Scapular Dyskinesis is Detrimental to Shoulder Tendon Properties and Joint Mechanics in a Rat Model

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Introduction

Shoulder injuries including rotator cuff and biceps tendinitis are common clinical conditions and are a significant source of pain and dysfunction.These tendinopathic conditions are frequently seen in the presence of abnormal scapulothoracic joint kinematics.[1,2,3] Specifically, altered scapular motion and position (termed scapular dyskinesis) has been observed in 68-100% of patients with shoulder injuries,including shoulder impingement.[1] However, the cause and effect relationships these mechanical alterations have on the shoulder joint are unknown and therefore, it is unclear whether abnormal scapulothoracic joint kinematics contributes to (or instead compensates for) these disorders. Therefore, the objectives of this study were to examine the effect of scapular dyskinesis on the initiation and progression of pathological changes in the rotator cuff and biceps tendon and to define the mechanical processes that lead to these changes.We hypothesized that scapular dyskinesis will: H1) alter joint function and passive joint mechanics and H2) permanently diminish supraspinatus and biceps tendon mechanical properties.

Methods

A rat model of scapular dyskinesis was developed and used.This condition was created by denervating the trapezius and serratus anterior muscles through surgical transection of the spinal accessory and long thoracic nerve, respectively. 60 adult male Sprague-Dawley rats (400-450 grams) were randomized into two groups: nerve transection (SD) or sham nerve transection (Control).All rats were sacrificed at 4 or 8 weeks after transection and frozen at −20°C. Forelimb gait and ground reaction forces were quantified over time in all animals using an instrumented walkway.[4] Data was collected one day prior to nerve transection to obtain baseline values and then collected at days 3, 7, 14, 28, 42, and 56 post-surgery. Ground reaction force data, including medial/lateral (ML), propulsion, braking, and vertical forces were collected for each walk. Parameters were normalized to the animal's body weight.

Passive shoulder joint range of motion and stiffness were measured over time using a custom instrument and methodology.[5] Parameters were normalized to baseline. Measurements were taken one day prior to nerve transection, and at days 14, 28, and 56 days post-surgery. Briefly, under anesthesia, the arm was secured into the rotating clamp at 90° of elbow flexion and 90° of glenohumeral forward flexion. The scapula was manually stabilized in order to isolate glenohumeral motion.The arm was then rotated through the full range of internal and external rotation three times. Range of motion was determined using data from all three cycles. A bilinear fit was applied to calculate joint stiffness in the toe and linear regions for both internal and external rotation.

At the time of testing, the animals were thawed and the scapula and humerus were dissected with the biceps and supraspinatus tendons intact. Stain lines, for local optical strain measurement, were placed on the supraspinatus and biceps tendons. Cross sectional area was measured using a custom laser device. To determine biomechanical properties, tensile testing was performed as follows: preload to 0.08 N, preconditioning (10 cycles of 0.1-0.5 N at a rate of 1% strain/s), stress relaxation to 4% (biceps) or 5% (supraspinatus) strain at a rate of 5% strain/s for 600s, and ramp to failure at 0.3% strain/s. Stress was calculated as force divided by initial area, and 2D Lagrangian optical strain was determined from stain line displacements measured using custom texture tracking software.

For the ambulatory assessment, multiple imputations were conducted using the Markov chain Monte Carlo method for missing data points. For both ambulatory assessment and passive joint mechanics, significance was assessed using a 2-way ANOVA with repeated measures on time with follow-up t-tests between groups at each time point. Tissue mechanics between groups were assessed using a t-test. Significance was set at $p<0.05$.

Results

Gross observational examination demonstrated clear alterations in scapular movements, consistent with scapular "winging."

Poster No: 1007
2014 Annual Meeting
Orthopaedic Research Society
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Additionally, joint function was significantly altered in the SD group (Figure 1). Specifically, ML force was significantly altered at early time points (5 and 7 days post-transection), with the SD group demonstrating less of a laterally directed force than control (Figure 1A). No differences between groups were observed in braking force (Figure 1B). However, propulsion force was significantly increased and vertical force was significantly decreased at all time-points compared to control (Figure 1C, 1D). Passive joint mechanics were also significantly altered (Table 1). Internal range of motion was significantly greater in the SD group compared to control at all post-surgical time-points. No other differences were observed, except for an increase in toe region stiffness in external rotation in the SD group compared to control at 4 weeks post-surgery.

