

EXPERIMENTALLY INDUCED LOW BACK PAIN CAUSES CHANGES IN MOTOR CONTROL OF THE TRUNK MUSCLES

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SUMMARY

Electromyographic (EMG) activity of the trunk muscles associated with arm movement was recorded during low back pain induced by injection of hypertonic saline into the longissimus muscle. While changes were observed in the timing and amplitude of muscle activity in all the trunk muscles, changes in transversus abdominis (TrA) were most consistent and were similar to those changes observed in clinical low back pain populations. Our data provide the first evidence that low back pain causes motor control changes of the trunk muscles, in particular TrA.

BACKGROUND AND PURPOSE

Biomechanical and in-vitro data suggest that abnormal motor control of the trunk may result in microtrauma of spinal tissues and cause pain. Corresponding data has demonstrated deficits in motor control of the trunk muscles in clinical low back pain (LBP) populations. On the basis of these findings, attention has been placed on motor control training in the rehabilitation of LBP patients, with the aim to restore normal control of spinal motion. Despite the reported success of this clinical approach, it is not known whether motor control changes precede or follow the onset of LBP. One method to investigate this fundamental issue, is by experimentally inducing low back pain in asymptomatic subjects. The technique of choice is intra-muscular injection of hypertonic saline, which results in a short duration pain that closely simulates clinical pain. The purpose of this study was to determine the effect of experimentally induced LBP, on motor control of trunk muscles during upper limb movements.

MATERIALS AND METHODS

In 7 subjects with no history of LBP, fine wire EMG electrodes were inserted into TrA, obliques internus (OI) and externus (OE), and deep (DM) and superficial multifidus (SM). Surface electrodes were placed over erector spinae (ES), and posterior and anterior deltoid. Standing subjects performed rapid arm flexion in response to a visual cue, and rapid repetitive arm movements in the sagittal plane about shoulder neutral. Data were

collected prior to and after the blinded injection of isotonic and hypertonic saline (NRS ³ 5/10), and at follow up 20 minutes after injection (NRS < 1/10).

RESULTS

During single movements, 5/6 subjects in which TrA activity was recorded, demonstrated a consistent pattern of delay in the onset of TrA EMG after the injection of hypertonic saline ($P < 0.01$). Other trunk muscles demonstrated delay or augmentation, variable between subjects. During repetitive arm movements, there was a decrease in mean amplitude, peak amplitude and minimum amplitude of TrA EMG ($P < 0.002$). There was no change in the activity of the all other trunk muscles except ES ($P < 0.001$)

CONCLUSION

Experimental LBP induced by intramuscular injection of hypertonic saline, caused widespread changes in the timing and amplitude of EMG activity associated with arm movements. Changes in motor control of TrA associated with pain were consistent across the group and were similar to those changes reported in clinical LBP populations. Changes in motor control of the other trunk muscles were more variable, but were also consistent with findings from clinical populations. This data provides new evidence that LBP causes motor control deficits of the trunk muscles, in particular TrA, and supports motor control training in LBP rehabilitation.