

The effects of Swimming Fatigue on Shoulder Strength, Range of Motion, Joint Control, and Performance in Swimmers.

Abstract

**Purpose:** To investigate the effects of training induced fatigue on shoulder strength, ROM, joint position sense, and stroke length in elite competitive swimmers.

**Methods:** Seventeen national level swimmers performed maximum isometric strength (internal and external rotation), ROM, and joint position sense tests before and after a fatiguing 8x100m training set. Stroke length, heart rate, blood lactate and blood glucose levels were recorded throughout.

**Results:** Peak blood lactate, blood glucose levels, and heart rate increased significantly ( $P<0.001$ ) post-training confirming fatigue. Reductions were observed in stroke length of both arms ( $P<0.001$ ), external rotation range of motion ( $P<0.001$ ,  $-5.29^\circ$ , Right shoulder;  $P=0.04$ ,  $-3.18^\circ$ , Left shoulder) and joint position sense in their dominant (breathing side) arm ( $P=0.03$ ).

**Conclusions:** This investigation revealed a reduction in stroke length across both arms and also an arm bias in swimming whereby a greater reduction in both external rotation range and joint position sense was observed in the dominant arm when fatigued. This has highlighted a relationship between fatigue and potential mechanism of shoulder pathology in swimmers.

## Introduction

Shoulder pain in swimming is a major cause for concern (Beach et al., 1992; Yanai & Hay., 2000; Wolf et al., 2009; Lynch et al., 2010; Tate et al., 2012), with shoulder injuries occurring in 30% to 91% of swimmers (Beach et al., 1992; Wolf et al., 2009; Walker et al., 2011; Tate et al., 2012), and a wide acceptance that shoulder pain is a normal part of swimming (Hibberd & Myers., 2013). This pathology, often characterised as pain following repeated shoulder impingement in swimmers, is described as “swimmers shoulder” [sic] (Kennedy and Hawkins, 1974).

Competitive swimmers practice 6 -7 days a week, swim on average between 10,000m and 14,000m on each day (Kluemper et al., 2006; Lynch et al., 2010; Sein et al., 2010; Allegrucci et al., 1995), and perform an estimated 16000- 25000 shoulder rotations during a typical training week (Scovazzo et al. 1991). With 80% of training time spent on freestyle stroke (Beach et al., 1992), and 90% of the propulsion for this coming from the upper extremities (Barbosa et al., 2008), excessive training volumes has been identified as a possible contributing factor to **shoulder pathology** (Beach et al., 1992; Tate et al., 2012; Walker et al., 2011; Wolf et al., 2009; Hibberd & Myers., 2013). **There is a clear relationship between distances trained and shoulder pathology, with swimmers who train for longer distances appearing up to four times as likely to have a shoulder pathology as those who train for less** (Beach et al., 1992; Tate et al., 2012; Walker et al., 2011; Sein et al., 2010).

**Mechanisms of overuse shoulder injury appear to be related to the inability to maintain optimal posture and movement patterns, resulting in continuous aggravation of susceptible tissues that is worsened under fatigue due to detriments in strength, proprioception, and**

ROM (Beach et al., 1992; Rupp et al., 1995; Pink & Tibone, 2000; Yanai & Hay, 2000; Kluemper et al., 2006; Lynch et al., 2010). Competitive swimmers with decreased muscle endurance in external rotation and abduction may be more likely to develop shoulder pathology (Beach et al., 1992). This may be compounded by a protracted shoulder posture further predisposing swimmers to a higher risk of shoulder injuries (Kluemper et al., 2006; Lynch et al., 2010; Pink & Tibone, 2000). If infraspinatus or teres minor are weak, not activating correctly, or are fatigued (Tyler et al., 2000; Michener et al., 2003; Koester et al., 2005; Heyworth and Williams 2009; Ellenbecker and Cools 2010; Heyworth and Page 2011), or if the scapula is already in a forward position, preventing further protraction, (Kluemper et al., 2006; Lynch et al., 2010; Pink & Tibone, 2000), abnormal movement of the gleno-humeral and scapulothoracic joints may predispose an athlete to compression of the sub-acromial space, leading to impingement of supraspinatus tendon and the subacromial bursa (Yanai & Hay, 2000; Lynch et al., 2010). Shoulder weakness or fatigue in outer range may result in rotator cuff impingement, predominantly of supraspinatus during the abduction and internal rotation that is characteristic of the recovery phase of the freestyle stroke (Yanai & Hay, 2000).

