Suprascapular nerve decompression in addition to rotator cuff repair: a prospective, randomized observational trial

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Abstract: BACKGROUND Tear and retraction of the supraspinatus (SS) and infraspinatus (IS) musculotendinous units and/or their repair may be associated with traction damage to the suprascapular nerve, potentially responsible for pain or weakness of the rotator cuff (RC). Arthroscopic release of the transverse scapular ligament at the suprascapular notch has been advocated to prevent or treat suprascapular nerve impairment associated with RC retraction and/or repair. The effect of this procedure on preoperative normal nerve function is, however, not well studied. We hypothesize that (1) decompression of the suprascapular nerve without preoperative pathologic neurophysiological findings will not improve clinical or imaging outcome and (2) suprascapular decompression will not measurably change suprascapular nerve function. METHODS Nineteen consecutive patients with a magnetic resonance arthrography documented RC tear involving SS and IS but normal preoperative electromyography (EMG)/nerve conduction studies of the SS and IS were enrolled in a prospective, controlled trial involving RC repair with or without suprascapular nerve decompression at the suprascapular notch. Nine patients were randomized to undergo, and 10 not to undergo, a decompression of the suprascapular nerve. Patients were assessed clinically (Constant score, mobility, pain, strength, subjective shoulder value), with magnetic resonance imaging and neurophysiology preoperatively and at 3- and 12-month follow-up. RESULTS There was no clinically relevant difference between the release and the non-release group in any clinical parameter at any time point. At magnetic resonance imaging, there was a slightly greater increase of fatty infiltration of the IS in the release group without any other differences between the 2 groups. Electromyographically, there were no pathologic findings in the non-release group at any time point. Conversely, 3 of the 9 patients of the release group showed pathologic EMG findings at 3 months, of whom 2 had recovered fully and 1 only partially at 12 months. CONCLUSION In the presence of normal EMG findings, suprascapular nerve release added to arthroscopic RC repair is not associated with any clinical benefit, but with electromyographically documented, postoperative impairment of nerve function in 1 of 3 cases. Suprascapular nerve release does not therefore seem to be justified as an adjunct to RC repair if preoperative EMG findings document normal suprascapular nerve function. Based on these findings, the ongoing prospective randomized trial was terminated.

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Suprascapular nerve decompression in addition to rotator cuff repair: a prospective, randomized observational trial

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Background: Tear and retraction of the supraspinatus (SS) and infraspinatus (IS) musculotendinous units and/or their repair may be associated with traction damage to the suprascapular nerve, potentially responsible for pain or weakness of the rotator cuff (RC). Arthroscopic release of the transverse scapular ligament at the suprascapular notch has been advocated to prevent or treat suprascapular nerve impairment associated with RC retraction and/or repair. The effect of this procedure on preoperative normal nerve function is, however, not well studied. We hypothesize that (1) decompression of the suprascapular nerve without preoperative pathologic neurophysiological findings will not improve clinical or imaging outcome and (2) suprascapular decompression will not measurably change suprascapular nerve function.

Methods: Nineteen consecutive patients with a magnetic resonance arthrography documented RC tear involving SS and IS but normal preoperative electromyography (EMG)/nerve conduction studies of the SS and IS were enrolled in a prospective, controlled trial involving RC repair with or without suprascapular nerve decompression at the suprascapular notch. Nine patients were randomized to undergo, and 10 not to undergo, a decompression of the suprascapular nerve. Patients were assessed clinically (Constant score, mobility, pain, strength, subjective shoulder value), with magnetic resonance imaging and neurophysiology preoperatively and at 3- and 12-month follow-up.

Results: There was no clinically relevant difference between the release and the non-release group in any clinical parameter at any time point. At magnetic resonance imaging, there was a slightly greater increase of fatty infiltration of the IS in the release group without any other differences between the 2 groups. Electromyographically, there were no pathologic findings in the non-release group at any time point. Conversely, 3 of the 9 patients of the release group showed pathologic EMG findings at 3 months, of whom 2 had recovered fully and 1 only partially at 12 months.

Conclusion: In the presence of normal EMG findings, suprascapular nerve release added to arthroscopic RC repair is not associated with any clinical benefit, but with electromyographically documented, postoperative impairment of nerve function in 1 of 3 cases. Suprascapular nerve release does not therefore seem to be justified as an adjunct to RC repair if preoperative EMG findings document normal suprascapular nerve function. Based on these findings, the ongoing prospective randomized trial was terminated.

