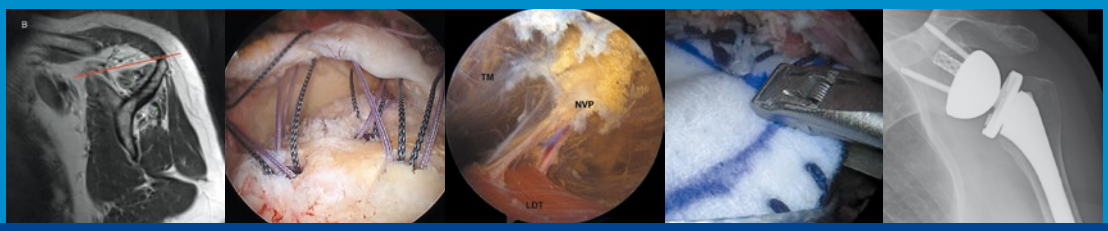
Nuno Sampaio Gomes · Ladislav Kovačič
Frank Martetschläger · Giuseppe Milano
Editors



Massive and Irreparable Rotator Cuff Tears

From Basic Science to Advanced Treatments

European Society of Sports Traumatology, Knee Surgery & Arthroscopy
ESSKA

Springer

Massive and Irreparable Rotator Cuff Tears

Nuno Sampaio Gomes
Ladislav Kovačič
Frank Martetschläger
Giuseppe Milano
Editors

Massive and Irreparable Rotator Cuff Tears

From Basic Science to Advanced Treatments

Springer

European Society of Sports Traumatology, Knee Surgery & Arthroscopy
ESSKA

Editors
Nuno Sampaio Gomes
Hospital das Forças Armadas
and Hospital da Luz
Porto
Portugal

Ladislav Kovačič
Department of Traumatology
University Medical Centre Ljubljana
Ljubljana
Slovenia

Frank Martetschläger
ATOS Klinik München
Deutsches Schulterzentrum
München
Germany

Giuseppe Milano
Department of Orthopaedics
University of Brescia
Brescia
Italy

ISBN 978-3-662-61161-6 ISBN 978-3-662-61162-3 (eBook)
https://doi.org/10.1007/978-3-662-61162-3

“To my two sons, André and Simão.” (NG)

“To my dear Eva. Thank you for your endless love and dedication to our family. Sharing the path of life together is a wonderful, joyful, and fulfilling experience.” (LK)

“To Andrea and Aaron Julius. To my parents Alfred and Irmtrud and my sister Silke.” (FM)

“To Virginia, Edoardo and Costanza. I wish you could live the life you’ve imagined in your endless imaginary world!” (GM)

Foreword

It is a pleasure to introduce this well done and much needed text on the rotator cuff. My friends and colleagues Drs. Gomes, Kovačič, Martetschlager, and Milano are all internationally recognized experts in the management of disorders of the rotator cuff. Injuries to the shoulder are quite common, but unfortunately our patients often delay treatment until the shoulder becomes severely dysfunctional.

The severely dysfunctional shoulder is usually the result of chronic, displaced, massive rotator cuff pathology. These troubled patients have become all too common, thus requiring significant improvement in management techniques. This textbook by these excellent surgeons should help our patients by improving diagnosis, planning and surgical techniques.

In short, this represents an excellent treatise on difficult problems. I think it is a must-read for any surgeon treating chronic shoulder issues.

New Orleans, LA, USA — Felix H. "Buddy" Savoie III

Preface

As some of the readers of this book may well remember, the array of common surgical procedures performed on the shoulder at the early stages of their practice as surgeons was considerably less vast than it is today. With the advent of arthroscopy, a whole new universe of possibilities was disclosed, not only because new diagnosis and successful treatment techniques were described but also because keyhole surgery, due to its inherent appeal, seduced many of us to eagerly contribute to its development over the past years.

Scientific knowledge of the shoulder joint is, therefore, profound these days. The European Shoulder Associates (ESA) of ESSKA, gathering many of the current international leading shoulder surgeons, is a good example of the dynamism and strength of the scientific and technical evolution that exists today on this matter. Its biennial meeting in 2019 was, for the first time, organized as a joint event with the other sections of ESSKA—the Speciality Days—in a brand new format that proved to be a success. Especially for ESA, awarded the Best Section Performance prize of the meeting! It was held in Madrid on November 2019, focusing on a topic that continues to impel many shoulder surgeons towards the best possible solutions for a frequent shoulder problem, the massive rotator cuff tear.

The scientific chairs are proud to present this monograph, based on the same theme they explored at the Speciality Days. Besides the contribution of the meeting participants, other related topics are also covered in this book that include the role of both conservative and surgical management, from new biological options to all potential surgical techniques. In addition, the value of anesthesia and regional blocks, as well as guidance on the management of treatment complications and failures, are also included.

We are thankful to the ESSKA Board for supporting this project and to Springer for their high professionalism.

Porto, Portugal — Nuno Sampaio Gomes

Contents

Part I

Basic Science

Massive and Irreparable Rotator Cuff Tears: Defining the Problem

1

Lukas N. Muench, Felix Dyrna, and Knut Beitzel

1.1 Introduction

Massive and irreparable rotator cuff tears remain a major challenge in shoulder surgery [1]. Due to pain, loss of range of motion, and insufficient function, these tears significantly affect the patients' quality of daily living [1]. Representing up to 40% of all rotator cuff tears, massive tears are associated with persistent defects and poorer clinical outcomes [2, 3]. Imbalance of the force couples results in unstable kinematics of the glenohumeral joint, causing the remaining shoulder function to be sustained by a significantly increased compensatory deltoid force [4, 5].

This article tries to provide a structured overview about the biological challenges and biomechanical consequences of massive irreparable rotator cuff tears, as understanding of these factors is essential to initiate a differentiated therapeutic approach. Beginning with the different existing classification systems as well as initiation and progression of massive rotator cuff tears, the authors try to outline successively the biological problems including healing potential and tissue degeneration, followed by the main biomechanical problems. These mainly comprise the effects on tractive forces, shoulder function, glenohumeral joint centering and the development of osteoarthritis. In clinical practice, all of these factors have to be considered, in order to achieve satisfactory improvement in functional outcomes.

1.2 Structural Problem

1.2.1 Classifications of Massive Rotator Cuff Tears

Massive rotator cuff tears can be characterized by size, chronicity, and location. Regarding the tear size, different definitions exist. DeOrio and Cofield [6] defined massive tears as those whose greatest diameter exceeds 5 cm. Contrarily, Gerber et al. [7] characterized massive tears as those including complete tears of at least two tendons. This definition may show a more consistent correlation to the patients' function, prognosis, and outcome [5, 7, 8]. Considering chronicity, massive tears can be classified as acute, acute-on-chronic, and chronic tears [5]. Acute tears are relatively rare, commonly occurring

L. N. Muench
Department of Orthopaedic Sports Medicine, Technical University, Munich, Germany

Department of Orthopaedic Surgery, University of Connecticut, Farmington, CT, USA
e-mail: lukas.muench@tum.de

F. Dyrna
Department of Trauma, Hand and Reconstructive Surgery, University of Münster, Münster, Germany

K. Beitzel (✉)
Department of Orthopaedic Sports Medicine, Technical University, Munich, Germany

Department of Shoulder Surgery, ATOS Clinic, Cologne, Germany

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_1

after a traumatic event in younger patients [5]. In contrast, chronic massive tears are mostly observed in the elderly [5]. Regarding location, massive tears mostly follow the two distinct patterns of anterosuperior and posterosuperior tears [5].

1.2.2 Tear Initiation and Progression

Previous studies have proposed that degenerative rotator cuff tears start with the supraspinatus tendon, typically initiating at the anterior part of the humeral insertion near the biceps tendon, and propagate posteriorly over time [9–12]. In contrast, more recent studies have found that full-thickness as well as partial-thickness rotator cuff tears most commonly initiate at a location approximately 10–15 mm posterior to the biceps tendon, and may even begin with the infraspinatus tendon [10, 11, 13, 14]. An explanation for this finding is based on the "rotator crescent" concept, first described by Burkhart et al. [11, 15]. The rotator crescent is a term describing the thin, crescent-shaped rotator cuff sheet, which spans from the biceps tendon to the inferior border of the infraspinatus tendon, and is bound proximally by an arch-shaped thick bundle of fibres, called the "rotator cable" [15]. The rotator cable preserves the rotator crescent from stress through a "suspension bridge" configuration [15].

As people age, relative avascularity may lead to progressive thinning of the crescent, thus increasing dependence on the rotator cable [11, 15]. The location found by Kim et al. [11] 15 mm posterior to the biceps tendon is approximately at the center of the rotator crescent [15]. However, a recent MRI study located the initial tear site 5 mm more anterior (9–10 mm posterior to the biceps tendon) than as described by Kim et al. [10, 11]. This leads to the assumption that tears might propagate in both anterior and posterior directions [10]. Given the fact that the supraspinatus footprint is much smaller than previously believed, this location may be regarded as either the junction between the supraspinatus and infraspinatus, or being purely within the infraspinatus tendon [11, 16].

As torn tendons cannot participate in load distribution, the increasing tensile load on the remaining fibres can easily lead to tear propagation, particularly if the remaining tendon is of poor quality [17].

1.3 Biological Problem

1.3.1 Healing

The tear size can directly affect the clinical outcome and tendon healing [18–20]. A series of arthroscopic rotator cuff repairs have demonstrated that postoperative healing usually occurs between 71 and 89% of cases [19, 20]. However, this rate of tendon healing may drop to 47 or 50% in the treatment of massive rotator cuff tears [19, 20]. Even though hypovascularity has been hypothesized to facilitate tear initiation and limit biological healing after repair, the complexity of the healing process has not been fully understood [21].

The cells contributing to natural tendon healing originate from loose connective tissue surrounding the tendon fascicles and tendon body [22]. In response to the injury, these cells proliferate and migrate toward the tear site where they form collagenous healing tissue [22–24]. As the endogenous healing potential of the tendon seems to be limited, biologic augmentation techniques have recently garnered more and more attention, including the application of growth factors, platelet concentrates, or mesenchymal stem cells (MSCs) [25, 26]. Despite bone marrow being the traditional source for MSCs for biologic augmentation of tendon injuries, recent studies have highlighted subacromial bursal tissue being a source of MSCs, demonstrating superior proliferation potential, tissue engraftment, and survival [22, 26–29].

1.3.2 Atrophy, Fatty Infiltration, Retraction, and Loss of Elasticity

In addition to tear propagation, the process of atrophy, fibrosis, and fatty infiltration may occur in the rotator cuff tendon, as well as in the associated muscle belly over time (Fig. 1.1) [7, 20,

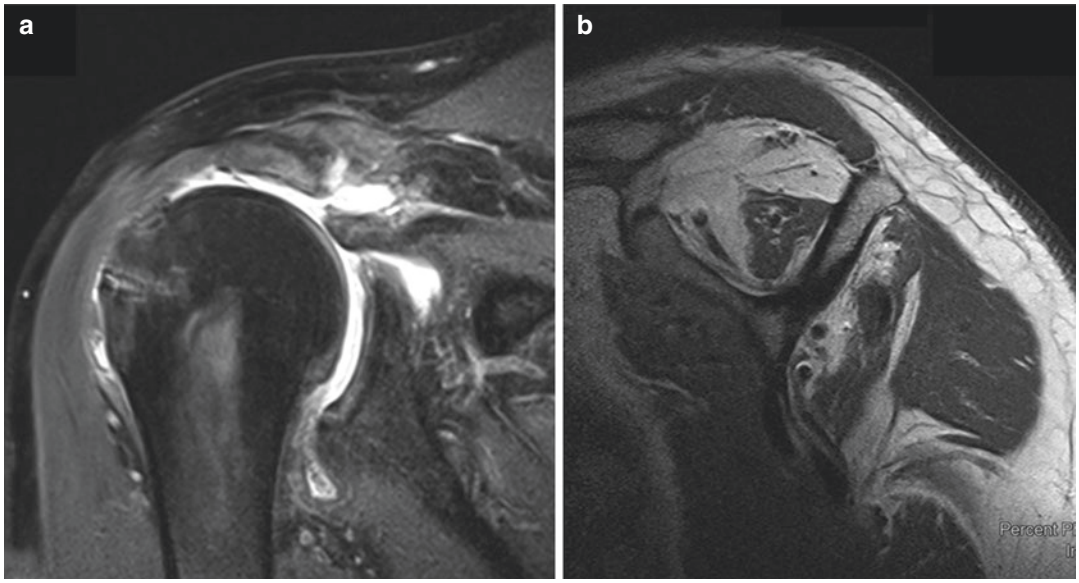


Fig. 1.1 MRI scan demonstrating a massive re-tear of the rotator cuff tendons with retraction, atrophy, and fatty infiltration. (**a**) Coronal view and (**b**) sagittal view

30]. Moreover, these tears often cause the tissue to become less compliant and stiffer [20, 30]. Particularly, in combination with tissue atrophy or fatty infiltration, this may result in severe tendon retraction [20, 30]. A widely retracted tendon margin coupled with poor tissue quality makes surgical mobilization difficult and sometimes impossible (Fig. 1.2) [20].

Muscle atrophy and fatty infiltration have been reported to be independent factors predicting outcomes and success rate after rotator cuff repair [31]. As tears of the rotator cuff result in mechanical unloading and denervation due to suprascapular nerve injury, consistent pathological changes can occur in the muscles' myotubes [32–34]. This may lead to alterations in the central molecular pathways, which regulate muscle atrophy and hypertrophy through mechanical load signaling [32–34].

A cell subpopulation of interstitial pluripotent stem cells, named fibro-adipo-progenitor cells (FAPs) and resident in muscle tissue, has been identified to be the cellular source of fatty infiltration [35, 36]. As shown in a mouse model, FAPs proliferate and differentiate into cells primarily expressing fat genes and cellular markers of adipogenesis, after inducing cuff injury [35, 36].

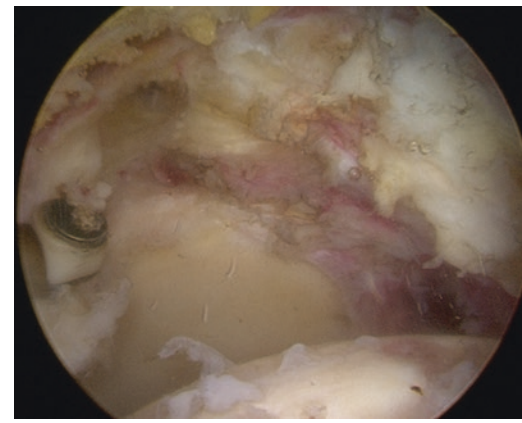


Fig. 1.2 Arthroscopic view of a large, retracted massive rotator cuff tear with concomitant tissue degeneration

Large, retracted tears have also been shown to cause traction on the suprascapular nerve, and may contribute to the progression of atrophy and fatty infiltration of the supraspinatus and infraspinatus muscles [17]. Moreover, the tear initiation location found by Kim et al. [11] may explain why fatty degeneration of the infraspinatus is seen in some patients with a presumed isolated tear of the supraspinatus tendon, highlighting the need to assess its integrity.

1.4 Biomechanical Problem

1.4.1 Tractive Forces

Coordinated action between the rotator cuff and deltoid muscles is essential for a sufficient glenohumeral abduction motion [37]. As the anterior and middle deltoid show preferential muscle activity and loading from 30° to 90° of glenohumeral abduction, the supraspinatus is the dominant muscle during the first 30°, and therefore the main initiator of abduction [37, 38]. Rotator cuff tears may lead to kinematic alterations, potentially causing a significant change in the biomechanical synergy between deltoid and rotator cuff muscles [39]. As the cuff tear size propagates posteriorly, considerably greater amounts of force are placed upon the middle portion of the deltoid, showing a major increase between 10° and 45° of abduction [4, 40, 41]. At the same time, the mechanical advantage of the deltoid may be disrupted due to loss of balanced concavity-compression and superior translation caused by tear progression [4]. This results in greater forces required to maintain joint stability and decreased abduction capability [4, 41, 42].

A recent biomechanical study highlighted the required compensatory deltoid function to compensate for abduction motion loss in the presence of simulated rotator cuff tears [4]. Anterosuperior (combined supraspinatus and subscapularis) tears resulted in the largest loss in glenohumeral abduction motion, despite the greatest increase in deltoid force [4]. On the other hand, isolated subscapularis tears increased the anterior deltoid force, compensating for the loss of anterior joint compression without a reduction in abduction [4].

1.4.2 Shoulder Function and Pseudoparalysis

The rotator cuff muscles are important contributors to a smooth glenohumeral motion and sufficient joint stability [17]. Acting as force couples, they collaborate to stabilize the inherently unstable glenohumeral joint [17]. The deltoid and the inferior portion of the rotator cuff act as the coronal force couple, compressing the humeral head to the glenoid in abduction [43]. Subscapularis and infraspinatus/teres minor represent the axial force couple, providing a fulcrum for the actions of the deltoid and supraspinatus, which is essential to maintain joint stability by a compressive joint reaction force in the axial plane [17, 37, 43].

Massive rotator cuff tears may disrupt these force couples resulting in superior migration of the humeral head and dysfunction of the shoulder (Fig. 1.3) [5, 17]. The importance of the force couples was highlighted by introducing the "suspension bridge" concept [43]. Accordingly, shoulder function may be maintained in isolated supraspinatus tears due to intact force couples [43]. However, as tears propagate into the anterior or posterior cuff direction, force coupling is disturbed, resulting in unstable kinematics and loss of function [43].

In addition, instability of the glenohumeral joint results in increased internal rotation in the setting of posterosuperior tears, external rotation in anterosuperior tears, and the total rotational range of motion in all abduction angles [44]. To maintain normal kinematics in the presence of massive cuff tears, greater forces by both the deltoid and the corresponding force couple muscle

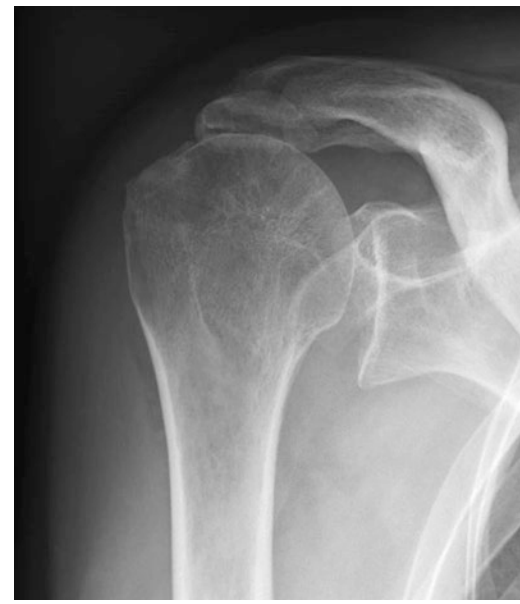


Fig. 1.3 X-ray demonstrating the superior migration of the humeral head

are required to achieve a coordinated abduction motion [4, 40].

Due to these kinematic changes, pseudoparalysis of the shoulder may occur (Fig. 1.4). The most common definition is active elevation less than 90° with full passive elevation [1, 45–47]. Risk factors are considered to be disruption of the entire subscapularis or of the three rotator cuff muscles [45]. However, recently it has been reported that pseudoparalysis should rather be described as no active elevation with maintained passive elevation of chronic nature, usually with anterior–superior escape and being refractory following an injection [1]. This definition may be more adequate, as pseudoparalysis is often confused with pain [1]. Therefore, pain should be ruled out as a cause of apparent pseudoparalysis, since patients may benefit from a pain-relieving treatment alone [1]. Sometimes, an injection of lidocaine for pain elimination will clarify the diagnosis in the face of a massive rotator cuff tear [1].

1.4.3 Decentralization, Glenohumeral Joint Pressure, and Osteoarthritis

Sufficient function of the rotator cuff muscles is essential to ensure glenohumeral stability through the concavity compression principle [48, 49]. Loss of rotator cuff integrity may significantly alter the joint-reaction forces, which are required to maintain glenohumeral stability [48, 49]. Dysfunction of the infraspinatus and subscapularis may lead to superior humeral head translation and joint instability by displacing the glenoidal contact point superiorly [4, 41].

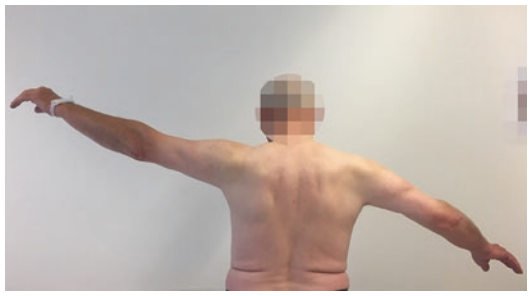


Fig. 1.4 Patient with severe pseudoparalysis of the right cuff-deficient shoulder

The abnormal joint loading due to rotator cuff insufficiency may cause various erosion patterns, frequently seen in type B glenoids of osteoarthritic patients [50, 51]. Recent literature suggests that this wear pattern is not axisymmetric to the superoinferior axis of the glenoid, but rather orientated in the posteroinferior region [50, 51]. Over time, these erosion patterns may lead to significant glenoid bone loss, presenting a major challenge in reverse shoulder arthroplasty [50–52]. However, three-dimensional reconstruction has allowed further analysis of glenoid erosion patterns. This is much needed, since the two-dimensional CT images inaccurately represent the wear pattern in osteoarthritic glenoids [51]. Unfortunately, it still remains uncertain if osteoarthritis results in altered kinematics and subluxation, or if the changed kinematics with subluxation is instigating this inflammatory disease [51].

Finally, massive cuff tears may lead to cuff tear arthropathy (CTA), which is defined as muscle degeneration, including fatty infiltration and atrophy, along with bony alterations, such as humeral head erosion and acetabularization of the acromion [53]. The underlying pathway may be induced by a massive cuff tear with anterosuperior escape, followed by a mechanical conflict between the humeral head and the superior glenoid and acromion [54]. In addition to the collapse of cartilage and bony structures, enzymes may be released that impair the surrounding tissue, thus leading to pain and limited shoulder function [53]. Maintenance of a sufficient rotator cuff function has been shown to be vital to delay the development of glenohumeral arthritis, highlighting the necessity of a good repair technique [55]. However, in the presence of severe CTA, these repair techniques may be infeasible, calling for reverse total shoulder arthroplasty instead.

1.5 Conclusion

Due to the combination of impaired biological healing potential and joint affecting biomechanical changes, massive irreparable rotator cuff tears

Table 1.1 Key factors

Nature of the problem	Key factors
Structural	• Most massive tears follow distinct patterns (antero- and posterosuperior) • Progressive thinning of the rotator crescent facilitates tear initiation • Increased tensile load on remaining fibres leads to tear progression in both anterior and posterior directions
Biological	• Hypovascularity limits biological healing potential • Tissue degeneration includes atrophy, fatty infiltration, retraction, and loss of elasticity making surgical repair difficult
Biomechanical	• Mechanical advantage of the deltoid muscle is comprised of higher tractive forces and loss of balanced concavity-compression • Disruption of force couples leads to superior humeral head migration and shoulder dysfunction (pseudoparalysis) • Glenohumeral instability causes humeral head decentralization • Glenoidal erosion patterns may progress to severe CTA

remain a major challenge in shoulder surgery. For the treatment of these patients and defining the underlying problems (Table 1.1), the interaction of biological and biomechanical pathomechanisms has to be considered. As biological healing may be impaired by hypovascularity as well as tissue degeneration including atrophy, fatty infiltration, and tendon retraction, concomitant biomechanical alterations of glenohumeral joint kinematics may result in shoulder dysfunction and lead to the development of cuff tear arthropathy in the long term.

References

1. Tokish JM, Alexander TC, Kissenberth MJ, Hawkins RJ. Pseudoparalysis: a systematic review of term definitions, treatment approaches, and outcomes of management techniques. J Shoulder Elbow Surg. 2017;26(6):e177–87.
2. Bedi A, Dines J, Warren RF, Dines DM. Massive tears of the rotator cuff. J Bone Joint Surg Am. 2010;92(9):1894–908.
3. Harryman DT, Mack LA, Wang KY, Jackins SE, Richardson ML, Matsen FA. Repairs of the rotator cuff. Correlation of functional results with integrity of the cuff. J Bone Joint Surg Am. 1991;73(7):982–9.
4. Dyrna F, Kumar NS, Obopilwe E, et al. Relationship between deltoid and rotator cuff muscles during dynamic shoulder abduction: a biomechanical study of rotator cuff tear progression. Am J Sports Med. 2018;46(8):1919–26.
5. Neri BR, Chan KW, Kwon YW. Management of massive and irreparable rotator cuff tears. J Shoulder Elbow Surg. 2009;18(5):808–18.
6. DeOrio JK, Cofield RH. Results of a second attempt at surgical repair of a failed initial rotator-cuff repair. J Bone Joint Surg Am. 1984;66(4):563–7.
7. Gerber C, Fuchs B, Hodler J. The results of repair of massive tears of the rotator cuff. J Bone Joint Surg Am. 2000;82:505–15.
8. Galatz L, Ball C, Teefey S, Middleton W, Yamaguchi K. The outcome and repair integrity of completely arthroscopically repaired large and massive rotator cuff tears. J Bone Joint Surg Am. 2004;86(2):219–24.
9. Hijioka A, Suzuki K, Nakamura T, Hojo T. Degenerative change and rotator cuff tears. An anatomical study in 160 shoulders of 80 cadavers. Arch Orthop Trauma Surg. 1993;112(2):61–4.
10. Jeong JY, Min SK, Park KM, Park YB, Han KJ, Yoo JC. Location of rotator cuff tear initiation: a magnetic resonance imaging study of 191 shoulders. Am J Sports Med. 2018;46(3):649–55.
11. Kim HM, Dahiya N, Teefey SA, et al. Location and initiation of degenerative rotator cuff tears: an analysis of three hundred and sixty shoulders. J Bone Joint Surg Am. 2010;92(5):1088–96.
12. Lehman C, Cuomo F, Kummer FJ, Zuckerman JD. The incidence of full thickness rotator cuff tears in a large cadaveric population. Bull Hosp Jt Dis. 1995;54(1):30–1.
13. Shimizu T, Itoi E, Minagawa H, Pradhan RL, Wakabayashi I, Sato K. Atrophy of the rotator cuff muscles and site of cuff tears. Acta Orthop Scand. 2002;73:40–3.
14. Wening JD, Hollis RF, Hughes RE, Kuhn JE. Quantitative morphology of full thickness rotator cuff tears. Clin Anat. 2002;15:18–22.
15. Burkhart SS, Esch JC, Jolson RS. The rotator crescent and rotator cable: an anatomic description of the shoulder's "suspension bridge". Arthroscopy. 1993;9(6):611–6.
16. Mochizuki T, Sugaya H, Uomizu M, et al. Humeral insertion of the supraspinatus and infraspinatus. New anatomical findings regarding the footprint of the rotator cuff. J Bone Joint Surg Am. 2008;90(5):962–9.
17. Greenspoon JA, Petri M, Warth RJ, Millett PJ. Massive rotator cuff tears: pathomechanics, current treatment options, and clinical outcomes. J Shoulder Elbow Surg. 2015;24(9):1493–505.
18. Bennett WF. Arthroscopic repair of massive rotator cuff tears: a prospective cohort with 2- to 4-year follow-up. Arthroscopy. 2003;19(4):380–90.

19. Bigliani LU, Cordasco FA, McIlveen SJ, Musso ES. Operative treatment of failed repairs of the rotator cuff. J Bone Joint Surg Am. 1992;74(10):1505–15.
20. Nho SJ, Delos D, Yadav H, et al. Biomechanical and biologic augmentation for the treatment of massive rotator cuff tears. Am J Sports Med. 2010;38(3):619–29.
21. Gamradt SC, Gallo RA, Adler RS, et al. Vascularity of the supraspinatus tendon three months after repair: characterization using contrast-enhanced ultrasound. J Shoulder Elbow Surg. 2010;19(1):73–80.
22. Dyrna F, Zakko P, Pauzenberger L, McCarthy MB, Mazzocca AD, Dyment NA. Human subacromial bursal cells display superior engraftment versus bone marrow stromal cells in murine tendon repair. Am J Sports Med. 2018;46(14):3511–20.
23. Dyment NA, Galloway JL. Regenerative biology of tendon: mechanisms for renewal and repair. Curr Mol Biol Rep. 2015;1(3):124–31.
24. Dyment NA, Hagiwara Y, Matthews BG, Li Y, Kalajzic I, Rowe DW. Lineage tracing of resident tendon progenitor cells during growth and natural healing. PLoS One. 2014;9(4):e96113.
25. Hernigou P, Flouzat Lachaniette CH, Delambre J, et al. Biologic augmentation of rotator cuff repair with mesenchymal stem cells during arthroscopy improves healing and prevents further tears: a case-controlled study. Int Orthop. 2014;38(9):1811–8.
26. Imam MA, Holton J, Horriat S, et al. A systematic review of the concept and clinical applications of bone marrow aspirate concentrate in tendon pathology. SICOT J. 2017;3:58.
27. Song N, Armstrong AD, Li F, Ouyang H, Niyibizi C. Multipotent mesenchymal stem cells from human subacromial bursa: potential for cell based tendon tissue engineering. Tissue Eng Part A. 2014;20(1-2):239–49.
28. Steinert AF, Kunz M, Prager P, et al. Characterization of bursa subacromialis-derived mesenchymal stem cells. Stem Cell Res Ther. 2015;6:114.
29. Utsunomiya H, Uchida S, Sekiya I, Sakai A, Moridera K, Nakamura T. Isolation and characterization of human mesenchymal stem cells derived from shoulder tissues involved in rotator cuff tears. Am J Sports Med. 2013;41(3):657–68.
30. Nho SJ, Yadav H, Shindle MK, Macgillivray JD. Rotator cuff degeneration: etiology and pathogenesis. Am J Sports Med. 2008;36(5):987–93.
31. Gladstone JN, Bishop JY, Lo IK, Flatow EL. Fatty infiltration and atrophy of the rotator cuff do not improve after rotator cuff repair and correlate with poor functional outcome. Am J Sports Med. 2007;35(5):719–28.
32. Joshi SK, Kim HT, Feeley BT, Liu X. Differential ubiquitin-proteasome and autophagy signaling following rotator cuff tears and suprascapular nerve injury. J Orthop Res. 2014;32(1):138–44.
33. Joshi SK, Liu X, Samagh SP, et al. mTOR regulates fatty infiltration through SREBP-1 and PPARgamma after a combined massive rotator cuff tear and suprascapular nerve injury in rats. J Orthop Res. 2013;31(5):724–30.
34. Liu X, Joshi SK, Samagh SP, et al. Evaluation of Akt/mTOR activity in muscle atrophy after rotator cuff tears in a rat model. J Orthop Res. 2012;30(9):1440–6.
35. Joe AW, Yi L, Natarajan A, et al. Muscle injury activates resident fibro/adipogenic progenitors that facilitate myogenesis. Nat Cell Biol. 2010;12(2):153–63.
36. Liu X, Ning AY, Chang NC, et al. Investigating the cellular origin of rotator cuff muscle fatty infiltration and fibrosis after injury. Muscles Ligaments Tendons J. 2016;6(1):6–15.
37. Alpert SW, Pink M, Jobe FW, McMahon PJ, Mathiyakom W. Electromyographic analysis of deltoid and rotator cuff function under varying loads and speeds. J Shoulder Elbow Surg. 2000;9(1):47–58.
38. Otis JC, Jiang CC, Wickiewicz TL, Peterson MG, Warren RF, Santner TJ. Changes in the moment arms of the rotator cuff and deltoid muscles with abduction and rotation. J Bone Joint Surg Am. 1994;76(5):667–76.
39. Kijima T, Matsuki K, Ochiai N, et al. In vivo 3-dimensional analysis of scapular and glenohumeral kinematics: comparison of symptomatic or asymptomatic shoulders with rotator cuff tears and healthy shoulders. J Shoulder Elbow Surg. 2015;24(11):1817–26.
40. Hansen ML, Otis JC, Johnson JS, Cordasco FA, Craig EV, Warren RF. Biomechanics of massive rotator cuff tears: implications for treatment. J Bone Joint Surg Am. 2008;90(2):316–25.
41. Oh JH, Jun BJ, McGarry MH, Lee TQ. Does a critical rotator cuff tear stage exist?: a biomechanical study of rotator cuff tear progression in human cadaver shoulders. J Bone Joint Surg Am. 2011;93(22):2100–9.
42. Terrier A, Reist A, Vogel A, Farron A. Effect of supraspinatus deficiency on humerus translation and glenohumeral contact force during abduction. Clin Biomech. 2007;22(6):645–51.
43. Burkhart SS. Fluoroscopic comparison of kinematic patterns in massive rotator cuff tears. A suspension bridge model. Clin Orthop Relat Res. 1992;284:144–52.
44. Oh JH, McGarry MH, Jun BJ, et al. Restoration of shoulder biomechanics according to degree of repair completion in a cadaveric model of massive rotator cuff tear: importance of margin convergence and posterior cuff fixation. Am J Sports Med. 2012;40(11):2448–53.
45. Collin P, Matsumura N, Ladermann A, Denard PJ, Walch G. Relationship between massive chronic rotator cuff tear pattern and loss of active shoulder range of motion. J Shoulder Elbow Surg. 2014;23(8):1195–202.
46. Denard PJ, Ladermann A, Jiwani AZ, Burkhart SS. Functional outcome after arthroscopic repair of massive rotator cuff tears in individuals with pseudoparalysis. Arthroscopy. 2012;28(9):1214–9.

47. Oh JH, Kim SH, Shin SH, et al. Outcome of rotator cuff repair in large-to-massive tear with pseudoparalysis: a comparative study with propensity score matching. Am J Sports Med. 2011;39(7):1413–20.
48. Apreleva M, Parsons IM, Warner JJ, Fu FH, Woo SL. Experimental investigation of reaction forces at the glenohumeral joint during active abduction. J Shoulder Elbow Surg. 2000;9(5):409–17.
49. Parsons IM, Apreleva M, Fu FH, Woo SL. The effect of rotator cuff tears on reaction forces at the glenohumeral joint. J Orthop Res. 2002;20(3):439–46.
50. Beuckelaers E, Jacxsens M, Van Tongel A, De Wilde LF. Three-dimensional computed tomography scan evaluation of the pattern of erosion in type B glenoids. J Shoulder Elbow Surg. 2014;23(1):109–16.
51. Knowles NK, Keener JD, Ferreira LM, Athwal GS. Quantification of the position, orientation, and surface area of bone loss in type B2 glenoids. J Shoulder Elbow Surg. 2015;24(4):503–10.
52. Gupta A, Thussbas C, Koch M, Seebauer L. Management of glenoid bone defects with reverse shoulder arthroplasty-surgical technique and clinical outcomes. J Shoulder Elbow Surg. 2018;27(5):853–62.
53. Rugg CM, Gallo RA, Craig EV, Feeley BT. The pathogenesis and management of cuff tear arthropathy. J Shoulder Elbow Surg. 2018;27(12):2271–83.
54. Collins DN, Harryman DT. Arthroplasty for arthritis and rotator cuff deficiency. Orthop Clin North Am. 1997;28(2):225–39.
55. Ecklund KJ, Lee TQ, Tibone J, Gupta R. Rotator cuff tear arthropathy. J Am Acad Orthop Surg. 2007;15(6):340–9.

Biology of Rotator Cuff Injury and Repair

2

Alessio Giai Via, Davide Cucchi, and Laura de Girolamo

2.1 Histological Features

2.1.1 The Normal Tendon Tissue

Tendons are the anatomical structures responsible for the transmission of forces from muscles to bones, and thus are crucial in allowing joint movement [1]. They are mainly made of collagen type I molecules hierarchically organized into tropocollagen (a polypeptide chain made of a triple helix, the building blocks of fibrils), which are bundled in fibers surrounded by the endotenon and eventually compose fascicles [2]. Fascicles associated together represent the tertiary bundles, which altogether constitute the tendon itself [3, 4]. A number of different collagen molecules, proteoglycans (PGs), glycosaminoglycans (GAGs) and noncollagenous proteins (tenascin, fibronectin, elastin, decorin, etc.) are responsible for this complex structural organization. While collagen provides tensile strength, the other components of tendon extracellular matrix (ECM) allow for structural support and regulate fibril and fiber assembly [4]. The epitenon, a thin membrane surrounding the tendon, provides supply to the tissue in terms of vascularization and innervation. At the outer layer, the epitenon is surrounded by the paratenon, a sheat of connective tissue made of collagen fibrils (mainly type I and III) [5].

Resident tendon cell population comprise both tenocytes, displaying an elongated shape, and tenoblasts, rounded progenitor cells [6]. These cells, embedded in tendon ECM, are responsible for the synthesis and the remodeling of the molecules composing the fibers.

The remodeling activity of these cells is important for the maintenance of tissue homeostasis and function, particularly in tendons with high functional demand, such as the supraspinatus and the Achilles tendons [7, 8], where it represents a protective mechanism against tissue damage [9]. Remodeling is also crucial in the early phases of tissue repair and healing, while failure in this process may cause the formation of scar tissue, thus compromising the mechanical properties of tendons [10]. Matrix Metalloproteinase-1 (MMP-1) is the main enzyme involved in collagen type I degradation [11] and its activity is controlled at many levels to prevent aberrant matrix disruption leading to tissue degeneration [12]. The importance of the remodeling mechanisms was

A. Giai Via
Department of Orthopaedic and Traumatology, Sant'Anna Hospital, Como, Italy

D. Cucchi
Department of Orthopaedics and Trauma Surgery, Universitätsklinikum, Bonn, Germany

L. de Girolamo (✉)
Orthopaedic Biotechnology Laboratory, IRCCS Orthopaedic Institute Galeazzi, Milan, Italy
e-mail: laura.degirolamo@grupposandonato.it

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_2

clearly demonstrated by Millar and colleagues, who showed the correlation between MMP inhibition and the onset of painful lesions [13].

The portions of tendons towards muscle, as well as those close to the bone, are specialized regions with different ECM compositions and cellularities. In particular, in the bone-tendon junction the transition of the tissue from tendon towards bone comprises zones of fibrocartilage and mineralized fibrocartilage [14].

Upon light microscopy, the normal tendon appears as a dense tissue made of parallel collagen bundles, with a few scattered cells showing elongated nuclei. Small vessels are found in the endotenon, following a parallel orientation with respect to the collagen fibers [1, 4, 15] (Fig. 2.1a).

The supraspinatus tendon in the rotator cuff is responsible for the stabilization of the shoulder joint, and it is an example of the complex organization characterizing some tendons. Indeed, it is composed of a multilayered structure, each layer presenting a different fiber orientation [16]. Fifty-six percent of its dry weight is collagen [8], with type I and type III collagen cross-links to form each fibril [17]. The cross-link among collagen molecules is crucial to obtain the definitive tendon structure, and in the supraspinatus tendon these elements are more present with respect to the average tendon, probably due to higher functional demand in terms of shear stress and load it has to sustain [18]. For the same reason, the supraspinatus tendon contains a higher amount of proteoglycans, in particular, aggrecan and byglican [19].

2.1.2 The Pathological Tendon Tissue

Tendon pathologies are commonly classified with the term "tendinopathy," comprising a wide spectrum of conditions, from inflammation to ruptures, through different grades of tears and tissue degeneration [20]. In this condition, several modifications of tissue histopathology occur: collagen fibers separate, reducing their dimension and losing the parallel orientation, thus resulting in decreased tissue density; microtears may be observed, in the form of erythrocyte accumulation in the presence of fibrin and fibronectin deposits; increase of vascularization; infiltration of adipose tissue within the tendon (Fig. 2.1b). Moreover, tendinopathy is associated with an overall increase of collagen type III, contributing to tissue density reduction and loss of fiber orientation, and influencing the appearance of the tissue under polarized light, with reduced reflectivity if compared to the normal tissue [21]. Another characteristic of this condition is the uneven distribution of tendon cells within the tissue, with some areas containing a high cell density, where tenocytes may show a

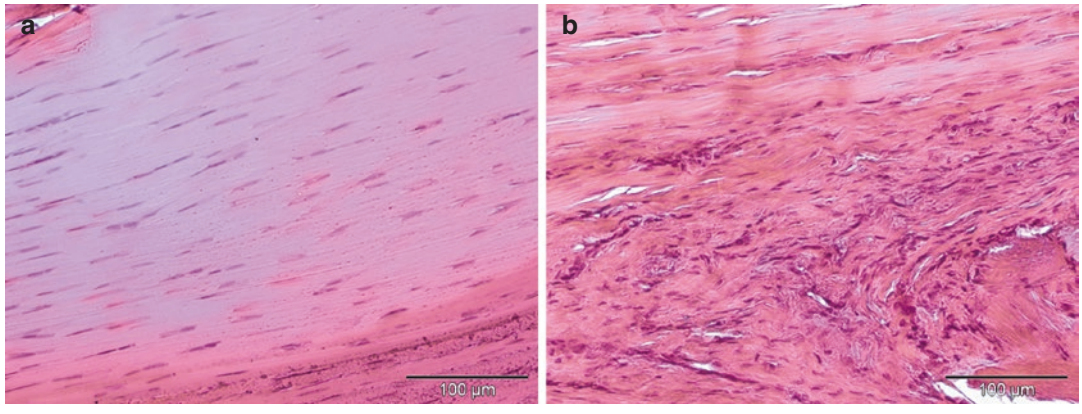


Fig. 2.1 Hematoxylin and eosin staining of tendons. (**a**) Normal tendon appearance with parallel collagen fibers and few elongated cells; (**b**) degenerated tendon tissue, with loss of fiber orientation and an increased number of cells showing a round shape. Scale bar: 100 µm

chondroid appearance, with rounded nuclei and a cytoplasm rich in PGs and GAGs, and other areas with decreased density, where a few cells have a small nuclei with an apoptotic/necrotic appearance [21, 22]. The role of inflammation in tendinopathy has been considered negligible for most of the twentieth century, but it is nowadays well recognized, thanks to the introduction of more sophisticated techniques, in particular in the field of immunohistochemistry. In fact, lymphocytes T and B and macrophages have been described in chronic Achilles tendinopathy, while granulocytes characterize the asymptomatic tendon ruptures [23]. At the same time, different markers of inflammation have been found in tendon pathological contexts, for example cyclooxygenase 2 and interleukin-1β (IL -1β) [24, 25].

As already mentioned, neovascularization is a key feature of tendinopathy, and since it is usually accompanied by innervation, it may be responsible for the symptomatic pain in tendon disorders [26].

The hierarchical structure of tendons allows for the establishment of a fail-safe mechanism, since the failure of few fibers would not affect the functionality of the whole tendon. Nevertheless, the etiology of tendinopathy is usually described as derived from the unequal distribution of load across the tendon or by repeated strain, possibly leading to fatigue failure of multiple fiber bundles and to the separation of layers within the tendon tissue [27, 28], causing overuse injury.

2.1.3 Histological Findings of Rotator Cuff Tendinopathy

As mentioned before, tendons from the rotator cuff have a complex organization and they are characterized by a higher content in PGs and GAGs as well as a higher density of cross-links among the collagen fibers, with respect to other tendons. Nevertheless, the degeneration of this tissue results in further accumulation of GAGs, especially between fibers, as well as a morphological change of tendon cells, from an elongated to rounded shape, forming fibrocartilage-like areas [29–31]. In addition, reduction in cell density, tissue calcification, neovascularization, and lipid infiltration are common features of the degenerated supraspinatus tendon that usually occur in patients over 60 years old [32]. In general, ruptured supraspinatus tendons usually show severe degeneration, in particular characterized by reduced cellularity and fiber disorganization [32]. Typical features also comprise the onset of tears and the reduction of fiber dimension. In advanced stages, cell necrosis and tissue calcification may occur [33–35]. From the biochemical point of view, the key markers of inflammation and tendon remodeling, IL-1β and MMP-1, resulted in an increase in ruptured supraspinatus tendons [24]. Nevertheless, it is still unclear whether the presence of these markers represents an acute response to rupture or if they are related to the degeneration process. On the other hand, a higher proportion of collagen type III in the tissue is an indicator of previous injuries and traumas, resulting in scar tissue with a lesser quality with respect to native tendon and thus more prone to rupture [36, 37].

2.1.4 The Role of Hypercholesterelemia in Tendinopathy

Besides traumas and overuse, hypercholesterolemia (HC), i.e., a high blood content of cholesterol (>240 mg/dL), recently emerged as a possible cause of tendon degeneration. Histological studies showed that high cholesterol levels may alter the tendon microenvironment via local changes in gene expression, protein synthesis and ECM turnover. In particular, high serum cholesterol levels allow for the accumulation of oxidized-low-density lipoproteins (LDL), which are lipids carrier proteins. Lipid accumulation within tendon ECM may affect the mechanical properties of the tissue [38, 39] correlating with a lower healing potential after surgical repair, as observed in animal models [40, 41]. Clinical studies often show conflicting results regarding the relationship between HC and tendon disorders [42, 43], but the majority of the studies investigating the correlation of

HC, hyperlipidemia or dyslipidemia and tendon pathologies demonstrated a correlation between the systemic levels of lipids and the development of tendon disorders, in particular rotator cuff disease [42, 44]. A possible explanation of the relation between body mass index (BMI) and tendinopathy could be the excessive loading on the musculoskeletal system exerted by overweight individuals. Nevertheless, this explanation is simplistic, given the molecular changes that have been observed in tendons of subject affected by hypercholesterolemia and normal tendons. Indeed, in HC condition, cholesterol deposits are found within tendon ECM and cells, with a direct influence of the mechanical properties of the tissue [40, 45]. In addition, inflammation is a direct consequence of HC, also causing cardiovascular diseases, and the infiltration of macrophages in the tendons of patients affected by HC has been observed [46]. At the same time, HC results in an alteration of matrix deposition in different tissues, affecting the synthesis of noncollagenous proteins [47], the proportion of collagen type III [48], and the production of PGs [49]. Taken together, these observations indicate that HC may significantly alter the composition of tendon ECM, explaining the reduction of the supraspinatus tendon biomechanical properties, the higher rates of tendon injury, and the decrease in tissue healing observed in animal models of HC [41, 50]. Based on these results, HC emerged as a risk factor for tendon degeneration, causing structural/mechanical modifications and fostering inflammation. Indeed, while overuse is considered the most common cause of tendinopathy, the involvement of HC would explain the onset of these disorders in the population of inactive patients with high body mass index (BMI) [51, 52].

2.2 Rotator Cuff Injury

Rotator cuff injury has a multifactorial pathogenesis, which includes anatomical, mechanical (or extrinsic), and biological (or intrinsic) factors. Many theories have been postulated to explain the pathogenesis of rotator cuff tears (RCTs), trying to unify intrinsic and extrinsic theories, but the precise role of each factor is not fully understood yet. However, recent evidence strongly suggests that most of the tendinopathies and tendon ruptures are caused by primary failed healing response [53].

2.2.1 Mechanical and Extrinsic Factors

2.2.1.1 Anatomy

Anatomic differences have been considered as a risk factor for RCTs. The critical shoulder angle (CSA) is the angle between a line connecting the inferior and superior margins of the glenoid fossa, and a line drawn from the inferior edge of the glenoid to the lateral aspect of the acromion, measured on anteroposterior plain radiographs (Fig. 2.2). Moor et al. found that a large CSA was an independent predictor for a posterosuperior RCT [54]. Gerber et al. showed that a large CSA increased the instability ratio (the ratio of join shear force to joint compression force), in particular at about 60° of abduction, and that the load on the supraspinatus tendon increased by 33% in response to the increased shoulder instability [55]. This could produce the supraspinatus tendon

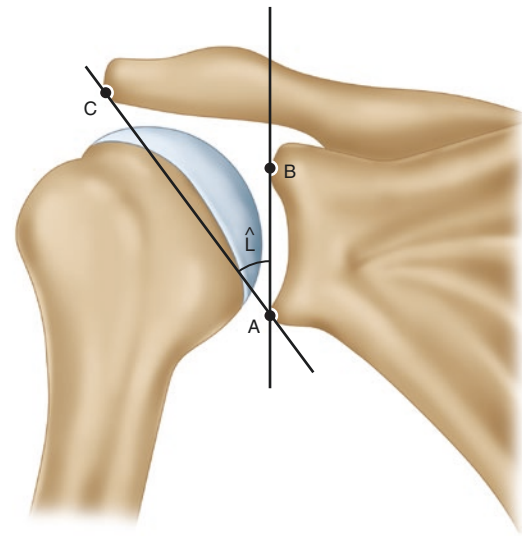


Fig. 2.2 The critical shoulder angle (CSA) is the angle between a line connecting the inferior and superior margin of the glenoid fossa, and a line drawn from the inferior margins of the glenoid to the lateral aspect of the acromion

tear. The size of the acromion is also an additional anatomic risk factor. Nyffeler et al. demonstrated that the lateral acromion index (the distance from the lateral border of the acromion to the glenoid plane divided by the distance from the lateral border of the humeral head to the glenoid plane) was significantly larger in patients with full-thickness RCTs compared to controls [56].

Mechanical factors have been proposed to be a cause for RCTs, because the confined position of the supraspinatus tendon within the subacromial space makes this tendon more susceptible to degenerative changes. Neer and Poppen first described a subacromial impingement theory in 1972. Based on the intraoperative findings, the authors found that RCTs mainly occurred in the supraspinatus tendon, in an area that contatcted the coracoacromial ligament, the anterior acromion, and sometimes the acromion-clavicular joint during forward elevation, concluding that 95% of RCTs were caused by subacromial impingement [57]. In support of this theory, the acromial shape was classified into three types by Bigliani et al. Type II (curved) and type III (hooked) have been statistically associated with RCTs, and type III acromion has been considered responsible for about 70% of supraspinatus tendon tears [58]. A systematic review by Seitz et al. concluded that patients with full-thickness RCTs have a significantly smaller acromiohumeral space than controls. However, although the narrowing of the subacromial space has been classically associated with RCTs, it not clear whether impingement induces tendon injury or a primary rotator cuff dysfunction leads to subacromial impingement by the resulting superior humeral translation [59]. Acromial shapes can be both congenital and acquired. Age determines the progression from a flat to a curved or hooked acromion, possibly because of traction forces. This would partially explain the epidemiological evidence of higher incidence of RCTs with increased age, but also suggests a primary intrinsic moving factor [60].

