DO SHOULDER MOTOR CONTROL IMPAIRMENTS REMAIN IN THE YEAR FOLLOWING THE LAST PAIN EPISODE?

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Introduction

The high dependency of the shoulder complex on muscle control for mid-range stability supports the evidence demonstrating that most abnormal biomechanics and overuse injuries that occur in this region are related to alterations in the function of the scapular-stabilizing muscles (Ciubotariu et al., 2004; Kuhn et al., 1995).

Changes in motor control have been claimed to be at the origin, but also in the perpetuation, of the injury/pain if the altered motor strategy or movement leads to excessive loading of tissues. On the other hand, according to the pain adaptation theory, the pain experience is associated with an adaptation in motor behaviour that involves redistribution of activity within and between muscles at multiple levels of the motor system with potential long-term consequences (Hodges et al., 2011). There is evidence that pain results in gradual decrease in the motor unit discharge rate (Ervilha et al., 2005) via central mechanisms' adaptation (Thunberg et al., 2002), both in related muscles (Graven-Nielsen et al., 1997) as well as in synergistic and antagonist muscles (Ciubotariu et al., 2004). Specifically, studies on shoulder pain demonstrated decreased magnitude and delayed activation of the serratus anterior (SA) (David et al., 2000) and the lower trapezius (LT) muscles (Ludewig et al., 2000) and increased

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upper trapezius' (UT) activity (Ludewig et al., 2000). Also, a bilateral reorganization in the coordination among the divisions of the trapezius muscle was demonstrated in response to induced muscle pain (Falla et al., 2007).

Despite the known relation between pain and motor control, the treatment of pain is unlikely to be sufficient to restore motor control. Studies involving motor control in low back pain found that many aspects of the motor adaptation persist after the resolution of the pain (MacDonald et al., 2009). To the best of our knowledge, no study evaluated shoulder motor control between shoulder pain episodes in tasks involving mainly concentric activity, but also in tasks involving mainly excentric activity. Considering that impairments in trunk motor control between pain episodes (Ferreira et al., 2004; Hodges et al., 1996) can be restored with motor relearning strategies (Tsao et al., 2007) and that these changes persist after cessation of training (Tsao et al., 2008), it is important to identify shoulder motor control impairments between pain episodes to guide effective motor relearning strategies.

The purpose of the present study is to evaluate shoulder motor control expressed to muscle timings during raised and lowered scaption in subjects with shoulder pain episodes in the last year but currently without pain.

Methods

Participants Fifty adult subjects participated in this study and were distributed in two groups. Subjects with history of shoulder pain episodes in the year before the time of evaluation were included in the group of shoulder pain, while the subjects with no pain episodes in the two years before the time of evaluation were included in the control group (Table 1). Subjects were excluded if they presented pain at the time of evaluation, as well if they had history of pain in the cervical or in regions of the upper limb other than the shoulder, history of traumatic injuries, chirurgical and/or conservative intervention in the upper limb and/or neurological impairments.

Table 1

Mean and standard deviation (SD) values of age, height and weight of the control and the shoulder pain groups

Variables	Mean (SD)		
	Control group	Soulder pain group	p-value
Age (years)	21.6 (2.02)	21.8 (1.75)	0.339
Height (m)	1.7 (0.08)	1.7 (0.10)	0.918
Body weight (Kg)	66.3 (14.20)	65.6 (13.07)	0.992
Number of previous pain episodes in the last year	-	1 (n=2); 2 (n=7); 3 (n=2); 4 (n=1); >5 (n=13)	-
Pain intensity	-	Low, n=6 Moderate, n=18 Severe, n=1	-
Mean duration of pain episodes (days)	-	< 3, n=13 4-7, n=8 15-30, n=4	-
	n=25	*n*=25	

The study was approved by the local ethics committee and was implemented according to the Declaration of Helsinki. All individuals gave their written consent to participate in the study.

Instrumentation The activity of UT, LT, SA, infraspinatus (IS) and middle deltoid (MD) muscles was assessed through surface electromyography using a bioPLUX system. The signals were collected at a sampling frequency of 1000 Hz and were preamplified in each electrode and then fed into a differential amplifier with an adjustable gain setting (20-500 Hz; common-mode rejection ratio: 110 dB at 50 Hz, input impedance of 100 MΩ and gain of 1000). Self-adhesive silver chloride electromyographic electrodes were used in a bipolar configuration with a distance of 20 mm between detection surface centres. The skin impedance was measured with an Electrode Impedance Checker. The electromyography signals were analysed with Matlab and **Acqknowledge software** A Qualysis Track Manager system was used to assess shoulder kinematic data during scaption with an acquisition frequency of 100Hz.

Procedures The skin surface was prepared to reduce the electrical resistance to <5000Ω and the electrodes were placed according to anatomic

references. Reflector markers were placed at the acromion, lateral epicondyle and midpoint of iliac crest to assess scaption amplitude. Participants were positioned in a standard sitting position and performed ten cycles of raised (2s) and lowered scaption (2s) interleaved by two isometric periods (2s each). Auditory (metronome) and visual (video) feedback was provided to standardize the task within and between subjects.