Level of evidence: Level II; Randomized Controlled Trial; Treatment Study
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Keywords: Shoulder; arthroscopic rotator cuff repair; suprascapular nerve neuropathy; fatty infiltration; electromyography; magnetic resonance imaging

Institutional review board approval for this study was received from the Cantonal Ethics Committee Zürich (number: 2017-00425).

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Suprascapular nerve dysfunction can occur not only due to inflammatory phenomena like Parsonage-Turner syndrome, \textsuperscript{12} compression by cysts and tumors, \textsuperscript{19,23,30,38} or entrapment under the transverse scapular ligament at the scapular or spinoglenoid notch, but also due to trauma \textsuperscript{20,36,41,44} and repetitive traction, especially in overhead sports. \textsuperscript{10,21,26,32,34,43} Suprascapular nerve impairment can, however, allegedly also be caused by retraction of the torn posterosuperior rotator cuff (RC) or during its repair involving lateral advancement of the supraspinatus (SS) and infraspinatus (IS) muscle tendon units.\textsuperscript{17,28,42}

The prevalence of combined suprascapular nerve lesion and RC tears is largely unknown and varies strongly in the literature. It appears that the combination of tear and nerve lesion is more frequent in larger tears. Prospective studies document that objective, preoperative suprascapular nerve dysfunction is exceptional in RC tears with a prevalence of no more than 2\%.\textsuperscript{6} In patients with massive RC tears, much higher prevalences of 8\% to 60\%\textsuperscript{35,23,39} have been reported, albeit not always well validated methodologically.

Whether decompression of the suprascapular nerve at the suprascapular notch in addition to an arthroscopic RC repair improves postoperative outcome is still controversial. Some authors reported better clinical outcomes by additional decompression of the nerve.\textsuperscript{35,39} Others reported excellent clinical outcome and reversal of a suprascapular nerve pathology by RC repair without additional decompression.\textsuperscript{8,28} The risk of suprascapular nerve decompression, however, has never been assessed quantitatively.

The purpose of this prospective, randomized, controlled study was to assess the value of suprascapular nerve decompression in addition to RC repair. We hypothesized that (1) the decompression of the suprascapular nerve without preoperative pathologic electromyography (EMG) findings does not improve clinical or imaging outcome as measured by fatty infiltration of the SS and IS muscles and that (2) suprascapular decompression does not measurably change suprascapular nerve function.

Material and methods

Inclusion criteria

All patients gave written consent according to the approved consent form. Patients prospectively seen between December 2013 and April 2016 with SS or SS and IS tears willing to participate in this trial were randomly assigned to the release and non-release group in a 1:1 ratio. Randomization was done by designated study staff drawing a sealed envelope containing a randomization card. Patients are informed about the assignment by the treating investigator.

- Inclusion criteria were: (1) signed informed consent for the planned study, (2) patient at least 18 years old, (3) partial or complete RC tear of the SS and/or IS muscle with typical clinical symptoms and documentation in magnetic resonance arthrography, and (4) a repairable RC tear with maximum fatty infiltration stage II according to the magnetic resonance adaptation of the Goutallier classification.\textsuperscript{11,16}

- Exclusion criteria were (1) any previous surgery to the affected shoulder, (2) fatty degeneration stages III or IV according to Fuchs et al,\textsuperscript{11} (3) previous infections of the shoulder joint, (4) systemic corticosteroid therapy, (5) diabetes mellitus, (6) bleeding disorders and oral anticoagulation, (7) pregnancy or breastfeeding, (8) contraindications for magnetic resonance imaging (MRI) or EMG, (9) drug or substance addiction, and (10) previous surgery to the opposite shoulder.

