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The Study of the Variability of Anticipatory Postural Adjustments in Recurrent Non-specific LBP Patients

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***Abstract*—**The study of the variability of the postural strategies in low back pain patients, as a criterion in evaluation of the adaptability of this system to the environmental demands is the purpose of this study. A cross-sectional case-control study was performed on 21 recurrent non-specific low back pain patients and 21 healthy volunteers. The electromyography activity of Deltoid, External Oblique (EO), Transverse Abdominis/Internal Oblique (TrA/IO) and Erector Spine (ES) muscles of each person was recorded in 75 rapid arm flexion with maximum acceleration. Standard deviation of trunk muscles onset relative to deltoid muscle onset were statistically analyzed by MANOVA . The results show that chronic low back pain patients exhibit less variability in their anticipatory postural adjustments (APAs) in comparison with the control group. There is a decrease in variability of postural control system of recurrent non-specific low back pain patients that can result in the persistence of pain and chronicity by decreasing the adaptability to environmental demands.

***Keywords*—**EMG Onset Latency, Variability, Posture, Non - specific Low Back Pain

I. Introduction

THE altered trunk postural muscles activity during functional tasks in the group with LBP has been first reported by Arendt-Nielsen and Hodges and Richardson in 1996 respectively. These changes are in the form of delay in the deep trunk muscles activity and increase in the activity of some superficial trunk muscles [1]. Despite the fact that the altered feedforward responses of trunk muscles in people with LBP have been the focuses of diverse studies [1],[2],it seems to have remained unknown in some aspects and the current treatments have failed to improve these strategies. One of the aspects of postural control system evaluation is the study of the variability of postural responses. Nowadays, the motor control researchers consider it as a key element in the organization of central control system that can reveal a lot of information about movement control systems [3]. The study of the variability for the purpose of diagnosis and of the basic of pathological motor behavior has attracted the attention of a lot of researchers [4],[5]. Decrease in variability is one of the characteristics of the rigid and unchangeable biological systems and its excessive increase leads the system to randomness and instability. These two criteria decrease the adaptability to perturbation. The study of the variability of anticipatory postural adjustments (APA) was first performed by Moseley and Hodges in 2006 .They studied the variability of feed-forward responses of external oblique (EO) muscle in 16 healthy subjects, they showed that only the subjects whose variability postural strategies decrease as a result of inducing pain did not return to normal strategies even when pain stopped. They concluded that reduced variability in postural responses caused less adaptability to environmental demands. Recently, Jacobs and et al studied the variability of APA onset latencies in LBP Patients in 2009. They concluded that the reduced variability in the timing of APA is also evident in people with chronic low back pain. The authors examined the standard deviations of electromyography onset latencies from the bilateral internal oblique (IO) and erector spinae(ES) muscles in 10 people with LBP [6]. In spite of these studies having been conducted, some questions have remained. Firstly, considering the different functional role of trunk muscles (local and global muscles), are different patterns of variability of the timing of APA to be expected? Secondly, considering the fact that postural activity of the abdominal muscles varies body positions [7] are the changes observed in the timing of APA of trunk muscles in the sitting position (as Moseley and Jacob's study) different in other body postures such as the standing position. Answers to these questions about the postural strategies of low back pain patients can reveal more details about the postural system behavior toward perturbation.

