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Central and Peripheral Fatigue in Physical Effort: A Mini-Review

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ABSTRACT

Costa PNC, Musialowski R, Palma A. Central and Peripheral Fatigue in Physical Effort: A Mini-Review. **JEPonline** 2019;22(5):220-226. Fatigue is considered to be a defensive mechanism of the organism, as a means to preserve the neuromuscular system when facing intense physical effort situations, reducing the capacity of producing force. However, there is evidence of peripheral mechanisms, pointing to changes in the neuromuscular junction and the muscle fiber membrane (sarcolemma) that lead to a marked decrease in the mechanisms of ATP production, which to a certain extent hinders the connection between myosin and actin. The result is a type of fatigue, together with the combined accumulation of muscle metabolite sub-products such as extracellular K⁺, H⁺, ADP, Pi ions, Mg⁺, and La⁻, as well as the formation of reactive oxygen species, the decrease in the resting potential of the membrane, and the ATP and PC production plus changes in the calcium pump of the sarcoplasmic reticulum. When a central fatigue is detected, there are neuromuscular junction faults as a result of the neural signal alteration that reaches the muscle, as well as other aspects, such as: increased K⁺, H⁺, inorganic phosphate, bradykinin, prostaglandin, ammonia, and decreased plasma glucose, muscle and liver glycogen, hyperemia, plasma tryptophan and 5-hydroxytryptophan. However, the motivation reduction, pain tolerance, deficit of reasoning and attention, decreased peripheral motor

coordination, central nervous structures, transmission of electrical impulses from the spinal cord to the motor nerves and motor neuron recruitment are also considered as indicators of central fatigue. These factors depend on the intensity, frequency, duration, types of exercises, locations where the exercises are performed, environmental factors, and levels of individual physiological conditioning and biochemical adaptations. Coaches and athletes should adjust their athletes' training loads and nutritional aspects to minimize the deleterious effects of fatigue to obtain performance improvement.

Key words: Central Fatigue, Fatigue, Performance, Peripheral Fatigue

Biochemical, physiological, hormonal, hematological, mechanical, and psychological factors can influence the fatigue that occurs during physical exercise, and it can be considered as the disability or loss of strength production by the muscular system (33). On the other hand, exhaustion refers to the intense feeling of effort that results in reduced motor power and the lack of willingness to continue exercising and, consequently, interrupts the workload (4).

Fatigue is considered as a defense mechanism of the human body. In fact, it is a way to preserve the integrity of the neuromuscular system when faced with situations of intense physical effort. Fatigue decreases the capacity to produce muscle contractions according to the required work (36). For this reason, one of the measuring mechanisms is muscle torque (27) and biochemical tests to analyze metabolic and other components that either decrease or increase the capacity of the neuromuscular system to continue working. (3).

However, one of the first responsible nucleotides for the storage and source of energy is the ATP molecule, adenosine triphosphate (1). Continuous exercise can decrease ATP, which has a negative effect on continued contraction of the muscles. Hence, when the ATP levels are low, they need to be regenerated to allow for the continuous production of force during physical exertion (18).

Also, it is important to point out that fatigue can be divided into: (a) central fatigue that results from changes in cortical and medullary mechanisms; and (b) peripheral fatigue that results from changes in muscle structure and/or junction, which are caused by complex factors that are yet fully understood (5,12). However, this division does not mean exclusivity in an area (13) that will depend on the intensity, volume, and type of physical exercise (8).

Blundell et al. (6) reported that the peripheral mechanisms are derived from changes in the functioning of the peripheral nerves, neuromuscular junctions, and electrical activities in the muscle fiber membrane (sarcolemma) or muscle structure that lead to a marked decrease in the mechanisms that produce ATP (6). For example, the decreased production of ATP is directly related to the depletion of phosphocreatine, which causes a low re-phosphorylation of ADP to ATP. The resulting effect is an increase in the concentration of ADP (i.e., adenosine diphosphate) and cyclic adenosine monophosphate (cAMP) that to some extent will make it difficult to connect the cross-bridges between myosin and actin (18,19,26).

