

Muscle biopsy investigations on neuromuscular insufficiency of the rotator cuff: A contribution to the functional impingement of the shoulder joint

Ulrich Irlenbusch, MD, and Heike-Kathrin Gansen, MD, Arnstadt, Germany

The possibility that functional impingement results from muscular imbalance has been discussed. This study investigates whether disturbance of muscular coordination is reflected in a shift in the balance of fast-twitch and slow-twitch fibers, as fast-twitch fibers enable a rapid reaction or contraction and slow-twitch fibers enable slow contractions and sustained performance. The supraspinatus and deltoid muscles in 37 patients with a supraspinatus syndrome and partial or complete rupture of the rotator cuff underwent biopsy. All three groups of patients showed a reduction in size, increased variability, and change in the frequency distribution of fiber cross sections. The fast-twitch fibers were especially affected. These findings were confirmed objectively by morphometric measurements. It might be concluded from the changes mentioned above that disturbance in coordination of the musculature can cause a functional impingement. We term this neuromuscular insufficiency of the rotator cuff. The mechanical factors relating to subacromial impingement may only have a role in promoting its manifestation by restricting the compensatory range of the joint. (J Shoulder Elbow Surg 2003;12:422-6.)

The studies of Neer²² focused on subacromial impingement instead of the inflammatory pathogenesis described by Duplay (1872).⁶ This simple and graphic concept mainly led to a mechanistic view of the problem.

In recent years interest has increasingly concentrated on the neuromuscular system. Muscular imbalance, instability impingement, and overuse are now viewed as factors in the pathogenesis of the impingement syndrome.^{2,4,10,17,21,23}

From the Department of Orthopedic Surgery, Orthopädische Klinik des Marienstifts Arnstadt, Arnstadt, Germany.

Reprint requests: Ulrich Irlenbusch, MD, Orthopädische Klinik des Marienstifts Arnstadt, Wachsenburgallee 12, D-99130, Arnstadt, Germany. (E-mail: irlenbusch@ms-arn.de).

Copyright © 2003 by Journal of Shoulder and Elbow Surgery Board of Trustees.

1058-2746/2003/\$35.00 + 0

doi:10.1016/S1058-2746(03)00036-3

Nirschl²³ pointed out that recurrent overloading of the supraspinatus muscle might lead to functional impingement. In addition, other authors did not relate the impingement syndrome to osseous narrowing. They discussed it as a superior migration of the humerus resulting from insufficiency of the rotator cuff (ie, muscular imbalance).^{2,5,18}

The importance of the neuromuscular system for motion of the shoulder joint has likewise been described in various reports dealing with proprioception.^{7,12,13,28} In particular, studies on athletes performing throwing or overhead sports led to the concept of secondary or non-outlet impingement.^{15-17,32}

Until now, functional disorders have not been adequately investigated at the muscular level. We decided to investigate this problem, drawing on experience in neuromuscular disorders in the knee joint and spine through muscle biopsy^{25,26} and an earlier investigation by Irlenbusch and Pieper¹¹ on the shoulder joint.

In striated muscle two fiber types are distinguished.^{9,25,26,33} These can be demonstrated with different frequency and distribution in the phasic and tonic muscles. Slow-twitch (ST) fibers (type I) are slowly contracting structures that are resistant to fatigue. Fast-twitch (FT) fibers (type II) are rapidly contracting elements that fatigue quickly. FT fibers are able to produce brief sequences of explosive contraction. For this reason, they are of particular importance for rapid fine adjustment of musculature to stabilize joints.

The objective of this study is to establish the morphologic changes in the supraspinatus muscle and in the acromial part of the deltoid muscle in patients with a supraspinatus syndrome and a partial or complete supraspinatus rupture. Conclusions relating to the pathogenesis may be possible from changes in the fiber distribution pattern of the ST and FT fibers with progression of the disease.

MATERIALS AND METHODS

Samples were taken exclusively by an open technique because of strict criteria for biopsy.^{14,29} In this way, arti-

facts from the accumulation of fluid during arthroscopy were also avoided.³⁰

All patients with comorbidities such as myopathies, neurologic diseases, chronic polyarthritis, and long-term steroid intake were excluded. Taking biopsy specimens was associated with different logistic problems (refrigerated transport to the laboratory is required immediately after surgery). For these reasons, biopsy specimens were taken in only 58 of 142 patients.

