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Original article

Spinal kinematics and trunk muscle activity in cyclists: a comparison between healthy controls and non-specific chronic low back pain subjects—a pilot investigation

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Abstract

The aim of this pilot study was to examine whether differences existed in spinal kinematics and trunk muscle activity in cyclists with and without non-specific chronic low back pain (NSCLBP). Cyclists are known to be vulnerable to low back pain (LBP) however, the aetiology of this problem has not been adequately researched. Causative factors are thought to be prolonged forward flexion, flexion–relaxation or overactivation of the erector spinae, mechanical creep and generation of high mechanical loads while being in a flexed and rotated position. Nine asymptomatic cyclists and nine cyclists with NSCLBP with a flexion pattern disorder primarily related to cycling were tested. Spinal kinematics were measured by an electromagnetic tracking system and EMG was recorded bilaterally from selected trunk muscles. Data were collected every five minutes until back pain occurred or general discomfort prevented further cycling. Cyclists in the pain group showed a trend towards increased lower lumbar flexion and rotation with an associated loss of co-contraction of the lower lumbar multifidus. This muscle is known to be a key stabiliser of the lumbar spine. The findings suggest altered motor control and kinematics of the lower lumbar spine are associated with the development of LBP in cyclists.

1. Introduction

The aim of a competitive cyclist is to produce maximal power at the pedals to propel the bike in the desired direction (Burke, 1996). To maximise bike velocity for a given power output however, the cyclist must reduce their frontal cross-sectional area to reduce aerodynamic drag (Kyle, 1994) and consequently the cyclist must bend forward from the hips in addition to flexing the thoracolumbar spine. The extent to which pelvic and spinal flexion contributes towards the cyclist reaching the handlebars determines whether the cyclists adopts a "round-back" or "flat-back" posture (Burke, 1996; Usabiaga et al., 1997). The fact the cyclist is seated increases the tendency towards a kyphotic lumbar spine posture (Lord et al., 1997; Salai et al., 1999; Bressel and Larson, 2003) unless there is well-developed flexibility of the hamstrings and hips.

Cyclists are known to be vulnerable to low back pain (LBP) (Weiss, 1985; Mellion, 1991; Brier and Nyfield, 1995; Wilber et al., 1995; Callaghan and Jarvis, 1996; Manninen and Kallinen, 1996) however, there is little evidence of radiographic abnormality in the majority of back pain disorders resulting in them being diagnosed with non-specific chronic low back pain (NSCLBP) (Dillingham, 1995). Because of this there is a growing emphasis upon sub-classifying LBP patients on criteria other than radiological abnormality. Patients that present with NSCLBP have been reported to show distinctly different clinical patterns although this notion has not been well scientifically validated (Delitto et al., 1995; O'Sullivan, 2000). A proposed sub-group of

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NSCLBP patients have been classified on the directional basis of back pain provocation and their individual clinical presentation. One of these groups has been classified as a ''flexion pattern'' pain disorder (O'Sullivan, 2000). Flexion strain pain disorders are clinically characterised by LBP, which is reproduced by sustained and repeated flexion of the lumbar spine and is relieved by extension of the lumbar spine. This clinical pattern is reported to be associated with no spinal mobility impairment but a loss of lower lumbar lordosis with associated dysfunction of the lumbar multifidus muscles and a compensatory upper lumbar lordosis and increased tone in the thoracic erector spinae muscles (O'Sullivan, 2000). Flexion pattern pain disorders are thought to result from a loss of neutral zone control of the spinal motion segment with resultant repetitive strain of the spinal segment at the end of range of flexion (O'Sullivan et al., 2003). Cyclists with LBP are thought to commonly represent subjects with a flexion strain pain disorder of the lumbar spine.

