

## **UNILATERAL SHOULDER PAIN IS ASSOCIATED WITH ASYMMETRIES IN TENDON THICKNESS FOLLOWING MAXIMUM EXERTION IN AN ELITE WHEELCHAIR RUGBY ATHLETE: A CASE IN POINT TOWARDS INDIVIDUALIZED FEEDBACK**

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The purpose of this study was to describe changes in tendon thickness following maximum exertion in the symptomatic and asymptomatic shoulder of a highly trained wheelchair rugby athlete with tetraplegia. Tendon thickness of the biceps and supraspinatus on both sides were measured with ultrasound before any physical tests, after a treadmill test up to maximum exertion to define aerobic capacity, and after a 30s Wingate test to define anaerobic capacity. The athlete reported moderate to severe shoulder pain, quantified with the upper extremity pain symptom questionnaire (PSQ) on the left shoulder and no pain on the right shoulder. There was a meaningful reduction (i.e.,  $> 2 \times \text{SD}$ ) in biceps and supraspinatus tendon thickness after the physical tests at the asymptomatic shoulder with no meaningful changes at the symptomatic shoulder. Reduction in tendon thickness has been related to increased alignment of the collagen fibres or creep which is a typical response to tensile loading. While the symptomatic shoulder presented a higher peak power output, there was a significantly greater drop in maximum power output during the Wingate test. Subsequently, the greater decline in power output with fatigue in the symptomatic shoulder, may have increased loads on the asymptomatic shoulder and be related to the greater reduction in tendon thickness following exertion. This demonstrates the importance to monitor and reduce asymmetries to improve performance and prevent injury and pain.

**KEYWORDS:** Shoulder Pain, Wheelchair Rugby, Fatigue, Tendons, Ultrasound

**INTRODUCTION:** Wheelchair athletes experience high stress on their upper extremities with the highest prevalence of soft tissue injuries reported at the shoulder joint (Nyland et al. 2000). Common sites of shoulder injuries are located at the supraspinatus and biceps tendon both localised within the sub-acromial space (Brose et al. 2008). Repetitive activities in specific, have a direct impact on tendon health and with a disturbed balance between loading and unloading, may cause micro-damage, chronic tendon degeneration, and ultimately tendon rupture (Magnusson et al. 2008). Pain itself may cause inhibition of the muscles that produce the painful movement which is described as a protective adaptation (Hodges et al. 2010). Furthermore, close to maximum exertion, the musculotendonous system is unstable and especially vulnerable for adaptations and/or compensations (Pol et al. 2019). Shoulder pain in highly trained wheelchair rugby athletes was previously found to be associated with asymmetries in scapular kinematics during wheelchair propulsion (Mason et al. 2018). Furthermore, fatiguing propulsion in a community-based sample of wheelchair users with a paraplegia was found to cause compensation strategies in muscular activation and contact time to the wheelchair pushrim (Bossuyt et al. 2020a) and caused changes in tendon thickness (Bossuyt et al. 2020b). However, it is unclear how tendons adjust with maximum exertion if pain is apparent. Therefore, this study aimed to identify acute changes in tendon appearance following maximum exertion during wheelchair propulsion in a highly trained elite wheelchair rugby athlete who experiences unilateral shoulder pain and to directly compare results of the asymptomatic and symptomatic shoulder.

**METHODS:** Following written informed consent, one male elite wheelchair rugby athlete (complete lesion at C5/C6, 5 yrs since injury, height: 182 cm, right handed) completed the upper extremity pain symptom questionnaire (PSQ) (van Drongelen et al. 2006) and the wheelchair user's shoulder pain index (WUSPI) (Harvey et al. 2019) to define shoulder pain. The athlete had been playing wheelchair rugby for 4 yrs and his training load included 5-8 hrs of wheelchair rugby, 2 hrs strength training and 2 hrs aerobic training per week. Body mass and the mass of the day chair and rugby chair were obtained to the nearest 0.1 kg with seated balance scales (Seca, Birmingham, UK). Two tests were collected to determine the aerobic and anaerobic capacity: a submaximal (duration ca. 30 min) and maximal test on a wheelchair treadmill (duration ca. 12 min) aimed to determine aerobic capacity and a 30s Wingate test on a dual roller wheelchair ergometer (WERG: Lode B.V., Groningen, The Netherlands) aimed to determine anaerobic capacity. These measurements are part of the annual monitoring programme of training and were all performed in the rugby chair.

