Suprascapular nerve entrapment

Neurophysiological localization in 6 cases

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We report 6 cases of a suprascapular nerve lesion: 3 of them presented a nerve entrapment at the suprascapular notch and 3 at the spinoglenoid notch. Electrophysiological investigation localized the site of entrapment in all cases.

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Symptoms of entrapment of the suprascapular nerve at the suprascapular notch (SSN) are shooting shoulder pain and hypotrophy of the supraspinatus (SSM) and infraspinatus (ISM) muscles (Kopell and Thompson 1963). The lesion of the nerve is proximal to the sensory and motor branches innervating the acromioclavicular and glenohumeral joints, and the SSM and ISM (Figure 1). Ganzhorn et al. (1981) reported entrapment of the suprascapular nerve at the spinoglenoid notch (SGN). In this case, the entrapment affects terminal motor branches innervating the ISM, while sensory branches to the acromioclavicular and glenohumeral joints and motor branches to the SSM remain unaffected. Thus, SSM function is spared and shoulder pain is usually slight or absent (Figure 1).

Although many cases of suprascapular nerve lesions have been reported (Ferretti et al. 1987, Steiman 1988, Black and Lombardo 1990), the differential diagnosis between the two commonest entrapment locations has not been given much consideration. This may be one cause of the high rate of surgical failure (Clein 1975, Vastamäki and Göransson 1993).

We investigated electrophysiologically 6 patients with clinically suspected suprascapular nerve entrapment to locate the site of the lesion.

Patients and methods

We studied 5 men and 1 woman with a suspected suprascapular neuropathy: in all cases, physical, radiographic and neurophysiological evaluations were carried out. A follow-up was also performed in all cases but one.

Bilateral shoulder and arm muscle strength was assessed (BMRC scale). Special attention was paid to the evaluation of the SSM, ISM, trapezius, latissimus dorsi, deltoideus, biceps brachii and triceps brachii.

Neurophysiological evaluation was performed with a Medelec Sapphire 4ME electromyograph and included EMG of SSM, ISM and of muscles innervated by the C4-T1 roots, and distal motor latency (DML) of the SSM and ISM. EMG was performed with concentric needle electrodes. In studies of DML, supramaximal stimulation (0.1 ms duration) was delivered with a bipolar surface stimulator at Erb's point and potentials recorded by concentric needle electrodes from the SSM and ISM (normal values < 3.3 ms and < 4 ms, respectively (Gassel 1964).

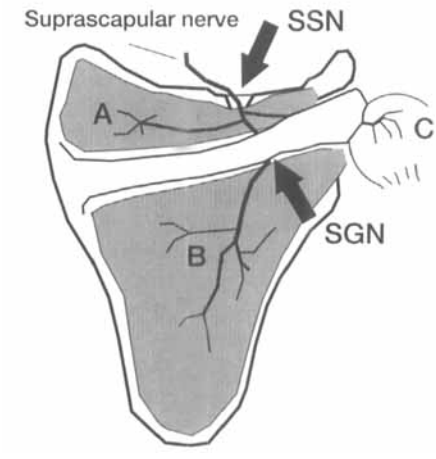

Figure 1. Schematic illustration of suprascapular nerve entrapment sites. A – supraspinatus muscle; B – infraspinatus muscle; C – humerus; SSN – suprascapular notch; and SGN – spinoglenoid notch

Table 1. Suprascapular nerve entrapment. Data in 6 cases

Patient data					Symptoms		Motor strength		EMG findings [a]		Distal motor latency (ms)		Location of lesion	Putative cause of lesion
	age	sex	side	activity	weak-ness	pain	SSM	ISM	SSM	ISM	SSM	ISM		
1	28	M	r	volley ball	no	no	5	3	f–, n–	f+, n+	2.9	6.2	SGN	microtrauma
2	21	M	r	tennis	yes	no	5	2	f–, n–	f–, n+	2.9	12	SGN	microtrauma
3	41	F	l	physiotherapist	no	no	5	3	f–, n–	f–, n+	2.4	6.2	SGN	arm hyperextension
4	23	M	l	military service	no	yes	3	3	f+, n+, r+	f+, n+, r+	15	24	SSN	weight lifting
5	23	M	r	student	yes	yes	3	3	f+, n+	f+, n+	8.9	12.2	SSN	motorbike accident
6	38	M	r	secretary	yes	yes	0	0	f+ no volunt. activity	f+ no volunt. activity	no motor response	no motor response	SSN	abnormal sleeping position

Radiographs showed an old fracture in the scapula of case 5, but were normal in all other cases
[a] EMG findings: f– no fibrillation, f+ fibrillation; n– normal recruitment, n+ neurogenic recruitment; r+ regeneration potentials

Case reports (Table 1)

Case 1

A volleyball player with severe hypotrophy and weakness of right ISM (Figure 2). Neurophysiological examination disclosed abnormal findings in the right ISM alone, suggesting a lesion of the right suprascapular nerve at the SGN, with signs of nerve regeneration. Sport activity was discontinued. 6 months later, a slight clinical improvement of right ISM function was noted with a decrease of DML of the ISM (5.6 ms), but the patient complained of shoulder pain due to bursitis or tendinitis.

