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HOW DOES BACK PAIN AFFECT STABILITY AND MUSCLE ACTIVITIES OF THE TRUNK?

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SUMMARY

The purpose of this study was to examine the muscular activity and kinematics of the trunk during unstable sitting and to determine differences between healthy and low back pain (LBP) participants. The participant sat on a custom-made chair that was allowed to swing freely in the sagittal plane. They were asked to regain balance after the chair was tilted forward 20° and released. The movements of the lumbar spine and pelvis as well as the chair motion were recorded using Fastrak Sensors. Electromyographic activities of the trunk muscles were recorded using EMG sensors. Trunk stability was evaluated using balancing error and the time required to regain balance. Data were compared between the two groups of participants in order to detect possible differences due to LBP.

INTRODUCTION

Instability of the spine may be a cause of back pain. The main factors that contribute to this are believed to be the intrinsic stiffness of the structures that form the spine, the active contraction of the muscles that control the movements of the trunk, and the modulation of the neural system [1].

Panjabi [1] proposed that an alteration of the passive structures can be related to a decrease in the intrinsic stiffness. This can lead to an increase of the muscular activity as a compensatory response in order to sustain the stability of the trunk. Co-contraction of the trunk muscle has been found necessary to stabilise the spine for both healthy and LBP subjects [2]. Differences in muscular activities between LBP and healthy subjects have been already evaluated. Previous studies revealed evidence of sustained and increased activity of the trunk muscles and hip extensors due to LBP [3,4]. Researchers found differences in kinematics strategy adopted by LBP subjects while performing different tasks, and their mobility was found to decrease when compared to healthy subjects [5]. However, the relationship between kinematic and electromyographic activities and how these affect trunk stability are still unclear.

The purpose of this study was to investigate kinematics along with muscular activity of the trunk while participants were trying to balance on an unstable chair and to evaluate possible differences between healthy and LBP subjects.

METHODS

A total of 54 participants were recruited and divided in two groups: 24 with chronic no-specific LBP and 30 healthy. No significant differences were found between the two groups regarding age, weight, height and BMI. Exclusion criteria for all participants included presence of ankylosing spondylitis, fractures/dislocation of the spine or hips, history

of spinal or hip surgery, pregnancy, neurological disorders, cancer and osteoporosis. Participants were asked to sit on a custom-made seat that was free to swing in the sagittal plane (20° backward and 20° forward). Belt and straps were used for lower limb immobilization. Participants were asked to relax and then the chair was tilted and then released without warning the participants and they were asked to achieve a steady balanced position. During the experiment the participant was asked to maintain their arms folded across the chest and to look forward. Each participant had three attempts to reach the balanced position. Balance was considered successfully achieved again when the participant was able to maintain the steady position for 1 second without touching the mechanical stops and with a maximal oscillation of less than $\pm 1^\circ$. Trunk motion was measured using a three-dimensional motion track system (3SPACE FASTRAK®, Polhemus Inc.). One sensor was placed on the sacrum and one on the first lumbar vertebral. One further sensor was placed on the chair to track its movement. These data were used to derive the angle of the chair, the lumbar spine angle (angle between the first lumbar vertebrae and sacrum) and the hip angle (angle between sacrum and the thigh).

Motion data were filtered at 2 Hz. Electromyographic electrodes (Biometrics Ltd, type Nos. SX230) were used to record the electrical activities (EMG) of the paraspinal muscles. Electrodes were placed on the erector spinae, rectus abdominus, external oblique and internal oblique. The placements of the sensors were based on the recommendations of previous studies [6]. EMG data were rectified and the linear envelop obtained by applying a low pass filter. Contraction of each muscle and co-contraction time was measured for each trial. Balancing error, calculated as the difference between the ideal value of the chair angle for the steady balanced position (0°) and the steady balanced position achieved by participants, and the time required to regain balance were evaluated.

RESULTS AND DISCUSSION

The results of the experiment are summarised in table 1. Figure 1 shows the percentage of number of LBP and control subjects showing muscle co-contraction throughout the whole trial. Hip and lumbar spine angular displacements were found to be significantly different in LBP subjects: in particular hip motion was increased while lumbar spine motion decreased (table 1). The magnitude of the electromyographic signals were increased and latency of contraction was found to be shorter in LBP for all the muscles tested. However, there were no differences in balancing error and the time to regain balance between the two groups (table 1). We hypothesised that the increased