Surgery and aftercare

Surgery was performed by 1 of the 2 most experienced consultants in the shoulder unit of the respective department (C.G. and D.C.M.). The patient was placed in a beach chair position with the arm in the arm holder (SPIDER Limb Positioner, Tenet Medical Engineering, Calgary, Alberta, Canada). For pain control an interscalene catheter was installed preoperatively and removed 2 days postoperatively. The surgery was done using general anesthesia or sedation. Disinfection with Betaseptic and draping was done in a standardized manner. For the procedure, the surgeons created a posterior, lateral, anterolateral, anterior, and superior (Nevisier) portal. After diagnostic arthroscopy, suprascapular nerve release was performed as described by Lafosse et al with the scope in the posterior portal or, if necessary, in one of the lateral portals in patients randomized into the release group. Subacromial bursectomy was performed by dissecting toward the medial origin of the supraspinate muscle at its anterior border. Through the Nevisier portal, tissue dissection was performed along the coracoclavicular ligaments using blunt preparation and radiofrequency ablation. Once the transverse ligament was visible and the surrounding tissue was removed, arthroscopic dissectors were used to cut the superior transverse ligament of the scapula. After visual integrity and free mobility of the suprascapular nerve were documented, standard reconstruction of the RC using suture anchors in a single row configuration was completed. According to the intraoperative findings, the surgeons performed additionally acromioplasty (14 cases), biceps tenotomy (12 cases) or biceps tenodesis (2 cases), acromioclavicular joint resection (2 cases), decompression of a calcium deposit (1 case), and humeral head microfracturing (1 case).

Aftercare consisted of wearing an abduction brace for 6 weeks allowing passive mobilization under physiotherapeutical control. Active range of motion exercises were carried out without weight through weeks 7 to 12. Strengthening was started at 13 weeks postoperatively.

Clinical examination

The patients underwent clinical examination, imaging (conventional X-ray and Arthro-MRI) and neurophysiological evaluation (needle EMG of the IS muscle and nerve conduction study [NCS] of the suprascapular nerve) preoperatively and at 3- and 12-month follow-up. The clinical examination included an assessment of the subjective shoulder value,\textsuperscript{15} the absolute\textsuperscript{7} and relative Constant-Murley score\textsuperscript{13} including measurement of abduction strength with a validated dynamometer.
Magnetic resonance imaging

The MRIs were performed using the same specific protocol for each patient at each time point. MRI at our institution was obtained in a 1.5 Tesla (Magnetom Avanto Fit; Siemens Healthcare, Erlangen, Germany) or a 3.0 Tesla magnet (Magnetom Skyra Fit; Siemens Healthcare). We obtained at least 4 sequences in 3 standard imaging planes (axial, coronal, and sagittal). Sequences included at least 1 T1-weighted and several fluid-sensitive sequences (proton-density weighted with fat suppression).

Radiographic evaluation was performed by 3 examiners, including 1 radiologist, specialized in musculoskeletal imaging and head of the department, 1 senior consultant in orthopedic surgery, and 1 orthopedic resident. Deviating measurements were discussed and read out in consensus. We analyzed (1) tendon integrity pre- and postoperatively (classification as (a) no tear, (b) partial tear, (c) full-thickness tear, or (d) tendinopathy), (2) the number of affected tendons, and (3) the degree of fatty infiltration according to modified Goutallier on MRI.\textsuperscript{15,16}

Neurologic examination

Neurologic and neurophysiological examination was performed by a senior consultant, board certified in electrodiagnostic medicine, using Dantec Keypoint G4 (Neuroline AG, Belp, Switzerland). Testing was performed using the same specific protocol and method for each patient. For the examination, the patient was placed in a sitting position with resting shoulders on the side. For EMG testing, a needle was inserted into the IS muscle 2 cm inferior to the scapular spine. NCS was performed using the same recording needle with supramaximal stimulation through a surface electrode at Erb’s point.

EMG was used to determine normal and pathologic nerve activity. It included measurement of acute spontaneous pathologic activity in the form of fibrillations and positive sharp waves indicating recent axonal damage while complex repetitive discharge and fasciculation were assumed to be signs of chronic axonal damage on the spinate muscles of both shoulders. Recruitment and motor unit potentials were evaluated while tensioning the muscle. NCS included latency (not shown for all patients) and amplitude values of compound muscle action potentials on electrical stimulation. NCS data were measured but not used as a criterion for abnormality due to questionable reproducibility and the absence of established cutoff values for pathology.