It is also possible that there is a disparity in the levels of fatigue observed in different muscles. In tennis, Ellenbecker & Roetert (1999) observed that the internal rotator muscles were more fatigue resistant than external rotators. Although this may not directly replicate fatigue in swimming, the relative shift in internal and external rotator strength with fatigue may indicate that internal rotators retain more strength under fatigue than the much smaller external rotators. Indeed, there is some evidence that training the external rotators has a positive effect on reducing shoulder pain in swimmers (Lynch et al., 2010).

As well as detriments to strength, several studies have also identified a detrimental effect of fatigue on proprioception (Skinner et al., 1986; Lee et al., 2003), with as much as a 73% increase in the threshold of detection of movement (Carpenter et al., 1993). This may have implications for the accurate replication of movement patterns that are central to both performance and injury prevention.

These cumulative effects of fatigue on strength, range of motion, and proprioception appear to have a profound impact on swimming performance. Figueiredo et al., (2012) found while testing 10 male competitive swimmers aged 21.6 ( $\pm 2.4$ ) with 11.00 years of competitive swimming, a significant decrease in biomechanical and coordinative parameters of swimmers occurred under fatigue. Speed decreased by 13.8% ( $P=0.00$ ), stroke rate (frequency) by 5.1% ( $P=0.00$ ) and stroke length by 6.9% ( $P=0.01$ ) with an increase in blood lactate levels of 11.12  $\text{mmol}^{-1}$  ( $\pm 1.65 \text{ mmol}^{-1}$ ). This test was able to show biomechanical changes due to fatigue, but only over a very short period of time (96.1 seconds  $\pm 2.1$ ); this is representative of a race distance but does not mimic the cumulative effects of training. It does however show that swimmers have reduced joint control and proprioception, which may contribute to pathology under fatigue (Carpenter et al., 1993).

Previous work has suggested that spatial trajectory and stroke parameters of international swimmers is not easily changed even by the impairments imposed by fatigue, suggesting that stroke rate becomes the most determinant factor of swimming velocity for top level swimmers and not stroke length (Anjouannet et al., 2006; Toussanit et al., 2006). When power output declines, both swim speed and stroke rate decrease, but stroke length remains constant (Toussanit et al., 2006). It appears that the more experienced the swimmer the less deviation in stroke length while under fatigue (Figueiredo et al., 2012; Anjouannet et al.,

2006) and may be a factor in the reduced occurrence in shoulder pathology observed in more experienced swimmers (Wolf et al., 2009).

It is therefore hypothesised that interventions that mitigate fatigue and promote the maintenance of optimal posture, proprioception, mobility, and movement patterns during swimming may reduce the chances of shoulder pathology. To identify potentially helpful interventions this study aims to investigate the impact of training induced fatigue on strength, mobility, joint reposition ability, and parameters of performance.

## Methodology

### Participants

Seventeen national level youth (8 male, 9 female) swimmers from a North West swimming squad, aged 15.9 years ( $\pm 1.09$ ), height 174cm ( $\pm 7.52$ cm), weight 66.06kg ( $\pm 8.07$ kg), were targeted as part of a convenience sample and invited to participate. The seventeen participants constituted every member of the youth squad. Each participant gave informed consent prior to participation and gave permission for all collected data to be used in this study. All swimmers competed regularly at national level championships, were right hand dominant, had approximately 7 years of regular training experience, and considered freestyle their preferred stroke. Ethical approval was gained prior to the study from the institution's RGECC.

