

SPINO-PELVIC KINEMATICS AND TRUNK MUSCLE ACTIVATION IN PROLONGED ERGOMETER ROWING: MECHANICAL ETIOLOGY OF NON-SPECIFIC LOW BACK PAIN IN ADOLESCENT ROWERS

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The aim of this study was to determine whether adolescent rowers with and without low back pain (LBP) displayed differences in spino-pelvic kinematics and trunk muscle activation during prolonged ergometer rowing. Ten rowers with LBP and twelve rowers without LBP performed a 20 minute ergometer trial with kinematics, muscle activation and self reported perception of pain data (VAS) collected during the trial. Results of this study show that rowers with LBP postured their lumbar spine in flexion for a greater proportion of the drive phase and nearer to their end range of flexion when compared to those without LBP. This study highlights potential mechanisms for the ramping of back pain in adolescent rowers.

Key Words: adolescent, rowing, low back pain, posture, motor control

INTRODUCTION:

A recent study by Perich et al. (2006) reported that the point prevalence of low back pain (LBP) in a large group of adolescent female rowers was 47.5% when compared to the incidence of LBP in an age matched control group (15.5%). It was also reported in the study of Perich and associates that mechanical factors appeared to be dominant in the development of LBP. These mechanical factors that may contribute to increasing the sensitisation of spinal structures include; reduced lower limb and back muscle endurance which may in turn result in increased forces being transferred to the passive spinal structures (Perich et al., 2006).

A classification method of chronic LBP has been proposed, whereby patients' pain is associated with deficits in segmental spinal control resulting in peripheral generation of back pain (O'Sullivan, 2000). Five sub-groups of non-specific chronic LBP patients have been reliably identified by musculoskeletal physiotherapists (Dankaerts et al., 2006). Of these groups, it is the 'flexion' pattern disorder that is most common in adolescent female rowers (Perich, Unpublished data). This pattern is defined as flexion pain provocation associated with a loss of control of the lumbar spine into flexion placing flexion-related strain on spinal structures.

Although it is clear that rowing is commonly associated with LBP, there has been little examination of the LBP ramping mechanisms in rowers. Therefore, the aim of the study was to determine whether differences in spino-pelvic kinematics data and surface electromyography (EMG) exist in rowers with LBP (with flexion pattern classification) and those without LBP during a prolonged rowing trial.

METHODS:

Data Collection: In this study, 22 rowers (10 males, 12 females) between the ages of 14-17 years, with and without LBP, completed testing (Table 1). Subjects with LBP rated their usual levels of pain and their rowing related pain levels using a visual analog scale (VAS). A battery of clinical tests in conjunction with subjective pain evaluation (O'Sullivan, 2000) was used to positively identify subjects with a 'flexion' pattern classification of LBP. The inclusion criteria for this study were; a typical increase in the level of back pain to above 3/10 within 30 minutes of rowing training, performing training at least 3 times a week and competing in rowing regattas.

Table 1: Characteristics of the no-LBP and LBP groups.

	No-LBP (N = 12–6 Males, 6 Females)	LBP (N = 10-4 Males, 6 Females)
Age (years)	15.8 (0.7)	16.0 (1.0)
Height (m)	1.77 (0.09)	1.73 (0.08)
Mass (kg)	69.5 (11.7)	67.9 (8.8)
Pain - Usual (/10)	0	5.3 (2.3)
Pain – Rowing (/10)	0	4.7 (2.8)

In the period before testing, other forms of exercise were not restricted. Prior to undergoing the experimental protocol subjects were asked to perform a warm-up that included ergometer rowing and stretching. Subjects were then requested to row on a modified rowing ergometer (ferrous supports replaced with wood) for a maximum of 20 minutes at a rate of 22 strokes per minute (spm). Subjects in both groups were asked to row at an exertion of greater than 17 on the Borg scale (range of 6 to 20) and ratings of exertion and VAS scores were collected every minute. Testing ceased if the level of back pain experienced by the subjects exceeded that experienced during normal rowing sessions (as determined by individual VAS scores).

