13 Suprascapular Nerve Release: Fact or Fiction

Bartlomiej Kordasiewicz, Michał Janyst, and Andrzej Boszczyk

13.1 Introduction

The suprascapular nerve (SSN) pathology is a uncommon clinical diagnosis, however its incidence alone or in association with some other concomitant pathologies has been recently reported more regularly [1–3]. Anatomy of SSN makes it susceptible to compression or traction injuries [4–6]. In recent years it was reported as an important cause of shoulder pain in overhead athletes, often as a gradually progressing "cummulative neuropraxia" [7]. Another investigated subject in recent studies, as well as in this chapter, remains a correlation between SSN pathology and massive rotator cuff tears (RCT) [8, 9].

13.2 Anatomy

SSN is formed by the ventral rami of C5, C6 and sometimes C4 roots. The nerve courses laterally through the posterior cervical triangle deep to the trapezius and omohyoideus muscles, then passing though the foramen formed by the suprascapular notch and its roof—transverse scapular ligament (TSL). This bone and ligamentous structures can have many anatomical variants creating risk for potential nerve entrapment [6]. The SSN passes under TSL and major supraspinatus nerve branch arises usually distal, however possibly also proximal to the ligament. In this area some motor sensory branches arise to supply the supraspinatus muscle, glenohumeral and acromioclavicular joint. Also the small cutaneous branch arising in TSL area supplies posterior—infraspinatus and scapular spine region of the shoulder [10]. The nerve continuous through the spinoglenoid notch under spinoglenoid ligament winding around the lateral border of the scapular spine to enter the infraspinatous fossa. The spinoglenoid ligament is quadrangle in shape and extends from the posterior glenoid neck and glenohumeral capsule to insert into the scapular spine [11]. The SSN terminates in two motor branches to the infraspinatus muscle and smaller branches to the glenohumeral joint and scapula.

13.3 Pathophysiology

In 1886 Dörrien presented the first case of an isolated SSN lesion [12]. In 1959 Kopell and Thompson described suprascapular neuropathy at the suprascapular notch and in 1982 Aiello

B. Kordasiewicz (✉) · A. Boszczyk
Trauma and Orthopaedics Department,
SPSK im. A. Grucy, Otwock, Poland

Centre of Postgraduate Medical Education,
Warsaw, Poland

M. Janyst
Trauma and Orthopaedics Department,
SPSK im. A. Grucy, Otwock, Poland

Medical University of Warsaw, Warsaw, Poland

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et al. presented two points of entrapment: at the suprascapular notch at the spinoglenoid notch [13, 14]. Various ethologies of the SSN pathologies have been presented. Direct trauma to SSN is very rare, but reported as iatrogenic injury or as a result of fracture [15, 16]. Parsonage-Turner Syndrome, a rare neurological entity of unknown reason, also should not be forget, as this is usually self-limiting disease and if correctly diagnosed using electromyographic (EMG) studies—surgical intervention can be avoided [17, 18]. Despite these above described rare conditions, the usual two anatomic sites of compression can generate two separate clinical entities. A compression at the suprascapular notch generally leads to weakness of both the supraspinatus and infraspinatus (Fig. 13.1). A compression at the spinoglenoid notch leads to isolated infraspinatus weakness (Fig. 13.2). It is believed that addressing the problem (usually TSL at the suprascapular notch and paralabralcyst at the spinoglenoid notch) can resolve the compression. According to recent reports, more usual and more probable reasons of SSN pathology could be divided into compression or traction related [19]. Ganglions (spinoglenoid cysts), ossified TSL, bone or soft tissue

