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Posterior Shoulder Pain and Arthroscopic Decompression of the Suprascapular Nerve at the Transverse Scapular Ligament

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The etiology of posterior shoulder pain can be elusive for the treating physician. Compression of the suprascapular nerve at either the transverse scapular ligament or the spinoglenoid ligament can lead to resultant posterior shoulder pain, muscle weakness, and permanent muscle atrophy. Compression at the transverse scapular ligament, the more common finding than realized in the past in patients, can often result in weakness and atrophy of both the supraspinatus and infraspinatus muscles as compression occurs at the suprascapular notch prior to the nerve giving off motor branches to each of these muscles. Patients with this pathology often have a long-standing disease course of missed diagnoses and even wrong surgical procedures. This paper will discuss the anatomy, pathophysiology, and presentation of symptoms in patients. A thorough discussion of the physical examination as well as appropriate adjunct diagnostic procedures will follow to aid the clinician in making a correct diagnosis with detailed recommendations for appropriate arthroscopic decompression allowing the athlete and patient to return to activities of daily living with a short recuperation period.
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An article in the New England Journal of Medicine in 1959 opened the gates for discussion about suprascapular nerve entrapment in the clinical setting.[1] Various authors including ourselves have described the transverse scapular ligament and spinoglenoid notch as the 2 potential sites of entrapment[2-5] (Fig. 1). This injury can lead to a multitude of symptoms including pain and weakness with an inability to hold even a 5-kg weight in the horizontal plane. The pain and disability associated with this condition can be unduly prolonged, but it is easily cured now with advanced arthroscopic techniques. Although this entity represents a small percentage of the average shoulder surgeon's practice, recent advancements as well as diagnostic testing and the aforementioned treatment options have brought this diagnosis of exclusion to the forefront and minds of many surgeons.

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Although always thought of as a disease for only overhead athletes, compression of the suprascapular nerve is now recognized to be associated with patients with massive rotator cuff disease especially when fat atrophy is noted, those with a labral tears with or without a paralabral cyst, those with large space-occupying lesions in both notches, and those whose magnetic resonance imaging (MRI) may show evidence of the enlarged nerve implying compression or even detection of weakness of external rotation of the shoulder on physical examination. Confirmation of suprascapular disease remains elusive at times because the etiology (e.g., direct trauma or indirect trauma [eg, traction], repetitive overuse, rotator cuff disease, and anatomical variations) as well as the indications for decompression of this nerve remain fraught with its advocates and critics.

Anatomy of the Suprascapular Nerve

The suprascapular nerve has been classically thought to arise from the upper trunk of the brachial plexus (C5-C6) at Erb's Point. However, there are reports of the nerve receiving

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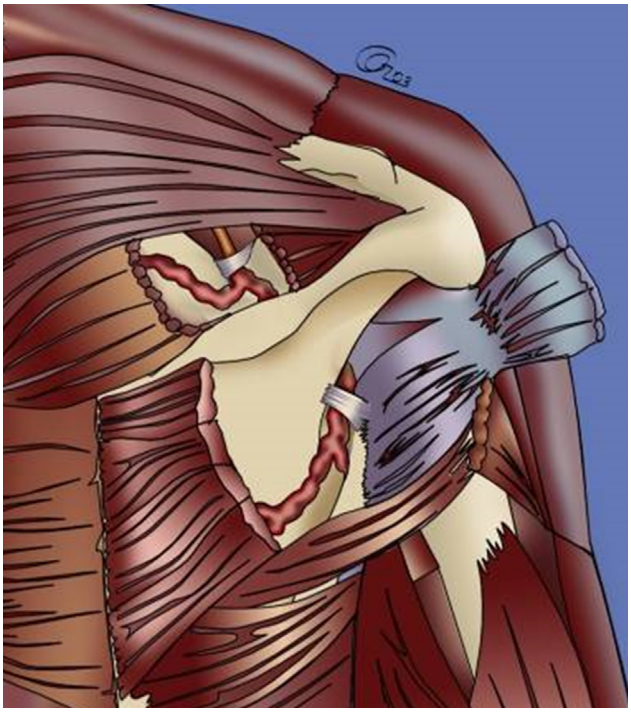