As cyclists spend a large amount of time training on their bikes to elicit a physiological training effect this may increase the chance of low back injury via three mechanisms. Firstly, the flexion–relaxation phenomenon, which manifests itself as myoelectrical silence of the erector spinae at the end of range of forward flexion (Floyd and Silver, 1955; McGill and Kippers, 1994; Kaigle et al., 1998; Callaghan and Dunk, 2002) may be problematic as it has been found that when muscle forces are reduced in lifting, passive structures such as the ligaments and intervertebral discs are placed at higher risk (Kong et al., 1996). A study of non-cycling athletes by Juker et al. (1998) suggested that flexion–relaxation may occur in certain cycling postures. Secondly, NSCLBP in cyclists may result from the generation of excessive activation of the spinal extensors resulting in increased tissue strain across the lower lumbar spine. This mechanism has been previously suggested as a basis for NSCLBP (Indahl, 1999). Thirdly, prolonged forward flexion may be an important etiological factor towards LBP as the posterior annulus may develop accumulated micro-damage (Callaghan and McGill, 2001). Loading of the passive structures of the lumbar spine which leads to LBP as discussed above, may be further exacerbated by two factors. Firstly, mechanical creep may increase the stretch on the posterior structures (McGill and Brown, 1992) however, this is questionable as a portion of the cyclist's mass is supported by the handlebars (Bolourchi and Hull, 1985) and therefore, is different to the open-ended system that is typically evident in occupational settings (McGill and Brown, 1992). Secondly, intersegmental joint reaction forces and moments are generated by the lower limbs and must be transferred through the thoracolumbar spine whilst the trunk is in a flexed, and sometimes rotated position.

As there is little data pertaining to the development of LBP in cyclists, the aim of this pilot study was to examine whether differences in trunk muscle activity and spinal kinematics existed in cyclists with and without NSCLBP. It was hypothesised that cyclists with NSCLBP develop a flexion pattern pain disorder due to repeated strain of the lower lumbar spine into end range of flexion/axial rotation and altered motor control of their spinal stabilising muscles.

2. Methods

2.1. Sample

Eighteen (8 male and 10 female) middle-level to high-level cyclists/triathletes, aged between 18 and 57 years were recruited for this study. Cyclists were matched as closely as possible by specific criteria (see below) into a non-pain and a pain group. The non-pain group contained nine cyclists (mean age 37.6 ± 7.9 years, weight 67.2 ± 7.0 kg, height 1.70 ± 0.07 m, body mass index 23.4 ± 2.0) with no history of LBP. The pain group contained nine cyclists (mean age 42.3 ± 9.7 years, weight 67.0 ± 7.0 kg, height 1.70 ± 0.07 m, body mass index 22.9 ± 1.7) that had a history of NSCLBP. The details of this group are outlined in Table 1.

Two experienced manipulative physiotherapists independently assessed the NSCLBP group, and only the cyclists presenting with a flexion pattern pain disorder, that was considered directly attributable to the activity of cycling, were selected (O'Sullivan, 2000). The pain group had a baseline Visual Analog Score (VAS) for the level of pain of 2.3 ± 1.7. This VAS score was determined by the subject's average pain over the week prior to clinical investigation. Cyclists with known structural pathology such as spondylolisthesis of the spine were excluded from the study. Ethical clearance for the study was provided by the Edith Cowan University Human Research Ethics Committee and informed consent was obtained from subjects prior to testing. Subjects were instructed not to partake in any heavy training or physical activity 24 h prior to their clinical assessment or testing day.

2.2. Data collection

Subjects rode their own road bicycles on an indoor wind trainer and were instructed to remain seated as much as possible and to ride on either their tri-bars or drop bars (Fig. 1). Subjects were also instructed to ride at 75% of their age-predicted maximum heart rate and at a cadence between 90 and 100 rpm until the onset of LBP (pain group—total ride time was 38.5 ± 12.7 min) or until the general discomfort was too great (non-pain group—total ride time was 54.5 ± 12.3 min). The cycling

Table 1
Non-specific chronic low back pain (NSCLBP) group subject general details, pain characteristics and level of pain measured by a Visual Analog Scale (VAS)

Subject (pain duration)	Sex	Age (yrs)	Baseline pain (VAS)	Level of pain	Side of pain	Cycling pain (VAS)
JV (3 months)	M	28	5/10	L5/S1	R > L	5/10
JP (14 years)	M	57	3/10	L4/L5	L = R	5/10
NC (2 years)	F	30	2/10	L2/3, L3/4, L5/S1	L = R	4/10
SM (10 years)	F	40	5/10	L4/5, L5/S1	L = R	5/10
DW (7 months)	F	44	2/10	L4/5, L5/S1	L = R	5/10
MM (18 months)	F	49	2/10	L4/5, L5/S1	L = R	6/10
DB (4 months)	M	45	2/10	L4/5, L5/S1	R > L	6/10
DM (3 years)	F	48	0/10*	L4/5, L5/S1	L	6/10
AK (6 years)	M	36	0/10*	L4/5, L5/S1	L = R	7/10

*—Denotes pain was experienced whilst cycling only.