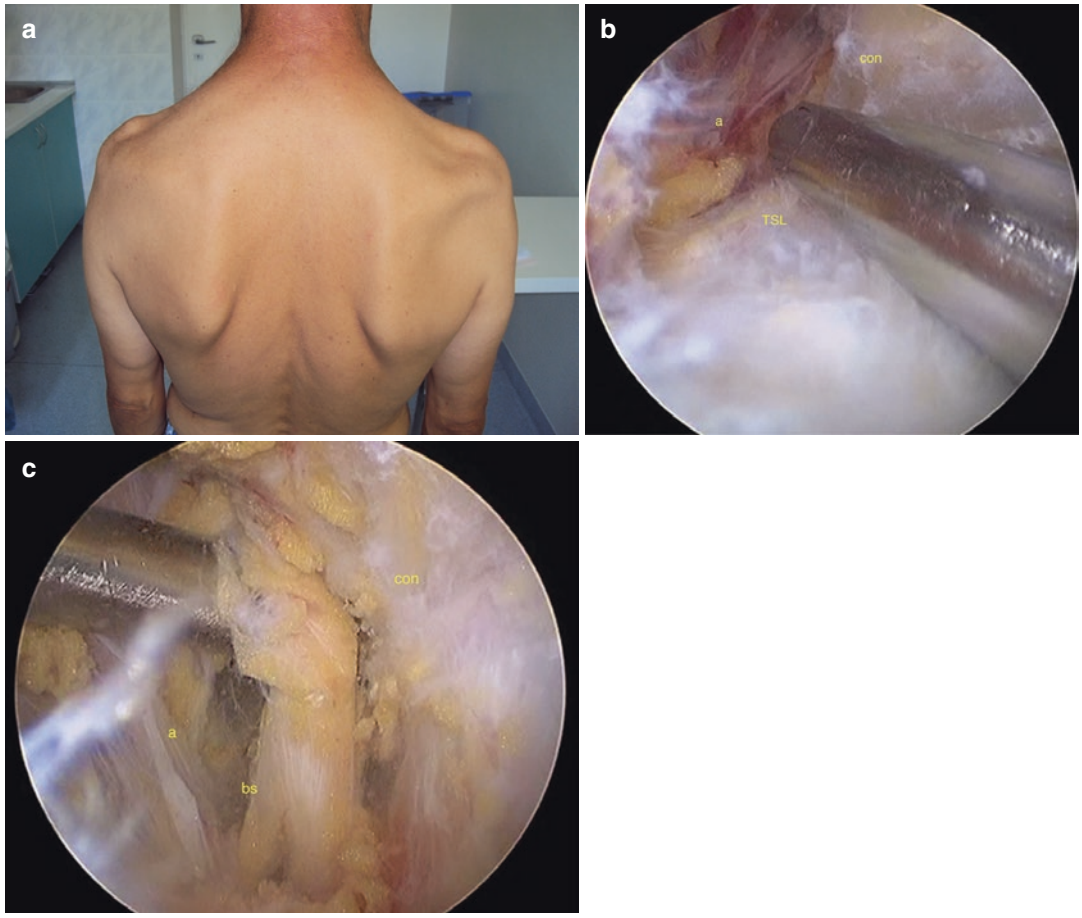

Fig. 13.1 The SSN pathology at the suprascapular notch. (**a**) The SSN disturbances in a patient with massive rotator cuff lesion. Clinical image of the supraspinatus and the infraspinatus muscles atrophy is the same as in the SSN compression at the suprascapular notch. (**b**) Arthroscopic view of the suprascapular notch area; right shoulder, beach chair position, arthroscope in the lateral portal, shaver in the antero-lateral portal. (**c**) The SSN after ligament release, trocar releasing the nerve in the G poral (the modified Neviaser portal). Conoid ligament (con), transverse scapular ligament (TSL), suprascapular artery (a), the branch of the supraspinatus muscle (bs)