2.2.1.2 Overuse

Mechanical overuse is also involved in rotator cuff disease, as suggested by the frequent observation of symptomatic disease in the dominant arms rather than in the nondominant arms. Mechanical overuse of the supraspinatus tendon has been studied by Soslowsky et al. in a rat model [61, 62]. Tendons in the overuse model exhibited an increased cross-sectional area, hypercellularity, and collagen disorganization. Maximum stress and elastic modulus were significantly lower in the overuse group compared to control rats. However, chronic overuse injury is only one of the several factors contributing to the pathogenesis of RCTs, as 28% of patients present full-thickness tear in the nondominant arm, and 36–50% of patients have bilateral full-thickness tear, especially in older patients [63, 64].

2.2.1.3 Cigarette Smoking

The other extrinsic factor is cigarette smoking, which has been associated with rotator cuff injury and poorer outcomes after repair. Many studies reported that nicotine and carbon monoxide decrease microperfusion and tissue oxygenation, leading to tissue hypoxia [65]. The supraspinatus tendons in smokers showed significantly more advanced degenerative changes, with increased density of apoptotic cells, reduced tenocyte density, and down regulation of proliferative activity [66]. This relationship is dose-dependent and time-dependent [67].

2.2.2 Biological Factors

2.2.2.1 The Role of Vascular Supply

A hypovascular zone, the so-called "critical zone," has been traditionally described 10–15 mm proximal to the insertion of the supraspinatus tendon [68]. It is unclear whether this hypoperfusion contributes to tendon's degeneration, because histological and intraoperative studies showed relative hyperperfusion at this area and at the tear edge [31]. Brooks et al. showed that both the vessel diameter and their number were approximately reduced by third at 5 mm from the cuff edge compared with 30 mm, but no significant hypovascularization has been identified [69]. However, when the arm is in full adduction, the supraspinatus is compressed by the humeral head into the subacromial space, and blood perfusion may significantly be reduced.

2.2.2.2 The Role of Diabetes Mellitus

Diabetes mellitus has been associated with compromised tendon function, increased susceptibility to tendon injury, and reduced healing ability [70]. Worse results after rotator cuff repair and higher incidence of re-ruptures have been reported in diabetic patients compared to nondiabetic controls [63]. Accumulation of advanced glycation end-products (AGEs) impaired collagen production and ECM formation, and compromised angiogenesis caused by elevated glucose concentration may be suggested as a probable pathogenesis of tendinopathy in patients with diabetes mellitus. Protein glycation is a spontaneous reaction depending on the degree and duration of hyperglycemia, the half-life of protein, and permeability of the tissue to free glucose. Glycated proteins can undergo further reactions giving rise to AGEs, which are complex, heterogeneous molecules that cause protein cross-linking, and alter the physical characteristics of collagen fibers [71]. In tendons, AGEs formation affects the interactions between collagen fibers, ECM protein, and tenocytes [72]. These changes have been associated with both reduced healing capacity and altered mechanical properties of connective tissues. The effects of AGEs on the mechanical properties of tendons have been studied in a rat model [70]. The formation of AGEs would change the way tendons respond to loading, in particular reducing tissue viscoelasticity by severely limiting fibril–fibril sliding, making tendon more susceptible to injury. This has been recently confirmed by an in vitro study by Gautieri et al. [73]. Interestingly, Chung et al. found a significant overexpression of MMP-9 and IL-6 genes in the torn supraspinatus tendon of diabetic patients compared to controls, concluding that the increased MMP-9 and IL-6 synthesis might significantly compromise the integrity of tendon ECM and predispose patients with diabetes to tendinopathy or rupture [74].

2.2.2.3 The Role of Thyroid Hormones

The relationship between thyroid disorders and shoulder pain has been suspected since the late 1920s [75]. More recently, such association has been formally hypothesized. A recent epidemiological study by Oliva et al. showed that nearly 60% of patients that received arthroscopic rotator cuff repair were also affected by thyroid disorders [76]. The influence of thyroid hormones on the pathogenesis of tendinopathy has been confirmed by an in vitro study that showed the presence of thyroid hormones receptors on tenocytes. Thyroid hormones were able to induce tenocyte growth, to reduce the doubling time, and they also counteracted apoptosis in a dose- and time-dependent manner. Thyroid hormones are also able to influence tenocyte secretion of ECM proteins [77]. When tenocytes have been cultivated in the presence of T3 or T4 individually or in combination with ascorbic acid, thyroid hormones significantly increase the expression of collagen type I. Furthermore, the synthesis of cartilage oligomeric matrix protein (COMP) was also increased. COMP is a glycoprotein that binds collagen type I, II, and IX and fibronectin, and it is largely present in tendon exposed to compressive load. All these data confirm the essential role of thyroid hormones in regulating tenocytes' proliferation and ECM homeostasis [78].

2.2.2.4 The Role of Cholesterol and Lipids

As already reported (2.1.4), hypercholesterolemia has been implicated as a risk factor for tendinopathy, including rotator cuff injury [79, 80].

High serum cholesterol levels allow for the accumulation of oxidized-low-density lipoproteins (LDLs), which are lipid carrier proteins [43]. The clinical manifestation of LDL accumulation in human tendons is xanthoma, which is the major tendon disorder in patients with familial dyslipidemias. Lipids found within xanthomas derive from the circulating plasma rather than being synthesized locally [81].

2.2.3 Genetic Factors

2.2.3.1 The Role of Apoptosis

Recent studies suggested the contribution of genetic factors in the pathogenesis of RCTs. Many authors described the increased risk of

experiencing symptoms (five times) and of developing RCT (more than twice) among siblings and second-degree relatives [82–84]. Even if the exact genetic profile is still under investigation, some genes responsible for being more susceptible to RCT have been described.

Animal studies showed a higher gene expression of glutamate, and high levels of intratendinous glutamate have been revealed in a rat model of supraspinatus tendon tear [85]. Glutamate is a neurotrasmitter of the central nervous system, and high extracellular glutamate concentration has been related with neurodegenerative disorders such as Huntington and Alzheimer diseases, and seems to be deleterious for cells ("excitotoxicity") [86]. Interestingly, glutamate cascade has been related to functional adaptation of bone to mechanical loading, and in vitro study showed that it has a pro-apoptotic effect in cultured tendon fibroblasts, by regulating the expression of apoptosis-related genes [87, 88]. Therefore, even if the exact significance of high glutamate synthesis is not completely understood, the gene-expression may be related to rotator cuff tears.

Recent studies pointed out that apoptosis plays a key role in the pathogenesis of tendon injury [89]. Apoptosis is a highly regulated cellular process involved in the development of multicellular organisms, and because of its role in the control of cell population, it is essential for the homeostasis of adult tissues. Excessive apoptosis within the rotator cuff tendon can alter the balance of normal tissue turnover, and promote increased tendon degradation. Yuan et al. showed an increased prevalence of apoptotic tissue within the edges of torn supraspinatus compared to the control subscapularis tendon [90]. Wu et al. showed that the percentage of cells undergoing apoptosis increased gradually with the degree of ECM breakdown [91]. Many biochemical events lead to apoptosis, as modification of the cellular membrane (blebbing), nuclear fragmentation, DNA fragmentation, and modification of cell adhesion [92]. Cytochrome C proteins are cellular signaling proteins which activate the synthesis of caspases, a protease enzyme family which promotes the degradation of cellular contents [93]. In an in vitro study, Lee et al. found an increased expression of cytochrome C and caspases in injured rotator cuff tendons compared to controls, confirming the increased apoptosis in turned supraspinatus tendons [94]. On the other hand, the turnover of ECM is mediated by MMPs, which are able to denature collagen fibers. The fine balance between suppression and induction of the MMPs is of primary importance for the homeostasis of the ECM. An increased activity of MMP-1 and a reduction of MMP-2 and MMP-3 have been described by many authors in a supraspinatus tendon rupture, confirming that the failure of the normal matrix remodeling process is an important element in RCTs [95]. Castagna et al. found these increased enzyme levels not only at the edges of the torn supraspinatus, but also in uninjured portions of the supraspinatus and the subscapularis tendons, which suggests that a more global breakdown of tissue may occur [96].

The variances in the genetic code between individuals are termed single nucleotide polymorphisms (SNPs), and Tashjian et al. identified 2 SNPs associated with RCTs [97]. They were located within two genes SAP30BP (on chromosome 17) and SASH1 (on chromosome 6). These genes both play a marked role in apoptosis, regulating tendon cell apoptosis and predisposing individuals to RCTs. Recently the same authors identified an SNP within the estrogen-related receptor beta (ESRRB) gene that appears to promote increased susceptibility to retears after a rotator cuff repair [98].

2.3 Rotator Cuff Healing

2.3.1 The Tendon Repair Process

The tendon healing process can be divided into three successive and overlapping phases, defined respectively as inflammatory phase (0–7 days), proliferative phase (5–25 days), and remodeling phase (>21 days) [99].

The goals of tendon repair are to restore its force transmission function and recreate the relationships with the surrounding tissues which allow the tendon to move smoothly. The success of tendon healing depends on the activation

of cellular elements able to synthesize a new ECM and to remodel it with structural properties suitable for sustaining tensile loads. Two mechanisms have been proposed to explain the recruitment of these cellular elements [99]: the first hypothesis, of "extrinsic repair," states that fibroblasts responsible for the synthesis of ECM are not resident in the tendon but migrate to the lesion from the bloodstream; the second hypothesis, of "intrinsic repair," states that these cells are resident in the endothenonium and in the epithenonium and from there directly migrate to the lesion site. It is likely that both the mechanisms coexist and are activated in two successive moments; the initial stages of the repair would be guided by nonresident cells, recalled by chemotactic factors released at the time of injury, while the remodeling phases would be promoted by resident cell populations, which migrate later to the lesion site [100].

2.3.1.1 Inflammatory Phase

In the initial inflammatory phase, damage to the vascular structures causes extravasation of blood inside the tendon and formation of hematoma. Activation of the coagulation cascade and of platelets releases chemotactic factors, vasodilatory substances, and proinflammatory molecules, which attract inflammatory cells at the site of the lesion. These cells release other cytokines such as IL-1β and tumor necrosis factor α, which further promote the inflammatory cascade [101].

This early response to rotator cuff tears leads to apoptosis of tenocytes and degradation of muscle fibers; cellular debris, clot, and foreign material are then removed by phagocytosis from granulocytes and macrophages, which secrete additional signaling molecules involved in chemotaxis and in the regulation of cell differentiation. Most of these factors are members of the transforming growth factor β superfamily and are the key regulators of gene expression, allowing subsequent tissue regeneration to occur [102]. At the end of the inflammatory phase, fibroblasts are recruited, and begin to synthesize the components of the ECM and release angiogenic factors, which activate vascular proliferation and promote the formation of a new capillary network [103].

2.3.1.2 Proliferative Phase

In the proliferative (or fibroblastic or "repair") phase, the recruitment of fibroblasts continues and their proliferation increases, as well as the synthesis of collagen and of other molecules of the ECM. Collagen production during tendon repair begins with the synthesis of type III collagen, which takes on a disordered disposition and gives the lesion a histological appearance similar to that of dermal scars [104].

In the muscle, anti-inflammatory macrophages express myogenic regulatory factors, which in combination with other endocrine growth factors can induce the development of mature myocytes from precursor cells [105, 106].

2.3.1.3 Remodeling Phase

The remodeling phase is characterized by a reduction of the synthesis of ECM and of type III collagen and an increase in the synthesis of type I collagen. Type I collagen fibers are arranged longitudinally along the axis of the tendon load and cross-links are formed that stabilize the fibers. At the end of this phase, the maximum stiffness and tensile strength of the tendon is reached, which however remains lower than that of a healthy tendon [100, 104].

2.3.2 Patients' Factors Affecting Tendon Healing

Numerous studies described factors influencing rotator cuff healing after surgical repair, including age, smoking habits, comorbidities, tear size, and fatty infiltration of the rotator cuff muscles. These factors, together with variables related to the surgical technique and the rehabilitation protocol, contribute to define the healing potential of a repaired rotator cuff.

2.3.2.1 Age

Increasing age negatively affects rotator cuff healing [107, 108]. This is both related to the reduction of the tendons' intrinsic healing prop-

erties and to the fact that age also increases the probability of having concomitant extrinsic factors, which increase the likelihood of postoperative retears. A recent retrospective cohort study on 1600 consecutive rotator cuff repairs identified age as an independent factor strongly related to increasing retear after rotator cuff repair. Interestingly, the retear risk appeared to increase at different rates as the patient age increased (minimally under 50 years of age, by 5% for each decade between the ages of 50 and 70 years and substantially over 70 years of age) [109]. An age >70 years at the time of surgery was also identified as an independent risk factor in a study designed to determine the prognostic factors that predict rotator cuff healing after surgical repair [110]. These findings are confirmed by ex vivo and animal studies, which identified a loss of the structural organization of tendons and a decrease of collagen organization and repair integrity with increasing age [111, 112].

2.3.2.2 Cigarette Smoking

Smoking affects the biomechanical and histological properties of rotator cuff tendons: this has a consequence both on the risk of developing rotator cuff tears [67] and on the healing properties after rotator cuff repair. The fact that smokers have a significantly higher healing failure rate than nonsmokers was postulated considering the results of animal studies and was recently confirmed in a prospective cohort study [113, 114]. Abstinence or at least decrease in nicotine use is recommended to improve healing after rotator cuff repair [99].

2.3.2.3 Endocrine Disorders and Hormones

Abnormal glucose levels impair the biomechanical properties of rotator cuff in animal models and increase the number of complications (infections and repair failures) after rotator cuff repairs in human patients [115, 116]. It is therefore recommended to evaluate and normalize the blood glucose levels pre- and postoperatively.

Hyperlipidemia and hypercholesterolemia decrease the biomechanical properties of rotator cuff tendons in animal models and have been considered as risk factors for the development of RCTs [41, 44, 50]. Recently, the negative role of hyperlipidemia has been documented also in a clinical study with a retrospective design [117].

Estrogens could also play a role influencing rotator cuff healing: estrogen deficiency, in fact, was associated with decreased biomechanical properties and poor development of chondroid tissue at the tendon-to-bone junction after rotator cuff repair in an animal model: this could encourage investigations on agents which modulate bone metabolism, to improve tendon-to-bone healing in patients with an estrogen deficiency who undergo rotator cuff surgery [118].

Low levels of vitamin D showed to negatively influence early healing of the rotator cuff after repair in animal models [119], but these effects could not be proved in human clinical studies [120].

2.3.3 Intraoperative Variables and Surgical Technique

The surgical technique can also affect rotator cuff healing. Numerous procedures have been described to address rotator cuff lesions, evolving from open to fully arthroscopic techniques; simultaneously, sutures and devices to secure the repaired tendon to the humeral head have been optimized, with the constant aim of providing a mechanically stable repair at the tendon-to-bone junction [121, 122]. However, not every massive RCT can be treated with the same surgical technique. The macroscopic observation of the tendon and its grade of mobilization can guide the surgeon to the best choice among the numerous options available; furthermore, predictive scores, like the ARoCuS score, have been developed to help the surgeon in this decision-making process [123].

Drilling into the footprint or performing microfractures of the greater tuberosity have been proposed as solutions to enhance tendon healing, with contrasting results among the available reports. Although not visible in postoperative magnetic resonance imaging, healing seems to be positively affected by microfractures, especially in case of larger tears [124]. Drilling into the footprint is another solution which could contribute

to rotator cuff healing [125, 126]. The rationale behind these techniques, which demonstrated to improve the quality of repair tissue and biomechanical strength at the tendon-to-bone insertion after rotator cuff repair in animal models, is the stimulation of bone marrow-derived cell infiltration into the repaired rotator cuff [127]. To perform a debridement of the torn tendons is another surgical trick which could improve postsurgical rotator cuff healing. This is commonly recommended in presence of large retracted lesions, although recent evidence suggests that detaching the intact tendon, completing and repairing the rotator cuff lesion, could enhance healing as compared to in situ repair techniques for partial lesions [128].

2.3.4 Timing of Surgery and Rehabilitation

Animal studies suggest that, when possible, early surgical repair of traumatic massive RCTs should be performed, since this leads to improved biomechanical properties of the tissue after healing [129]. This recommendation, however, still deserves to be confirmed in the clinical setting [130]. Delaying repair of massive lesions induces fatty degeneration of the involved tendons, which can also influence structural and clinical outcomes: fatty degeneration of the rotator cuff tendons is, in fact, an independent risk factor for rotator cuff retears and for worse outcome in patients with large to massive tears who had intact tendons after repair [107, 131, 132].

A high quality meta-analysis by Riboh et al. showed no statistically significant difference between immobilization and early passive motion in rotator cuff retear rates at minimum of 1 year of follow-up, but suggested the possibility of an increase in retears with early passive motion protocols [133]. Kluczynski et al. concluded their meta-analysis associating early active motion with increased risk of structural defects for small and large rotator cuff tears. The current, best available evidence regarding postoperative rehabilitation after rotator cuff repair is a recently published systematic review of overlapping meta-analyses, which suggests that early motion improves the range of motion after rotator cuff repair but increases the risk of rotator cuff retear [134].

2.3.5 Predictive Models to Quantify Tendon Healing

As this chapter synthetically illustrated, numerous patient-related factors may play a role in determining both subjective and objective outcomes of rotator cuff surgery. The rotator cuff healing index was recently developed as a numerical scoring system to predict rotator cuff healing after surgical repair. This promising system, for which validation studies are expected, includes clinical and radiological factors and has been designed to help predict the adequacy of the repair and assist in deciding the appropriate treatment options [110].

Acknowledgments The authors thank Dr. Marco Viganò, IRCCS Orthopaedic Institute Galeazzi, for his precious help with the chapter.

References

1. Józsa L, Kannus P. Histopathological findings in spontaneous tendon ruptures. Scand J Med Sci Sports. 1997;7(2):113–8.
2. Kastelic J, Galeski A, Baer E. The multicomposite structure of tendon. Connect Tissue Res. 1978;6(1):11–23.
3. Kannus P. Etiology and pathophysiology of chronic tendon disorders in sports. Scand J Med Sci Sports. April 1997;7(2):78–85.
4. Aström M, Rausing A. Chronic Achilles tendinopathy. A survey of surgical and histopathologic findings. Clin Orthop. 1995;316:151–64.
5. Kvist M, Józsa L, Järvinen M, Kvist H. Fine structural alterations in chronic Achilles paratenonitis in athletes. Pathol Res Pract. 1985;180(4):416–23.
6. Kannus P. Structure of the tendon connective tissue. Scand. J Med Sci Sports. 2000;10(6):312–20.
7. Bank RA, Robins SP, Wijmenga C, Breslau-Siderius LJ, Bardoel AF, van der Sluijs HA. Defective collagen crosslinking in bone, but not in ligament or cartilage, in Bruck syndrome: indications for a bone-specific telopeptide lysyl hydroxylase on chromosome 17. Proc Natl Acad Sci U S A. 1999;96(3):1054–8.
8. Riley GP, Harrall RL, Cawston TE, Hazleman BL, Mackie EJ. Tenascin-C and human tendon degeneration. Am J Pathol. 1996;149(3):933–43.

9. Perez-Castro AV, Vogel KG. In situ expression of collagen and proteoglycan genes during development of fibrocartilage in bovine deep flexor tendon. J Orthop Res. 1999;17(1):139–48.
10. Spindler KP, Andrish JT, Miller RR, Tsujimoto K, Diz DI. Distribution of cellular repopulation and collagen synthesis in a canine anterior cruciate ligament autograft. J Orthop Res. 1996;14(3):384–9.
11. Cawston TE, Curry VA, Summers CA, Clark IM, Riley GP, Life PF. The role of oncostatin M in animal and human connective tissue collagen turnover and its localization within the rheumatoid joint. Arthritis Rheum. 1998;41(10):1760–71.
12. Murphy G, Willenbrock F. Tissue inhibitors of matrix metalloendopeptidases. Methods Enzymol. 1995;248:496–510.
13. Millar AW, Brown PD, Moore J. Results of single and repeat dose studies of the oral matrix metalloproteinase inhibitor marimastat in healthy male volunteers. Br J Clin Pharmacol. 1998;45:21–6.
14. Cooper RR, Misol S. Tendon and ligament insertion. A light and electron microscopic study. J Bone Joint Surg Am. 1970;52(1):1–20.
15. Khan KM, Bonar F, Desmond PM, Cook JL, Young DA, Visentini PJ. Patellar tendinosis (jumper's knee): findings at histopathologic examination, US, and MR imaging. Victorian Institute of Sport Tendon Study Group. Radiology. 1996;200(3):821–7.
16. Clark JM, Harryman DT. Tendons, ligaments, and capsule of the rotator cuff. Gross and microscopic anatomy. J Bone Joint Surg Am. 1992;74(5):713–25.
17. Lapiere CM, Nusgens B, Pierard GE. Interaction between collagen type I and type III in conditioning bundles organization. Connect Tissue Res. 1977;5(1):21–9.
18. Bailey AJ. Tissue and species specificity in the cross-linking of collagen. Pathol Biol. 1974;22(8):675–80.
19. Berenson MC, Blevins FT, Plaas AH, Vogel KG. Proteoglycans of human rotator cuff tendons. J Orthop Res. 1996;14(4):518–25.
20. Riley G. Tendinopathy—from basic science to treatment. Nat Clin Pract Rheumatol. 2008;4(2):82–9.
21. Järvinen M, Józsa L, Kannus P, Järvinen TL, Kvist M, Leadbetter W. Histopathological findings in chronic tendon disorders. Scand J Med Sci Sports. 1997;7(2):86–95.
22. Movin T, Kristoffersen-Wiberg M, Shalabi A, Gad A, Aspelin P, Rolf C. Intratendinous alterations as imaged by ultrasound and contrast medium-enhanced magnetic resonance in chronic achillodynia. Foot Ankle Int. 1998;19(5):311–7.
23. Schubert TEO, Weidler C, Lerch K, Hofstädter F, Straub RH. Achilles tendinosis is associated with sprouting of substance P positive nerve fibres. Ann Rheum Dis. 2005;64(7):1083–6.
24. Gotoh M, Hamada K, Yamakawa H, Tomonaga A, Inoue A, Fukuda H. Significance of granulation tissue in torn supraspinatus insertions: an immunohistochemical study with antibodies against interleukin-1 beta, cathepsin D, and matrix metalloprotease-1. J Orthop Res. 1997;15(1):33–9.
25. Zhang J, Wang JH-C. Production of PGE(2) increases in tendons subjected to repetitive mechanical loading and induces differentiation of tendon stem cells into non-tenocytes. J Orthop Res. 2010;28(2):198–203.
26. Alfredson H, Lorentzon R. Intratendinous glutamate levels and eccentric training in chronic Achilles tendinosis: a prospective study using microdialysis technique. Knee Surg Sports Traumatol Arthrosc J. 2003;11(3):196–9.
27. Uhthoff HK, Sano H. Pathology of failure of the rotator cuff tendon. Orthop Clin North Am. 1997;28(1):31–41.
28. Ker RF, Wang XT, Pike AV. Fatigue quality of mammalian tendons. J Exp Biol. 2000;203(Pt 8):1317–27.
29. Chard MD, Cawston TE, Riley GP, Gresham GA, Hazleman BL. Rotator cuff degeneration and lateral epicondylitis: a comparative histological study. Ann Rheum Dis. 1994;53(1):30–4.
30. Chard MD, Hazleman BL. Tennis elbow—a reappraisal. Br J Rheumatol. 1989;28(3):186–90.
31. Fukuda H, Hamada K, Yamanaka K. Pathology and pathogenesis of bursal-side rotator cuff tears viewed from en bloc histologic sections. Clin Orthop. 1990;254:75–80.
32. Riley GP, Goddard MJ, Hazleman BL. Histopathological assessment and pathological significance of matrix degeneration in supraspinatus tendons. Rheumatology (Oxford). 2001;40(2):229–30.
33. Nixon JE, DiStefano V. Ruptures of the rotator cuff. Orthop Clin North Am. 1975;6(2):423–47.
34. Kjellin I, Ho CP, Cervilla V, Haghighi P, Kerr R, Vangness CT. Alterations in the supraspinatus tendon at MR imaging: correlation with histopathologic findings in cadavers. Radiology. 1991;181(3):837–41.
35. Pettersson G. Rupture of the tendon aponeurosis of the shoulder joint in anterio-inferior disclocation: a study on the origin and occurrence of ruptures. Acta Chir Scand. 1942;87(suppl 77):1–184.
36. Pajala A, Melkko J, Leppilahti J, Ohtonen P, Soini Y, Risteli J. Tenascin-C and type I and III collagen expression in total Achilles tendon rupture. An immunohistochemical study. Histol Histopathol. 2009;24(10):1207–11.
37. Eriksen HA, Pajala A, Leppilahti J, Risteli J. Increased content of type III collagen at the rupture site of human Achilles tendon. J Orthop Res. 2002;20(6):1352–7.
38. Soslowsky LJ, Fryhofer GW. Tendon homeostasis in hypercholesterolemia. Adv Exp Med Biol. 2016;920:151–65.
39. Taylor B, Cheema A, Soslowsky L. Tendon pathology in hypercholesterolemia and familial hypercholesterolemia. Curr Rheumatol Rep. 2017;19(12):76.
40. Beason DP, Abboud JA, Kuntz AF, Bassora R, Soslowsky LJ. Cumulative effects of hypercholesterolemia on tendon biomechanics in a mouse model. J Orthop Res. 2011;29(3):380–3.

41. Beason DP, Tucker JJ, Lee CS, Edelstein L, Abboud JA, Soslowsky LJ. Rat rotator cuff tendon-to-bone healing properties are adversely affected by hypercholesterolemia. J Shoulder Elbow Surg. 2014;23(6):867–72.
42. Lin TT-L, Lin C-H, Chang C-L, Chi C-H, Chang S-T, Sheu WH-H. The effect of diabetes, hyperlipidemia, and statins on the development of rotator cuff disease: a nationwide, 11-year, longitudinal, population-based follow-up study. Am J Sports Med. 2015;43(9):2126–32.
43. Longo UG, Franceschi F, Spiezia F, Forriol F, Maffulli N, Denaro V. Triglycerides and total serum cholesterol in rotator cuff tears: do they matter. Br J Sports Med. 2010;44(13):948–51.
44. Abboud JA, Kim JS. The effect of hypercholesterolemia on rotator cuff disease. Clin Orthop. 2010;468(6):1493–7.
45. Kruth HS. Cholesterol accumulation in vascular smooth muscle cells incorporated into platelet-rich plasma clots. Lab Investig J Tech Methods Pathol. 1985;53(6):634–8.
46. Artieda M, Cenarro A, Junquera C, Lasierra P, Martínez-Lorenzo MJ, Pocoví M. Tendon xanthomas in familial hypercholesterolemia are associated with a differential inflammatory response of macrophages to oxidized LDL. FEBS Lett. 2005;579(20):4503–12.
47. Rönnemaa T, Juva K, Kulonen E. Effect of hyperlipidemic rat serum on the synthesis of collagen by chick embryo fibroblasts. Atherosclerosis. 1975;21(3):315–24.
48. Nunes RLV, Bruschini H, Utsunomia K, Silveira MA, Teodoro WR, Leite KRM. Influence of a hypercholesterolemic diet on the collagen composition of the bladder wall extracellular matrix in rats. Histol Histopathol. 2012;27(6):745–52.
49. Oberkersch R, Maccari F, Bravo AI, Volpi N, Gazzaniga S, Calabrese GC. Atheroprotective remodelling of vascular dermatan sulphate proteoglycans in response to hypercholesterolaemia in a rat model. Int J Exp Pathol. 2014;95(3):181–90.
50. Chung SW, Park H, Kwon J, Choe GY, Kim SH, Oh JH. Effect of hypercholesterolemia on fatty infiltration and quality of tendon-to-bone healing in a rabbit model of a chronic rotator cuff tear: electrophysiological, biomechanical, and histological analyses. Am J Sports Med. 2016;44(5):1153–64.
51. Rolf C, Movin T. Etiology, histopathology, and outcome of surgery in achillodynia. Foot Ankle Int. 1997;18(9):565–9.
52. Franceschi F, Papalia R, Paciotti M, Franceschetti E, Di Martino A, Maffulli N. Obesity as a risk factor for tendinopathy: a systematic review. Int J Endocrinol. 2014;2014:670262.
53. Giai Via A, Papa G, Oliva F, Maffulli N. Tendinopathy. Curr Phys Med Rehabil Rep. 2016.
54. Moor BK, Röthlisberger M, Müller DA, Zumstein MA, Bouaicha S, Ehlinger M. Age, trauma and the critical shoulder angle accurately predict supraspinatus tendon tears. Orthop Traumatol Surg Res. 2014;100(5):489–94.
55. Gerber C, Snedeker JG, Baumgartner D, Viehöfer AF. Supraspinatus tendon load during abduction is dependent on the size of the critical shoulder angle: a biomechanical analysis. J Orthop Res. 2014;32(7):952–7.
56. Nyffeler RW, Werner CML, Sukthankar A, Schmid MR, Gerber C. Association of a large lateral extension of the acromion with rotator cuff tears. J Bone Joint Surg Am. 2006;88(4):800–5.
57. Neer CS. Anterior acromioplasty for the chronic impingement syndrome in the shoulder: a preliminary report. J Bone Joint Surg Am. 1972;54(1):41–50.
58. Bigliani LU, Ticker JB, Flatow EL, Soslowsky LJ, Mow VC. The relationship of acromial architecture to rotator cuff disease. Clin Sports Med. 1991;10(4):823–38.
59. Seitz AL, Michener LA. Ultrasonographic measures of subacromial space in patients with rotator cuff disease: a systematic review. J Clin Ultrasound. 2011;39(3):146–54.
60. Maffulli N, Longo UG, Berton A, Loppini M, Denaro V. Biological factors in the pathogenesis of rotator cuff tears. Sports Med Arthrosc Rev. 2011;19(3):194–201.
61. Soslowsky LJ, Thomopoulos S, Tun S, Flanagan CL, Keefer CC, Mastaw J, Neer Award 1999. Overuse activity injures the supraspinatus tendon in an animal model: a histologic and biomechanical study. J Shoulder Elbow Surg. 2000;9(2):79–84.
62. Soslowsky LJ, Thomopoulos S, Esmail A, Flanagan CL, Iannotti JP, Williamson JD. Rotator cuff tendinosis in an animal model: role of extrinsic and overuse factors. Ann Biomed Eng. 2002;30(8):1057–63.
63. Gia Via A, De Cupis M, Spoliti M, Oliva F. Clinical and biological aspects of rotator cuff tears. Muscles Ligaments Tendons J. 2013;3(2):70–9.
64. Yamaguchi K, Ditsios K, Middleton WD, Hildebolt CF, Galatz LM, Teefey SA. The demographic and morphological features of rotator cuff disease. A comparison of asymptomatic and symptomatic shoulders. J Bone Joint Surg Am. 2006;88(8):1699–704.
65. Carbone S, Gumina S, Arceri V, Campagna V, Fagnani C, Postacchini F. The impact of preoperative smoking habit on rotator cuff tear: cigarette smoking influences rotator cuff tear sizes. J Shoulder Elbow Surg. 2012;21(1):56–60.
66. Lundgreen K, Lian OB, Scott A, Nassab P, Fearon A, Engebretsen L. Rotator cuff tear degeneration and cell apoptosis in smokers versus nonsmokers. Arthrosc J Arthrosc Relat Surg. 2014;30(8):936–41.
67. Baumgarten KM, Gerlach D, Galatz LM, Teefey SA, Middleton WD, Ditsios K. Cigarette smoking increases the risk for rotator cuff tears. Clin Orthop. 2010;468(6):1534–41.
68. Lohr JF, Uhthoff HK. The microvascular pattern of the supraspinatus tendon. Clin Orthop. 1990;254:35–8.

69. Brooks CH, Revell WJ, Heatley FWA. quantitative histological study of the vascularity of the rotator cuff tendon. J Bone Joint Surg Br. 1992;74(1):151–3.
70. Snedeker JG, Gautieri A. The role of collagen crosslinks in ageing and diabetes—the good, the bad, and the ugly. Muscles Ligaments Tendons J. 2014;4(3):303–8.
71. Ahmed N. Advanced glycation endproducts—role in pathology of diabetic complications. Diabetes Res Clin Pract. 2005;67(1):3–21.
72. Li Y, Fessel G, Georgiadis M, Snedeker JG. Advanced glycation end-products diminish tendon collagen fiber sliding. Matrix Biol J Int Soc Matrix Biol. 2013;32(3–4):169–77.
73. Gautieri A, Passini FS, Silván U, Guizar-Sicairos M, Carimati G, Volpi P. Advanced glycation end-products: mechanics of aged collagen from molecule to tissue. Matrix Biol J Int Soc. 2017;59:95–108.
74. Chung SW, Choi BM, Kim JY, Lee Y-S, Yoon JP, Oh K-S. Altered gene and protein expressions in torn rotator cuff tendon tissues in diabetic patients. Arthrosc J Arthrosc Relat Surg. 2017;33(3):518–526.e1.
75. Duncan WS. Relationship of thyroid disease to chronic nonspecific arthritis. JAMA. 1932;99(15):1239.
76. Oliva F, Osti L, Padulo J, Maffulli N. Epidemiology of the rotator cuff tears: a new incidence related to thyroid disease. Muscles Ligaments Tendons J. 2014;4(3):309–14.
77. Oliva F, Berardi AC, Misiti S, Verga Falzacappa C, Falzacappa CV, Iacone A. Thyroid hormones enhance growth and counteract apoptosis in human tenocytes isolated from rotator cuff tendons. Cell Death Dis. 2013;4:e705.
78. Berardi AC, Oliva F, Berardocco M, la Rovere M, Accorsi P, Maffulli N. Thyroid hormones increase collagen I and cartilage oligomeric matrix protein (COMP) expression in vitro human tenocytes. Muscles Ligaments Tendons J. 2014;4(3):285–91.
79. Beason DP, Hsu JE, Marshall SM, McDaniel AL, Temel RE, Abboud JA. Hypercholesterolemia increases supraspinatus tendon stiffness and elastic modulus across multiple species. J Shoulder Elbow Surg. 2013;22(5):681–6.
80. Beason DP, Hsu JE, Edelstein L, Lee CS, Tucker JJ, Abboud JA. Effect of diet-induced hypercholesterolemia on rotator cuff tendon mechanics in a rat model. Trans Orthop Res Soc. 2011;36:22.
81. Bhattacharyya AK, Connor WE, Mausolf FA, Flatt AD. Turnover of xanthoma cholesterol in hyperlipoproteinemia patients. Lab Clin Med. 1976;87:503–18.
82. Tashjian RZ, Farnham JM, Albright FS, Teerlink CC, Cannon-Albright LA. Evidence for an inherited predisposition contributing to the risk for rotator cuff disease. J Bone Joint Surg Am. 2009;91(5):1136–42.
83. Harvie P, Ostlere SJ, Teh J, McNally EG, Clipsham K, Burston BJ. Genetic influences in the aetiology of tears of the rotator cuff. Sibling risk of a full-thickness tear. J Bone Joint Surg Br. 2004;86(5):696–700.
84. Tashjian RZ, Farnham JM, Granger EK, Teerlink CC, Cannon-Albright LA. Evidence for an environmental and inherited predisposition contributing to the risk for global tendinopathies or compression neuropathies in patients with rotator cuff tears. Orthop J Sports Med. 2016;4(4):2325967116642173.
85. Molloy TJ, Kemp MW, Wang Y, Murrell G. Microarray analysis of the tendinopathic rat supraspinatus tendon: glutamate signaling and its potential role in tendon degeneration. J Appl Physiol. 2006;101(6):1702–9.
86. Hynd MR, Scott HL, Dodd PR. Glutamate-mediated excitotoxicity and neurodegeneration in Alzheimer's disease. Neurochem Int. 2004;45(5):583–95.
87. Skerry TM. Identification of novel signaling pathways during functional adaptation of the skeleton to mechanical loading: the role of glutamate as a paracrine signaling agent in the skeleton. J Bone Miner Metab. 1999;17(1):66–70.
88. Orth T, Paré J, Froehlich JE. Current concepts on the genetic factors in rotator cuff pathology and future implications for sports physical therapists. Int J Sports Phys Ther. 2017;12(2):273–85.
89. Osti L, Buda M, Del Buono A, Osti R, Massari L, Maffulli N. Apoptosis and rotator cuff tears: scientific evidence from basic science to clinical findings. Br Med Bull. 2017;122(1):123–33.
90. Yuan J, Murrell GAC, Wei A-Q, Wang M-X. Apoptosis in rotator cuff tendonopathy. J Orthop Res. 2002;20(6):1372–9.
91. Wu B, Chen J, Rosa TD, Yu Q, Wang A, Xu J. Cellular response and extracellular matrix breakdown in rotator cuff tendon rupture. Arch Orthop Trauma Surg. 2011;131(3):405–11.
92. Suzanne M, Steller H. Letting go: modification of cell adhesion during apoptosis. J Biol. 2009;8(5):49.
93. Wolf BB, Green DR. Suicidal tendencies: apoptotic cell death by caspase family proteinases. J Biol Chem. 1999;274(29):20049–52.
94. Lee H-J, Kim Y-S, Ok J-H, Song H-J. Apoptosis occurs throughout the diseased rotator cuff. Am J Sports Med. 2013;41(10):2249–55.
95. Riley GP, Curry V, DeGroot J, van El B, Verzijl N, Hazleman BL. Matrix metalloproteinase activities and their relationship with collagen remodelling in tendon pathology. Matrix Biol J Int Soc Matrix Biol. 2002;21(2):185–95.
96. Castagna A, Cesari E, Garofalo R, Gigante A, Conti M, Markopoulos N. Matrix metalloproteases and their inhibitors are altered in torn rotator cuff tendons, but also in the macroscopically and histologically intact portion of those tendons. Muscles Ligaments Tendons J. 2013;3(3):132–8.
97. Tashjian RZ, Granger EK, Farnham JM, Cannon-Albright LA, Teerlink CC. Genome-wide association study for rotator cuff tears identifies two significant single-nucleotide polymorphisms. J Shoulder Elbow Surg. 2016;25(2):174–9.

98. Tashjian RZ, Granger EK, Zhang Y, Teerlink CC, Cannon-Albright LA. Identification of a genetic variant associated with rotator cuff repair healing. J Shoulder Elbow Surg. 2016;25(6):865–72.
99. Zumstein M-A, Lädermann A, Raniga S, Schär M-O. The biology of rotator cuff healing. Orthop Traumatol Surg Res. 2017;103(1S):S1–10.
100. Beredjiklian PK. Biologic aspects of flexor tendon laceration and repair. J Bone Joint Surg Am. 2003;85-A(3):539–50.
101. Andrianjafiniony T, Dupré-Aucouturier S, Letexier D, Couchoux H, Desplanches D. Oxidative stress, apoptosis, and proteolysis in skeletal muscle repair after unloading. Am J Physiol Cell Physiol. 2010;299(2):C307–15.
102. Arnold L, Henry A, Poron F, Baba-Amer Y, van Rooijen N, Plonquet A. Inflammatory monocytes recruited after skeletal muscle injury switch into antiinflammatory macrophages to support myogenesis. J Exp Med. 2007;204(5):1057–69.
103. Murphy PG, Loitz BJ, Frank CB, Hart DA. Influence of exogenous growth factors on the synthesis and secretion of collagen types I and III by explants of normal and healing rabbit ligaments. Biochem Cell Biol Biochim Biol Cell. 1994;72(9–10):403–9.
104. Hays PL, Kawamura S, Deng X-H, Dagher E, Mithoefer K, Ying L. The role of macrophages in early healing of a tendon graft in a bone tunnel. J Bone Joint Surg Am. 2008;90(3):565–79.
105. Zanou N, Gailly P. Skeletal muscle hypertrophy and regeneration: interplay between the myogenic regulatory factors (MRFs) and insulin-like growth factors (IGFs) pathways. Cell Mol Life Sci. 2013;70(21):4117–30.
106. Frey E, Regenfelder F, Sussmann P, Zumstein M, Gerber C, Born W. Adipogenic and myogenic gene expression in rotator cuff muscle of the sheep after tendon tear. J Orthop Res. 2009;27(4):504–9.
107. Lee YS, Jeong JY, Park C-D, Kang SG, Yoo JC. Evaluation of the risk factors for a rotator cuff retear after repair surgery. Am J Sports Med. 2017;45(8):1755–61.
108. Tashjian RZ, Hollins AM, Kim H-M, Teefey SA, Middleton WD, Steger-May K. Factors affecting healing rates after arthroscopic double-row rotator cuff repair. Am J Sports Med. 2010;38(12):2435–42.
109. Diebold G, Lam P, Walton J, Murrell GAC. Relationship between age and rotator cuff retear: a study of 1,600 consecutive rotator cuff repairs. J Bone Joint Surg Am. 2017;99(14):1198–205.
110. Kwon J, Kim SH, Lee YH, Kim TI, Oh JH. The rotator cuff healing index: a new scoring system to predict rotator cuff healing after surgical repair. Am J Sports Med. 2019;47(1):173–80.
111. Chillemi C, Petrozza V, Garro L, Sardella B, Diotallevi R, Ferrara A. Rotator cuff re-tear or non-healing: histopathological aspects and predictive factors. Knee Surg Sports Traumatol Arthrosc. 2011;19(9):1588–96.
112. Plate JF, Brown PJ, Walters J, Clark JA, Smith TL, Freehill MT. Advanced age diminishes tendon-to-bone healing in a rat model of rotator cuff repair. Am J Sports Med. 2014;42(4):859–68.
113. Galatz LM, Silva MJ, Rothermich SY, Zaegel MA, Havlioglu N, Thomopoulos S. Nicotine delays tendon-to-bone healing in a rat shoulder model. J Bone Joint Surg Am. 2006;88(9):2027–34.
114. Park JH, Oh K-S, Kim TM, Kim J, Yoon JP, Kim JY. Effect of smoking on healing failure after rotator cuff repair. Am J Sports Med. 2018;46(12):2960–8.
115. Bedi A, Fox AJS, Harris PE, Deng X-H, Ying L, Warren RF. Diabetes mellitus impairs tendon-bone healing after rotator cuff repair. J Shoulder Elbow Surg. 2010;19(7):978–88.
116. Chen AL, Shapiro JA, Ahn AK, Zuckerman JD, Cuomo F. Rotator cuff repair in patients with type I diabetes mellitus. J Shoulder Elbow Surg. 2003;12(5):416–21.
117. Garcia GH, Liu JN, Wong A, Cordasco F, Dines DM, Dines JS. Hyperlipidemia increases the risk of retear after arthroscopic rotator cuff repair. J Shoulder Elbow Surg. 2017;26(12):2086–90.
118. Tanaka K, Kanazawa T, Gotoh M, Tanesue R, Nakamura H, Ohzono H. Effects of estrogen-deficient state on rotator cuff healing. Am J Sports Med. 2019;47(2):389–97.
119. Angeline ME, Ma R, Pascual-Garrido C, Voigt C, Deng XH, Warren RF. Effect of diet-induced vitamin D deficiency on rotator cuff healing in a rat model. Am J Sports Med. 2014;42(1):27–34.
120. Ryu KJ, Kim BH, Lee Y, Dan J, Kim JH, Low Serum Vitamin D. Is not correlated with the severity of a rotator cuff tear or retear after arthroscopic repair. Am J Sports Med. 2015;43(7):1743–50.
121. Randelli P, Cucchi D, Ragone V, de Girolamo L, Cabitza P, Randelli M. History of rotator cuff surgery. Knee Surg Sports Traumatol Arthrosc J. 2015;23(2):344–62.
122. Spennacchio P, Banfi G, Cucchi D, D'Ambrosi R, Cabitza P, Randelli P. Long-term outcome after arthroscopic rotator cuff treatment. Knee Surg Sports Traumatol Arthrosc J. 2015;23(2):523–9.
123. Walter SG, Stadler T, Thomas TS, Thomas W. Advanced rotator cuff tear score (ARoCuS): a multi-scaled tool for the classification and description of rotator cuff tears. Musculoskelet Surg. 2019;103(1):37–45.
124. Milano G, Saccomanno MF, Careri S, Taccardo G, De Vitis R, Fabbriciani C. Efficacy of marrow-stimulating technique in arthroscopic rotator cuff repair: a prospective randomized study. Arthrosc J Arthrosc Relat Surg. 2013;29(5):802–10.
125. Kida Y, Morihara T, Matsuda K-I, Kajikawa Y, Tachiiri H, Iwata Y. Bone marrow-derived cells from the footprint infiltrate into the repaired rotator cuff. J Shoulder Elbow Surg. 2013;22(2):197–205.
126. Jo CH, Shin JS, Park IW, Kim H, Lee SY. Multiple channeling improves the structural integ-

rity of rotator cuff repair. Am J Sports Med. 2013;41(11):2650–7.
127. Nakagawa H, Morihara T, Fujiwara H, Kabuto Y, Sukenari T, Kida Y. Effect of footprint preparation on tendon-to-bone healing: a histologic and biomechanical study in a rat rotator cuff repair model. Arthrosc J Arthrosc Relat Surg. 2017;33(8):1482–92.
128. Gereli A, Kocaoglu B, Ulku TK, Silay S, Kilinc E, Uslu S. u. a. Completion repair exhibits increased healing characteristics compared with in situ repair of partial thickness bursal rotator cuff tears. Knee Surg Sports Traumatol Arthrosc. 2018;26(8):2498–504.
129. Cavinatto L, Malavolta EA, Pereira CAM, Miranda-Rodrigues M, Silva LCM, Gouveia CH. Early versus late repair of rotator cuff tears in rats. J Shoulder Elbow Surg. 2018;27(4):606–13.
130. Mukovozov I, Byun S, Farrokhyar F, Wong I. Time to surgery in acute rotator cuff tear: a systematic review. Bone Jt Res. 2013;2(7):122–8.
131. Ohzono H, Gotoh M, Nakamura H, Honda H, Mitsui Y, Kakuma T. Effect of preoperative fatty degeneration of the rotator cuff muscles on the clinical outcome of patients with intact tendons after arthroscopic rotator cuff repair of large/massive cuff tears. Am J Sports Med. 2017;45(13):2975–81.
132. Jeong HY, Kim HJ, Jeon YS, Rhee YG. Factors predictive of healing in large rotator cuff tears: is it possible to predict retear preoperatively. Am J Sports Med. 2018;46(7):1693–700.
133. Riboh JC, Garrigues GE. Early passive motion versus immobilization after arthroscopic rotator cuff repair. Arthrosc J Arthrosc Relat Surg. 2014;30(8):997–1005.
134. Houck DA, Kraeutler MJ, Schuette HB, McCarty EC, Bravman JT. Early versus delayed motion after rotator cuff repair: a systematic review of overlapping meta-analyses. Am J Sports Med. 2017;45(12):2911–5.

3 Biomechanics of Rotator Cuff Repair

Olaf Lorbach

3.1 Introduction

Arthroscopic and open repair of the rotator cuff leads to good clinical and functional results. In order to achieve a successful repair, detailed knowledge of the biomechanics of the intact as well as ruptured rotator cuff and the superior capsule is essential.

The superior capsule of the glenohumeral joint is a thin fibrous structure which is attached to 30–61% of the greater tuberosity [25, 53, 61].

The rotator cable, or semicircular humeral ligament, courses from the anterior supraspinatus and upper subscapularis to the inferior infraspinatus and is located on the perimeter of the avascular zone [57, 63]. It is in continuity with the superior capsule and prevents excess stress from being transferred from the rotator cuff to the thin aspects of the capsule [6, 7]. As the rotator cable attachments are thickenings of the superior capsule, superior capsular integrity may be essential for maintenance of normal biomechanics and kinematics of the shoulder joint [1].

The rotator cable creates a suspension bridge phenomenon that may explain why patients with massive cuff tears or also after partial repair of the rotator cuff may show good to excellent shoulder function if the rotator cable remained intact or could be repaired [6, 15, 24].

Especially in massive tears, delamination of the superior capsule is more frequent. Therefore, in these massive tears of the rotator cuff, it may be of more importance to address both layers (rotator cuff and superior capsule) separately in order to improve the healing rate of the repair construct [1].

3.2 Biomechanics of the Intact Rotator Cuff

The rotator cuff muscles work as a motion actuator (abduction, external, and internal rotations). Moreover, they have an important impact on stabilization of the shoulder joint. In the end range of shoulder motion (abduction and maximum external rotation), the capsuloligamentous structures such as the inferior glenohumeral ligament contribute significantly to shoulder stability whereas in the mid-range of shoulder motion, where the capsuloligamentous structures are more lax, shoulder stability is mainly provided by the glenoid concavity and the compressive force generated by the rotator cuff muscles [32].

During shoulder motion, the anterior (M. subscapularis) as well as the posterior (M. infraspinatus and M. teres minor) rotator cuff mainly contribute to keep the humeral head in the glenoid socket. Considering that the cross-sectional

O. Lorbach (✉)
Department of Shoulder Surgery and Sports Medicine, Schön-Clinic Lorsch, Lorsch, Germany

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_3

areas of the anterior and posterior rotators are approximately equal [27], it seems viable that the torques generated by these two groups are balanced and represent something like a force couple that resists humeral head translation [68].

The deltoid muscle and the rotator cuff muscles work together in order to perform the physiological shoulder movement without any significant changes of the center of rotation. During abduction by the deltoid muscle, the rotator cuff keeps the humerus centered in the glenoid socket and prevents further migration of the humeral head [22]. The role of the supraspinatus is described as a compressive force; the subscaplaris balances the anterior aspect of the shoulder whereas the posterior rotator cuff (M. infraspinatus and M. teres minor) balances the posterior part of the shoulder [4, 5, 67].

