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Trunk muscle activation patterns in subjects with low back pain

Obrasci aktivacije mišića trupa kod osoba sa lumbalnim sindromom

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Introduction

Low back pain (LBP) is nowadays one of the most widely experienced health-related problems. First symptoms usually appear between the age of thirty and fifty, although it has been recorded in athletes in early twenties [1–3]. Etiology of LBP is multifactorial and it is thought that non-specific LBP is associated with lumbar spine instability [4, 5]. It is generally believed that spinal instability (such as in circumstances with no neurological deficit, deformity, or the presence of pain) is associated with reduced capabilities of neuromuscular system to respond to physiological loadings [4].

The spine is a mechanically complex structure which is inherently unstable. Due to the redundancy in the neuromuscular and spinal system, there is a large set of possible muscle activation patterns to meet the stability constraints [6]. However, different muscle activation patterns can significantly affect the magnitude and direction of the intervertebral loadings and, therefore, the spinal and core stability.

Muscle activation patterns when either retaining different postures or performing various movements and their relationship with LBP is of much interest for researchers and the clinical practice. Theoretically, the unbalanced activation and coactivation may lead to a mechanical imbalance of the whole body system [4, 6]. However, there is a disagreement among the researchers to what extent the changes in trunk muscles activation level and recruitment patterns contribute to the presence of pain (and later on to its reduction) and the altered core stability [7]. However, research in the area of motor control has made a significant contribution to understanding of the neuromuscular reorganization due to LBP.

Core stability and the role of deep trunk muscles

Core (or trunk) stability has been frequently emphasized in the literature. It is usually operationally defined as the body's ability to control the spine (e.g. to maintain or regain the balance) in response to internal and external perturbations. Panjabi [4] presented a conceptualization of core stability based on 3 systems of control: active (muscles), passive (passive stabilizers) and neural control unit. Conversely, Borghuis et al. [7] see the core stability as a product of motor control and the muscular capacity of the lumbar-pelvic-hip complex.

The importance of the two deep (local) trunk muscles, *m. transversus abdominis* (TrA) i *m. mulitfidus* (MF), has been particularly emphasized in the concept of core stability [2, 8, 9]. In particular, TrA has received a lot of attention as the main factor which provides anterior core stability, while MF provides dynamic control of segmental inter-vertebral motions inside the neutral zone. Cocontraction of these muscles increases the intra-abdominal pressure and presumably provides the stability and stiffness of the lumbar spine [8, 10] and which could possibly be either a cause or consequence of the nonspecific LBP.

Trunk muscles' activity

Differences in motor control and trunk muscle function of LBP in healthy individuals have been frequently reported in the literature [1, 5, 10–19]. These differences may either constitute a predisposing factor for low back injuries or a compensation mechanism aimed to stabilize the lumbar spine [5, 11–13]. Different hypothesis and models have been pro-

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posed in the attempt to explain the effects and mechanisms of LBP related changes in motor control, but the majority of them could be grouped into the following two main theories: changes in muscle activity cause spinal pain ("pain-spasm-pain model"), and changes in muscle activity serve to restrict spinal motion ("pain adaptation model") [11].

Under the assumption that the muscle activation pattern is altered in patients with LBP (e.g. an altered recruitment, delayed activity, asymmetrical activity of contralateral muscles and others), the researchers have recorded their elektromiographic (EMG) activity under various conditions (different contraction types, difficulty and movement complexity [1, 3, 6, 11, 12, 16, 18, 20–30]). The results have indicated that TrA and MF are primarily affected by LBP [10], as well as that the individuals with LBP (as compared with the healthy ones) have a decreased ability of activation of deep muscles during the trunk flexion motion [12, 13], suggesting that the presence of differences in activity between deep and superficial trunk muscle is consistent [12, 16, 24]. Even though changes in their function relates to LBP etiology, some researchers emphasize that the muscle weakness could be a consequence of pain and the associated inactivity [9, 24, 25]. Earlier findings of Hodges [8], Hodges et al. [12] and Hodges and Moseleyet [16] revealed an association of the rapid limb movements and delayed latency in TrA activity of LBP subjects. Changes were also observed within MF [12, 16, 29] in the form of both a hypoactivity and delayed activation during the expected and unexpected perturbations. In more complex bimanual aiming tasks, an additional load applied upon the subjects' hands caused a delayed onset of MF and errector spinae (ES) as compared to deltoid muscle, associated with both a lack of activity of abdominal muscles and longer overall movement time than in their healthy peers [15]. During the period of induced pain, the response of TrA, *obliquus extermus abdominis* (OE), and ES to arm flexion was both delayed and reduced, but with earlier onset of MF [12, 18, 19], while changes in other muscles were more variable and also dependable on the movement phase. The muscle activation pattern remained altered even after the pain disappeared, which implies the possibility that even the smallest exposure to pain stimuli may have long lasting consequences on motor control. It also remains possible that the pain represents a delayed adaptation which develops and progresses over time, in an attempt to provide the necessary trunk stifness and stability through enhanced muscle activation, and thus fights with the symptoms [1, 4, 7, 8, 18].