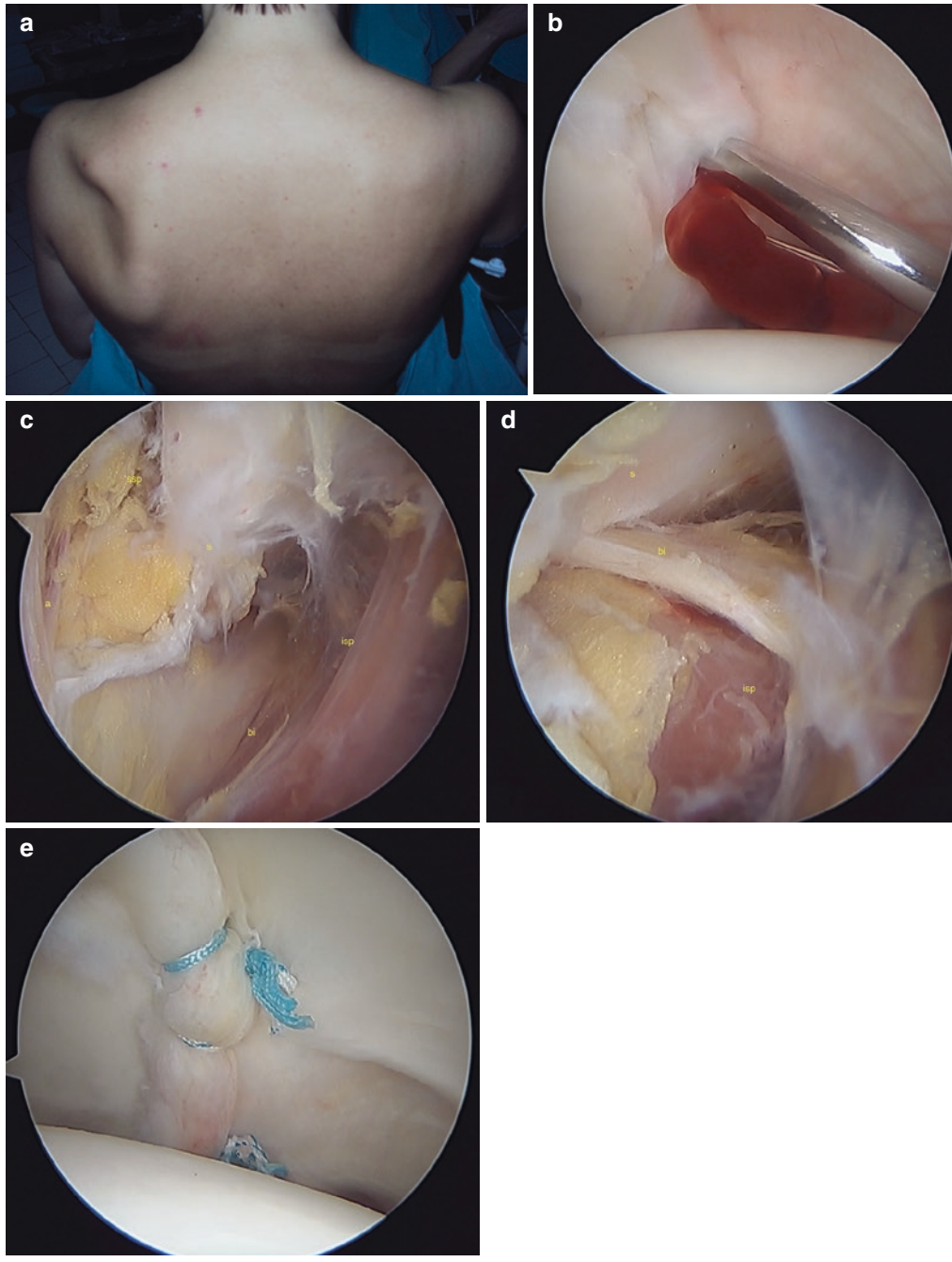

Fig. 13.2 The SSN pathology at the spinoglenoid notch. (**a**) The infraspinatus insufficiency—clinical image of the patient with a spinoglenoid cyst. (**b**) Arthroscopic intraarticular view, left shoulder, beach chair position, arthroscope in the anterolateral portal. Spinoglenoid cyst evacuation, the tissue liberator and the needle below are introduced from the posterolateral portal. (Six weeks before arthroscopy an ultrasound guided evacuation of the cyst was performed in a different centre—not successful—it explains the blood clots in the cyst). (**c**, **d**) decompression of the SSN at the spinoglenoid notch; arthroscope in the subacromial space in the lateral portal, scapular spine visible from above the rotator cuff muscles. (**e**) Arthroscopic intraarticular view—posterior labrum repair. Authors preferred method is the spinoglenoid cyst and the SSN decompression (at the spinoglenoid notch) followed by posterior labrum repair. Supraspinatus muscle (ssp), infraspinatus muscle (isp), a scapular spine (s), branches of the infraspinatus muscle (bi), the suprascapular artery at the spinoglenoid notch (a)

tumours or vascular anomaly could compress the nerve. Repetitive overhead activity in athletes is believed to create some traction leading to SSN dysfunction. It was also proven that spinoglenoid ligament tightens in a overhead position in throwing, resulting in increased pressure on the SSN [20]. Another traction related problem is SSN pathology related with massive RCT—retraction of supraspinatus tendon is responsible for increasing the tension by changing the angle between the nerve and its motor branches [19].

