Title:	Electromyography of the trunk and abdominal muscles in golfers with							
	and without low back pain							
Running Head:	EMG patterns of low back pain golfers							
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#### Abstract

Twelve male golfers who experienced low back pain (LBP) whilst playing or practicing golf and eighteen asymptomatic golfers were recruited and divided into handicap-specific groups; low-handicap golfers, with a handicap between 0 and 12 strokes; and high-handicap golfers with a handicap of between 13 and 29 strokes. The myoelectric activity of the lumbar erector spinae (ES) and the external obliques (EO) was recorded via surface electromyography (EMG), whilst the golfers performed 20 drives. The root mean square (RMS) was calculated for each subject and the data for the ES and EO were normalised to the EMGs recorded whilst holding a mass equal to 5% of the subjects' body mass at arms length and whilst performing a double-leg raise, respectively. The results showed that the low-handicap LBP golfers tended to demonstrate reduced ES activity at the top of the backswing and at impact and greater EO activity throughout the swing. The high-handicap LBP golfers demonstrated considerably more ES activity compared with their asymptomatic counterparts, whilst EO activity tended to be similar between the high-handicap groups. The reduced ES activity demonstrated by the low-handicap LBP group may be associated with a reduced capacity to protect the spine and its surrounding structures at the top of the backswing and at impact, where the torsional loads are high. When considering this with the increased EO activity demonstrated by these golfers, it is reasonable to suggest that these golfers may be demonstrating characteristics/mechanisms that are responsible for or are a cause of LBP.

## 1. Introduction

The modern golf swing is a complex, asymmetrical movement that is reliant on the powerful and precisely timed contractions of a number of skeletal muscles [1]. Although the game of golf has traditionally been perceived as a low-impact sport, the stresses placed on the body during the performance of the swing have been linked to numerous acute and overuse injuries [2, 3]. The most common site of injury in both amateur and professional golfers is the lower back and this reportedly accounts for up to 63 percent and 36 percent of all injuries developed in these sub-populations, respectively [1, 3, 4]. Although many low back injuries are idiopathic in nature, a reduction in lumbar spinal stability has been identified as a risk-factor for the development of these disorders [5, 6]. According to Panjabi [6] and Cholewicki and McGill [7], the mechanical stability of the spine is primarily controlled by the system of muscles surrounding the spinal column. However, previous research has indicated that the functional efficacy of the abdominal [8-10] and paraspinal muscles [9] is reduced in those individuals suffering from low back pain. It has been suggested that this dysfunction in individuals with low back pain may compromise the integrity of the spine and lead to development of a low back injury [7, 11-14]. In addition to this, persuasive evidence has been presented to suggest that this neuromuscular dysfunction may lead to reduced strength and endurance in the affected trunk muscles [14, 15].

Although research continues to refine the understanding of the functional deficiencies evident in individuals with low back pain, few researchers have sought to assess these characteristics in the golfing population [16]. Some of the first attempts to quantify the activity patterns of the trunk musculature during the performance of the golf swing were performed with healthy male golfers [17, 18]. However, to date, only three studies [1, 19, 20] have investigated the activity of the trunk muscles in golfers with low back pain (LBP). Evans and Oldreive [20] assessed the endurance of the Transversus Abdominus (TrA) muscle in a group of 10 golfers with LBP. Their findings suggested that golfers with a history of LBP had significantly reduced TrA endurance, which was indicative of a motor control deficit in this muscle. Alternatively, Grimshaw and Burden [19] measured the activity of the lower thoracic and lumbar muscles in a professional golfer suffering with LBP using surface EMG. The authors reported a reduction in the activity of the lumbar erector spinae during the downswing phase after three-months of muscle conditioning and technique modification, resulting in a possible decrease in the compression forces acting on the lumbar spine during this phase. In a previous investigation conducted by Horton and colleagues [1] surface EMG was used to assess the muscle activity patterns of the superficial abdominal muscles in golfers with and without LBP. This research indicated that the magnitude of the muscle activity for the rectus abdominis, external oblique and internal oblique did not differ significantly between those golfers with LBP and those without [1]. However, the authors reported that the lead external oblique (left in right-handed golfers) was activated significantly later during the backswing in the golfers with LBP when compared to the asymptomatic controls. Although these delays may suggest altered neuromuscular control in LBP golfers, they are unlikely to provide insight into the possible causes of their injuries, as the backswing has not typically been associated with a high risk of injury [2, 4].