3.3 Biomechanics of the Intact Capsule

When the shoulder is abducted, the deltoid applies a superiorly directed force to the proximal humerus, whereas the rotator cuff muscles apply a force vector that is directed medially and inferiorly to the humeral head. Balancing these coupled forces prevents proximal migration of the humerus. At the end range of motion, the glenohumeral ligaments passively tighten or relax to maintain the position of the humeral head within the glenoid cavity, with the rotator cuff muscles providing secondary dynamic stabilization [23, 48].

3.4 Biomechanics of the Ruptured Rotator Cuff

Intrinsic changes or ruptures of the rotator cuff tendons may lead to a significant disturbance of the shoulder kinematics, of shoulder joint stabilization, and may further lead to subacromial impingement [22].

Burkhart et al. [5] reported three different types based on the localization and extent of the rotator cuff rupture:

- **Type I lesion:** Rotator cuff rupture with stable fulcrum. The supraspinatus tendon is ruptured with contribution of the upper M. infraspinatus. A normal kinematic of the shoulder joint is still present.
- **Type II lesion:** The fulcrum becomes unstable, if a complete rupture of the cranial (supraspinatus tendon) as well as the dorsal rotator cuff occurs (M. infraspinatus). The force couple is uncoupled preventing the centralization of the humeral head.
- **Type III lesion:** permanent static destabilization (captured fulcrum). A rupture of the supraspinatus, as least one third of the posterior rotator cuff as well as half of the subscapularis muscle is present.

3.5 Biomechanics of Rotator Cuff Repairs

The ideal biomechanical rotator cuff repair should provide low gap formation under cyclic loading with a high ultimate failure load [18]. The biomechanical performance of the construct is potentially influenced by many factors like the anchor material and design, the suture material, the suture configuration, and the surgical technique (double-row vs. single-row repair) [38].

With the introduction of a second row of suture anchors, the so-called double-row repair was described with superior biomechanical findings compared to single-row repairs [3, 28, 43, 51, 55].

However, these tested double-row repairs were compared to simple suture repairs. As the suture tendon interface is the weakest part of the linkage, it seems viable to compare double-row constructs to single-row repairs using modified suture configurations.

Single-row repairs using modified suture configurations were able to show comparable biomechanical findings to double-row repairs with no significant differences in ultimate load-to-failure [13, 33–37, 39–41, 49, 59] or cyclic displacement [33–37, 39, 49, 59].

The transosseous equivalent rotator cuff repair (TOE) is a modification of the initially used

two separated rows of suture anchors and was described with several potential benefits compared to single-row as well as "simple" double-row repairs such as decreased knot impingement, improved footprint coverage as well as superior biomechanical properties. However, TOE rotator cuff repairs were also associated with several concerns about possible tendon strangulation and necrosis [11], the potential risk of over-tensioning the construct medially [52] as well as a reduced intratendinous blood flow [12]. Moreover, the technique seems to be highly associated with a difference in the re-tear pattern where the construct fails at the musculotendinous junction which is associated with a more difficult re-reconstruction in a failure situation [11, 19, 55, 62, 64].

Especially in chronic massive tears with a decreased remnant tendon length (<10 mm), it may be more viable to use a single-row repair as double-row repair was significantly associated with a higher re-rupture rate for TOE double-row repairs compared to single-row repairs (46% vs. 6%) [29].

Furthermore, despite any potential benefit in biomechanical studies, no significant differences were further seen in the majority of clinical studies, neither in the clinical scores [2, 9, 10, 14, 16, 19, 21, 30, 54, 56, 58, 65] nor in the reported re-rupture rate evaluated by CT-arthrogram[7] or MRI [9, 14, 16, 30]. Merely superior structural results were described for the double-row constructs concerning the quality of the repair as well as the rate of partial re-tears [10, 14, 60]. These results were mainly found in subgroup analysis of small patient cohorts.

Therefore, published evidence of biomechanical as well as clinical superiority for any type of double-row repairs compared to single-row repairs is, based on the current literature, at least questionable. As the weakest point of the repair is the suture tendon interface, the biomechanical performance is more dependent on the number of sutures, which penetrate the tendon as well as the suture configuration and not by the number of anchors in the rotator cuff insertion. Moreover, the stich position may further play an important role as positioning the stitch just medial to the rotator cable seems to provide the most sufficient biomechanical results [66]. High level studies are needed in order to further support a potential benefit of the double row construct compared to simple sutures as well as single-row repairs in order to justify the increased surgical time as well as increased implant costs for the double row constructs.

Published evidence of rotator cuff repairs has been investigated in a systematic review of 2383 articles (1980–2012) of those 108 met inclusion criteria.

Inspite of the dramatic increase in publications per year as well as the high amount of introduced new implants as well as surgical technique modifications, little evidence is present that the results of rotator cuff repairs are improving over the years with a mean re-tear rate of 27% at 2 year follow-up [42].

Therefore, biology seems to be the most important affecting factor concerning the healing rate of the repaired rotator cuff.

Moreover, not every rupture of the rotator cuff is still repairable. Fatty infiltration of the RC muscles is described as one of the most important predicting factors [20]. Meyer et al. [44] further described a reduced tendon length (<15 mm) as an important factor leading to failure of the repairs in 2/3 of the patients. A positive tangent sign according to Zanetti [70] is reported as an important prognostic factor as well. If a significant static instability of the shoulder is already present, this is described as a contraindication for rotator cuff repair [31].

Patients with an exhibited re-tear of the rotator cuff may have inferior clinical outcome compared to patients with an intact tendon after repair [69]. However, failure of the repaired construct is not generally associated with inferior clinical results. It is described to significantly decrease pain and increase function and strength even if the repair has failed suggesting that the potential for re-rupture should not be considered as a contraindication for repair [26].

Therefore, in patients where an anatomic repair is uncertain or the suggested re-tear rate is high; a partial rotator cuff repair may still be recommended. The aim of the partial repair is the restoration of the force couple in the shoulder with

refixation of the rotator cable attachments. The technique included the refixation of the often involved upper subscapularis tendon as well as the posterior rotator cuff (infraspinatus muscle). The use of additional side-to-side repair sutures between the anterior and posterior rotator cuff may further reduce the remaining defect to a subtotal or complete (nonanatomic) repair in the majority of cases. Moreover, the applied forces especially on the repaired posterior rotator cuff can be reduced which may have a significant impact on rotator cuff healing as well [50].

Galasso et al. [17] could show that partial repair of the rotator cuff led to improved shoulder function and pain reduction even in the long term. Different from pure symptomatic procedures, it was able to at least partially restore shoulder joint functionality [17].

Furthermore, the concept of partial rotator cuff repair may be combined with reconstruction of the superior capsule which is reported as a new concept in the treatment of irreparable tears of the rotator cuff [45–47].

3.6 The Biomechanical Concept of Superior Capsule Reconstruction

The biomechanical concept of reconstruction of the superior capsule in irreparable rotator cuff tears was described by Mihata and coworkers [45–47]. They could show in a biomechanical study that complete discontinuity of the supraspinatus muscle with superior capsule leads to increased superior translation, increased subacromial contact pressure, and reduction of the glenohumeral contact pressure. Interposition patch autograft was only able to partially restore shoulder kinematics to the intact level, whereas fixation of the graft at the rotator cuff footprint on the humeral head as well as at the superior glenoid, miming the physiological insertion of the superior capsule, resulted in complete restoration of shoulder kinematics [45].

Even if a superior capsule reconstruction in combination with a partial repair of the rotator cuff is not able to achieve a normal muscle function, a type III or type II lesion may be transferred into a type I lesion resulting in an acceptable shoulder function and even the reversal of the pseudoparalysis in selected cases [8].

3.7 Summary

In order to adequately treat even complex tears of the rotator cuff, profound anatomic and biomechanical knowledge is necessary in order to restore shoulder kinematics by a complete repair or a partial repair with or without concomitant superior capsule reconstruction.

Acknowledgments *Conflicts of Interest*: No potential conflict of interest declared.

References

1. Adams CR, DeMartino AM, Rego G, Denard PJ, Burkhart SS. The rotator cuff and the superior capsule: why we need both. Arthroscopy. 2016;32(12):2628–37.
2. Aydin N, Kocaoglu B, Guven O. Single-row versus double-row arthroscopic rotator cuff repair in small- to medium-sized tears. J Shoulder Elbow Surg. 2010;19:722–5.
3. Baums MH, Spahn G, Steckel H, et al. Comparative evaluation of the tendon-bone interface contact pressure in different single- versus double-row suture anchor repair techniques. Knee Surg Sports Traumatol Arthrosc. 2009;17:1466–72.
4. Burkhart SS. Arthroscopic treatment of massive rotator duff tears: clinical results an biomechanical rationale. Clin Orthop. 1991;267:45–56.
5. Burkhart SS. Fluoroscopic comparison of kinematic patterns in massive rotator cuff tears. A suspension bridge model. Clin Orthop Relat Res. 1992;(284):144–52.
6. Burkhart SS, Esch JC, Jolson SR. The rotator crescent and rotator cable: an anatomic description of the shoulder's "suspension bridge". Arthroscopy. 1993;9:611–6.
7. Burkhart SS. Reconciling the paradox of rotator cuff repair versus debridement: a unified biomechanical rationale for the treatment of rotator cuff tears. Arthroscopy. 1994;10:4–19.
8. Burkhart SS, Hartzler RU. Superior capsular reconstruction reverses profound pseudoparalysis in patients with irreparable rotator cuff tears and minimal or no glenohumeral arthritis. Arthroscopy. 2019;35(1):22–8.

9. Burks RT, Crim J, Brown N, Fink B, Greis PE. A prospective randomized clinical trial comparing arthroscopic single- and double-row rotator cuff repair: magnetic resonance imaging and early clinical evaluation. Am J Sports Med. 2009;37:674–82.
10. Charousset C, Grimberg J, Duranthon LD, Bellaiche L, Petrover D. Can a double-row anchorage technique improve tendon healing in arthroscopic rotator cuff repair?: a prospective, nonrandomized, comparative study of double-row and single-row anchorage techniques with computed tomographic arthrography tendon healing assessment. Am J Sports Med. 2007;35:1247–53.
11. Cho NS, Lee BG, Rhee YG. Arthroscopic rotator cuff repair using a suture bridge technique: is the repair integrity actually maintained? Am J Sports Med. 2011;39:2108–16.
12. Christoforetti JJ, Krupp RJ, Singleton SB, et al. Arthroscopic suture bridge transosseus equivalent fixation of rotator cuff tendon preserves intra-tendinous blood flow at the time of initial fixation. J Shoulder Elbow Surg. 2012;21(4):523–30.
13. Cummins CA, Appleyard RC, Strickland S, et al. Rotator cuff repair: an ex vivo analysis of suture anchor repair techniques on initial load to failure. Arthroscopy. 2005;21:1236–41.
14. Dehaan AM, Axelrad TW, Kaye E, et al. Does double-row rotator cuff repair improve functional outcome of patients compared with single-row technique?: a systematic review. Am J Sports Med. 2012;40:1176–85.
15. Denard PJ, Koo SS, Murena L, Burkhart SS. Pseudoparalysis: the importance of rotator cable integrity. Orthopedics. 2012;35:e1353–7.
16. Franceschi F, Ruzzini L, Longo UG, et al. Equivalent clinical results of arthroscopic single-row and double-row suture anchor repair for rotator cuff tears: a randomized controlled trial. Am J Sports Med. 2007;35:1254–60.
17. Galasso O, Riccelli DA, De Gori M, De Benedetto M, Orlando N, Gasparini G, Castricini R. Quality of life and functional results of arthroscopic partial repair of irreparable rotator cuff tears. Arthroscopy. 2017;33(2):261–8.
18. Gerber C, Schneeberger AG, Beck M, Schlegel U. Mechanical strength of repairs of the rotator cuff. J Bone Joint Surg Br. 1994;76:371–80.
19. Gerhardt C, Hug K, Pauly S, Marnitz T, Scheibel M. Arthroscopic single-row modified mason-allen repair versus double-row suture bridge reconstruction for supraspinatus tendon tears: a matched-pair analysis. Am J Sports Med. 2012;40:2777–85.
20. Goutallier D, Postel JM, Bernageau J, Lavau L, Voisin MC. Fatty muscle degeneration in cuff ruptures. Pre- and postoperative evaluation by CT scan. Clin Orthop Relat Res. 1994;(304):78–83.
21. Grasso A, Milano G, Salvatore M, et al. Single-row versus double-row arthroscopic rotator cuff repair: a prospective randomized clinical study. Arthroscopy. 2009;25:4–12.
22. Habermeyer P, Lichtenberg S, Magosch P, editors. Schulterchirurgie. 4th ed. München: Urban & Fischer, Elsevier GmbH; 2010.
23. Halder A, Kuhl S, Zobitz M, Larson D, An K. Effects of the glenoid labrum and glenohumeral abduction on stability of the shoulder joint through concavity-compression: an in vitro study. J Bone Joint Surg Am. 2001;83:1062–9.
24. Halder AM, O'Driscoll SW, Heers G, et al. Biomechanical comparison of effects of supraspinatus tendon detachments, tendon defects, and muscle retractions. J Bone Joint Surg Am. 2002;84:780–5.
25. Ishihara Y, Mihata T, Tamboli M, et al. Role of the superior shoulder capsule in passive stability of the glenohumeral joint. J Shoulder Elbow Surg. 2014;23:642–8.
26. Jost B, Pfirrmann CW, Gerber C, Switzerland Z. Clinical outcome after structural failure of rotator cuff repairs. J Bone Joint Surg Am. 2000;82(3):304–14.
27. Keating JF, Waterworth P, Shaw-Dunn J, Crossan J. The relative strengths of the rotator cuff muscles. A cadaver study. J Bone Joint Surg Br. 1993;75:137e140.
28. Kim DH, Elattrache NS, Tibone JE, et al. Biomechanical comparison of a single-row versus double-row suture anchor technique for rotator cuff repair. Am J Sports Med. 2006;34:407–14.
29. Kim YK, Moon SH, Cho SH. Treatment outcomes of single- versus double-row repair for larger than medium-sized rotator cuff tears: the effect of preoperative remnant tendon length. Am J Sports Med. 2013;41:2270–7.
30. Koh KH, Kang KC, Lim TK, Shon MS, Yoo JC. Prospective randomized clinical trial of single- versus double-row suture anchor repair in 2- to 4-cm rotator cuff tears: clinical and magnetic resonance imaging results. Arthroscopy. 2011;27:453–62.
31. Lädermann A, Denard PJ, Collin P. Massive rotator cuff tears: definition and treatment. Int Orthop. 2015;39(12):2403–14.
32. Lippitt SB, Vanderhooft JE, Harris SL, Sidles JA, Harryman DT II, Matsen FA III. Glenohumeral stability from concavity-compression: a quantitative analysis. J Shoulder Elbow Surg. 1993;2:27e35.
33. Lorbach O, Anagnostakos K, Vees J, Kohn D, Pape D. Three-dimensional evaluation of the cyclic loading behavior of different rotator cuff reconstructions. Arthroscopy. 2010;26:S95–105.
34. Lorbach O, Bachelier F, Vees J, Kohn D, Pape D. Cyclic loading of rotator cuff reconstructions: single-row repair with modified suture configurations versus double-row repair. Am J Sports Med. 2008;36:1504–10.
35. Lorbach O, Kieb M, Raber F, et al. Comparable biomechanical results for a modified single-row rotator cuff reconstruction using triple-loaded suture anchors versus a suture-bridging double-row repair. Arthroscopy. 2012a;28:178–87.

36. Lorbach O, Kieb M, Raber F, Kohn D, Pape D. Three-dimensional evaluation of cyclic displacement in single-row and double-row rotator cuff reconstructions under a static external rotation. Am J Sports Med. 2013;41(1):153–62.
37. Lorbach O, Pape D, Raber F, et al. Influence of the initial rupture size and tendon subregion on three-dimensional biomechanical properties of single-row and double-row rotator cuff reconstructions. Knee Surg Sports Traumatol Arthrosc. 2012b;20(11):2139–47.
38. Lorbach O. Rotator cuff repair. Why it fails. In: Milano G, Grasso A, Calvo A, Brzoska R, editors. Management of failed shoulder surgery. New York, NY: Springer; 2018. p. 201–5. Chapter 17.
39. Ma CB, Comerford L, Wilson J, Puttlitz CM. Biomechanical evaluation of arthroscopic rotator cuff repairs: double-row compared with single-row fixation. J Bone Joint Surg Am. 2006;88:403–10.
40. Mahar A, Tamborlane J, Oka R, Esch J, Pedowitz RA. Single-row suture anchor repair of the rotator cuff is biomechanically equivalent to double-row repair in a bovine model. Arthroscopy. 2007;23:1265–70.
41. Mazzocca AD, Millett PJ, Guanche CA, Santangelo SA, Arciero RA. Arthroscopic single-row versus double-row suture anchor rotator cuff repair. Am J Sports Med. 2005;33:1861–8.
42. McElvany MD, McGoldrick E, Gee AO, Neradilek MB, Matsen FA III. Rotator cuff repair: published evidence on factors associated with repair integrity and clinical outcome. Am J Sports Med. 2015;43(2):491–500.
43. Meier SW, Meier JD. The effect of double-row fixation on initial repair strength in rotator cuff repair: a biomechanical study. Arthroscopy. 2006;22:1168–73.
44. Meyer DC, Wieser K, Farshad M, Gerber C. Retraction of supraspinatus muscle and tendon as predictors of success of rotator cuff repair. Am J Sports Med. 2012;40(10):2242–7.
45. Mihata T, McGarry MH, Pirolo JM, Kinoshita M, Lee TQ. Superior capsule reconstruction to restore superior stability in irreparable rotator cuff tears: a biomechanical cadaveric study. Am J Sports Med. 2012;40(10):2248–55.
46. Mihata T, McGarry MH, Kahn T, Goldberg I, Neo M, Lee TQ. Biomechanical role of capsular continuity in superior capsule reconstruction for irreparable tears of the supraspinatus tendon. Am J Sports Med. 2016a;44(6):1423–30.
47. Mihata T, McGarry MH, Kahn T, Goldberg I, Neo M, Lee TQ. Biomechanical effect of thickness and tension of fascia lata graft on glenohumeral stability for superior capsule reconstruction in irreparable supraspinatus tears. Arthroscopy. 2016b;32(3):418–26.
48. Milano G, Grasso A. Shoulder arthroscopy: principles and practice. 1st ed. London: Springer; 2013.
49. Nelson CO, Sileo MJ, Grossman MG, Serra-Hsu F. Single-row modified mason-allen versus double-row arthroscopic rotator cuff repair: a biomechanical and surface area comparison. Arthroscopy. 2008;24:941–8.
50. Oh JH, McGarry MH, Jun BJ, Gupta A, Chung KC, Hwang J, Lee TQ. Restoration of shoulder biomechanics according to degree of repair completion in a cadaveric model of massive rotator cuff tear: importance of margin convergence and posterior cuff fixation. Am J Sports Med. 2012;40(11):2448–53.
51. Park MC, Idjadi JA, Elattrache NS, et al. The effect of dynamic external rotation comparing 2 footprint-restoring rotator cuff repair techniques. Am J Sports Med. 2008;36:893–900.
52. Park MC, McGarry MH, Gunzenhauser RC, et al. Does transosseous-equivalent rotator cuff repair biomechanically provide a “self-reinforcement” effect compared with single-row repair? J Shoulder Elbow Surg. 2014;23(12):1813–21.
53. Pouliart N, Somers K, Eid S, Gagey O. Variations in the superior capsuloligamentous complex and description of a new ligament. J Shoulder Elbow Surg. 2007;16:821–36.
54. Reardon DJ, Maffulli N. Clinical evidence shows no difference between single- and double-row repair for rotator cuff tears. Arthroscopy. 2007;23:670–3.
55. Rhee YG, Cho NS, Parke CS. Arthroscopic rotator cuff repair using modified Mason-Allen medial row stitch: knotless versus knot-tying suture bridge technique. Am J Sports Med. 2012;40:2440–7.
56. Saridakis P, Jones G. Outcomes of single-row and double-row arthroscopic rotator cuff repair: a systematic review. J Bone Joint Surg Am. 2010;92:732–42.
57. Sheah K, Bredella MA, Warner JJ, Halpern EF, Palmer WE. Transverse thickening along the articular surface of the rotator cuff consistent with the rotator cable: identification with MR arthrography and relevance in rotator cuff evaluation. AJR Am J Roentgenol. 2009;193:679–86.
58. Sheibani-Rad S, Giveans MR, Arnoczky SP, Bedi A. Arthroscopic single-row versus double-row rotator cuff repair: a meta-analysis of the randomized clinical trials. Arthroscopy. 2013;29:343–8.
59. Smith CD, Alexander S, Hill AM, et al. A biomechanical comparison of single and double-row fixation in arthroscopic rotator cuff repair. J Bone Joint Surg Am. 2006;88:2425–31.
60. Sugaya H, Maeda K, Matsuki K, Moriishi J. Functional and structural outcome after arthroscopic full-thickness rotator cuff repair: single-row versus dual-row fixation. Arthroscopy. 2005;21:1307–16.
61. Terry GC, Chopp TM. Functional anatomy of the shoulder. J Athl Train. 2000;35:248–55.
62. Trantalis JN, Boorman RS, Pletsch K, Lo IK. Medial rotator cuff failure after arthroscopic double-row rotator cuff repair. Arthroscopy. 2008;24:727–31.
63. Trumble T, Cornwall R, Budoff J. Core knowledge in orthopaedics: hand, elbow and shoulder. Philadelphia, PA: Mosby Elsevier; 2006.
64. Voigt C, Bosse C, Vosshenrich R, Schulz AP, Lill H. Arthroscopic supraspinatus tendon repair with suture-bridging technique: functional outcome and

magnetic resonance imaging. Am J Sports Med. 2010;38:983–91.
65. Wall LB, Keener JD, Brophy RH. Clinical outcomes of double-row versus single-row rotator cuff repairs. Arthroscopy. 2009;25:1312–8.
66. Wieser K, Rahm S, Farshad M, et al. Stitch positioning influences the suture hold in supraspinatus tendon repair. Knee Surg Sports Traumatol Arthrosc. 2013;21:1587–92.
67. Wülker N, Roetman B, Plitz W, Knop W. Function of the supraspinatus muscle in a dynamic shoulder model. Unfallchirurg. 1994;97:308–13.
68. Yamamoto N, Itoi E. A review of biomechanics of the shoulder and biomechanical concepts of rotator cuff repair. Asia Pac J Sports Med Arthrosc Rehabil Technol. 2015;2(1):27–30. https://doi.org/10.1016/j.asmart.2014.11.004.
69. Yang J Jr, Robbins M, Reilly J, Maerz T, Anderson K. The clinical effect of a rotator cuff retear: a meta-analysis of arthroscopic single-row and double-row repairs. Am J Sports Med. 2017;45(3):733–41.
70. Zanetti M, Gerber C, Hodler J. Quantitative assessment of the muscles of the rotator cuff with magnetic resonance imaging. Invest Radiol. 1998;33(3):163–70.

4 Re-rupture or Non-healing? Factors Determining an Unsuccessful Repair

Berte Bøe

4.1 Introduction

There is a need to understand the reasons why a high proportion of rotator cuff repairs fail to heal. Several studies have noted that increasing age is a significant factor for diminished rotator cuff healing, while biomechanical studies have suggested that the reasons for this may be an inferior healing environment in older patients.

Rotator cuff tears may cause significant pain and loss of function. Patient outcomes are reported in outcome scores, including strength, pain, and active range of motion. Healing of the tendon after surgical repair is thought to improve functional outcomes and is the reason why patients with persistent symptoms are commonly offered surgical treatment. Even though rotator cuff repair results in improved clinical outcome, several studies report failure of healing in up to 94% of patients [1]. An explanation of this discrepancy is that the success of a repair is commonly based on patient-related outcomes and not on the healing of the tendon. The outcome is often irrespective of healing; however, there is a tendency that outcome scores that include strength and active range of motion provide better results with tendon healing, whereas those scores that evaluate subjective patient outcomes fail to show a difference between patients with healed tendons and those with discontinuity of the rotator cuff tendon [2].

Healing indicates a continuous layer of tissue from the rotator cuff muscle belly to the insertion on the greater tuberosity. The healing process is divided into three overlapping stages: inflammation (1 week), repair (week 1–4), and remodeling (after 3 weeks) [3].

To assess healing structural and qualitative assessment of the rotator cuff can be done with either MRI or ultrasound. Most often repair integrity is determined by MRI, according to the classification described by Sugaya et al. [4]. This classification distinguishes five repair categories with the use of oblique coronal, oblique sagittal, and transverse T2-weighted images. Type I indicates a repaired rotator cuff that has sufficient thickness with homogeneously low intensity on each image; type II, sufficient thickness with a partial high-intensity area; type III, insufficient thickness without discontinuity; type IV, the presence of a minor discontinuity in more than one slice of each image, suggestive of a small tear; and type V, the presence of a major discontinuity on each image, suggestive of a large tear (Fig. 4.1).

The term re-rupture is used throughout the literature when rotator cuff healing is assessed, but most studies do not specifically document healing before recurrent tears. Reported healing rates from rotator cuff repairs in general range from 6 to 100% [1], in other words a huge variation.

B. Bøe (✉)
Division of Orthopaedic Surgery, Oslo University Hospital, Oslo, Norway

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_4

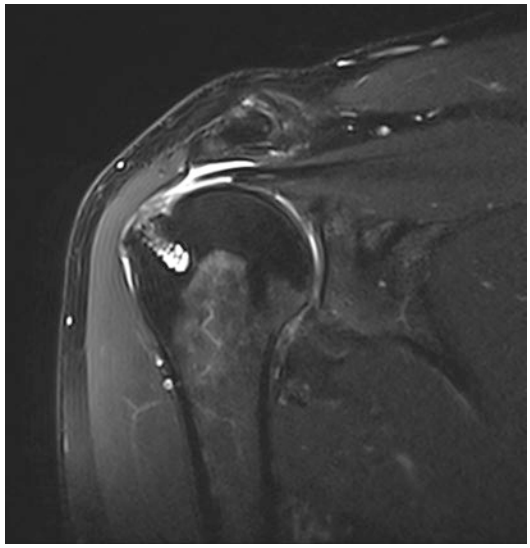


Fig. 4.1 Re-rupture after rotator cuff repair, Sugaya 5

Agout et al. published healing rates of 68–81% 10 years after rotator cuff repair, depending on the initial type of tear [5]. Tears with posterior extension have a higher retear rate, while the risk of arthritis is higher in tears with anterosuperior extension.

Isolated supraspinatus tears show the best results, with a lower failure rate than the other types.

The factors associated with healing are multifactorial in nature. Interpretation of scientific studies is confounded by variations in the definition of healing, the time point at which healing is assessed and the imaging modality and method used to assess healing.

A randomized trial of open vs. arthroscopic repair (UKUFF trial) revealed that 40% of repairs fail within 12 months irrespective of the surgical technique used and that a failed repair adversely affected patient outcomes [6].

The factors that are most consistently reported to influence healing are patient age and tear size. Other commonly cited factors include fatty infiltration, muscle atrophy [7], muscle tendon retraction, workers compensation, compliance with rehabilitation, smoking, manual workers, injections, type of repair, use of orthobiologics [8], and hypercholesterolemia.

4.2 Patients' Age

Patients with well-healed repair tend to be younger [9]. Age is found to be an independent predictor for a structurally intact rotator cuff repair. Gumina et al. [10] reported that the mean age of patients with a healed repair was 3.0 years younger than those who had a recurrent tear.

Diebold et al. [11] studied a cohort of 1600 patients normally distributed in terms of age, with a mean age of 59 years and a range from 15 to 91 years. The 212 patients (13%) who had a retear at 6 months were also normally distributed in terms of age, with a mean age of 65 years and a range from 15 to 88 years. They found that the re-rupture rate in patients below 50 years of age was 5%. This increased to 10% in patients aged 50–59 years, 15% in those aged 60–69 years, 25% in those aged 70–79 years, and 34% in those aged above 80 years. Multiple logistic regression analysis showed that the patient age was an independent factor strongly associated with re-ruptures.

Older age should not be viewed as a contraindication to surgical repair. A study from the French Arthroscopy Society in 2013 showed better outcomes of rotator cuff repair compared to bursectomy and acromioplasty in patients older than 70 years [12].

4.3 Tear Size

The healing rate depends chiefly on the initial size of the tear [6, 13]. Tear size measurements are often made in the anterior posterior direction according to the footprint defect. Medial-lateral dimensions are retraction measurements from the leading edge of the tear to the lateral edge of the footprint [14]. Small and medium tears are more likely to heal than large and massive tears. Park et al. found the failure rate to be significantly higher in patients with a tear of more then 2 cm in size anterior to posterior (34.2%) compared with patients with a tear of less then 2 cm (10.6%) [14]. Rodeo et al. concluded that at 12 weeks post-repair, 71% of small tears healed

compared with 82% of medium tears, and 56% of large tears [15].

The influence of infraspinatus delamination and of a subscapularis tear remains debatable [16, 17]. An infraspinatus tear longer than 1 cm has also been identified as a risk factor for failure to heal [18].

Agout et al. reported 10-year results of 511 patients in 2014. The healing rate was from 68 to 81% depending on the initial type of tear. Tears with posterior extension had a higher retear rate, while the risk of arthritis was higher in tears with anterosuperior extension. Isolated supraspinatus tears showed the best results, with a lower failure rate than the other types. However, surgical repair gave good functional results in the long term whatever the type of tear was, in spite of a 10% complication rate and a 12% revision rate.

Shimokobe et al. [19] found that the re-rupture rate was significantly higher in a group with posterosuperior tears compared to two groups with anterosuperior tears or anterioposterior-extending tears (P = 0.02). In this study of 102 patients multivariate analysis showed that decreased preoperative active external rotation range was a unique risk factor for postoperative re-rupture in both the posterosuperior and the anteriorposterior-extended groups.

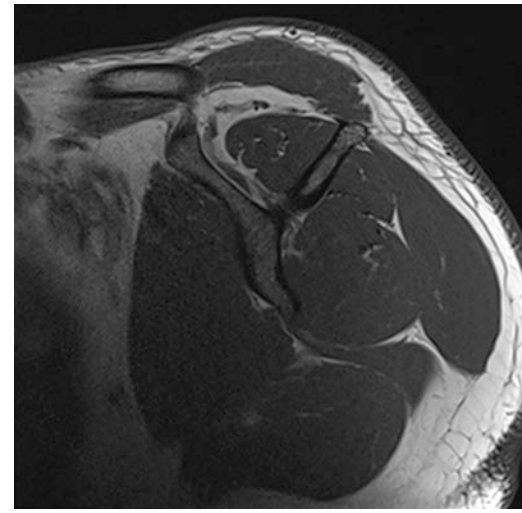


Fig. 4.2 Fatty infiltration is evaluated in the sagittal plane at the level where the scapular spine and body form a Y shape

4.4 Fatty Infiltration

To assess the degree of fatty infiltration the T1-weighted oblique sagittal sequences are frequently used and graded based on Goutallier's classification scheme [20]. Previous studies have found that this staging system can be appropriately applied to MRI [21]. The scan is evaluated in the sagittal plane at the level where the scapular spine and body form a Y shape (Fig 4.2).

In 2016 Gasbarro et al. analyzed risk factors to predict structural failure after arthroscopic cuff repair [18]. The material consisted of 30 failures compared to 60 controls. The presence of supraspinatus fatty infiltration grade 2 or above was significantly more common in the failure group. Fatty infiltration in any rotator cuff muscle was present upon MRI in 30% of the failures vs. 25% of the controls. There was no difference in the average number of muscles being affected. Conditional logistic regression confirmed supraspinatus fatty infiltration and supraspinatus tear size as risk factors for failure.

4.4.1 Muscle Atrophy and Muscle Tendon Retraction

In the muscle, there is significant migration of inflammatory cells within the first few days of a tear and the muscle fibers undergo apoptosis [22]. In the ensuing weeks to months, this early response leads to muscular retraction, degeneration, and atrophy. The progressive loss of muscle volume is due to the loss of sarcomeres, which causes an enlargement of the inter- and intramyofibrillar spaces. If the muscle remains unloaded and retracted, the myogenic precursor cells may be reprogrammed into the adipogenic pathway, with mature adipocytes infiltrating the free inter- and intramyofibrillar spaces. This phenomenon is termed fatty infiltration [23].

4.5 Compliance with Rehabilitation

A number of rehabilitation protocols have been described for use following rotator cuff repair. While some surgeons recommend early, aggressive rehabilitation programs, others recommend a more conservative rehabilitation program. Early aggressive rehabilitation protocols may result in a slightly higher incidence of retear compared with more conservative protocols.

Slower, less aggressive rehabilitation programs have demonstrated improved healing with no negative effect on the final range of motion and are therefore recommended after repair of most full-thickness tears [24].

4.6 Smoking

Galatz et al. [25] demonstrated that nicotine impairs biomechanical as well as histological properties after rotator cuff tendon repair in a rat model. In a clinical study, a dose- and time-dependent relationship between smoking and the presence of rotator cuff tears was noted [26]. This data suggests that abstinence or at least a decrease in nicotine use might help to improve healing after rotator cuff repair and smoking cessation should be strongly encouraged.

4.7 Manual Workers

Return to work may prove impossible, despite workplace adjustments. Collin et al. found that one-fifth of patients had not returned to their previous occupation 6 months after rotator cuff repair. The factors associated with failure to resume work were female gender, heavy manual work, and persistent bursitis detected by follow-up sonography [27]. They were unable to show a difference in Constant scores between a group returning to work and the other group not returning to normal activities at 6 months after the univariate analysis. Lack of healing was not correlated with the group not returning to normal activities. This finding corroborates with previous studies [28] and confirms that lack of healing does not necessarily result in clinical failure, except in labour intensive workers who represent a high-risk group for poor outcome among patients with failure [29].

Despite studies that suggest that tendon healing can affect the final outcome [30], the lack of tendon healing is not associated with the inability to return to work or activity, at least at 6 months postoperatively.

4.8 Injections/Medication

Nonsteroidal anti-inflammatory drug therapy is very effective in relieving the postoperative pain but may adversely affect tendon healing in rats [31].

There is some basic research evidence that the application of NSAIDs postoperatively may alter rotator cuff healing [32]. The common practice of administering NSAIDs should therefore be reconsidered during the first 6 postoperative weeks. After this period of time, NSAIDs do not seem to have an influence on healing and there is evidence that they positively influence the remodeling of the collagen matrix during that time [33].

4.9 Type of Repair

The surgical technique might play a role. During the past decade, several innovative arthroscopic techniques have been introduced for rotator cuff reconstructions. Strong fixation can be achieved, but no suturing or anchoring method has been proven superior over the others. The surgeon must adapt the surgical technique to the local conditions, notably the shape and direction of the tear and the flexibility of its borders. The double-row repair has been shown to increase the biomechanical properties relative to simple suture single-row as well as trans osseous rotator cuff repairs.

In a metaanalysis by Brown et al. there were no significant differences in re-rupture rates for modified Mason Allen sutures compared to simple sutures in single row repairs of tears

measuring less than 3 cm [34]. For single row repairs of tears measuring more than 3 cm there was no significant difference in mattress sutures compared to simple sutures. The rates of re-rupture did not differ between the single row and double row for tears measuring less than 3 cm or 3 cm and more. These findings suggest that suture technique may not affect re-rupture rates after rotator cuff repair.

Hein et al. did a systematic review where their imaging diagnosed re-rupture rate was stratified by preoperative tear size at a minimum of 1 year follow-up, and re-ruptures were diagnosed by either MRI, ultrasound, or arthrogram. Both double row and suture bridge had significantly lower re-rupture rates than single row repair for all sizes. There was no significant difference between double row and suture bridge. To date, no level I study has shown whether the suture bridge technique yields superior healing rates compared with conventional double row.

This study only investigated the incidence of postoperative re-ruptures as diagnosed by clinical imaging, and as such, no conclusions regarding clinical outcomes as a function of repair technique could be made.

Asymptomatic retears in the intermediate term may progress to larger, symptomatic tears requiring revision, and massive retears are inherently difficult to revise. Therefore future studies are necessary to track the long-term re-rupture rates of rotator cuff repair techniques to determine how re-ruptures affect long-term clinical outcomes.

Despite the apparent benefits of newer constructs, double-row rotator cuff repairs lead to increased surgical time and higher implant costs compared to single-row repairs, and therefore, there is still a debate about the ideal repair construct.

4.10 Orthobiologics

In the past two decades, orthopaedic research has focused on biologically augmenting the rotator cuff reconstruction, and therefore improving healing at the tendon-bone interface as well as trying to stop muscular degeneration or even accomplish regeneration of the rotator cuff muscle. This biological augmentation has included applying different platelet concentrates containing growth factors, mesenchymal stem cells, scaffolds, and a combination of the above. The biology of rotator cuff tears and repairs has gained more interest as growth factors might positively influence tendon to bone ingrowth and, therefore, might positively influence the re-rupture rate.

The additional augmentation with platelet-rich plasma did not reveal any significant differences in the healing rate compared to conventional rotator cuff repair. No definitive evidence supports the use of platelet rich plasma or mesenchymal stem cells (MSC) regarding improvement of healing rates and clinical outcomes [24].

In a rabbit model hyalorunic acid (HA) accelerated tendon-to-bone healing in the rotator cuff repair model, enhancing the biomechanical strength and increasing chondroid formation and tendon maturity at the tendon-bone interface. Based on the data of in vitro experiments, HA activated MSCs may play a crucial role in the acceleration of tendon-to-bone healing [35].

Bone marrow vents are a cheap possibility to stimulate the interface between tendon and bone. There are some articles publishing good clinical and radiological results after rotator cuff repair with bone marrow vents; however, as long as there are no control groups, it is impossible to conclude that this really enhances the healing [36].

4.11 Hypercholesterolemia and Diabetes Mellitus

There seems to be a relationship between an individual's lipid levels and tendon pathologies [37]. Therefore the question arises if high serum cholesterol levels should be treated before rotator cuff surgery.

Diabetes may have an impact on rotator cuff healing. Bedi et al. [38] reported that diabetes mellitus decreased the biomechanical properties in a rat model. Chen et al. [39] reported a higher rate of postoperative complications, namely infections and to a lesser extent also failures.

4.12 Discussion

The healing rate of rotator cuff repair went down from 94% in 2007 to 71% in 2015. The reason for this is likely to be multifactorial. One possibility is publication bias and an increased tendency to report not just clinical outcomes but also imaging of repair integrity after surgery. Some series reporting these outcomes of failed biologic healing are based on ultrasound evaluation and others are based on MRI.

There has been a dramatic increase in the incidence of rotator cuff repair surgery, reflecting a probable change in the selection criteria for surgery.

Park et al. [14] used a univariate analysis in their case-control study to conclude that both age and tear size were prognostic factors for healing. They also noted that the mean age of patients with healed repairs was 59 vs. 63 years in those with a failed repair and that tears greater than 2 cm in size healed less often than those less than 2 cm (66% vs. 89%). They did not adjust for age and tear size together.

Repair failure is usually defined as a need for further surgery in the short- or medium-term. In the retrospective SoFCOT study [5] of 511 patients who underwent repair surgery for an isolated supraspinatus tear in 2003, 35 (7%) patients required revision surgery within 10 years (repeat repair, $n = 17$; arthroplasty, $n = 7$; and other procedures, $n = 11$).

After a retear discovered at follow up there will be a new assessment whether to perform a revision surgery or not. In general the results of revision surgery are inferior to primary cases. Most of the cuff re-ruptures do not require surgery, given their good clinical tolerance and stable outcomes over time. Information must be obtained about the circumstances of the first repair procedure; a possible diagnostic inadequacy or technical error might be present. Trauma after repair might be due to an aggressive rehabilitation program. Revision cuff repair, when indicated by pain or functional impairment, can improve pain and function at midterm follow-up [40]; however, the clinical outcome scores were comparable in patients with an intact repair and those with failed rotator cuff healing after revision repair. Therefore, tendon integrity is not necessarily correlated with better clinical outcomes after revision rotator cuff repair at final follow-up.

4.13 Summary

Clinical decision-making should take into account the overriding importance of increasing age as a risk factor when considering the suitability of rotator cuff surgery for patients.

Non-operative treatment (rehabilitation and local injections) should be considered in patients with manageable pain and limited physical activities.

A rotator cuff re-rupture is a multifactorial process with no single preoperative or intraoperative factor being overwhelmingly predictive of it. Nevertheless, rotator cuff tear size shows stronger association with re-ruptures at 6 months after surgery than measures of tissue quality and concomitant shoulder injuries.

Knowledge about risk factors of re-ruptures or non-healing may help surgeons and patients to predict the success of arthroscopic rotator cuff repair, and this information is likely to assist preoperative counseling, preoperative planning, and selection of patients for surgery.

References

1. Galatz LM, Ball CM, Teefey SA, Middleton WD, Yamaguchi K. The outcome and repair integrity of completely arthroscopically repaired large and massive rotator cuff tears. J Bone Joint Surg Am. 2004;86-A(2):219–24.
2. Charousset C, Bellaiche L, Kalra K, Petrover D. Arthroscopic repair of full-thickness rotator cuff tears: is there tendon healing in patients aged 65 years or older? Arthroscopy. 2010;26(3):302–9.
3. Angeline ME, Rodeo SA. Biologics in the management of rotator cuff surgery. Clin Sports Med. 2012;31(4):645–63.
4. Sugaya H, Maeda K, Matsuki K, Moriishi J. Functional and structural outcome after arthroscopic full-thickness rotator cuff repair: single-row versus dual-row fixation. Arthroscopy. 2005;21(11):1307–16.
5. Agout C, Berhouet J, Bouju Y, Godeneche A, Collin P, Kempf JF, et al. Clinical and anatomic results of rota-

tor cuff repair at 10 years depend on tear type. Knee Surg Sports Traumatol Arthrosc. 2018;26(8):2490–7.
6. Carr AJ, Cooper CD, Campbell MK, Rees JL, Moser J, Beard DJ, et al. Clinical effectiveness and cost-effectiveness of open and arthroscopic rotator cuff repair [the UK Rotator Cuff Surgery (UKUFF) randomised trial]. Health Technol Assess. 2015;19(80):1–218. Pubmed Central PMCID: 4781041.
7. Dwyer T, Razmjou H, Henry P, Gosselin-Fournier S, Holtby R. Association between pre-operative magnetic resonance imaging and reparability of large and massive rotator cuff tears. Knee Surg Sports Traumatol Arthrosc. 2015;23(2):415–22.
8. Jo CH, Shin JS, Shin WH, Lee SY, Yoon KS, Shin S. Platelet-rich plasma for arthroscopic repair of medium to large rotator cuff tears: a randomized controlled trial. Am J Sports Med. 2015;43(9):2102–10.
9. Rashid MS, Cooper C, Cook J, Cooper D, Dakin SG, Snelling S, et al. Increasing age and tear size reduce rotator cuff repair healing rate at 1 year. Acta Orthop. 2017;88(6):606–11. Pubmed Central PMCID: 5694804.
10. Gumina S, Campagna V, Ferrazza G, Giannicola G, Fratalocchi F, Milani A, et al. Use of platelet-leukocyte membrane in arthroscopic repair of large rotator cuff tears: a prospective randomized study. J Bone Jt Surg Am. 2012;94(15):1345–52.
11. Diebold G, Lam P, Walton J, Murrell GAC. Relationship between age and rotator cuff retear: a study of 1,600 consecutive rotator cuff repairs. J Bone Jt Surg Am. 2017;99(14):1198–205.
12. Flurin PH, Hardy P, Abadie P, Desmoineaux P, Essig J, Joudet T, et al. Rotator cuff tears after 70 years of age: a prospective, randomized, comparative study between decompression and arthroscopic repair in 154 patients. Orthop Traumatol Surg Res. 2013;99(8 Suppl):S371–8.
13. Zumstein MA, Rumian A, Thelu CE, Lesbats V, O'Shea K, Schaer M, et al. SECEC Research Grant 2008 II: use of platelet- and leucocyte-rich fibrin (L-PRF) does not affect late rotator cuff tendon healing: a prospective randomized controlled study. J Shoulder Elbow Surg. 2016;25(1):2–11.
14. Park JS, Park HJ, Kim SH, Oh JH. Prognostic factors affecting rotator cuff healing after arthroscopic repair in small to medium-sized tears. Am J Sports Med. 2015;43(10):2386–92.
15. Rodeo SA, Delos D, Williams RJ, Adler RS, Pearle A, Warren RF. The effect of platelet-rich fibrin matrix on rotator cuff tendon healing: a prospective, randomized clinical study. Am J Sports Med. 2012;40(6):1234–41.
16. Zilber S, Carillon Y, Lapner PC, Walch G, Nove-Josserand L. Infraspinatus delamination does not affect supraspinatus tear repair. Clin Orthop Relat Res. 2007;458:63–9.
17. Boileau P, Brassart N, Watkinson DJ, Carles M, Hatzidakis AM, Krishnan SG. Arthroscopic repair of full-thickness tears of the supraspinatus: does the tendon really heal? J Bone Jt Surg Am. 2005;87(6):1229–40.
18. Gasbarro G, Ye J, Newsome H, Jiang K, Wright V, Vyas D, et al. Morphologic risk factors in predicting symptomatic structural failure of arthroscopic rotator cuff repairs: tear size, location, and atrophy matter. Arthroscopy. 2016;32(10):1947–52.
19. Shimokobe H, Gotoh M, Honda H, Nakamura H, Mitsui Y, Kakuma T, et al. Risk factors for retear of large/massive rotator cuff tears after arthroscopic surgery: an analysis of tearing patterns. J Orthopaed Surg Res. 2017;12(1):140. Pubmed Central PMCID: 5613358.
20. Goutallier D, Postel JM, Bernageau J, Lavau L, Voisin MC. Fatty muscle degeneration in cuff ruptures.Pre- and postoperative evaluation by CT scan. Clin Orthop Relat Res. 1994;304:78–83.
21. Fuchs B, Weishaupt D, Zanetti M, Hodler J, Gerber C. Fatty degeneration of the muscles of the rotator cuff: assessment by computed tomography versus magnetic resonance imaging. J Shoulder Elbow Surg. 1999;8(6):599–605.
22. Lundgreen K, Lian OB, Engebretsen L, Scott A. Tenocyte apoptosis in the torn rotator cuff: a primary or secondary pathological event? Br J Sports Med. 2011;45(13):1035–9. Pubmed Central PMCID: 3951987.
23. Frey E, Regenfelder F, Sussmann P, Zumstein M, Gerber C, Born W, et al. Adipogenic and myogenic gene expression in rotator cuff muscle of the sheep after tendon tear. J Orthop Res. 2009;27(4):504–9.
24. Abtahi AM, Granger EK, Tashjian RZ. Factors affecting healing after arthroscopic rotator cuff repair. World J Orthop. 2015;6(2):211–20. Pubmed Central PMCID: 4363803.
25. Galatz LM, Silva MJ, Rothermich SY, Zaegel MA, Havlioglu N, Thomopoulos S. Nicotine delays tendon-to-bone healing in a rat shoulder model. J Bone Jt Surg Am. 2006;88(9):2027–34.
26. Baumgarten KM, Gerlach D, Galatz LM, Teefey SA, Middleton WD, Ditsios K, et al. Cigarette smoking increases the risk for rotator cuff tears. Clin Orthop Relat Res. 2010;468(6):1534–41. Pubmed Central PMCID: 2865623.
27. Collin P, Abdullah A, Kherad O, Gain S, Denard PJ, Ladermann A. Prospective evaluation of clinical and radiologic factors predicting return to activity within 6 months after arthroscopic rotator cuff repair. J Shoulder Elbow Surg. 2015;24(3):439–45.
28. Russell RD, Knight JR, Mulligan E, Khazzam MS. Structural integrity after rotator cuff repair does not correlate with patient function and pain: a meta-analysis. J Bone Jt Surg Am. 2014;96(4):265–71.
29. Namdari S, Donegan RP, Chamberlain AM, Galatz LM, Yamaguchi K, Keener JD. Factors affecting outcome after structural failure of repaired rotator cuff tears. J Bone Jt Surg Am. 2014;96(2):99–105.
30. Denard PJ, Ladermann A, Burkhart SS. Prevention and management of stiffness after arthroscopic rotator cuff repair: systematic review and implications for rotator cuff healing. Arthroscopy. 2011;27(6):842–8.

31. Connizzo BK, Yannascoli SM, Tucker JJ, Caro AC, Riggin CN, Mauck RL, et al. The detrimental effects of systemic Ibuprofen delivery on tendon healing are time-dependent. Clin Orthop Relat Res. 2014;472(8):2433–9. Pubmed Central PMCID: 4079885.
32. Cohen DB, Kawamura S, Ehteshami JR, Rodeo SA. Indomethacin and celecoxib impair rotator cuff tendon-to-bone healing. Am J Sports Med. 2006;34(3):362–9.
33. Chan KM, Fu SC. Anti-inflammatory management for tendon injuries—friends or foes? Sports Med Arthrosc Rehab Ther Technol. 2009;1(1):23. Pubmed Central PMCID: 2770552.
34. Brown MJ, Pula DA, Kluczynski MA, Mashtare T, Bisson LJ. Does suture technique affect re-rupture in arthroscopic rotator cuff repair? A meta-analysis. Arthroscopy. 2015;31(8):1576–82.
35. Honda H, Gotoh M, Kanazawa T, Ohzono H, Nakamura H, Ohta K, et al. Hyaluronic acid accelerates tendon-to-bone healing after rotator cuff repair. Am J Sports Med. 2017;45(14):3322–30.
36. Dierckman BD, Ni JJ, Karzel RP, Getelman MH. Excellent healing rates and patient satisfaction after arthroscopic repair of medium to large rotator cuff tears with a single-row technique augmented with bone marrow vents. Knee Surg Sports Traumatol Arthrosc. 2018;26(1):136–45.
37. Tilley BJ, Cook JL, Docking SI, Gaida JE. Is higher serum cholesterol associated with altered tendon structure or tendon pain? A systematic review. Br J Sports Med. 2015;49(23):1504–9. Pubmed Central PMCID: 4680137.
38. Bedi A, Fox AJ, Harris PE, Deng XH, Ying L, Warren RF, et al. Diabetes mellitus impairs tendon-bone healing after rotator cuff repair. J Shoulder Elbow Surg. 2010;19(7):978–88. Pubmed Central PMCID: 5257255.
39. Chen AL, Shapiro JA, Ahn AK, Zuckerman JD, Cuomo F. Rotator cuff repair in patients with type I diabetes mellitus. J Shoulder Elbow Surg. 2003;12(5):416–21.
40. Willinger L, Lacheta L, Beitzel K, Buchmann S, Woertler K, Imhoff AB, et al. Clinical outcomes, tendon integrity, and shoulder strength after revision rotator cuff reconstruction: a minimum 2 years' follow-up. Am J Sports Med. 2018;46(11):2700–6.