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II. METHODS

Twenty one (15 males and 6 females) recurrent non-specific low back pain patients who were pain free during the test or whose maximum pain was two or less according to visual analogue scale participated in a case-control cross sectional study. Twenty one healthy subjects who did not report a history of pain at least during the previous year or a back pain lasting for over three months were matched according to sex, age and body mass index(BMI) with the LBP patients. The subjects gave written informed consent to the test procedures that were approved by the institutional medical research ethics committee of Tarbiat Modares University. Surface electrodes were placed over the bulk of anterior deltoid muscle of the dominant hands and EO and TrA/IO and ES muscles of the opposing side to minimize the artifact caused by the arm movement [8],[9]then, an accelerometer was put on the dominant wrist. At this stage, each participant stood in the specified place and raised the dominant arm from the neutral position (arm beside the body) as fast as possible to about 90 degrees of arm flexion in response to auditory stimulus "go" and remained still responding to "stop". The order and the time of auditory stimulus were random to minimize the probability of anticipating which auditory stimulus was heard. The electromyography activity of the mentioned muscles was recorded during the arm flexion. The rapid arm raise was restricted to 90 degrees since in arm raises over 90 degrees the postural responses complexity is increased because of the changes in the reaction torques and consequently the variability between trials might increase. Since the timing of the muscle responses can be influenced by the background EMG activity, the background activity was controlled at the beginning of each trial and if necessary, the participants were given the essential feedback to relax their muscles. The rapid arm raise and EMG activity of the muscles were performed 75 times for each person. In each trial, the recording would be eliminated if the onset of deltoid muscle was less than 100 ms or over 200 ms after hearing the auditory stimulus. The onset time of the trunk muscles was accepted if it happened between 100 ms before the onset of deltoid muscle and 200 ms after it since it is unlikely that trunk muscle activity out of this time span is generated by internal perturbation. In order to stabilize the amount of internal perturbation in all trials, the mean and the standard deviation of maximum acceleration of arm flexion were measured during 20 rapid arm raises for each person. In each trial if the acceleration of arm raise was beyond the mean ± 2SD range that trial would be eliminated. In order to prevent fatigue, each subject would rest every 20 trials and if needed, they would be given more time to rest. The EMG activity of muscles was recorded by an electromyography eight-channel device made in the Biometrics Company. Data were sampled at 1000 Hz and band pass filtered at 20-450 Hz. Signals from the accelerometer were recorded at 200 Hz. The EMG data were analyzed in MATLAB software program. The timing of muscle response was measured as the latency between the onsets of EMG of trunk muscles relative to the latency of deltoid muscle. The onset latency of each muscle was determined by software devised by "Staude" [10]. The standard deviation of the timing of trunk muscles relative to deltoid onset time that was regarded as the variability of the timing of APA was compared in 75 trials of rapid arm raises between the two groups by MANOVA. Differences in average peak arm raise accelerations between participants with and without LBP were determined by two-tailed t tests.

III- RESULTS

The result indicated that the patients with chronic low back pain showed less variability in the timing of the APA of TrA/IO muscle compared to the control group (Wilk's lambda F=4.21, P=.047). Whereas the reduction of the variability in EO (Wilk's lambda F=.59, P=.45) and ES muscle (Wilk's lambda F=.27, P=.6) was not observed in the patients (figure1).

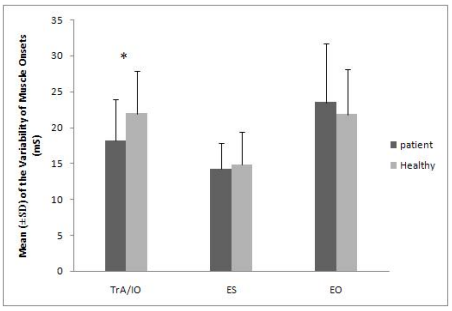

Fig. 1 The results of the comparison between the variability of APA in patients and healthy subjects.

III. DISCUSSION

The study demonstrates that the variability of the timing of APA in TrA/IO is reduced on Chronic LBP patients. The findings are in line with Moseley and Jacobs study. The result of this research exhibits less variability of the APA onset latencies in some muscles of chronic LBP patients. Some of the factors affecting the APA constitute direction, velocity and acceleration of movement and fatigue [11]. As the subjects' direction of movement, their position and internal perturbation intensity were similar in all trials and enough resting time had been considered to avoid fatigue, the observed changes cannot be attributed to mechanical factors. The patients did not feel any pain during the trials. Therefore, these changes cannot be relevant to pain. As the method of evaluation was a choice reaction time task, a positive feedback was given to the participants so as to prevent the result of the performance being affected by the concern over the quality of the task. So, these changes are expected to have occurred by the influence of the other factors rather than pain, physical activity or the stress about the quality of the task. Motor control deficiency in LBP patients is reported ranging from increase in reaction time of fingers to delay in recruitment of muscle trunk accompanied by predictable and unpredictable perturbations [2]. Rapid arm raise threatens the whole body equilibrium and