The requested quality specifications for biopsy^{14,29} resulted in a further reduction to 37 samples that could be evaluated. The following criteria had to be fulfilled: no artificial damage, muscle fibers cut exactly orthograde, greater extensive and continuous parenchymatous tissue, and clear contours of the individual muscle fiber cross sections. Especially in cases of long-standing ruptures, technically perfect preparations could not be obtained because of marked retraction of the tendon stump and thus poor accessibility of the muscle. In other cases fatty degeneration of the muscle was far too advanced to permit evaluation.^{8,20} In these cases the biopsy specimens were excluded.

A sample 3 × 3 × 5 mm in size was taken from the anterior portion of the acromial part of the deltoid muscle (surgical access between the clavicular and acromial part of the deltoid muscle) as well as from the supraspinatus muscle about 1.5 to 2 cm medial to the musculotendinous junction. Details of the biopsy, fixation, and histologic techniques of the material are given in the references mentioned above. The most important steps were freezing the biopsy specimens in liquid nitrogen and preparing 8- μ m thin crosscut sections with the cryostat at -9.4°F (-23°C).

In addition to hematoxylin-eosin and Gomori trichrome staining, the nicotinamide adenine dinucleotide dehydrogenase reaction was used. It enables differentiation of muscle fiber types. FT fibers were stained bright blue, and ST fibers were stained deep blue/black.

For performance of measurements, all samples were photographed and paper prints were made with a final enlargement of 1:125. The fiber cross sections were later determined with a semiautomatic image analysis system. After manual marking of the areas, their contents could be calculated automatically.

Age and sex distribution, right- or left-handedness, affected side, and disease duration before surgery in the 37 patients evaluated are shown in Table I.

RESULTS

The histologic samples showed greater changes for FT fibers than for ST fibers. With increasing progression of the clinical picture, from the supraspinatus syndrome to partial to complete rupture, a reduction in fiber diameter and a relative shift in the frequency distribution of FT and ST fibers were observed. A greater variability in fiber diameter was especially conspicuous (Figure 1).

The observations were confirmed objectively by morphometric investigations. Figure 2 shows the histograms for the supraspinatus and deltoid muscles. The average areas of FT and ST fibers for the three