In the presence of scapular dyskinesis, viscoelastic parameters were significantly altered (Figure 2). Tendon percent relaxation was significantly greater in the SD group compared to control at 8 weeks, for both the biceps and supraspinatus tendons, indicative of inferior tissue properties (Figure 2A). No differences were observed in any tendon for cross-sectional area (data not shown) or insertion elastic modulus (Figure 2B). However, tendon mid-substance elastic parameters were significantly altered. Tendon mid-substance elastic modulus was significantly decreased in the SD group compared to control, at both 4 and 8 weeks for the supraspinatus and at 8 weeks for the biceps, also indicative of inferior tissue properties (Figure 2C).

Discussion

While the prevalence of shoulder impingement and its association with scapulothoracic kinematic abnormalities is well-documented,[3] the cause and effect relationships behind them are not well-established, making optimal clinical management difficult. In this animal model, we were able to prescribe scapular dyskinesis and rigorously evaluate the effect in a controlled manner. Results of this study demonstrate that scapular dyskinesis alters joint loading and leads to compromised tendon mechanical properties. The changes observed in the presence of scapular dyskinesis may be a result of reduced subacromial space, leading to tendon mechanical abrasion and wear. Tendon changes were localized to the mid-substance region, likely due to its anatomic location underneath the acromial arch during forward flexion, resulting in tendon impingement. Additionally, the increased internal range of motion observed may be due to a loss of dynamic restraint. The unstable scapula may not allow the rotator cuff to effectively compress the humeral head into the glenoid fossa thereby requiring the joint to rely on static restraints, such as the joint capsule, for stability and placing it at increased risk for injury. This is the first study to directly identify scapular dyskinesis as a mechanical mechanism for the development of pathological changes in the rotator cuff and biceps tendon. Future studies will examine the effect of scapular dyskinesis in the presence of overuse and following supraspinatus repair in order to help define the in vivo mechanical processes

