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

Marco Orsini¹,², Mauricio de Sant’ Anna Jr³, Marcos RG de Freitas⁴, Eduardo Trajano², Manuel Leite Lopes⁵, Acary Bulle Oliveira⁶, Silmar Teixeira¹⁰,¹¹, Sergio Nader⁸, Nélio Souza⁹ and Victor Hugo Bastos⁷*

¹Postgraduate Program in Science Rehabilitation, UNISUAM, Brazil
²Professional Master’s Degree in Applied Science in Health, Severino Sombra University, Vassouras, Brazil
³Professor of the Federal Institute of Rio de Janeiro IFRJ, Department of Physical Therapy, Brazil
⁴Medicine Department, Rio de Janeiro Federal University, UFRJ, Brazil
⁵Physiotherapist, Clinica Sinapse, RJ, Brazil
⁶São Paulo Federal University, UNIFESP, SP, Brazil
⁷Associate Professor, LAMCEF/LAMPLACE, Professor of Master’s and PhD in Biomedical Sciences and Biotechnology UFPI-CMRV, Brazil
⁸Professor from Iguacu University, Brazil
⁹Professor of the Physiotherapy Course at the Serra dos Órgãos University (UNIFESCO); Doctoral Student in Neurosciences by the Federal Fluminense University (UFF), Niteroi, RJ, Brazil
¹⁰Associate Professor, LAMPLACE, Professor of Master’s and PhD in Biomedical Sciences and Biotechnology UFPI-CMRV, Brazil
¹¹Associate 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.

Received: March 30, 2017; Published: April 21, 2017

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 deplored 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].

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

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 (VO2) 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% VO2 peak, or 60 - 85% HRmax 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 metabolorreflex 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].

Bibliography


Volume 6 Issue 1 April 2017
© All rights reserved by Victor Hugo Bastos, et al.