The electromyographic signals were filtered using a zero-lag, second-order Butterworth filter with an effective band pass of 20 to 450 Hz, and the root mean square was calculated. The muscle latency was detected in two time windows using a combination of computational algorithms and visual inspection: 1) from –450ms in relation to the beginning of raised scaption to the end of this phase; and 2) from -450ms in relation to the beginning lowered scaption to the end of this phase. The latency for a specific muscle was defined as the instant lasting for at least 50 ms when its EMG amplitude was higher (activation-raised scaption) or lower (inhibition-lowered scaption) than the mean of its baseline value plus/minus 3 standard deviation (SD), measured from –500 to –450 ms in relation to the beginning of raised and lowered scaption, respectively.

Statistical analysis The acquired data were analysed using the Statistic Package Social Science (SPSS) software from IBM Company (USA).Mean and standard deviation were used for descriptive analysis. The Independent Sample T-test was used to compare muscle latencies during raised and lowered scaption between the control and the shoulder pain groups. A 0.05 significance level was used for inferential analysis.

Results

In general, the shoulder pain group presented a tendency to present delayed activation of the LT, SA and IS and earlier activation of the TA during raised scaption when compared to controls (Figure 1). Statistical significant differences between groups were observed for the LT (p=0.004), SA (p=0.024) and IS (p=0.017) muscles. The opposite tendency was observed in the lowered scaption as delayed UT deactivation timing was observed in the shoulder pain group together with earlier deactivation timing of the LT, DA and IS. Statistical significant differences be-

tween groups were observed for the UT (p=0.016), LT (p=0.003) and SA (p=0.019).

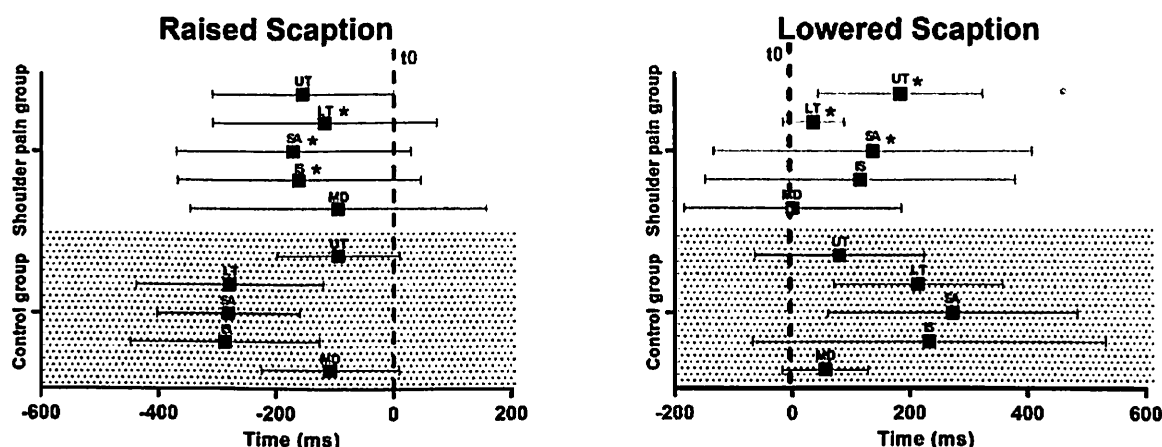

Fig. 1

*Representation of timings of activation during raised scaption and timings of deactivation during lowered scaption in the control and the shoulder pain groups. Significant differences between groups are represented (*p<0.05).*

Discussion

The findings obtained in the present study indicate that motor control impairments in shoulder pain conditions (David et al., 2000; Falla et al., 2007; Ludewig et al., 2000) remain after the resolution of the pain episode. This demonstrates that shoulder motor control adaptation does not require ongoing nociceptor stimulation for maintenance. It should be noted that the majority of the participants included in the shoulder pain group presented recurrent pain episodes in the last year, reinforcing the arguments for considering that the observed changes can perpetuate excessive tissue loading. According to (Falla et al., 2011) the intensity of pain is the parameter that best correlates to the activation timing of the deep cervical flexor muscles in cases of cervical pain. In the present study it was interesting to note that for the majority of the participants the pain episodes were of short duration and of moderate to low intensity.

Participants with previous shoulder pain episodes presented delayed activation timing of the agonist for scapular (LT and SA) and glenohumeral stability (IS) and a tendency to an earlier activation of the TS

muscle as a compensatory strategy during raised scaption that can compromise scapular orientation and stability (Mottram, 1997). This inefficient compensatory strategy was more pronounced during lowered scaption as the earlier deactivation of the LT and SA muscles was compensated by a later deactivation of the TS muscle. These results are in agreement with clinical observations demonstrating that subjects with shoulder pain present scapular kinematic changes and often more pain in shoulder depression than during elevation (Faria et al., 2008). Motor impairments observed in the shoulder pain group, when compared to those in the control group, together with epidemiological data indicating that the recurrence of pain episodes occurs within 1 year following the first episode (Cadogan et al., 2011; Croft et al., 1996) sustain the argument that shoulder pain recurrence results from a deregulation of the shoulder stabilizing muscle synergy. Studies involving motor relearning strategies to restore optimal shoulder muscle motor control are required.

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