Figure 1 Right shoulder, posterior view, artwork demonstrating the 2 possible sites of compression of the suprascapular nerve. (Copyright: Plancher K.)

contributions from C4 in approximately 25% of individuals[6,7] (Fig. 2). The nerve exits the upper trunk approximately 3 cm above the clavicle to run laterally and parallel to the muscle belly of the omohyoid muscle and deep to the anterior border of the trapezius along the posterior cervical triangle (Fig. 3). As it passes through the posterior triangle, it travels with the suprascapular artery and vein. The nerve then travels along the posterior border of the clavicle to reach the superior border of the scapula. The nerve must diverge now from the artery to take a posterior approach diving into the suprascapular notch (Fig. 4). This nerve is approximately 3 cm away from the supraglenoid tubercle.[8] The artery instead takes an anterior position and enters the suprascapular notch over the ligament. Variations, though infrequent, do occur with the artery staying with the nerve as it passes posterior to the ligament.[9] The ligament that divides the nerve and artery in most cases is termed the transverse scapular ligament. The nerve now in the supraspinatus fossa gives off 2 motor branches to the supraspinatus muscle belly. The nerve also gives off sensory and sympathetic branches to two-thirds of the glenohumeral joint, coracoclavicular ligament, coracohumeral ligament, subacromial bursa, and the posterior capsule of the acromioclavicular (AC) joint.[10-12]

The roof of the notch is formed by the transverse scapular ligament. Hypertrophy of the transverse scapular ligament is possible and can cause stenosis of the notch. The geometry of the notch may also vary leading to compression of the nerve. Classification of these variations and the 6 types of notches has been reported[6] (Fig. 5). This narrowed notch may compress the nerve leading to a neuropraxia. The transverse scapular ligament extends from the base of the coracoid to the superior border of the scapula and can ossify in 25% of clinical cases.[13]

The nerve travels along the supraspinatus fossa heading laterally and coming within 2.0 cm of the posterior glenoid rim at the level of the spine of the scapula.[14] The suprascapular nerve travels laterally around the scapular spine to descend into the infraspinatus fossa only to pass under the spinoglenoid ligament (inferior transverse scapular ligament) and give off 2-4 branches to this muscle belly (Fig. 4).

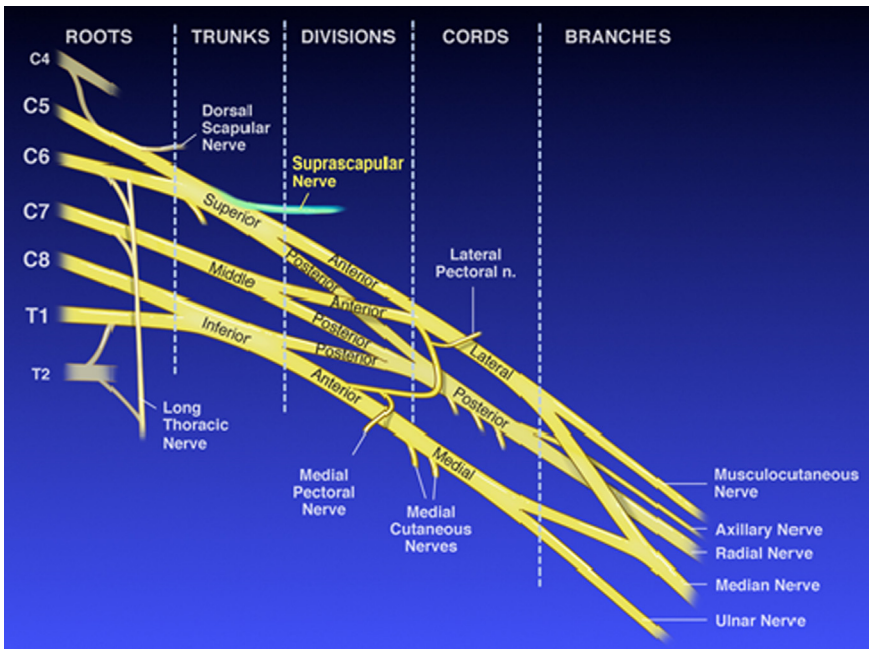

Figure 2 The suprascapular nerve arising from the upper trunk of the brachial plexus, which receives contributions from C4 in 25% of individuals. (Copyright: Plancher K.)

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