Based on these findings, it is evident that there is still much confusion regarding the role that the muscles of the lower back play in the prevention of low back injuries. For example, some researchers have suggested that higher muscle forces are required to ensure that the structural integrity of the spine is maintained during dynamic tasks [e.g. 6, 7], whilst separate studies have postulated that larger forces should be avoided to lessen the loads acting on the spine [e.g. 2, 19]. Consequently, there is still a clear need for further research, which focuses on providing information on the muscle activity patterns of golfers with LBP and on the ability of these muscles to function as a unit to facilitate trunk motion and stability. Therefore, it was the aim of this investigation to assess the muscle activity of the lumbar erector spinae and the external oblique muscles in a population of golfers suffering with LBP and a population of golfers without LBP.

## 2. Methodology

## 2.2 Subjects

Golfers who reported experiencing golf-related LBP whilst playing or practicing golf were asked to complete the Short-Form McGill Pain Questionnaire (SF-MPQ) prior to participating in this study in order to establish the severity of their condition [21]. This questionnaire incorporates a visual analogue scale (100 mm line) that subjects use to rank the intensity of their pain, with '0' representing 'no pain' and '100' denoting 'worst possible pain' [21]. The SF-MPQ was logically constructed from the Long-Form McGill Pain Questionnaire (LF-MPQ) described by Melzack [22]. Previous research has provided evidence for the test-retest reliability [23], content validity [21], construct validity [e.g. 24], concurrent criterion validity [e.g. 21] and predictive validity [25] of both the SF-MPQ and the LF-MPQ, supporting their use in the assessment of clinical pain. Those golfers who recorded that the pain associated with their lower back was at either a mild or greater level ( $\geq 20$  mm) were recruited to participate in this project (n = 12 right-handed golfers). A further eighteen asymptomatic right-handed golfers who had no prior history of spinal deformities or spinal surgery were also recruited to serve as control subjects (NLBP). As those golfers comprising the LBP and NLBP groups had playing handicaps of between 0 and 29 strokes, the groups were further sub-divided into two handicap-specific groups (Table 1). Those golfers who reported having a playing handicap of 12 strokes or less were classified as the low-handicap golfers (LBP = 8 golfers; NLBP = 8 golfers). Similarly, those golfers with a self-reported handicap of 13 strokes or greater were considered high-handicap golfers (LBP = 4 golfers; NLBP = 10 golfers). All subjects provided written informed consent to participate in the investigation and the experimental methodology of this study was approved by the Human Research Ethics Committee at the University of South Australia.

## Insert Table 1 about here.

# 2.2 Task.

For the purposes of this research, each golfer was asked to perform a total of twenty drives towards a flag positioned 320 metres from the tee-off area using their own driver and employing their 'normal' swinging technique. So as to allow the golfers to perform the golf swing in an uninhibited fashion, all data collection took place on a grassed area at a local driving range. A two-metre square tee-off area was defined on the grass using a custom set of markers and the subjects were asked to position themselves within this box to ensure that both their body and the club were in this space throughout the swing. Prior to the collection of data, all of the subjects were encouraged to take the time to perform an appropriate warmup and to familiarise themselves with the surrounding experimental equipment.

## 2.3 Data Collection

Whilst performing the tee-shots, the activity of the erector spinae (ES) and the external obliques (EO) was measured using an AMLAB II surface EMG system (AMLAB International, AU). To perform this assessment, pairs of pre-gelled silver-silver chloride (Ag-AgCl) surface electrodes (Red Dot 2258-3, 3M, Ontario, CA), were positioned with a centre-to-centre distance of 2 cm over the muscles of interest. Each pair of electrodes was attached to a differential amplifier (gain x 1000, input impedance = 500 M $\Omega$ , common mode rejection ratio >110 dB, noise = <2  $\mu$ V) with a bandpass frequency of 15 – 480 Hz. The amplifier was connected to an IBM-compatible computer via a 12-bit analog-to-digital conversion board and the EMGs were collected at a sampling frequency of 1000 Hz using the AMLAB II (Build 19.8) software.

