Title: Trunk muscle onset and cessation in golfers with and without low back pain.

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Abstract

The knowledge of the onset and cessation timing of the paraspinal muscles that surround the lumbar spine is an important area of research for the understanding of low back pain. This study examined the timing of the erector spinae and external oblique muscle activity in a group of golfers with and without low back pain. The study compared the results of surface electromyography measurements for two groups of golfers. Twelve male golfers who had reported a mild or greater level of pain in the lower back that was experienced while playing golf were examined. A further fifteen male golfers who had reported no history of lower back pain in the previous 12 months were recruited as controls. The results showed that the low back pain golfers switched-on their erector spinae muscle significantly in advance of the start of the backswing. This finding was not evident in the group who did not have low back pain symptoms. Low back pain golfers therefore, may use the erector spinae muscle as a primary spinal stabiliser instead of the stronger deeper muscles such as transversus abdominis and multifidus. These results may have important implications for conditioning programmes for golfers with low back pain.

1. Introduction

During the golf swing, neuromuscular control of the trunk is important in ensuring the accuracy of the technique and providing sufficient muscle force to stabilise and protect the vertebral column from injury (Cholewicki and VanVliet, 2002; Hodges and Moseley, 2003). Research has demonstrated that in the presence of acute and chronic low back pain (LBP), neuromuscular control of the paraspinal and abdominal muscles is adversely affected, which may compromise the stability of the spine (Hodges and Richardson, 1996; Roy, DeLuca, and Casavant, 1989).

Hodges and Richardson (1996; 1997; 1999) reported that transversus abdominis (TA) activation preceded the movement of the upper and lower limbs in healthy subjects. However, while performing the same repeated upper or lower limb movements, LBP patients demonstrated a significant delay in the activation of this muscle. As the TA does not contribute significantly to trunk motion (Hodges, 1999; McGill, 1996), it was theorised that the early onset of this muscle served to stabilise the lumbar spine and protect it from possible injury (Hodges, 1999). Hides, Richardson and Jull (1996) reported a similar dysfunction for the multifidus muscle in patients with LBP.

Such deficiencies in the lumbar stabilising musculature may be one of the factors that contribute to high recurrence rates of LBP in the years following an initial episode (Hides et al., 1996). This notion is supported by separate research, which suggests that disturbances in the functioning of the musculoskeletal system can result in abnormal motion and excessive spinal loads, which may lead to the development of LBP (e.g. Colloca and Keller, 2004).

In addition to these clinical studies, several investigations have assessed the neuromuscular differences in LBP patients in the golfing population (Cole and Grimshaw, 2008; Evans and Oldreive, 2000; Horton, Lindsay, and Macintosh, 2001). In their investigation, Cole and Grimshaw (2008) reported that low-handicap LBP golfers demonstrated increased external oblique (EO) activity at the top of the backswing and reduced erector spinae (ES) activity at impact, when compared with asymptomatic controls. The authors suggested that whilst the reduced ES activity could lead to smaller compressive loads, it could also reduce spinal stability that could be hazardous when coupled with increased EO activity (and possible torsional forces). Evans and Oldreive (2000) used a static abdominal hollowing technique to assess the endurance of the TA in golfers with and without a history of LBP. Their findings suggested that golfers with a history of LBP demonstrated significantly reduced TA endurance compared with golfers with no history of the disorder. This was indicative of a motor control deficit, which implied that the function of the TA was not spontaneously recovered following an episode of LBP. Using surface electromyography, Horton et al. (2001) assessed the muscle onset patterns of the superficial abdominal muscles in golfers with and without chronic LBP. This research indicated that the lead EO (left in right-handed golfers) was activated significantly later during the backswing in golfers with LBP when compared with asymptomatic controls.

Although this delayed muscle activity may provide evidence of altered neuromuscular control in the injured golfers, it is unlikely to offer insight into the possible cause of their injuries, as previous research has not identified the backswing as a high risk component (Hosea and Gatt, 1996; Sugaya, Tsuchiya, Moriya, Morgan, and Banks, 1999). The study conducted by Horton and colleagues (2001) is the only research to date that quantifies abdominal onset in

LBP golfers, while less is known about the activation patterns of the paraspinal muscles in this population.