Fig 1. The experimental set up.

positions were utilised to accelerate the onset of LBP in the pain group. Based on the similarity in the mean age of the pain and non-pain groups there would be little difference between the resulting power output between these two groups (Grazzi et al., 1999). The VAS scores at the end of the ride for the pain group were 5.4 ± 0.9. Subjects in the pain group were matched to subjects in the non-pain group by three criteria, they being; total ride time (data were collected every 5 min and the files corresponding to the time where the LBP occurred were eventually analysed in both the pain and non-pain groups), ride position (tri-bars and drop bars) and subject height. Subjects were not matched for physical activity level as training activities outside cycling could not be controlled for however, all subjects were all very physically active. Synchronised trunk muscle electromyography (EMG) and spinal kinematics data were collected at the beginning of each trial and then every 5 min throughout the duration of the ride.

Six pairs of trunk muscles were investigated in this study; three pairs of abdominal muscles and three pairs of back muscles. The muscles that were investigated (with their abbreviations in brackets) and their electrode placements were as follows:

- Left and Right Rectus Abdominis (LRA, RRA)—approximately 3 cm lateral to the midline, half way between the tip of the xyphoid process to the umbilicus (Ng et al., 1998);
- Left and Right External Oblique (LEO, REO)—at the approximate edge of the lateral border of the 8th rib (Ng et al., 1998);
- Left and Right Internal Oblique (LIO, RIO)—approximately 1 cm lateral to the border of the anterior superior iliac crest (Ng et al., 1998);
- Left and Right Lumbar Multifidus (LLM, RLM)—approximately 2–3 cm lateral to the midline of the L4/5 level of the spinous process (De Foa et al., 1989);
- Left and Right Erector Spinae Thoracic 12 (LEST12, REST12)—approximately 5 cm lateral of the midline of the vertebral column at the level of the T12 spinous process (Danneels et al., 2001a);
- Left and Right Erector Spinae Thoracic 9 (LEST9, REST9)—approximately 5 cm lateral of the midline of the vertebral column at the level of the T9 spinous process (Callaghan et al., 1998).

Excess body hair was removed and the area was abraded, then cleaned with an alcohol swab. Ag/AgCl disposable electrodes (30-mm diameter, 20-mm inter-electrode distance), were adhered to the skin along the muscle fibre orientation. An impedance meter was then used to ensure an impedence reading less then 5 KΩ. To ensure that normal cycling movement was not compromised, two portable patient units and two receiving units (Bortec Electronics, Ont., Canada) received the left and right-sided EMG signals.

Prior to data collection on the bicycle, subjects performed a maximum voluntary isometric contraction (MVIC) for all trunk muscles. All MVICs were collected for 5 s and three trials were performed. MVICs for all

back muscles were generated using the one test. From the prone position with hands behind their head subjects then pushed maximally against a maximal manual isometric resistance. To generate MVIC for the abdominal muscles three tests were used (Danneels et al., 2001b; Dankaerts et al., 2004). The subject was positioned supine with the legs straight and strapped with a belt. A resisted curl-up with maximal manual isometric resistance applied in a symmetrical manner through the shoulders of the subject by the investigator (standing at the head end of the couch) was used for left and right rectus abdominis (RA). A resisted crossed curl-up, with the right shoulder moving towards the left and maximal manual isometric resistance applied through the right shoulder by the investigator (standing at the left side) for left internal oblique (LIO) and right external oblique (REO) muscles. For the right internal oblique (RIO) and the left external oblique (LEO) the same procedure was repeated to the right with the investigator standing at the right side applying resistance to the left shoulder. The highest generated contraction from each muscle during the three normalisation trials was deemed to be the MVIC. This calculation of maximum activity was based upon a 25 ms moving window. This approach of normalisation of trunk muscles has previously been shown to exhibit excellent within-day reliability for both healthy controls and patients with NSCLBP (Dankaerts et al., 2004).