13.4 SSN Pathology and Rotator Cuff Tears

In 2003 Albritton et al. presented cadaver study describing correlation between the SSN tension and supraspinatus tendon retraction [5]. They also proved the motor branch to the supraspinatus muscle was taut if the tendon retraction reached 2–3 cm. Authors concluded, that medial retraction "drastically" changes the course of the SSN particularly at the spinoglenoid notch. Massimini et al. found that tear and retraction of the supraspinatus muscle resulted in medial translation of the nerve at the suprascapular notch and significantly increased the nerve tension [4]. Kong et al. reported the results of evaluation of massive RCT with severe fatty infiltration in the infraspinatus muscle. The mean retraction of the infraspinatus was 3.6 cm in patients with more severe fatty degeneration in the infraspinatus, versus 3.0 cm in those with more severe degeneration in the supraspinatus ($p = 0.003$). Authors concluded that fatty degeneration affecting the infraspinatus more than the supraspinatus may be due to entrapment of the suprascapular nerve at the spinoglenoid notch [21]. Another SSN related question could be lateral advancement of retracted tendons during their release and repair. Warner et al. described the SSN anatomy performing dissections on 18 cadavers and concluded, that normal anatomy limits the possibility of the lateral tendon advancement. They reported that supraspinatus muscle can be laterally mobilised up to 1 cm—then the motor branches are damaged. Releasing the SSN at the suprascapular notch would be another 5 mm added to above distance of 1 cm [22]. Also Greiner et al. demonstrated increased tension in medial motor branches when advancing the supraspinatous tendon laterally [23]. Savoie et al. proposed a hypothesis of SSN correlation with RCT. Disruption of the tendon causes subsequent retraction of the rotator cuff changing the SSN tension and additionally scar tissue formation in this area. This scar tissue not only limits the mobility of the tendon, but also compresses the nerve. Whilst mobilisation and repairing the rotator cuff tendons, the tension in the nerve increases, leading to clinical signs. Authors found it might be a potential indication for nerve release at the suprascapular notch [8].

13.5 Examination and Diagnosis

Clinical findings in SSN pathology can vary according to nerve function, duration of symptoms and associated pathologies. Infraspinatus atrophy, decrease of strength of external rotation and abduction can direct the physician to the diagnosis. Lafosse et al. described "the suprascapular stretch test"—a provocative maneuver increasing the symptoms due to the traction of the SSN [24]. MRI studies can present atrophy and fatty infiltration of supraspinatus and infraspinatus muscle depending on site of compression. MRI can also identify any lesions responsible for the SSN compression—tumours and ganglion cysts. The situation remains more difficult in case of massive RCT—clinical tests are usually linked with the tendons rupture and MRI findings may be correlated to fatty infiltration and atrophy due to RCT [11]. EMG studies remain the gold standard and the only tool to detect the SSN disturbances. It is particularly helpful if physical examination and imaging studies present no obvious pathology or massive RCT. The usual nerve motor latency varies in the range of 1.7–3.7 ms for the supraspinatus and 2.4–4.2 ms for the infraspinatus at the stimulation performed at Erb's point. A value above 2.7 and 3.3 ms indicates abnormality for compression of the supraspinatus and infraspinatus respectively [11, 25]. Other EMG findings suggesting the SSN pathology are a decrease in

the amplitude or in the spontaneous or marked polyphasicity of the evoked potentials. Reduction in the interference pattern can be seen in long-standing neuropathy. Additional findings could also be positive sharp waves and fibrillation potentials and absent or decreased numbers of motor unit action potentials (MUAP) in muscles and features of reinnervation of MUAP [25]. It is important to remember that the SSN dysfunction can be present with a normal nerve conduction studies—it was proven that EMG and nerve motor latency are accurate in 91% [26]. It is to notice that diagnosis of the SSN neuropathy can be sometimes difficult. Momaya et al. reported that a mean time from onset of symptoms to decompression was 19 months. In their review study authors found, that the most common symptom was deep, posterior shoulder pain—a symptom difficult to differentiate from other pathologies [1].