Peripheral fatigue may lead to muscle fiber ischemia, which limits the blood flow to the muscles and, therefore, induces the activation of the anaerobic pathway (5). The ischemia

can also lead to the accumulation of muscle metabolites produced during anaerobic metabolism, such as the release of hydrogen ions (H^+), sodium lactate (La^-), and inorganic phosphate (Pi) (3,5). However, McCully et al. (20) indicate that the decrease in the hydrogen ionic potential (pH) is more pronounced at low temperatures than at high temperatures. The low pH causes a buffering by bicarbonate that leads to the release of carbon dioxide, which can change the respiratory quotient (31).

These factors lead to a decrease in the concentration of intracellular calcium (Ca^{+2}) by the calcium pump of the sarcoplasmic reticulum (16), which will decrease the intracellular pH (20). This limitation of the release of Ca^{+2} and inhibition in the opening of Ca^{+2} channels may lead to the accumulation of magnesium ions (Mg^+) in the sarcoplasm (2,28). High levels of Ca^{+2} lead to increased production of reactive oxygen species (15,24), and that oxidative stress may be one of the main causes of the decreased capacity to generate strength by the muscle fibers (11). In the extracellular environment, there is an increase in potassium (K^+) that can block the action potentials, altering the process of excitation and contraction (21). As for the mechanical alterations, ruptures occur between the transverse tubules and the sarcoplasmic reticulum, compromising muscle contraction (2).

Central fatigue is commonly related to long-term exercises (endurance), there is evidence of changes in the neural signal with a decrease in the frequency of stimuli that cause neuromuscular junction failure (6). Other factors causing failure include: (a) an increase in extracellular and intracellular K^+ and Na^+ that will reduce the Na^+ and K^+ pump function with a reduction in the potential of the action for depolarization of the muscle fibers (2,34); (b) an increase in the output of H^+ ions from the mitochondrial matrix to the intermembrane space; (c) an increase in Pi , with its entry into the sarcoplasmic reticulum, linking to Ca^{+2} , reducing the number of cross-bridges formed (7), bradykinin (in response to vasodilation); and (d) an increase in prostaglandin (response to the process of muscle regeneration) (29). They may transmit inhibitory information to the central nervous system (CNS) through the afferent sensory nerves (9,16,18). The increase of ammonia (NH_3) when produced by the skeletal muscle through the hydrolysis of ATP or amino acids crosses the blood-brain barrier, causing an accumulation in plasma, increasing the brain uptake of NH_3 and may influence the brain neurotransmitters (23). There is also a release of interleukin 6 (IL-6) that is considered a predecessor of the feeling of fatigue (25,30). On the other hand, there is a decrease in plasma glucose, in which the neural cells responsible for motor control will consume too much of it (23). The metabolization of liver and muscle glycogen may be slower (in trained individuals) or faster (non-trained individuals) that also contributes to central fatigue (12).

There is also an increase in plasma tryptophan and 5-hydroxytryptophan (a precursor of cerebral serotonin), which is a neurotransmitter that induces central fatigue (4). The degradation of free fatty acids in plasma induced by exercise must be transported by albumin (32). However, tryptophan competes with albumin to be transported. When there are too many fats, albumin prefers to transport fats, leaving tryptophan free. Then, this amino acid passes to the CNS, which will be transformed into the neurotransmitter serotonin that will cause fatigue (14). At the same time, the stimulation of the sympathetic nervous system will occur and produce an increase in cardiac contractility and its frequency along with bronchodilation and an increase in respiratory breaths per minute (22).

In addition to the above mentioned factors, a decrease in motivation, pain tolerance, deficit of reasoning and attention, peripheral motor coordination, changes in the CNS structures, changes in the transmission of electrical impulses from the spinal cord to the motor nerves and/or recruitment of motor neurons are considered as indicators of central fatigue (17). Therefore, these factors depend on the intensity, frequency, duration, types of exercises, places where the exercises are performed, environmental factors, and levels of individual conditioning specific to physiological, hematological, biochemical, and hormonal adaptations (10).

Coaches, athletes, and recreational athletes should adjust their training loads and evaluate their nutritional status to minimize the deleterious effects of fatigue in order to obtain performance improvement.

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