5 Biological Augmentation in Rotator Cuff Repair: Growth Factors

Claudio Rosso and Patrick Vavken

Currently, roughly 250,000 rotator cuff repairs per year are performed in the United States alone [1]. Retear rates especially in massive rotator cuff tears are still an issue. We are still facing up to 94% retear rate in large to massive rotator cuff tears even with double-row repairs [2, 3] while the clinical importance of retears remains unclear [4–6].

Retears of tendons have been associated with the same patho-mechanisms that led to the initial tear: mechanical stress on a degenerated tendon. This might lead to retearing just medial to the repair site [7, 8].

In a prospective trial, tendon pulling through the sutures is the most common type of failure (so-called cheese wiring) followed by new tears through the already degenerated tissue and anchor failure, respectively [8]. It was also found that retears are a multifactorial process associated with tear size [9].

One possible solution to this problem might be to augment rotator cuff repairs with matrices, patches, or growth factors. Reinforcing the rotator cuff with synthetic or xenografts adds a high complexity to arthroscopic rotator cuff repair while mixed results have been reported [10, 11].

Due to promising animal studies, the use of growth factors such as platelet-rich plasma (PRP) has been implemented as an alternative. It was hypothesized that PRP would improve rotator cuff healing by propelling regeneration of the degenerated tendons by means of stimulating the differentiation of scar tissue [7, 12]. Platelet-rich plasma (PRP) is the most commonly used term for an autologous, concentrated platelet suspension. Autologous conditioned plasma (ACP) is another platelet suspension form with a low level of white blood cells. Autologous conditioned serum (ACS) is a platelet suspension which has a high content of IL-1Ra (Interleukin-1 receptor antagonist) and is therefore used for anti-rheumatic and anti-inflammatory purposes. Direct comparisons of individual products have shown a relatively large variability of the contents, and for use in clinical practice a great overlap of PRP/ACP/ACS in nature and effect can be assumed [13].

In clinical practice, PRP was initially used primarily in plastic, cardiovascular, and maxillofacial surgery [14, 15]. Early studies in these fields have shown beneficial effects on wound healing, tissue regeneration, and fracture healing/bone remodeling. Responsible for this were bioactive proteins and growth factors [16, 17]. However, the extracellular matrix of the coagulated PRP has also been discussed as a potential

C. Rosso (✉)
ARTHRO Medics, Shoulder and Elbow Center, Basel, Switzerland

University of Basel, Basel, Switzerland
e-mail: c.rosso@arthro.ch

P. Vavken
Alphaclinic Zurich, Zurich, Switzerland

Boston Children's Hospital, Harvard Medical School, Boston, USA
e-mail: vavken@alphaclinic.ch

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_5

signaling mechanism for regenerative processes because of its mechanical and structural properties [18]. Furthermore, it was confirmed that the autologous cells do not induce any unwanted immunological reactions or even diseases that may be associated with blood transfusions, for example. In short, PRP therapy can fill or cover a defect, locally release growth factors that attract the cells needed for wound healing, and stimulate these cells to increased activity. In the context of these processes, a PRP therapy can stimulate and sustain wound healing.

PRP preparations at various cell concentrations are used both experimentally and clinically. Unequivocal evidence for optimal platelet concentration does not exist so far, and the relationship between concentration and effect is unclear. Some arguments suggest that high concentration produces a high impact, and most commercial kits produce PRP in the range of 9–14× physiological platelet concentration. Others, in turn, could not show any clinical differences with higher concentrations, or even report detrimental effects, e.g., the inhibition of osteoblasts [19]. For example, animal experiments have shown equally good biomechanical results for ligament and tendon regeneration with 3–5× concentrated PRP compared to higher concentrations [20]. This could also be shown specifically for human rotator cuff cells [21]. Finally, it could even be shown that a 1.2–2× concentration of PRP, i.e., nearly normal blood, can achieve a good effect in soft tissue healing [20]. Too high a concentration of growth factors may result in an unorganized reaction of the cells involved in healing, resulting in a poorly differentiated, i.e., mechanically weak, scar [22]. PRP is not a pure platelet suspension, and with ACP or ACS even a reduced addition of white blood cells is advertised. Both red blood cells and leukocytes have been shown to modulate the effect of platelets on mesenchymal cells [23]. Due to the fact that PRP is supposed to support but not overstimulate wound healing, this interaction with erythrocytes and leukocytes is desirable, although the evidence here is less. Currently, there is no gold standard regarding the ion or electrolyte concentration, nor is it determined how high the protein fraction should be.

Lastly, the application form of PRP is a matter of debate. It can be injected as a suspension, or used as a spray or as a gel (on a carrier). There is very little comparative data on these applications or on the effect depending on the application form. However, influence on the enzyme kinetics by the application form is probably nonexistent. What needs to be considered, especially in the context of an arthroscopic application, is feasibility. A solid clot can be manipulated and threaded on a suture. A fluid application is at risk of dilution or to be flushed out of the defect.

Promising results are obtained from animal studies. Recently, it was shown that a freeze-dried chitosan implant solubilized in PRP could enhance tendon-to-bone healing and thus improve rotator cuff healing in a rabbit model [24].

In our systematic review, we could show that the use of PRP did not improve tendon healing and reduce retears in large tears but was beneficial in small- and medium-sized tears [25].

Alternatively, microfracturing of the humeral head in order to influx connective tissue progenitor cells into the healing site during rotator cuff repair has been advocated [26]. A recent meta-analysis revealed a positive effect to reduce retear rates by promoting tendon-to-bone healing [27]. However, no significant improvement in clinical outcomes was shown.

Cost-Effectiveness

In recent years, terms like cost-effectiveness, value-based health care, and sustainability in health care financing have become ubiquitous and physicians are increasingly confronted with demands for cost-effectiveness and cost-containment by legislators and insurance companies. Hence it makes sense to approach biological augmentation not only from a clinical-impact perspective, but also from one of economic feasibility. A simple economic analysis has two scopes. In the narrow scope, the question whether the incremental cost of adding growth factors to a cuff repair is offset by a commensurate gain in clinical outcome needs to be answered. In the wider scope, the question whether the incremental cost of adding growth factors to a cuff repair that is offset by a commensurate gain in clinical

outcome is preferable to a surrogate treatment form (e.g., an RSA).

For the smaller scope, some data exist to build an analytical model. This model includes the various possible developments of a patient undergoing cuff repair (i.e., healing, retear, and revision) into a decision tree. Each branch represents a specific outcome with the likelihood of achieving this outcome. A value, called *utility*, for each outcome is developed from patient information. The utility describes the value of an event or outcome to a patient and its unit of measurement is the quality-adjusted life year (QaLY). The utility of healing obviously is higher than the utility of a retear. The utility of a successful revision is lower than the utility of a successful primary repair due to a principle called *time preference*. Time preference describes the simple fact that achieving a preferred outcome sooner has a higher utility than arriving at the same endpoint later or via a circuitous route. For economic analysis, the gain of utilities is compared to the additional cost, usually within a range of outcomes (i.e., with a risk of revision ranging from +10% to −10% of what is seen in the literature) to account for clinical variability. The findings are compared to benchmarked thresholds, with a rule of thumb that an extra cost of US$ 100,000.00 for an additional QaLY is considered cost-effective. One study exists using such a standardized framework to assess the cost-effectiveness of biological augmentation of cuff repair with PRP. Its findings show that the overall cost (including consumables, OR time, and fixed cost) should be below (2015) US$ 650. Given the current cost of most commercially available kits, and the mostly negative growth of reimbursement rates of shoulder surgery it is questionable if this is a sustainable business case outside well-structured ASCs and comparable institutions. However, microfracturing as described above has very little additional cost in time and consumables.

In the larger scope, confronted with a massive or irreparable cuff tear, substitutes to arthroscopic repair exist and are well delineated and described in following chapters. A considerably larger decision model could include cuff repair, shoulder replacement, debridement, spacers, etc. like the model described above. There is some data on the comparison of cuff repair with primary replacement in patients with massive tears favoring cuff repair in the short- and mid-term. However, this hinges to a greater extent on the lower initial cost of arthroscopic repair, a low revision rate, and no arthritic degeneration, rather than on clinical outcomes. This preference changes drastically if a cuff repair is to be revised. Hence, the additional cost of PRP in cuff repair, if reducing revision-worthy retears, may be well within cost-effectiveness thresholds. We could show that although retear rates can be reduced in small- and mid-size tears using PRP this procedure is not cost-effective [25].

In conclusion, the use of growth factors, especially PRP, does not reduce retear rates and is currently not cost effective due to the additional OR time and costs of the harvesting systems. However, results from animal studies using structural grafts loaded with PRP are promising. Upon reviewing the current literature, the authors have the impression that we are at a turnaround to enhance tendon healing with growth factors using scaffolds [28, 29]. Currently, the easiest and most cost-effective procedure is microfracturing of the tuberosities to get stem cells into the healing site [27].

References

1. Edwards SL, Lynch TS, Saltzman MD, Terry MA, Nuber GW. Biologic and pharmacologic augmentation of rotator cuff repairs. J Am Acad Orthop Surg. 2011;19(10):583–9.
2. Galatz LM, Ball CM, Teefey SA, Middleton WD, Yamaguchi K. The outcome and repair integrity of completely arthroscopically repaired large and massive rotator cuff tears. J Bone Joint Surg Am. 2004;86-A(2):219–24.
3. Gerber C, Fuchs B, Hodler J. The results of repair of massive tears of the rotator cuff. J Bone Joint Surg Am. 2000;82(4):505–15.
4. Genuario JW, Donegan RP, Hamman D, Bell JE, Boublik M, Schlegel T, et al. The cost-effectiveness of single-row compared with double-row arthroscopic rotator cuff repair. J Bone Joint Surg Am. 2012;94(15):1369–77.
5. Klepps S, Bishop J, Lin J, Cahlon O, Strauss A, Hayes P, et al. Prospective evaluation of the effect of rotator cuff integrity on the outcome of open rotator cuff repairs. Am J Sports Med. 2004;32(7):1716–22.

6. Russell RD, Knight JR, Mulligan E, Khazzam MS. Structural integrity after rotator cuff repair does not correlate with patient function and pain: a meta-analysis. J Bone Joint Surg Am. 2014;96(4):265–71.
7. Cheung EV, Silverio L, Sperling JW. Strategies in biologic augmentation of rotator cuff repair: a review. Clin Orthop Relat Res. 2010;468(6):1476–84.
8. Cummins CA, Murrell GA. Mode of failure for rotator cuff repair with suture anchors identified at revision surgery. J Shoulder Elbow Surg. 2003;12(2):128–33.
9. Iannotti JP, Deutsch A, Green A, Rudicel S, Christensen J, Marraffino S, et al. Time to failure after rotator cuff repair: a prospective imaging study. J Bone Joint Surg Am. 2013;95(11):965–71.
10. Iannotti JP, Codsi MJ, Kwon YW, Derwin K, Ciccone J, Brems JJ. Porcine small intestine submucosa augmentation of surgical repair of chronic two-tendon rotator cuff tears. A randomized, controlled trial. J Bone Joint Surg Am. 2006;88(6):1238–44.
11. Barber FA, Burns JP, Deutsch A, Labbe MR, Litchfield RB. A prospective, randomized evaluation of acellular human dermal matrix augmentation for arthroscopic rotator cuff repair. Arthroscopy. 2012;28(1):8–15.
12. Vavken P, Sadoghi P, Murray MM. The effect of platelet concentrates on graft maturation and graft-bone interface healing in anterior cruciate ligament reconstruction in human patients: a systematic review of controlled trials. Arthroscopy. 2011;27(11):1573–83.
13. Qiao J, An N, Ouyang X. Quantification of growth factors in different platelet concentrates. Platelets. 2017;28(8):774–8.
14. Taylor DW, Petrera M, Hendry M, Theodoropoulos JS. A systematic review of the use of platelet-rich plasma in sports medicine as a new treatment for tendon and ligament injuries. Clin J Sport Med. 2011;21(4):344–52.
15. El-Sharkawy H, Kantarci A, Deady J, Hasturk H, Liu H, Alshahat M, et al. Platelet-rich plasma: growth factors and pro- and anti-inflammatory properties. J Periodontol. 2007;78(4):661–9.
16. Fufa D, Shealy B, Jacobson M, Kevy S, Murray MM. Activation of platelet-rich plasma using soluble type I collagen. J Oral Maxillofac Surg. 2008;66(4):684–90.
17. Cenni E, Perut F, Ciapetti G, Savarino L, Dallari D, Cenacchi A, et al. In vitro evaluation of freeze-dried bone allografts combined with platelet rich plasma and human bone marrow stromal cells for tissue engineering. J Mater Sci Mater Med. 2009;20(1):45–50.
18. Vavken P, Gruber M, Dorotka R. Tissue engineering in orthopaedic surgery—clinical effectiveness and cost effectiveness of autologous chondrocyte transplantation. Z Orthop Unfall. 2008;146(1):26–30.
19. Huh JY, Choi BH, Zhu SJ, Jung JH, Kim BY, Lee SH. The effect of platelet-enriched fibrin glue on bone regeneration in autogenous bone grafts. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2006;101(4):426–31.
20. Mastrangelo AN, Vavken P, Fleming BC, Harrison SL, Murray MM. Reduced platelet concentration does not harm PRP effectiveness for ACL repair in a porcine in vivo model. J Orthop Res. 2011;29(7):1002–7.
21. Sadoghi P, Lohberger B, Aigner B, Kaltenegger H, Friesenbichler J, Wolf M, et al. Effect of platelet-rich plasma on the biologic activity of the human rotator-cuff fibroblasts: a controlled in vitro study. J Orthop Res. 2013;31(8):1249–53.
22. Rodeo SA, Potter HG, Kawamura S, Turner AS, Kim HJ, Atkinson BL. Biologic augmentation of rotator cuff tendon-healing with use of a mixture of osteoinductive growth factors. J Bone Joint Surg Am. 2007;89(11):2485–97.
23. Harrison SL, Vavken P, Murray MM. Erythrocytes inhibit ligament fibroblast proliferation in a collagen scaffold. J Orthop Res. 2011;29(9):1361–6.
24. Depres-Tremblay G, Chevrier A, Snow M, Rodeo S, Buschmann MD. Freeze-dried chitosan-platelet-rich plasma implants improve supraspinatus tendon attachment in a transosseous rotator cuff repair model in the rabbit. J Biomater Appl. 2019;33(6):792–807.
25. Vavken P, Sadoghi P, Palmer M, Rosso C, Mueller AM, Szoelloesy G, et al. Platelet-rich plasma reduces retear rates after arthroscopic repair of small- and medium-sized rotator cuff tears but is not cost-effective. Am J Sports Med. 2015;43(12):3071–6.
26. Mazzocca AD, McCarthy MB, Chowaniec DM, Cote MP, Arciero RA, Drissi H. Rapid isolation of human stem cells (connective tissue progenitor cells) from the proximal humerus during arthroscopic rotator cuff surgery. Am J Sports Med. 2010;38(7):1438–47.
27. Li Z, Zhang Y. Efficacy of bone marrow stimulation in arthroscopic repair of full thickness rotator cuff tears: a meta-analysis. J Orthop Surg Res. 2019;14(1):36.
28. Hoberman AR, Cirino C, McCarthy MB, Cote MP, Pauzenberger L, Beitzel K, et al. Bone marrow-derived mesenchymal stromal cells enhanced by platelet-rich plasma maintain adhesion to scaffolds in arthroscopic simulation. Arthroscopy. 2018;34(3):872–81.
29. Barber FA. PRP as an adjunct to rotator cuff tendon repair. Sports Med Arthrosc Rev. 2018;26(2):42–7.

6 Biological Augmentation in Rotator Cuff Repair: Cell Therapies

Carlo Alberto Stoppani, Sonia Maggi, Alessandra Menon, Chiara Fossati, and Pietro Randelli

Rotator cuff tears (RCTs) are one of the most common soft-tissue-related pathologies affecting up to half of the patients over age 60 [1, 2], and they continue to be an insidious challenge for orthopaedic surgeons. In the last decades, increasingly more patients undergo surgery to repair RCTs, but, even though the great advances made in technology and surgical techniques, the risk of re-ruptures after a rotator cuff repair is still high and remains a problem for the surgeons, especially in the treatment of massive RCTs [1].

According to the Snyder arthroscopic classification (Southern California Orthopedic Institute—SCOI—rotator cuff classification system), it is defined "massive" the complete RCTs larger than 3–4 cm involving two or more tendons with advanced fatty infiltration of tendons, important tendon retraction, and poor-quality tissues [3].

These kinds of lesions are also known as "irreparable cuff tears" because of the high intrinsic risk of failure of the surgical repair. It has been reported that recurrences after RCTs repair range between 20% and 40% for small-medium tears, and it is up to 94% for large or chronic tears [4, 5].

The high rate of failure is due to the complexity of tendon-to-bone integration because of the great biomechanical imbalance between tendon and bone and the insufficient regenerative potential of native tissues [1]. In fact, in most cases the tear occurs at the fibrocartilage zone of the tendon-to-bone junction, which is poorly vascularized, so the healing process is slow and it ends with a fibrous scar, subverting native biomechanical and histological structures.

Proximal humeral epiphysis is known to contain a pool of mesenchymal stem cells (MSCs) but little is known about their amount, healing potential, and changes after rotator cuff injuries.

Some authors in their study tried to determine whether RCTs could modify great tuberosity MSCs' number and in which way they may contribute to poor healing response. They found out that the size of the tear and the time between injury and surgery are directly proportional to the

C. A. Stoppani · S. Maggi
Laboratory of Applied Biomechanics, Department of Biomedical Sciences for Health, Università degli Studi di Milano, Milan, Italy

1° Clinica Ortopedica, ASST Centro Specialistico Ortopedico Traumatologico Gaetano Pini-CTO, Milan, Italy

A. Menon (✉) · C. Fossati · P. Randelli
Laboratory of Applied Biomechanics, Department of Biomedical Sciences for Health, Università degli Studi di Milano, Milan, Italy

1° Clinica Ortopedica, ASST Centro Specialistico Ortopedico Traumatologico Gaetano Pini-CTO, Milan, Italy

Research Center for Adult and Pediatric Rheumatic Diseases (RECAP-RD), Department of Biomedical Sciences for Health, Università degli Studi di Milano, Milan, Italy
e-mail: pietro.randelli@unimi.it

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_6

decrease of MSCs of the great tuberosity, especially at the tendon-bone interface [6].

Therefore, in the last years greater attention has been given to biological augmentation for surgical repair of RCTs in order to improve the healing rate [1].

This chapter will focus on biological augmentation strategies based on the use of tissue-derived MSCs cell and, in particular, bone marrow and adipose tissue-derived ones.

6.1 Mesenchymal Stem Cells (MSCs)

MSCs are multipotent stromal cells that have the potential to differentiate into various cell types, including osteoblasts, chondrocytes, tenocytes, myocytes, and adipocytes [7].

MSCs have generated great excitement among orthopaedists and researchers as a potential source for cell-based treatment strategies, thanks to their intrinsic ability to self-renew and differentiate into different connective tissue cell types [8].

Furthermore, MSCs have shown great potential for the replacement of damaged tissues such as bone, cartilage, tendon, and ligament, so they are becoming increasingly promising biological augmentation for surgery in the treatment of different orthopaedic pathology.

They can be collected from a variety of tissues and their ubiquity is correlated to their origin from perivascular cells (pericytes) known to be located in all vascularized tissues [8].

The majority of MSCs used for orthopaedic applications are obtained from bone marrow tissue, which is relatively easy to access and provide relatively high numbers of MSCs. More recent studies have focused their attention also on adipose-derived MSCs, which can be even more easily collected by needle biopsy or liposuction aspiration.

Other alternative sources of stem cells have been identified, such as muscles, tendons, cartilage, synovium, blood, skin, testes, hair, and scalp tissue [9]; since they are less commonly used, they are not the subject of extensive discussion.

Mesenchymal stem cells own great heterogeneity, as demonstrated in both in vivo and in vitro studies, because the MSC pool includes not only mesenchymal stem cells themselves but also subpopulation of cells at different stages of differentiation [8].

MSCs have also been demonstrated to have immune modulation properties and trophic potential in response to injuries, so that they have been defined also as "drugstore." They are able to home-in on injured sites and to secrete cytokines and growth factors in order to enhance healing response and tissue repair [10].

6.2 Bone Marrow–Derived Mesenchymal Stem Cells (BM-MSCs)

Bone marrow–derived mesenchymal stem cells (BM-MSCs) can be easily collected and harvested from long-bone epiphysis, such as the humeral head, or iliac crest and re-injected in the injured site to improve its healing. Starting from the notion that the newly formed fibrovascular scar tissue at the tendon-to-bone interface after surgical RCT repair has poor biological properties, different studies have been made to assess the effectiveness of BM-MSCs on tendon-to-bone surface healing and also on the enhancement of tendon attachment strength [11, 12].

In a recent animal study, Gao et al. tested the healing effectiveness of MSCs with the transducer of ErbB2.1 (TOB1) deficiency transplanted at the injured tendon-bone junction [13]. TOB1 is a negative regulation of BMP/Smad signaling, which is involved in osteoblast differentiation and tendon-bone healing process. MSCs were isolated and collected from the tibia and femur of male Sprague-Dawley rats at the age of 12 weeks; they were harvested and TOB1 suppression was induced. Gao and colleagues surgically detached supraspinatus tendon from rats and then they performed the sutures. Before tying the sutures, they randomly separated the rat pools into four different groups: three groups received a fibrin glue carrier with three different MSC augmentations at the tendon-bone interface and one group,

the control group, did not receive any augmentation to surgery. At 8 weeks after surgery, in MSC groups there was evidence of more orderly collagen fibers in contrast to the control group, and a small amount of chondrocytes was detected at the tendon-bone interface, showing a promising greater healing potential using MSCs with TOB1 suppression [13].

Gulotta et al. studied the use of MSCs in a *Lewis*-type rat model after unilateral surgical detachment and repair of the supraspinatus tendon [14]. They used the MSCs transducted with the product of a gene involved in tendon-to-bone embryogenesis (Ad-hMT1-MMP) compared to untransducted cells. Both groups were analyzed at 4 weeks after surgery: biomechanical tests showed better ultimate load-to-failure rate and more new cartilage formation in the group with MT1-MMP gene overexpression, introducing an interesting cue for further studies on tendon repair by recreating similar local conditions as they happen during embryogenesis [14].

This study was repeated using the BMP-13 enzyme, which is known to be part of the tendon healing process, but no significant results were obtained [11].

In a more recent study, Thangarajah et al. investigated the efficacy of BM-MSCs augmentation to scaffolds in supraspinatus tendon tear repair in a *Wistar*-type rat model. Eighteen rats underwent surgical unilateral detachment of the right supraspinatus tendon and one additional animal was used to collect stem cells. The surgical repair was performed after 3 weeks and the animals were divided into three different groups: the first group received BM-MSC augmentation on a demineralized bone matrix, the second group received BM-MSCs on a human dermal matrix, and the last group received the BM-MSC augmentation alone, without any scaffold. After 6 weeks, specimens were collected for postoperative analyses: MSCs' tracking showed that they remained at the tendon-to-bone interface where they were implanted; furthermore there was a complete closure of the tendon-bone gap and the demineralized bone matrix scaffold with MSCs reached a total bone mineral density at the surgical site similar to the contralateral side [12].

To our knowledge, clinical trials have been rare so far, but they give promising results [15–17].

In 2015, Havlas et al. published their preliminary results on a limited number of patients regarding the safety of cultured human MSCs in orthopaedic treatments and their effect on tendon healing. They collect a small group of ten patients with RCTs who had met the indication for arthroscopic repair. Two patients were excluded from the study due to exclusion criteria. Pain intensity questionnaires have been submitted to the eight remaining patients, including VAS, UCLA, and Constant-Murley scores. The authors harvested patients' bone marrow 3–4 weeks before surgery; a suspension of cultured BM-MSCs was applied to the suture site during the cuff repair. Postoperative questionnaires were submitted at 6 months after surgery and they showed an improvement of all the average values (VAS: 0, UCLA: 32, Constant-Murley: 84). At the same time MRI was performed, showing a fully healed tendon-to-bone surface tissue in all the patients. No adverse events were recorded [15].

In a case-controlled study, Hernigou et al. treated 90 patients for complete supraspinatus tears divided into two different groups of 45 patients each. The groups were matched taking count of these parameters: size and location of the tear, dominant side, gender, age, and same surgical repair technique. The first group underwent surgery with BM-MSC augmentation while the second one without any biological adjuvant. Results were assessed with MRI postoperatively at different timings: 3 and 6 months, 1 and 2 years, and at the most recent follow-up MRI (minimum 10 years follow-up). They found that 100% of the first group patients had the tear healed in 6 months against 67% of patients treated without the augmentation. Furthermore, the first group had a less failure rate in the following 10 years: 87% of intact rotator cuffs in the BM-MSC treated group against 44% in the untreated group [16].

An even larger study was performed by Taniguchi et al. who treated 111 patients with chronic medium-to-large RCTs. They used a novel arthroscopic technique described by Yamaguchi et al. for rotator cuff repair using medial anchors and lateral transosseous sutures

(surface-holding repair technique—ASH). The patients were divided into two different homogeneous groups: BM-group, composed of 67 patients treated with ASH plus BM-MSCs, and non-BM-group, composed of 44 patients treated with the ASH procedure alone. BM-MSCs were recruited from the humeral bone after drilling its surface with 4–6 holes in the footprint area with a metal bar during arthroscopy, after anchor insertion. MRI was performed before the surgery and at least 12 months postoperatively to assess the rate of healing and re-tears. Rotator cuff integrity was evaluated by using a score derived from Sugaya's classification, dividing cuff integrity in five different groups from type I (repaired cuff with sufficient thickness, 1 point) to type V (major discontinuity, 5 points). At 1 year follow-up, the re-tear rate was 23.9% in the non-BM-group versus 9.1% in BM-group and the cuff integrity score was significantly higher in the non-BM-group for larger tears, but it did not differ significantly for medium-size lesions. No complications were observed either intraoperatively or postoperatively. These results showed a great effectiveness on RCT healing, especially in large chronic tears, and reduction of re-tear rate by using BM-MSC augmentation [17].

6.3 Muscle-Derived Mesenchymal Stem Cells (MD-MSCs)

Another source of MSCs is muscular tissue; encouraging results have been reported in animal model studies of rotator cuff healing, opening the way to investigate the role of muscle-derived MSCs (MD-MSCs) in humans.

Pelinkovic et al. in their study injected high purified M-MSCs into native supraspinatus tendon of 8-week-old athymic rats and monitored them for 3 weeks. From 7 days after the injection, the cells were histologically integrated into the tendon collagen bundles showing fibroblastic phenotype differentiation [18].

Tsai et al. starting from the idea that if injured rotator cuff tissues can differentiate into other type of tissues such as bone (rotator cuff calcifications) and fat (fatty degeneration of rotator cuff muscles), then they could have an intrinsic differentiation potential and endogenous stem cells could also be isolated from rotator cuff tissues. Firstly, they isolated the cells from rotator cuff and bone marrow of five patients and then they analyzed their superficial markers: same surface protein profiles were expressed in both rotator cuff–derived stem cells (RC-MSCs) and BM-MSCs, showing their potential to differentiate into osteogenic, adipogenic, and chondrogenic progenitors. Furthermore, they demonstrated an enhancement of RC-MSCs' myogenic potential both in vivo and in vitro models of myogenic injury [19].

Coleman et al. compared the effectiveness of M-MSCs and BM-MSCs in an adult sheep model. They made the hypothesis that a single dose injection of MSC in rotator cuff muscle at the time of surgical repair could promote the healing rate by improving muscular function and decreasing fatty infiltration. Twenty-four adult sheep underwent surgery for detachment of supraspinatus tendon that was then repaired 6 weeks later. At the time of the surgical repair, animals were divided into three groups: surgery plus M-MSCs, surgery plus BM-MSCs, or surgery alone.

BM-MSCs were collected from the iliac crest and the M-MSCs from muscle tissues of three donor animals and then cultured and expanded before injection. Three months after surgical repair, the infraspinatus generated an increase in average loads of 29% for M-MSCs and 40% for BM-MSCs compared to the control group [20].

6.4 Adipose-Derived Mesenchymal Stem Cells (AD-MSCs)

As initially described by Zuk et al. [21], adipose tissue has proven to be a valid source of multipotent MSCs that are commonly defined as adipose-derived MSCs (AD-MSCs). These cells can proliferate and differentiate into different types of mesenchymal cells, like tenocytes, myocytes, chondrocytes, and also express their paracrine effect, releasing growth factor and

cytokines. In comparison with BM-MSCs, this source of stem cells presents some considerable advantages: easy harvest procedure such as liposuction, high cellularity, and minimal discomfort for the patient.

Stated the high biological capability of this stem cell, the injection of processed lipoaspirate has been used in different clinical conditions like osteoarthritis, chondromalacia, meniscus tear, osteonecrosis of femoral head, Achilles tendinopathy, lateral epicondylitis [22], and also in cardiac fibrosis [23], diabetic ulcers [24], skin repair [25], and nerve injury [26].

Focus on RCTs, one of the first studies that evaluates the effect of AD-MSC injection during rotator cuff repair, has been performed by Oh et al. [27]. In this study, four groups of eight rabbits were treated for subscapularis lesions, respectively, with AD-MSC plus suture, saline plus suture, AD-MSC only, and saline only. Six weeks after the procedure, electromyographic, biomechanical, and histological analyses were performed. The AD-MSC plus suture group demonstrated the best and more statistically significant results for all the analyzed variables with larger compound muscle action potential area, higher load-to-failure, and smaller proportion of fatty infiltration in comparison with all the other groups.

Recently, Rothrauff et al. [28] did not find positive results with the use of AD-MSCs on sutures of massive RCTs. They evaluated 48 rats with a chronic tendon lesion and 48 rats with acute lesion treated with no repair, repair only or repair augmented with fibrin, gelatin methacrylate (GelMA), fibrin plus AD-MSCs, GelMA plus AD-MSCs, fibrin plus AD-MSCs plus TGF-β3, or GelMA plus AD-MSCs plus TGF-β3. At the 4-week follow-up, no significant differences emerged in histologic appearance or structural properties (load-to-failure in ultimate load, maximum elongation, energy absorption). The only advantage of AD-MSC addition has been founded in terms of reduction of the bone loss (Bone Mineral Density) of the proximal humeral epiphysis in rats with chronic lesions.

Moving to studies in humans, it is important to mention the article of Kim et al. [29] that considered the effect of AD-MSCs loaded in fibrin glue after a rotator cuff repair. The authors evaluated the outcomes of the surgery in terms of VAS, range of motion, Constant-Murley score, and UCLA score, and they also assessed the integrity of the repaired tendon with an MRI at minimum of 12 months follow-up. Stated the common improvement of patients' performances after surgery, there is no significant difference in terms of clinical results between patients operated conventionally and those that received AD-MSC injection. Conversely, the radiological results highlight a significant difference in the re-tear rate in favor of patients treated with the addition of stem cells (28.5% vs. 14.3%).

Jo et al. [30] evaluates the efficacy and safety of intratendinous injection of AD-MSCs in patients with partial thickness RCTs. The first part of the study aims to verify the safety and tolerability of increasing dose of stem cells: no significant difference was found between low dose (1.0×10^7 cells) and high dose (1.0×10^8 cells) injections. The second part of the study evaluated the efficacy of high-dose intratendinous injection under ultrasonographic guidance after arthroscopic evaluation of the lesion. At the 6-month follow-up, the Constant-Murley score significantly increased (20%) and the VAS pain-on-motion significantly decreased (71%) in the high-dose group. The positive results are also confirmed by arthroscopic evaluation of the lesion with a decrease of bursal and articular defect of more than 80%.

At the moment, different studies on humans are underway of realization and probably only future long-term randomized control trials would clarify the effects of AD-MCSs on rotator cuff repair.

6.5 Tendon-Derived Mesenchymal Stem Cells (TD-MSCs)

In the research for the best source of MSCs, the tendon has also been studied in the last few years, and different articles stated the existence of stem cells coming from the tendon (TD-MSCs) [19, 31–33]. In particular, in 2013 Randelli et al. [32] collected samples of supraspinatus and long-

head biceps tendon and after adequate preparation, culture, and stimulation, they isolated adult stem cells with high regenerative potential and capability to differentiate into osteoblasts, adipocytes, and skeletal muscle cells. Nagura et al. [31] found similar results with samples of edges of the rotator cuff harvested during arthroscopic repair, suggesting a potential regenerative and self-repair capacity in the torn tendon.

Research on TD-MSCs on animals began in 2012 when Shen et al. [34] treated a group of 14 rabbits with an allogenic rotator cuff TD-MSC-enriched knitted silk-collagen scaffold in addition to rotator cuff repair. In comparison with the non-loaded scaffold group, the treatment group showed increased fibroblastic cell ingrowth, reduced infiltration of lymphocytes, and improved biomechanical and structural qualities at 12 weeks after repair.

Unfortunately, no clinical studies on humans have been performed yet. This is probably due to some complexity that are intrinsic in this procedure (two steps, high cost, cell expansion time, infection risk), and it is necessary to define a safety and reproducible technique for the use of TD-MSCs before these could be used in clinical practice.

6.6 Conclusions

In the last decades, great attention has been focused on the use of MSCs as a biological adjuvant for the treatment of RCTs because of their ability to differentiate and directly participate in the healing process. They are known to produce cytokines and growth factors that improve healing mechanisms at the site of inflammation or injury [10].

They have become an attractive option in biological augmentation strategies because they can be easily collected from many different tissues (bone marrow, adipose, tendon and muscular tissues, etc.), and their use has proven to be feasible and safe both for the direct injection of MSC suspension alone into the site of injury and for the injection with a matrix-carrier-like fibrin glue [35].

In a recent systematic review, Ahmad et al. have concluded that MSCs have a positive effect on healing process by producing a tissue that is similar to the pre-injury state, but the available evidence is still limited [36].

On the contrary, Haiko et al. claimed that, with the current state of knowledge, there is no high-quality evidence to support the use of MSCs for tendon disorders because many studies are at high risk of bias [37].

Further investigations are, therefore, needed to assess the real effectiveness of these promising cell-based therapies for tendon healing until a safe and satisfactory procedure can be developed for routine use.

References

1. Patel S, Gualtieri AP, Lu HH, Levine WN. Advances in biologic augmentation for rotator cuff repair. Ann N Y Acad Sci. 2016;1383:97–114.
2. Tashjian RZ. Epidemiology, natural history, and indications for treatment of rotator cuff tears. Clin Sports Med. 2012;31:589–604.
3. Kim S-J, Lee I-S, Kim S-H, Lee W-Y, Chun Y-M. Arthroscopic partial repair of irreparable large to massive rotator cuff tears. Arthroscopy. 2012;28:761–8.
4. Galatz LM, Ball CM, Teefey SA, Middleton WD, Yamaguchi K. The outcome and repair integrity of completely arthroscopically repaired large and massive rotator cuff tears. J Bone Joint Surg Am. 2004;86-A:219–24.
5. Sears BW, Choo A, Yu A, Greis A, Lazarus M. Clinical outcomes in patients undergoing revision rotator cuff repair with extracellular matrix augmentation. Orthopedics. 2015;38:e292–6.
6. Hernigou P, Merouse G, Duffiet P, Chevalier N, Rouard H. Reduced levels of mesenchymal stem cells at the tendon–bone interface tuberosity in patients with symptomatic rotator cuff tear. Int Orthop. 2015;39:1219–25.
7. Ankrum JA, Ong JF, Karp JM. Mesenchymal stem cells: immune evasive, not immune privileged. Nat Biotechnol. 2014;32:252–60.
8. Baksh D, Song L. Tuan RS Adult mesenchymal stem cells: characterization, differentiation, and application in cell and gene therapy. J Cell Mol Med. 2004;8:301–16.
9. Grogan SP, Miyaki S, Asahara H, D'Lima DD, Lotz MK. Mesenchymal progenitor cell markers in human articular cartilage: normal distribution and changes in osteoarthritis. Arthritis Res Ther. 2009;11:R85.

10. Caplan AI, Correa D. The MSC: an injury drugstore. Cell Stem Cell. 2011;9:11–5.
11. Gulotta LV, Kovacevic D, Packer JD, Ehteshami JR, Rodeo SA. Adenoviral-mediated gene transfer of human bone morphogenetic protein–13 does not improve rotator cuff healing in a rat model. Am J Sports Med. 2011;39:180–7.
12. Thangarajah T, Sanghani-Kerai A, Henshaw F, Lambert SM, Pendegrass CJ, Blunn GW. Application of a demineralized cortical bone matrix and bone marrow–derived mesenchymal stem cells in a model of chronic rotator cuff degeneration. Am J Sports Med. 2018;46:98–108.
13. Gao Y, Zhang Y, Lu Y, Wang Y, Kou X, Lou Y, Kang Y. TOB1 deficiency enhances the effect of bone marrow-derived mesenchymal stem cells on tendon-bone healing in a rat rotator cuff repair model. Cell Physiol Biochem. 2016;38:319–29.
14. Gulotta LV, Kovacevic D, Montgomery S, Ehteshami JR, Packer JD, Rodeo SA. Stem cells genetically modified with the developmental gene MT1-MMP improve regeneration of the supraspinatus tendon-to-bone insertion site. Am J Sports Med. 2010;38:1429–37.
15. Havlas V, Kotaška J, Koníček P, Trč T, Konrádová Š, Kočí Z, Syková E. [Use of cultured human autologous bone marrow stem cells in repair of a rotator cuff tear: preliminary results of a safety study]. Acta Chir Orthop Traumatol Cech. 2015;82:229–234.
16. Hernigou P, Flouzat Lachaniette CH, Delambre J, Zilber S, Duffiet P, Chevallier N, Rouard H. Biologic augmentation of rotator cuff repair with mesenchymal stem cells during arthroscopy improves healing and prevents further tears: a case-controlled study. Int Orthop. 2014;38:1811–8.
17. Taniguchi N, Suenaga N, Oizumi N, Miyoshi N, Yamaguchi H, Inoue K, Chosa E. Bone marrow stimulation at the footprint of arthroscopic surface-holding repair advances cuff repair integrity. J Shoulder Elbow Surg. 2015;24:860–6.
18. Pelinkovic D, Lee J-Y, Engelhardt M, Rodosky M, Cummins J, Fu FH, Huard J. Muscle cell-mediated gene delivery to the rotator cuff. Tissue Eng. 2003;9:143–51.
19. Tsai CC, Huang TF, Ma HL, Chiang ER, Hung SC. Isolation of mesenchymal stem cells from shoulder rotator cuff: a potential source for muscle and tendon repair. Cell Transplant. 2013;22:413–22.
20. Coleman S, Ehteshami J, Kisiday J, Altchek D, Warren R, Turner A. The effects of mesenchymal stem cells on rotator cuff muscle in a chronic injury model in sheep. In: 55th Annual Meeting of the Orthopaedic Research Society.
21. Zuk PA, Ph D, Zhu MIN, Mizuno H, Benhaim P, Lorenz HP. Multilineage cells from human adipose tissue: implications for cell-based therapies. Tissue Eng. 2001;7:211–29.
22. Pak J, Lee JH, Park KS, Park M, Kang L, Lee SH. Current use of autologous adipose tissue-derived stromal vascular fraction cells for orthopedic applications. J Biomed Sci. 2017;24:9.
23. Bai X, Alt E. Myocardial regeneration potential of adipose tissue-derived stem cells. Biochem Biophys Res Commun. 2010;401:321–6.
24. Gadelkarim M, Abushouk AI, Ghanem E, Hamaad AM, Saad AM, Abdel-Daim MM. Adipose-derived stem cells: effectiveness and advances in delivery in diabetic wound healing. Biomed Pharmacother. 2018;107:625–33.
25. Chavez-Munoz C, Nguyen KT, Xu W, Hong S-J, Mustoe TA, Galiano RD. Transdifferentiation of adipose-derived stem cells into keratinocyte-like cells: engineering a stratified epidermis. PLoS One. 2013;8:e80587.
26. Kingham PJ, Kolar MK, Novikova LN, Novikov LN, Wiberg M. Stimulating the neurotrophic and angiogenic properties of human adipose-derived stem cells enhances nerve repair. Stem Cells Dev. 2014;23:741–54.
27. Oh JH, Chung SW, Kim SH, Chung JY, Kim JY. 2013 Neer Award: effect of the adipose-derived stem cell for the improvement of fatty degeneration and rotator cuff healing in rabbit model. J Shoulder Elbow Surg. 2014;23:445–55.
28. Rothrauff BB, Smith CA, Ferrer GA, Novaretti JV, Pauyo T, Chao T, Hirsch D, Beaudry MF, Herbst E, Tuan RS, Debski RE, Musahl V. The effect of adipose-derived stem cells on enthesis healing after repair of acute and chronic massive rotator cuff tears in rats. J Shoulder Elbow Surg. 2019; 28:654.
29. Kim YS, Sung CH, Chung SH, Kwak SJ, Koh YG. Does an injection of adipose-derived mesenchymal stem cells loaded in fibrin glue influence rotator cuff repair outcomes? A clinical and magnetic resonance imaging study. Am J Sports Med. 2017;45:2010–8.
30. Jo CH, Chai JW, Jeong EC, Oh S, Kim PS, Yoon JY, Yoon KS. Intratendinous injection of autologous adipose tissue-derived mesenchymal stem cells for the treatment of rotator cuff disease: a first-in-human trial. Stem Cells. 2018;36:1441–50.
31. Nagura I, Kokubu T, Mifune Y, Inui A, Takase F, Ueda Y, Kataoka T, Kurosaka M. Characterization of progenitor cells derived from torn human rotator cuff tendons by gene expression patterns of chondrogenesis, osteogenesis, and adipogenesis. J Orthop Surg Res. 2016;11:40.
32. Randelli P, Conforti E, Piccoli M, Ragone V, Creo P, Cirillo F, Masuzzo P, Tringali C, Cabitza P, Tettamanti G, Gagliano N, Anastasia L, Bergante S, Ghiroldi A. Isolation and characterization of 2 new human rotator cuff and long head of biceps tendon cells possessing stem cell–like self-renewal and multipotential differentiation capacity. Am J Sports Med. 2013;41:1653–64.
33. Utsunomiya H, Uchida S, Sekiya I, Sakai A, Moridera K, Nakamura T. Isolation and characterization of human mesenchymal stem cells derived from shoulder tissues involved in rotator cuff tears. Am J Sports Med. 2013;41:657–68.

34. Shen W, Chen J, Yin Z, Chen X, Liu H, Heng BC, Chen W, Ouyang HW. Allogenous tendon stem/progenitor cells in silk scaffold for functional shoulder repair. Cell Transplant. 2012;21:943–58.
35. Kwon DR, Park G-Y. Adult mesenchymal stem cells for the treatment in patients with rotator cuff disease: present and future direction. Ann Transl Med. 2018;6:432.
36. Ahmad Z, Wardale J, Brooks R, Henson F, Noorani A, Rushton N. Exploring the application of stem cells in tendon repair and regeneration. Arthroscopy. 2012;28:1018–29.
37. Pas HIMFL, Moen MH, Haisma HJ, Winters M. No evidence for the use of stem cell therapy for tendon disorders: a systematic review. Br J Sports Med. 2017;51:996–1002.

7 Biological Augmentation in Rotator Cuff Repair: Scaffolds

Emre Bilgin, Mehmet Kapicioglu, and Kerem Bilsel

7.1 Introduction to scaffolds

Despite the improved implant designs and surgical techniques, the failure rates remain high following rotator cuff (RC) repair. Incidence of re-tears were reported to be 11% in smaller tears but increased up to 94% in massive tears [1, 2]. Hence, studies initially focused on surgical strategies such as the "double row" suture technique to restore the mechanical strength of RC. However, the double row-technique was not found to be superior to the single-row technique based on re-tear rates, and failure of the repair has remained a significant issue in shoulder surgery [3, 4].

The poor healing capacity of RC led to orthopedic research interested in biological augmentation. Thereafter, various approaches have been investigated to improve the healing potential of cuff repair [5, 6]. Cellular tendon augmentation via either allogenic or autogenic sources is determined to be a way of biological augmentation [7]. Allografts from cadaveric Achilles, quadriceps, and patellar tendon were transplanted for massive RC tears. However, improvement of functional scores was not found to be significant compared to the patients with similar conditions who underwent subacromial decompression and RC debridement surgery alone. In addition, increased infection and rejection risk were reported as disadvantages of allograft materials [8].

Augmentation with autologous tenotomized biceps tendon is another way of reinforcement with cellular components. Regarding the decreased possibility of host response and ability of performing readily without secondary incision to harvest the graft, tenotomized long head of biceps seemed to be a useful option [7]. Although biceps-augmented patients showed greater muscle strength and lower structural failure rate, equivalent clinical outcomes were demonstrated in terms of range of motion, pain, and functional scores with non-augmented controls [9].

At date, biological augmentation methods include the use of growth factors, stem cell therapies, and scaffolds or a combination thereof. Scaffolds used in RC surgery are typically classified under three main designations: biological

E. Bilgin
Department of Orthopaedics and Traumatology, Tepecik Training and Research Hospital, Health Science University, İzmir, Turkey

M. Kapicioglu
Faculty of Medicine, Department of Orthopaedics and Traumatology, Bezmialem Vakif University, İstanbul, Turkey

K. Bilsel (✉)
Faculty of Medicine, Department of Orthopaedics and Traumatology, Bezmialem Vakif University, İstanbul, Turkey

Ortopedi ve Travmatoloji Anabilim Dali, Bezmialem Vakif Universitesi Hastanesi, Istanbul, Turkey

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_7

scaffolds (extracellular matrix patches), synthetic scaffolds, or combinations. Extracellular matrix (ECM) scaffolds are manufactured from mammalian tissues. Cell sources can be from porcine small intestinal submucosa (SIS), porcine dermis, bovine dermis, equine pericardium, human dermis, and human fascia lata. By definition, xenografts are ECMs derived from non-human sources, and allografts are ECMs derived from human sources [10–12].

On the other hand, synthetic scaffolds are manufactured from chemical compounds and consist of various polymers including polyester, polypropylene, polyarylamid, Dacron, carbon, silicone, or nylon [13]. The advantages of synthetic scaffolds are that they do not carry a risk of disease transmission and have superior mechanical properties compared with biological scaffolds [12]. Synthetic patches can provide a strong structural environment until the host tissue heals. However, they have a limited biological impact on RC healing in contrast to biological ECM patches [10]. Furthermore, their poor biocompatibility can cause long-term complications such as degeneration of the implants, failure associated with impaired stability, infection, synovitis, foreign body reaction (FBR), osteolysis, and osteoarthritis [13].

As an alternative approach, new generation devices which are synthetic, degradable, and biomimetic polymers have been emerging. These patches provide non-permanent support to the tissues due to their progressively resorbing structure and promote self-healing potential of the repair construct. Enhanced biocompatibility, sufficient mechanical properties, and flexible design are determinant characteristics of these degradable synthetic scaffolds. Poly-L-lactic acid (PLLA), poly-lactic-co-glycolic acid (PLGA), polycaprolactone, and polydioxanone are commonly used members of these implants [14].

7.2 Host Response to Scaffolds

ECM scaffolds are associated with an acute and intensive host response. Cellular components of the augmented scaffolds can be responsible for this antigenicity. Therefore, one goal of the manufacturer is to completely remove or eliminate the cellular components from ECMs for minimizing the risk of host response [10]. Decellularization techniques vary depending on the manufacturer's choice and mainly include physical, chemical, and enzymatic methods. Physical approaches are performed via freezing procedures or mechanical agitation of the harvested tissues. In chemical approaches, the cellular remnants are removed via consecutive washing steps after dissolving the tissue cells with detergents and hypotonic solutions. The enzymatic approach can be performed using a number of enzymes, such as trypsin, to lyse the cellular components [7, 15]. When combined with gamma irradiation, its effect may improve [16]. Each method has distinctive advantages and disadvantages. Hence, a combination of these techniques has been used to achieve complete decellularization.

Besides the cellular components, host response in the recipient may depend on the chemical structure of the scaffold, sterilization method, surgical exposure, and mechanical loading. Architecture of the biomaterial may both affect its degradation characteristics and remodeling potential as well as the recipient immune response. Researches to date reveal that cross-linking is associated with undesirable host response regardless of the ECM type [10, 17].

After implantation, scaffolds are recognized as foreign by the host tissues and induce inflammation defined as FBR. In non-biologic synthetic and modified biologic scaffolds, FBR creates a capsule formation that surrounds the scaffold, and this environment can lead to prolonged inflammatory response. In tissue-engineered scaffolds which are cell embedded, the cells within the patch may respond to this environment and stimulate the migration of inflammatory cells such as macrophages into the scaffolds [18].

The macrophages are known as orchestrators of the FBR. Following the migration they interact with cellular components and manage the inflammatory process through paracrine or juxtacrine signaling mechanisms [18]. Macrophages are classified into two main groups: either M1 or M2 types. The M1 type is associated with a proinflammatory response and typically represents the

inflammatory process associated with FBR. In contrast, the M2 type is associated with the remodeling process and stimulates tissue regeneration [19]. Thus, the macrophage phenotype can be considered as a predictive factor for the outcome of the scaffold augmentation. Currently, it has not been clearly identified yet which factor determines the macrophage type. However, chemically cross-linked scaffolds are prone to cause proinflammatory responses with the M1 type and not cross-linked (rapidly degraded) scaffolds which are more likely to cause a remodeling response with M2 type of macrophages [17].