Whilst rowing on the ergometer, synchronised trunk and quadriceps muscle activation and spino-pelvic kinematics were collected for a period of 15 seconds every minute. Spino-pelvic kinematic data were collected using the 3-Space Fastrak™ (Polhemus Navigation Science Division, Kaiser Aerospace, Vermont) which measure angles to 0.2°. During rowing trials, four sensors were affixed to the skin overlying the spinous processes of T6, T12, L3 and S2. Prior to testing, subjects' spinal ranges of motion in sitting were also obtained by subject's slumping their spine to maximum flexion. Subjects were then positioned into a neutral spinal posture by an experienced musculoskeletal physiotherapist. Three trials for flexion range of movement and neutral spine position were captured and a mean value was obtained. The following spino-pelvic angles were defined: *Pelvis* – S2 relative to the magnetic source; *Lower Lumbar* – L3 relative to S2; *Upper Lumbar* – T12 relative to L3; *Lower Thoracic* – T6 relative to T12.

Muscle activation was recorded bilaterally from three muscles at 1000Hz (bandwidth 10-500 Hz and the common mode rejection ratio >115 db at 60 Hz) using an Octopus Cable Telemetric system (Bortec Electronics Inc., Calgary, Canada). Data were recorded from the vastus lateralis (VL), superficial lumbar multifidus (SLM) and the erector spinae at the level of T9 (EST9). Two silver/silver chloride disposable surface electrodes (inter-electrode distance - 20mm) were placed on the skin after the skin was abraded and cleaned with ethanol so that the resistance was less than 5Ω. A ground electrode was placed over the left anterior superior iliac crest. Raw EMG data were demeaned and then amplitude normalised using sub-maximal voluntary isometric contractions (sub-MVIC). To determine the sub-MVIC for SLM and EST9, subjects were asked to lie prone with knees flexed 90 degrees and lift their knees off the plinth for 3 seconds (Dankaerts et al., 2004). For VL, the MVIC values were taken as the maximum value recorded over an average of 100ms in the first minute of the rowing trial.

Data Analysis: EMG and spino-pelvic kinematic data were simultaneously collected and synchronised using the length of chain on the ergometer. Drive and recovery phases were identified using a rotary encoder. On the basis of these data, drive phase duration, stroke rate and stroke length were calculated. Muscle activation data were calculated using Root Mean Square (RMS) with a window length of 50ms. All data were time normalized (0-100%) using an interpolative spline and ensemble averages were obtained from five completed rowing cycles within the 15-second window. All kinematics (spinal-pelvic angles) and EMG muscle activation variables at catch and at the finish position were screened for normality (Shapiro-Wilks test) and data were deemed to be normally distributed. Therefore, a two-way ANOVA with one-between subjects variable (LBP status) and one repeated measures

variable (time) was conducted. All statistical procedures were conducted using SPSS V13.0 and the level of significance was set at $p < 0.05$.

RESULTS AND DISCUSSION:

There was a gradual increase in level of LBP experienced by the LBP group during the 20 minute rowing ergometer trial (Figure 1). Two rowers (1 male and 1 female) ceased testing after 15 minutes of the rowing trial as the level of pain exceeded that of normal training. One subject in the LBP group did not report pain during the rowing trial, but complained of pain the following day. This subject was included in the pain group as this is a common clinical finding in rowers after training.

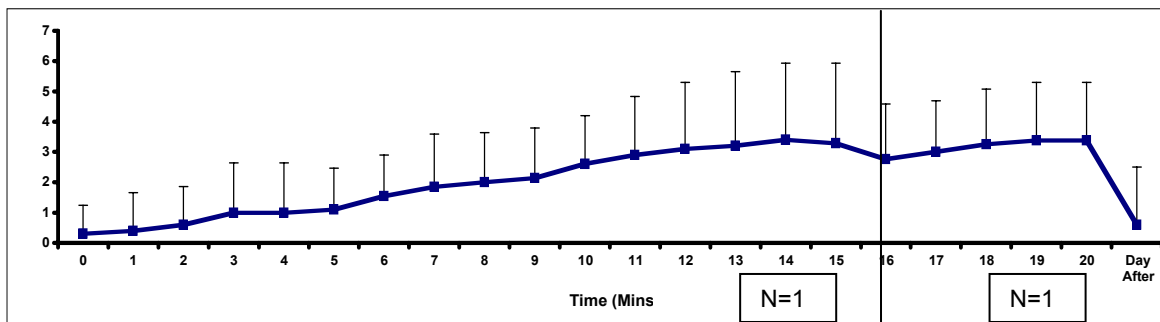


Figure 1: Average of reported levels of LBP (VAS - /10) during the rowing ergometer trial.