### Testing procedure

Swimmers completed their normal standardised, pre-training, warm up (1600m consisting of full stroke, arms only, legs only, and drills) before having baseline blood lactate and glucose levels recorded.

Maximum isometric strength was measured in internal and external rotation using a handheld dynamometer that had been calibrated with digital scales to a known weight and had previously been deemed to be a reliable method (Dover and Powers, 2003). Swimmers stood upright and each arm was positioned in 90° of shoulder abduction and 90° of elbow flexion in the frontal plane. The swimmer then positioned themselves so that the hand held dynamometer (Micro FET 2, Hoggan Health Industries) could be pressed maximally against a pillar, with the swimmers pressing forwards (to measure internal rotation strength) and backwards (to measure external rotation strength). This position was chosen as it was deemed to be the most suitable compromise between replicating the arm position during the stroke, while also being repeatable. Three attempts were taken with the best score being recorded. Pilot testing was conducted on the swimmers prior to this study, during which the repeatability of each, measure was determined using intra-class correlation coefficient (model 2,1). The comparison of first and second measurement indicated a strong correlation between the two measures of .96 ( $p < 0.01$ ) (internal rotation) and .92 ( $p < 0.01$ ) (external rotation).

Internal and external range of motion was assessed at the glenohumeral joint using a goniometer (Baseline HiRes, 12-1000HR) with arm abducted to 90°, elbow flexed at 90° while lying on a mobile plinth. Swimmers were then instructed to actively move their arm as far as they were able into internal and external rotation. Pilot testing was

conducted on the swimmers prior to this study, during which the repeatability of each, measure was determined using intra-class correlation coefficient (model 2,1). The comparison of first and second measurement indicated a strong correlation between the two measures of .92 ( $p < 0.01$ ) (internal rotation) and .93 (external rotation) ( $p < 0.01$ ).

Joint position sense was measured with swimmers lying prone with arm abducted to  $90^\circ$ , elbow flexed at  $90^\circ$ . The arm was then passively externally rotated to  $45^\circ$  before returning to the initial position. With their eyes closed swimmers were asked to relax and then to resume the arm position (Herrington et al., 2010).

Following the initial test session, swimmers performed 8 x 100m training swims off a 2-minute interval. Target times were prescribed for each swimmer based on 85% of their best 100m times (Maglischo, 2003; Barden & Kell, 2009). The 8 x 100m swim set of a 2-minute interval is a regular training session undertaken by these participants and was chosen as it was deemed by the coaches as the most reliable method of inducing fatigue and that was also representative of typical training practice.

Immediately on completion of the last swim, swimmers exited the pool and repeated the isometric strength, range of motion, and joint position tests. Efforts were made to ensure that this began as soon as possible post-swim (within 60s) and that the order and timing of the post-fatigue tests replicated those of the pre-fatigue tests.

Blood samples were taken 3 minutes post-exercise (Gore, 2000; Maglischo, 2003; Aujouannet et al., 2006).

Swim stroke was recorded using a high-speed camera (Panasonic Lumix FT5) at 30 frames per second (Anjouannet et al., 2006; Toussanit et al., 2006) and was analysed using Quintic, using the 3<sup>rd</sup> 25m of each 100m swim. (Figure 1).

### Data analysis

All data were analysed using SPSS (Version 20) with ANOVA and post-hoc tests used to determine difference pre to post fatigue. Prior to analysis both visual inspection of the histogram and Shapiro-Wilk results (no sig deviation from normality) indicated the data were normally distributed. Pearson's correlation coefficient was used to determine the correlations between stroke length and the tested variables.