Prior to positioning the Ag-AgCl electrodes on the skin, the sites were shaved and cleaned thoroughly with an alcohol wipe to reduce the effects of impedance at the interface between the electrode and the skin. The recording electrodes were then positioned bilaterally over the erector spinae (posterior muscle) at the level of the fourth lumbar vertebra (L4), whilst the reference electrodes were located over the spinous processes of the second and third lumbar vertebrae. The bilateral activity of the external obliques (anterior muscle) was recorded by a pair of electrodes placed 15 cm lateral to the umbilicus at the transverse level [5] and reference electrodes, which were placed bilaterally over the tenth rib. Although, in this later case the reference position is not ideal (i.e. a moving rib in the rotation associated with the golf swing) it was all that could be achieved within the experimental constraints for this muscle group.

## 2.4 Data Analysis.

For the purposes of identifying any differences in the muscle activity patterns of the LBP and NLBP golfers, the data from the best three performances were considered. These trials were identified qualitatively by the principal researcher and were 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. In this context, it is important to add that the principal researcher was an experienced golfer who, at the beginning of the experiment, was coached by a professional in the identification of characteristics that constitute a 'good' golf drive. The raw EMG data for the three best trials were processed using the root mean square (RMS) method over consecutive periods of 200 ms and then averaged. Additionally, to facilitate the comparison of the muscle activity patterns of different individuals, each subject's RMS EMGs were normalised by expressing them as a multiple of the RMS EMGs recorded during two standardised tests. Although normalisation of EMG data typically involves the performance of a maximal isometric voluntary contraction [17, 18], research shows that this measure is unsuitable for use in LBP populations and could exacerbate the pre-existing injury in these

individuals [26]. Therefore, the test used to normalise the ES EMG data required the subjects to *s*tand with shoulders in 90° flexion whilst holding a mass equivalent to 5% body mass. Sub-maximal muscle contractions have been used previously to normalise EMG data for the back muscles in individuals who experience low back pain [e.g. 27]. The EMG data for the EO muscles was normalised using the EMGs collected during the performance of a seated double-leg raise, similar to that used by Horton and colleagues [1]. The normalised EMG for the best three trials for each subject in each group were averaged and examined at address (pre-swing); at the top of the backswing/start of the downswing (TBS/SDS); and at impact between the clubhead and the ball.

## 2.5 Statistical Analysis.

For the purposes of assessing any statistically significant differences between the groups and the left and right sides, the SPSS 12.0 statistical software package (SPSS Inc., USA) was used to conduct an independent samples one-way analysis of variance (ANOVA), with the level of significance set at p < 0.01. In addition to this, effect sizes were calculated using the Cohen's *d* method [28] to account for the small sample sizes used in this research. Effect sizes of less than 0.2 and between 0.2 and 0.5 were considered a negligible effect and a small effect, respectively. A reported effect size between 0.5 and 0.8 was deemed to be a medium effect, whilst a value greater than 0.8 represented a large effect [28]. A larger effect size suggested that it was more probable that a statistically significant difference would be identified during the statistical analysis [29].

## 3. Results

## 3.1 L4 Erector Spinae

#### Insert Figure 1 about here.

The normalised L4 ES myoelectric activity for the low-handicap LBP and NLBP golfers (Figure 1) tended to show a progressive increase in amplitude between address and impact. At address, the right- and left-side activity of the L4 ES for the LBP and NLBP golfers did not differ significantly. However, the EMGs for the LBP group tended to be smaller than those reported for the NLBP golfers at both TBS/SDS and impact. These results show a reduced muscle activity level in the LBP group when comparing the LBP and NLBP groups for left and right muscle activity at TBS/SDS and impact (for example, right-side ES activity in the LBP group at TBS/SDS compared with right-side ES activity of the NLBP group at TBS/SDS). Although these bilateral reductions were found to be significantly different at impact (Right: p = 0.007, d = 0.86; Left: p = 0.002, d = 0.98), they did not achieve statistical significance at TBS/SDS (Right: p = 0.02, d = 0.77; Left: p = 0.21, d = 0.34).