Data collection and statistical analyses

Patient’s data were collected in the REDCap electronic data capture system version 8.6.1 (Vanderbilt University, Nashville, TN, USA) anonymously.\textsuperscript{18}

For statistical analyses, IBM SPSS Statistics software for Windows version 23.0 (IBM Corp., Armonk, NY, USA) was used.

Values were expressed as mean ± standard deviation for parametric data and as median ± interquartile range for nonparametric data. Data were tested for normal distribution using the Kolmogorov-Smirnov test. We used paired and unpaired t-tests for parametric data, the Wilcoxon or Mann-Whitney U test for nonparametric data, and Fisher’s exact test for categorical variables. A P value of less than .05 was considered significant.

Results

Demographics

We recruited 23 patients, meeting the inclusion criteria. Three patients dropped out, 1 due to incompliance in wearing the abduction brace. Two patients had to undergo revision surgery, 1 due to a postoperative surgical site infection and 1 due to repair failure after trauma. The other 20 completed the study protocol, resulting in a follow-up of 100%. Baseline characteristics are displayed in Table I.

We included 19 patients with preoperative normal EMG values in our analysis (see Fig. 1). One patient (ID20) randomized into the release group had a single tendon tear but showed pathologic preoperative EMG findings with positive sharp waves and fibrillation. He underwent repair with release of the suprascapular nerve and achieved an excellent clinical outcome. The measured amplitude increased from 0.14 mV preoperatively to 0.83 mV at 3-month and to 7.0 mV at 12-month follow-up. The latency was pathologic with 10.1 ms preoperatively and at 3-month follow-up, but decreased over the course to 2.21 ms at 12-month follow-up. The preoperatively presented positive sharp waves and fibrillation had resolved completely at 12 months.

Clinical evaluation

A comparison between the release and non-release group showed no significant differences for all clinical examinations preoperatively and postoperatively at 3 and 12 months.

The results of the surgery led to a significant improvement in most of the parameters. A comparison between

| Table I | Comparison of basic demographic data using the Mann-Whitney U test (age) and Fisher’s exact test (side, sex, tendons) |
|---------|---------------------------------------------------------------------------------------------------------------|
| Patients | Release group | Non-release group | P value |
| n =9 | n = 10 |
| Age (mean) | 60 ± 7 | 59 ± 9 | .743 |
| Side | 6 right, 3 left | 9 right, 1 left | .582 |
| Sex | 3 male, 6 female | 5 male, 5 female | .650 |
| Affected tendons | | | .187 |
| 1-tendon injury | 3 | 2 |
| 2-tendon injury | 6 | 5 |
| 3-tendon injury | 0 | 3 |
Preoperative and 12-month follow-up documented improvement in the absolute and relative Constant score, subjective shoulder value, and pain in the release group ($P = .004$, $P = .004$, $P = .004$, $P = .004$) and in the non-release group ($P = .002$, $P = .002$, $P = .002$, $P = .002$) (Figs. 2-4).

Mean abduction strength increased significantly in the release and the non-release group ($P = .012$, $P = .020$), but there was no difference between the groups ($P = .89$).

In terms of mobility, improvement was documented for both groups. There was a significant improvement in flexion and abduction in the release group (preop vs. 12-month follow-up, $P = .02$ and $P = .02$) and for abduction in the non-release group (preop vs. 12-month follow-up, $P = .02$) (Fig. 5). There was no significant improvement for flexion in the non-release group ($P = .20$). Flexion had been lower in the release group, but the final results were identical for the 2 groups ($P = .95$).

The intervention did not improve external rotation and internal rotation in neither the release (preop vs. 12-month follow-up, $P = .14$, $P = .81$) nor the non-release group (preop vs. 12-month follow-up, $P = .31$, $P = .27$).

We compared the subgroup of patients with abnormal EMGs with the other study patients. We did not identify any differences between absolute or relative Constant scores, pain, strength, flexion, abduction, external rotation, and internal rotation at any time point.

**Magnetic resonance imaging**

In MRI, no retear or failed healings were detected up to 12 months postoperatively.

For fatty infiltration, we detected no differences between the 2 groups preoperatively and at 3- and 12-month follow-up for the SS ($P = .93$, $P = .63$, $P = .72$) (Table II), subscapularis ($P = .66$, $P = .85$, $P = .85$), and IS muscles ($P = .33$, $P = .10$, $P = .66$) (Table III).