Spinal kinematics data were recorded using an electromagnetic tracking device (3-Space Fastrak, Polhemus Navigation Sciences Division, Vermont, USA). The device consists of an electromagnetic source (transmitter), a systems electronic unit and four receivers (each of which have a three-dimensional coordinate system embedded). A validity study of the Fastrak system in the presence of metal (from EMG electrode studs, bike seat posts and bike wheel spokes) was carried out prior to data collection. This preliminary study showed that the accuracy of the Fastrak system was not compromised in the testing environment as the static variation was less than 0.1°. The magnetic source was securely fixed to a wooden frame and the four receivers were placed on the subject's back as follows:

- Sensor 1—spinous process of the 2nd sacral vertebrae (S2);
- Sensor 2—spinous process of the 3rd lumbar vertebrae (L3);
- Sensor 3—spinous process of the 12th thoracic vertebrae (T12);
- Sensor 4—spinous process of the 6th thoracic vertebrae (T6).

To obtain a neutral posture for the spinal kinematics analysis subjects were required to sit upright on their bike seats with their legs hanging unsupported. Three trials of five seconds were recorded. A digital switch (± 10 V) was positioned to synchronise the collection of the EMG and Fastrak signals and to identify top dead centre (TDC). Raw EMG and spinal kinematics data were saved to file for latter analysis.

2.3. Data analysis

The files at the initiation and completion of the ride were analysed. A customised software program written in LabVIEW V6.1 (National Instruments Inc., Texas, USA) was used to process the raw data. EMG data from five continuous crank revolutions (TDC to TDC) were generated during each trial of interest. Each of these sub-samples was full wave rectified and low pass filtered at 4 Hz to generate a linear envelope. Data was then amplitude normalised to the previously recorded MVC values for each muscle. To allow comparison between subjects, data was time normalised to 0–1000 via a cubic spline. To reduce within-subject variability, an ensemble average was then calculated from the five crank revolutions for each muscle. EMG activity was quantified by obtaining the average activation, during this period.

The calculation of the three-dimensional relative rotations of one electromagnetic sensor to another whilst subjects were cycling was based upon the Joint Coordinate System of Grood and Suntay (1983). The output of the Fastrak data was changed from a lateral bending, flexion/extension and axial rotation sequence of rotation to a flexion/extension, lateral bending axial rotation order of rotation as recommended by McGill et al. (1997) then all data were calculated with reference to the neutral position. The matrix algebra procedures for these calculations are outlined by Burnett et al. (1998). Flexion and axial rotation angular displacement values were then defined for the following spinal regions:

- 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.

2.4. Statistical analysis

Independent sample *t*-tests were performed to determine whether differences existed between the non-pain and pain groups at the start and finish of the ride and whether differences existed in EMG activation of the left and right sides of the selected trunk muscles. Prescreening of the data revealed the assumptions of the normality and equality of variance could be made in the vast majority of cases. If the assumption of equality of group variance could not be made, the degrees of

freedom were altered according to the results of a Levene's test. All statistical testing was carried out using the Statistical Package for Social Sciences Version 10.0 (SPSS V10.0) software. Differences were considered statistically significant at $p<0.05$. Due to the small sample size, effect sizes were also calculated and values greater than 0.8 were considered as large (Cohen, 1988).

3. Results

Statistical significance was not reached for a number of the variables measured in this pilot study due to two reasons. Firstly, a limitation in the study was the small sample size due to the requirement of the study to maintain the homogeneity of the NSCLBP group. This affected the ability to make definite conclusions however, there were trends in the data that were worthy of consideration. Secondly, the cyclists in this study adopted two differing postures (riding on their tri-bars and drop bars) as it was considered more clinically relevant that the cyclists adopt the riding posture that provoked their back pain. However, this resulted in large SD values for the spinal kinematics data (see paragraph below), which in turn, decreased the size of the effect between the non-pain and pain groups. However, trends were observed in the data that appear clinically significant which may provide insight into the possible mechanisms contributing to LBP in cyclists. Variables that displayed statistical significance or a large effect size were considered for discussion.