7.3 Mechanical Properties of Scaffolds

The purpose of scaffold augmentation in RC repair include providing a mechanical support by "off-loading" the surgical repair at time zero and/or a biological environment to enhance the healing potential at the tendon-bone interface. The ECM scaffolds provide more biological advantages rather than the mechanical support. In contrast, synthetic scaffolds can degrade over time and maintain biomechanical support for longer periods depending on its chemistry. Some non-degradable synthetic scaffolds may remain in the tissues till the end of the patient's life. Variable degradation characteristics of ECM or synthetic scaffolds may affect their mechanical performance. On one hand, degradation can elicit impaired mechanical strength, on the other hand host cell integration and remodeling of the implant can concomitantly strengthen the repair construct [10].

Previously, numerous in vitro studies were performed to determine the mechanical characteristics of the various scaffold types to help identify their appropriate clinical usage. Barber et al. [20] biomechanically compared a number of human dermis–derived (GraftJacket, Permacol, TissueMend) and porcine SIS-derived scaffolds (CuffPatch, Restore). They reported that dermis-derived grafts had greater load-to-failure than SIS-derived grafts. GraftJacket Extreme (thicker form of the original patch) demonstrated the highest failure load (229 N). The failure was associated with suture pull-out in almost all cases, except one in which graft tearing occurred.

In another study, ECMs were found to be less elastic than the reported values of the human infraspinatus tendon, which may result in failure of the repair in regard to a decreased load-bearing capacity. SIS-derived patches (Restore, CuffPatch) demonstrated greater elastic modulus than dermis-derived (GraftJacket, TissueMend) patches. In addition, ECM scaffolds required 10–30% stretch before they started to bear significant load. Authors concluded that although ECMs have more biological benefits rather than mechanical, prestretching before the implantation may offer more functional contribution [11].

To better understand the mechanical characteristics of scaffold devices, the following studies used human cadaveric specimens. In one study, the mean load-to-failure in the non-augmented control group was found to be 273 N whereas it was 325 N in the GraftJacket Extreme augmented group in which single-row repair was performed. Failures were observed at the tendon-suture interface in 8 of 10 non-augmented and 6 of 10 augmented repairs. Suture breakage was observed in two and four in non-augmented and augmented repairs, respectively [21]. Omea et al. [22] demonstrated in single-row repaired constructs that the human dermal graft augmented group had significantly higher failure load compared to the non-augmented group (560 N and 345 N, respectively). Failures were observed through three different mechanisms: tendon cut-out ($n = 7$), suture breakage ($n = 3$), and suture anchor pull-out ($n = 3$).

Synthetic scaffolds were also studied in terms of their mechanical properties in human cadaveric shoulders. McCarron et al. [23] reported significantly increased yield load and ultimate failure load but not the initial stiffness of repair construct at time zero after PLLA graft (X-Repair) augmentation.

Recently, Smith et al. [24] investigated a number of synthetic (X-Repair, LARS ligament, Poly-Tape) and biologic ECM scaffolds via comparing their mechanical properties with cadaveric fresh frozen human supraspinatus. Synthetic scaffolds demonstrated greater load-to-failure. Among the scaffolds, LARS and X-Repair were the best performing on the macroscale. However, none of them entirely matched the native tendon in terms of macro- and micro-mechanical proper-

ties, probably because none of these devices were originally designed for RC repair.

Beitzel et al. [25] found that dermis patches (ArthroFlex) augmented on top of the reconstruction and collagen grafts (Mucograft) that are interposed between bone and tendon with double-row repair showed significantly higher load-to-failure (575 N and 573 N, respectively) under cyclic loads whereas interposed dermis patches showed also higher but non-significant load-to-failure (469 N) compared to the non-augmented controls (348 N). Consequently, collagen scaffolds demonstrate more biomechanical advantages when interposed between bone and tendon. However, dermis scaffolds seem to be more effective when augmented on the top of the tendon repair.

The results of previous in vitro studies confirmed that not only the scaffold type, but also the surgical technique including location, number, and type of the sutures as well as the location of the graft may affect the mechanical strength of augmented repair. Moreover, postoperative rehabilitation and existing joint pathology are associated with mechanical performance [10]. Above all, the mechanical strength of the primary tendon-bone reconstruction is the main determinant factor for the overall mechanical performance of the repair construct. The main goal should be to achieve a stable reconstruction of the tendon-bone interface even where scaffold augmentation is intended [26]. One should bear in mind that, however, results of in vitro studies may not entirely represent the in vivo biomechanical characteristic of a scaffold device.

7.4 Specific Scaffold Devices

7.4.1 Biological Scaffolds (Extracellular Matrix Patches)

Currently, more than 20 scaffolds are commercially available for surgical use of RC repair [24] (Tables 7.1 and 7.2). To date, natural ECMs are the most commonly used method to augment RC repair [17] After harvesting, the tissues are processed through various steps including general cleaning, removal of lipids and cellular compounds, cross-linking, and sterilization [27]. Eventually, scaffolds consist of a protein-based

Table 7.1 This table includes a number of commercially available synthetic scaffolds for RC surgery

Synthetic patch	Material	Degradation characteristics	Company
SportMesh	Polyurethane-urea	Partial degradable	Biomet Sports Medicine
X-Repair	Poly-L-lactic acid	Degradable	Synthasome
Poly-tape	Polyethylene terephthalate	Non-degradable	Xiros Ltd, Neoligaments
LARS ligament	Polyethylene terephthalate	Non-degradable	LARS
Biomerix RCR patch	Polycarbonate polyurethane-urea	Non-degradable	Biomerix
BioFiber	Poly-4-hydroxybutyrate	Degradable	Tornier
Gore-Tex patch	Expanded polytetraflouroethylene	Non-degradable	Gore Medical

Table 7.2 This table includes a number of commercially available biological ECM scaffolds for RC surgery

ECM patches	Tissue type	Source	Cross-linked	Company
Xenografts				
Restore Orthobiologic Implant	SIS	Porcine	No	DePuy Orthopaedics
CuffPatch	SIS	Porcine	Yes	Arthrotek
Zimmer Collagen Repair	Dermis	Porcine	Yes	Zimmer
Connexa	Dermis	Porcine	No	Tornier
Arthrex DX Reinforcment Matrix	Dermis	Porcine	No	Arthrex
TissueMend	Dermis	Bovine	No	Stryker Orthopaedics
OrthADAPT Bioimplant	Pericardium	Equine	Yes	Pegasus Biologics
Allografts				
GrafJacket	Dermis	Human	No	Wright Medical Technology
ArthroFlex	Dermis	Human	No	Arthrex
AlloPatch	Dermis	Human	No	MTF

SIS small intestinal submucosa, *MTF* Musculoskeletal Tissue Foundation

ECM, and they contain predominantly type I collagen fibers [11, 12].

The US Food and Drug Administration (FDA) regulation considers ECMs derived from non-human sources (xenografts) as medical devices through the 510(k) application, and these products have been cleared as augmentation devices "for reinforcement of the soft tissues that are repaired by suture or suture anchors during tendon repair including RC surgery." Animal and human studies are not required to prove the efficacy of these devices. However, ECMs derived from human sources (allografts) are classified as human tissue for transplantation, and FDA clearance is not mandatory for their use [10, 17]. Therefore, these devices have been used for augmentation as well as interpositional grafts for bridging the tendon repair (Fig. 7.1).

7.4.1.1 Xenografts

Restore Orthobiologic Implant (DePuy Orthopaedics) is an acellular, structural non-cross-linked porcine SIS. ECM contains over 90% collagen, approximately 5–10% lipids, and small amounts of carbohydrates and growth factors. The patch consists of 10 layers and is dry packaged [11, 12].

Early clinical researches on Restore yielded mixed results. Metcalf et al. [28] reported a 2-year follow up of 12 patients who underwent arthroscopic massive chronic RC repair augmented with porcine SIS. Significant improvement of shoulder range of motion (ROM) in each direction, abduction strength, and functional outcomes were demonstrated. Graft failure with complete resorption was observed in 1 of 12 patients within 12 weeks. Schlamberg et al. [29] evaluated 11 patients with large and massive RC tears treated with open repair. In contrast to former studies, they reported that magnetic resonance (MR) revealed re-tears in 10 out of 11 shoulders. ROM did not change and shoulder pain improved in seven patients postoperatively. However, the lack of non-augmented control groups was an important limitation of these studies.

In a prospective randomized control trial of the Restore implant, Iannoti et al. [30] reported

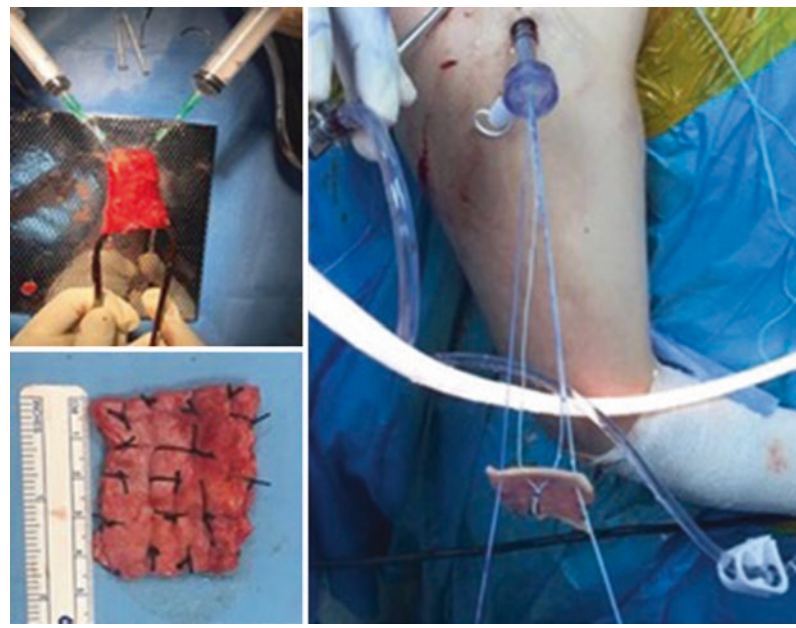


Fig. 7.1 Arthroscopic augmentation of a massive rotator cuff tear with an autologous tensor facia lata graft after preparation according to measured tear size

that RC tears healed in 4 out of 15 patients in the augmented group and in 9 out of 15 patients in the non-augmented group ($p = 0.11$). PENN score was significantly higher in the control group than in the augmented group (83–91, $p = 0.07$). Non-augmented repairs were found to be 7% more likely to heal than those in the control group. The authors concluded that Restore augmentation was insufficient to treat large and massive RC tears. Walton et al. [31] reported impaired muscle strength, greater impingement in external rotation, slower resolution of pain in activity, and longer duration of participation in sports in the Restore augmented group compared to the non-augmented group. At 2-year follow-up, re-tears were comparable between the two groups (6 of 10 in the study group, 7 of 12 in the control group). In the augmented group, four patients underwent open debridement for severe inflammation. Thus, the authors did not recommend the use of Restore as an augmentation graft for RC repairs.

CuffPatch Bioengineered Tissue Reinforcement (Arthrotek) is an acellular, 8–layered, porcine SIS sheet. The product is artificially cross-linked and packaged hydrated [11]. In rats, various types of host responses like multinucleated giant cells, proliferation of blood vessels, and tissue edema were observed at the implantation site [32]. Negligible amounts of porcine DNA were demonstrated inside the CuffPatch [11], and there is limited data on the clinical use and efficacy of the implant. Therefore, the CuffPatch is neither recommended nor contraindicated based on current researches [33].

Zimmer Collagen Repair Patch (Zimmer) is an acellular sheet of cross-linked single layer xenograft derived from porcine dermal tissue also known as the **Permacol Surgical Implant**. It is packaged hydrated [11]. Soler et al. [34] reported the early results of four patients who underwent massive RC repairs with Permacol as a bridging device. The graft failed in all of four patients between 3 and 6 months after the surgery.

The following studies demonstrated more promising results. Badhe et al. [35] evaluated ten patients with a mean age of 66 years where the Zimmer Collagen Patch was used to augment extensive RC tears. At the final follow-up (mean 4.5 years), the mean Constant scores increased from 41 to 62 ($p = 0.0003$). ROM and abduction strength significantly improved postoperatively. Radiologic evaluation revealed intact grafts in 8 of 10 patients and no adverse effect was observed during the follow-up period. Cho et al. [36] reported the results of five patients with massive RC tears who underwent mini-open surgery. Repairs were augmented with Permacol. All the patients showed improved pain relief and functional scores. No intraoperative and postoperative complications were noted. At MRI evaluation, repair was intact in four patients and re-tear was observed in one patient at an average of 8 months postoperatively.

Conexa Reconstructive Tissue Matrix (Tornier) is another ECM scaffold device made from porcine dermis. Gupta et al. [37] reported the augmentation results of 27 RC with massive or two tendon tears with the use of Conexa patch. At an average of 32 months, improved outcomes were reported in terms of active ROM, supraspinatus and external rotation strength, as well as functional scores. The mean American Shoulder and Elbow Surgeons (ASES) score increased from 62.7 to 91.8 ($p = 0.0007$), and Short-Form-12 (SF-12) score increased from 48.4 to 56.6 ($p = 0.044$) postoperatively. Ultrasound evaluations of 22 shoulders were obtained at a minimum of 2 years follow-up. Sixteen shoulders had an intact repair, five had a partially intact repair, and one shoulder had complete disruption at the graft-bone interface.

Arthrex DX Reinforcement Matrix (Arthrex) is also a porcine dermis-derived

ECM. In a recent retrospective comparative trial, Flury et al. [38] concluded that using a porcine dermal xenograft for RC repairs in over 60-year-old patients is insufficient to reduce the re-tear rates and improve the functional outcomes.

7.4.1.2 Allografts

GraftJacket Regenerative Tissue Matrix (Wright Medical Technology) is derived from human skin and composed of mainly collagen, elastin, and proteoglycans. The implant is single layered, not artificially cross-linked, and packaged dry [11]. GraftJacket has been also widely studied in clinical researches.

Bond et al. [39] arthroscopically repaired massive immobile RC tears using GraftJacket as an interpositional bridging graft. At a mean 26.8 months of follow-up period, 15 out of 16 patients were satisfied with their outcome. Significant improvement of Constant (from 53.8 to 84) and UCLA scores (from 18.4 to 30.4) were seen postoperatively. In addition, shoulder pain, forward flexion, and external rotation strength were improved. No complication was noted. MR evaluation revealed that 13 grafts completely incorporated into the native tissue. Failure of the graft was observed in three patients.

Wong et al. [40] reported 45 patients with massive irreparable RC tears arthroscopically treated using GraftJacket with a minimum follow-up of 2 years. Modified UCLA scores increased from 18.4 to 27.5 postoperatively ($p < 0.01$) and no graft rejection was observed. However, one patient who suffered from deep infection underwent arthroscopic debridement.

In a prospective randomized controlled trial, Barber et al. [41] compared the results of two groups of patients with greater than 3 cm, two-tendon tears. The patients in group 1 ($n = 22$) underwent arthroscopic RC repair with GraftJacket augmentation whereas the repairs in group 2 ($n = 20$) were performed without graft augmentation. ASES and Constant scores significantly improved in group 1 ($p = 0.035$, and $p = 0.008$, respectively). At a mean 14.5 months, 85% demonstrated intact cuff on MR evaluation in graft-augmented patients compared with 40% of non-augmented patients.

Similarly, Gupta et al. [42] demonstrated improved ASES scores (from 66.6 to 88.7) after interpositional repair of massive RC tears with GraftJacket. All the 24 patients were satisfied with their clinical result. Moreover, significant improvements were noted in terms of mean active forward flexion and external rotation, mean shoulder abduction, as well as supraspinatus and infraspinatus strength. No infection, inflammatory reaction, or graft rejection was observed. Partial graft re-tear occurred in one case because of patient noncompliance with postoperative rehabilitation.

ArthroFlex (Arthrex) is also a decellularized human dermal allograft (Fig. 7.2). The implant is packaged hydrated and terminally sterilized. Gilot et al. [43] examined the arthroscopic repair of 35 patients with massive RC tear with or without ArthroFlex augmentation. Re-tear incidence was 26.8% in control group (4 of 15) versus 10.4% in augmented group (2 of 20). ASES and SF-12 scores both significantly improved in the augmented group compared with the control group ($p = 0.02$ and 0.031, respectively).

AlloPatch HD (Musculoskeletal Tissue Foundation) is a human skin allograft. Agrawal [44] reported that 12 of 14 patients had intact repair based on MR evaluation after reinforcement with the AlloPatch of massive or previously failed RC repairs at a mean of 16.8 months. Constant score, pain score, scapular plane abduction, and strength were found to be significantly improved postoperatively. The authors concluded that the use of the implant is beneficial for the treatment of massive to large revision RC tears.

Fig. 7.2 Acellular human dermal allograft (ArthroFlex—Arthrex)

7.4.2 Synthetic Scaffolds

Few clinical studies investigated the outcomes of synthetic scaffolds for the treatment of massive RC tears. Encalada-Diaz et al. [45] reported the results of ten patients with full thickness supraspinatus or infraspinatus tears that underwent open repair with the Biomerix RCR Patch (polycarbonate polyurethane-urea) augmentation. Significant improvement in ASES scores, Simple Shoulder Test, ROM, and pain at both 6 and 12 months was observed. MRI and ultrasound evaluation showed 90% of intact repairs at 12 months. No adverse effect was reported.

Proctor [46] evaluated the functional results of X-Repair (PLLA)-augmented large to massive RC repairs. At 12 months, 15 of 18 patients demonstrated intact repair on MRI and ultrasound. At 42 months, intact repairs decreased to 14. Postoperative ASES scores significantly improved from 25 to 71 and 70 at 12 months and 42 months, respectively. In another study which investigated the same scaffold, 13 patients with massive and recurrent RC tears were evaluated [47]. At a mean 1.5 year follow-up, only five patients had an intact repair radiologically despite the significant improvement in ASES and PENN scores postoperatively (from 32.8 to 74.2 and from 50.9 to 77.6, respectively).

Audenaert et al. [48] reported the results of massive RC repairs of 41 patients using a polyester graft at a mean follow-up of 43 months. The mean Constant and Murley scores significantly improved postoperatively (from 25.7 to 72.1). The study group demonstrated significant pain relief and improvement in overhead activities. Nada et al. [49] also reported significantly improved clinical outcomes in terms of Constant score, ROM, and pain in the treatment of massive tears with a polyester patch (Dacron).

The LARS ligament (polyethylene terephthalate) was also used to reinforce the repair of massive RC tears. Petrie and Ismaiel [50] demonstrated significantly increased Oxford Shoulder scores and acromiohumeral distance in 31 shoulders with chronic massive cuff tear after LARS ligament augmentation.

Although these researches display promising results, they lacked a control group. Ciampi et al. [51] clinically compared 152 patients with massive RC tears who underwent surgical repair alone (n = 51) and with bovine pericardium-derived collagen patch (n = 49) or polypropylene patch (n = 51) augmentation. The results showed that

mean UCLA score, shoulder elevation, strength, and re-tear rates were significantly improved in the polypropylene group at 36 months.

7.4.3 Combinations

Recently, novel strategies have been developed such as electrospinning which was predominantly used to closely mimic the native orientation of tendon collagen bundles with structurally aligned synthetic scaffolds. This method provides an opportunity to create combinations via the incorporation of bioactive growth factors or embedding the stem cells into the scaffold devices [6, 14]. In this way, both improved mechanical performance and enhanced cellular activity at the repair site can be obtained simultaneously.

Zahao et al. [52] investigated PLGA and basic fibroblast growth factor (bFGF)-loaded PLGA membranes that are prepared via the electrospinning method. In rats, the authors found that the membranes had excellent biocompatibility and biodegradability. After in vivo RC surgery, bFGF-PLGA significantly improved the collagen organization compared with control and PLGA groups at 2, 4, and 8 weeks. Electrospun membranes demonstrated higher ultimate load-to-failure than the control group.

Yokoya et al. [53] compared control infraspinatus tendon defects in rats with reconstructed tears with poly-glycolic acid (PGA) sheets and autologous cultured mesenchymal stem cell (MSC)-seeded PGA sheet. They found higher volume of type I collagen than type III collagen in the MSC-PGA group compared to the PGA-only group, and besides, regenerated tendons in the MSC-PGA group demonstrated better tensile strength than the PGA-only and control groups at 16 weeks.

Combinations can be prepared via loading growth factors into collagen ECMs. Hee et al. [54] demonstrated higher load-to-failure as well as improved morphologic appearance including tendon-to-bone integration in the repair of ovine infraspinatus tears with augmentation of recombinant human platelet-derived growth factor–loaded bovine collagen matrix compared to the collagen matrix patch alone. Despite promising results in animals, we are not aware of any clinical data based on these novel combinations.

7.5 Summary

Currently, scaffolds have been the most common tissue-engineered approach used to obtain improved outcomes after RC augmentation. The rationale behind the usage of a scaffold device includes mechanical reinforcement of the repair construct as well as the biological enhancement of the healing potential of a RC tear [10].

The ideal scaffold should biomechanically match the physical characteristics of the tendon-bone interface and maintain a support until the healing completes. The implant should be cell-instructive and present with artificially oriented structures to closely mimic native tissue. In addition, it should be biodegradable to enable the new tendon-bone interface to completely integrate and regenerate without causing any side effects because of the degraded material. Finally, the device should artificially permit incorporation of growth factors, stem-cells, or minerals [6, 55]. Future directions may be focused on scaffold devices which meet these demands.

Researches to date have confirmed that porcine SIS-derived xenografts demonstrated higher failure rates with little to no clinical improvement. Because of causing severe inflammatory reactions due to high residual porcine DNA [56], further use of the Restore implant was not recommended for RC repair in humans [31]. Although porcine dermal xenografts and dermal allografts demonstrated more promising results, most of the studies lacked a control group. Moreover, concern still remains that allografts may also create an inflammatory response due to DNA remnants [11, 57].

Synthetic grafts are an alternative approach for the augmentation of RC repair. Several clinical studies demonstrated low complication and decreased re-tear rates and improved outcomes after implantation of various types of synthetic

patches. However, most of them were also unable to compare the study group with a control group (Table 7.3).

Table 7.3 Literature results of ECM and synthetic scaffolds based on clinical researches

Scaffold source	Tissue/material type	Literature results
Porcine	Small intestinal submucosa	Decreased healing potential of RC, high re-tear rates, increased impingement, and shoulder pain, associated with severe inflammatory reaction due to residual DNA content
Porcine	Dermis	Low adverse effect and complication risk, significantly improved functional outcomes. However, ineffective to treat massive tears in >60-year-old patients and when used as a bridging device
Human	Dermis	High satisfaction rates, improved functional scores, decreased re-tear rate, decreased inflammatory reaction, and rejection risk. Safe and useful for augmentation in revision RC surgery
Synthetic	Polycarbonate polyurethane-urea	Improved functional outcomes and 90% intact repair at 12 months. Low complication risk. No observed adverse effect
Synthetic	Poly-L-lactic acid	Improved functional scores and 78% intact repair at 42 months. In contrast, one study reported 62% re-tear rate despite significantly improved functional scores
Synthetic	Polyester	Significantly increased Constant scores. Improved pain and ROM
Synthetic	Polyethylene terephthalate	Successful clinical outcomes, radiologically decreased acromiohumeral distance
Synthetic	Polypropylene	Decreased re-tear rates, improved functional scores, and shoulder elevation at 36 months

Based on the current researches, the data is limited on the use of combination types of scaffolds in humans. There are only a few animal studies available with promising results in the literature. Nevertheless, further clinical studies are required to warrant the reliability and efficacy of these novel tissue-engineered devices.

References

1. Frank JB, ElAttrache NS, Dines JS, Blackburn A, Crues J, Tibone JE. Repair site integrity after arthroscopic transosseous-equivalent suture-bridge rotator cuff repair. Am J Sports Med. 2008;36(8):1496–503.
2. Galatz LM, Ball CM, Teefey SA, Middleton WD, Yamaguchi K. The outcome and repair integrity of completely arthroscopically repaired large and massive rotator cuff tears. J Bone Joint Surg Am. 2004;86(2):219–24.
3. Burks RT, Crim J, Brown N, Fink B, Greis PE. A prospective randomized clinical trial comparing arthroscopic single-and double-row rotator cuff repair: magnetic resonance imaging and early clinical evaluation. Am J Sports Med. 2009;37(4):674–82.
4. Franceschi F, Ruzzini L, Longo UG, Martina FM, Beomonte Zobel B, Maffulli N, Denaro V. Equivalent clinical results of arthroscopic single-row and double-row suture anchor repair for rotator cuff tears: a randomized controlled trial. Am J Sports Med. 2007;35(8):1254–60.
5. Zumstein MA, Lädermann A, Raniga S, Schär MO. The biology of rotator cuff healing. Orthop Traumatol Surg Res. 2017;103(1S):S1–S10.
6. Patel S, Gualtieri AP, Lu HH, Levine WN. Advances in biologic augmentation for rotator cuff repair. Ann N Y Acad Sci. 2016;1383(1):97–114.
7. Cheung EV, Silverio L, Sperling JW. Strategies in biologic augmentation of rotator cuff repair: a review. Clin Orthop Relat Res. 2010;468(6):1476–84.
8. Moore DR, Cain EL, Schwartz ML, Clancy WG. Allograft reconstruction for massive, irreparable rotator cuff tears. Am J Sports Med. 2006;34(3):392–6. https://doi.org/10.1177/0363546505281237.
9. Cho NS, Yi JW, Rhee YG. Arthroscopic biceps augmentation for avoiding undue tension in repair of massive rotator cuff tears. Arthroscopy. 2009;25(2):183–91. https://doi.org/10.1016/j.arthro.2008.09.012.
10. Ricchetti ET, Aurora A, Iannotti JP, Derwin KA. Scaffold devices for rotator cuff repair. J Shoulder Elbow Surg. 2012;21(2):251–65. https://doi.org/10.1016/j.jse.2011.10.003.
11. Derwin KA, Baker AR, Spragg RK, Leigh DR, Iannotti JP. Commercial extracellular matrix scaffolds for rotator cuff tendon repair: biomechanical, biochemical, and cellular properties. J Bone Joint Surg Am. 2006;88(12):2665–72.

12. McCormack RA, Shreve M, Strauss EJ. Biologic augmentation in rotator cuff repair: should we do it, who should get it, and has it worked? Bull Hosp Jt Dis. 2014;72(1):89–96.
13. Longo UG, Lamberti A, Khan WS, Maffulli N, Denaro V. Synthetic augmentation for massive rotator cuff tears. Sports Med Arthrosc Rev. 2011;19(4):360–5. https://doi.org/10.1097/jsa.0b013e318224e359.
14. Hakimi O, Mouthuy PA, Carr A. Synthetic and degradable patches: an emerging solution for rotator cuff repair. Int J Exp Pathol. 2013;94(4):287–92.
15. Gilpin A, Yang Y. Decellularization strategies for regenerative medicine: from processing techniques to applications. Biomed Res Int. 2017;2017:9831534.
16. Thangarajah T, Pendegrass CJ, Shahbazi S, Lambert S, Alexander S, Blunn GW. Augmentation of rotator cuff repair with soft tissue scaffolds. Orthop J Sports Med. 2015;3(6):2325967115587495.
17. Derwin KA, Badylak SF, Steinmann SP, Iannotti JP. Extracellular matrix scaffold devices for rotator cuff repair. J Shoulder Elbow Surg. 2010;19(3):467–76.
18. Saleh LS, Bryant SJ. The host response in tissue engineering: crosstalk between immune cells and cell-laden scaffolds. Curr Opin Biomed Eng. 2018;6:58–65. https://doi.org/10.1016/j.cobome.2018.03.006.
19. Valentin JE, Stewart-Akers AM, Gilbert TW, Badylak SF. Macrophage participation in the degradation and remodeling of extracellular matrix scaffolds. Tissue Eng Part A. 2009;15(7):1687–94.
20. Barber FA, Herbert MA, Coons DA. Tendon augmentation grafts: biomechanical failure loads and failure patterns. Arthroscopy. 2006;22(5):534–8.
21. Barber FA, Herbert MA, Boothby MH. Ultimate tensile failure loads of a human dermal allograft rotator cuff augmentation. Arthroscopy. 2008;24(1):20–4.
22. Omae H, Steinmann SP, Zhao C, Zobitz ME, Wongtriratanachai P, Sperling JW, An KN. Biomechanical effect of rotator cuff augmentation with an acellular dermal matrix graft: a cadaver study. Clin Biomech (Bristol, Avon). 2012;27(8):789–92.
23. McCarron JA, Milks RA, Chen X, Iannotti JP, Derwin KA. Improved time-zero biomechanical properties using poly-L-lactic acid graft augmentation in a cadaveric rotator cuff repair model. J Shoulder Elbow Surg. 2010;19(5):688–96.
24. Smith RD, Zargar N, Brown CP, Nagra NS, Dakin SG, Snelling SJ, Carr A. Characterizing the macro and micro mechanical properties of scaffolds for rotator cuff repair. J Shoulder Elbow Surg. 2017;26(11):2038–46.
25. Beitzel K, Chowaniec DM, McCarthy MB, Cote MP, Russell RP, Obopilwe E, Mazzocca AD. Stability of double-row rotator cuff repair is not adversely affected by scaffold interposition between tendon and bone. Am J Sports Med. 2012;40(5):1148–54.
26. Aurora A, McCarron JA, van den Bogert AJ, Gatica JE, Iannotti JP, Derwin KA. The biomechanical role of scaffolds in augmented rotator cuff tendon repairs. J Shoulder Elbow Surg. 2012;21(8):1064–71.
27. Longo UG, Lamberti A, Petrillo S, Maffulli N, Denaro V. Scaffolds in tendon tissue engineering. Stem Cells Int. 2012;2012:517165.
28. Metcalf MH, Savoie FH III, Kellum B. Surgical technique for xenograft (SIS) augmentation of rotator-cuff repairs. Oper Tech Orthop. 2002;12(3):204–8.
29. Sclamberg SG, Tibone JE, Itamura JM, Kasraeian S. Six-month magnetic resonance imaging follow-up of large and massive rotator cuff repairs reinforced with porcine small intestinal submucosa. J Shoulder Elbow Surg. 2004;13(5):538–41.
30. Iannotti JP, Codsi MJ, Kwon YW, Derwin K, Ciccone J, Brems JJ. Porcine small intestine submucosa augmentation of surgical repair of chronic two-tendon rotator cuff tears: a randomized, controlled trial. J Bone Joint Surg Am. 2006;88(6):1238–44.
31. Walton JR, Bowman NK, Khatib Y, Linklater J, Murrell GA. Restore orthobiologic implant: not recommended for augmentation of rotator cuff repairs. J Bone Joint Surg Am. 2007;89(4):786–91.
32. Valentin JE, Badylak JS, McCabe GP, Badylak SF. Extracellular matrix bioscaffolds for orthopaedic applications: a comparative histologic study. J Bone Joint Surg Am. 2006;88(12):2673–86.
33. Gillespie RJ, Knapik DM, Akkus O. Biologic and synthetic grafts in the reconstruction of large to massive rotator cuff tears. J Am Acad Orthop Surg. 2016;24(12):823–8.
34. Soler JA, Gidwani S, Curtis MJ. Early complications from the use of porcine dermal collagen implants (Permacol) as bridging constructs in the repair of massive rotator cuff tears. A report of 4 cases. Acta Orthop Belg. 2007;73(4):432–6.
35. Badhe SP, Lawrence TM, Smith FD, Lunn PG. An assessment of porcine dermal xenograft as an augmentation graft in the treatment of extensive rotator cuff tears. J Shoulder Elbow Surg. 2008;17(1):S35–9.
36. Cho CH, Lee SM, Lee YK, Shin HK. Mini-open suture bridge repair with porcine dermal patch augmentation for massive rotator cuff tear: surgical technique and preliminary results. Clin Orthop Surg. 2014;6(3):329–35.
37. Gupta AK, Hug K, Boggess B, Gavigan M, Toth AP. Massive or 2-tendon rotator cuff tears in active patients with minimal glenohumeral arthritis: clinical and radiographic outcomes of reconstruction using dermal tissue matrix xenograft. Am J Sports Med. 2013;41(4):872–9.
38. Flury M, Rickenbacher D, Jung C, Schneider MM, Endell D, Audigé L. Porcine dermis patch augmentation of supraspinatus tendon repairs: a pilot study assessing tendon integrity and shoulder function 2 years after arthroscopic repair in patients aged 60 years or older. Arthroscopy. 2018;34(1):24–37.
39. Bond JL, Dopirak RM, Higgins J, Burns J, Snyder SJ. Arthroscopic replacement of massive, irreparable rotator cuff tears using a GraftJacket allograft: technique and preliminary results. Arthroscopy. 2008;24(4):403–9.

40. Wong I, Burns J, Snyder S. Arthroscopic GraftJacket repair of rotator cuff tears. J Shoulder Elbow Surg. 2010;19(2):104–9.
41. Barber FA, Burns JP, Deutsch A, Labbé MR, Litchfield RB. A prospective, randomized evaluation of acellular human dermal matrix augmentation for arthroscopic rotator cuff repair. Arthroscopy. 2012;28(1):8–15.
42. Gupta AK, Hug K, Berkoff DJ, Boggess BR, Gavigan M, Malley PC, Toth AP. Dermal tissue allograft for the repair of massive irreparable rotator cuff tears. Am J Sports Med. 2012;40(1):141–7.
43. Gilot GJ, Alvarez-Pinzon AM, Barcksdale L, Westerdahl D, Krill M, Peck E. Outcome of large to massive rotator cuff tears repaired with and without extracellular matrix augmentation: a prospective comparative study. Arthroscopy. 2015;31(8):1459–65.
44. Agrawal V. Healing rates for challenging rotator cuff tears utilizing an acellular human dermal reinforcement graft. Int J Shoulder Surg. 2012;6(2):36–44.
45. Encalada-Diaz I, Cole BJ, MacGillivray JD, Ruiz-Suarez M, Kercher JS, Friel NA, Valero-Gonzalez F. Rotator cuff repair augmentation using a novel polycarbonate polyurethane patch: preliminary results at 12 months' follow-up. J Shoulder Elbow Surg. 2011;20(5):788–94.
46. Proctor CS. Long-term successful arthroscopic repair of large and massive rotator cuff tears with a functional and degradable reinforcement device. J Shoulder Elbow Surg. 2014;23(10):1508–13.
47. Lenart BA, Martens KA, Kearns KA, Gillespie RJ, Zoga AC, Williams GR. Treatment of massive and recurrent rotator cuff tears augmented with a poly-l-lactide graft, a preliminary study. J Shoulder Elbow Surg. 2015;24(6):915–21.
48. Audenaert E, Van Nuffel J, Schepens A, Verhelst M, Verdonk R. Reconstruction of massive rotator cuff lesions with a synthetic interposition graft: a prospective study of 41 patients. Knee Surg Sports Traumatol Arthrosc. 2006;14(4):360–4.
49. Nada AN, Debnath UK, Robinson DA, Jordan C. Treatment of massive rotator-cuff tears with a polyester ligament (Dacron) augmentation: clinical outcome. J Bone Joint Surg Br. 2010;92(10):1397–402.
50. Petrie MJ, Ismaiel AH. Treatment of massive rotator-cuff tears with a polyester ligament (LARS) patch. Acta Orthop Belg. 2013;79(6):620–5.
51. Ciampi P, Scotti C, Nonis A, Vitali M, Di Serio C, Peretti GM, Fraschini G. The benefit of synthetic versus biological patch augmentation in the repair of posterosuperior massive rotator cuff tears: a 3-year follow-up study. Am J Sports Med. 2014;42(5):1169–75.
52. Zhao S, Zhao J, Dong S, Huangfu X, Li B, Yang H, Cui W. Biological augmentation of rotator cuff repair using bFGF-loaded electrospun poly (lactide-co-glycolide) fibrous membranes. Int J Nanomedicine. 2014;9:2373–85.
53. Yokoya S, Mochizuki Y, Natsu K, Omae H, Nagata Y, Ochi M. Rotator cuff regeneration using a bioabsorbable material with bone marrow–derived mesenchymal stem cells in a rabbit model. Am J Sports Med. 2012;40(6):1259–68.
54. Hee CK, Dines JS, Dines DM, Roden CM, Wisner-Lynch LA, Turner AS, Santoni BG. Augmentation of a rotator cuff suture repair using rhPDGF-BB and a type I bovine collagen matrix in an ovine model. Am J Sports Med. 2011;39(8):1630–40.
55. Chainani A, Little D. Current status of tissue-engineered scaffolds for rotator cuff repair. Tech Orthop. 2016;31(2):91–7.
56. Zheng MH, Chen J, Kirilak Y, Willers C, Xu J, Wood D. Porcine small intestine submucosa (SIS) is not an acellular collagenous matrix and contains porcine DNA: possible implications in human implantation. J Biomed Mater Res B Appl Biomater. 2005;73(1):61–7.
57. Gilbert TW, Freund JM, Badylak SF. Quantification of DNA in biologic scaffold materials. J Surg Res. 2009;152(1):135–9.

Imaging of Repaired Rotator Cuff

8

Ricardo Sampaio, Carlos Abel Ribeiro,
and Nuno Sampaio Gomes

8.1 Introduction

Imaging of the repaired rotator cuff can be accomplished with several imaging modalities, including radiography, ultrasonography (US), computed tomography (CT), and magnetic resonance (MR) imaging. Usually, the imaging algorithm follows the same algorithm used locally for imaging the nonrepaired cuff. Radiography remains a cornerstone of shoulder imaging and a first-line imaging modality, whether on post-op shoulders or not. In expert hands, US is an excellent modality to image the cuff tendons, including the repaired cuff. For small tears, US may even be better than MR imaging. Magnetic resonance imaging allows the most comprehensive assessment of the shoulder, including its surrounding muscles and bone structures. In particular cases, CT or MR with intraarticular contrast (CT or MR arthrography) may be used to characterize the repaired rotator cuff.

R. Sampaio (✉)
Radiology Department, Hospital Lusíadas Porto, Porto, Portugal

C. Abel Ribeiro
Radiology Department, Hospital da Luz Arrábida, Porto, Portugal

N. Sampaio Gomes
Orthopedics Department, Hospital da Luz Arrábida, Porto, Portugal

Imaging of the postoperative shoulder usually begins with radiography of the shoulder. The purpose of the radiograph is not only to detect obvious osseous complications but also to identify the type of surgical procedure performed and to assess the amount of metallic implantation that may be present [1]. From there, usually one proceeds to MR imaging or ultrasound of the shoulder.

8.1.1 MR Imaging Protocol

Different MR imaging sequences are used for imaging follow-up of the repaired rotator cuff. There are two basic types of MR sequences: (1) short time-of-echo (TE) sequences that depict anatomy using fat as the natural contrast (T1-weighted and proton density (PD) weighted sequences) and (2) long TE sequences that depict fluid in white shades (T2-weighted and STIR sequences). On long TE (T2-weighted) sequences in musculoskeletal imaging, fat is usually suppressed (FatSat) and, therefore, shown in dark shades on images (T2-weighted FatSat sequences). After intravenous paramagnetic contrast medium administration (gadolinium compounds), the T1-weighted sequences are also acquired with fat suppression (T1-weighted FatSat Gad sequences). STIR sequences naturally suppress fat. There are many variants of these basic sequences; some of them designed for

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_8

patients with metal implants (Figs. 8.1, 8.2, 8.3, 8.4, 8.5, 8.6, 8.7, 8.8 and 8.9).

Metal of any kind (such as in screws, anchors, sutures, wires, buttons, tacks or plates) distorts the magnetic field and results in susceptibility artifacts on MR images. Different MR sequences in different magnetic field stregths have different sensitivities to magnetic susceptibility artifacts. As rules of thumb, the susceptibility artifact increases as one goes from T1-weighted to T2-weighted sequences, fat saturation blooms the artifact, as the use of gradient-echo T2-weighted

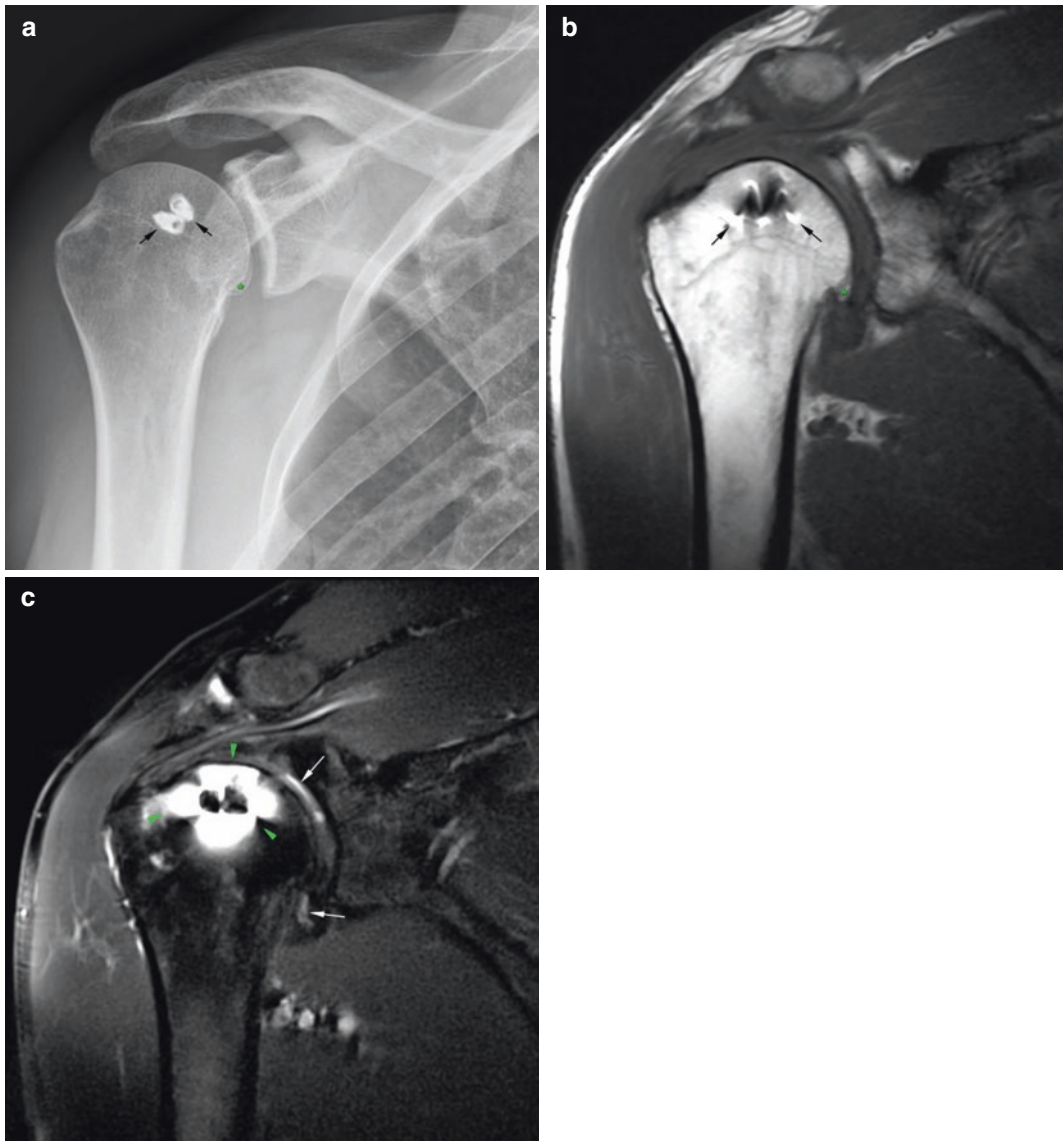


Fig. 8.1 (**a**–**c**) Metal susceptibility artifact. (**a**) Frontal radiograph of the shoulder. Two metal suture anchors are seen in the greater tuberosity. (**b**) T1-weighted coronal MR image. The suture anchors metallic artifact distorts the anatomy (black arrows). The anchors themselves are not really visualized. Subcutaneous fat is bright as is the fatty bone marrow in the marginal osteophyte in the humeral head (green asterisk). (**c**) T2-weighted FatSat coronal MR image. The artifact from the suture anchors blooms, with a halo of artifactual blurring of the image around the anchors (arrow heads). Tiny amounts of fluid in the joint cavity are seen as bright spots (white arrows). Subcutaneous and bone marrow fat is suppressed and depicted in dark shades on the image

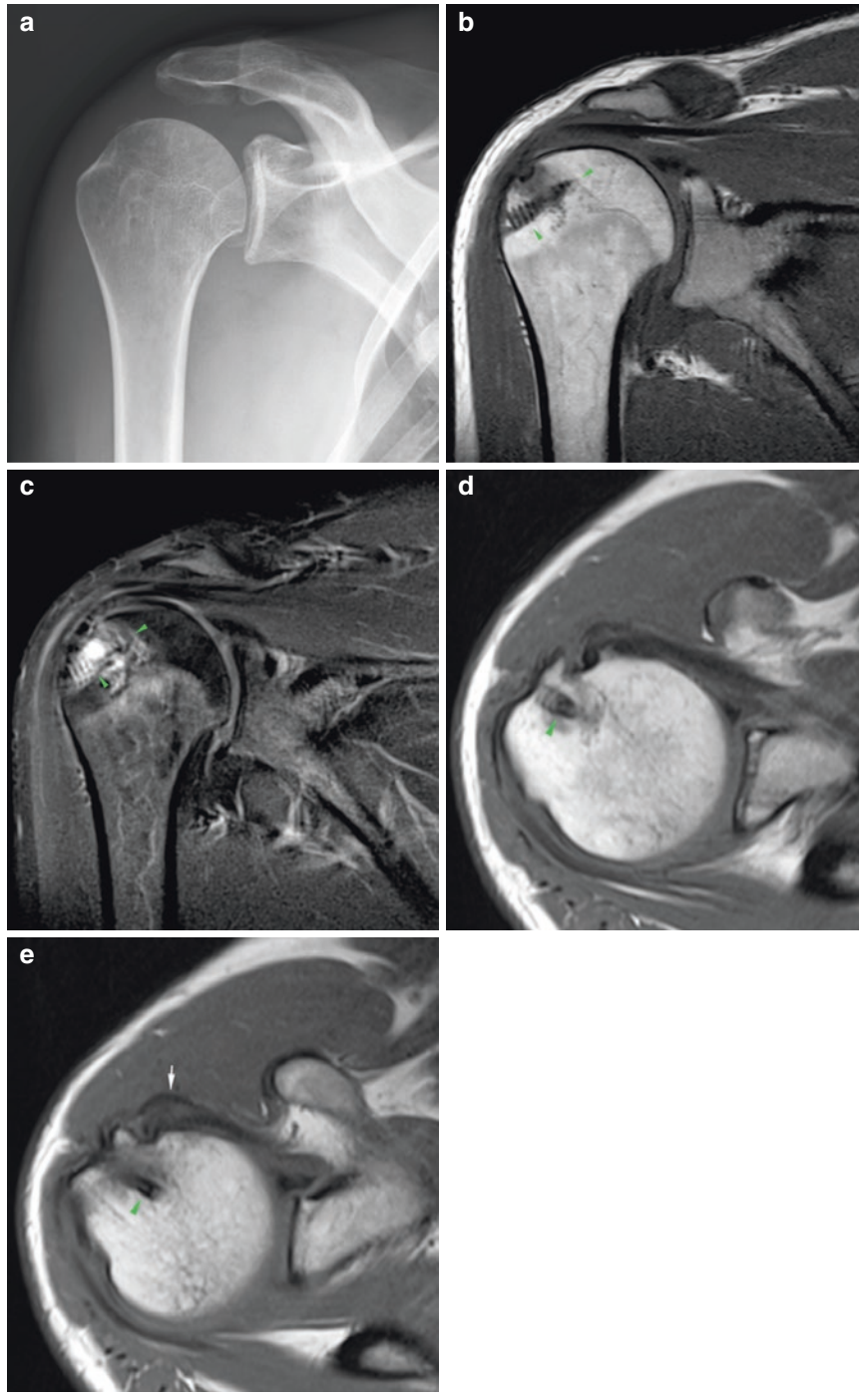


Fig. 8.2 (**a**–**e**) Nonmetal suture anchor artifact. (**a**) Frontal radiograph of the shoulder post supraspinatus reinsertion. The bioabsorbable suture anchor is not appreciated on the radiograph. There are small calcifications in the acromioumeral space. (**b**) T1-weighted coronal MR image. The nonmetallic anchor is readily seen with no significant artifact (arrowheads). Normal osteointegration of the anchor. (**c**) T2-weighted FatSat coronal MR image. There is some artifact caused by the anchor (arrowheads) but not enough to prevent the supraspinatus tendon from being evaluated. The supraspinatus tendon looks normal. (**d**, **e**) T1-weighted axial MR images. The anchor is defined in two adjacent planes (arrowheads). Subscapularis tendinosis (white arrow): the tendon is thickened at its insertion and maybe delaminated due to subluxation of the long head of the biceps tendon

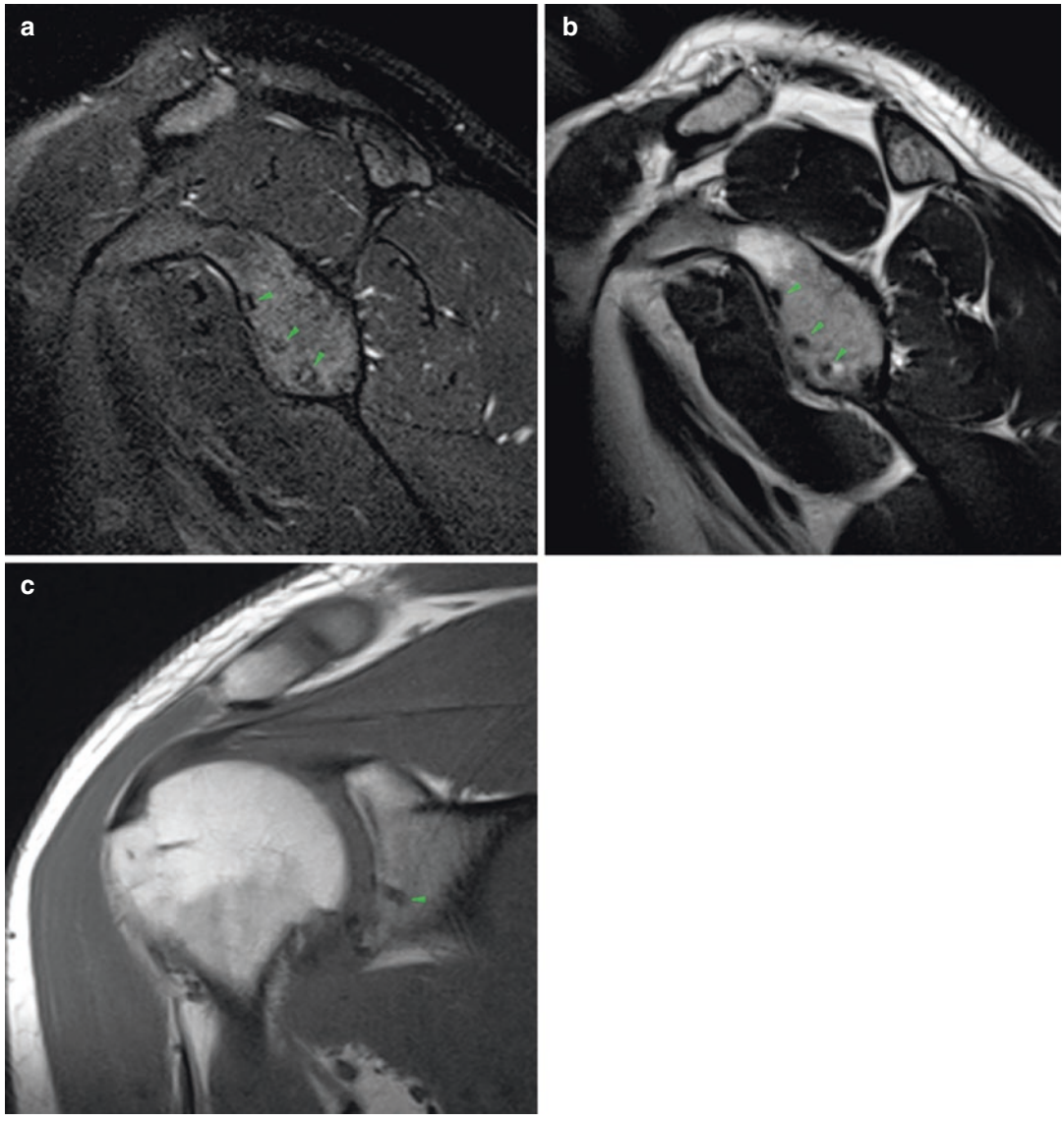


Fig. 8.3 (**a**–**c**) Nonmetal suture anchor post arthroscopic repair of a Bankart lesion with bioabsorbable suture anchors. (**a**) T2-weighted FatSat sagittal MR image. The suture tracks are readily appreciated with normal appearance (arrowheads). (**b**) T2-weighted MR image, nonfat saturated. Small dots along the anterior margin of the glenoid represent the osteointegrated bioabsorbable suture anchors (arrowheads). (**a**) T1-weighted coronal MR image, same patient. One of the suture anchors is seen normally osteointegrated (arrowhead). The anatomy is otherwise normal

(T2∗-weighted) sequences does (Figs. 8.1, 8.6, and 8.7), and the greater the magnetic field strenght the greater de artifact. On postoperative MR images, one should be aware of the possibility of metal artifact, so not to misconstrue artifact for pathology. Metal sources in bone and soft tissues after open or arthroscopic rotator cuff repair can be not only screws, anchors, and sutures, but also microscopic metal particles from the surgical instruments themselves, such as guides, probes, forceps, and cutting tools (Figs. 8.6 and 8.7).