Comparing the status preoperatively and at 12-month follow-up, there were no significant changes in...
fatty infiltration in the release and non-release group for the SS ($P = .08$ and $P = .48$) and the subscapularis ($P = .32$ and $P = .32$). For the IS muscle, the difference in the non-release group ($P = .10$) was not significant, but in the release group, it was ($P = .03$). The analysis of the individual data, however, shows that fatty infiltration also increased in the SS and IS muscle in individual cases.

Electromyography and nerve conduction studies

Preoperatively 22 of 23 analyzed patients had entirely normal EMG findings and constitute the body of this study. Three months postoperatively, we identified 3 patients in the release group with abnormal EMG findings (33.3% in the release group). In the non-release group, no abnormal EMG was recorded at any time point. At the 1-year follow-
up examination, 1 EMG in the release group continued to be abnormal, but with a significant improvement over the findings at 3 months. In the 2 other patients, the previously pathologic findings were normalized. NCS using intra-muscular needle recordings to assess compound muscle action potentials in follow-up examinations from the IS muscle resulted in largely variable compound muscle action potential amplitudes, as can be seen in Table IV. Inconsistent with EMG criteria, several cases of amplitude loss of more than 50% were found during follow-up, which in NCS studies of distal peripheral nerves would be assumed to indicate axonal damage. However, with similarly inconsistent reproducibility of amplitude data also obtained on the nonaffected contralateral side (control data not shown), this was deemed to indicate shortcomings in reproducible positioning of the recording needle, or stimulation, or both. Therefore only EMG signs of acute spontaneous activity were used as a most conservative criterion for axonal nerve damage in the operated side in follow-up examinations.

Nevertheless, evaluation between the 2 groups at 3- and 12-month follow-up revealed significantly lower amplitude values in the release group. The analysis of the latency showed similar results between the groups at all time points (see Supplementary figures).

The abnormal EMG findings in the 3 patients were as follows:

- ID1: High-frequency, abnormal potentials (acute spontaneous pathologic activity in the form of fibrillations and positive sharp waves) at 3-month follow-up that had resolved at 12 months. The decrease in suprascapular nerve amplitude was from 12.6 to 1.44 mV (preoperative vs. 3-month follow-up). At 12-month follow-up, the value was 5.4 mV.

- ID2: Few abnormal potentials (acute spontaneous pathologic activity in the form of fibrillations and positive sharp waves) at 3 months, again absent at 12 months. The amplitude increased from 6.20 preoperatively to 12.30 mV at 3 months and 14.70 mV at 12 months. In this case, NCS data point to effective release.

- ID5: Few abnormal potentials (acute spontaneous pathologic activity in the form of fibrillations and positive sharp waves) as well as increased polyphasic waves and signs of neurogenic reorganization at 3 months with mild regression at 12 months. A decrease in suprascapular nerve amplitude from 10.1 preoperatively to 3 mV at 3 months and to 2.2 mV at 12 months was measured.

### Complications

We terminated our study because of safety concerns with 3 not clinical, but electromyographic complications in 3 patients in the release group and because no benefit whatsoever could be identified or suspected with the data available for such a particular cohort.

We had 3 dropouts, 1 patient with an infection, 1 patient with a retear, and 1 patient dropped out due to noncompliance by not using the abduction brace.

### Discussion

It was the original study plan to include 60 patients, constituting 4 groups of 15 patients each. We had expected to be able to recruit 30 patients with preoperatively abnormal and 30 with normal EMG findings for the suprascapular nerve. In each group, patients were planned to either receive or not receive a nerve decompression. The first finding of this study was that it is almost impossible to find RC tears with measurable preoperative suprascapular nerve dysfunction. Within the first 20 recruited patients, only 1 had abnormal preoperative nerve function. By the time we had included 20 patients, we already had 3—albeit clinically irrelevant—but clear electromyographic complications without any clinical benefit in the release group without any complications in the non-release group, so it was decided to terminate the trial.