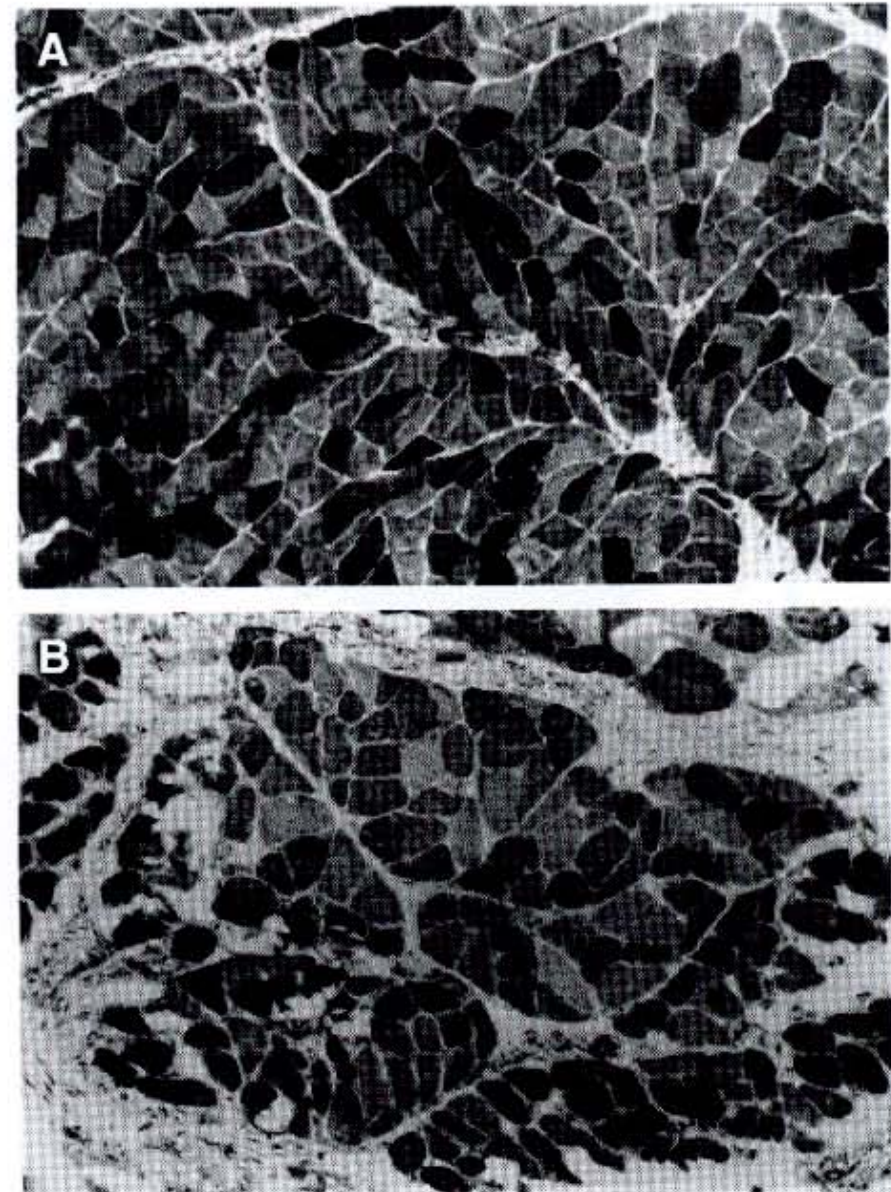


Figure 1 Sections through the supraspinatus muscle in a patient with supraspinatus syndrome (**A**) and in a patient with complete rotator cuff rupture (**B**) (magnification proportion 1:125, nicotinamide adenine dinucleotide staining); FT fibers (type II) appear light and ST fibers (type I) dark. **A**, A case of supraspinatus syndrome showing normal distribution of the fiber types with a mild caliber variation and a decrease in fiber diameter for single fibers. **B**, A case of complete rotator cuff rupture. In contrast to **A**, marked caliber variations are shown in both ST and FT fibers, in addition to perivascular atrophy and intermuscular fibromatosis.

diagnostic groups are presented separately for each muscle. On the basis of the investigations of Pongratz,²⁷ a Gauss distribution is to be expected for healthy probands.

For the deltoid muscle, an approximately normal distribution of ST fibers was found in all diagnosis groups. On the other hand, there was a left shift in the case of the supraspinatus muscle, corresponding to a decrease in fiber diameter. There was a deviation from the Gauss distribution, corresponding to a higher variability in supraspinatus syndrome. On the whole, the changes were more pronounced than in the deltoid muscle.

A flattening and broadening of the histogram with several peaks occurs in the early phase in FT fibers of the deltoid muscle (ie, in supraspinatus syndrome).

The changes in FT fibers are more pronounced in the supraspinatus muscle. In all diagnosis groups,