The aim of this investigation was to assess the patterns of erector spinae and external oblique muscle activation in golfers suffering with low back pain when compared with those of asymptomatic controls.

2. Methodology

2.1 Subjects

Golfers who previously reported experiencing LBP were asked to complete the Short-Form McGill Pain Questionnaire (SF-MPQ) to establish the severity of their condition (Melzack, 1987). The SF-MPQ was logically created from the Long-Form McGill Pain Questionnaire (LF-MPQ) (Melzack, 1975) and incorporates a visual analogue scale (100 mm line) that patients use to rank the intensity of pain, with '0' representing 'no pain' and '100' denoting 'worst possible pain' (Melzack, 1987). Importantly, the reliability, content validity, construct validity, concurrent criterion validity and predictive validity have been demonstrated for the SF-MPQ and the LF-MPQ (Flaherty, 1996; Grafton, Foster, and Wright, 2005; Melzack, 1987; Pearce and Morley, 1989), supporting their use in clinical research. Golfers who recorded a mild or greater ($\geq$ 20 mm) level of pain associated with their lower back were recruited to form the LBP group (n = 12 right-handed male golfers). In addition, golfers who presented with no history of spinal surgery, fracture or deformity and who had not experienced LBP in the previous 12 months (n = 15 right-handed male golfers) were recruited to comprise the no low back pain group (NLBP). All subjects provided written informed consent and the experimental protocol was approved by the Human Research Ethics Committee at the University.

Insert Table 1 about here.

2.2 Task.

Prior to testing, a two-metre square tee-off area was defined on the grass using custom markers, whilst the golfers performed an appropriate warm-up to familiarise themselves with the surrounding conditions. Following this, the golfers positioned themselves within the tee-

off area, to ensure that their body and club were in this box at all times. Using their own driver, each golfer performed twenty drives towards a flag positioned 320 m from the tee-off box. All data collection took place on a grassed tee-off area at a public driving range, to ensure that the movements closely replicated playing conditions and allowed the golfers to perform the swing in an uninhibited manner.

2.3 Data Collection

During the performance of the drives, myoelectric activity was collected for the ES and the EO using an AMLAB II surface EMG system (AMLAB International, AU). For the purpose of assessing these muscles, pairs of circular pre-gelled silver-silver chloride (Ag-AgCl) surface electrodes (Red Dot 2258-3, 3M, Ontario, CA), were positioned over the muscle of interest at a centre-to-centre distance of 2 cm apart. Each pair of electrodes was attached to a differential amplifier (gain x 1000, input impedance = 500 MΩ, common mode rejection ratio >110 dB, noise = <2 μV) with a bandpass frequency of 15 - 480 Hz. The amplifier was connected to a computer via a 12-bit A/D conversion board and the myoelectric data were collected at a sampling frequency of 1000 Hz using the AMLAB II (Build 19.8) software.

Before the Ag-AgCl electrodes were positioned, the sites were shaved and cleaned with alcohol to reduce the impedance at the interface between the skin and the electrodes. Following the preparation of the skin, the recording electrodes were placed bilaterally at a distance of 3 cm from the spinous processes of the second (L2) and fourth lumbar vertebra (L4) over the lumbar ES muscle. The bilateral activation patterns of the EO muscles were recorded by a pair of electrodes placed 15 cm lateral to the umbilicus at the transverse level (Horton et al., 2001). Reference electrodes were located over the spinous processes of the first to fourth lumbar vertebrae and bilaterally over the tenth rib for the ES and EO muscles,

respectively. Whilst the reference position used for the EO was not ideal (i.e. a moving rib in rotation associated with the golf swing) it was all that could be achieved within the experimental constraints.

To facilitate the identification of swing events, the EMG data was synchronized with a three-dimensional videography system (50 Hz) via an Event and Video Control Unit (E&VCU) (Peak Performance Technologies Inc., USA). The E&VCU and video cameras were linked with the EMG system via a breakout box, which facilitated the use of an external trigger to simultaneously mark the video and EMG data. The events of the golf swing (start of the backswing, top of the backswing and impact) were identified for the video data and then synchronised for the corresponding EMG.