Metal alloys always result in susceptibility artifacts, although not the same amount of artifact is seen in the same sequence for different implant compositions. The degree of artifact in the presence of metal implants is related to the

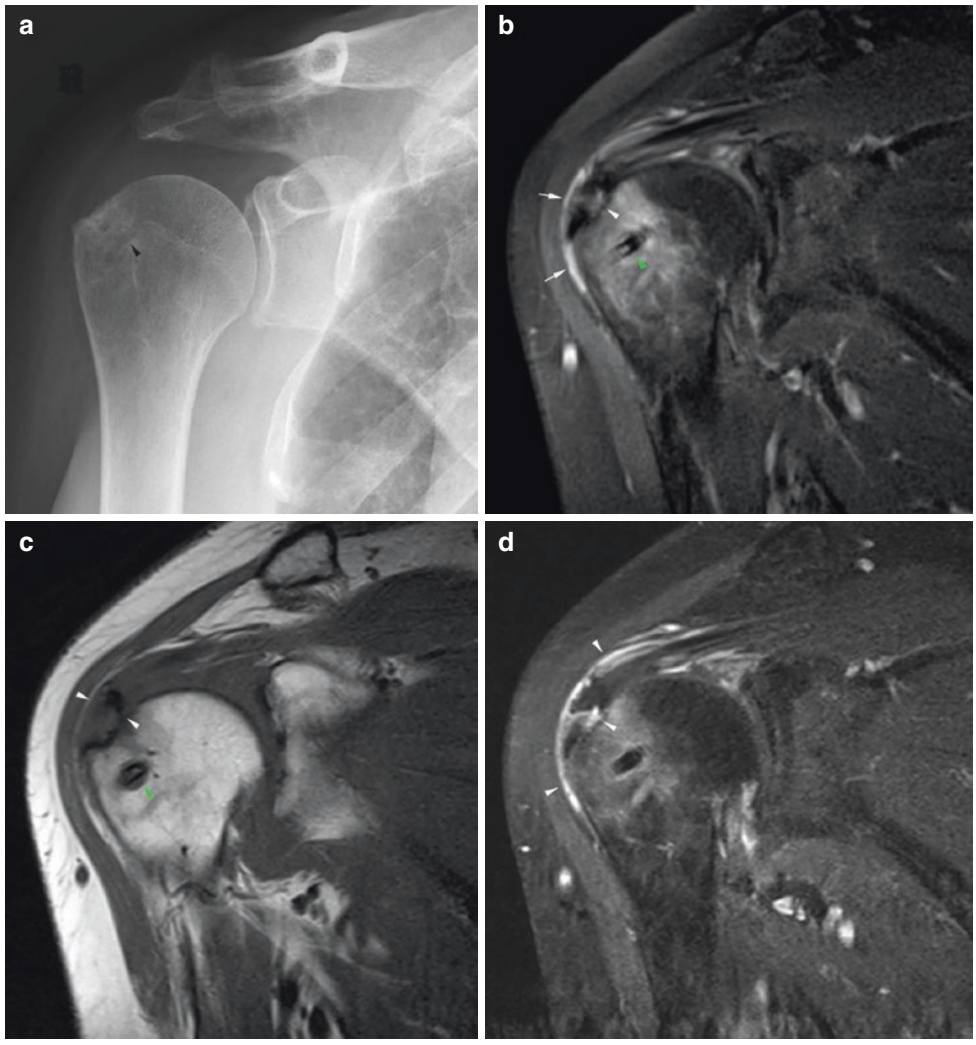


Fig. 8.4 (**a**–**h**) Supraspinatus tendon repair follow-up. (**a**) Frontal radiograph of the shoulder post supraspinatus reinsertion. The bioabsorbable suture anchor track is barely appreciated (arrowhead). (**b**) T2-weighted FatSat coronal MR image. The suture anchor is visible with mild osteolysis and edema of the surrounding bone marrow, normal for several weeks to months after surgery. There is no dislocation of the anchor (green arrowhead). The supraspinatus tendon is thickened, and its signal is very heterogeneous, also normal in the postoperative period. There is a small focus of fluid signal intensity in the articular side of the tendon footprint (white arrowhead), nonspecific (could be granulation tissue, a small residual communication or a small type 1 retear of the tendon). Bursitis-like signal intensity is seen in the subacromial bursal space, a normal finding for many months after surgery. (**c**) The corresponding T1-weighted coronal image. (**d**) T1-weighted FatSat Gad coronal image. This is a T1-weighted image with fat saturation and intravenous gadolinium. Inflammatory and granulation tissue takes up contrast (enhances). There is granulation tissue in the small communication in the supraspinatus tendon footprint (arrowhead). (**e**) T2-weighted FatSat coronal MR image, 10 months latter from image B. In the follow-up 10 months later, some loss of thickness and fissuring are seen in the supraspinatus tendon with a small bursal-side partial type 2 tear (arrowhead). (**f**) T2-weighted FatSat coronal MR image, 17 months later from image B. In the follow-up 17 months later, the small bursal-side partial type 2 tear (arrowhead) is better appreciated. This tear is not necessarily functionally relevant. The supraspinatus tendon is thinner than on the first postoperative MR study. (**g**) T2-weighted FatSat sagittal MR image, 17-months follow-up. The supraspinatus tendon is very heterogeneous, with several small transtendinous fissures (arrowheads), but for the most part reattached to the greater tuberosity. (**h**) Final frontal radiograph of the shoulder 17-months follow-up. New hyperostosis has developed in the supraspinatus footprint, as compared to the initial radiograph (**a**) (arrowhead)

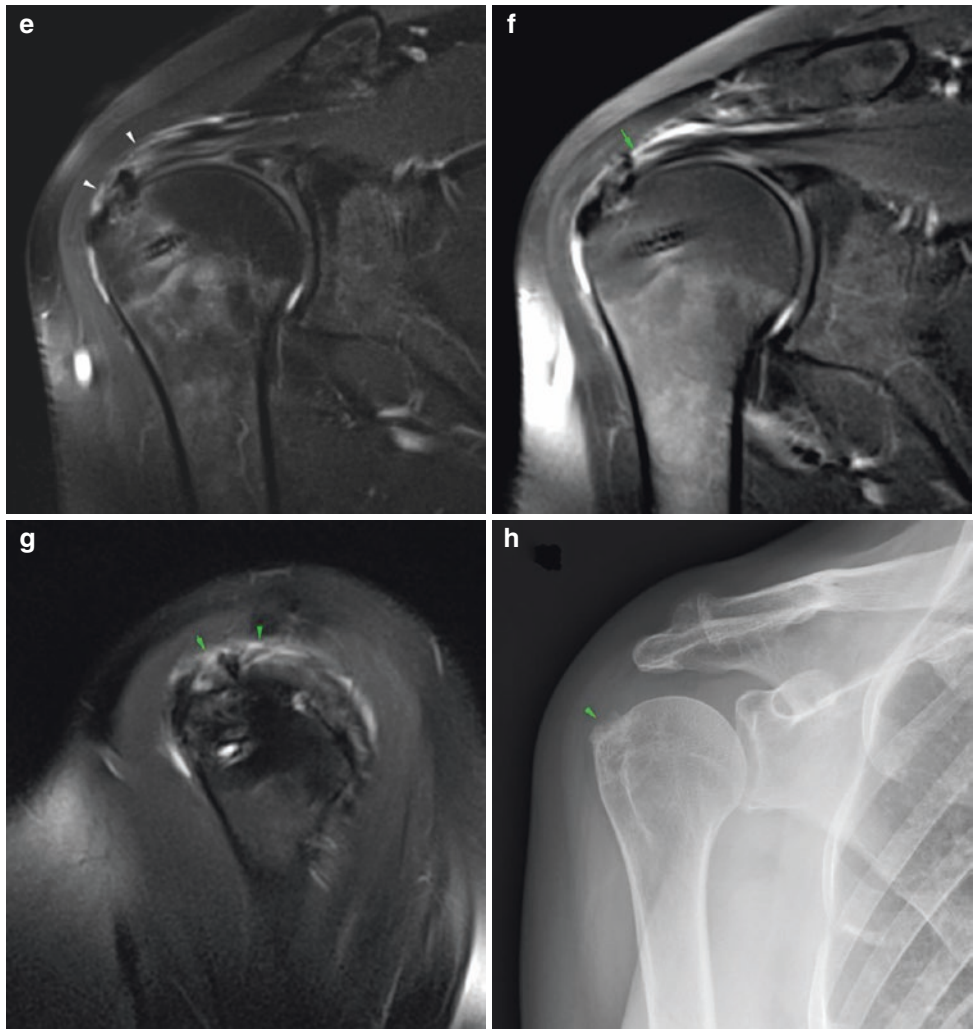


Fig. 8.4 (continued)

quantity of metal, the particular elements of the metal alloy, the geometry of the implant, the MR acquisition parameters, and the field strength of the MR equipment. Metallic artifact is greater with ferromagnetic implants (steel, for instance) than with nonferromagnetic metal implants, such as titanium. Susceptibility to artifact increases in a linear fashion with magnetic field strength. Therefore, the artifact will be greater at 3.0 Tesla than at 1.5 Tesla and will be the least in low-field permanent magnets (0.3 Tesla, for instance), such as in dedicated extremity MR installations.

Nonmetal implants (such as bioabsorbable interference screws or suture anchors) do not cause susceptibility artifacts per se, but susceptibility artifacts may still be present due to the microscopic metal particles from the surgical instrumentation (Figs. 8.2 and 8.3).

8.1.2 Normal Cuff Repair

Surgery intended to repair the shoulder rotator cuff will change the anatomy in several different ways. The tendon may be reinserted into the bone; the tendon may be cut and inserted somewhere else as in bicipital tenodesis or in cuff tendon transposition, or may be cut off

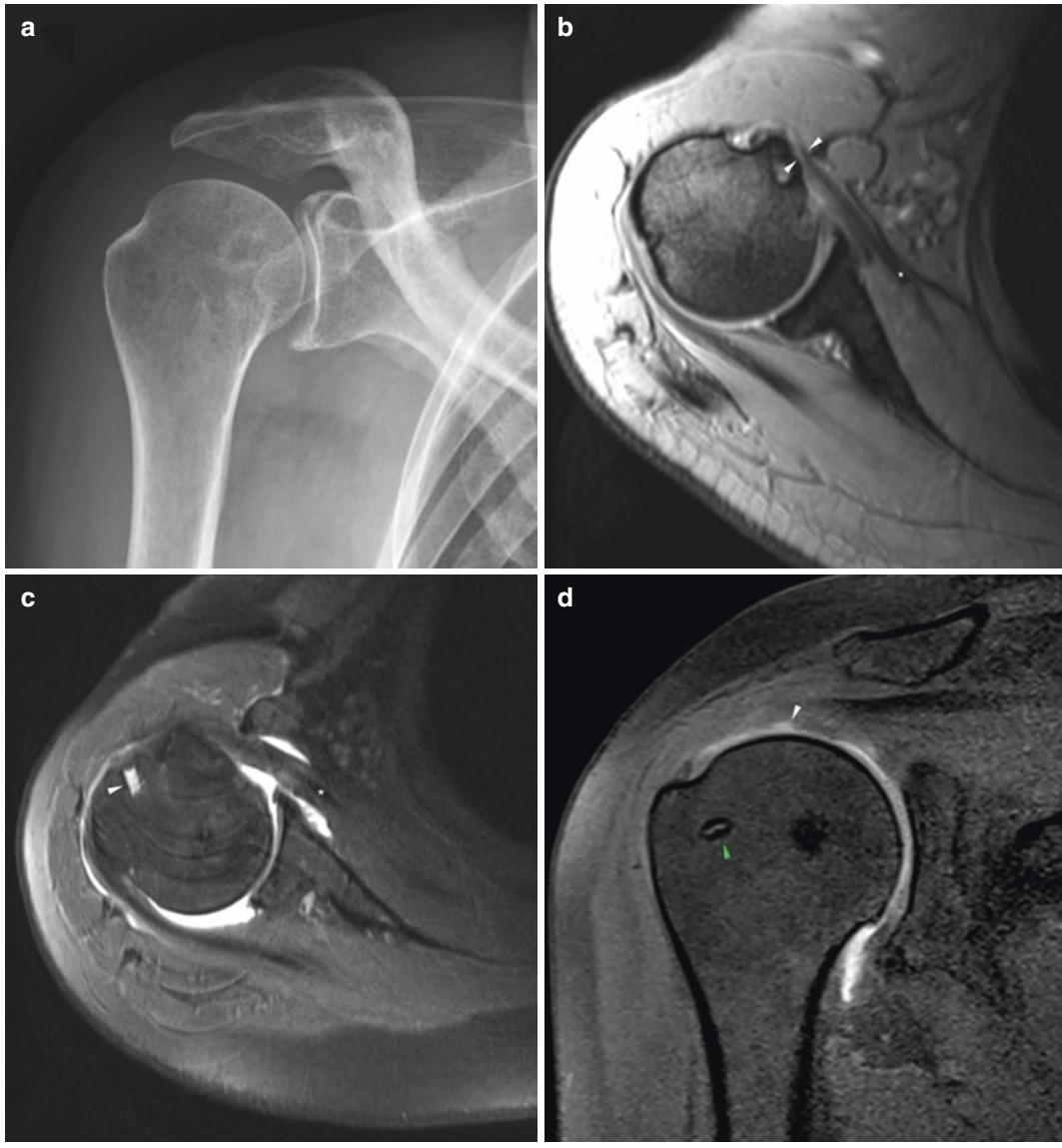


Fig. 8.5 (**a**–**d**) Subscapularis tendon repair follow-up. (**a**) Frontal radiograph of the shoulder post subscapularis tendon reinsertion. There are no metal implants. (**b**) Pre-op T2∗-weighted axial MR image. There is a full-thickness subscapularis tendon tear (arrowheads) with moderate myotendinous retraction (asterisk). (**b**) Postoperative PD-weighted axial MR image with intraarticular contrast (MR arthrography). The subscapularis tendon has been successfully reattached and the subscapularis myotendinous junction has now a normal position (asterisk). One of the nonmetallic suture anchors is also seen (arrowhead). (**c**) Postoperative T1-weighted FatSat coronal MR image with intraarticular contrast (MR arthrography), same day. There are small partial bursal-sided tears in the undersurface of the supraspinatus tendon (white arrowhead), apparently new as compared to the preoperative MR study. One of the suture anchors is seen normally osteointegrated

altogether as in bicipital tenotomy; the bony environment to the cuff may be modified as in the acromioplasty procedure; enthesophytes or osteophytes may be excised, and muscle may the cut open and then sutured, as in transdeltoid open surgery [2, 3].

Arthroscopic surgery results in different changes from the ones seen after open surgery,

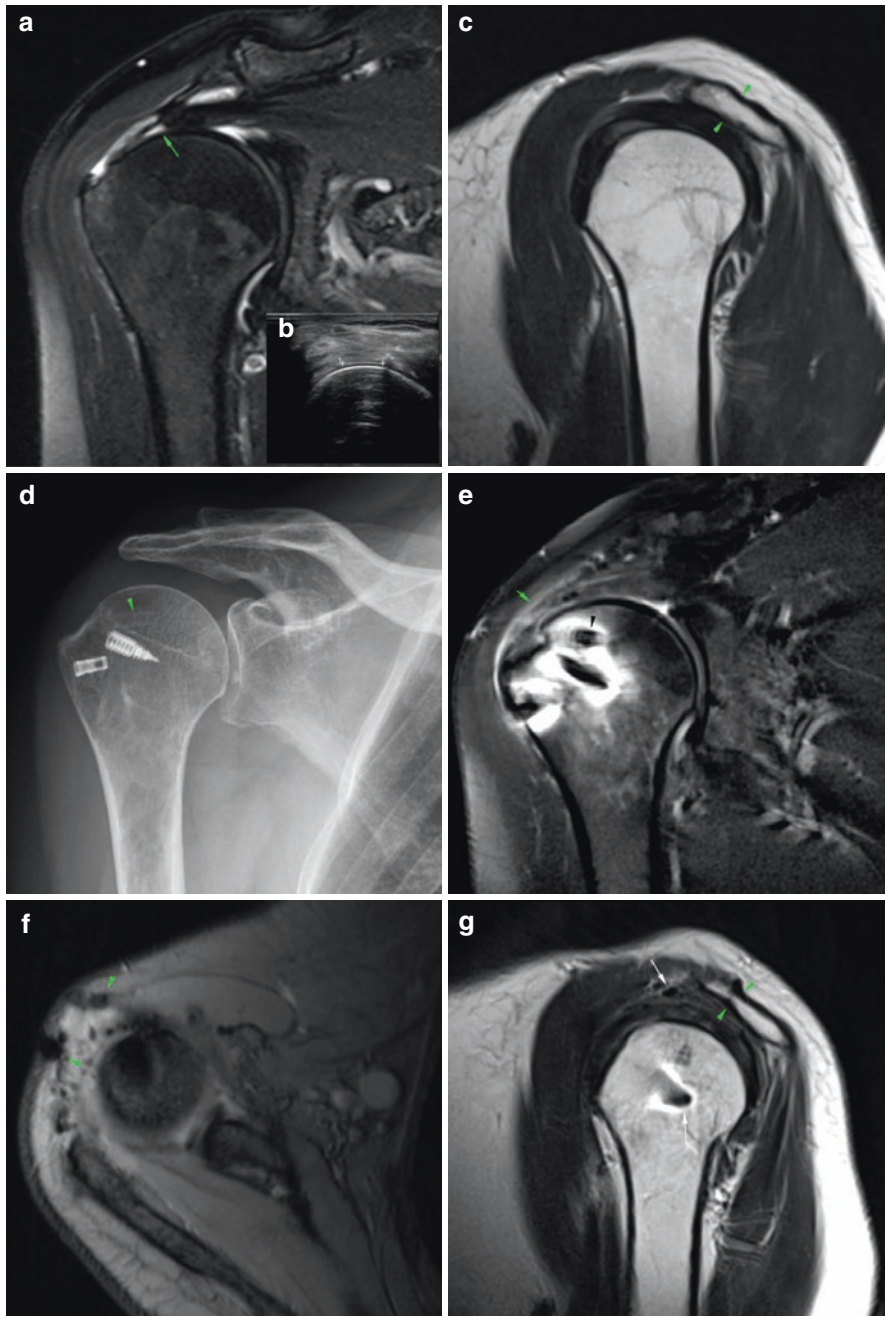


Fig. 8.6 (**a**–**g**) Cuff repair follow-up, acromioplasty. (**a**) Preoperative T2-weighted FatSat coronal MR image. There is a full-thickness tear of the supraspinatus tendon with moderate retraction of the tendon (Patte stage 2). The tendon tear was also demonstrated on ultrasound (Insert **b**). (**c**) Preoperative T2-weighted sagittal MR image. Normal thickness of the acromion. (**d**) Frontal radiograph of the shoulder post supraspinatus tendon repair. There are two metal suture anchors and a nonmetal anchor (arrowhead). (**e**) Postoperative T2-weighted FatSat coronal MR image. The supraspinatus tendon has been partially reattached to the humerus. The tendon is not as thick as it would be if fully reattached. Black arrowhead: nonmetal suture anchor. (**f**) Postoperative T2∗-weighted axial MR image. Open surgery with susceptibility artifact foci in the soft tissues in the deltoid area (arrowheads) from surgical instrumentation (no metal is seen in the soft tissues on the same day radiograph—Fig. **d**). (**g**) Preoperative T2-weighted sagittal MR image. Acromioplasty has been performed. The undersurface of the acromion has been shaved and the thickness of the acromion is reduced as compared to the preoperative images

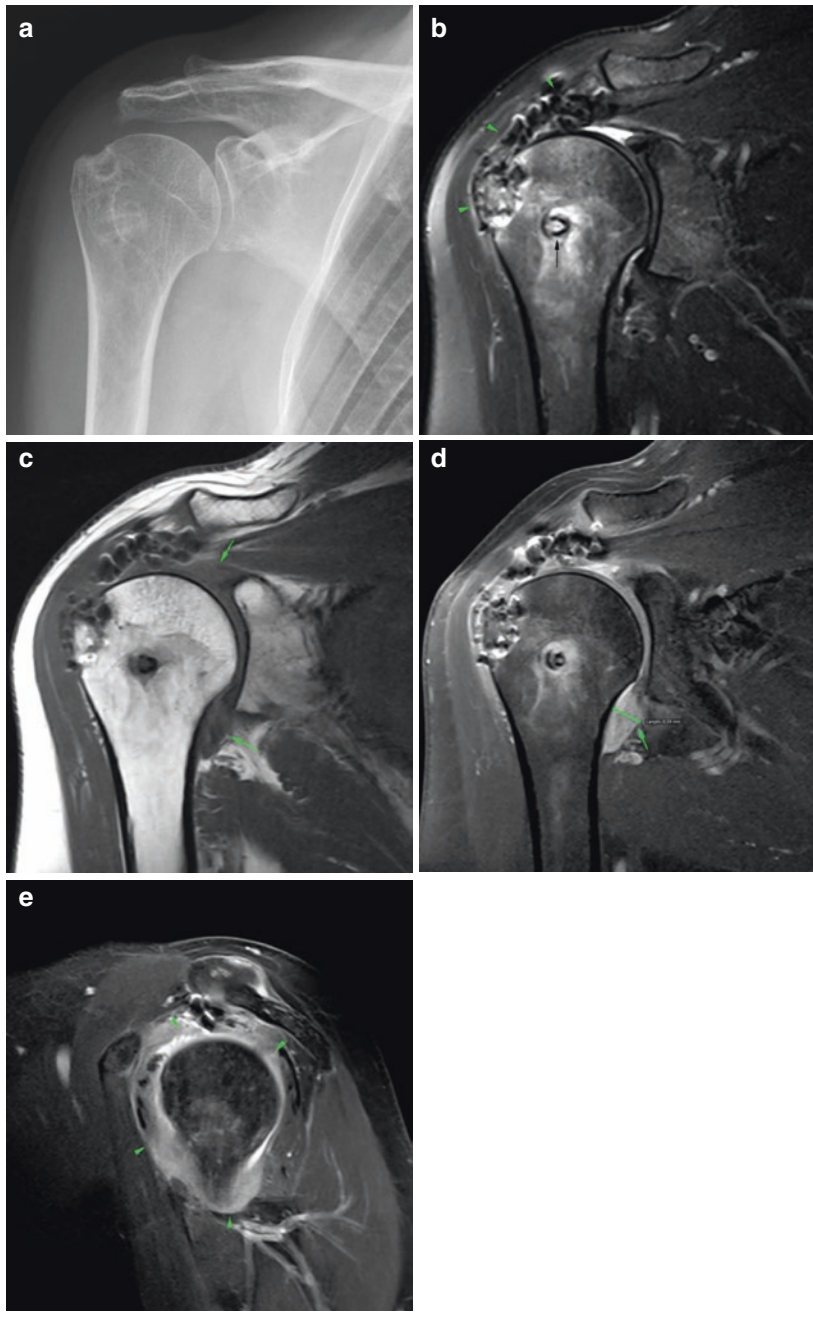


Fig. 8.7 (**a**–**e**) Cuff repair follow-up. Adhesive capsulitis. A Frontal radiograph of the shoulder post cuff repair. No metal implants are seen. A postoperative trough is present in the supraspinatus footprint. There has been acromioplasty. (**b**) Postoperative T2-weighted FatSat coronal MR image. There is extensive susceptibility artifact in the supraspinatus tendon despite the fact that no metal particles are seen in the tendon on the same day radiograph (**a**). The supraspinatus tendon appears to be reattached to the tuberosity. The tip of a suture anchor is visible (black arrow). (**c**) Postoperative T1-weighted coronal MR image. The capsular axillary recess appears thickened, and there is also apparent capsulosinovial thickening or fluid in the superior recess (arrows). Note the susceptibility artifact in the supraspinatus tendon. (**d**) Postoperative T1-weighted FatSat coronal MR image with intravenous gadolinium. (**e**) Postoperative T1-weighted FatSat sagittal MR image with intravenous gadolinium. There is capsular enhancement and thickening and no fluid in the joint, consistent with adhesive capsulitis. The capsular fibroblastic reaction is circumferential although most prominent in the axillary and subcoracoid recesses of the glenohumeral joint

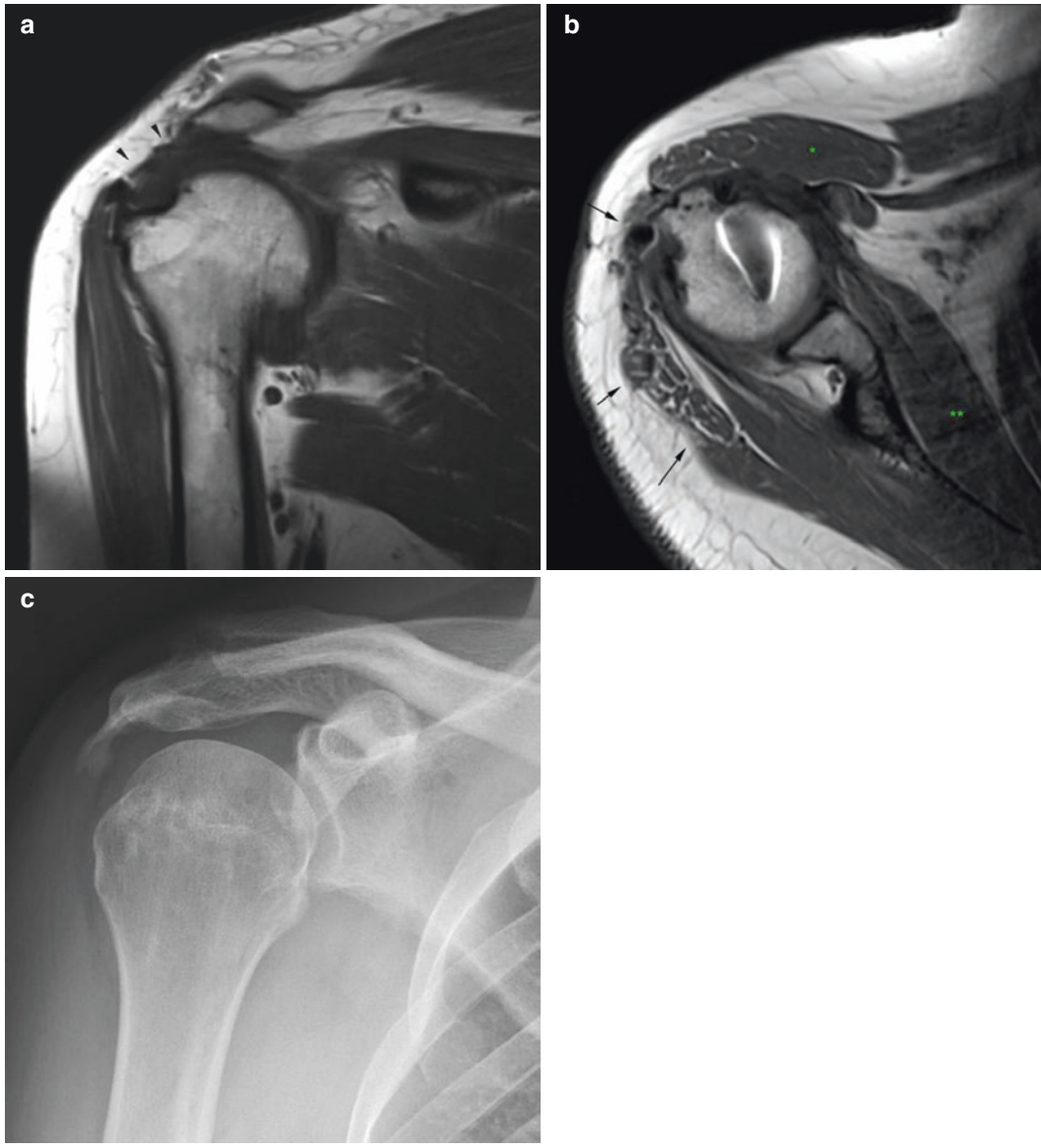


Fig. 8.8 (**a**–**c**) Cuff repair follow-up. Chronic deltoid dehiscence. (**a**) Postoperative T1-weighted coronal MR image. (**b**) Postoperative T1-weighted axial MR image. There has been detachment of the deltoid muscle from the acromion (arrows) with atrophy and fatty degeneration of the acromial belly of the deltoid muscle (arrows). For comparison, see the clavicular portion of the deltoid (asterisk) or the subscapularis muscle belly (double asterisk). (**c**) Different patient, a frontal radiograph of the shoulder shows heterotopic ossification of the deltoid muscle origin in the acromion, post open surgical repair of the rotator cuff

and certain complications are particular to the type of surgery performed, such as the deltoid dehiscence that may occur after open surgery. All of these changes are amenable to imaging investigation, particularly, MR imaging (Figs. 8.4, 8.5, 8.6, 8.7, 8.8, and 8.9).

Surgery may fail for any number of reasons, or the patient's symptoms may fail to resolve. Up to 25% of patients remain symptomatic after surgical repair of the rotator cuff [4]. Imaging is usually warranted to study these patients and when that happens, MR is usually called upon to image

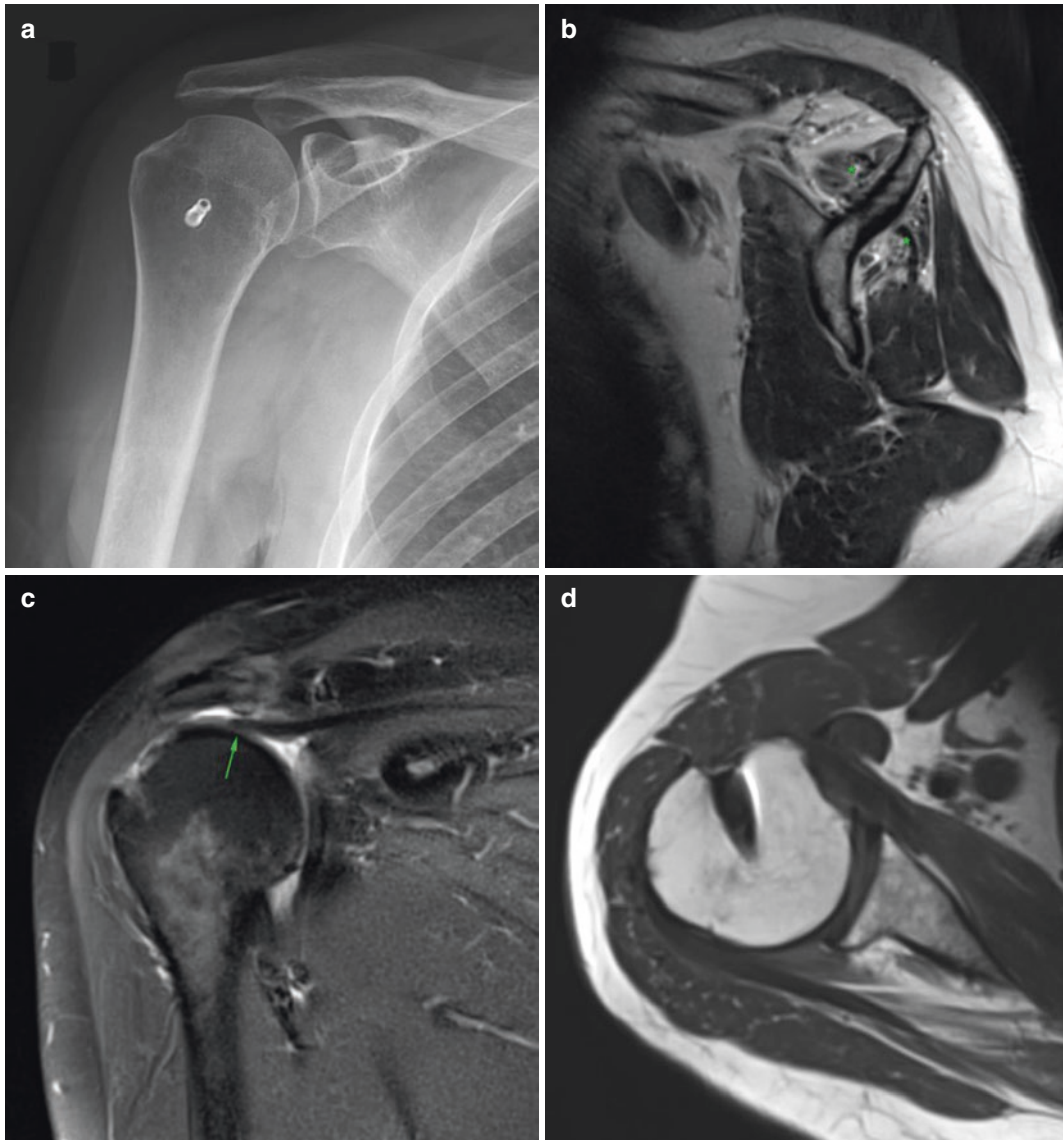


Fig. 8.9 (**a**–**d**) Cuff repair follow-up. Long head of the biceps tenodesis. (**a**) Frontal radiograph of the shoulder shows a metallic anchor or interference screw in the humeral metaphysis. (**b**) T2-weighted sagittal MR image at the level of the spine of the scapula. There is massive atrophy and fatty degeneration of the supraspinatus and infraspinatus muscles (Thomazeau Stage 3), most likely beyond repair (asterisks). (**c**) Postoperative T2-weighted FatSat coronal MR image. Complete tear of the supraspinatus tendon with massive retraction of the tendon (arrow). The repair of the supraspinatus tendon was either not attempted or failed. (**d**) T1-weighted axial MR image. The screw was used to fix the long head of the biceps tendon into the biceps tendon gutter (biceps tenodesis). There is no apparent adverse reaction to the implant

the shoulder [4–6]. On MR imaging, the repaired tendon will show heterogeneous signal intensity, from postsurgical changes, healing response, and preexisting tendinopathy (Fig. 8.4). These signal changes are most conspicuous on short TE MR images (such as T1-weighted and PD-weighted images). They are less conspicuous on long TE images (such as on T2-weighted images). The tendon will usually be thicker than a normal tendon, but not always. If the quality of the pre-

op tendon edges is poor, or the tendon is asymmetrically retracted, it might be only partially reattached to the bone, and the final result will be a tendon less thick or equal in thickness to a normal tendon (Fig. 8.6). A repaired tendon thinner or equal in thickness to a normal tendon has been either partially reinserted or, if initially fully reinserted, has suffered a partial retear. The repaired tendon, if fully reattached, is always thicker than a normal tendon and usually thicker than the preoperative tendon (Figs. 8.4 and 8.5). The thickness of the tendon is a much more reliable indicator of pathology than signal changes on MR imaging.

On long TE MR images (T2-weigheted images), fluid within the tendon usually indicates tendinous communication, either remnant communication or retear of the tendon. After surgical repair of a cuff tear, the tear is not always completely sealed, and small transtendinous communications may persist. They are easily depicted on MR arthrography and US and may be less conspicuous on conventional MR studies. However, they may not be clinically relevant. Small residual defects or retears (<1 cm) of the rotator cuff are not necessarily associated with clinical symptoms. Partial and even full-thickness tears are common in asymptomatic patients after surgery. Likewise, subacromial bursitis-like MR abnormalities are almost always seen after rotator cuff repair, even in patients without residual complaints (Fig. 8.4). Fluid in the subacromial bursa may persist for several years after rotator cuff repair and appear to be clinically irrelevant [1, 4].

In practice, tendon repairs are often thinner than a normal tendon. Interestingly, cuff repair footprint coverage may improve by the end of the first postoperative year. The appearance of the repaired rotator cuff on MR imaging shows considerable variability in the first postoperative year and does not correlate to outcome. The tendon appearance often becomes more like normal tendon by 1 year after surgery [7]. Improvement in signal changes, when it occurs, generally develops between 3 and 12 months.

To avoid magnetic susceptibility artifacts at MR imaging, T2-weighted inversion recovery (STIR) imaging may be used instead of fat saturation, and fast spin-echo sequences may be used instead of conventional spin-echo sequences or gradient-echo sequences. The technical parameters of the MR sequences may have to be modified to minimize metallic artifact or some sequences may have to be substituted for others. There are specially designed sequences for metal MR imaging, but they are not available in all MR scanners.

MR arthrography may also be used, instead of conventional MR imaging (Fig. 8.5). The advantages of MR arthrography include better definition of the rotator cuff articular side, more accurate assessment of capsule volume and delineation of labral ligamentous structures, and aid in the differentiation of partial- and full-thickness tears [6]. However, contrast accumulation within the subacromial bursa (a characteristic sign for a full-thickness tear in nonoperated shoulders) has substantially less impact in patients after surgery because the rotator cuff does not need to be watertight to be functional [4].

Ultrasound is able to look at the entire distal cuff, provided that the patient is able to freely move the shoulder into the positions required to access the supraspinatus and the subscapularis tendons (extension, adduction and external rotation). Patients unable to move the shoulder, for instance patients with adhesive capsulitis, are not good candidates for US of the repaired cuff. Otherwise, US can show suture dehiscence, tendon avulsion, or retear of the repaired tendon as well as adverse reactions to intratendinous sutures [8, 9]. A subdeltoid bursal effusion representing residual arthroscopic fluid and/or hematoma may persist for several months after surgery.

Ultrasound is usually requested to detect suture failure or retear of a repaired cuff tendon. Although the repaired tendons demonstrate altered echogenicity and thickness, in comparison to a normal tendon, US can accurately predict the localization and extent of a cuff tear, with a comparable accuracy to MR imaging [10]. Ultrasound can be used to serially monitor the postoperative cuff changes after surgical repair [9]. Ultrasound is cheaper than MR imaging and

more patient friendly and a lot faster, but counterintuitively may be less available, due to the long learning curve it takes the operator to become proficient in shoulder US.

Ultrasound is not so good as an imaging method to look at intraosseous adverse reactions related to suture anchors, tacks or screws, or bone vascular changes. A combination of radiography and US may overcome the limitations of each of the methods, but glenohumeral chondral and labral pathology, bone marrow, complex ganglion cysts, and muscle pathology remain best studied with MR imaging. If a physician with similar experience with MR and US is available, the preference for either one of these tests should not be based on the accuracy of the imaging modality, but rather on patient tolerance, cost, and the importance of detecting non-rotator cuff pathology, such as labral, capsular, or bone lesions.

Mild superior subluxation of the humeral head may persist after open or arthroscopic cuff repair, maybe due to capsular tightening, scarring, cuff atrophy, or bursectomy, and can be appreciated on radiographs of the shoulder. Changes to the subacromial bursa and to the acromion and lateral end of the clavicle are easier to evaluate with MR imaging. Postacromioplasty acromial changes are surprisingly hard to see on radiographs (Fig. 8.6).

8.1.3 Failed Cuff Repair and Complications

A number of events may cause a repaired cuff to fail. The tendon may suffer a repeat tear (for instance, during over vigorous physical therapy), or the anchor or suture may fail, and the tendon may detach from the bone. Despite anatomically correct repair, pain may persist, or a second lesion may develop. Complications may arise at the coracoacromial arch or the deltoid incision. General adverse events such as adhesive capsulitis or infection may arise. When pain or disability occurs after rotator cuff surgery, postoperative imaging is frequently performed, usually MR imaging [1].

8.1.4 Retear of the Repaired Cuff

Retear of a previously repaired rotator cuff tendon is relatively common and does not necessarily compromise functional outcome or patient satisfaction [7]. Two patterns of retear have been described: in type 1 retear, the tendon fails at the tendon-bone interface, and in type 2 retear, the cuff failure happens medially, about the myotendinous junction 1.5–2 cm medial to the tendon insertion, with the remnant cuff still attached to the humeral tuberosity [11]. Type 1 tears tend to occur earlier in the postoperative period and may result from failure of the tendon-to-bone fixation. Type 2 tears tend to occur later in the postoperative period and may be related to progression of tendon disease, impaired vascularity, or increased tension after reattachment of the tendon (Fig. 8.4).

Magnetic resonance imaging will allow for the detection of the cuff retear as well as its staging and classification. On MR imaging, a recurrent tendon tear will be seen as a fluid-filled defect within or across the tendon on long TE (T2-weighted) images. Secondary signs of tendon retear include tendon retraction and loss of tendon thickness. It may be difficult to differentiate tendinosis, granulation tissue, and healing response from a partial retear although the healing response will evolve over time, with the signal intensity expected to decrease on long TE MR images for about one year [7]. Full thickness retears are easier to demonstrate on MR images. The presence of a recurrent tear is not necessarily symptomatic, but the size of the tear may be correlated with the development of symptoms [1] (Figs. 8.4, 8.5, and 8.9).

Ultrasound will depict type 1 retears, but may fail to show type 2 retears, particularly if the patient is unable to fully extend, adduct, and externally rotate the shoulder.

8.1.5 Suprascapular, Axillary Nerve Palsy

Muscle edema when the muscle is anchored in the humerus usually reflects acute muscle denervation or myositis. Muscle denervation may

occur for a variety of reasons, the two most common being neuritis (as in the Parsonage-Turner syndrome) and nerve entrapment or injury. Nerve entrapment and nerve injury may occur during and after surgery. Long TE MR fat-suppressed images (T2-weighted FatSat or STIR images) are very sensitive to muscle edema, not only detecting acute signs of denervation (days or weeks after the nerve lesion) but also mapping the involved muscles, so the involved nerve is identified [12]. In the postoperative shoulder, localized edema or soft-tissue reaction may cause enough pressure on a nerve (suprascapular nerve, for instance) to result in acute denervation in that particular nerve territory. Intraoperative axillary nerve injury will result in teres minor and deltoid muscle denervation, and MR is well suited to diagnose the muscle edema associated with such instances of muscle denervation.

Chronic muscle denervation will result in muscle atrophy and fat infiltration, and both phenomena are also very well depicted on MR images (Fig. 8.9).

8.1.6 Postoperative Bursitis, Synovitis, Adhesive Capsulitis, Scarring

Postoperative bursitis is a relatively frequent finding in patients with persistent or recurrent pain. Inflammation of the bursae is best depicted on T1-weighted fat-saturated MR images with gadolinium. Care should be taken not to overcall subacromial bursitis, as bursal signal abnormalities are very common in asymptomatic patients. These bursitis-like changes may persist for a long time after surgery (up to 4–5 years) [4].

Adhesive capsulitis may be a cause of persistent symptoms after cuff repair. This complication usually occurs shortly after cuff repair. Adhesive capsulitis may not be recognized on standard MR images as the capsulosinovial thickening may be subtle and the inflamed synovium may be difficult to tell from joint fluid on fat-suppressed long TE MR images, aside from the postoperative changes in the rotator cuff interval and in the subcoracoid fat triangle. MR T1-weighted imaging with intravenous contrast medium (gadolinium) is better able to show thickening and enhancement of the joint capsule and synovial membrane along with the tightness of the joint cavity that characterizes adhesive capsulitis (Fig. 8.7).

Patients after rotator cuff repair may have persistent pain and dysfunction caused by exuberant postoperative reaction and scar formation. This reaction may involve the subacromial area, the joint capsule, the tendons, and adjacent soft tissues. Scar tissue is made up of collagen and show up on MR images as bulky masses or bands of low-signal intensity tissue on short and long TE images, reflecting dense fibrotic adhesions and even calcified or ossified scar tissue. Myositis ossificans and heterotopic ossification may occur in muscle incisions and tracts, particularly in the deltoid muscle (Fig. 8.8c).

Tendon dystrophic calcifications are a secondary manifestation of tendon degeneration. Calcifications are easier to detect on gradient-echo ($T2^*$) MR images because these imagens are more sensitive to magnetic susceptibility. Unfortunately, this increased sensitivity to calcifications of $T2^*$ images also make them more prone to artifacts due to metal implants and debris. On ultrasound, sutures and microcalcifications can be difficult to tell apart. On radiography, only calcifications that are not superimposed on the humeral head are readily visualized.

8.1.7 Infection, Loose Bodies

A significant fluid collection about the joint without focal tendon discontinuity suggests the presence of complication, including infection, perisutural inflammation, synovitis associated with loose bodies, or loose surgical implants. Intravenous paramagnetic contrast medium administration greatly enhances the ability of MR to show inflammatory tissue and fluid collections. Bone marrow edema and periarticular and subchondral erosions are findings better depicted on MR imaging. Imaging (usually ultrasound) can also serve as a guide to direct a needle to an optimal site for aspiration of joint fluid, should the possibility of a septic joint be considered.

8.1.8 Deltoid Dehiscence

Open cuff repair usually involves an incision through the acromial part of the deltoid muscle. This incision is closed on surgery completion, and the muscle is reattached to the acromion, but the suture may fail, or the muscle may tear along the incision or acromial reattachment and result in deltoid dehiscence. MR imaging can show a fluid-filled defect within the muscle with or without retraction of the muscle. If the dehiscence is chronic, muscle atrophy and fatty degeneration may be present [1] (Fig. 8.8).

8.1.9 Implant (Suture Anchors, Tacks, Screws) Complications

Surgical implants used to repair the cuff and facilitate the soft tissue-to-bone repair, such as suture anchors and screws, can be either made up of metal alloys or nonmetal bioabsorbable materials (bioabsorbable polymers and calcium ceramics) [13]. They can fail or complicate by several mechanisms, such as loosening or migration, foreign body reaction, cyst formation or infection, impingement upon surrounding bone or soft tissue structures, fracture of the host bone, loose intraarticular foreign bodies, synovitis, or cartilage damage [13–16]. Among these complications, osteolysis and cyst formation, enlargement of drill holes, loosening and anchor pull-out, and suture break away from the anchor are probably the most important in rotator cuff repair.

Nonmetal bioabsorbable implants allow for decreased artifact during MR imaging, although still some artifacts may be seen (Figs. 8.2 and 8.3). Resorption of the anchor is the desirable course for bioabsorbable implants, which have different degradation profiles and are, over time, replaced by bone or calcified fibrosis within the screw track. However, exposure to the debris of absorbable anchors in the joint can cause synovitis and pain. MR imaging is able to demonstrate the implant location and its surrounding environment as well potential complications related to the implant, such as cyst formation, track enlargement, implant displacement, or infection [1, 15, 17].