### Results

#### Lactate

A significant increase ( $P < 0.001$ ;  $d = 4.8$ ;  $r = .92$ ) was found in blood lactate, post- 8 x100m test, compared to baseline blood lactate ( $8.81 \text{ mmol}^{-1} \pm 2.09$  vs  $1.55 \text{ mmol}^{-1} \pm 0.42$ ). Blood glucose levels also increased from  $5.01 \text{ mmol}^{-1} \pm 0.91$  pre-test to  $7.12 \text{ mmol}^{-1} \pm 1.22$  post-test ( $P < 0.001$ ;  $d = 1.96$ ;  $r = .7$ )(Table 1), confirming the high intensity nature of the training session (Pyne et al., 2001; Aujouannet et al., 2006; Anderson et al., 2006; Anderson et al., 2008; Carr et al., 2011; Figueiredo et al., 2012).

#### Time, Heart rate and stroke length

Swimmers were asked to maintain swim pace at a level equivalent to 85% of their best 100m time. No significant difference was found in swimming times during the test ( $P = 0.19$ ) demonstrating that swimmers maintained an even pace throughout. A significant increase in



heart rate was observed between the first ( $167 \pm 17$  BPM) and final ( $199 \pm 21$  BPM) repetitions of the 8 x100m training set ( $P < 0.001$ ;  $d = 1.67$ ;  $r = .64$ ). (Table 2)

A significant reduction in left arm stroke length ( $P < 0.001$ ;  $d = 1.26$ ;  $r = .53$ ) and right arm stroke length ( $P < 0.001$ ;  $d = 1.14$ ;  $r = .5$ ) was recorded during the test, from 1.36m ( $\pm 0.14$ m) left and 1.37m ( $\pm 0.14$ m) right on the first swim to 1.22m ( $\pm 0.07$ m) left and 1.24m ( $\pm 0.08$ m) right on the final swim. (Table 3) (Figure 2.)

### Joint reposition sense

A significant increase in mean joint reposition variance (as defined by the magnitude of error from the 45° reposition target angle) was observed following fatigue on the right arm from  $4.92^\circ \pm 4.71$  pre to  $9.62^\circ \pm 6.63$  post ( $P = 0.03$ ; Cohen's  $d = .82$ ;  $r = .38$ ), representing a 95.31% increase in variance. No significant differences were observed on the left arm. (Table 4).

No significant correlation between joint position sense and stroke length was found pre- or post- fatigue.

### Range of motion

Range of motion into external rotation was also significantly reduced in both right ( $-5.29^\circ$ ;  $P < 0.001$ ;  $d = .75$ ;  $r = .35$ ) and left arms ( $-3.18^\circ$ ;  $P = 0.04$ ;  $d = .42$ ;  $r = .2$ ) post fatigue. (Table 3). No significant differences were observed for internal rotation.

No significant correlation between external rotation range and stroke length was found pre- or post- fatigue.

### Strength

No significant strength differences were observed pre- to post-fatigue in either the internal or external rotators of either shoulder.

There was, however, a statistically significant relationship between right external rotation strength and stroke length, pre- ( $R=-0.635$ ) and post- ( $R=-0.643$ ) fatigue using a Pearson correlation coefficient.

### Discussion

Following 8 x 100m training swims, off a 2-minute interval at 85% of maximum swim time, several changes were observed. These included a significant reduction in stroke length in both right (13cm;  $P<0.001$ ;  $d = 1.14$ ;  $r = .5$ ) and left (14cm;  $P<0.001$ ;  $d = 1.26$ ;  $r = .53$ ), arms, reduced range of motion into external rotation in both right ( $-5.29^\circ$ ;  $P<0.001$ ;  $d = .75$ ;  $r = .35$ ) and left arms ( $-3.18^\circ$ ;  $P=0.04$ ;  $d = .42$ ;  $r = .2$ ), and a 95.3% increase in joint reposition variance on the dominant right arm ( $P=0.03$ ; Cohen's  $d = .82$ ;  $r = .38$ ).