muscular activity may be related to a compensatory and protective strategy from LBP participants. The simultaneous co-activation of flexor and extensor trunk muscles was observed much more frequently in LBP subjects. It appears that the increase in spine stiffness associated with LBP is due to this co-contraction. This may be a compensatory strategy as a result of the decrease in the intrinsic stiffness due to the LBP [1], a protective response in order to limit spine motion and reduce the risks of further damage to the spine tissue. The increased muscular activity or co-contraction may be a sign of muscles spasms that was generally believed to be present in LBP subjects. The finding of decrease in contraction latency does not agree with that of previous research [7], which reported the contrary. A possible explanation could be that the LBP subjects may contract their muscles more quickly to protect the spine. More than 50% of the LBP participants in our study have some of the muscles already activated before the starting of the trial. This suggests that the participants were psychologically and physiologically prepared for the swing, and they are more ready to activate the muscles earlier, compared to subjects in other studies. Correlation of the electromyographic activities of the left and right sides of the trunk (table 1) showed that there were asymmetrical patterns of muscle contraction [8]. Although there were major kinematic and electromyographic changes, there were no differences in seat angle, balance error and the time to regain balance between LBP and healthy subjects (table 1). This indicates the effectiveness of the compensatory strategies adopted by LBP subjects. This was in accordance with previous study which found no differences in the postural control in unstable sitting between LBP and healthy participants [9]. This study showed that LBP subjects had

altered motor control mechanisms but such postural control was effective in maintaining trunk stability.

CONCLUSIONS

The present study shows the kinematic response to perturbation and the changes in muscular activity of the trunk in subjects with LBP. There were altered muscle contraction patterns and significant increases in muscle co-contraction. These active mechanisms appear to be effective in maintaining the stability of the spine, but trunk balance was not compromised in this group of people. It is concluded that LBP is a complex process involving compensatory strategies in both motion and muscular activities. Future research should look at whether the altered muscular mechanism is a cause or consequence of LBP. Treatment of LBP should be focused on muscle functions and pain relief rather than the stability of the trunk.

ACKNOWLEDGEMENTS

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Table 1: Mean and SD of the hip and spine angles, balancing error, time to regain balance, co-contraction latency and activity and correlation of the erector spinae muscle for both the groups performing the 20° initial tilt angle trial.

	Hip angle (°)	Spine angle (°)	Balancing error (°)	Balance time (s)	Co-contraction activity (%)	Co-contraction latency (s)	Correlation Erector Spinae
Healthy	25.3 ± 9.1	13.3 ± 7.6	2.0 ± 2.0	4.6 ± 2.0	12.6 ± 8.3	0.91 ± 0.86	0.67 ± 0.18
LBP	31.8 ± 8.6*	9.2 ± 5.3*	2.3 ± 1.7	4.3 ± 1.6	35.2 ± 11.2*	0.34 ± 0.16*	0.56 ± 0.18*

* Indicates significant difference (p -value < 0.05) between healthy and LBP participant's groups.

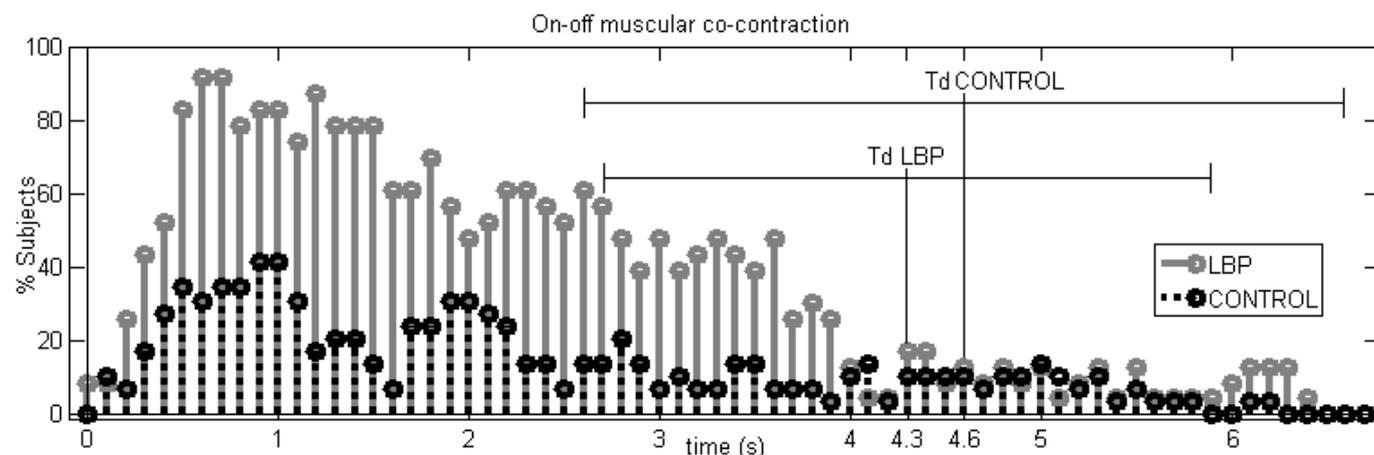


Figure 1: On-off muscular co-contraction as percentage of number of LBP subjects during the trial compared with control subjects. Trial duration (Td) mean and SD for LBP and control participants are shown.