Metallic implants are easily spotted with radiography and the amount of susceptibility artifact they create may render them invisible on MR images. Worse, the artifact creates a blind zone around the implant that may prevent complications in the vicinity of the implant from being visualized (Fig. 8.1). Metallic implants create their own risks in the MR environment, such as overheating of the implant and implant-induced internal burns. Certain metal compositions (steel, for instance) are deflected by the magnetic field and may be displaced by simply placing the patient inside the magnet of the MR machine.

New not bioabsorbable implants (such as polyetheretherketone or PEEK) are radiolucent and, therefore, not visible on radiographs. Similar to metallic implants, they do not resorb, but similar to bioabsorbable implants they are visible on MR imaging, and their susceptibility artifact profile renders them accessible to MR imaging interrogation.

8.1.10 Other Causes of Pain After Cuff Repair

In patients with failed rotator cuff surgery, it is possible that other pathologies are generating pain and may be the primary source of symptoms, rather than the rotator cuff [18].

Long head of biceps tendon subluxation is common in patients with chronic rotator cuff tear. If not repaired at the time of surgery, biceps tendon subluxation may persist after surgery. Biceps tendon subluxation may also develop de novo in a patient already submitted to cuff repair. Superior labrum anterior posterior (SLAP) lesions may also account for the patient's symptoms.

US is ideally suited to diagnose biceps tendon subluxation due to its dynamic nature. If the tendon spontaneously reduces in the neutral position of the shoulder, MR imaging may miss the biceps tendon subluxation.

8.2 Conclusion

The imaging assessment of patients who have had rotator cuff surgery includes a radiographic examination followed by either MR imaging (sometimes MR arthrography) when a comprehensive shoulder evaluation is desired, or ultrasound when targeted to identifying rotator cuff recurrent tears. Knowledge of common surgical procedures and expected postoperative findings on various imaging techniques and potential complications are important for the imaging evaluation of the shoulder after rotator cuff repair.

References

1. Thakkar RS, Thakkar SC, Srikumaran U, McFarland EG, Fayad LM. Complications of rotator cuff surgery—the role of post-operative imaging in patient care. Br J Radiol. 2014;87:20130630–12.
2. MD LWB, MD CW, MD CP, MD TF. Postoperative shoulder magnetic resonance imaging. Magn Reson Imaging Clin N Am. 2012;20:313–25.
3. Beltran LS, Bencardino JT, Steinbach LS. Postoperative MRI of the shoulder. J Magn Reson Imaging. 2014;40:1280–97.
4. Zanetti M, Jost B, Hodler J, Gerber C. MR imaging after rotator cuff repair: full-thickness defects and bursitis-like subacromial abnormalities in asymptomatic subjects. Skeletal Radiol. 2000;29:314–9.
5. Gusmer PB, Potter HG, Donovan WD, O'Brien SJ. MR imaging of the shoulder after rotator cuff repair. Am J Roentgenol. 1997;168:559–63.
6. Mohana-Borges AVR, Chung CB, Resnick D. MR imaging and MR arthrography of the postoperative shoulder: spectrum of normal and abnormal findings. Radiographics. 2004;24:69–85.
7. Crim J, Burks R, Manaster BJ, Hanrahan C, Hung M, Greis P. Temporal evolution of MRI findings after arthroscopic rotator cuff repair. AJR Am J Roentgenol. 2010;195:1361–6.
8. de Beer J. Journal AKSO, 2009 Sonographic evaluation of the arthroscopically repaired rotator cuff. scielo.org.za
9. Yoo HJ, Choi J-Y, Hong SH, Kang Y, Park J, Kim SH, Kang HS. Assessment of the postoperative appearance of the rotator cuff tendon using serial sonography after arthroscopic repair of a rotator cuff tear. J Ultrasound Med. 2015;34:1183–90.
10. Lee SC, Williams D, Endo Y. The repaired rotator cuff: mri and ultrasound evaluation. Curr Rev Musculoskelet Med. 2018;11:1–10.
11. Bedeir YH, Schumaier AP, Abu-Sheasha G, Grawe BM. Type 2 retear after arthroscopic single-row, double-row and suture bridge rotator cuff repair: a systematic review. Eur J Orthop Surg Traumatol. 2018;19:1–10.
12. Kim S-J, Hong SH, Jun WS, Choi J-Y, Myung JS, Jacobson JA, Lee JW, Choi J-A, Kang HS. MR imaging mapping of skeletal muscle denervation in entrapment and compressive neuropathies. Radiographics. 2011;31:319–32.
13. Dhawan A, Ghodadra N, Karas V, Salata MJ, Cole BJ. Complications of bioabsorbable suture anchors in the shoulder. Am J Sports Med. 2012;40:1424–30.
14. Willemot L, Elfadalli R, Jaspars KC, Ahw MH, Peeters J, Jansen N, Declerq G, Verborgt O. Radiological and clinical outcome of arthroscopic labral repair with all-suture anchors. Acta Orthop Belg. 2016;82:174–8.
15. Kim SH, Oh JH, Lee O-S, Lee H-R, Hargens AR. Postoperative imaging of bioabsorbable anchors in rotator cuff repair. Am J Sports Med. 2014;42:552–7.
16. Take Y, Yoneda M, Hayashida K, Nakagawa S, Mizuno N. Enlargement of drill holes after use of a biodegradable suture anchor: quantitative study on consecutive postoperative radiographs. Arthroscopy. 2008;24:251–7.
17. Stein T, Mehling AP, Reck C, Buckup J, Efe T, Hoffmann R, Jäger A, Welsch F. MRI assessment of the structural labrum integrity after Bankart repair using knotless bio-anchors. Knee Surg Sports Traumatol Arthrosc. 2011;19:1771–9.
18. Pierce JL, Nacey NC, Jones S, Rierson D, Etier B, Brockmeier S, Anderson MW. Postoperative shoulder imaging: rotator cuff, labrum, and biceps tendon. Radiographics. 2016;36:1648–71.

Part II

Controversies in Massive Rotator Cuff Tears

9 Clinical Outcome vs. Structural Integrity: What Really Matters?

John Bampis, John Swan, and Achilleas Boutsiadis

9.1 Introduction

Symptomatic rotator cuff (RC) tears are a very common musculoskeletal disorder that can cause severe disability, weakness, and persistent pain [1]. Therefore, surgical repair, with the transition from open to mini-open and to fully arthroscopic approaches, became one of the most increasingly commonly performed procedures [2–4]. In recent decades, authors have reported an overall increase of 238% [3], and importantly, a significant shift towards arthroscopic procedures with a 600% increase [2].

However, the repair of massive rotator cuff tears (MRCT) remains a surgical challenge with unpredictable outcomes due to the substantial fatty infiltration, tendon retraction, and tendon tissue degeneration [5–8]. In 2004 Galatz et al. reported 94% re-tear rates after arthroscopic repair of large and massive rotator cuff tears. Additionally, the authors found that the initial pain relief and the ability to perform daily activities were not constant. Their results deteriorated significantly in 2 years postoperatively [9]. Since then, rotator cuff surgery has evolved, where techniques have progressed from single-row [SR] suture anchor repairs, to double-row [DR], and finally to transosseous-equivalent speed bridge [SB] techniques. Additionally, several alternative surgical methods have been proposed to solve the challenging problem of massive repairable tears such as patch augmented repair, interval slide techniques, and the use of biological factors [10].

However, despite advances in surgical technique, are we really performing "a better operation" for the patient? The purpose of this chapter is to examine the evidence whether these newer methods have in fact improved the healing rate for MRCT and whether this correlates with clinical and functional outcomes. Finally, we will discuss whether or not a healed MRCT is actually a prognostic factor of a good final result.

9.2 Factors Affecting Rotator Cuff Integrity After Rotator Cuff Repair

Currently, reported re-tear rates following rotator cuff repair (RCR) vary between 13% and 68% [11]. Many authors have attempted to analyze the influence of several different factors on the final anatomical results [10, 12–15]. These

J. Bampis
2nd Orthopaedic Department, 401 Military Hospital of Athens, Athens, Greece

J. Swan
Department of Orthopaedic Surgery, Centre Osteoarticulaire des Cèdres, Grenoble, France

A. Boutsiadis (✉)
2nd Orthopaedic Department, 401 Military Hospital of Athens, Athens, Greece

Chalandri, Attiki, Greece

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_9

factors could be categorized into two groups: (a) patient-related nonmodifiable factors and (b) surgeon-related modifiable factors [10].

9.2.1 Patient-Related Nonmodifiable Factors Affecting Rotator Cuff Healing

These factors are critically important and the surgeon must carefully evaluate them before any decision-making.

9.2.1.1 Age

Increasing patient age has been associated with lower rates of tendon healing after RCR in multiple studies [10, 16, 17]. The authors reported that due to aging, there is a biological limitation at the repair site that appears to be the most important factor influencing tendon healing, even after maximizing repair biomechanical strength with a double-row construct.

However, the most recent studies have shown that age over 70 or 75 years old is not a contraindication to proceed with arthroscopic rotator cuff repair, even in cases of large or massive lesions [18, 19]. Probably, the detrimental effect of increasing age on tendon healing after rotator cuff repair may be due to other factors affecting tendon healing rather than the age itself. Therefore, age may be a surrogate for other anatomical factors like decreased bone mineral density, fatty infiltration, and tendon retraction, which correlate with impaired healing after rotator cuff repair [10].

9.2.1.2 Tear Size and Location

As previously mentioned, repaired large and massive tears are prone to increased rates of anatomical failures [9, 10, 12]. Massive rotator cuff tears represent approximately 20% of all RCTs that require surgery and account for 80% of the cases with postoperative structural failure [20]. However, in the international literature, the failure rates of MRCTs are reported to be between 17.6% and 94%. One reason for this large discrepancy could simply be the lack of a universal definition for a "massive rotator cuff tear." By using the term "massive rotator cuff tear," the surgeon should take into account not only a tear diameter ≥5 cm or a complete tear of two or more tendons, but also other factors such as tendon retraction, muscle atrophy, arthritis, and intraoperative tendon mobilization [21].

Once an MRCT is identified, it should be further classified into subgroups according to the location of the tear, as this helps to determine the likelihood for a successful repair. For this reason, either the classification of Gerber et al. [22] or Collin et al. [23] can be used. Recent studies have shown that postero-superior tears have the highest postoperative failure rate followed by anteroposterior subtypes of MRCTs [20, 24, 25].

9.2.1.3 Fatty Infiltration and Atrophy

It is reported that Goutallier grade 2 or higher degrees of fatty infiltration are significantly associated with poorer healing after repair [10]. This is also supported by several studies [5, 12, 26, 27] where the fatty infiltration of both the supraspinatus and infraspinatus can predict the final tendon integrity. A new meta-analysis showed that an increase in supraspinatus or infraspinatus fatty infiltration by one grade increases the risk of re-tear by approximately 2.5 times [13].

However, we should comment that the current body of literature is sometimes confusing and the final multivariate analysis performed by some authors shows that fatty infiltration may not be a risk factor for re-tear [28], nor is a negative prognostic factor for bad functional outcome even in postoperative cases with intact tendon [29]. Furthermore, some authors propose that only decreased preoperative active external rotation rather than atrophy is a risk factor for postoperative re-tear in the postero-superior and anteroposterior tear groups [25]. One could conclude that the pathoanatomy of MRCTs is multifactorial.

9.2.1.4 Muscle-Tendon Unit Retraction and Tissue Quality

The Patte classification is usually used for the evaluation of the RC tendon retraction, and it assesses the degree of tendon retraction in the coronal plane on MRI. According to this classification, RCTs are divided into three groups: (1) Full-thickness tear

with little tendon retraction, (2) tendon retraction to the level of the humeral head, and (3) tendon retraction to the level of the glenoid [30]. Lädermann et al. have further defined an MRCT as requiring at least one of the two torn tendons to be retracted beyond the top of the humeral head [21].

Tendon retraction has been directly correlated with the tear size, tear chronicity, and muscle fatty infiltration [10, 31]. Furthermore, it has been reported to be an independent prognostic factor of re-tear [12, 32, 33].

9.2.1.5 Other Patient-Related Factors

Several other patient-related factors have been reported to affect rotator cuff tendon healing.

- *Smoking*
  Smoking can increase the risk for rotator cuff tears, can influence the size of the tear, and also compromise healing [10].
- *Hypercholesterolemia and Diabetes*
  Total cholesterol, triglycerides, and low-density lipoprotein cholesterol concentrations are increased in patients with rotator cuff tears and may also affect the healing rates [10]. Similarly the body fat (expressed as body mass index) [34] and the sustained hyperglycemia increase the possibility of anatomic failure of a repaired cuff [35].
- *Osteoporosis*
  In older patients and especially in women, the surgeon should account for not only diminished bone mineral density, but also possible vitamin D deficiency, which may have a negative effect on rotator cuff tendon healing following surgical repair [10].

9.2.2 Surgeon-Related Modifiable Factors Affecting Rotator Cuff Healing

9.2.2.1 Single-Row, Double-Row, or Suture Bridge Techniques

After understanding the importance of different technical factors like the orientation and the depth of anchors, the type and the strength of the knots used, the rotator cuff surgical trends have gradually passed from the classic open transosseous [TO] repairs to single-row [SR] suture anchor repairs, to double-row [DR] suture anchor repairs, and finally to transosseous-equivalent suture bridge [SB] techniques [10]. Double-row and speed bridge transosseous-equivalent techniques seem to produce a better biomechanical environment that theoretically could improve healing rates and superior functional outcomes.

The initial reports with SR techniques showed healing rates from 71% to 78%. However, large or massive tears with antero-superior, and principally, postero-superior lesions showed up to 50% tear recurrence [16, 36]. Furthermore, Barth et al. reported their anatomical and functional outcomes in 212 patients operated with the classic DR technique. The authors used the postoperative RC integrity classification as described by Sugaya and found an overall 13% recurrence rates. However, large and massive tears were also more prone to anatomical failure (25.5%), but with significantly better results compared to previous SR studies [12]. Duquin et al. after including 1252 patients from 23 studies concluded that the re-tear rates are significantly lower for DR techniques. In detail, they reported failures for DR of 7% for small (<1 cm), 8% for medium (1–3 cm), 25% for large (3–5 cm), and 43% for massive tears (>5 cm). Respectively, the values for the SR were 18% for small, 31% for medium, 44% for large, and 65% for massive tears [37]. The introduction of the SB technique had initially given encouraging results with Frank et al. [38] reporting 88% healing rates in 25 patients [100% success in massive tears (3/3 patients)]. Later Neyton et al. evaluated the arthroscopic SB repair for only small- to medium-sized supraspinatus tears and reported 10.3% recurrence (one case rupture of musculotendinous junction, 0.9%) [39]. Furthermore, Kim et al. stated that the overall anatomical failure rate for SB was 15%, and more specifically 12%, 21%, and 22% for medium, large, and massive tears, respectively [40].

Newer reports have shown equivalent outcomes between double-row and single-row repairs in small and medium lesions. However, in large and massive tears DR or TOE fixation may provide a functional advantage over SR [10, 25, 41].

9.2.2.2 Interval Slide Techniques for Massive Immobile Rotator Cuff Tears

In cases of massive, contracted, and immobile rotator cuff tears, Lo and Burkhart in 2004 proposed the interval slide technique. This can be either anterior, by incising the posterior part of the coracohumeral ligament (release of the supraspinatus from the rotator interval), or posterior by releasing the interval between the supraspinatus and infraspinatus tendons [42]. Initially, the authors reported no complications and significant improvement in the active range of motion and muscle strength [42]. However, despite the functional improvement, Berdusco et al. demonstrated that the healing rates did not exceed 45% [43]. Finally, according to Kim et al., the aggressive interval slide techniques with complete tendon repair have a 91% re-tear rate, which is not superior to partial repair [44].

9.2.2.3 Biologics (More Details Regarding the Biologics Are Provided in Chaps. 5, 6, and 7)

- *Patch Augmentation for Large and Massive Rotator Cuff Tears*

  Due to the significant rates of rotator cuff failures, especially in large and massive tears, several patch augmentation materials have been developed. Their purpose is either to enhance mechanically the strength of the repair and or provide a better biologic healing environment. Additionally, the mechanical support could be obtained either by augmentation of the repair either by interposition or bridging of the patch between tendon and bone.

  The current literature shows that regardless of the type of material (xenografts, allografts, and autografts), the interposing or bridging patches that span the defect from the retracted and stiff tendon stump to the greater tuberosity show superior healing rates than augmentation patches (75–90% successful rates vs. 50–60% respectively) [45–47]. This may be explained by the fact that in chronic massive tears, the relative tendon to muscle ratio of the musculotendinous unit is severely altered, resulting in significant loss of the muscle force [48].

- *Platelet Rich Plasma (PRP)*

  Despite the promising advantages and the results of animal model studies, great controversy exists regarding the effectiveness of the PRP when clinically applied during rotator cuff repair [10]. Interestingly, regarding their intraoperative clinical use during rotator cuff surgery, there is an abundance of review and meta-analysis articles, with 64 published to date. All authors concluded that the use of PRP during rotator cuff surgery results in no differences in the overall re-tear rates and in functional outcomes compared with patients treated without the application of any biological factor, except probably the small- and medium-sized tears [49, 50].

The most important prognostic factors affecting rotator cuff integrity after rotator cuff repair are shown in Table 9.1, as presented in the meta-analysis of Saccomanno et al. [14] and McElvany et al. [13].

Table 9.1 The most important prognostic factors affecting rotator cuff healing presented by Saccomanno et al. and McElvany et al.

	Saccomanno et al. [14]	McElvany et al. [13]
Re-tear risk factors	Older age	Older Age (per 10 years)
	Severe fatty infiltration	SSP fatty infiltration
	Larger tear size	ISP fatty infiltration
	Multiple tendons involved	Global fatty degenerative index
	Poor tendon quality	Tear size >3 cm
	Tendon delamination	Traditional double-row technique (no suture bridge)
	Single-row technique	Single-row technique
	ACJ procedures	Delay of active ROM and strengthening
	LHB procedures	
	Lower bone mineral density	
	Smaller AHD	
	Preoperative tendon length <15 mm	

9.3 What Is the Clinical Effect of a Rotator Cuff Re-tear in Cases with a Massive Lesion?

Despite surgical evolution and a better preoperative diagnostic approach, structural failures of MRCTs after arthroscopic repair remain high [10, 14]. This raises several important questions that will be discussed in turn:

(a) *What is the clinical effect of a rotator cuff re-tear?*
(b) *Is it worth repairing a massive rotator cuff tear when 20–90% of repairs will re-tear anyway?*
(c) *What are the prognostic factors that influence the outcome of MRCT repair most?*

9.3.1 What Is the Clinical Effect of a Rotator Cuff Re-tear?

Due to the heterogeneity in their study design, the different surgical techniques used, the type of the tears included, and the different imaging and functional evaluations utilized, the conclusions regarding the clinical effect of a postoperative rotator cuff re-tear are often confusing. For example, numerous studies have shown that both objective and subjective results are significantly superior in cases with confirmed healed tendon [12, 51–55]. However, others report that the presence of a postoperative tendon defect is not always correlated with an inferior outcome [53, 56]. However, evaluation of the published articles reveals that the functional evaluation of the patients is most often performed using the ASES, UCLA, and Constant scores. The ASES score is a validated outcome measure in patients with shoulder pathology, including rotator cuff tears. However, the minimal clinically important difference in the ASES score has to be in the range of 6.4–12.00 points, which is quite large [56]. The UCLA score was originally designed to measure outcomes after shoulder arthroplasty, and heavily weights pain relief, passive shoulder motion, and patient satisfaction. Therefore the use of the UCLA as an outcome measure is less reliable in terms of distinguishing the difference between patients with or without rotator cuff re-tear [56].

In the majority of published studies, the patients with a healed tendon had greater strength in forward flexion (by approximately 2.5 kg) and marginally improved strength in external rotation. Furthermore, the Constant score is largely influenced by strength, which accounts for worse Constant scores in cases with RC repair failure [56]. This is also in accordance with the findings of Kim et al., who showed poorer outcomes after RC re-tear in patients of younger age and lower education level and laborers [57]. Also, we know that worse clinical outcomes are found not in small and partial tears, but mainly in large recurrent defects (>4 cm) [58, 59].

From the aforementioned studies it is understood that re-tear may not significantly affect the final functional scores nor patient satisfaction. However, surgeons should rather focus on the postoperative strength of the operated shoulder, which is detrimentally affected by an anatomical failure, and correlate this with the occupational demands of the patient [7, 11].

9.3.2 Is It Worth Repairing a Massive Rotator Cuff Tear When 20–90% of Repairs will Re-tear Anyway? (Table 9.2)

As mentioned, the landmark article by Galatz et al. demonstrated a 94% re-tear rate of massive tear repairs at 1 year postoperatively in 18 patients, but patients had a high degree of satisfaction, most achieved overhead arm function, and the American Shoulder and Elbow Surgeons (ASES) score was >80 in two-thirds of the patients.

In 2000, Gerber et al. reported 44% failure rates (12/27 patients) 2 years postoperatively in patients with MRCTs treated with open repair. The authors also reported that patients with a re-tear showed significant improvement in the shoulder compared with the preoperative state, but they showed less improvement than those with a successful repair who had excellent results

Table 9.2 Table showing the clinical and structural outcomes after massive rotator cuff tear repair

	Patients	Surgical technique	Re-tear results	Follow up	Constant		VAS		ASES		UCLA		Strength	Subjective shoulder value (SSV)
Park et al. (2016) [24]	92 patients I: 42 subscapularis (SSC) Intact S: 22 ≤ ½ SSC tear L: 28 > ½ SSC tear	SR for SSC DR for SSP + ISS	Overall re-tear: 27% (25/92) I: 21% (9/42)[a] S: 18% (4/22)[a] L: 43% (12/28)[a]	24 months	L tears postoperative Intact 66.4 Failed 64.4[a]		L tears postoperative Intact 1.1 Failed 3.8p		L tears Postoperative Intact 90.6 Failed 58.5p					
Heuberer et al. (2016) [60]	68 patients 23 DB 22 PR 23 CR	SB/SR/CR	CR re-tear (29%) PR re-tear (53%)	45 months	Postoperative DB: 65.8 ± 14.7 PR: 67.5 ± 9.9 CR: 80.3 ± 8.9[b]									
Jung et al. (2017) [19]	64 patients	Open	Re-tear: 26% (12/46)	24–60 months	Preoperative 44 ± 18	Postoperative 76 ± 7	Preoperative 6.4 ± 2.2	Postoperative 2.3 ± 1.1	Preoperative 42 ± 16	Postoperative 84 ± 8			Elevation strength from 51% to 78% Ex. rotation strength from 59% to 81%	
Ohzono et al. [29]	55 patients	Arthroscopic rotator cuff repair	Overall re-tear: 25.6% (19/74)	30 ± 12 months			Rest 26.3 Motion 59.8 Night 52.3 Postoperatively				Preoperative 18.1 ± 4.4	Postoperative 29.8 ± 4.5	Elevation 66.7[c] Abduction 65.5[c] Int rotation 67.5[c] Ext rotation 74.3[c]	
Shimokobe et al. [25]	102 patients 59 antero-superior tear (AS) 21 postero-superior tear (PS) 22 antero-posterior tear (APE)	SB/SR/DR	Overall re-tear 25.5% (26/102) AS: 10/59 (16.9%) PS: 9 (42.9%)[d] APE: 7 (31.8%)	>12 months			Stat. significant improvement than preoperative				Preoperative AS: 18.2 ± 5 PS: 17 ± 4.7 APE: 15.9 ± 4	Postoperative 28.3 ± 7.2 29.4 ± 3.8 28.3 ± 5.9	Stat. significant improvement than preoperative	
Agout et al. (2018) [26]	511 patients 289 isolated SS tears 92 anterior extension (A) 94 posterior extension (P) 36 antero-posterior (AP)	Open (254) Arthroscopic (257) SR/DR/TRANSOS-SEOUS	SS: 40/289 (19%) A: 19/92 (25%) P: 24/94 (32%) AP: 8/36 (31%)	10 years	Preoperative A: 59.3 ± 16.2 P: 54 ± 14.9 AP: 51.2 ± 18.3	Postoperative 77.4 ± 14.1 78.4 ± 12.2 78.5 ± 8.6								Postoperative A: 84.5 ± 15.9 P: 83.8 ± 15.8 AP: 82.5 ± 15.3

Godenèche et al. [61]	73 patients 50 CR 23 PR	SB/SR	PR: 11/23 (47.8%) CR: 10/50 (20%)	29–55 months	Preoperative PR: 32.2 ± 9.1 CR: 30.8 ± 8.7	Postoperative PR: 75.3 ± 13.9 CR: 79.7 ± 12							Postoperative PR: 11/23 (47.8%) CR: 10/50 (20%)	Postoperative PR: 70.2 ± 23.7 CR: 79.2 ± 20.2
Kim et al. [62]	73 patients	Arthroscopic	Re-tear (R): 39/73 (53%) Healed (H): 34/73 (47%)	2 years			H: 1 ± 1 R: 2.1 ± 1.1		H: 90.8 ± 5.8 R: 76.6 ± 14.2		H: 31.0 ± 2.9 R: 24.9 ± 5.8			H: 90.2 ± 6.7 R: 77.4 ± 14.5
Ok et al. [20][e]	104 patients 34 antero-superior tear (A) 54 postero-superior tear (P) 16 antero-posterior tear (AP)	SB	Overall 45/104 (43.3%) A: 8/34 (23.5%) P: 28/54 (51.9%) AP: 9/16 (56.3%)	2 years	Preoperative 56.9 ± 15.4	Postoperative 74.3 ± 10.5	Preoperative 5.0 ± 2.73	Postoperative 2.4 ± 1.31	Preoperative 52.3 ± 11.4	Postoperative 79.4 ± 11.4	Preoperative 19.1 ± 8.31	Postoperative 27.1 ± 8.46		
Collin et al. [5]	130 patients	Open (64%) Arthroscopic (47%) SR/ Transosseous	Overall 34%	10 years	Preoperative 53.1 ± 15.9	Postperative 78.5 ± 11.3								Postoperative 83.4 ± 15.7
Henry et al. (2015) [63]	954 patients	Open/ Arthroscopic SR/DR	Re-tear 79%	33–52 months	Preoperative 49 Postoperative 74		Preoperative 5.9 Postoperative 1.7		Preoperative 40 Postoperative 84		Preoperative 13.5 Postoperative 29.6			
Lee et al. (2017) [64]	122 patients I: 45 intact SSC P: <1/3 tear of SSC C: >1/3 tear of SSC	SR for SSC SR/SB for SSP, ISP	Overall re-tear 37/122 (31.1%) I: 10/45 (22.2%) P: 7/35 (20%) C: 20/45 (47.6)p	39.5 months	Preoperative I: 48 ± 11.6 P: 45 ± 15.6 C: 44.3 ± 13	Postoperative 82 ± 11.8 82 ± 13.5 82.5 ± 12.8	Preoperative I: 4.6 ± 1.8 P: 4.9 ± 1.2 C: 5.1 ± 1.3	Postoperative 1.8 ± 1 1.9 ± 1.3 1.9 ± 1.2	Preoperative I: 45.5 ± 15 P: 43.5 ± 16 C: 42.7 ± 14	Postoperative 85.7 ± 12.5 81.7 ± 13.4 82.7 ± 14.7				
Papadopoulos et al. [58]	27 shoulders	Mini open SR	Overall re-tear 14/27 (51.9) <2 mm than initial 12/14 (small) >2 mm than initial 2/14 (large)	36–60 months	Postoperative Intact 85 Small 77.1 Large 67.5						Postoperative Intact 32.23 Small 29.8 Large 25.5			
Zumstein et al. [65]	23 patients	Open transosseous technique	Re-tear 13/23 (57%) 9.9 years Re-tear 10/27 (37%) 3.1 years[a]	9.9 years	Intact 81 Failed 64p								Abduction strength Intact 5.5 Failed 2.6p	

[a]not statistically significant
[b]Statistical significance between PR (partial repair)/DB (debridement)/CR (complete repair)
[c]Measured as the percentage of unaffected side
[d]Statistical significance higher than AS and APE
[e]Functional outcomes between groups were not significantly different

[22]. Eight years later the same authors presented the results of the same case series of 23 patients. Twenty-two of the 23 patients remained very satisfied or satisfied with the result. The subjective shoulder value and the Constant score were slightly improved compared with results from 2000 (82% vs. 80% and 85 vs. 83, respectively). However, the re-tear rate (57% vs. 44%) and the re-tear size were increased. Patients with a failed repair had a worse result than those with an intact reconstruction but still better than their preoperative conditions [65].

Papadopoulos et al., using mini-open repair, reported 52% failure rates 3 years postoperatively, but with good overall Constant and UCLA scores. Only large (>5 cm) defects were correlated with significantly worse clinical outcomes [58].

Collin et al. in a recent multicenter study with 10 years follow up of postero-superior MRCTs reported an overall prevalence of re-tears of 34%. Final Constant scores were significantly associated with cuff integrity, but even in cases with Sugaya type 5 re-tears, the mean score was 75, which is 20 points higher than the mean preoperative value. Also, their multivariate analysis revealed that the functional outcomes were only associated with preoperative infraspinatus retraction. Finally, the anterior extension of the tear and the involvement of the subscapularis did not have any negative effect on the Constant score or re-tear rates [5].

In antero-superior MRCTs, the percentage of structural failure also remains high. Kim et al. reported that after 2 years follow up there were 53% re-tears. Again the functional outcomes were significantly worse in patients with re-tears and even worse when the subscapularis was torn. Again, within both groups (healed or not), all scores and the range of motion improved significantly compared with preoperative values [62].

Rotator cuff surgery has also been studied in patients older than 75 years with MRCTs. Jung et al. reported on 64 patients with results of 26% re-tears but 80% patient satisfaction. The most important finding of this study was that beyond improvements in ASES and Constant scores, these elderly patients showed significant functional independence during their daily activities (Katz index and Functional independence measurement motor) [19].

Ozhono et al. reported that tendon integrity after repair of MRCTs is not the panacea for an excellent postoperative outcome. In their case series, they found that preoperative fatty degeneration of the infraspinatus and or subscapularis with Goutallier stage 2 or higher was significantly associated with worse outcome in patients who had intact tendons after arthroscopic repair [29].

Finally, Godenèche et al. in their study tried to answer the question whether we should reconstruct an MRCT even when it is partially repairable. The authors found 20% re-tears in patients with complete repair and approximately 50% in those with partial repair. However, the Constant score was only slightly higher for completely reparable tears (81.5) than for partially reparable tears (79). The authors reported that even two tendon repairs can produce "equivalent" improvement, patient satisfaction, and autonomy [61].

9.3.3 What Are the Prognostic Factors that Influence the Outcome of MRCT Repair Most?

Review of the literature focused on MRCT repair shows that possible prognostic factors associated with the outcomes of the procedure include the following:

- The recognition of *a reparable MRCT* is of great importance. An MRCT is described as a tear with a diameter of 5 cm or more, and or as a complete tear of two or more tendons, with at least one of the two tendons retracted beyond the top of the humeral head. It is important to exclude arthritis and the cuff arthropathy should be Hamada stage 2 or less.
- The location of MRCT seems to be very important. The classifications proposed by Gerber et al. [22] or by Collin et al. [23] are very useful for the clinician during decision making. The *postero-superior and antero-posterior tears* are prone to worse outcomes in terms of postoperative tendon integrity, reduced acromiohumeral distance, and functional results [20]. Additionally when over half of the subscapularis tendon is involved in

a postero-superior tear some authors propose other treatment options [24].

- Advanced preoperative fatty infiltration and atrophy. The preoperative fatty infiltration of the infraspinatus and or subscapularis with Goutallier stage 2 or higher are associated with worse postoperative outcomes even in patients with intact tendons [29].
- The pre-operative infraspinatus retraction may have a significant association with the 10-year Constant-Murley score [5].
- The decreased preoperative active external rotation in patients with postero-superior tears [25].
- The size of the postoperative tendon re-tear is significantly correlated with worse outcomes [9, 58].
- Older age is not always a contraindication of an MRCT repair [19].
- The onset of the tear is also important with traumatic tears showing better and more predictable outcomes [61].
- Zumstein et al. proposed that the wide lateral extension of the acromion is a risk factor of re-tear in MRCTs [65]. Recently, Taniguchi et al. described a new scale for measuring humeral head translation. The T-scale is the perpendicular distance from the head center to the coracoacromial line. The authors support that a negative T-scale value is a useful prognostic factor for considering reverse shoulder arthroplasty in patients with MRCTs [66].

9.4 Conclusions

Before any decision making, the recognition of a repairable MRCT and its location is of great importance. Despite the high failure rates after MRCT repair, in most cases the patients' functional outcomes are significantly better than their preoperative condition. However, surgeons should focus on the postoperative strength of the operated shoulder, which is still reduced in cases of anatomical failure and consider this along with the patients' occupational demands. Older (fatty infiltration and tear location) and newer (T-scale) prognostic factors should be preoperatively systematically evaluated before considering all possible treatment modalities, from arthroscopic repair to reverse shoulder arthroplasty.

References

1. Yamaguchi K, Ditsios K, Middleton WD, Hildebolt CF, Galatz LM, Teefey SA. The demographic and morphological features of rotator cuff disease. J Bone Joint Surg Am. 2006;88:1699–704.
2. Colvin AC, Egorova N, Harrison AK, Moskowitz A, Flatow EL. National trends in rotator cuff repair. J Bone Joint Surg Am. 2012;94:227–33.
3. Ensor KL, Kwon YW, DiBeneditto MR, Zuckerman JD, Rokito AS. The rising incidence of rotator cuff repairs. J Shoulder Elbow Surg. 2013;22:1628–32.
4. Zhang AL, Montgomery SR, Ngo SS, Hame SL, Wang JC, Gamradt SC. Analysis of rotator cuff repair trends in a large private insurance population. Arthroscopy. 2013;29:623–9.
5. Collin P, Colmar M, Thomazeau H, Mansat P, Boileau P, Valenti P, Saffarini M, Nover L, Kempf J-F. Clinical and MRI outcomes 10 years after repair of massive posterosuperior rotator cuff tears. J Bone Joint Surg Am. 2018;100:1854–63.
6. Kim KC, Shin HD, Cha SM, Kim JH. Repair integrity and functional outcomes for arthroscopic margin convergence of rotator cuff tears. J Bone Joint Surg Am. 2013;95:536–41.
7. Kim Y-K, Jung K-H, Kim J-W, Kim U-S, Hwang D-H. Factors affecting rotator cuff integrity after arthroscopic repair for medium-sized or larger cuff tears: a retrospective cohort study. J Shoulder Elbow Surg. 2018;27:1012–20.
8. Thès A, Hardy P, Bak K. Decision-making in massive rotator cuff tear. Knee Surg Sports Traumatol Arthrosc. 2015;23:449–59.
9. Galatz LM, Ball CM, Teefey SA, Middleton WD, Yamaguchi K. The outcome and repair integrity of completely arthroscopically repaired large and massive rotator cuff tears. J Bone Joint Surg Am. 2004;86-A:219–24.
10. Abtahi AM, Granger EK, Tashjian RZ. Factors affecting healing after arthroscopic rotator cuff repair. World J Orthop. 2015;6:211–20.
11. Haque A, Pal Singh H. Does structural integrity following rotator cuff repair affect functional outcomes and pain scores? A meta-analysis. Shoulder Elbow. 2018;10:163–9.
12. Barth J, Fotiadis E, Barthelemy R, Genna S, Saffarini M. Ultrasonic evaluation of the repair integrity can predict functional outcomes after arthroscopic double-row rotator cuff repair. Knee Surg Sports Traumatol Arthrosc. 2015;23:376–85.
13. McElvany MD, McGoldrick E, Gee AO, Neradilek MB, Matsen FA. Rotator cuff repair: published evidence on factors associated with repair integrity and clinical outcome. Am J Sports Med. 2015;43:491–500.

14. Saccomanno MF, Sircana G, Cazzato G, Donati F, Randelli P, Milano G. Prognostic factors influencing the outcome of rotator cuff repair: a systematic review. Knee Surg Sports Traumatol Arthrosc. 2016;24:3809–19.
15. Yang J, Robbins M, Reilly J, Maerz T, Anderson K. The clinical effect of a rotator cuff retear: a meta-analysis of arthroscopic single-row and double-row repairs. Am J Sports Med. 2017;45:733–41.
16. Boileau P, Brassart N, Watkinson DJ, Carles M, Hatzidakis AM, Krishnan SG. Arthroscopic repair of full-thickness tears of the supraspinatus: does the tendon really heal? J Bone Joint Surg Am. 2005;87:1229–40.
17. Tashjian RZ, Hollins AM, Kim H-M, Teefey SA, Middleton WD, Steger-May K, Galatz LM, Yamaguchi K. Factors affecting healing rates after arthroscopic double-row rotator cuff repair. Am J Sports Med. 2010;38:2435–42.
18. Flurin P-H, Hardy P, Abadie P, Boileau P, Collin P, Deranlot J, Desmoineaux P, Duport M, Essig J, Godenèche A, Joudet T, Kany J, Sommaire C, Thelu C-E, Valenti P, French Arthroscopy Society (SFA). Arthroscopic repair of the rotator cuff: prospective study of tendon healing after 70 years of age in 145 patients. Orthop Traumatol Surg Res. 2013;99:S379–84.
19. Jung HJ, Sim G-B, Bae KH, Kekatpure AL, Chun J-M, Jeon I-H. Rotator cuff surgery in patients older than 75 years with large and massive tears. J Shoulder Elbow Surg. 2017;26:265–72.
20. Ok HS, Kim BG, Choi WC, Hong CG, Kim JW, Kim JH. Clinical relevance of classifying massive rotator cuff tears: results based on functional and radiological findings after arthroscopic repair. Am J Sports Med. 2017;45:157–66.
21. Lädermann A, Denard PJ, Collin P. Massive rotator cuff tears: definition and treatment. Int Orthop. 2015;39:2403–14.
22. Gerber C, Fuchs B, Hodler J. The results of repair of massive tears of the rotator cuff. J Bone Joint Surg Am. 2000;82:505–15.
23. Collin P, Matsumura N, Lädermann A, Denard PJ, Walch G. Relationship between massive chronic rotator cuff tear pattern and loss of active shoulder range of motion. J Shoulder Elbow Surg. 2014;23:1195–202.
24. Park J-Y, Chung SW, Lee S-J, Cho H-W, Lee JH, Lee J-H, Oh K-S. Combined subscapularis tears in massive posterosuperior rotator cuff tears: do they affect postoperative shoulder function and rotator cuff integrity? Am J Sports Med. 2016;44:183–90.
25. Shimokobe H, Gotoh M, Honda H, Nakamura H, Mitsui Y, Kakuma T, Okawa T, Shiba N. Risk factors for retear of large/massive rotator cuff tears after arthroscopic surgery: an analysis of tearing patterns. J Orthop Surg Res. 2017;12:140.
26. Agout C, Berhouet J, Bouju Y, Godenèche A, Collin P, Kempf J-F, Favard L. Clinical and anatomic results of rotator cuff repair at 10 years depend on tear type. Knee Surg Sports Traumatol Arthrosc. 2018;26:2490–7.
27. Cho NS, Yi JW, Lee BG, Rhee YG. Retear patterns after arthroscopic rotator cuff repair: single-row versus suture bridge technique. Am J Sports Med. 2010;38:664–71.
28. Kim I-B, Kim M-W. Risk factors for retear after arthroscopic repair of full-thickness rotator cuff tears using the suture bridge technique: classification system. Arthroscopy. 2016;32:2191–200.
29. Ohzono H, Gotoh M, Nakamura H, Honda H, Mitsui Y, Kakuma T, Okawa T, Shiba N. Effect of preoperative fatty degeneration of the rotator cuff muscles on the clinical outcome of patients with intact tendons after arthroscopic rotator cuff repair of large/massive cuff tears. Am J Sports Med. 2017;45:2975–81.
30. Patte D. Classification of rotator cuff lesions. Clin Orthop Relat Res. 1990;(254):81–6.
31. Meyer DC, Farshad M, Amacker NA, Gerber C, Wieser K. Quantitative analysis of muscle and tendon retraction in chronic rotator cuff tears. Am J Sports Med. 2012;40:606–10.
32. Meyer DC, Wieser K, Farshad M, Gerber C. Retraction of supraspinatus muscle and tendon as predictors of success of rotator cuff repair. Am J Sports Med. 2012;40:2242–7.
33. Shin YK, Ryu KN, Park JS, Jin W, Park SY, Yoon YC. Predictive factors of retear in patients with repaired rotator cuff tear on shoulder MRI. Am J Roentgenol. 2017;210:134–41.
34. Gumina S, Candela V, Passaretti D, Latino G, Venditto T, Mariani L, Santilli V. The association between body fat and rotator cuff tear: the influence on rotator cuff tear sizes. J Shoulder Elbow Surg. 2014;23:1669–74.
35. Cho NS, Moon SC, Jeon JW, Rhee YG. The influence of diabetes mellitus on clinical and structural outcomes after arthroscopic rotator cuff repair. Am J Sports Med. 2015;43:991–7.
36. Cole BJ, McCarty LP III, Kang RW, Alford W, Lewis PB, Hayden JK. Arthroscopic rotator cuff repair: prospective functional outcome and repair integrity at minimum 2-year follow-up. J Shoulder Elbow Surg. 2007;16:579–85.
37. Duquin TR, Buyea C, Bisson LJ. Which method of rotator cuff repair leads to the highest rate of structural healing? Am J Sports Med. 2010;38:835–41.
38. Frank JB, ElAttrache NS, Dines JS, Blackburn A, Crues J, Tibone JE. Repair site integrity after arthroscopic transosseous-equivalent suture-bridge rotator cuff repair. Am J Sports Med. 2008;36:1496–503.
39. Neyton L, Godenèche A, Nové-Josserand L, Carrillon Y, Cléchet J, Hardy MB. Arthroscopic suture-bridge repair for small to medium size supraspinatus tear: healing rate and retear pattern. Arthroscopy. 2013;29:10–7.
40. Kim KC, Shin HD, Lee WY, Han SC. Repair integrity and functional outcome after arthroscopic rotator cuff repair double-row versus suture-bridge technique. Am J Sports Med. 2012;40:294–9.
41. Hantes ME, Ono Y, Raoulis VA, Doxariotis N, Venouziou A, Zibis A, Vlychou M. Arthroscopic single-row versus double-row suture bridge technique for rotator cuff tears in patients younger than 55 years:

a prospective comparative study. Am J Sports Med. 2018;46:116–21.
42. Lo IKY, Burkhart SS. Arthroscopic repair of massive, contracted, immobile rotator cuff tears using single and double interval slides: technique and preliminary results. Arthroscopy. 2004;20:22–33.
43. Berdusco R, Trantalis JN, Nelson AA, Sohmer S, More KD, Wong B, Boorman RS, Lo IKY. Arthroscopic repair of massive, contracted, immobile tears using interval slides: clinical and MRI structural follow-up. Knee Surg Sports Traumatol Arthrosc. 2015;23:502–7.
44. Kim S-J, Kim S-H, Lee S-K, Seo J-W, Chun Y-M. Arthroscopic repair of massive contracted rotator cuff tears: aggressive release with anterior and posterior interval slides do not improve cuff healing and integrity. J Bone Joint Surg Am. 2013;95:1482–8.
45. Gupta AK, Hug K, Berkoff DJ, Boggess BR, Gavigan M, Malley PC, Toth AP. Dermal tissue allograft for the repair of massive irreparable rotator cuff tears. Am J Sports Med. 2012;40:141–7.
46. Ono Y, Dávalos Herrera DA, Woodmass JM, Boorman RS, Thornton GM, Lo IKY. Graft augmentation versus bridging for large to massive rotator cuff tears: a systematic review. Arthroscopy. 2017;33:673–80.
47. Wong I, Burns J, Snyder S. Arthroscopic GraftJacket repair of rotator cuff tears. J Shoulder Elbow Surg. 2010;19:104–9.
48. Ditsios K, Boutsiadis A, Kapoukranidou D, Chatzisotiriou A, Kalpidis I, Albani M, Christodoulou A. Chronic massive rotator cuff tear in rats: in vivo evaluation of muscle force and three-dimensional histologic analysis. J Shoulder Elbow Surg. 2014;23:1822–30.
49. Zhang Q, Ge H, Zhou J, Cheng B. Are platelet-rich products necessary during the arthroscopic repair of full-thickness rotator cuff tears: a meta-analysis. PLoS One. 2013;8:e69731.
50. Zhao J-G, Zhao L, Jiang Y-X, Wang Z-L, Wang J, Zhang P. Platelet-rich plasma in arthroscopic rotator cuff repair: a meta-analysis of randomized controlled trials. Arthroscopy. 2015;31:125–35.
51. Anderson K, Boothby M, Aschenbrener D, van Holsbeeck M. Outcome and structural integrity after arthroscopic rotator cuff repair using 2 rows of fixation: minimum 2-year follow-up. Am J Sports Med. 2006;34:1899–905.
52. Flurin P-H, Landreau P, Gregory T, Boileau P, Brassart N, Courage O, Dagher E, Graveleau N, Guillo S, Kempf J-F, Lafosse L, Laprelle E, Toussaint B, et la Société Française d'Artroscopie. [Arthroscopic repair of full-thickness cuff tears: a multicentric retrospective study of 576 cases with anatomical assessment]. Rev Chir Orthop Reparatrice Appar Mot. 2005;91:31–42.
53. Galanopoulos I, Ilias A, Karliaftis K, Papadopoulos D, Ashwood N. The impact of re-tear on the clinical outcome after rotator cuff repair using open or arthroscopic techniques – a systematic review. Open Orthop J. 2017;11:95–107.
54. Gartsman GM, Drake G, Edwards TB, Elkousy HA, Hammerman SM, O'Connor DP, Press CM. Ultrasound evaluation of arthroscopic full-thickness supraspinatus rotator cuff repair: single-row versus double-row suture bridge (transosseous equivalent) fixation. Results of a prospective, randomized study. J Shoulder Elbow Surg. 2013;22:1480–7.
55. Lafosse L, Brozska R, Toussaint B, Gobezie R. The outcome and structural integrity of arthroscopic rotator cuff repair with use of the double-row suture anchor technique. J Bone Joint Surg Am. 2007;89:1533–41.
56. Russell RD, Knight JR, Mulligan E, Khazzam MS. Structural integrity after rotator cuff repair does not correlate with patient function and pain: a meta-analysis. J Bone Joint Surg Am. 2014;96:265–71.
57. Kim HM, Caldwell J-ME, Buza JA, Fink LA, Ahmad CS, Bigliani LU, Levine WN. Factors affecting satisfaction and shoulder function in patients with a recurrent rotator cuff tear. J Bone Joint Surg Am. 2014;96:106–12.
58. Papadopoulos P, Karataglis D, Boutsiadis A, Fotiadou A, Christoforidis J, Christodoulou A. Functional outcome and structural integrity following mini-open repair of large and massive rotator cuff tears: a 3-5 year follow-up study. J Shoulder Elbow Surg. 2011;20:131–7.
59. Vastamäki M, Lohman M, Borgmästars N. Rotator cuff integrity correlates with clinical and functional results at a minimum 16 years after open repair. Clin Orthop Relat Res. 2013;471:554–61.
60. Heuberer PR, Kölblinger R, Buchleitner S, Pauzenberger L, Laky B, Auffarth A, Moroder P, Salem S, Kriegleder B, Anderl W. Arthroscopic management of massive rotator cuff tears: an evaluation of debridement, complete, and partial repair with and without force couple restoration. Knee Surg Sports Traumatol Arthrosc. 2016;24:3828–37.
61. Godenèche A, Freychet B, Lanzetti RM, Clechet J, Carrillon Y, Saffarini M. Should massive rotator cuff tears be reconstructed even when only partially repairable? Knee Surg Sports Traumatol Arthrosc. 2017;25:2164–73.
62. Kim S-J, Choi Y-R, Jung M, Lee W, Chun Y-M. Arthroscopic repair of anterosuperior massive rotator cuff tears: does repair integrity affect outcomes? Am J Sports Med. 2017;45:1762–8.
63. Henry P, Wasserstein D, Park S, Dwyer T, Chahal J, Slobogean G, Schemitsch E. Arthroscopic Repair for Chronic Massive Rotator Cuff Tears: A Systematic Review. Arthroscopy: The Journal of Arthroscopic & Related Surgery. 2015;31:2472–80.
64. Lee SH, Nam DJ, Kim SJ, Kim JW. Comparison of clinical and structural outcomes by subscapularis tendon status in massive rotator cuff tear. Am J Sports Med. 2017;45:2555–62.
65. Zumstein MA, Jost B, Hempel J, Hodler J, Gerber C. The clinical and structural long-term results of open repair of massive tears of the rotator cuff. J Bone Joint Surg Am. 2008;90:2423–31.
66. Taniguchi N, D'Lima DD, Suenaga N, Chosa E. A new scale measuring translation of the humeral head as a prognostic factor for the treatment of large and massive rotator cuff tears. J Shoulder Elbow Surg. 2018;27:196–203.

10 Fatty Infiltration and Muscle Atrophy. What It Means and What Happens After Repair?

Michael Hantes and George Komnos

10.1 Introduction

Rotator cuff tears (RCTs) are a common cause of shoulder pain and disability. Fatty infiltration (FI) and muscle atrophy can be established and progress following RC tears, and are closely correlated to worse outcomes following rotator cuff repair. These two terms are usually used interchangeably. Fatty degeneration (FD) of the rotator cuff muscles appears due to tendon rupture or less often due to nerve damage. Rotator cuff tears were at the beginning associated with muscle degeneration as reported by Goutallier et al. [1]. FD can involve all muscles with ruptured tendons.

In terms of RC muscles, fatty degeneration takes place in the whole muscle mass of supraspinatus, while it appears in whole infraspinatus only if more than 50% of the tendon is torn [1]. Subscapularis degenerates only in the part of the tendon that is ruptured. Nevertheless, severe fatty infiltration can appear in subscapularis too. There are cases when infraspinatus degenerates without rupture, when the supraspinatus and subscapularis are torn on the antero-superior side. The duration of symptoms seems to cause increase of fatty infiltration, especially of the infraspinatus muscle [1].