Two images of the biceps and supraspinatus of the symptomatic and asymptomatic side were taken at three different time points following previously validated quantitative ultrasound protocols (QUS) (Collinger et al. 2010) with an ultrasound device (Legic E9, GE Healthcare, USA) to identify acute changes in tendon thickness following maximum exertion. Additionally, at the first measuring time point, three images of the acromio-humeral distance (AHD) were taken (Mackenzie et al. 2016). The first QUS images were taken before any tasks took place (duration ca. 30 min) (Pre-test, time = 0 min), subsequently, QUS images were taken after the tests that aimed to determine aerobic capacity (Post 1, time = 1hr45min) and after the test that aimed to determine anaerobic capacity (Post 2, time = 2hr20min).

Maximum exertion was defined as the moment when the person could not continue the task anymore and quantified with the local, central and overall rate of perceived exertion (RPE). Aerobic capacity was defined with scaled  $VO_{2peak}$  (Goosey-Tolfrey et al. 2003). Peak speed and power were defined during the Wingate test. The fatigue index was calculated as a % drop from the highest peak power output to the lowest peak power output. The shortest distance between the anterior inferior edge of the acromion and the humerus represented the AHD and the average AHD of the three repetitions, was used for further analyses. All QUS images were analysed in a randomized order and the mean tendon thickness, of the two images taken at the same time point, was used for further analysis (Collinger et al. 2010). One of the images (supraspinatus thickness Post 1) had to be excluded because of bad quality (i.e., reference lines from reference markers were not clearly visible) which resulted in one image for this time point.

**RESULTS:** The wheelchair rugby athlete reported moderate to severe left shoulder pain 3 times per week (WUSPI = 40.3). His body weight was 84.6 kg, day chair weighted 14.8 kg and his rugby chair 16.3 kg. At maximum exertion, the local, central and overall RPE were each 19/20 representing a perceived exertion of "extremely hard". Aerobic capacity represented by the scaled  $VO_{2peak}$  was 18.39 ml/kg/min (peak heart rate: 105 bpm, peak blood lactate: 2.9 mmol/l). Anaerobic capacity, represented by the peak power and fatigue index, was compared between sides. Peak power of the asymptomatic side was 22% lower (157 W) as compared to the symptomatic side (202 W) while the fatigue index was 42% higher for the symptomatic side (63.4%) as compared to the asymptomatic side (36.5%) (peak heart rate: 106 bpm, peak blood lactate: 3.8 mmol/l, peak speed: 2.41 m/s).

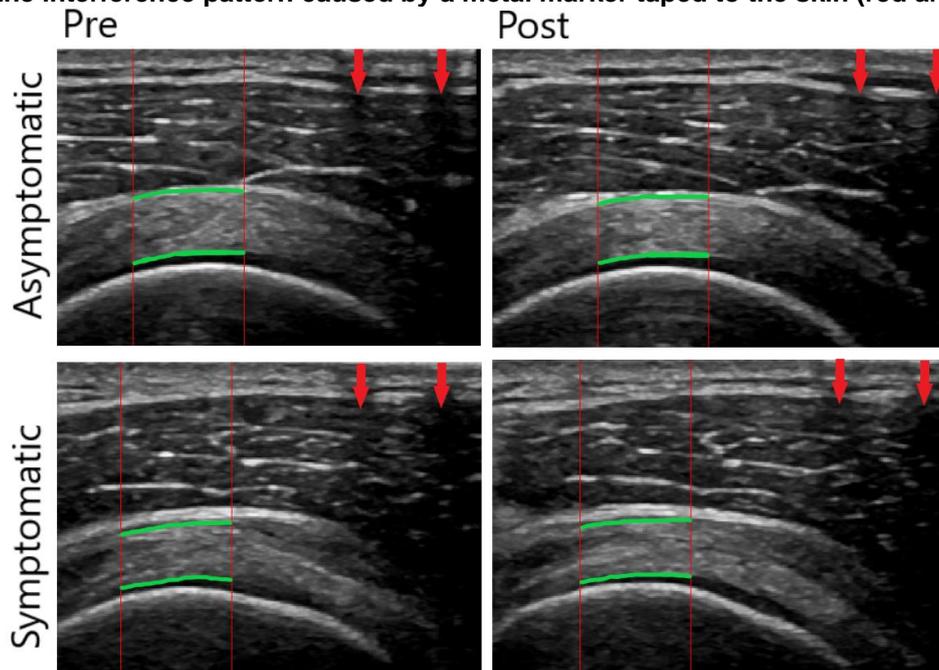
The AHD of the symptomatic shoulder ( $1.09 \pm 0.04$  cm) and the asymptomatic shoulder ( $1.06 \pm 0.01$  cm) did not present meaningful differences (i.e., greater than two times the standard deviation). For QUS, the greatest standard deviation found at any of the three time points was used as a reference of reliability and changes greater than two times this standard deviation were defined as meaningful. In the asymptomatic shoulder, there was a meaningful reduction in supraspinatus tendon thickness in both post-tests compared to the pre-test (Post 1: -0.35 mm (-8 %); Post 2: -0.37 mm (-9 %)) and in biceps tendon thickness after the last post-test (-0.25 mm (-9 %)) (Table 1, Figure1). The symptomatic shoulder did not present meaningful changes in supraspinatus or biceps tendon thickness in both post-tests.

