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Short Communication

Therapeutic Exercise in Amyotrophic Lateral Sclerosis: What do we Expect from Anabolism Versus Catabolism?

Marco Orsini[1,2], Mauricio de Sant' Anna Jr[3], Marcos RG de Freitas[4], Eduardo Trajano[2], Manuel Leite Lopes[5], Acary Bulle Oliveira[6], Silmar Teixeira[10,11], Sergio Nader[8], Nélio Souza[9] and Victor Hugo Bastos[7]*

[1]Postgraduate Program in Science Rehabilitation, UNISUAM, Brazil

[2]Professional Master's Degree in Applied Science in Health, Severino Sombra University, Vassouras, Brazil

[3]Professor of the Federal Institute of Rio de Janeiro IFRJ, Department of Physical Therapy, Brazil

[4]Medicine Department, Rio de Janeiro Federal University, UFRJ, Brazil

[5]Physiotherapist, Clinica Sinapse, RJ, Brazil

[6]São Paulo Federal University, UNIFESP, SP, Brazil

[7]Associate Professor, LAMCEF/LAMPLACE, Professor of Master's and PhD in Biomedical Sciences and Biotechnology UFPI-CMRV, Brazil

[8]Professor from Iguaçu University, Brazil

[9]Professor of the Physiotherapy Course at the Serra dos Órgaos University (UNIFESO); Doctoral Student in Neurosciences by the Federal Fluminense University (UFF), Niteroi, RJ, Brazil

[10]Associate Professor, LAMPLACE, Professor of Master's and PhD in Biomedical Sciences and Biotechnology UFPI-CMRV, Brazil

[11]Associate Professor, LAMCEF, Professor of Master's in Biomedical Sciences UFPI-CMRV, Brazil

***Corresponding Author**: Victor Hugo Bastos, Associate Professor, LAMCEF/LAMPLACE, Professor of Master's and PhD in Biomedical Sciences and Biotechnology UFPI-CMRV, Brazil.

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Amyotrophic lateral sclerosis (ALS) can be defined as a progressive, degenerative and inexorable neurological disease, which genesis is still difficult to understand. Several factors contribute to deprogramming and early cell death. The search for a better interrelationship in new markers and associations with distinct cellular and/or molecular types seems to be the great challenge. The design of clinical research in (ALS), with information related to drug type, action and medicine dose, emerges as new theories are presented and added to the current model [1,2].

Definitely ALS is a multifactorial disease. Oxidative stress, glutamate-mediated excitotoxicity, effects caused by mutation of superoxide dismutase, abnormal protein-specific aggregation, disruption of intermediate neurofilaments, alteration of anterograde and retrograde axonal transport, microglial activation, inflammation, and growth factor disorders, have been considered as potential aggressors to motor neurons. Genetic factors, excessive influx of intracellular calcium and apoptosis are also part of this understandable but still indecipherable theoretical model [3].

Based on the above on the pathophysiology of ALS, as well as on the clinical manifestations of this disease, it becomes evident the need for multidisciplinary action, especially as far as the physiotherapist is concerned, since the moment of clinical diagnosis, as he will be responsible for the prescription of therapeutic exercises.

But what we expect from the relation between anabolism vs. catabolism? Since the initial phase of ALS, therapeutic exercises of the most diverse natures (aerobic, resistive, respiratory, etc.) are recommended, however the chain of devastating events inherent to the pathophysiology of ALS is already in progress. Respecting both the therapeutic objectives and the phases of ALS, the prescription of muscle strengthening and physical reconditioning become components of the treatment program established by physical therapy. Several studies are found in the literature - many support their practice - but some have controversial results [4-6].

We can divide the daily energy expenditure into three components: basal metabolic rate, thermal effect of food and energy expenditure associated with physical activity. Performing a physical exercise promotes an increase in total energy expenditure, both acutely and chronically. Regarding the acute effect, it is well established that after the end of the activity, the oxygen consumption (VO_2) does not return to the resting values right away. This energy demand during the post-exercise recovery period, which is necessary to "remove assumed metabolic debt during activity" is defined as excess post exercise oxygen consumption or excessive oxygen consumption after exercise (EPOC) [7,8]. Due to the great metabolic imbalance that involves ALS, and the frank catabolic state, the question to be asked is: Won't the intensity of the prescribed exercise produce an EPOC effect, speeding up the disease process? Would then that "metabolic debt" be another work to be carried out by a weakened body and in open catabolism?

There are new therapeutic possibilities in a pathophysiological framework still under construction, one of them is the proposal of the Deanna protocol [9] that suggests the supplementation of arginine-alpha-ketoglutarate, complex B, among others could attenuate mitochondrial dysfunction, glutamate excitotoxicity and oxidative stress. It is essential that the exercise prescription be at submaximal intensity (40 - 60% VO_2peak, or 60 - 85% HR_{max} or 10-13 on the Borg scale) both by the aspect involving the EPOC effect, as well as by the effects directly related to the metaborreflex of the peripheral and respiratory musculature. Based on these concepts, we do not recommend the prescription of muscle strengthening and physical reconditioning and ALS patients, but we suggest that all these aspects be included in the prescription [10].

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