2.4 Data Analysis.

For the purposes of identifying differences in the onset and cessation patterns of the paraspinal and abdominal muscles in the LBP and NLBP golfers, the data from the best three performances were considered. The principal researcher was responsible for the selection of these trials, which was based on the accuracy (i.e. directed toward the target) and flight path (i.e. no slice/hook and limited draw/fade) of the ball following impact. It is important to note that the principal researcher, who was an experienced golfer, was coached by a professional in the identification of characteristics that constitute a 'good' golf drive prior to the commencement of the study. The raw EMG data for the best three trials was full-wave rectified to give the absolute value for all EMGs and the onset and cessation of the ES and EO muscles was computed using the methods described by Hodges and Bui (1996). Onset of muscle activity was deemed to occur once the mean myoelectric activity over a 50 ms window exceeded the average baseline activity by one standard deviation. The timing of this

event was determined by advancing the 50 ms window one sample at a time and once onset had been identified the time of the first sample in that window was recorded. Similarly, cessation of muscle activity was considered to occur when the myoelectric activity fell below the pre-defined threshold using the same process outlined for onset. The onset and cessation times for the best three trials for each golfer were averaged and presented for the relative phases of the swing.

2.5 Statistical Analysis.

A one-way analysis of variance (ANOVA) was conducted using SPSS 12.0 (SPSS Inc., USA) to assess for any statistically significant differences between the LBP and NLBP groups, with respect to EMG onset and cessation. The level of significance was set at $p < 0.05$ and effect sizes were calculated using the Cohen's *d* method (Cohen, 1992) to account for the small sample sizes. Effect sizes measure the degree of association between independent (IV) and dependent variables (DV) and assess what proportion of the total variance in the DVs can be predicted by knowledge of the levels of the IVs (Tabachnick and Fidell, 2007). For this study, effect sizes of less than 0.2 and between 0.2 and 0.5 were considered negligible and small effects, whilst values between 0.5 and 0.8 and greater than 0.8 represented medium and large effects (Cohen, 1992).

3. Results

3.1 Lumbar Erector Spinae

Insert Table 2 about here.

The onset and cessation patterns of the ES (Table 2) demonstrated that, for the LBP golfers only, activation of the ES preceded the initiation of the backswing and this occurred significantly earlier when compared with the asymptomatic golfers. During the backswing, both groups tended to demonstrate a short bilateral cessation in activity before the top of the backswing, although this was not evident in all golfers. This was followed by a secondary onset that was presented relative to the start of the downswing. The timing of this bilateral period of cessation and the secondary onset was similar between the LBP and NLBP golfers, with the statistical analysis failing to show any differences between the groups. Interestingly, the secondary onset of the right-side ES preceded the start of the downswing in both golfer groups, whilst the activation of the left ES did not occur until shortly after the downswing had commenced. The ES remained active throughout the downswing and follow-through for both groups and no significant differences were observed during these phases.

3.2 External Oblique

Insert Table 3 about here.

The data presented in Table 3 depicts the onset and cessation timing of the EO for the LBP and NLBP golfers. Similar to the data presented for the lumbar ES, only the LBP golfers demonstrated left-side EO activation that preceded the initiation of the backswing; however

this difference was not found to be statistically significant. In both the LBP and NLBP golfers, the right-side EO activity preceded the beginning of the downswing by an equal time period (LBP = -638.3 ± 198.2 ms; NLBP = -633.3 ± 305.3 ms), with the statistical analysis showing no significant difference between the groups. Following onset, both the left- and right-side EO remained active throughout the swing in both groups, with activity cessation being recorded late in the follow-through.