There was minimal change in the pelvic and spinal angles across the duration of the ride (maximal difference for any angle was 1.1°) therefore, an average value was calculated from the start and finish of the ride for each variable (Table 2). Spinal flexion and range of axial rotation data for the regions of the spine (pelvis, lower lumbar, upper lumbar and lower thoracic) are shown in Tables 2 and 3, respectively. There were no statistically significant differences found between the non-pain and pain groups for these variables. However, large effect sizes which suggested a trend towards increased spinal flexion in the lower thoracic region at the start and finish of the ride ($d=0.96$ and 0.80,

Table 2
Spinal kinematics in the sagittal plane at the start and finish of the ride and the resulting differences (°)

	Non-pain ($n=9$)	Pain ($n=9$)	p-value	Effect size
Pelvic flexion—start	23.2 (16.6)	16.1 (15.4)	0.40	0.44
Pelvic flexion—finish	23.4 (17.4)	15.0 (15.4)	0.30	0.51
Pelvic flexion—difference	−0.1 (1.6)	1.1 (3.3)	0.32	0.49
Lower lumbar flexion—start	25.3 (19.3)	38.6 (19.0)	0.16	0.69
Lower lumbar flexion—finish	24.9 (20.2)	38.6 (19.9)	0.17	0.68
Lower lumbar flexion—difference	0.5 (1.8)	0.0 (2.7)	0.71	0.22
Upper lumbar flexion—start	26.8 (13.5)	19.3 (21.6)	0.39	0.43
Upper lumbar flexion—finish	27.2 (13.5)	18.9 (20.9)	0.34	0.48
Upper lumbar flexion—difference	−0.4 (1.9)	0.4 (1.0)	0.29	0.55
Lower thoracic flexion—start	2.7 (5.9)	10.8 (10.9)	0.07	0.96
Lower thoracic flexion—finish	3.8 (5.7)	11.0 (12.2)	0.13	0.80
Lower thoracic flexion—difference	−1.1 (1.8)	−0.2 (2.5)	0.39	0.46

Table 3
Range of axial rotation relative to the magnetic source at the start and finish of the ride and the resulting differences (°)

	Non-pain ($n=9$)	Pain ($n=9$)	p-value	Effect size
Pelvic axial rotation—start	5.6 (1.9)	8.1 (7.0)	0.30	0.56
Pelvic axial rotation—finish	6.4 (4.0)	5.2 (3.2)	0.47	0.33
Pelvic axial rotation—difference	−0.9 (3.5)	2.9 (7.5)	0.19	0.71
Lower lumbar axial rotation—start	2.2 (0.9)	3.4 (1.8)	0.08	0.89
Lower lumbar axial rotation—finish	1.6 (3.0)	3.4 (1.7)	0.15	0.77
Lower lumbar axial rotation—difference	0.6 (3.2)	0.0 (1.5)	0.68	0.21
Upper lumbar axial rotation—start	3.4 (1.2)	5.3 (4.0)	0.19	0.73
Upper lumbar axial rotation—finish	7.8 (7.1)	5.1 (5.9)	0.40	0.41
Upper lumbar axial rotation—difference	−4.4 (7.7)	0.2 (2.7)	0.11	0.89
Lower thoracic axial rotation—start	2.5 (2.1)	5.0 (6.1)	0.25	0.61
Lower thoracic axial rotation—finish	4.2 (4.7)	3.5 (2.4)	0.69	0.20
Lower thoracic axial rotation—difference	−1.7 (4.5)	1.5 (5.7)	0.19	0.65

respectively) and increased range of axial rotation in the lower lumbar spine for the pain group at the start of the ride ($d=0.89$) were evident. Although there was not a large effect size evident for the lower lumbar flexion angle, the mean for the pain ($38.6^\circ \pm 19.9^\circ$) and non-pain ($24.9^\circ \pm 20.2^\circ$) groups suggest clinically significant variation in the thoracolumbar flexion posture between these two groups when the difference in the lower thoracic flexion angle is also considered.