Using Rhea's (2004) interpretation of effect sizes for highly trained individuals, the effect of fatigue on stroke length can be considered as large; the reduction in the right and left arm range of motion can be considered moderate-to-large and moderate respectively; the increase in joint reposition variance can be considered moderate. The observed reduction of stroke length under fatigue is in contrast to previous findings (Anjouannet et al., 2006), but supports Figueiredo et al., (2012) who observed that the reduction in stroke length accompanied the rise in blood lactate during a maximal 200m swim test, with stroke frequency increasing to maintain velocity. The contrast with Anjouannet et al may be due to the relatively low volume of their fatiguing protocol (4 x 50m) compared with the volume and intensity of this particular swim training session (8 x 100m). Figueiredo et al., (2012), also observed that the right arm phase took longer than the left arm phase, associating this with 'handedness' of the swimmers and suggested that the dominant arm is preferentially used for propulsion and

the non-dominant hand is preferentially used for control and support. This may explain our findings that the dominant arm showed a greater reduction in external rotation range of motion, compared to the non-dominant, and also a significant reduction in joint position sense, that was not observed in the non-dominant. This fatigue in the dominant arm tends to agree with previous results suggesting that fatigue affected joint position sense and proprioception (Carpenter et al., 1998; Lee et al., 2003).

Although there were large decreases observed in some individuals, fatigue, however, had no significant effect on maximal isometric strength in both internal and external rotation in either arm. Despite this, we still observed a significant strong correlation between external rotation strength and stroke length, both pre- and post- fatigue. This may reflect the fact that there was a general (but non-specific) trend towards reduced strength with fatigue. This may be related to the muscle endurance capabilities of swimmers who regularly undergo endurance based swimming training (Kluemper et al., 2006; Lynch et al., 2010; Sein et al., 2010; Allegrucci et al., 1995), with isometric strength, as assessed in this study, not directly affected in the same way that joint position sense and range are. It is unlikely that, due to the repetitive nature of the sport, swimmers ever produce a maximal internal rotation contraction when swimming. It is possible that a muscle strength or local muscular endurance test that focuses on multiple repetitions may produce different results with endurance trained athletes, with previous research observing significant reduction in internal and external rotation strength over 50 repetitions on an isokinetic dynamometer (Beach et al., 1992).

### Practical Recommendations

These results, along with the clear link between training volume and impingement injuries (Lynch et al., 2010; Beach et al., 1992; Walker et al., 2011), leads us to question the wisdom

of adolescents swimming the high volumes reported (Lynch et al., 2010; Sein et al., 2010; Allegrucci et al., 1995), without first having an injury prevention and strengthening programme focused on the functional eccentric strength of the external stabilising muscles of the shoulder. In each concentric ‘power stroke’ the small stabilising muscles must work synergistically to prevent the anterior translation of the humeral head. Continued fatigue and poor joint position sense could result in anterior humeral head translation due to the dominance of overactive pectoral minor and latissimus dorsi muscles leading to laxity of the weaker posterior muscles (Tate et al., 2012). Anterior translation places the shoulder in a position whereby the subacromial space is compressed, potentially aggravating tissues such as the subacromial bursa and supraspinatus, leading to the impingement syndrome known as ‘swimmers shoulder’. Anterior translation will also cause an internal rotation of the humerus putting additional strain on the long head of biceps tendon.

Given the significant reduction in external rotation range and increased proprioceptive variance as a result of fatigue, along with the significant correlation observed between stroke length and strength, it is possible that competitive swimmers may benefit from a shoulder external rotation strengthening programme that includes inner range external rotation activities, mobility training aimed at restoring external rotation range of motion, and proprioception training. It is possible that such a programme could help swimmers maintain stroke length and external rotation range after fatiguing swimming. This may be relevant to injury prevention because it may help swimmers offset the pull from the dominant internal rotators and reduce potential for impingement of supraspinatus tendon (Kluemper et al., 2006).

