Suprascapular nerve anatomy during shoulder motion: a cadaveric proof of concept study with implications for neurogenic shoulder pain

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\textbf{Background:} The suprascapular nerve (SSN) carries sensory fibers which may contribute to shoulder pain. Prior anatomic study demonstrated that alteration in SSN course with simulated rotator cuff tendon (RCT) tears cause tethering and potential traction injury to the nerve at the suprascapular notch. Because the SSN has been implicated as a major source of pain with RCT tearing, it is critical to understand nerve anatomy during shoulder motion. We hypothesized that we could evaluate the SSN course with a novel technique to evaluate effects of simulated RCT tears, repair, and/or release of the nerve.

\textbf{Methods:} The course of the SSN was tracked with a dual fluoroscopic imaging system in a cadaveric model with simulated rotator cuff muscle forces during dynamic shoulder motion.

\textbf{Results:} After a simulated full-thickness supraspinatus/infraspinatus tendon tear, the SSN translated medially 3.5 mm at the spinoglenoid notch compared to the anatomic SSN course. Anatomic footprint repair of these tendons restored the SSN course to normal. Open release of the transverse scapular ligament caused the SSN to move 2.5 mm superior-posterior out of the suprascapular notch.

\textbf{Conclusion:} This pilot study demonstrated that the dynamic SSN course can be evaluated and may be altered by a RCT tear. Preliminary results suggest release of the transverse scapular ligament allowed the SSN to move upward out of the notch. This provides a biomechanical proof of concept that SSN traction neuropathy may occur with RCT tears and that release of the transverse scapular ligament may alleviate this by altering the course of the nerve.

\textbf{Level of evidence:} Basic Science Study, Anatomic Study, Cadaver Model.

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Recent clinical research has demonstrated that suprascapular nerve (SSN) dysfunction may occur in patients with rotator cuff tendon tears.\textsuperscript{4,6,18} Moreover, experimental anatomic dissection studies have implicated tethering of the SSN at the suprascapular notch as a potential pain generator with retraction of the supraspinatus muscle.\textsuperscript{2,11}
Hence, the inherent anatomy of the SSN makes it susceptible to compression and/or traction injury in association with rotator cuff tendon tears. Additionally, a further risk factor may be the variable anatomy of the suprascapular notch; from a deep, narrow notch with an ossified transverse scapular ligament, to no discrete notch at all.\textsuperscript{8,15}

The SSN originates from the C5 and C6 nerve roots (with contribution from C4 in some individuals) and courses through the suprascapular notch and into the supraspinatus fossa, underneath the supraspinatus muscle, and abruptly changes course around the spine of the scapula at the spinoglenoid notch as it forms 2-4 motor branches to the infraspinatus.\textsuperscript{21} As such, if the supraspinatus and infraspinatus tendons retract medially with a tendon tear, then the SSN will be vulnerable to a traction injury.\textsuperscript{6} Albritton et al verified this theory using a simulated rotator cuff tendon tear model. They found that with a simulated rotator cuff tendon tear, the SSN was pulled medially and tethered at the suprascapular notch, suggesting that the SSN may be at risk of traction along its course on the scapula.\textsuperscript{2}

Suprascapular neuropathy is usually diagnosed by both physical examination and electrodiagnostic study of the shoulder.\textsuperscript{3} Literature regarding this entity has increased significantly in the last decade. The increased incidence of suprascapular neuropathy is likely based on awareness amongst clinicians of the common causes; paralabral cysts adjacent to the spinoglenoid notch,\textsuperscript{1,9,24,29} mass effect,\textsuperscript{14,30} traction during high level overhead sport,\textsuperscript{10,17,22} scapular fracture,\textsuperscript{5} iatrogenic injury,\textsuperscript{27} and traction from a retracted rotator cuff tendon with resulting fatty infiltration of the posterosuperior rotator cuff.\textsuperscript{6,18}

Treatment for suprascapular neuropathy has traditionally involved open release, and, more recently, arthroscopic release of the transverse scapular ligament. This approach is based on the assumption that the nerve course is altered in the diseased state, and that traction is relieved by surgical release. A 2007 cadaveric study by Plancher et al demonstrated that arthroscopic release of the spinoglenoid ligament directly relieved pressure on the distal suprascapular nerve motor branch.\textsuperscript{21} Despite the recent enthusiasm for suprascapular nerve pathology, significant controversy remains regarding the precise mechanism and location of nerve injury caused by massive rotator cuff tendon tears, and the mechanism by which transverse scapular ligament release appears to relieve this inciting trauma.