4. Discussion

The current investigation reports on the onset and cessation patterns of the lumbar erector spinae and external oblique muscles in golfers with and without low back pain. Whilst it was clear that the pre-emptive onset of ES activity would assist in stabilising the spine during the dynamic golf swing, it was not clear why this muscle was contracted pre-emptively in the LBP group only. When considering the muscular system of the spine, it is important to recognise that the muscles are often divided into two groups, based on their structural properties and orientation (Bergmark, 1989). The global muscles are torque-producing muscles that facilitate movement of the trunk, but do not actually attach to vertebral structures (e.g. rectus abdominis, external obliques). These muscles provide general trunk stability, but due to their disassociation with the vertebral components they are not capable of controlling the segmental motion of the vertebrae (Evans and Oldreive, 2000; O'Sullivan, 2000). Conversely, the local muscle system comprises muscles that directly attach to the vertebral column, which stabilise and control the motion of the spinal segments (Bergmark, 1989; Hodges, 2000; O'Sullivan, 2000). The local muscle system encapsulates the deep spinal and abdominal muscles (e.g. multifidus and transversus abdominis through the lumbodorsal fascia (Bogduk, 2005)), which are suggested to co-contract prior to the performance of a dynamic task to provide the global muscles with a stable platform to perform the movement (Hodges, 1999; Hodges and Richardson, 1996, 1999; Marshall and Murphy, 2003). However, previous research has established that in the presence of acute and chronic LBP, the functioning of the local muscle system is preferentially affected when compared with the global muscles (Hides et al., 1996; Hodges and Richardson, 1996; Roy et al., 1989). Therefore, it could be postulated that the earlier onset of ES activity observed in the LBP golfers was related to this muscle playing a more significant primary role in the protection of the spine. This may be supported by previous research, which reported no

significant reduction in the endurance of the erector spinae musculature in LBP golfers when compared with NLBP golfers (Evans, Refshauge, Adams, and Aliprandi, 2005) and asymptomatic non-golfers (Suter and Lindsay, 2001). Similarly, it would be feasible to suggest that the later onset of ES activity in the NLBP golfers (i.e. after the beginning of the swing) was related to the fact that the unaffected functioning of the deeper local muscles only required the ES to provide a secondary support role in spinal stabilisation. However, Cole and Grimshaw (2008) demonstrated that low-handicap LBP golfers had reduced ES activity at the top of the backswing and at impact when compared with asymptomatic players. Therefore, although the ES may be activated earlier in the LBP group to compensate for the dysfunction of the deeper multifidus, the reduced amplitudes may suggest that this adaptation is ineffective in increasing the stability of the lumbar spine at critical points such as the top of the backswing and at impact.

The findings presented in the current study and previously by Cole and Grimshaw (2008) demonstrate altered recruitment patterns for the ES in LBP golfers, which would be likely to affect the strength and endurance of this muscles in injured players. Weishaupt and co-workers (2002) reported that LBP golfers showed considerable strength deficits in many of the trunk muscles, including the trunk extensors. However, Evans et al. (2005) and Suter and Lindsay (2001) reported no significant reduction in the static endurance of the trunk extensors for LBP golfers when compared with asymptomatic players and healthy non-players, respectively.

Considering the onset and cessation patterns of the right- and left-side EO muscles, the data indicated that the injured and uninjured golfers did not differ significantly. However, the right-side EO, which initiates the downswing, was activated prior to the start of the

downswing in the injured group. Horton and co-workers (2001) reported only one significant difference between a group of chronic LBP golfers and asymptomatic controls, with respect to the onset patterns of the EO, with the injured golfers activating the left EO later in the backswing. Although statistical significance was not reached, the LBP golfers in Horton and co-workers' (2001) study also demonstrated an earlier triggering of the right-side EO on the downswing compared to healthy golfers (LBP = -94.8 ± 61.3 ms; NLBP = -63.2 ± 37.9 ms). The earlier onset times of the EO demonstrated by the current study and Horton et al. (2001) combined with the increased EO activity amongst low handicap players shown by Cole and Grimshaw (2008) would suggest that golfers with LBP have a greater functional requirement for this muscle during the swing. Moseley and Hodges (2005) observed increased EO activity in individuals with experimentally-induced LBP, which they believed evidenced an alternate postural strategy that compensated for the neuromuscular dysfunction of the deeper TA. Interestingly, Lindsay and Horton (2006) found that golfers with LBP had reduced trunk rotation endurance towards the non-dominant side (left rotation in right-handed golfers). This endurance deficit in combination with the increased onset and activity of EO would likely lead to earlier fatigue and an increased injury risk in this population (Lindsay and Horton, 2006).