Average muscle activation data are presented for the start and finish of the ride (Tables 4 and 5, respectively). Table 6 presents the differences between the left and right sides of the muscle examined in this study at the start and finish of the ride. These data suggest there were trends evident between the non-pain and pain groups and across the cycling period which may provide evidence of altered motor control of the lumbar spine in the pain group. The pain group exhibited greater levels of activation of the REST9 ($d=0.83$), LLM (1.24), RRA ($d=0.81$) and reduced levels of activation of the LIO ($d=0.81$) at the end of the ride (Table 5). Furthermore, trends for asymmetrical activation of the lower portion of the lumbar multifidus (LM) were observed in the pain group both at the beginning ($d=0.81$) and end ($d=0.99$) of the ride (Table 6).

4. Discussion

The aim of this pilot study was to examine whether differences in spinal kinematics and trunk muscle activity existed in cyclists with and without NSCLBP whilst performing a continuous bike ride. A longitudinal study would have been a preferable design to determine the natural history of LBP in cyclists (specifically development of a flexion pattern disorder) however, in this study, two independent groups were analysed. In selecting the subjects for the two groups in this study, two considerations were important. Firstly, the pain group was homogeneous as possible by selecting NSCLBP subjects with a classification of a flexion pattern pain disorder with the clinically determined symptomatic level being either of the two lower spinal levels. Secondly, subjects were matched between groups

Table 4
Average trunk muscle activation at the start of the ride (%MVC)

	Non-pain ($n=9$)	Pain ($n=9$)	p-value	Effect size
Left multifidus	5.6 (1.8)	9.4 (8.0)	0.19	0.78
Left erector spinae (T12)	6.3 (4.7)	4.6 (2.7)	0.36	0.46
Left erector spinae (T9)	15.6 (7.9)	18.8 (5.7)	0.35	0.47
Left internal oblique	20.6 (16.7)	16.2 (16.1)	0.58	0.27
Left rectus abdominus	10.5 (12.4)	5.7 (5.1)	0.30	0.55
Left external oblique	13.1 (12.3)	11.0 (10.4)	0.69	0.19
Right multifidus	5.1 (2.3)	4.8 (3.3)	0.82	0.11
Right erector spinae (T12)	7.1 (11.7)	4.5 (3.2)	0.53	0.35
Right erector spinae (T9)	12.3 (7.1)	19.6 (16.9)	0.25	0.61
Right internal oblique	16.8 (14.6)	17.7 (16.6)	0.90	0.06
Right rectus abdominus	3.4 (2.2)	8.6 (6.5)	0.04	1.20
Right external oblique	7.5 (7.0)	5.3 (4.0)	0.44	0.40

Table 5
Average trunk muscle activation at the finish of the ride (%MVC)

	Non-pain ($n=9$)	Pain ($n=9$)	p-value	Effect size
Left multifidus	3.9 (1.1)	8.8 (6.8)	0.05	1.24
Left erector spinae (T12)	4.4 (4.1)	4.0 (2.4)	0.80	0.12
Left erector spinae (T9)	11.6 (11.6)	20.1 (13.8)	0.11	0.67
Left internal oblique	30.9 (20.4)	16.2 (15.8)	0.11	0.81
Left rectus abdominus	5.9 (7.8)	5.6 (3.9)	0.92	0.05
Left external oblique	11.7 (8.4)	10.2 (10.4)	0.74	0.16
Right multifidus	4.2 (2.4)	5.2 (2.9)	0.41	0.38
Right erector spinae (T12)	2.6 (2.0)	3.8 (2.1)	0.24	0.58
Right erector spinae (T9)	9.7 (6.7)	15.2 (6.6)	0.10	0.83
Right internal oblique	17.7 (19.0)	16.9 (14.4)	0.92	0.05
Right rectus abdominus	4.0 (2.3)	6.9 (4.9)	0.13	0.81
Right external oblique	8.0 (5.6)	6.3 (4.4)	0.49	0.34