The purposes of our proof of concept pilot study are: (1) to evaluate a validated 3-dimensional (3D) fluoroscopic imaging technique\textsuperscript{19} to empirically model the anatomic course of the SSN during shoulder motion; and (2) to evaluate the SSN course with and without a simulated rotator cuff tendon tear and its repair in a cadaver model and/or with the effect of release of the transverse scapular ligament. We hypothesized that this novel model could evaluate the SSN to determine if it would be subject to traction at the suprascapular notch, and that the change in orientation and position of the nerve with a simulated rotator cuff tear would increase the likelihood of traction on the nerve at this location as well as at the spinoglenoid notch. Our secondary hypothesis was that repair of the tendons would restore anatomy of the nerve to normal, and that the release of the transverse scapular ligament would result in elimination of traction of the nerve at the suprascapular notch by altering its course.

**Methods and materials**

**Specimen preparation**

One male fresh-frozen cadaver torso (age 30) with upper extremities intact and cephalus removed cranial to the C5 vertebrae was acquired. The specimen was stored at minus 4°F until thawed at room temperature for data acquisition. To provide fixed radio-opaque markers to assist bony tracking, titanium spheres 1/8” in diameter were implanted within the cortical shell of the scapula and humerus of the right shoulder. All dissections and operations were performed by a fellowship-trained orthopaedic shoulder surgeon (A.S.). For the humerus, a limited deltopectoral approach was utilized to directly visualize the lateral aspect of the humeral head. Five spheres were uniformly implanted into the lateral cortical bone of the humeral head and away from the articular surface. For the scapula, a posterior approach was utilized along the scapular spine. One sphere was implanted into the lateral acromion, 3 spheres along the scapular spine, and 1 sphere near the spinoglenoid notch. The cadaver was noted to have a typical U-shaped suprascapular notch with a nonossified ligament.

To mark the SSN and its terminal branches, 7 tungsten wires (OD = 0.025”) and 1 tungsten wire (OD = 0.045”), all 0.160” in length, were inserted coaxially into the SSN and along its course in select accessible locations of interest. Utilizing the existing posterior approach along the scapular spine, the SSN was directly visualized at select locations of interest from proximal to the suprascapular notch to the terminal motor branches. An attempt was made to minimize disruption of native adhesions to surrounding fat, muscle, and bone to minimize alteration of nerve kinematics. To this end, less than 5 mm of nerve had to be visualized for each insertion, as an angiocatheder was inserted coaxially into the nerve, and the tungsten wires inserted centrally. Small barbs on each end of the wires served to prevent pistoning or toggle from their initial position. A running locking stitch was used to close the deltopectoral and posterior approaches. Wire locations relative to the SSN anatomy are shown in Figure 1.

**Bone model reconstruction**

The specimen underwent high resolution computer tomography in a LightSpeed Pro 16 (GE Healthcare, Little Chalfont, Bucks, UK) with axial slices spaced 0.625 mm. The image resolution was 512 × 512 pixels with a field of view of 280 × 420 mm. DICOM images of the scan were transferred to a personal computer and
automatically segmented by a custom MATLAB (The Mathworks Inc, Natick, MA, USA) script based on the intensity gradient of each pixel. The segmented contours were transferred into the Rhinoceros 3D v4.0 program (Robert McNeel & Associates, Seattle, WA, USA) and B-Splines were meshed to create 3D surface models of the scapula and humerus.

Testing protocol

The specimen was rigidly secured to a custom apparatus via pedicle screws through the spine. The right shoulder was placed with the glenohumeral joint centered in the imaging volume created by the dual fluoroscopic imaging system (DFIS) (Fig. 2). This configuration allowed for unconstrained scapulothoracic motion.