13.6 Surgical Technique

Up to date no proper comparative studies have indicated superiority of arthroscopic technique over open one [1]. Nevertheless, for shoulder surgeons, possibility to address all other pathologies in one arthroscopic procedure seem to be more tempting and justified. In 2007 Lafosse et al. described an arthroscopic technique of the SSN decompression at the suprascapular notch [27]. A patient is operated on in the beach-chair position. After glenohumeral joint inspection, subacromial space is approached—the arthroscope is placed in lateral portal and working instruments are introduced in antero-lateral portal. The coraco-acromial ligament is followed to find the lateral border and base of the coracoid. More medial coraco-clavicualar ligaments are exposed. Directly medial to the conoid ligament, the suprascapular notch is located. In order to expose its structures an additional portal is performed between the clavicle and the scapular spine (G portal or modified Neviaser portal). Using trocar (if blunt decompression is possible) or arthroscopic scissors the TSL ligament is released, paying attention to the suprascapular artery—in 2.5% artery passes under the ligament [28]. A bony notch resection might be necessary in case of anatomic variations [6]. Arthroscopic spinoglenoid notch decompression was usually performed in association with paralabral ganglion cysts decompression. Bhatia et al. proposed cyst decompression using intraarticular method—a shaver and probe (or switching stick) are introduced from anterior and posterior portals under the rotator cuff tendons to achieve cloudy fluid outflow from the cyst [29]. Other authors proposed to achieve the cyst from subacromial space [11, 30]. Plancher and Petterson reported decompression of the SSN using an additional posterior viewing portal located 8 cm medial to the posterolateral corner of the acromion, so the surgeon looks at the scapula spine from medial following the fibers of the infraspinatus muscle [11]. Starting the entire arthroscopic procedure from the SSN decompression at the spinoglenoid notch is recommended to avoid swelling.

13.7 Controversies

In 2018 Momaya et al. reported a first systematic review about outcomes of the SSN decompression [1]. They reported 21 studies (including together 275 patients—276 shoulders), the mean age at surgery was 41.9 years and the mean follow-up 32.5 months. Ninety-four percent of patients had EMG (85% with positive results). It is interesting to realize that of the 21 above studies 11 involved decompression at the spinoglenoid notch only, 5 at the suprascapular notch only and 5 in both places (combined). Six of these studies concerned open and 15 arthroscopic technique. Only two complications were reported (0.74%): one soft tissue infection and one adhesive capsulitis. No studies comparing operative versus nonoperative treatment are found. Several case-studies presented successful results in patients with isolated symptomatic SSN entrapment regardless of age. Shah et al. reported significant improvement in 24 patients who underwent arthroscopic SSN nerve decompression (at suprascapular and/or spinoglenoid notch) at an average of 9.4 weeks after surgery [31]. Lafosse et al. reported an

increase in the average Constant score from 60.3 points preoperatively to 83.4 points in ten patients, with significant improvement in EMG results and also pain and function. The mean time to return to activity was 3 weeks [27]. Garcia et al. presented the outcomes of nine patients after arthroscopic SSN decompression at an older age (mean: 69.5 years). They reported significant improvements after surgery: in the UCLA score from 11.7 to 26.1, SF-36 questionnaire was 122.9 and the raw pain scale was 88% [32].