**Table 1: Tendon thickness (mm) before and after aerobic - (Post 1) and Wingate test (Post 2).**

		Pre	Post1	Post2
Supraspinatus	Symptomatic	3.64(0.16)	3.73(0.00)	3.79(0.00)
	Asymptomatic	4.2(0.14)	<b>3.85(0.04)*</b>	<b>3.84(0.01)*</b>
Biceps	Symptomatic	2.31(0.11)	2.23(0.03)	2.15(0.07)
	Asymptomatic	2.78(0.08)	2.66(0.06)	<b>2.53(0.06)*</b>

Note 1: \* represents meaningful difference (more than two times greatest standard deviation) with pre test.

**Figure 1: Supraspinatus tendon thickness (average distance between green horizontal lines) after maximum exertion (Post). Red vertical lines present borders of region of interest (defined from the interference pattern caused by a metal marker taped to the skin (red arrows)).**



**DISCUSSION:** A highly trained wheelchair rugby athlete with unilateral shoulder pain presented asymmetries in changes in tendon thickness following maximum exertion. The observed reduction in biceps and supraspinatus tendon thickness of the asymptomatic shoulder are in line with the overall changes reported following fatiguing propulsion in a population based sample of wheelchair users (Bossuyt et al. 2020b) and could be explained by tendon creep or alignment of the collagen fibres along the applied stress (Allen 2008; Magnusson et al. 2008). With insufficient time to recover, this may cause micro-damage and tendon degeneration or pain (Allen 2008). This typical response to tensile loading was not as clearly observed in the symptomatic shoulder.

While the symptomatic shoulder was capable of producing a greater maximum power output, the higher fatigue index presented a greater drop in maximum power output during the Wingate test. These changes support the previously described theory that pain may cause inhibition of agonistic muscles and change behaviour (Hodges et al. 2010). However, this may have caused increased loads on the asymptomatic shoulder, presented in the smaller drop in maximum power output during the Wingate test, and have resulted in greater changes in tendon thickness following maximum exertion on this side.

Interestingly, there were also overall differences in supraspinatus and biceps tendon thickness between sides in the pre-test. Different changes following exertion may have caused chronic tendon adaptations that resulted in the observed differences. It is however unclear, whether these differences between sides present degeneration or strengthening of the tendon structures and warrants further investigation.

Current findings support previous recommendations to monitor and reduce asymmetries in wheelchair athletes to improve performance (Goosey-Tolfrey et al. 2018) and improve prevention of further tissue degeneration and pain. In particular, it may be important to

improve the fatigue-index of the symptomatic side. However, further research is needed to identify appropriate training strategies if pain is apparent. This case-study has multiple limitations and does not allow to make any inferences, but offers the opportunity to directly compare a symptomatic and asymptomatic shoulder within a highly trained wheelchair rugby athlete with a complete tetraplegia. Due to the high variation in functioning in wheelchair rugby athletes, an individualized approach is needed and provides valuable feedback.

**CONCLUSION:** Asymmetrical changes following maximum exertion in shoulder tendons of a highly trained wheelchair rugby athlete with unilateral shoulder pain call for the need to monitor asymmetries and define appropriate training strategies as this may reduce the risk for further injury and pain. Further studies with larger samples should support these results.

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