Table 6
Differences in left and right side muscle activation at the start and finish of the ride (%MVC)

	Non-pain ($n=9$)	Pain ($n=9$)	p-value	Effect size
Multifidus—start	0.5 (3.0)	4.6 (7.1)	0.13	0.81
Multifidus—finish	−0.3 (2.2)	3.6 (5.7)	0.08	0.99
Erector spinae (T12)—start	−0.8 (12.3)	0.0 (1.8)	0.84	0.11
Erector spinae (T12)—finish	1.7 (3.2)	0.1 (1.6)	0.20	0.67
Erector spinae (T9)—start	3.4 (12.5)	−0.8 (18.5)	0.58	0.27
Erector spinae (T9)—finish	1.9 (9.8)	4.9 (14.3)	0.61	0.25
Internal oblique—start	3.8 (23.8)	−1.5 (13.5)	0.57	0.28
Internal oblique—finish	22.9 (17.1)	9.9 (13.4)	0.09	0.85
Rectus abdominus—start	7.1 (10.5)	−2.9 (4.3)	0.02	1.35
Rectus abdominus—finish	1.8 (8.1)	−1.4 (5.0)	0.33	0.49
External oblique—start	5.7 (8.5)	5.6 (8.2)	0.99	0.01
External oblique—finish	−0.6 (3.6)	−1.0 (3.0)	0.97	0.01

for cycling position (i.e. drop bars and tri-bars). A discussion of the spinal kinematics, the trunk muscle activation and the clinical implications is outlined below.

4.1. Spinal kinematics

The magnitude of the spinal angles measured in this study were higher than those obtained in a previous study which examined spinal posture in cyclists using X-ray methods (Usabiaga et al., 1997). The reason for this is that the electromagnetic tracking system used in this study was a skin-mounted measuring system therefore, skin distraction over the underlying vertebrae is being measured which consequently overestimates the true thoracolumbar spine motion (Pearcy and Hindle, 1989; Pope et al., 1992; McGill et al., 1997). However, as the same measuring system was used to compare both the non-pain and pain groups this was not considered to be detrimental to the design of the study.

The findings of this pilot study clearly showed that the spinal kinematics for both the non-pain and pain groups were remarkably stable across the cycling period which dismissed the concept that spinal creep occurred as the cyclists developed back pain, nor did the cyclists alter their spinal posture in response to the development of pain during cycling. Usabiaga et al. (1997) stated that hip flexion rather than lumbar spine flexion changed with cycling position or bicycle. This study however, suggests that spinal posture varied between asymptomatic and symptomatic patients. The pain group displayed a trend towards greater lower lumbar spine rotation and flexion when compared to the non-pain group. Conversely, the non-pain group displayed a trend towards greater upper lumbar spine flexion and rotation compared to the pain group. These findings, although clinically significant, should be viewed with caution as they did not reach statistical significance due to the low subject numbers examined in this study. Rotation of the lower lumbar spine in flexed postures has been well documented as a risk factor in the development of injury to the annulus fibrosis (Nachemson, 1999). Furthermore, end of range strain is known to increase the risk of back injury however, it was not known where these cyclists positioned their spines relative to end of range. It is possible that prolonged end of range strain into flexion and rotation was a factor in the pain group (McGill and Cholewicki, 2001).

4.2. EMG activation

Lumbar multifidus is known to be a key stabiliser of the lower lumbar spine controlling both flexion and rotation moments of the spine (Bogduk, 1997). Symmetrical patterns of activation of the LM have been reported in a number of normative EMG studies when investigating the lumbar spine during flexion/rotation tasks, supporting this muscle's stabilising role (Danneels et al., 2001b). Dysfunction of the LM has been documented in the LBP population, with a loss of symmetrical co-contraction being reported (Grabiner et al., 1992). The findings from this study suggest that the pain group presented with greater asymmetry of the superficial LM at both the beginning and end of the ride when compared to the non-pain group. This finding appears to be consistent with the trend towards increased axial rotation observed in the flexed lower lumbar spine typical of the pain group. It is unclear whether the trend towards an increase in LLM and REST (T9) and decrease in RIO at the end of the ride in the pain group represented an attempt to compensate for the flexion and rotation moments across the low back by increasing the extension moment across the spine, or whether this change was a reflex response to pain. Regardless of the mechanism involved, the spinal kinematics remained unaltered across the ride time and the pain reached a point where cycling had to cease. The reason for the trend towards an increase in the