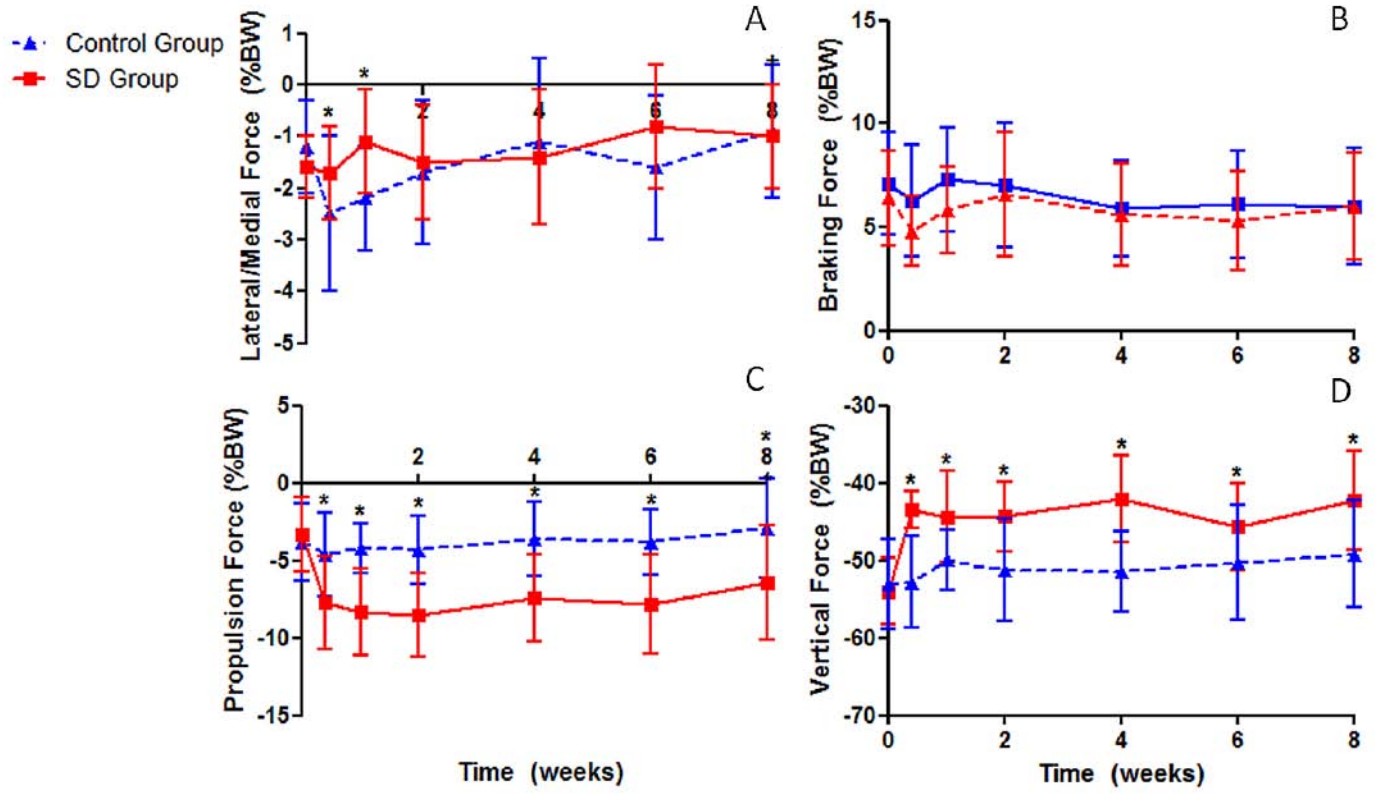

Figure 1. (A) The SD group had a significantly decreased lateral force at 5 and 7 days post-surgery compared to the control group. (B) No differences were observed in braking force between groups. (C) The SD group had a significantly increased propulsion force compared to controls at all time-points. (D) The SD group had a significantly decreased vertical force compared to control at all time-points. Data are shown as mean and standard deviation (SD) (Significance *$p<0.05$).

Table 1. Results for passive joint mechanics demonstrated increased internal range of motion (ROM) in the SD group compared to control at all time-points. Data are shown normalized by baseline values and as mean and SD (Significance *P<0.05).

Direction	Measurement	Time (wks)	Control	SD
Internal	ROM	2	0.93±0.21	1.09±0.19*
		4	0.89±0.07	1.01±0.17*
		8	0.87±0.12	1.02±0.16*
	Toe Stiffness	2	3.62±2.99	1.21±1.43
		4	3.87±3.83	1.66±1.75
		8	2.99±3.64	1.18±1.16
	Linear Stiffness	2	1.50±0.36	1.35±0.23
		4	1.47±0.31	1.30±0.28
		8	1.65±0.26	1.51±0.22
External	ROM	2	1.20±0.13	1.15±0.07
		4	1.03±0.10	0.98±0.09
		8	1.26±0.16	1.18±0.11
	Toe Stiffness	2	0.96±0.49	0.75±0.49
		4	0.82±0.29	1.50±0.86*
		8	0.87±0.42	1.03±0.58
	Linear Stiffness	2	1.10±0.28	1.10±0.32
		4	1.16±0.34	0.94±0.21
		8	1.25±0.43	1.24±0.29

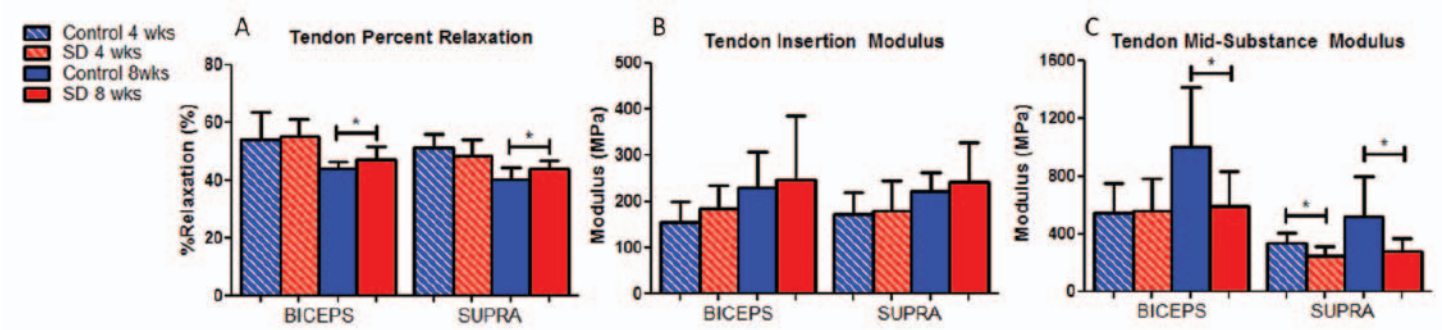

Figure 2. (A) The biceps and supraspinatus tendons demonstrated significantly increased percent relaxation at 8 weeks post-surgery in the SD group compared to control. (B) No differences were observed at the insertion site of any tendon at any time-point. (C) The biceps and supraspinatus tendons demonstrated significantly decreased tendon modulus at 8 weeks and both 4 and 8 weeks, respectively, in the SD group compared to control. Data are shown as mean and SD (Significance *p<0.05).

which lead to tendon degeneration and compromise healing potential following repair.

Significance

This study presents a new model of scapular dyskinesis and identifies scapular dyskinesis as a mechanical mechanism for shoulder tendon injury.

Acknowledgments

This study was funded by the NIH/NIAMS.

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