In this study, rowers with LBP spent a significantly longer time in flexion as compared to those without LBP during the drive phase ($p=0.025$) although there was no difference within groups across time. Furthermore, rowers with LBP also spent a greater proportion of time during the drive phase near end range of lumbar spine flexion (above 90% of full flexion) ($p=0.026$) (Figure 2). These were consistent across time and there were no interaction between time and group. Similar findings were evident in the recovery phase, but the difference did not reach statistical significance ($p=0.082$ and $p=0.106$ respectively). No other significant differences or trends were noted in other spinal angles.

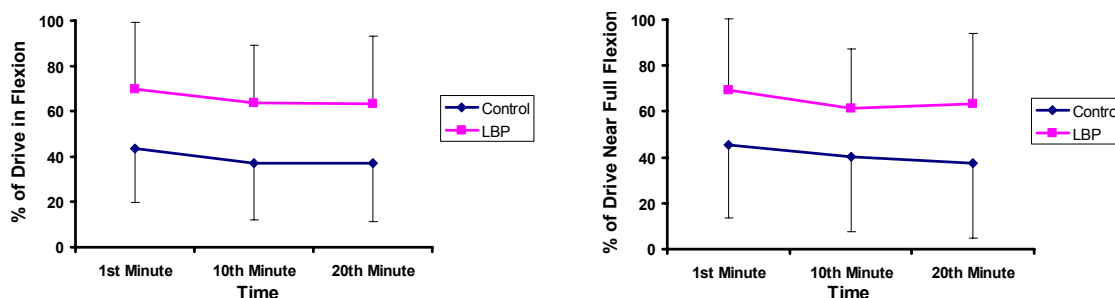


Figure 2: Percentage of drive phase with the lower lumbar spine spent in flexion (left) and spent in greater than 90% of full flexion (right).

With regards to muscle activation data, no significant differences were found for muscle activation between left and right sides. Therefore, these data were averaged to represent muscle activation of one muscle group. No differences were found at the start of the trial, however, although rowers with LBP had a trend towards greater activation in EST9 at the 20th minute when compared to rowers without LBP at catch (Figure 3). No differences or trends between pain and control subjects were found in the SLM (Figure 4) and VL at catch or finish.

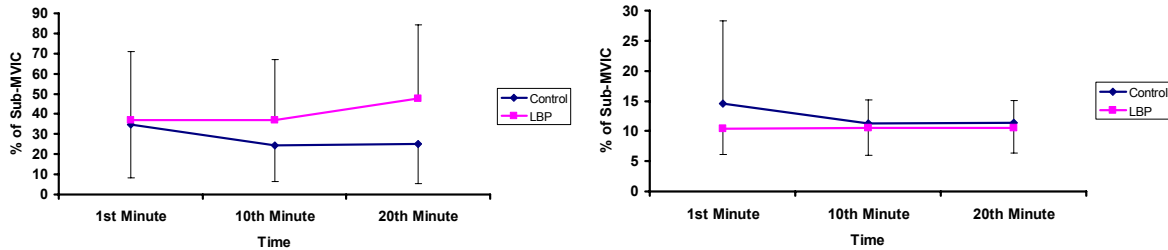


Figure 3. Muscle activation of the EST9 at the catch (left) and finish (right).

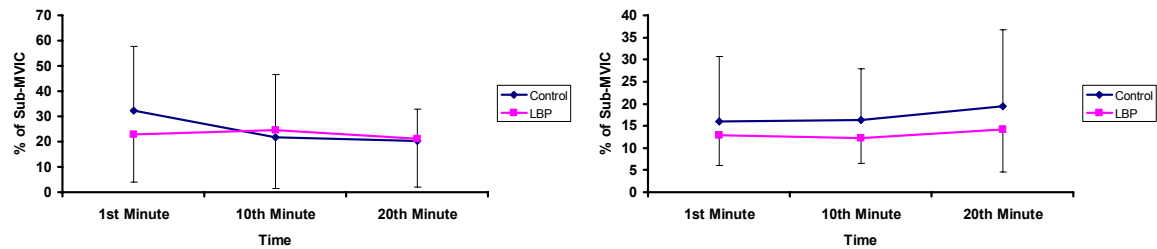


Figure 4. Muscle activation of the SLM at the catch (left) and the finish (right).

CONCLUSIONS:

This study suggests that rowers with LBP spent a greater proportion of their rowing stroke in flexion when compared to rowers without LBP during the drive phase. Furthermore, rowers with LBP spent a greater amount of time near full flexion in the lower lumbar spine when compared to rowers without LBP during a prolonged rowing trial on an ergometer. These findings indicate that rowers with LBP are exposed to greater flexion strain and potential passive structure loading which may represent a mechanism for their disorder.

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