## Insert Figure 2 about here.

Figure 2 shows the normalised EMGs recorded at the L4 ES site for the high-handicap LBP and NLBP golfers. The EMGs recorded for the high-handicap LBP golfers during the address phase were comparable to those reported for the NLBP golfers during the same phase. However, at TBS/SDS, the LBP group demonstrated significantly greater right- and left-side activation of the L4 ES compared with the NLBP group (Right: p=0.001, d=1.50; Left: p=0.002, d=1.15). At impact, both the LBP and NLBP golfers demonstrated similar right-side activity for the L4 ES, whilst the LBP group was shown to have reduced left-side activity at this point; although this difference was not significant (p=0.10, d=0.60).

#### Insert Figure 3 about here.

The data presented in Figure 3 depicts the normalised RMS EMGs recorded for the lowhandicap LBP and NLBP subjects over the EO muscle site. The bilateral (left and right) activity of the EO during the address phase was quite low in both the LBP and NLBP groups. The results of the statistical analysis demonstrated that the EO activity of the low-handicap LBP and NLBP golfers did not differ significantly during this phase of the movement (Right: p=0.02, d=0.70; Left: p=0.58, d=0.17). At TBS/SDS, the LBP golfers tended to record greater mean EMGs compared with the golfers in the NLBP group (Right: p=0.005, d=0.87; Left: p=0.040, d=0.62). Similarly, left versus left and right versus right EO EMGs were shown to be greater for the low-handicap LBP group at impact; however these differences were not found to be statistically significant.

#### **Insert Figure 4 about here.**

In contrast to the data presented for the low-handicap golfers, the muscle activity recorded for the high-handicap LBP and NLBP golfers at address, TBS/SDS and impact (Figure 4) only approached significance for the left EO during the address phase (p = 0.014; d = 0.90). Similarly, there was very little difference observed between the groups with respect to the myoelectric activity recorded at TBS/SDS or impact. The normalised EMGs recorded at the L4 ES and EO muscle sites for the low- and high-handicap groups comprising the LBP and NLBP golfers are summarised in Table 2.

#### Insert Table 2 about here.

# 3.3 Left- versus right-side comparisons within groups (i.e. within LBP high- and lowhandicap or within NLBP high and low-handicap)

Table 2 shows the left- versus right-side comparison for muscle activity within each group and each handicap classification. Statistical comparisons indicated similar levels of ES activity during all phases in the low-handicap LBP and NLBP groups. However, in the highhandicap sub-group, both the LBP and NLBP golfers significantly activated the right-side ES more than the left-side at TBS/SDS (LBP: p = 0.001; NLBP: p = 0.000).

For the EO muscle, similar left- versus right-side activity patterns were found in both the low- and high-handicap LBP and NLBP groups at the address and at TBS/SDS. However, at impact, the low-handicap LBP and NLBP and the high-handicap NLBP golfers tended to demonstrate increased right-side EO activity, although this findings was not significant.

## 4. Discussion

### 4.1 L4 Erector Spinae

At TBS/SDS, the high-handicap LBP and NLBP groups were shown to activate the right L4 ES to a significantly greater degree compared with the left-side L4 ES. Grimshaw and Burden [19] presented data that depicted increased right-side L4 ES activity during the downswing for a male professional golfer both prior to and following a three-month intervention period. According to Pink and colleagues [17] this increase in right-side ES activity is required to counteract the effects of gravity on the body during the early stages of the downswing.

In terms of the L4 ES muscle activity, there were a number of characteristic differences between the LBP and NLBP golfer groups. Excluding the address phase of the swing, it is evident that the low-handicap LBP golfers generally demonstrated less ES activity than the low-handicap NLBP golfers at TBS/SDS and significantly reduced levels at impact. It is well documented that without adequate support from its surrounding musculature, the lumbar spine is inherently unstable and prone to buckling under compressive loads of about 90 N [e.g. 7, 11]. However, a recent review highlighted that increased trunk stiffness is not always desirable in dynamic situations, as greater muscle forces are required to displace a stiffer spine [30], which may lead to injury in some specific situations. Therefore, although the reduced levels of ES activity may correspond with decreased lumbar spinal stability, it is plausible to suggest that this finding is due to an adaptation that these golfers have made due to their injury state. That is, a decrease in muscle force would equate to reduced spinal loads and less exacerbation of their low back injuries; however, possibly at the expense of trunk stability. Although the results presented for the high-handicap group at TBS/SDS tended not to support this notion, with the LBP group demonstrating significantly greater ES activity, the LBP group did show a considerable decrease in ES muscle activity at impact.