Leclere et al. reported four cases of complete fatty infiltration of supraspinatus and/or infraspinatus due to suprascapular neuropathy with intact rotator cuff and no specific traction or compression activity. Pain and function was immediately improved after arthroscopic SSN decompression. Improvement in strength was more predictable in abduction than in external rotation [19]. Similar results were reported by Kim et al. after open SSN decompression in 42 patients. They reported that 90% of patients improved abduction strength to grade 4 or better, as infraspinatus function improved to better than grade 3 only in 32% [33]. The management of SSN in association with concomitant shoulder pathology remains controversial. It is debatable, if SSN in such cases (particularly in case of paralabral cysts) should be liberated only in the site of compression or also in suprascapular or/and spinoglenoid notch. Additionally, it is debatable, if a cyst needs evacuation or whether repairing a concomitant labral tear will decompress the cyst thus resolving the SSN neuropathy. Kim et al. compared SLAP repair alone with SLAP repair with cyst decompression. The results were comparable suggesting that only simple SLAP repair was enough to resolve the problem [34]. The opposite results were presented by Pillai et al. [35]. They reported that cyst decompression led to greater strength increases than SLAP repair alone. Tsikouris et al. compared the clinical outcomes between elite overhead athletes who underwent SSN decompression associated with shoulder arthroscopy procedures and those without SSN decompression [2]. Thirty-five patients in SSN decompression group yielded superior outcomes then 21 patients after arthroscopy surgery only: Constant score mean 91 versus 82, UCLA score average 33 versus 28 and return to sport was 97% versus 84%, respectively. Twenty-seven patients had rotator cuff repair associated with SSN decompression comparing to 18 without SSN decompression. In the SSN decompression group all patients had significant improvement in postoperative EMG results at an average 6.2 months, except 3 patients (javelin throwers with symptomatic relief). In 2016 Savoie et al. presented a group of 22 patients who underwent revision repair of massive rotator cuff tears (retracted medial to the glenoid and Goutallier grade 4) and concomitant release of the SSN [8]. The results were compared to a similar group of 22 patients (Goutallier grade 3) who underwent revision rotator cuff repair without nerve release. Authors concluded that patients who underwent associated SSN release had better improvement in pain relief, active forward flexion and strength than a comparable group without SSN release. They also had noticed however, that SSN release did not improve tendon healing. Opposite to above studies Costouros et al. found that 7 out of 26 patients (38%) with massive RCT had electromyographic (EMG) and nerve conduction velocity (NCV) signs of SSN pathology [36]. In 6 of them (1 patient presented not reparable tear), after 6 months from partial or complete repair without nerve decompression, nerve recovery (partial or total) was confirmed in EMG/NCV. This correlated with complete pain relief and improvement in function. The authors concluded that arthroscopic rotator cuff repair could result in reversal of SSN pathology, which may correlate with improvement in pain and function. Authors believed that this recovery was related with SSN tension release—so called indirect decompression, due to the infraspinatus muscle and tendon lateral traction causing the SSN lateral translation away from the scapula spine—another point when the nerve could be tethered.

Aramberri, in his non-published study (thesis) on a pool of 100 patients operated by Lafosse due to massive RCT between 2004 and 2007, with a minimum follow-up of 24 months, reported 34.6% of the prevalence of the SSN pathology [37]. He found significant improvement in conduction of the supraspinatus branch in patients

after SSN release in the suprascapular notch. The infraspinatus branch ameliorated its conduction, but these findings were not significant [37].

Another topic raising controversies is SSN pathology in overhead athletes. Several studies reported that overhead athletes are prone to the SSN pathology due to repetitive overhead movement. Lajtai et al. reported that the prevalence of infraspinatus muscle atrophy in professional beach volley players was 30% [38]. They found that the Constant score was lower in players with atrophy: 87 versus 93 points in players without atrophy. They also noted the significant difference in external rotation strength (8.2 kg versus 9.5 kg). In another Lajtai et al. study concerning percutaneous EMG and NCV in volleyball players, decreased nerve conduction velocity was reported in all patients with atrophy, however lower activation patterns on electromyography were seen only in the severe athrophy group [39]. Players with atrophy had significantly greater loss of external rotation than those without atrophy. These changes confirm the hypothesis of a repetitive strain or traction injury of the SSN—stretching neuropathy. Cummins et al. found that infraspinatus atrophy was associated with a higher level and duration of sport activity [7]. It confirms that the repetitive overhead activity could lead to suprascapular nerve irritation at the sinoglenoid notch leading to "cumulative neuropraxia". Up to date most of authors had agreed that overhead athletes should be initially treated nonoperatively, however the last publication of Tsakuris et al. made this less clear [2].

13.8 Conclusions

The SSN pathology is rare but certainly existing entity. Surgical SSN release in case of proven pathology related with nerve compression is a well-described, low risk and successful treatment. It is to remember, however, that strength restitution is more predictable in abduction than in external rotation. The SSN entrapment in relationship with rotator cuff tears remains widely unclear and should be investigated.

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