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EDITORIAL

Skeletal muscle atrophy: relationship between basic and applied sciences (Kinesiology/Physiotherapy)

Skeletal muscle atrophy occurs due to a decrease in the synthesis pathways and/or an increase in protein degradation pathways. Once the size of the muscle fiber is affected, the individual loses muscle mass and strength and, consequently, the functional capacity.

Molecular biology techniques show that, in a condition of skeletal muscular atrophy, such as muscle disuse of a bed-bound patient, the markers involved in protein degradation pathways increase significantly in the first days (1-3 days), but decrease afterwards. On the other hand, the markers involved in the protein synthesis pathways suffer a constant decrease over the days without anabolic stimulus, which, in the case of the subject on bed rest, would be the mechanical load[1]. When the degradation pathways prevail over the synthesis pathways, the result is the loss of skeletal muscle mass and a decrease in the functionality of the subject[2,3].

As kinesiologists/physiotherapists, we see several conditions that can lead to a state of skeletal muscle atrophy, including: disuse, immobilization, catabolic diseases, such as cancer, or natural processes, such as aging. The effects may differ among the distinct causes of skeletal muscular atrophy, for example, young subjects submitted to seven days of immobilization had a loss of 220 grams muscle mass in their immobilized lower limb as opposed to being seven days on bed rest, when they had a loss of 140 grams muscle mass in each lower limb (with greater total body muscle loss). This accounts for ~0.6% loss of muscle mass in the lower limb per day of atrophy. Additionally, muscle strength was affected even further, showing ~1.3% loss per day[4].

Recent scientific literature and international congresses mention the concept of "anabolic resistance," which describes the reduction of skeletal muscle sensitivity/response to an anabolic stimulation. In general, this concept of "anabolic resistance" would be responsible, in part, for the typical loss of skeletal muscle mass in conditions such as disuse, aging and some diseases[5]. Several strategies have been used to counteract skeletal muscle atrophy: physical exercise, nutritional supplementation, electrical stimulation, among others. By measuring muscle protein synthesis in humans through muscle biopsy, resistance training has shown a greater potential to increase skeletal muscle mass. Moreover, the combination of physical exercise with an adequate nutritional protocol could increase those beneficial effects.

The evidences raised by basic sciences over the years, such as the ones mentioned above, show that kinesiologists/physiotherapists are indispensable from the first minute the subject is affected by skeletal muscular atrophy, such as entering an Intensive Care Unit or being partially/completely immobilized due to accident injuries. The rehabilitation practice should be intensified during conditions of disuse or immobilization in special populations (older adults, subjects with cancer or diabetes, among others), for those with a predisposition to develop skeletal muscle atrophy. Hence, the implementation of more hours of rehabilitation in different health facilities during the day, night and weekends is theoretically coherent, otherwise we will have subjects with a lower degree of functionality and, in the long run, greater functional dependency and health costs for the country.

The current recommendation for the aforementioned cases is to consume adequate amounts of proteins/amino acids, perform early stimulation using mechanical loading (through electrical stimulation, therapeutic exercise, ambulation, etc.) and increase, according to the conditions of the subject, the anabolic stimulus in special populations.

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