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Meeting Abstract

Arthrogenic Muscle Response Post Rupture of the Anterior Cruciate Ligament

Respuesta muscular artrogénica pos rotura de ligamento cruzado anterior

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Arthrogenic Muscle Response (AMR) is defined as the phenomenon of inhibition (AMI) or facilitation of the periarticular muscles of a joint that has suffered trauma. The AMR affects the muscles directly related to the traumatized joint, as well as those of neighbouring joints and even, according to some authors, in a minor degree to the joint muscles of the contralateral limb (1-3).

The AMR is described as a protective reflex mechanism that limits the normal use of a joint to prevent an increased damage. The duration of this mechanism should be consistent with the stages of tissue repair, but, for unknown reasons until today, sometimes persists for months and even years, even when the damaged tissues have been repaired (4,5).

The AMI appears in an early post trauma stage, generating functional impotence and its persistence over time inevitably leads to a muscle atrophy that is difficult to recover, which imposes a great challenge to the rehabilitation process (6).

In cases of a rupture of the anterior cruciate ligament (ACL), a pathology that primarily affects athletes aged between 15 and 30 years, the AMR causes the inhibition of the quadriceps, especially of the vastus medialis oblique (VMO) and increased the excitability the soleus, the hamstrings and hip extensor musculature during walking as a compensatory neuromuscular strategies to facilitate motion and protect the injured joint. This is because the action of the quadriceps is antagonistic to the action of ACL, so its action can increase the damage. In addition, the action of the soleus and hamstrings is synergistic with the action of the ligament, therefore acts as a protective mechanism. This phenomenon prevents the subject to achieve the full extent of his knee (7,8).

There is a close relationship between the ACL rupture and the development of premature osteoarthritis (OA), which occurs between 5 to 10 years post injury, even when a successful reconstruction surgery have been made within a sensible period and followed intensive rehabilitation treatment. This is a significant fact, as subjects may show signs of OA from the second or third decade of life, which has a great impact on personal and financial cost, since with the course of the years, it might be necessary to get a joint replacement. (9-11).

Persistent IMA may be related to the development of OA, since a poor control of the vastus medialis exposes the joint to a dynamic instability, which produces an abnormal distribution of the forces acting on the knee. This is so because the action of the quadriceps is not only motor but also protective by providing dynamic stability to the joint by properly distributing the forces acting on the joint.

Mechanisms involved in AMI

Regulation of the muscle contraction is done in the pool of neurons and interneurons located in the anterior horn of the spinal cord. In this center, peripheral afferents coming from proprioceptors are integrated with the efferent information coming from the higher centers. The structural and functional indemnity of this neural circuitry is essential for the proper control of the muscle activity.

The mechanisms involved in the AMI are still under study. In cases of a ruptured ACL, it is postulated that the AMI (injury with more published studies) would be mainly triggered by the discharge of the capsular mechanoreceptors Ruffini and Pacini, which are activated by the joint swelling that occurs next to the articulate damage and also, although in minor extent, by the loss of the afferents coming from the mechanoreceptors like Golgi that innervates the LCA, which are destroyed as a result of the injury. Meanwhile, the discharge of nociceptors from the damaged structures is apparently unrelated to the AMI's phenomenon since some studies showed the presence of AMI in the absence of pain, with intra articular experimental infusions of only 10 ml.
It is also relevant to consider the important effect of modulation, inhibition or facilitation of the muscle contraction applied by the higher centers through descending pathways on the interneurons circuit of the anterior horn of the spinal cord (1,8,12).
For the physiotherapist, it is necessary to early identify the presence of an intense and/or persistent AMI, since the treatment protocols normally applied on the rehabilitation post-reconstruction of ACL, are not focused on the treatment of AMI per se, but on the treatment of pain, of a swelling, of the recovery of motion range, of the muscle weakness, and in general terms of the functionality. These goals are not easily accomplished if we are in the presence of a persistent AMI; so in order to achieve the treatment goals, previously resolve this inhibition is a priority. Know the neurophysiological mechanisms involved in IMA, will allow us to deal with this phenomenon in order to achieve a more successful rehabilitation treatment.

Treatments based on cryotherapy, along with a neuromuscular electrical stimulation, the use of the miofeedback and some types of exercises are therapeutic alternatives to be explored to overcome AMI, so the subject can recover an optimal recruitment of the muscle fibers and hence

muscle function to achieving a dynamic stability and thus, decreasing the probability to present a premature OA (6,12-15).

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