## Conclusion

This investigation revealed a reduction in stroke length across both arms and also an arm bias in swimming whereby a greater reduction in both external rotation range and joint position sense was observed in the dominant arm when fatigued. This has highlighted a relationship between fatigue and potential mechanism of shoulder pathology in swimmers. Competitive swimmers may benefit from a shoulder external-rotation strengthening programme that includes inner range external rotation activities, mobility training aimed at restoring external rotation range of motion, and proprioception training. It is possible that such a programme could help swimmers maintain stroke length and external rotation range after fatiguing swimming with benefits for both performance and injury prevention.

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**Figure 1 – Picture of calibration square used to measure stroke length.**



**Table 1 – Table showing changes in blood lactate and blood glucose pre- and post-test.**

|   | <u>Mean</u> | <u>SD</u>   | <u>95%CI</u>       | <u>TTest</u>      |
|---|-------------|-------------|--------------------|-------------------|
| <u>Base lactate (mmol.l<sup>-1</sup>)</u> | <u>1.55</u> | <u>0.42</u> | <u>1.35 - 1.75</u> |                   |
| <u>Peak lactate (mmol.l<sup>-1</sup>)</u> | <u>8.81</u> | <u>2.09</u> | <u>7.82 - 9.8</u>  | <u>P&lt;0.001</u> |
| <u>Base glucose (mmol.l<sup>-1</sup>)</u> | <u>5.01</u> | <u>0.91</u> | <u>4.58 - 5.44</u> |                   |
| <u>Peak glucose (mmol.l<sup>-1</sup>)</u> | <u>7.12</u> | <u>1.22</u> | <u>6.54 - 7.7</u>  | <u>P&lt;0.001</u> |

**Table 2 – Table showing changes in joint position sense (proprioception) pre- and post-test.**

| <u>Proprioception Variance</u> | <u>Mean</u> | <u>SD</u>   | <u>95% CI</u>       | <u>% change</u> | <u>TTest</u>  |
|--------------------------------|-------------|-------------|---------------------|-----------------|---------------|
| <u>Pre-fatigue Right (°)</u>   | <u>4.92</u> | <u>4.71</u> | <u>2.68 – 7.16</u>  |                 |               |
| <u>Post-fatigue Right (°)</u>  | <u>9.62</u> | <u>6.63</u> | <u>6.47 – 12.77</u> | <u>95.31%</u>   | <u>p=0.03</u> |
| <u>Pre-fatigue-Left (°)</u>    | <u>7.85</u> | <u>6.25</u> | <u>4.88 – 10.82</u> |                 |               |
| <u>Post-fatigue Left (°)</u>   | <u>9.08</u> | <u>6.50</u> | <u>5.99 – 12.17</u> | <u>15.69%</u>   | <u>p=0.22</u> |

**Table 3 – Table showing external rotation range of motion pre- and post-test.**

| <u>External Rotation ROM</u> | <u>Mean</u>  | <u>SD</u>   | <u>95% CI</u>        | <u>% Change</u> | <u>TTest</u>      |
|------------------------------|--------------|-------------|----------------------|-----------------|-------------------|
| <u>Right Pre- (°)</u>        | <u>19.35</u> | <u>9.01</u> | <u>15.07 – 23.63</u> |                 |                   |
| <u>Right Post- (°)</u>       | <u>14.06</u> | <u>4.28</u> | <u>12.03 – 16.09</u> | <u>-27.36%</u>  | <u>p&lt;0.001</u> |
| <u>Left Pre- (°)</u>         | <u>19.41</u> | <u>9.24</u> | <u>15.02 – 23.8</u>  |                 |                   |
| <u>Left Post- (°)</u>        | <u>16.24</u> | <u>5.47</u> | <u>13.64 – 18.84</u> | <u>-16.36%</u>  | <u>p=0.04</u>     |

**Figure 2 – Graph showing changes in heart rate, stroke length and swim time**

# Heart rate, stroke length and swim time across 8 x 100m swims