#### 4.2 External Obliques

Although not significant, the findings of this study suggested that the high-handicap NLBP and the low-handicap LBP and NLBP golfers tended to activate the right-side EO more forcefully at impact compared with the left EO. Horton et al. [1] observed a similar increase in right-side EO activity in a group of elite male golfers, suffering with chronic LBP, but were also unable to report this as a statistically significance difference. During the final stages of the backswing and the early stages of the downswing, an increase in right EO activity is expected as this muscle plays an important role in contributing to the rotation of the trunk back toward the target [17]. Similarly, trunk rotation back toward the flag during the downswing phase would be expected to activate the left internal oblique (IO), as this muscle has been shown to assist the right EO with such movements [1].

The findings presented for the EO for the low-handicap LBP golfers suggested that these golfers tended to activate this muscle to a greater extent compared with their asymptomatic counterparts at address, TBS/SDS and impact. This increased EO activity evident in the low-handicap LBP golfers may have important implications for an understanding of the injury characteristics and/or mechanisms in this population. As the EO muscles are primarily involved in producing trunk flexion and rotation [17], increased EO activity in the low-handicap LBP golfers would suggest that these golfers rotated their trunks at a much greater velocity than their asymptomatic counterparts. Although an increased rate of trunk rotation during the downswing may increase the clubhead velocity at impact, larger torsional loads would also be expected. When considering this with the reduced ES activity (and possibly spinal stability) in these golfers, it is feasible that such an increase in rotatory force would exacerbate the pain in these golfers and possibly have contributed to their injuries.

It is important in any research to acknowledge a number of limitations when reviewing the findings presented in this study. Firstly, it is important to consider that the number of subjects comprising the groups was small (statistically), which may have effectively made it difficult to detect a real difference between the populations, with respect to the patterns of muscle activation in the golfers with or without LBP and with high- or low-handicaps. Therefore, effect sizes were determined for the statistical comparisons made between the groups and it is suggested that these data be considered when reviewing the results (expressed as *d*-values). Secondly, as the bilateral activity of the muscles was detected from the skin's surface, it is possible that a proportion of the signal was attributable to the muscles underlying and surrounding the muscles of interest; otherwise known as cross-talk. However, standardised techniques (i.e. location over the belly of the muscle, skin preparation, differential amplifiers and noise reduction techniques) used with EMG data collection were utilised to minimise this factor. Thirdly, as many of the subjects were recruited from the population of golfers who were practicing or playing golf at the venue during one of the scheduled testing sessions, it was not feasible to retrieve additional information related to their medical and rehabilitative treatment history. Finally, and perhaps the most important limitation of this investigation was that the LBP golfers had already had a history of golfrelated low back pain prior to their participation in this study and therefore, it was not possible to discern whether the differences observed between the groups might have contributed to their injury or have been a result from their injury. Hence, there still remains a need for longitudinal research aimed at identifying whether neuromuscular deficiencies are present prior to the development of the disorder or are a consequence of the disorder.

# 5. Conclusions.

The results of this investigation showed that, in general, the low-handicap LBP golfers demonstrated reduced ES activity and increased EO activity at TBS/SDS and impact, compared with their NLBP counterparts. Although reduced ES activity could be expected to contribute to reduced lumbar compression forces, it may also cause the spine to become unstable, which could be particularly hazardous when considering the increased EO activity (and possible torsional loads) demonstrated by these golfers. These findings may highlight neuromuscular deficiencies in the low-handicap LBP golfers and could have important implications for the understanding of and development of LBP during golf.

# **Practical Implications.**

- An improved understanding of any neuromuscular deficiencies for the trunk muscles in injured golfers will contribute to a better understanding of some of the possible mechanisms of low back injuries in this population.
- Research focussed on improving an understanding of the possible causes of such injuries, will help develop and refine effective interventions to reduce their prevalence in the golfing (and/or other) population.
- Any reduction in the prevalence of those injuries associated with golf (and other sports) will help to promote future participation in such activities, which will benefit the overall health and well-being of this community.

# Acknowledgements.

The authors thank the staff at the Kerry Elliss driving range, Adelaide, AUSTRALIA and the Riverside Golf Club, Mildura, AUSTRALIA for allowing this research to be carried out on their respective practice ranges. Additionally, the authors acknowledge the golfers who volunteered and made this research possible.

