anaerobic glycolysis is available after about five seconds from the onset of exertion. The reactions specific to anaerobic glycolysis take place at a relatively high speed and over a short period of time.

Anaerobic glycolysis predominates between 30 and 60 seconds of exercise, reducing to lower values as the exercise is prolonged and energy is provided by aerobic chemical reactions.

METHODS OF APPROACHING THERAPY WITH PHYSICAL EXERCISES

For healthy individuals, physical training is a beneficial method of increasing some parameters (such as muscle strength, endurance, mobility), improving cardiopulmonary function as well as preventing the decrease in muscle mass determined by the aging process. In people affected by DM, the effects related to the action of physical activity on the previously mentioned parameters are not fully proven, they continue to be the subject of many studies carried out in the last decade. Physical exercises have demonstrated their effectiveness in preventing osteoporosis, obesity, improving cognition, increasing the quality of life of patients with DM.

However, the specialized literature does not point out whether the physical exercises are intended to stimulate the regeneration processes of the skeletal muscles and not to cause their mechanical damage. This aspect is debated in many of the studies carried out to date [2].

The sustained physical activity carried out constantly takes place according to a protocol and is based on a plan that includes objectives related to the condition of the muscles and the physical possibilities of each individual. Strength training is based on repetitive resistance exercises focused on isometric muscle contractions that are designed to increase muscle strength and endurance.

Practicing aerobic exercise or cardiorespiratory fitness involves large muscle groups and the movement can be sustained for a long time, at the submaximal threshold in activities such as walking, running, swimming, cycling.

More than half of patients with DM have severe muscle fatigue, which is an early symptom of the disease. Muscle fatigue occurs when physical activity can no longer be continued by the patient because he feels discomfort accentuated by the exaggerated effort. The occurrence of fatigue is the result of the interdependence between the three types of force involved: the force required for the movement, the maximum force produced by the myofibrils and their resistance to fatigue. Muscle fatigue is caused by the progressive loss of motor unit components caused by various mechanisms of degeneration, depending on the genotype [12].

The effects of physical exercise have been highlighted by numerous studies conducted over the past ten years. Therapeutic exercise has been shown to be effective for DM, but the type of exercise that works best for the recovery of motor deficits caused by neuromuscular diseases is still unknown. There are controversies related to the type of training that should be applied (aerobic or strength training), its duration, frequency and intensity [22].

AEROBIC TRAINING

Moderate-intensity aerobic exercise can increase oxidative capacity and improve muscle activity in patients with preserved ambulation. It can also increase the maximum volume of oxygen (VO₂max) [13]. The exercises can be performed on the cycle ergometer for half an hour a day, with a frequency of three sessions per week for a period of ten to twelve weeks [21].

Submaximal aerobic physical exercises such as swimming and cycling (carried out without significant resistance) performed under the guidance and supervision of the physiotherapist, together with other types of activities carried out in such a way as not to exceed the limit of cardiopulmonary and muscular endurance, have proven effective [17].

It is thus observed that the constant performance of submaximal, progressive exercises has a significant role in maintaining but also increasing muscle strength and preventing muscle atrophy [8].

ENDURANCE TRAINING

Endurance training based on aerobic exercise at a moderate intensity is also considered beneficial. Researchers in the field presented the effects of this type of activity in a group of patients with limb girdle muscular dystrophy type 2I (LGMD2I). They performed a number of fifty sessions of thirty minutes each on the cycle ergometer over a period of three months, at a heart rate of 65% of VO₂max. Training was shown to have a positive effect on VO₂max and maximal effort volume by 21 - 27%, similar to the physiological response to training in healthy individuals.

Plasma creatine kinase (CK) values increased after exercise in both patients and some healthy subjects.

From the analysis of the results recorded in the conducted study, it appears that the majority of patients with LGMD2I achieved an increase in physical endurance, lower limb muscle strength and walking distances. Even if no progress was registered in a limited group of patients, no adverse effects, deterioration of the general condition or processes of muscle degeneration were reported [15].

RESISTANCE TRAINING

Studies related to the results obtained by carrying out both low-intensity and high-intensity resistance training in patients with LGMD2I, LGMD2A and Becker Muscular Dystrophy have also been conducted [16].