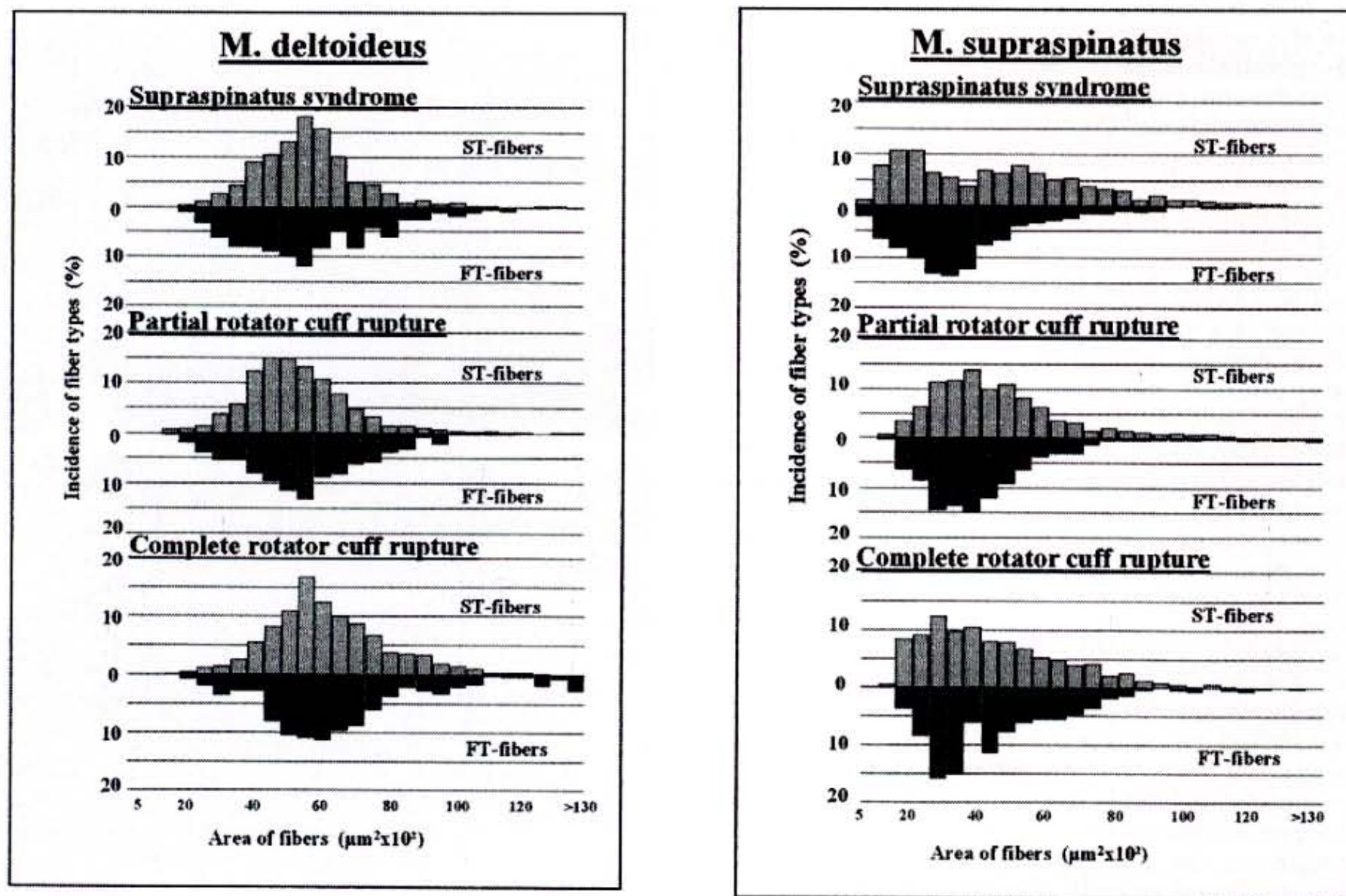


Figure 2 Histogram of the fiber cross sections of FT and ST fibers for the deltoid muscle and the supraspinatus muscle for the three investigation groups: supraspinatus syndrome, partial rotator cuff rupture, and complete rotator cuff rupture.

Table 1 Age and sex distribution, affected side, right or left handedness, and duration of disease prior to the operation in the three disease groups

	Supraspinatus syndrome	Partial rotator cuff rupture	Complete rotator cuff rupture	Total
Number	11	16	10	37
Mean age in years	49	47	54	49 (Min. 24, Max. 68)
Sex				
male	6	11	8	25
female	5	5	2	12
Affected side				
right	6	10	9	25
left	5	6	1	12
Handedness				
right	11	15	9	35
left	0	1	1	2
Duration of disease prior to the operation in months	19	7	9	11

there was a distinct left shift, and in complete rupture, this was combined with a multipeak histogram. This was interpreted as a manifestation of the increased

variability already described (ie, simultaneous hypotrophy and hypertrophy).

In summary, the variations from the norm were

present in an early stage of the disease. They were greater in FT fibers than in ST fibers. Furthermore, more pronounced changes were found in the supraspinatus muscle in comparison with the deltoid. Finally, a greater displacement to the left and greater variability of fiber cross section were found in rotator cuff rupture than in supraspinatus syndrome and in partial ruptures.

DISCUSSION

This study demonstrates a disturbance of the muscle fiber distribution in the deltoid and supraspinatus muscles (especially in FT fibers) even at an early stage of the disease—the supraspinatus syndrome. We interpret this as a primary disturbance of muscular coordination, as FT fibers are responsible for a rapid reaction and thus the fine motor control of the joint. Functional impingement develops as a consequence of this imbalance.

Our results suggest that a large number of subacromial syndromes have a functional origin. As a result of this hypothesis, we generally speak of neuromuscular insufficiency of the rotator cuff or of the shoulder girdle in this stage of disease. The known anatomic variations of the subacromial space (mechanical or outlet impingement)^{1,3,5,18,19,24,31} would, therefore, impair the ability to compensate.