Native rotator cuff muscle forces were modeled after a 2003 Mayo Clinic cadaveric study of rotator cuff tendon tears by Mura et al. Separate #5 Fiberwire sutures (Fiberwire; Arthrex Inc., Naples, FL, USA) were sewn into the native musculotendinous junction of each rotator cuff muscle, with 2 sutures in the subscapularis due to its large superior to inferior breath. Heavy gauge nylon rope was tied to each individual Fiberwire and passed through a custom jig to mimic the force vectors of in-vivo muscles according to their cross sectional area and direction of pull. The simulated muscle forces were: subscapularis 30N, supraspinatus 16N, infraspinatus 20N, and teres minor 12N. The shoulder was grossly well balanced after tensioning.

Simultaneous fluoroscopic images of the right shoulder were acquired at 30 frames per second with an 8 ms pulse width while the arm was manually manipulated in cycles of 0°-130°-0° abduction/adduction in the scapular plane at a rotation rate of approximately 45°/s. Five configurations were simulated in the following sequence: (case #1) intact rotator cuff; (case #2) an acute simulated full-thickness infraspinatus/supraspinatus tendon tear; (case #3) a transosseous double row repair of the tendons; (case #4) an acute simulated full-thickness infraspinatus/supraspinatus tendon tear with transection of the transverse scapular ligament; and (case #5) a transosseous double row repair of the tendons with transection of the transverse scapular ligament.

For case #2, the simulated rotator cuff tendon tear was effectuated with a #11 blade at the tendinous insertion of the superior cuff onto the greater tuberosity via the deltopectoral interval. The native footprint of both the supraspinatus and infraspinatus were both sharply incised. Open rotator cuff tendon repair was performed through the deltopectoral interval with internal rotation of the arm. The goal of repair was to accurately restore the native resting length of the tendon using soft tissue landmarks made during creation of the simulated tear. Three 1-cm wide mattress sutures with #5 Fiberwire were placed at the medial edge of the tuberosity and tied on the bursal aspect of the cuff. Two additional simple lateral row sutures were placed on the lateral aspect of the greater tuberosity to reduce the supraspinatus and infraspinatus tendons to their anatomic footprints and to minimize abrasion throughout the range of motion.
**Dual fluoroscopic imaging system (DFIS): 3D kinematics**

The system consists of two fluoroscopes (12” BV Pulsera; Phillips Medical, USA) arranged with the image intensifiers at approximately 120° to one another. The accuracy of the tracking system was ±0.3 mm in translation and ±0.5° in rotation. The nominal fluoroscope settings were 5.0 mA and 55 kV. The acquired fluoroscopic images were transferred to a personal computer and corrected for geometric distortion. A modified Gronenschild, global surface mapping technique was utilized. Within solid modeling software (Rhinoceros 3D v4.0; Robert McNeel & Associates, Seattle, WA, USA) a virtual DFIS was created to reproduce the physical geometry of the DFIS. Corrected fluoroscopic image pairs and 3D surface models of the scapula and humerus were imported into the virtual DFIS. The position of the scapula and humerus were adjusted in 6-DOF so that the 3D surface model spheres aligned with the intersection of the vectors projected from the spheres on the fluoroscopic images. As a result, the 3D positions of the scapula and humerus were recreated (Fig. 3). Bone kinematics were reproduced in approximately 0.33 second intervals (15 image pairs/poses per configuration) for the total abduction/adduction cycle for each configuration tested.

**DFIS: dynamic SSN tracking**

The SSN was tracked by reproducing the position of the implanted tungsten wires in the virtual DFIS. Each tungsten wire’s position was reproduced by constructing vectors from the virtual fluoroscopic source to the wire ends in the fluoroscopic images on the virtual fluoroscopic image planes. The intersection of corresponding vectors from the two image planes identified the 3D position of the wire ends in the virtual DFIS (Fig. 4). Corresponding wire ends were connected with a line to reproduce the position of the implanted tungsten wire in the virtual DFIS. In this manner, all wire ends and wire positions were found and manually checked by 2 investigators for each fluoroscopic image pair (15 image pairs/poses per configuration) for which bone kinematics were reproduced.