Although this trial is limited in the number of patients, it has a complete clinical, imaging, electromyographic, and

### Table II Fatty infiltration of the supraspinatus muscle between the release and non-release groups

| Musculus supraspinatus | Goutallier | 0 | 1 | 2 | 3 | 4 | $P$ value |
|------------------------|-----------|---|---|---|---|---|-----------|
| Preoperative Release    | 3         | 5 | 1 | 0 | 0 | 0 | .93       |
| Preoperative Non-release| 5         | 2 | 3 | 0 | 0 | 0 |           |
| 3-month Release        | 1         | 5 | 3 | 0 | 0 | 0 | .63       |
| 3-month Non-release    | 1         | 7 | 2 | 0 | 0 | 0 |           |
| 12-month Release       | 2         | 4 | 3 | 0 | 0 | 0 | .72       |
| 12-month Non-release   | 2         | 6 | 2 | 0 | 0 | 0 |           |
electroneurographic follow-up in all included patients. It documents that suprascapular nerve decompression at the suprascapular notch is not necessary for SS and IS tears of Goutallier stages 1 and 2, as the clinical outcome of repair of these tears without nerve decompression neither leads to measurable compromise or dysfunction of the suprascapular nerve nor is it associated with more fatty infiltration than RC repair with nerve decompression. Nerve decompression in addition to repair of the SS and IS tendons is not beneficial, as it does not provide any clinical benefit, be it in terms of overall subjective assessment, pain, range of movement or strength. Although this is merely an electromyographic finding, it is associated with measurable compromise of suprascapular nerve function in a substantial number of patients, so it should be considered a possible hazard without any clinical benefit.

It may be criticized that the technique of nerve release was not optimal: There are many techniques described to arthroscopically release the suprascapular nerve. In 2006, Bhatia et al presented their technique, which was then modified by Lafosse et al in 2007. Since then, multiple details have been modified and adapted, but the basic principles established by Bhatia and Lafosse have not changed. We have used the technique of Lafosse; the nerve was identified in all instances before and after its release and its structural integrity was verified after the

| Musculus infraspinatus | Goutallier | 0 | 1 | 2 | 3 | 4 | \( P \) value |
|------------------------|-----------|---|---|---|---|---|------------|
| Preoperative infraspinatus | | | | | | | |
| Release | 6 | 3 | 0 | 0 | 0 | .33 |
| Non-release | 5 | 3 | 2 | 0 | 0 | |
| 3-month infraspinatus | | | | | | | |
| Release | 3 | 6 | 0 | 0 | 0 | .10 |
| Non-release | 1 | 7 | 1 | 1 | 0 | |
| 12-month infraspinatus | | | | | | | |
| Release | 3 | 4 | 2 | 0 | 0 | .66 |
| Non-release | 2 | 6 | 1 | 1 | 0 | |

- **Table III**: Fatty infiltration of the infraspinatus muscle between the release and non-release groups

| ID | Release | NCS cMAP amplitude (mV) | Preop | 3 months | 12 months | EMG pathologic | 3 months | 12 months |
|----|---------|-------------------------|-------|----------|-----------|----------------|----------|----------|
| 1  | Yes     | 12.60                   | 1.44  | 5.40     | No        | Yes            | No       | No       |
| 2  | Yes     | 6.20                    | 12.30 | 14.70    | No        | Yes            | No       | No       |
| 3  | Yes     | 5.20                    | 9.70  | 6.10     | No        | No             | No       | No       |
| 4  | Yes     | 5.30                    | 5.20  | 5.60     | No        | No             | No       | No       |
| 5  | Yes     | 10.10                   | 3.00  | 2.20     | No        | Yes            | Yes      | Yes      |
| 6  | Yes     | 11.40                   | 0.58  | 4.90     | No        | No             | No       | No       |
| 7  | Yes     | 3.30                    | 6.70  | 1.78     | No        | No             | No       | No       |
| 8  | Yes     | 1.85                    | 0.76  | 2.20     | No        | No             | No       | No       |
| 9  | Yes     | 6.40                    | 6.90  | 4.00     | No        | No             | No       | No       |
| 10 | No      | 6.30                    | 14.00 | 18.00    | No        | No             | No       | No       |
| 11 | No      | 7.70                    | 17.00 | 7.70     | No        | No             | No       | No       |
| 12 | No      | 13.00                   | 7.70  | 5.80     | No        | No             | No       | No       |
| 13 | No      | 5.00                    | 8.40  | 13.70    | No        | No             | No       | No       |
| 14 | No      | 12.80                   | 9.30  | 5.10     | No        | No             | No       | No       |
| 15 | No      | 6.00                    | 11.80 | 10.10    | No        | No             | No       | No       |
| 16 | No      | 2.30                    | 7.40  | 5.10     | No        | No             | No       | No       |
| 17 | No      | 2.80                    | 11.20 | 3.60     | No        | No             | No       | No       |
| 18 | No      | 13.40                   | 11.30 | 11.60    | No        | No             | No       | No       |
| 19 | No      | 7.60                    | 13.10 | 8.60     | No        | No             | No       | No       |
| 20 | Yes     | 0.14                    | 0.82  | 7.0      | Yes       | Yes            | No       | No       |