The two most important factors that may interfere with a neuromuscular mechanism are the reduction of rotator cuff muscle strength and loss of precision in coordination. In part, these changes can be seen more precisely as an age-dependent reduction in fitness. With increasing age, there is a decrease in functioning motor units and a prolongation in the latency of monosynaptic reflexes. In the muscle system, the FT fibers responsible for fine motor control and rapid reaction are reduced in number.

In the shoulder girdle various disturbances of the neuromuscular system are possible. In addition to age-related changes, other disturbances such as a cervical syndrome, whiplash trauma (acceleration trauma of the cervical spine), thoracic outlet syndrome, or unilateral loading imbalances interfere with shoulder motion. However, until now, we have not been able to determine whether the muscular changes are the cause or the consequence of the tendon lesions.

Because we only examined the acromial part of the deltoid muscle and the supraspinatus muscle, we must limit our interpretation to these two muscles. Further investigation is required to clarify whether muscular imbalance also affects other muscles of the rotator cuff as well as the extrinsic muscles of the shoulder girdle.

This study confirms the results of several earlier publications that considered the functional components of the impingement syndrome so that therapy in

the early stages of the impingement syndrome should focus on physiotherapy rather than decompression the subacromial space. Therefore, more attention should be given to training of muscle coordination and proprioception, especially in the early stages of the disease as well as after surgery.

So that our hypothesis of a neuromuscular insufficiency of the rotator cuff can be elucidated, the disturbances of muscular coordination should be measured with noninvasive methods. Various investigatory techniques could be used in order to clarify the role of exogenous impingement or instability impingement on the one hand and muscular imbalances on the other.

REFERENCES

1. Banas MP, Miller RJ, Totterman S. Relationship between the lateral acromion angle and rotator cuff disease. *J Shoulder Elbow Surg* 1995;4:454-61.
2. Beach WR, Caspari RB. Arthroscopic management of rotator cuff disease. *Orthopedics* 1993;16:1007-15.
3. Bigliani LU. Impingement syndrome: aetiology and overview. In: Watson MS, editor. *Surgical disorders of the shoulder*. Edinburgh: Churchill Livingstone; 1991. p. 237-45.
4. Davies GJ, Dickhoff-Holtmann S. Neuromuscular testing and rehabilitation of the shoulder complex. *J Orthop Sports Phys Ther* 1993;2:449-58.
5. Deutsch A, Alichek DW, Schwartz E, Otis JC, Warren RF. Radiologic measurement of superior displacement of the humeral head in the impingement syndrome. *J Shoulder Elbow Surg* 1996;5:186-93.
6. Duplay S. De la péri-arthritis scapulo-humérale et des raideurs de l'épaule qui en sont la conséquence. *Arch Gen Med* 1872;20:513-42.
7. Gohlke F, Janssen E, Leidel J, Heppelmann B, Eulert J. Histopathological findings in the proprioception of the shoulder joint. *Orthopäde* 1998;27:510-7 [in German].
8. Goutallier D, Postel JM, Bernageau J, Lavau L, Voisin MC. Fatty muscle degeneration in cuff ruptures. *Clin Orthop* 1994;304:78-83.
9. Howald H. Morphologic and functional changes of muscle fibers during training. *Schweiz Z Sportmed* 1984;31:5-14 [in German].
10. Howell SM, Imobersteg M, Seger DH, Marone PJ. Clarification of the role of the supraspinatus muscle in shoulder function. *J Bone Joint Surg Am* 1986;68:398-404.
11. Irlenbusch U, Pieper KS. Muskelbiopsische Untersuchungen am Schultergelenk—ein Beitrag zur Pathogenese der sogenannten Periarthritis humeroscapularis. *Orthop Praxis* 1992;28:529-32.
12. Jerosch J, Steinbeck J, Schröder M, Wethues M, Reer R. Intraoperative EMG response of the musculature after stimulation of the glenohumeral joint capsule. *Acta Orthop Belg* 1997;63:8-14.
13. Jerosch J, Thorwesten L, Steinbeck J, Reer R. Proprioceptive function of the shoulder girdle in healthy volunteers. *Knee Surg Sports Traumatol Arthrosc* 1996;3:219-25.
14. Jerusalem F, Zierz S. *Muskelerkrankungen*. Stuttgart: Georg Thieme; 1991.
15. Jobe C. Oberer glenohumeraler Anschlag (superior glenoid impingement) beim Wurfsporler. In: Eulert J, Hedtmann A, editors. *Das Impingement-Syndrom der Schulter*. Stuttgart: Georg Thieme; 1996. p. 36-41.
16. Kvitne RS, Jobe FW, Jobe CM. Shoulder instability in the overhand or throwing athlete. *Clin Sports Med* 1995;14:917-35.
17. Kvitne RS, Jobe FW. The diagnosis and treatment of anterior