For each configuration tested (5 total), the positions of the tungsten wires relative to the scapula at each pose (15 poses per configuration) were used to find the average position of each wire over the abduction/adduction cycle. A 3D curve was fit through the average tungsten wire positions in the virtual DFIS to recreate the average course of the SSN during the simulated 0°-130°-0° abduction/adduction cycle in the,
scapular plane. The SSN courses for the 5 configurations tested were overlaid on the scapula for qualitative and quantitative assessment. Each SSN course was visually represented by a color coded 1.5-mm diameter tube in the final 3D model (Figs. 5-8).

Proof of concept

This pilot study was designed to determine whether we could biomechanically evaluate the SSN course with and without a simulated rotator cuff tendon tear and its repair in a cadaveric model using a highly accurate and validated DFIS methodology. The accuracy of the position of each tungsten wire was measured to \( \pm 0.3 \) mm, whereas the measured range of SSN translation between configurations was 2.5-3.5 mm. Accordingly, the magnitude of measured nerve translations compared to the accuracy of the tracking system supports the analysis of 1 cadaver shoulder to (1) show the technical feasibility of nerve tracking with DFIS during dynamic motion in a cadaveric model, and (2) demonstrate the concept that SSN anatomy may be empirically altered by a rotator cuff tendon tear and/or with release of the transverse scapular ligament.

Results

Case #1 - intact rotator cuff

An illustration of the anatomic SSN course is shown in Figure 5.

Case #2 - acute simulated full-thickness infraspinatus/supraspinatus tendon tear

The SSN translated medially 3.5 mm at the spinoglenoid notch compared to the anatomic SSN course (case #1). No change in the nerve course was observed at the suprascapular notch. An illustration of these nerve motions is shown in Figure 6.

Case #3 - transosseous double row repair of the tendons

The SSN translated laterally 3.5 mm at the spinoglenoid notch compared to the simulated full-thickness tendon tear (case #2), effectively restoring the anatomic nerve course at the spinoglenoid notch (Fig. 5, representing both case #1 and case #3). No change in the nerve course was observed at the suprascapular notch.

Case #4 - acute simulated full-thickness infraspinatus/supraspinatus tendon tear with transection of the transverse scapular ligament

The SSN translated medially 3.5 mm at the spinoglenoid notch and 2.5 mm superior-posterior at the suprascapular notch compared to the anatomic SSN course (case #1). An illustration of these nerve motions is shown in Figure 7.

Case #5 - transosseous double row repair of the tendons with transection of the transverse scapular ligament

The SSN translated 2.5 mm superior-posterior at the suprascapular notch, while no change in the nerve course was observed at the spinoglenoid notch compared to the anatomic SSN course (case #1). Effectively, the rotator cuff
tendon repair restored the anatomic nerve course at the spinoglenoid notch, while transection of the transverse scapular ligament allowed a more direct or shorter nerve path at the suprascapular notch (Fig. 8).

Discussion

This pilot study was designed as a proof of concept to evaluate a system designed to determine the SSN anatomy during shoulder motion with simulated rotator cuff muscle forces and with simulated rotator cuff tendon tears and/or with transection of the transverse scapular ligament. In a cadaver, we established a 3D model for the dynamic SSN course in the abducted/adducted shoulder. Empirically, we were able to detect medial traction about the spinoglenoid notch following the simulated massive rotator cuff tendon tear; and this effect was reversed by anatomic transosseous footprint repair. Furthermore, release of the transverse scapular ligament allowed the SSN to move superior-posterior out of the suprascapular notch, with a more direct or shorter nerve path to the suprascapular notch (Fig. 8).

The first cadaveric study to examine rotator cuff tendon retraction as a pathologic entity for the SSN was conducted in 2003 by Albritton et al. In that static anatomic study, they demonstrated that increasing retraction of the supraspinatus led to a reduction in the angle between the SSN and the first motor branch, thereby increasing tension on the nerve. That study established that anatomic change in nerve tracking was a potential causal link between a retracted rotator cuff tendon tear and SSN injury.

A number of proposed mechanisms for injury to the SSN exist in the literature. Repetitive traction in overhead athletes, microemboli from the axillary artery, a stenotic suprascapular notch, iatrogenic injury, or direct pressure from a cyst or bone tumor have all been proposed. The common pathway is generally direct pressure or stretch on the SSN. This pilot study provides proof for the concept that a retracted posterosuperior rotator cuff tendon tear puts the nerve in tension at the notches that constrain the SSN.