- **Table IV**: NCS amplitude data and EMG categorization as derived from the infraspinatus muscle obtained preoperatively, and at 3 and 12 months postoperatively in the release and non-release groups

NCS, nerve conduction studies; EMG, electromyography; cMAP, compound muscle action potential. Postoperative reduction of cMAP amplitude of >50% (usually indicative of axonal nerve damage) was not in accordance with EMG signs of axonal damage except in ID20 who was excluded from randomization due to assumed preoperative suprascapular nerve damage.
release. Nonetheless, we did identify 3 of 9 cases with nerve dysfunction and are unable to discuss these findings with the literature because standardized pre- and postoperative EMG/NCS findings have not been reported and specifically the question whether a nerve release as a preventive operation could cause damage has never been assessed.

If this study appears to answer the question regarding the use of prophylactic nerve release in stages 1 and 2 of Goutallier changes, it does not answer 2 questions: first, the study does not answer the question whether the decompression is beneficial in the repair of larger, chronic tears of stages Goutallier 3 and 4 with more advanced musculotendinous retraction. Currently, however, the repair of tears with fatty infiltration stage 3 or even 4 has become very rare, as they are known to fail in a very high proportion of cases, so it would be very difficult to recruit the necessary patients for such a study at least in a monocentric setting. The second question to be answered is whether nerve decompression at the suprascapular notch is truly beneficial if suprascapular nerve changes are present preoperatively. We found only 1 of 20 patients who had a measurable nerve dysfunction. He had been randomized to nerve release, and the clinical and electromyographic outcomes were good with recovery of nerve function. We do not know whether the nerve release assisted in this recovery or whether the cuff repair alone would have been sufficient, and this may be a question of a further study.

It is incompletely understood how damage to the suprascapular nerve occurs in association with RC tear. One might theorize that traction injury due to medialization of the muscle and consecutive tethering of the nerve against the suprascapular notch explains the injury. In cadavers, a change of the angle between the suprascapular nerve and the motor branches to the SS muscle has been shown due to medial retraction of the SS muscle. The angle decreased from 142.6° without a tear to 98.7° with 1 cm medial retraction and 34.6° with 5 cm medial retraction of the SS muscle.

A possible theory regarding how the nerve can be damaged is traction due to lateralization during surgery. Some authors stated that a maximum lateralization of 1 cm would be safe. More than 1 study, however, has documented that a lateralization of 3-4 cm without damage to the nerve in EMG is possible.

A possible mechanism of damaging the nerve during suprascapular nerve release may be inadvertent exposure to damaging heat during radiofrequency-assisted dissection in the fat tissue above to the transverse ligament. In our experience, the application of this device may be required if the blood pressure is not or cannot be lowered, but this damage mechanism is hypothetical. Another possible explanation of transient occurrence of abnormal EMG in our study could be a blunt physical contact with the nerve during the release procedure. Whether these findings are unique to our cohort is unknown. In the literature, there are only a few studies with postoperative EMG studies at quite different time points. As the identified findings were not associated with pathologic clinical findings, this pathology is likely to be missed unless systematically looked for.

**Conclusion**

In the presence of normal preoperative EMG findings, suprascapular nerve release added to arthroscopic repair of small and medium-sized RC tears is not associated with any clinical benefit, but with electromyographically documented, postoperative impairment of nerve function in 1 of 3 cases in this study. Suprascapular nerve release appears therefore unjustified if preoperative EMG findings document normal suprascapular nerve function in small and midsize RC tears. Risks and benefit of suprascapular nerve release in larger tears or in the presence of preoperative impairment of suprascapular nerve function remain to be established.

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