- instability in the throwing athlete. *Clin Orthop* 1993;291:107-23.
18. Lochmüller EM, Anetzberger H, Maier U, Habermeyer P, Müller-Gerbl M. Acromio-humeral distance and acromial shape in 3-dimensional computerized tomography reconstruction. Side comparison in supraspinatus outlet syndrome. *Unfallchirurg* 1997;100:874-9 [in German].
 19. Luo ZP, Hsu HC, Grabowski JJ, Morrey BF, An KN. Mechanical environment associated with rotator cuff tears. *J Shoulder Elbow Surg* 1998;7:616-20.
 20. Nakagaki K, Ozaki J, Tomita Y, Tamai S. Fatty degeneration in the supraspinatus muscle after rotator cuff tear. *J Shoulder Elbow Surg* 1996;5:194-200.
 21. Nakajima T, Rokuuma N, Hamada K, Tomatsu T, Fukuda H. Histologic and biomechanical characteristics of the supraspinatus tendon: reference to rotator cuff tearing. *J Shoulder Elbow Surg* 1994;3:79-87.
 22. Neer CS. Anterior acromioplasty for the chronic impingement of the shoulder. *J Bone Joint Surg Am* 1972;54:41-50.
 23. Nirschl RP. Rotator cuff tendinitis: basic concepts of pathoetiology. *Instr Course Lect* 1989;28:439-45.
 24. Patel VR, Singh D, Calvert PT, Bayley JI. Arthroscopic subacromial decompression: results and factors affecting outcome. *J Shoulder Elbow Surg* 1999;8:231-7.
 25. Pieper KS, Schmidt H, Bähr B, et al. Morphological and biochemical changes in the pars obliqua of the vastus medialis muscle in degenerative disorders of the knee joint. *Acta Histochem* 1986;80:183-90 [in German].
 26. Pieper KS, Bähr B, Biskop M, Paul B, Gärtner C, Förster E. Functional and structural changes in m. vastus lateralis and m. vastus medialis in cases of meniscopathy and chondropathia patellae. *Biomed Biochim Acta* 1986;45:S119-24.
 27. Pongratz D. Differentialdiagnose der Erkrankungen der Skelettmuskulatur an Hand von Muskelbiopsien. In: Scheid W, Wieck HH, Peters UH, editors. *Sammlung psychiatrischer und neurologischer Einzeldarstellungen*. Stuttgart: Georg Thieme; 1976. p. 1-107.
 28. Salomonow M, Guanche C, Wink C, et al. Mechanoreceptors and reflex arc in the feline shoulder. *J Shoulder Elbow Surg* 1996;5:139-46.
 29. Schröder JM. Pathologie der Muskulatur. In: Doerr W, Seifert G, Uehlinger E, editors. *Spezielle pathologische Anatomie*. Vol 15. Berlin: Springer Verlag; 1982:6-70.
 30. Sperber A, Wredmark T. Intramuscular pressure and fluid absorption during arthroscopic acromioplasty. *J Shoulder Elbow Surg* 1999;8:414-18.
 31. Uthoff HK, Hammond DI, Sarkar K, Hooper GJ, Papoff WJ. The role of coracoacromial ligament in the impingement syndrome: a clinical, radiological and histological study. *Int Orthop* 1988; 12:97-104.
 32. Werner A, Gohlke F. Impingement—Symptomatik des Sportlers. In: Euler J, Hedtmann A, editors. *Das Impingement-Syndrom der Schulter*. Stuttgart: Georg Thieme; 1996. p. 26-35.
 33. Ziegler J, Dippold A. Charakteristische morphologische Veränderungen des M. vastus medialis bei Gonarthrose. *Beitr Orthop Traumatol* 1985;32:26-29.