In contrast to healthy individuals, the patients with LBP also demonstrate significantly different muscle response pattern in response to a sudden load release. As found by Silfies et al. [14], adding an external load during the task performance induces increased activity of ES, MF and *rectus abdominis* (RA), but not in *internus obliquus abdominis* (IO) and *externus obliquus abdomimis* (EO). Abdominal (IO/RA) and extensor (MF/ES) synergist ratios become decreased, and since activity of ES is likely to be significanlty more increased than in RA, the flexor/extensor ratio could also decrease. Individuals with chronic LBP also demonstrate a decreased variability in anticipatory postural adjustments [19, 21], which suggests a reduction in the repertoire of motor control strategies utilized to reestablish the posture. It has been also observed that the gait of LBP patients is accompanied by poorly coordinated activity of the lumbar ES [17, 22]. Since the pain intensity, fear and disability were unrelated to the observed changes, it suggests that the discussed impaired coordination could be a direct consequence of LBP *per se* [16, 17, 22].

Measuring muscle activation patterns during various perturbations as a way to evaluate the efferent response to proprioception may be important in rehabilitation. If these patterns can be normalized, than proprioception may be improved through the well-planned exercise program. As it was found by Newcomer et al. [13] in both toes-up position and medium amplitude forward movement, RA shows decreased activation, while asymmetry was observed between muscle pairs both in RA and ES. Similar findings regarding the RA muscle were obtained by Hodges et al. [12, 16]. Recordings of muscle activity during different arm movement velocities showed absence of abdominal muscles activity when arms were moved slowly. The results suggested that LBP was associated with the increase in the velocity threshold required to induce abdominal muscle response. In addition, delayed activation of the abdominal (including IO and EO) and lumbar paraspinal muscles was observed prior to expected and following the unpredicted perturbations [5, 12, 19, 26].

LBP patients also demonstrate the inability to turn off the agonist and turn on the antagonist muscles during unanticipated extension moments around the trunk [24], accompanied with slower reaction times and less forceful corrections of ES activity. Jacobs et al. [21] believe that the history of LBP is associated with higher baseline of ES and RA activation, as well as that the EMG responses are modulated from this activated state rather than exhibiting sudden burst activity from a quiescent state. Consequently, if the ability to independently modulate, relax, or decouple muscle activity is compromised (as found in chronic LBP conditions; [12, 16, 19, 21, 24], the ability to safely reestablish posture and balance following an unexpected event could also be compromised.

Trunk muscle response to exercise interventions

Intents to remodulate the acitvation paterns of trunk musles with active therapy (general and stabilizing exercises) have revealed confounding results. Some researchers emphasized the importance of TrA specific exercises (e.g. bracing and hollowing) in prevention and treatment of LBP [8, 12, 16, 20, 27–30], wile others suggest that the importance of deep muscles has been overestimated and, therefore, specialized exercises unjustified [9, 10, 25]. Although causal relationships between the alterations in TrA activation and appearance of LBP cannot be implied, the research results [17–20] suggest that TrA specific exercises might contribute to the long-term symptom improvement through assistance in dynamic spine stabilization during functional tasks. Regaining neuromuscular control of the TrA and MF has been shown to reduce pain and improve function in chronic LBP patients, particularly in young athletes [3, 7, 28].

Contribution of exercise to changes in activity of other muscles, such as ES, has only partial effect on its activation. Active therapy contributes to an increase in ES activation, but has no effect on the loss of the ES's incomplete relaxation phenomenon which has been consistently observed in individuals with LBP [8, 9]. The sustained activity of ES at the end of the range of trunk flexion has been shown to limit the intervertebral motion. In addition, a high of impairment could be associated with an increase in hip flexion when moving to and from the full flexion, which could be associated with decreased alterations of movement patterns. Since the discussed changes were observed in the third quartile, the hip contribution to flexion might be a strategy put forth by the patients to limit both the motion and loading of the painful lumbar segments.

Conclusions

The results of a recent research on muscle activation in LBP do not reveal a consistent support for either the pain-spasm or the pain-adaptation model. Neither of the two models adequately predicts the effects of pain on trunk muscle activation, nor can fully explain the causality of LBP. Nevertheless, based on the presented research results, some recommendations for active therapy could be made. Specifically, the therapy based on the improvement of proprioception and motor re-education should be focused not only on pain reduction, but also on the long-term changes in trunk muscles' function.

The prescription of exercise as a conservative treatment for lumbar pain is a frequent approach that seems effective for the chronic cases of the nonspecific low back pain. However, there is no evidence for favoring one type of exercise over another. In general, TrA specific exercies should be included, as well as the gait training, to improve intersegmental and muscle coordination, stability and mobility.

Some important recommendations should be considered when designing an exercise program. First, a program should be systematic, progressive, and functional. More importantly, the program should also be individually designed. Second, since the musle response to perturbations is altered, the exercises should be not only proprioceptively rich, but also safe, challenging, and aimed to involve movements in multiple movement planes. Of importance is also incorporation of a multisensory environment and activities that are specificly aimed to improve the dynamic postural control. Therefore, achievement of appropriate muscular balance and joint arthrokinematics in the lumbo-pelvic-hip complex associated with the increase in neuromuscular efficiency throughout the entire body should lead to long term pain reduction and smaller incidence of pain recurence.

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