Case 2

A tennis player complaining about progressive difficulties during volleys, but with no shoulder pain since half a year. He had a severe hypotrophy and weakness of the right ISM. The electrophysiological studies suggested a lesion of the right suprascapular nerve at the SGN. 3 months later, after reduction of agonistic activity, the motor function of the right ISM had improved (improvement in EMG recruitment and decrease of DML to 9 ms); however, the patient had point shoulder pain when making a serve.

Figure 2. Case 1, volleyball player: the atrophy of the right ISM is evident, while SSM is not affected.

Case 3

The patient had hypotrophy in the left scapular region without pain, following a trauma with the left arm in hyperextension some time before. He had hypotrophy and weakness of the left ISM and the neurophysiological evaluation confirmed a lesion of the suprascapular nerve at the SGN. 2 years later, the clinical and neurophysiological findings were unchanged.

Case 4

The patient had atrophy of the left scapular region after a shoulder trauma with transitory shoulder pain during weightlifting 2 years before. He had marked hypotrophy and weakness of the left SSM and ISM. The neurophysiological studies confirmed the involvement of both SSM and ISM, suggesting a lesion of the suprascapular nerve at SSN. 5 months later, improvements in SSM and ISM function were observed (DML = 8.5 ms and 13.5 ms, respectively).

Case 5

6 months after a motorbike accident, the patient had decreased external rotation of the right shoulder accompanied by marked shoulder pain. He had hypotrophy and weakness of the right SSM and ISM (Figure 3). Neurophysiological data indicated a lesion of the right suprascapular nerve at the SSN and a surgical exploration was recommended, but the patient refused the operation. 2 years later, the clinical and neurophysiological findings were unchanged.

Case 6

The patient had proximal weakness of the right arm and shoulder pain. 1 month before the onset of symp-

Figure 3. Case 5, motorbike accident: the right ISM appears markedly atrophic. A minor degree of atrophy of the ipsilateral SSM is also evident.

toms, the patient slept in his car in a sitting position. He also had a history of an episode of neuralgic amyotrophy of right deltoid at the age of 18. He had paralysis of the right SSM and ISM. Neurophysiological evaluation indicated a complete lesion of the right suprascapular nerve at the SSN. The patient was lost to follow-up.

Discussion

The most commonly reported site of entrapment of the suprascapular nerve is the SSN (Kopell and Thompson 1963, Post and Mayer 1986, Kiss and Komar 1990, Callahan et al. 1991). This entrapment neuropathy should be considered in the differential diagnosis of chronic shoulder pain. In a series of 2520 patients with painful shoulder symptoms, the incidence of suprascapular nerve entrapment was 0.4% (10 cases) (Post and Mayer 1987). The diagnosis can be confirmed by an EMG showing signs of denervation of the SSM and ISM, with or without deficit of external rotation and abduction of the shoulder. Suprascapular nerve compression at the SSN may result from acute direct shoulder trauma, fracture, encroachment by intrinsic or extrinsic factors such as a tumor. More often it is classified as "idiopathic" (Rengachary et al. 1979, Post and Mayer 1987).

It appears possible that the frequency of a suprascapular nerve lesion is underestimated, as shoulder pain is considered by some authors the most important criterion in the diagnosis of suprascapular neuropathy (Post and Mayer 1987, Callahan et al. 1991). In our series, 3 patients did not complain of sensory symptoms or shoulder motor deficit, but came to our observation when they noted hypotrophy of the scapular region alone.

The localization of the entrapment site in suprascapular neuropathy is essential in planning the treatment. Data suggest a high sensitivity and specificity of electrophysiology in the diagnosis of suprascapular neuropathy (Ringel et al. 1990). In all our cases, the electrophysiological studies allowed us to confirm the diagnosis suspected on the basis of the clinical findings. Examination of the SSM is essential to distinguish between SSN and SGN nerve compression (Liveson et al. 1991). Moreover, electrophysiological studies allowed evaluation of the course of the lesion.

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