segmental stability causing center of mass displacement and buckling of different segments of the body. This threat must be defined by appropriate and timely functions of stability and postural control system [12]. In modeling and in vivo studies, it is observed that in such a condition LBP patients take advantage of co-contraction of their trunk muscles to increase stability [13]. This co-contraction and limiting excursion of the spine can be the result of control nervous system compensation for the deficiency of bony-ligamentus system or fear of pain or re-injury [14], whereas the APA production is more than a simple co-contraction and it must be in a way so as to cause movements that as far as direction and intensity are concerned, are compatible with the torques generated by voluntary movements. It is feasible that the patients with LBP reduce the variability of their APA onset latencies in order to minimize their spinal mobility and improve stability. These changes in postural control that are indicative of postural disturbance can stem from a new strategy [15]. Minimizing the movements caused by APA can be the result of fear of pain and injury [5]. Fear of pain and re-injury are enough to change the pattern of muscle activity in rapid arm raising [16]. The fear of pain can reduce the performance of the central nervous system because of the high priority of pain in this system. Al-obaidi reported that anticipation of pain, fear of pain and disability belief is a factor effective in decreasing walking velocity in chronic LBP patients [17].

These researches attributed these changes in the patients' walking patterns to their prior pain experience, memory of pain and their avoidance belief and mentioned that these effects are unlikely to be caused by a pain sensation. Lamoth et al. (2004) noted that the reduction of electromyography activity of ES in LBP patients during walking is relevant to the induced fear of pain [18]. Descarreaux et al. (2007), likewise, suggested the increase in the time to get to the maximum isometric force in trunk muscles stem from neurologic and cognitive adaptations to chronic pain modulated by fear of pain, movement and re-injury [19]. Fear of pain, movement and re-injury, like the other kinds of pain and anxiety, can interfere with the participants' cognitive function. These patients often attend to threaten signals and consequently the process of other cognitive activities is disrupted [20]. Therefore, the fear of pain in the participants in this study can be a factor effective in the reduction of variability of their postural responses. Variability is one of the aspects of behavioral complexity and its reduction can improve information processing capacity by restricting some degree of freedom of postural control system. The loss of variability in LBP patients can disturb the dynamic stability and cause spinal disability and disturbance of the whole body equilibrium in addition to imposing microtraumma and the increase of pain intensity following repetitive loading[4]. Mok et al suggested that the reduction in spinal motion induced by APA in LBP patients can finally increase the spinal motions [13].A reduction of the variability of APA onset latencies can cause the persistence of LBP and its chronicity by reducing the adaptability of patients to new demands. To preserve the dynamic equilibrium, optimal amount of variability is essential in neuromuscular strategies according to contextual constraints [4].

Another finding of this study is that there was no significant change in the variability of APA onset latencies of EO and ES muscles between the participants with and without LBP. This result can be attributed to different role of global muscles that produce torque, control orientation and prevent the buckling of the spinal vertebrae and local muscles that do fine tuning of the inter-segmental motions . Hodges and Richardson (1999) noted that the coordination between limbs movements and the associated APA is not restricted to a single strategy and can vary according to biomechanical role of that muscle in spinal stability [21]. These researchers reported that changing the reaction time of upper-limbs movement in a choice reaction time task does not affect the APA timing of TrA muscle whereas the activation time of other abdominal muscles is proportional the limb movement. They attributed two different patterns of abdominal muscle response to perturbation, to different models of coordination between the APA of postural muscles and limb movements.

This research implies that the direction-specific activation of more superficial abdominal muscles can follow the hierarchical model in coordinating the preparatory responses of trunk muscles and limb prime mover whereas postural activity of TrA muscle which is independent of the direction of movement is controlled in a parallel manner by central nervous system [14].

This emphasizes the importance of the coordination of the timing of trunk global muscles relative to limb prime movers.

APA in the muscles that cause movements such as EO and ES muscles must be in a way to produce some torque proportional to the direction and the amount of torque produced by perturbation so as to preserve the ideal posture. Since torque production by these muscles can cause movement, its amounts must be adjusted accurately according to the perturbing torque created by the limb movement. Otherwise, it can cause perturbation while TrA/IO has greater role in controlling trunk stiffness than torque production. Therefore, lack of coordination of the timing of these muscles with limb movement is less of a risk to instability. So, the central nervous system seems to prefer adjusting the APA timing of the muscles generate movements more accurately according to the limbs movements.

IV. CONCLUSION

There is a reduction of the variability in postural control system of recurrent, non-specific LBP patients that causes the persistence and chronicity of LBP by reducing the adaptability to environmental demands.

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