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Tables.

	L	.ow-Handicaj	pped Golfer	s	High-Handicapped Golfers						
		Low Back P	ain (n = 8)	Low Back Pain $(n = 4)$							
	Age (yrs)	Height (m)	<u>Mass (kg)</u>	Handicap		Age (yrs)	Height (m)	Mass (kg)	Handicap		
Mean	37.50	1.83	83.63	6.00		63.00	1.88	84.75*	19.50		
SD	14.56	0.09	4.98	5.10		9.76	0.09	6.50	6.14		
		Asymptoma	tic (n = 8)		Asymptomatic $(n = 10)$						
	Age (yrs)	Height (m)	Mass (kg)	<u>Handicap</u>		Age (yrs)	Height (m)	Mass (kg)	<u>Handicap</u>		
Mean	33.25	1.74	77.13	7.25		52.40	1.72	76.20*	18.60		
SD	14.54	0.11	10.18	3.15		10.93	0.10	14.02	6.22		

<u>Table 1</u>: The mean age, height, mass and handicap of the low-handicap and highhandicap golfers comprising the low back pain and asymptomatic control groups. N.B. \* indicates a significant difference (p < 0.01) between the LBP and Asymptomatic groups

		L4 Erector Spinae									
			Low-Handicap	)	High-Handicap						
		Address Right Left	TBS/SDS Right Left	<u>Impact</u> <u>Right</u> <u>Left</u>	Address Right Left	TBS/SDS Right Left	Impact Right Left				
LBP Golfers	Mean <i>SD</i>	0.94 0.97 0.25 0.34	1.29 2.03 0.86 2.19	3.30* 3.00* 0.97 1.40	0.98 0.87 0.17 0.35	4.74* 1.85* 1.16 1.44	3.19 2.23 1.41 1.45				
NLBP Golfers	Mean <i>SD</i>	1.12 1.11 0.37 0.28	3.603.002.523.46	5.67* 4.96* 2.66 2.54	1.23 1.08 0.26 0.27	2.01* 0.79* 1.33 0.66	3.08 3.19 1.34 1.72				
		External Obliques									
			Low-Handicap	)	High-Handicap						
		Address	TBS/SDS	<b>Impact</b>	Address	TBS/SDS	<b>Impact</b>				

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		<u>Right</u>	Left	<u>Right</u>	Left	<u>Right</u>	Left	<u>Right</u>	Left	<u>Right</u>	Left	<u>Right</u>	Left	
I DD Calfana	Mean	1.05	0.59	6.87*	5.59	9.38	6.70	0.68	0.90*	4.31	3.44	4.91	4.99	
LBP Gollers	SD	0.44	0.38	4.27	4.70	3.96	5.96	0.28	0.42	2.10	1.48	2.68	2.12	
NLBP Golfers	Mean SD	0.72 0.51	0.66 0.38	3.86* 2.43	3.38 2.01	6.52 4.33	4.93 2.87	0.60 <i>0.31</i>	0.55* <i>0.40</i>	4.25 3.60	3.59 2.96	6.40 5.67	4.11 2.59	

<u>Table 2</u>: Summary of the mean and standard deviation values of the normalised RMS EMGs recorded for the low- and high-handicap LBP and NLBP golfers at the L4 ES and EO muscle sites. N.B. \* denotes a significant difference (p < 0.01) between the LBP and NLBP groups.

Figures.



**<u>Figure 1</u>**: The average (± 1 SD) normalised L4 erector spinae EMGs recorded for the <u>low-handicap</u> LBP and NLBP golfers at address, TBS/SDS and impact.



**<u>Figure 2</u>**: The mean ( $\pm$  1 SD) normalised L4 erector spinae muscle activity at address, TBS/SDS and impact for the <u>high-handicap</u> LBP and NLBP groups.



**<u>Figure 3</u>**: The average (± 1 SD) normalised EMGs for the external obliques, recorded at address, TBS/SDS and impact for the low-handicap LBP and NLBP golfers.



<u>Figure 4</u>: The mean  $(\pm 1 \text{ SD})$  normalised muscle activity of the external obliques recorded for the high-handicap LBP and NLBP groups at address, TBS/SDS and impact.