activation of RRA for the pain group was less clear as this muscle is a powerful flexor of the spine. Previous research however, has reported dominant activation of the rectus abdominus to be associated with LBP disorders as a substitution strategy for a deficit in key stabilising muscles such as the transverse fibres of internal oblique (O'Sullivan et al., 1997). Further, this may be linked to the trend displayed for the difference in the lower thoracic spinal angle observed between the two groups. It should be acknowledged that the use of surface EMG prevented the measurement of the function of the deep spinal stabilising muscles.

4.3. Clinical implications

The underlying mechanism for NSCLBP is a source of great debate due to a lack of a patho-anatomical basis for the pain disorder. It has been hypothesised that the classification of NSCLBP subjects into homogenous subgroups may enhance the understanding of the underlying basis of these disorders and enhance treatment efficacy (Rose, 1989; Atlas et al., 1996; Leboeuf-Yde et al., 1997; Fritz and George, 2000). The subject group in this study reported having a NSCLBP disorder where pain was exacerbated by flexion activities and sustained postures of the lumbar spine, especially cycling. Conversely, pain was relieved with extension postures and activities. The majority of the NSCLBP subjects had sought various treatment interventions for their pain disorders however, they were still unable to cycle without the onset of significant pain. A majority of these subjects had to cease high level cycling due to their pain disorders.

The findings of this pilot study suggest that the cyclists with NSCLBP may have an underlying motor control disorder of the lumbo-pelvic region with an associated loss of co-contraction of the lower LM, and a trend towards an increase in flexion/axial rotation movement of the lower lumbar spine. This appears to represent either a maladaptive response or predisposition to a flexion/rotation strain pain disorder, as the movement pattern adopted by these subjects appears to increase the flexion/rotation strain on the lower lumbar spine already sensitised to movement and loading in these directions. Interestingly, this motor pattern preceded the onset of LBP during the cycling task, suggesting that it is an inherent movement fault rather than a reflex response to pain. Furthermore, with the onset of pain (related to flexion/rotation loading of the lower lumbar spine), there was no evidence of an effective adaptive response to pain to reduce the amount of rotation and flexion of the lower lumbar spine.

These findings lend support the clinical classification of flexion related pain disorders proposed by O'Sullivan (2000). These preliminary findings are in contrast to current theories that suggest the mechanism for NSCLBP is linked to a reflex extensor muscle response of the back extensor muscles, resulting in a loss of flexion relaxation of the back muscles and a reduction of spinal flexion resulting in secondary increased tissue strain (Indahl, 1999). In fact, the current study suggests contrary findings, they being; increased rotation movement in flexion postures of the lower lumbar spine and reduced co-contraction of the LM, which may result in increased flexion/axial rotation strain across the low back already pre-sensitised to strain in these movements. In order to test this hypothesis further, a motor learning intervention directed at facilitation of co-contraction of the lower LM to reduce the flexion/axial rotation strain at the low lumbar spine could be trialed, to assess its influence on these pain disorders. Clearly further investigations into similar populations with NSCLBP with a larger sample size are required to confirm or refute these preliminary findings.

5. Conclusions

The findings of this pilot study lend further credibility to the idea that clinical presentation of individuals suffering NSCLBP should be considered. During clinical evaluation, all subjects in this study reported that their LBP was precipitated by flexion related activities, in particular, during cycling. Cyclists in the pain group showed a trend towards increased lower lumbar rotation and flexion with associated loss of co-contraction of the muscles whose primary role is to control these movements (LM). Although these results should be viewed with caution due to the small sample size in this study, they do lend support to the presence of an underlying motor control disorder that predisposes the cyclists to flexion/rotation strain of the low lumbar spine. Further research into this group with a larger sample size is required and rehabilitation strategies to manage LBP in cyclists needs to be formerly assessed.

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