As with any research, there were a number of limitations that should be acknowledged. These are subject recruitment, EMG cross-talk, inter-subject variability (Abernethy, Neal, Moran, and Parker, 1990) and small statistical sample size, which may have effectively made it difficult to detect real differences between the populations. However, effect sizes were reported for each statistical comparison and it is suggested that these be taken into account when reviewing the findings. Additionally, it is important to acknowledge that whilst the slower sampling frequency of the three-dimensional videography system (50 Hz) would have

been sufficient to identify the initiation and top of the backswing (given their slow nature), the identification of high-frequency components, such as impact may have been subject to error. Finally, the LBP golfers had a history of golf-related low back pain and therefore, it was not possible to discern whether the differences observed between the groups were possible contributors to their injuries or present as a result of their condition.

5. Conclusions.

The results of this study identified that the low back pain group of golfers showed statistically different muscle activation patterns in the erector spinae muscle prior to the initiation of the backswing. It is suggested that this activation pattern is present in order for this muscle to provide a more primary stabilising spinal support during such an activity. This may be in response to the poor functioning of the deeper stronger spinal stabilising muscles (e.g. multifidus). However, there continues to be a need for longitudinal studies that aim to establish whether the observed neuromuscular differences are present prior to the development of or as a result of the injury.

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Table Captions

Table 1: Average age, height, mass, body mass index (BMI) and handicap of the golfers with and without low back pain. Note that * indicates a significant difference ($p < 0.05$) between the LBP and NLBP groups.

Table 2: Mean onset timing (ms) relative to the start of the backswing (BS) and downswing (DS) and cessation timing (ms) relative to the top of the backswing for the lumbar erector spinae muscles. **Bolded** *p*-values represent those comparisons that returned a significant difference between the LBP and NLBP golfers.

Table 3: Mean onset timing (ms) relative to the start of the backswing (BS) and downswing (DS) and cessation timing (ms) relative to impact for the right and left external obliques.

Tables

	Low Back Pain Golfers					No Low Back Pain Golfers				
	Age (yrs)	**Height** (m)	**Mass** (kg)	**BMI** (kg/m^2)	**Handicap** (Strokes)	**Age** (yrs)	**Height** (m)	**Mass** (kg)	**BMI** (kg/m^2)	**Handicap** (Strokes)
Mean	**46.00**	**1.85***	**84.00**	**24.77**	**10.50**	**39.60**	**1.77***	**82.73**	**26.48**	**10.40**
SD	*17.85*	*0.09*	*5.26*	*2.47*	*8.43*	*13.94*	*0.06*	*5.78*	*2.50*	*4.14*

Table 1.

		Onset Timing (ms) Relative to Start of BS				Cessation Timing (ms) Relative to Top of BS				Onset Timing (ms) Relative to Start of DS			
Site	**Group**	**Mean**	**SD**	***p***	***d***	**Mean**	**SD**	***p***	***d***	**Mean**	**SD**	***p***	***d***
R L2	**LBP**	-190.0	373.9	**0.04**	0.83	-218.2	98.6	0.83	0.09	-67.3	115.5	0.10	0.68
	NLBP	153.3	451.4			-230.8	166.4			-0.8	75.5		
L L2	**LBP**	-327.3	287.6	**0.03**	0.96	-308.0	95.1	0.67	0.20	32.0	49.8	0.77	0.13
	NLBP	111.3	579.1			-282.7	157.2			40.0	72.0		
R L4	**LBP**	-40.8	483.8	0.08	0.73	-262.2	175.3	0.30	0.49	-20.0	48.7	1.00	0.00
	NLBP	307.3	484.0			-351.0	187.5			-20.0	73.5		
L L4	**LBP**	-296.7	343.9	**0.03**	0.93	-284.2	58.7	0.24	0.50	16.7	78.2	0.33	0.42
	NLBP	137.3	581.3			-222.0	166.6			51.0	83.2		

Table 2.

		Onset Timing (ms) Relative to Start of BS				Onset Timing (ms) Relative to Start of DS				Cessation Timing (ms) Relative to Impact			
Site	**Group**	**Mean**	**SD**	***p***	***d***	**Mean**	**SD**	***p***	***d***	**Mean**	**SD**	***p***	***d***
R EO	**LBP**					-638.3	198.2	0.96	0.02	925.8	98.8	0.22	0.48
	NLBP					-633.3	305.3			874.7	109.5		
L EO	**LBP**	-47.5	143.0	0.26	0.46					906.7	113.3	0.66	0.17
	NLBP	64.0	311.0							888.7	96.2		

Table 3.