The link between the rotator cuff pathology and SSN dysfunction began with anatomic studies examining the surgical advancement of the superior rotator cuff during open repair. A 1992 study by Warner et al found that greater than 3 cm of rotator cuff advancement places tension on the medial motor branches. These findings were corroborated by a later anatomic study by Grenier et al that demonstrated measurable stretch on the motor branch with only 1 cm of tendon advancement.

Vad et al used electrodiagnostic testing to examine the prevalence of peripheral neurologic injury in 25 patients with full-thickness rotator cuff tendon tears and gross muscle atrophy. They found a 28% prevalence of nerve injury, most commonly of the axillary or suprascapular nerves. In a 2006 study, Mallon et al reported on 8 patients with massive rotator cuff tendon tears retracted at least 5 cm in greatest dimension. All eight had SSN denervation on electromyography (EMG). Four patients consented to partial repair with good functional results. Two of the repaired patients consented to follow up EMG, which demonstrated significant reinervation potentials.

In a recent study by Coustoros et al, they demonstrated that partial repair of the posterior rotator cuff tendon in patients with massive rotator cuff tendon tears leads to reversal of SSN denervation on EMG. At 6 months post repair, all 6 patients had evidence of recovery by EMG potentials. It was presumed this was the result of relieving traction on the nerve by restoring its normal course through the spinoglenoid notch when the posterior rotator cuff tendon was repaired. The results of our pilot study empirically support their presumption that the SSN moves lateral, away from the spinoglenoid notch, restoring SSN anatomy with repair of retracted posterosuperior rotator cuff tendons.

In 2007, Lafosse et al published an arthroscopic technique for release of the SSN at the transverse scapular notch. They demonstrated the effectiveness of this method in both alleviating pain and reversing EMG findings in patients with electrodiagnostically documented SSN neuropathy. Both Lafosse et al and Romeo et al have proposed that release of the SSN at the time of rotator cuff tendon repair is appropriate in select patients to relieve traction on the SSN, which is a direct result from tendon retraction.

Our empirical results provide a novel proof of concept model to further evaluate the previous clinical observations of clinical improvement and EMG reversal of SSN neuropathy, which is that repair of rotator cuff tendon
tears and/or release of the SSN at the transverse scapular notch reduces traction on the nerve. This anatomic pilot study is the first to devise a methodology to explore both the effect of rotator cuff tendon repair and/or transverse scapular ligament release on the SSN. As such, this study evaluated the SSN anatomy in a physiologically relevant model with applied rotator cuff muscle forces during dynamic shoulder abduction/adduction using a validated DFIS 3D modeling technique. The anatomic course of the SSN was modeled with and without a simulated rotator cuff tendon tear and repair and/or with transection of the transverse scapular ligament.

There are, however, several limitations of our study. First, as with all cadaver studies, an assumption must be made regarding physiologic muscle forces about the shoulder. We were careful to weight each muscle individually, dynamically, and in proportion to its cross-sectional area according to precedent set in prior study. We radiographically confirmed that the humeral head remained concentric on the glenoid during shoulder motion. Second, only one cadaver shoulder was used to quantify the motion of the SSN in a simulated massive rotator cuff tendon tear, tendon repair, and release of the transverse scapular ligament. Ultimately, our study goal to biomechanically demonstrate the concept that SSN anatomy can be evaluated with this novel DFIS technique to determine whether the course of the SSN under varying conditions such as a rotator cuff tendon tear and/or release of the transverse scapular ligament was accomplished. This pilot study provides evidence for future study regarding the influence of rotator cuff tendon tears and release of the transverse scapular ligament on SSN course alteration. The concepts of this pilot study, however, may have limited generalizability given the young age of the cadaver, lack of cuff pathology, and most common variant of SSN and suprascapular notch anatomy.

Conclusion
This pilot study demonstrated that a novel DFIS technique can evaluate dynamic SSN anatomy and that the anatomical course of the SSN may be altered by a rotator cuff tendon tear. Moreover, we believe that this model provides proof for the concept that SSN traction injury may occur in the clinical setting of a massive rotator cuff tendon tear, and has the potential to be reversed by anatomic repair and/or transverse scapular ligament release. Further study is warranted.

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References