For those who worked out at low intensity, the resistance training took place over a period of six months and targeted the quadriceps muscle and the biceps brachii muscle. The weight worked for knee extension and elbow flexion started at 40% of maximal strength and was increased by 5% in each of the following weeks, with a significant increase in maximal muscle strength and endurance (the maximum number of repetitions possible at 60% of maximum force) [16].

Plasma CK was analyzed every month throughout the duration of the training, being a relevant indicator of the damage produced by the muscular effort.

In the case of the group that worked at an increased intensity, the training took place over a period of three months with a frequency of three sessions per week. Several muscle groups were worked on, with patients being assessed every month for maximum endurance strength. At the end of the training, after three months, there was an increase in the strength of the subjects in the study group for flexion and extension movements of the stylo-radial joint.

The findings of this study highlight that resistance training poses no risk to the patient and is beneficial for improving muscle strength and endurance in DM patients with proximal muscle weakness (LGMD). The results obtained were satisfactory, the patients tolerated the working conditions well: the exercises performed did not cause them muscle damage, muscle fatigue or other types of accidents, but the basic condition is that the patients are constantly supervised and guided by the physiotherapist.

DM produces changes in cardiac and skeletal muscle that involve progressive loss of muscle mass and reduced muscle strength. The inability of muscle fibers to maintain their contractile capacity leads to the degradation of the sarcolemma, the degeneration of myofibrils and their necrosis [4].

In DM, the main reason considered responsible for the reduction in muscle strength is the loss of muscle mass. Various molecular processes are also involved here, such as the instability of the dystrophin-sarcoglycan complex [3] in dystrophinopathies and sarcoglycanopathies, the deregulation of calcium ion Ca^{2+} homeostasis in skeletal muscle, the connection with interstitial tissue, such as congenital DM, the excitability of the sarcolemma and the instability of messenger RNA in myotonic dystrophies [18]. In other situations, there are additional molecular pathophysiological mechanisms, such as changes in excitation–contraction coupling, congenital myopathies due to Ryanodine receptor 1-related myopathies, or protein catabolism in inclusion body myositis [7].

Low-intensity exercise with a number of exercise repetitions similar to strength training in healthy subjects has also been beneficial in patients with McArdle [10] and lipid myopathies (myopathies with abnormal lipid metabolism) [6].

Patients with LGMD2A and LGMD2B performed isometric leg flexions starting at 20% of maximal voluntary contraction (MVC) and gradually increasing by 10% MVC after every 30 seconds. After the end of exercise, the recovery rate of phosphocreatine in LGMD2A patients was considerably reduced compared to LGMD2B and healthy subjects which indicates the alteration of their oxidative metabolism [9].

Along with the decrease in intracellular pH due to anaerobic glycogenolysis produced after aerobic exercise, the consequences of ionic changes can lead to muscle fatigue [1].

CONCLUSIONS

The use of high-performance imaging methods (magnetic resonance, musculoskeletal ultrasound) allowed obtaining new data on skeletal muscle activity analyzed in studies investigating muscle fatigue [14], [5].

Advances in imaging allow an in-depth study of the muscle contraction process and implicitly how it is affected by neuromuscular diseases.

The role of physical exercises has been highlighted in many of the studies that have focused on demonstrating the effectiveness of therapeutic exercises in the process of slowing down the progression of functional disorders caused by muscular dystrophies. Recovery therapy based on the performance of physical exercise has proven effective in combating the appearance of secondary manifestations of muscular dystrophies, as well as in delaying the establishment of sedentarism caused by muscle weakness and degeneration.

The essential condition for physical exercise to be beneficial for patients affected by DM is that the recovery plan is individualized, adapted to the movement possibilities of each patient, so that the patient tolerates the effort and does not suffer additional or various injuries complications that affect his general condition and alter his quality of life.

The recovery of patients with DM must combine exercise therapy with new therapies discovered in the field of treating disorders caused by neuromuscular diseases. The literature mentions the importance of combining these types of therapy for effective multidisciplinary management of DM.

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