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Opinion

Neural Mechanisms and Perspectives about the Therapeutic Exercises for Knee Osteoarthritis

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DESCRIPTION

Osteoarthritis (OA) is the most common arthropathy in the worldwide, which is characterized by the degradation of articular cartilage and affects all joint structures such as the sub-chondral bone, fibrocartilage, ligament, capsule, and synovium. The knee OA is highly prevalent in individuals of both sexes over 50 years old, and the high levels of pain lead the loss of functional capacity and quality of life. The quadriceps weakness is a common finding in patients with OA, symptomatic or not. The disuse due to pain lead for muscle atrophy, however, the neural inhibition also has been reported in individuals with OA, and it's has been called of Arthrogenic Muscle Inhibition (AMI). This term describes the inability to contract a muscle completely, and is considered a reflex response to joint injury because it's beyond conscious and voluntary control, which can reduce about 60% of the quadriceps strength. In the OA the AMI and quadriceps weakness would be linked to inflammation, pain, joint laxity and damaged structures and therefore the IMA would be triggered by changes in the firing of sensory receptors due to damage in the knee joint, and changes in the excitability of spinal pathways, which could affect the performance of functional activities. Although studies have reported differences in the muscle recruitment pattern and kinematic and kinetic changes in functional activities such as walking, going up and down stairs and in simulations of muscle deficiency, other studies not found differences in these variables in the same conditions. This discrepancy of results may be related to the speed of the movement in which the task is performed. Individuals with OA tend to reduce the speed of movement because of the pain. Also, these differences can be related with subgroups of the individuals with OA. The modification of the spinal pathways could be related to pain, which may be local, but also affect distant areas of the knee. The pain can become chronic in patients with OA and the intense nociceptive information of the inflamed and painful structures lead to metabolic changes and reorganization of the spinal cord. The term secondary hyperalgesia refers to an increased sensitivity of neurons in the spinal segments corresponding to the primary site of pain. Identified clinically hyperalgesia in disabled individuals with knee OA. Generalized hyperalgesia over superficial and deep structures suggests that central and peripheral nervous system may be involved in the maintenance of chronic pain. It can be seen therefore that the relationship between pain, proprioception, and functional performance is not clear yet. For those reasons there are several conservative treatments for knee OA. The fact that the knee OA has multifactorial causes makes it plausible that the treatment can also be multimoda. However, therapeutic exercises are strongly recommended, especially quadriceps strengthening, aerobic and aquatic exercises. The quadriceps strengthening appears to have greater efficacy in reducing symptoms. The cause of this efficacy is the reduction of shear force and improves the distribution of forces on the knee, facilitating neuromuscular control.

CONCLUSION

There are not studies investigating the effect of the strengthening exercise in the mechanisms of peripheral and central pain, although the improvement in pain after strengthening exercise is frequently reported (assessed through questionnaires and scales). Furthermore, the characteristics of exercise, such as, how to make (uni or multi-joint exercise) and the application techniques (intensity, duration and frequency) more appropriate for treatment of knee OA is still a gap in the literature. Therefore, future research should be conducted to understand the mechanisms by which therapeutic exercise can help to reduce peripheral and central pain, in the neuromuscular control and its interaction with the other therapeutic modalities.

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