Infraspinatus FI is gaining interest recently, because it seems to be the most rapidly affected muscle [2, 3]. It is reported that infraspinatus fatty degeneration is higher when an infraspinatus tear exist, and worsens when multiple tendons are torn [4]. Age, besides the aforementioned duration of symptoms, is found to be a predisposing factor for increased infraspinatus FI [4]. FI of the infraspinatus tendon can appear even without tendon rupture [5].

The pathogenesis and pathophysiology of FI and atrophy are complex and not clearly understood. Tendon rupture has an important effect on muscle physiology, structure, and function. FI is characterized by increased fibrosis and fiber shortening while mechanical unloading and denervation also have been found to contribute to atrophy [6]. Architectural changes take place as a result of tendon release and musculotendinous retraction [7]. Fibrous tissue and fat serve to fill the gap space created among muscle fibers. Furthermore, increased tension on the suprascapular nerve (SSN) may be applied by retracted tears of supraspinatus, causing nerve injury which subsequently leads to muscle atrophy [6, 8].

Although there are limited studies refusing association between age and FI [9], recent data find correlation between age and FI [2, 4, 10]. In a large retrospective study, evaluation of FI with the use of CT arthrography, in intact cuffs, showed a significant influence of aging on FI of supraspinatus, infraspinatus, and subscapularis,

M. Hantes (✉) · G. Komnos
Department of Orthopaedic Surgery
and Traumatology, University Hospital of Larissa,
Larissa, Greece
e-mail: hantesmi@otenet.gr

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_10

with significant acceleration after 40 years [11]. Moreover, the age of 70 seemed to be a particular threshold for FI. Same conclusions were presented by Raz et al. [12], reporting age-related increase of FI for supraspinatus, infraspinatus, and subscapularis, with earlier onset of infiltration in supraspinatus and subscapularis. In late stages, FI and atrophy may even be seen during clinical examination. Patients with massive RCTs and atrophy may appear with diminished external or internal rotation [13, 14].

10.2 Radiological and Clinical Implications

In terms of radiological evaluation, anteroposterior plain radiographs of the shoulder are usually ordered at initial admission, being of minor diagnostic occurrence for FI. However, narrowing of the acromio-humeral interval may gradually cause muscle atrophy, especially in infraspinatus tendon [4]. Computed tomography (CT) was used by Goutallier to establish his grading system (Table 10.1) [1]. Later, Fuchs et al. [15] modified this grading system with the application of magnetic resonance imaging (MRI) (Table 10.2). Their modified grading system includes three grades of fatty infiltration based on Goutallier grading (0 + 1, 2, 3 + 4) (Figs. 10.1 and 10.2). They found that interobserver reproducibility turns from good to excellent by using their modified classification.

Two most popular systems for grading of RC atrophy are the occupational ratio and the so-called "tangent sign." Occupational ratio, described by Thomazeau et al. [16], is defined (Table 10.3) as the surface area of the supraspinatus muscle/surface area of the entire supraspinatus fossa (Fig. 10.3). "Tangent sign" is seen

Table 10.1 Goutallier grading system for fatty infiltration (based on CT)

0	Normal
1	Some fatty steaks
2	More muscle than fat
3	Equal amounts of fat and muscle
4	More fat than muscle

Table 10.2 Fuchs grading system for fatty infiltration (based on CT and MRI)

1(0 + 1)	No or some fatty steaks	Normal muscle
2(2)	More muscle than fat	Moderate degeneration
3(4 + 5)	As much muscle as fat or more fat than muscle	Advanced degeneration

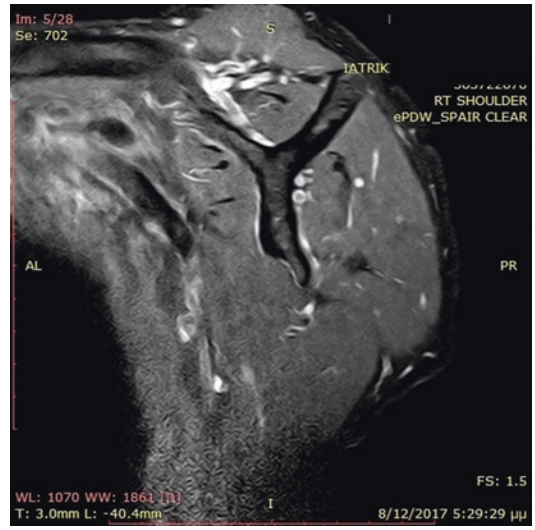


Fig. 10.1 Goutallier stage II/Fuchs grade II (more muscle than fat), MRI sagittal view

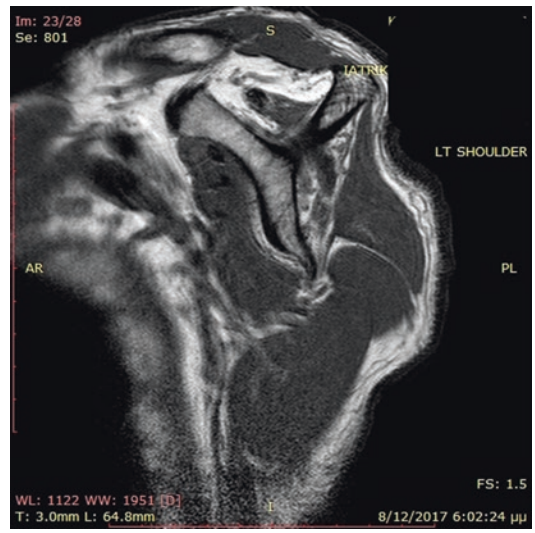


Fig. 10.2 Goutallier stage IV/Fuchs grade III (more fat than muscle), MRI sagittal view

Table 10.3 Thomazeau grading system for RC atrophy

Grade	Muscle atrophy	Occupation ratio
I	Normal/slight	0.6–1.0
II	Moderate	0.4–0.6
III	Severe	<0.4

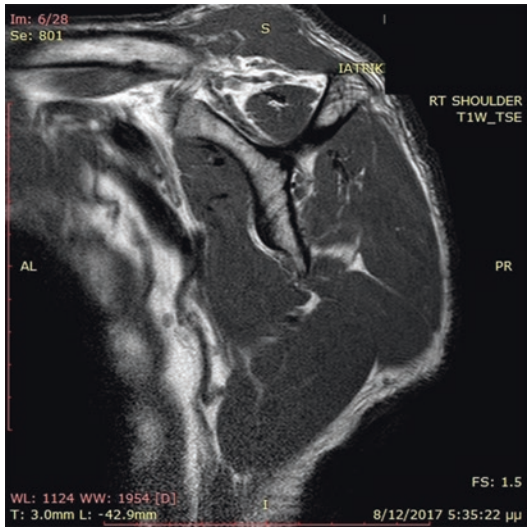


Fig. 10.3 Thomazeau grade II, MRI sagittal view

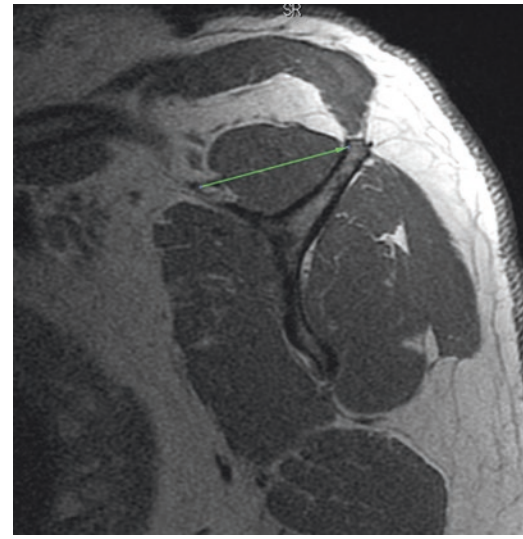


Fig. 10.4 Negative "tangent sign"—the supraspinatus crosses a line (green arrow) between the superior aspect of the coracoid process and the superior border of the scapular spine

on an oblique-sagittal MRI image ("Y" view) and is negative if the supraspinatus crosses a line between the superior aspect of the coracoids process and the superior border of the scapular spine (Fig. 10.4) [17]. Tangent sign is a useful MR sign for atrophy of the supraspinatus muscle. It is worth mentioning that interobserver agreement for evaluating FI in MRI is remarkably low according to some studies [18]. Ultrasonography has been also demonstrated to be a reliable means of diagnosing FI. Its advantages, such as low cost, fast and easy to perform, and dynamic evaluation of the RC, make it useful and preferable for the patient [6]. Nevertheless, the use of ultrasonography by an experienced physician is of major importance for proper diagnosis. Ultrasonography can also predict shoulder function after arthroscopic repair by evaluating morphological changes of the supraspinatus muscle [19].

Fatty degeneration is associated with tendon re-tear and postoperative muscle atrophy as well. As FI and muscle atrophy progress, the outcomes worsen. Fatty changes may negatively affect the clinical outcome regardless of tendon re-rupture. Nevertheless, both FI and muscle atrophy are considered as negative prognostic factors for shoulder function and clinical outcomes after repair of RC tears [20–23]. Goutallier et al. [24] proposed a cut-off of stage 2 according to their staging system for better results after repair. This finding was enhanced by Ohzono et al. [25] who reported that fatty degeneration of Goutallier stage 2 or more before surgery is a predisposing factor for unsatisfactory outcome. In another study, by Gladstone et al. [23], functional outcome and strength of the RC were compromised by FI [23]. The remarkable clue of their analysis was that FI and atrophy of the infraspinatus were found to be the only preoperative factors which deteriorate functional scores. Patients with stage 4 FI seem to experience much lower functional scores in comparison with patients with stage 1 FI after open repair [26]. Godeneche et al. [20] found that Constant scores were mainly influenced by preoperative FI of supraspinatus and not infraspinatus. Additionally Constant scores were significantly lower in patients with stage 1 or 2 FI compared to those with stage 0.

Valencia et al. [27] reported FI as a strong indicator of muscle weakness and subsequent loss of supraspinatus strength and function in an experimental study. Muscles with atrophy and FI are much weaker than muscles with atrophy alone.

Literature is controversial regarding the outcome of tendon repair on infiltration progression, regardless of the success or recurrent tears. Probably there is a level of damage in the muscles which cannot be regenerated. Therefore surgery should probably be performed before a high grade of fatty degeneration [4, 28]. Some authors have reported that infiltration and atrophy remain irreversible or progress even in successful repairs [23, 29]. They believe that atrophy remains irreversible, or is slowed by successful repair and advances after failure [30, 31]. Fuchs et al. [30] reported no significant change of muscle atrophy after RC repair, and statistically significant FI increase, despite repair. Nevertheless, RC atrophy was much greater in patients who experienced re-tear of RC after repair [30].

On the other hand, there are studies demonstrating reversal or improvement of atrophy after RC repair. Muscles seem to show an individual behavior regarding infiltration and atrophy after repair. In a study by Gerber et al. [32] with patients operated for massive RCTs, supraspinatus atrophy was slightly improved, while infraspinatus atrophy deteriorated even in successful repairs. In an experimental study in sheep, muscle atrophy was found to be partially reversible at 35 weeks following repair [33]. Butt et al. [34] demonstrated that atrophy is reversible following repair, since the mean cross-sectional area of muscle fibers of supraspinatus increases after repair. In a retrospective study, Fabbri et al. [35] compared two groups of patients with medium to large RCTs and found that surgery can stop muscular degenerative changes. They reported no progression of the muscle atrophy in the operative treatment group, while significant worsening was observed in the nonoperative group [35].

Improvement of muscle atrophy and FI can appear at 2 years after surgery, more than initially after operation according to the results of a study by Hamano et al. [36]. MRI findings confirmed the regeneration of muscle fibers at 2 years, while patients with improved FI experienced a better range of motion in flexion and abduction.

Nonoperative treatment is also proposed by some authors for RC tears. Success of nonoperative treatment seems to depend on FI of RC muscles. Jain et al. [37] reported that the absence of FI along with shorter duration of symptoms have more favorable outcomes in nonoperative treatment of RC tears.

10.3 Summary

In conclusion, fatty infiltration seems to be a progressive, aging process of the muscles. Trauma and/or rupture initiate, accelerate, or worsen this process. Rotator cuff repair after rotator cuff tear probably stops this degeneration process, improving or even sometimes reversing the established muscle atrophy.

References

1. Goutallier D, Postel JM, Bernageau J, Lavau L, Voisin MC. Fatty muscle degeneration in cuff ruptures. Pre- and postoperative evaluation by CT scan. Clin Orthop Relat Res. 1994;(304):78.
2. Barry JJ, Lansdown DA, Cheung S, Feeley BT, Ma CB. The relationship between tear severity, fatty infiltration, and muscle atrophy in the supraspinatus. J Shoulder Elbow Surg. 2013;22:18. https://doi.org/10.1016/j.jse.2011.12.014.
3. Chung SW, Oh JH, Gong HS, Kim JY, Kim SH. Factors affecting rotator cuff healing after arthroscopic repair: osteoporosis as one of the independent risk factors. Am J Sports Med. 2011;39:2099. https://doi.org/10.1177/0363546511415659.
4. Melis B, Wall B, Walch G. Natural history of infraspinatus fatty infiltration in rotator cuff tears. J Shoulder Elbow Surg. 2010;19:757. https://doi.org/10.1016/j.jse.2009.12.002.
5. Cheung S, Dillon E, Tham SC, Feeley BT, Link TM, Steinbach L, et al. The presence of fatty infiltration in the infraspinatus: its relation with the condition of the supraspinatus tendon. Arthroscopy. 2011;27:463. https://doi.org/10.1016/j.arthro.2010.09.014.
6. Kuzel BR, Grindel S, Papandrea R, Ziegler D. Fatty infiltration and rotator cuff atrophy. J Am Acad Orthop Surg. 2013;21:613. https://doi.org/10.5435/JAAOS-21-10-613.
7. Meyer DC, Hoppeler H, von Rechenberg B, Gerber C. A pathomechanical concept explains muscle loss and fatty muscular changes following surgical ten-

don release. J Orthop Res. 2004;22:1004. https://doi.org/10.1016/j.orthres.2004.02.009.
8. Albritton MJ, Graham RD, Richards RS, Basamania CJ. An anatomic study of the effects on the suprascapular nerve due to retraction of the supraspinatus muscle after a rotator cuff tear. J Shoulder Elbow Surg. 2003;12:497. https://doi.org/10.1016/S1058-2746(03)00182-4.
9. Goutallier D, Postel JM, Gleyze P, Leguilloux P, Van Driessche S. Influence of cuff muscle fatty degeneration on anatomic and functional outcomes after simple suture of full-thickness tears. J Shoulder Elbow Surg. 2003;12:550. https://doi.org/10.1016/S1058-2746(03)00211-8.
10. Kany J, Flurin PH, Richardi G, Hardy P. Rotator cuff tear imaging in patients over 70years: specific MRI findings? Orthop Traumatol Surg Res. 2013;99:S385. https://doi.org/10.1016/j.otsr.2013.10.003.
11. Gueniche J, Bierry G. Rotator cuff muscles fatty infiltration increases with age: retrospective review of 210 patients with intact cuff on computed tomography arthrography. J Shoulder Elbow Surg. 2019;28:617. https://doi.org/10.1016/j.jse.2018.09.020.
12. Raz Y, Henseler JF, Kolk A, Riaz M, van der Zwaal P, Nagels J, et al. Patterns of age-associated degeneration differ in shoulder muscles. Front Aging Neurosci. 2015;7:236. https://doi.org/10.3389/fnagi.2015.00236.
13. Walch G, Boulahia A, Calderone S, Robinson AHN. The "dropping" and "hornblower's" signs in evaluation of rotator-cuff tears. J Bone Joint Surg Br. 1998;80:624. https://doi.org/10.1302/0301-620x.80b4.8651.
14. Gerber C, Hersche O, Farron A. Isolated rupture of the subscapularis tendon. J Bone Joint Surg Am. 1996;78:1015.
15. Fuchs B, Weishaupt D, Zanetti M, Hodler J, Gerber C. Fatty degeneration of the muscles of the rotator cuff: assessment by computed tomography versus magnetic resonance imaging. J Shoulder Elbow Surg. 1999;80:599. https://doi.org/10.1016/S1058-2746(99)90097-6.
16. Thomazeau H, Boukobza E, Morcet N, Chaperon J, Langlais F. Prediction of rotator cuff repair results by magnetic resonance imaging. Clin Orthop Relat Res. 1997;(344):275.
17. Zanetti M, Gerber C, Hodler J. Quantitative assessment of the muscles of the rotator cuff with magnetic resonance imaging. Invest Radiol. 1998;33:163. https://doi.org/10.1097/00004424-199803000-00006.
18. Spencer EE, Dunn WR, Wright RW, Wolf BR, Spindler KP, McCarty E, et al. Interobserver agreement in the classification of rotator cuff tears using magnetic resonance imaging. Am J Sports Med. 2008;36:99. https://doi.org/10.1177/0363546507307504.
19. Kim YK, Choi ES, Kim KT, Yoon JR, Chae SH. Quantitative measurement of muscle atrophy and fat infiltration of the supraspinatus muscle using ultrasonography after arthroscopic rotator cuff repair. Ann Rehabil Med. 2018;42:260. https://doi.org/10.5535/arm.2018.42.2.260.
20. Godenèche A, Elia F, Kempf JF, Nich C, Berhouet J, Saffarini M, et al. Fatty infiltration of stage 1 or higher significantly compromises long-term healing of supraspinatus repairs. J Shoulder Elbow Surg. 2017;26:1818. https://doi.org/10.1016/j.jse.2017.03.024.
21. Barth J, Andrieu K, Fotiadis E, Hannink G, Barthelemy R, Saffarini M. Critical period and risk factors for retear following arthroscopic repair of the rotator cuff. Knee Surg Sport Traumatol Arthrosc. 2017;25:2196. https://doi.org/10.1007/s00167-016-4276-x.
22. Gerber C, Schneeberger AG, Hoppeler H, Meyer DC. Correlation of atrophy and fatty infiltration on strength and integrity of rotator cuff repairs: a study in thirteen patients. J Shoulder Elbow Surg. 2007;16:691. https://doi.org/10.1016/j.jse.2007.02.122.
23. Gladstone JN, Bishop JY, Lo IKY, Flatow EL. Fatty infiltration and atrophy of the rotator cuff do not improve after rotator cuff repair and correlate with poor functional outcome. Am J Sports Med. 2007;35:719. https://doi.org/10.1177/0363546506297539.
24. Goutallier D, Postel JM, Radier C, Bernageau J, Zilber S. Long-term functional and structural outcome in patients with intact repairs 1 year after open transosseous rotator cuff repair. J Shoulder Elbow Surg. 2009;18:521. https://doi.org/10.1016/j.jse.2008.11.006.
25. Ohzono H, Gotoh M, Nakamura H, Honda H, Mitsui Y, Kakuma T, et al. Effect of preoperative fatty degeneration of the rotator cuff muscles on the clinical outcome of patients with intact tendons after arthroscopic rotator cuff repair of large/massive cuff tears. Am J Sports Med. 2017;45:2975. https://doi.org/10.1177/0363546517724432.
26. Warner JJP, Higgins L, Parsons IM IV, Dowdy P. Diagnosis and treatment of anterosuperior rotator cuff tears. J Shoulder Elbow Surg. 2001;10:37. https://doi.org/10.1067/mse.2001.112022.
27. Valencia AP, Lai JK, Iyer SR, Mistretta KL, Spangenburg EE, Davis DL, et al. Fatty infiltration is a prognostic marker of muscle function after rotator cuff tear. Am J Sports Med. 2018;46:2161. https://doi.org/10.1177/0363546518769267.
28. Yamaguchi H, Suenaga N, Oizumi N, Hosokawa Y, Kanaya F. Will preoperative atrophy and fatty degeneration of the shoulder muscles improve after rotator cuff repair in patients with massive rotator cuff tears? Adv Orthop. 2012;2012:195876. https://doi.org/10.1155/2012/195876.
29. Kuenzler MB, Nuss K, Karol A, Schär MO, Hottiger M, Raniga S, et al. Neer Award 2016: reduced muscle degeneration and decreased fatty infiltration after rotator cuff tear in a poly(ADP-ribose) polymerase 1 (PARP-1) knock-out mouse model. J Shoulder Elbow Surg. 2017;26:733. https://doi.org/10.1016/j.jse.2016.11.009.
30. Fuchs B, Gilbart MK, Hodler J, Gerber C. Clinical and structural results of open repair of an isolated one-

tendon tear of the rotator cuff. J Bone Joint Surg Am. 2006;88:309. https://doi.org/10.2106/JBJS.E.00117.
31. Schaefer O, Winterer J, Lohrmann C, Laubenberger J, Reichelt A, Langer M. Magnetic resonance imaging for supraspinatus muscle atrophy after cuff repair. Clin Orthop Relat Res. 2002;(403):93. https://doi.org/10.1097/00003086-200210000-00015.
32. Gerber C, Fuchs B, Hodler J. The results of repair of massive tears of the rotator cuff. J Bone Joint Surg Am. 2000;82:505. https://doi.org/10.2106/00004623-200004000-00006.
33. Gerber C, Meyer DC, Schneeberger AG, Hoppeler H, Von Rechenberg B. Effect of tendon release and delayed repair on the structure of the muscles of the rotator cuff: an experimental study in sheep. J Bone Joint Surg Am. 2004;89:1973. https://doi.org/10.2106/00004623-200409000-00016.
34. Butt U, Rashid MS, Temperley D, Crank S, Birch A, Freemont AJ, et al. Muscle regeneration following repair of the rotator cuff. Bone Joint J. 2016;98-B:1389. https://doi.org/10.1302/0301-620X.98B10.37231.
35. Fabbri M, Ciompi A, Lanzetti RM, Vadalà A, Lupariello D, Iorio C, et al. Muscle atrophy and fatty infiltration in rotator cuff tears: can surgery stop muscular degenerative changes? J Orthop Sci. 2016;21:614. https://doi.org/10.1016/j.jos.2016.06.001.
36. Hamano N, Yamamoto A, Shitara H, Ichinose T, Shimoyama D, Sasaki T, et al. Does successful rotator cuff repair improve muscle atrophy and fatty infiltration of the rotator cuff? A retrospective magnetic resonance imaging study performed shortly after surgery as a reference. J Shoulder Elbow Surg. 2017;26:967. https://doi.org/10.1016/j.jse.2016.10.016.
37. Jain NB, Ayers GD, Fan R, Kuhn JE, Baumgarten K, Matzkin E, et al. Predictors of pain and functional outcomes after the nonoperative treatment of rotator cuff tears. Orthop J Sports Med. 2018;6:2325967118788531. https://doi.org/10.1177/2325967118788531.

Pseudoparalysis: Pathomechanics and Clinical Relevance

11

Emmanouil Brilakis and Dimitrios Gerogiannis

11.1 Introduction

Shoulder pseudoparalysis has been one of the most debatable issues in recent literature. Different opinions have been proposed for the definition of this condition, ranging from active forward flexion less than 90° due to massive rotator cuff (RC) tear [1–5] to no active elevation [6]. The same controversy also exists regarding the treatment options. Several treatment options have been proposed including conservative treatment [7], partial or complete RC repair [3, 4, 8], reverse total shoulder arthroplasty [1, 5, 9], tendon transfers [10], and more recently superior capsular reconstruction (SCR) [11–13].

Gschwend et al. were the first who mentioned the term pseudoparalysis and made the correlation between massive RC tears and decreased shoulder motion [14]. Massive RC tears (at least two tendons fully torn or tear dimension >5 cm) comprise approximately 20% of all RC tears but the clinical presentation may vary [15]. Some patients preserve their function with a full range of motion (ROM), limited pain, and small restrictions mainly due to the loss of strength, while others appear debilitated with nearly no shoulder motion and pain of different grades. In order to use the term pseudoparalysis, neurologic entities as well as cervical radiculopathy or axillary nerve damage must be excluded as they could provoke true paralysis because of deltoid and other parascapular muscle dysfunctions.

11.2 Definition

Shoulder pseudoparalysis should be defined as the clinical presentation of a patient with massive RC tear when shoulder active forward flexion is less than 45° with full passive range of motion. For the definition of pseudoparalysis, the absence of pain should be emphasized, because pain often may be the source of this limitation (Fig. 11.1). The shoulder inability for forward flexion could be described as a shoulder shrug, mainly produced from the scapulothoracic motion. In order to exclude pain as the main cause of the decreased elevation, a local anesthetic injection before the clinical evaluation has been proposed [6, 11].

As mentioned before, many articles refer to shoulder pseudoparalysis as shoulder elevation less than 90° with maintained passive motion without any pain reference. However, more recent articles [11, 27] clarify the term "pseudoparalysis" more rigorously. Burks et al. described the pseudoparalytic shoulder with 45° of elevation, unaffected passive motion, and also suggest that the condition should be chronic and with atraumatic onset [27]. Moreover, they also

E. Brilakis (✉) · D. Gerogiannis
3rd Orthopaedic Department, Hygeia General Hospital, Athens, Greece
e-mail: dgerogiannis@hotmail.gr

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_11

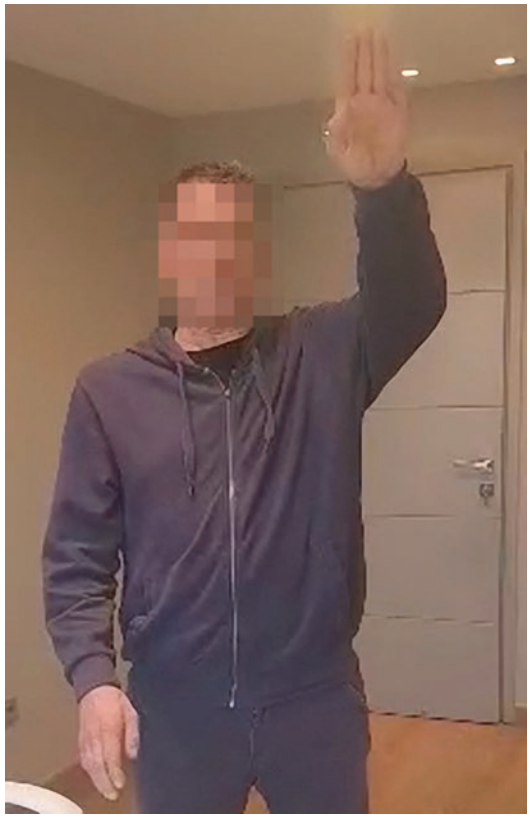


Fig. 11.1 Clinical presentation of pseudoparalysis. This patient has painless inability to actively flex forward his right arm over 45°. The absence of pain should be emphasized, because pain often may be the source of this limitation

mentioned that the massive RC tear should be combined with at least grade 2 fatty infiltration according to Goutallier classification [16]. Tokish et al. proposed two different terms, "pseudoparalysis" and "pseudoparesis" [6]. The term "pseudoparalysis" for patients with massive RC tears, 0° of active elevation and full passive elevation with anterior–superior escape, after injection of local anesthesia in order to eliminate the pain. The term "pseudoparesis," which was first mentioned by Werner et al., is for patients with massive RC tears, less than 90° anterior elevation with normal passive motion and no anterior–superior escape after the local anesthetic injection [5]. The authors also proposed the terms "external rotation (ER) pseudoparalysis" referring to patients without active ER, with ER lag and full passive ER and "external rotation (ER) pseudoparesis" for patients who can achieve active ER to neutral position but not in 30° of abduction. Burkhart and Hartzler suggested a shoulder shrug as a shoulder with profound pseudoparalysis, meaning less than 45° of active elevation, with full passive elevation after an injection of local anesthetic, making no reference to chronicity of the tear or the presence of anterior–superior escape [11, 17]. Finally, Mihata et al. divided pseudoparalysis to moderate and severe [12]. "Moderate pseudoparalysis" was defined as free passive ROM, less than 90° of active shoulder elevation but patients can maintain more than 90° elevation if the shoulder is brought to this position passively. On the other hand, "severe pseudoparalysis" was used for patients with free passive motion, less than 90° of shoulder active elevation and positive drop-arm test. Pain and the degree of muscle weakness could be a cause of the restricted active elevation in moderate pseudoparalysis; severe muscle weakness was highlighted as the main source of limited elevation in severe pseudoparalysis.

11.3 Biomechanics of Pseudoparalysis

Biomechanics of the pseudoparalytic shoulder is very interesting since the clinical relevance of massive RC tears could vary significantly, from patients with almost a full range of active motion to disabled patients with nearly no active motion of the shoulder. The answer to this controversy is found when considering the biomechanical function of the rotator cuff. Its main function is to create a balanced force couple around the glenohumeral joint. Coronal and transverse force couples are created between the subscapularis (SSC) anteriorly and the infraspinatus (IS) and teres minor (TM) posteriorly. This equilibrium centralizes the humeral head to the glenoid and allows the RC muscles to provide stability, acting as a stable fulcrum, for assisting the deltoid to elevate the arm. In massive RC tears, this equilibrium is disrupted creating unbalanced force couples and instability during active shoulder motion. During the deltoid contraction, the

humeral head migrates superiorly (cranially) as a result of the absence of the RC stabilizing action, leading to anterior–superior escape and eventually to limited shoulder active motion. There are several biomechanical analyses in the literature which attempt to specify the main source of pseudoparalysis in massive tears. The integrity of RC cable [2], the RC tear pattern [18], the volume of RC musculature [19], and the fatty infiltration, as well as the continuity of the superior capsule [20] have been highlighted as the key points of pseudoparalysis.

Denard et al. tried to find the connection between rotator cable integrity and pseudoparalysis [2]. The cable is a thickening of the RC right before its insertion to the humeral head. Forces are distributed through the cable, which acts as a suspension bridge, to its anterior and posterior attachments bypassing a potential RC tear. The anterior attachment of the rotator cable bifurcates, and the anterior portion passes over the biceps tendons before attaching to the lesser tuberosity, while the posterior portion of the anterior attachment inserts just posterior to the bicipital groove. The posterior attachment consists of the inferior part of infraspinatus insertion [21]. Denard et al. also studied the rotator cable attachments integrity in 127 patients with massive RC tears, 24 with pseudoparalysis and 103 with forward flexion more than 90° [2]. A tear that involved more than 50% of subscapularis insertion was defined as disruption of the anterior attachment while a tear of 100% of infraspinatus insertion was defined as disruption of posterior attachment. When both rotator cable attachments were intact the percentage of pseudoparalysis was zero. In the cases with one attachment disruption (anterior or posterior) pseudoparalysis was detected in 14.7% and when both attachments were disrupted pseudoparalysis was present in 44.8% of the cases.

Collin et al. examined the relationship between RC tear pattern and pseudoparalysis in 100 patients with massive RC tears, fatty infiltration beyond Goutallier stage 3 and without significant glenohumeral arthritis (lower than grade 3 according to Hamada classification [22]). The RC was divided into five elements: superior subscapularis, inferior subscapularis, supraspinatus (SS), infraspinatus, and teres minor (Fig. 11.2). The pattern of the RC tear was also classified into five types. Type A consists of supraspinatus and superior subscapularis tears; type B supraspinatus and entire subscapularis tears; type C supraspinatus, superior subscapularis, and infraspinatus tears; type D supraspinatus and infraspinatus tears; and type E supraspinatus, infraspinatus, and teres minor tears (Fig. 11.3). Pseudoparalysis was detected in 0% of patients with type A tears; in 80% of the patients with type B; 45.5% with type C; 2.9% with type D; and 33.3% with type E tears, respectively. Based on the results, the authors stated that subscapularis tears, especially those of the inferior portion, is the main contributing factor for developing pseudoparalysis and subscapularis repair is crucial for revering it [18]. The significantly high percentage of pseudoparalysis

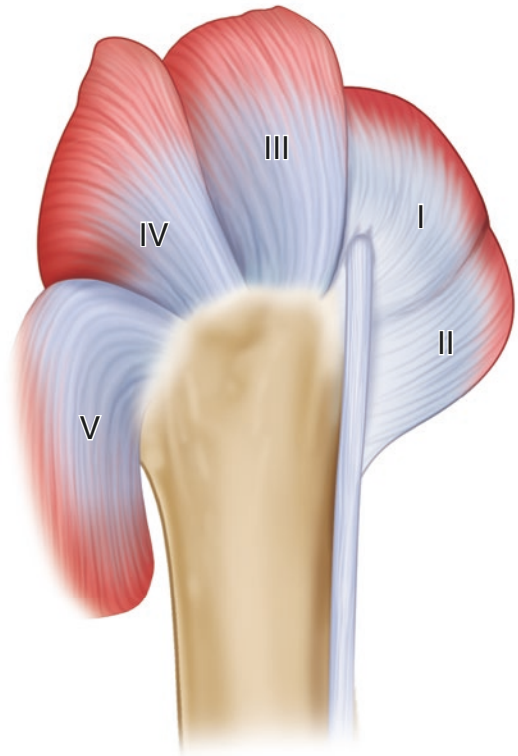


Fig. 11.2 Collin et al. examined the relationship between the RC tear pattern and pseudoparalysis [18]. The RC was divided into five elements: (I) superior subscapularis, (II) inferior subscapularis, (III) supraspinatus, (IV) infraspinatus, and (V) teres minor

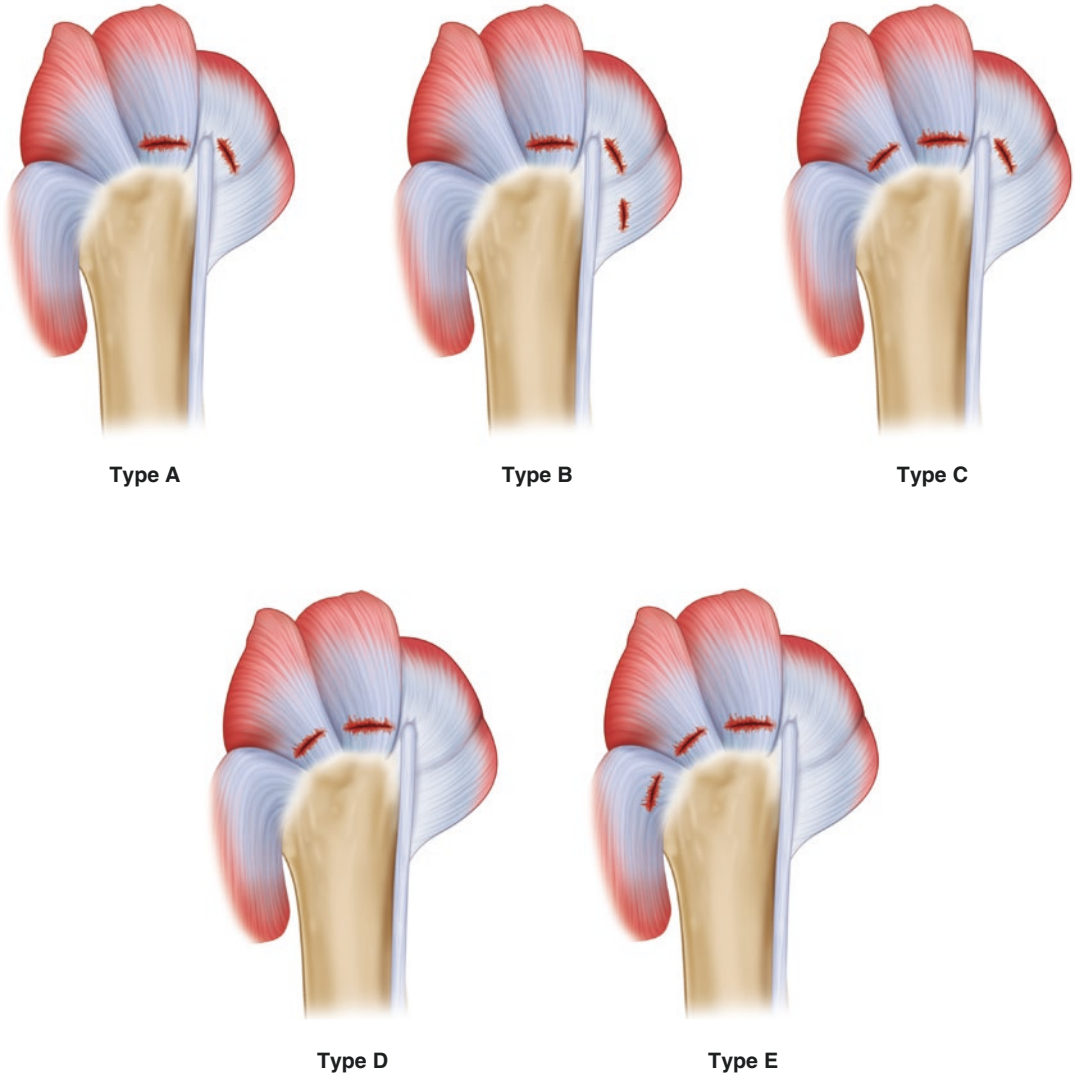


Fig. 11.3 The pattern of the RC tear is classified into five types according Collin et al. and the relationship between the tear pattern and pseudoparalysis was evaluated [18]. *Type A*: supraspinatus and superior subscapularis tears. *Type B*: supraspinatus and entire subscapularis tears. *Type C*: supraspinatus, superior subscapularis, and infraspinatus tears. *Type D*: supraspinatus and infraspinatus tears. *Type E*: supraspinatus, infraspinatus, and teres minor tears

in group B could be explained by the fact that a tear of inferior subscapularis represents a disruption of the anterior rotator cable attachment. This study also concluded that the dysfunction of three RC muscles, as they were previously divided, is a risk factor for pseudoparalysis.

In another biomechanical study, the volumes of the RC muscles and their ratio were measured in order to investigate the effect of a massive RC tear to active shoulder motion [19]. The volumes of subscapularis, divided into the superior and inferior parts, supraspinatus, infraspinatus, and teres minor, and their ratios were measured on MRI scan in 53 patients with massive irreparable RC tears (22 pseudoparalytic and 31 nonpseudoparalytic). The same measures were conducted in a control group of 25 individuals with no RC pathology. While an imbalance in the anterior to posterior cuff muscle volume ratio (SSC/IS + TM) was noticed in all patients

with massive RC tears, there was no significant difference between the pseudoparalytic and the nonpseudoparalytic group. On the contrary, the relationship between the decreased volume of inferior subscapularis and infraspinatus and the development of pseudoparalysis were statistically significant. The importance of subscapularis tear was again emphasized by the fact that 81% of patients with inferior subscapularis tear developed pseudoparalysis.

11.4 Management of Patients with Pseudoparalysis

The abovementioned controversy that exists on the definition and also the complexity of pseudoparalysis' biomechanics clarifies why so many different treatment options have been proposed. Partial or complete RC repair, reverse shoulder arthroplasty and superior capsular reconstruction with allograft or fascia lata autograft have been proposed in the recent literature as solutions for patients with massive RC tears and pseudoparalysis. Other factors besides tear size, such as tendon mobility, fatty infiltration of RC muscles [16, 23], tendon retraction based on Patte classification [24], and chronicity of the tear could influence the surgeons for selecting proper treatment. It should be mentioned that a study by Levy et al. reported significant increase of forward elevation in patients with massive RC tear (from 40° preoperatively to 160° postoperatively) with a 9-month-duration rehabilitation program of the anterior deltoid [7]. However, these patients had significant pain which could be the reason for motion limitation. In those patients, the rehabilitation program, in addition to the local injections used, might increase the forward elevation by pain elimination.

Reverse shoulder arthroplasty (RSA) has been stated as the "gold standard" for the treatment of RC arthropathy. In the case of an irreparable RC tear and significant osteoarthritic changes (greater than grade 4 according to Hamada classification), reverse shoulder arthroplasty provides a reliable solution [25]. Rotator cuff dysfunction leads to an unbalanced force couple in coronal plane, resulting in superior migration of the humeral head from the deltoid pull. The "reverse ball and socket" design provides the stable fulcrum for assisting the deltoid to elevate the shoulder, while anatomic total shoulder arthroplasty and hemiarthroplasty cannot compensate this biomechanical instability and less satisfactory results have been recorded [26]. The indications for RSA have been widely expanded nowadays and the RSA has been stated as well as the "gold standard" for the treatment of pseudoparalysis due to massive RC tear providing good outcomes in both, pain relief and motion restoration [1, 6, 9, 26–29]. Many surgeons supporting RSA, propose a rehabilitation program which should start preoperatively, even as long as 6 months.

Werner et al. was the first who evaluated the results of reverse shoulder arthroplasty in patients with painful pseudoparesis, defined as active shoulder elevation less than 90° with full passive motion, due to massive irreparable RC tear [5]. Both primary and revision cases were included (17 and 41, respectively) and good results were recorded as far as the pain relief, the active forward elevation (from 42° to 100°), and the constant score (from 29% to 64%) were concerned. The complication rate was higher than other studies (50%), but the author attributed this to the fact that even hematoma was recorded as a complication. It was mentioned that only in 6 of 29 patients with complication, revision or removal of prosthesis was required.

Mullieri et al. studied 58 patients (60 shoulders) with massive RC tear and no significant arthritis (Hamada stage 3 or less) who underwent RSA [9]. Fifty-six shoulders (93%) had less than 90° of elevation preoperatively, while all shoulders had free passive motion. Functional and pain scores improved significantly, forward flexion increased from 53° to 134°, and the complication rate was 20%. The author concluded that RSA is able to reduce pain and restore shoulder function in patients suffering from massive tears, with <90° forward elevation and no glenohumeral arthritis with or without antero-superior escape.

RC repair can also be used for the treatment of pseudoparalysis in the case of massive tears without arthritis. Undoubtedly, the repair of a massive

tear could be challenging even for experienced shoulder surgeons and advanced techniques, as anterior and posterior slides may be required. However, a small proportion of RC tears is irreparable. Denard et al. published his results for the treatment of pseudoparalysis (defined as less than 90° active forward elevation with normal passive ROM) with RC repair [3]. After 1-year follow-up, 95% reversal of pseudoparalysis was detected regardless of the tendon's fatty infiltration. It should be mentioned that in the total number of patients with a fatty degeneration of grade 3 or more, pseudoparalysis was reversed. On the other hand, the duration of pseudoparalysis was relatively small (3.9 ± 5.6 months before surgery) and acute in 80% of the cases. The percentage of pseudoparalysis reversal seems to be significantly different between primary and revision RC repairs. In a comparative study, 90% of pseudoparalysis was reversed in primary repairs compared to 43% in revision cases [8]. However, the mean duration of pseudoparalysis was significantly longer in revision cases (20.8 months) than in primary ones (4.6 months) and that may influence tendons' reparability. Oh et al. compared patients with massive RC tears with and without painful pseudoparalysis preoperatively [4]. Functional scores and ROM did not differ significantly postoperatively among the two groups apart from the constant score, specifically the strength measurement, and the active forward elevation. 24.1% of patients with pseudoparalysis failed to achieve active elevation more than 90°. A cost-related study of RC repair and arthroplasty was contacted in patients with massive RC tears and pseudoparalysis with no arthritic changes. Three case scenarios were studied: (1) arthroscopic cuff repair (ACR) with an option to arthroscopic revision, (2) ACR with conversion to RSA in case of failure, and (3) primary RSA. Arthroscopic RC repair with conversion to RSA was proved to be the most cost-effective solution.

Recently, superior capsular reconstruction demonstrates extremely promising results in patients with "true" pseudoparalysis (active elevation >45° with normal passive motion). SCR is documented to restore the shoulder function in massive irreparable RC tears with osteoarthritic changes less than grade 3 of Hamada classification. The biomechanics of this innovative technique is the restoration of the superior instability of massive tears by adding a biologic constraint [20]. This constraint represents the stable fulcrum which is amenable for deltoid normal function. Burkhart and Hartzler studied the results of SCR, using allograft, in pseudoparalytic patients with irreparable RC tears and minimum arthritis [11]. Pseudoparalysis was defined as an active elevation >45° with full passive elevation after pain elimination with a local anesthetic injection. Ninety percent of the patients (nine out of ten) succeeded in restoring active forward elevation (from 27° to 159°). Only in one patient (10%) pseudoparalysis was not reversed, but this patient also suffered from cervical radiculopathy with deltoid weakness. Mihata et al. divided patients with irreparable RC tears into three groups and studied the results of SCR with fascia lata graft [12]. The three groups were: (1) No pseudoparalysis, (2) moderate pseudoparalysis with no stiffness and shoulder active elevation <90° but not drop-arm sign, and (3) severe pseudoparalysis with active elevation <90° and positive drop-arm test. Pseudoparalysis was reversed in 96.4% of patients with moderate pseudoparalysis (27 of 28) and in 93.3% with severe pseudoparalysis (14 of 15). Both the patients who remained with pseudoparalysis had graft tears in the MRI performed. ROM and functional scores did not have significant differences among the three groups postoperatively. The authors concluded that SCR is able to reverse pseudoparalysis if the graft does not fail.

The supporters of joint replacement techniques argue that RC repair can restore the shoulder function only in patients with active shoulder elevation just less 90° and not in "true" pseudoparalysis. Moreover, there are some massive tears that are not amenable even for a partial repair. It was stated that RSA might be the only solution for patients with true pseudoparalysis due to an irreparable RC tear. SCR demonstrates very promising results for the treatment of pseudoparalysis in patients with irreparable RC tears without significant glenohumeral arthritis. Taking into account the low complication rate, SCR seems to

be a reasonable and reliable treatment for pseudoparalytic patients. However, it is technically quite a demanding procedure and its results on pseudoparalysis must be reproducible in further studies.

11.5 Conclusion

Pseudoparalysis is a very challenging condition for a shoulder surgeon to treat. The definition and the biomechanics of this entity vary significantly, rendering the diagnosis and the management difficult and patient specific. The optimal treatment for pseudoparalytic patients should be individualized for each patient, since there are many factors that influence the surgeons' selection.

Conflict of Interest Emmanouil Brilakis and Dimitrios Gerogiannis declare that they have no conflict of interest.

References

1. Boileau P, Chuinard C, Roussanne Y, Bicknell RT, Rochet N, Trojani C. Reverse shoulder arthroplasty combined with a modified latissimus dorsi and teres major tendon transfer for shoulder pseudoparalysis associated with dropping arm. Clin Orthop Relat Res. 2008;466:584–93.
2. Denard PJ, Koo SS, Murena L, Burkhart SS. Pseudoparalysis: the importance of rotator cable integrity. Orthopedics. 2012;35:e1353–7.
3. Denard PJ, Ladermann A, Brady PC, Narbona P, Adams CR, Arrigoni P, Huberty D, Zlatkin MB, Sanders TG, Burkhart SS. Pseudoparalysis from a massive rotator cuff tear is reliably reversed with an arthroscopic rotator cuff repair in patients without preoperative glenohumeral arthritis. Am J Sports Med. 2015;43:2373–8.
4. Oh JH, Kim SH, Shin SH, Chung SW, Kim JY, Kim SH, Kim SJ. Outcome of rotator cuff repair in large-to-massive tear with pseudoparalysis: a comparative study with propensity score matching. Am J Sports Med. 2011;39:1413–20.
5. Werner CM, Steinmann PA, Gilbart M, Gerber C. Treatment of painful pseudoparesis due to irreparable rotator cuff dysfunction with the Delta III reverse-ball-and-socket total shoulder prosthesis. J Bone Joint Surg Am. 2005;87:1476–86.
6. Tokish JM, Alexander TC, Kissenberth MJ, Hawkins RJ. Pseudoparalysis: a systematic review of term definitions, treatment approaches, and outcomes of management techniques. J Shoulder Elb Surg. 2017;26:e177–87.
7. Levy O, Mullett H, Roberts S, Copeland S. The role of anterior deltoid reeducation in patients with massive irreparable degenerative rotator cuff tears. J Shoulder Elb Surg. 2008;17:863–70.
8. Denard PJ, Lädermann A, Jiwani AZ, Burkhart SS. Functional outcome after arthroscopic repair of massive rotator cuff tears in individuals with pseudoparalysis. Arthroscopy. 2012;28:1214–9.
9. Mulieri P, Dunning P, Klein S, Pupell D, Frankle M. Reverse shoulder arthroplasty for the treatment of irreparable rotator cuff tears without glenohumeral arthritis. J Bone Joint Surg Am. 2010; 92:2544–56.
10. Birmingham PM, Neviaser RJ. Outcome of latissimus dorsi transfer as a salvage procedure for failed rotator cuff repair with loss of elevation. J Shoulder Elb Surg. 2008;17:871–4.
11. Burkhart SS, Hartzler RU. Superior capsular reconstruction reverses profound pseudoparalysis in patients with irreparable rotator cuff tears and minimal or no glenohumeral arthritis. Arthroscopy. 2019;35:22–8.
12. Mihata T, Lee TQ, Hasegawa A, Kawakami T, Fukunishi K, Fujisawa Y, Itami Y, Ohue M, Neo M. Arthroscopic superior capsule reconstruction can eliminate pseudoparalysis in patients with irreparable rotator cuff tears. Am J Sports Med. 2018;46(11):2707–16.
13. Mihata T, Lee TQ, Watanabe C, Fukunishi K, Ohue M, Tsujimura T, Kinoshita M. Clinical results of arthroscopic superior capsule reconstruction for irreparable rotator cuff tears. Arthroscopy. 2013;29:459–70.
14. Gschwend N, Ivosevic-Radovanovic D, Patte D. Rotator cuff tear - relationship between clinical and anatomopathological findings. Arch Orthop Trauma Surg. 1988;107:7–15.
15. Burkhart SS, Danaceau SM, Pearce CE Jr. Arthroscopic rotator cuff repair: analysis of results by tear size and by repair technique—margin convergence versus direct tendon-to-bone repair. Arthroscopy. 2001;17:905–12.
16. Goutallier D, Postel JM, Bernageau J, Lavau L, Voisin MC. Fatty muscle degeneration in cuff ruptures. Pre- and postoperative evaluation by CT scan. Clin Orthop Relat Res. 1994;304:78–83.
17. Burchart SS. Letter to the Editor regarding Tokish et al: "Pseudoparalysis: a systematic review of term definitions, treatment approaches, and outcomes of management techniques". J Shoulder Elb Surg. 2018;27:e56–7.
18. Collin P, Matsumura N, Lädermann A, Denard PJ, Walch G. Relationship between massive chronic rotator cuff tear pattern and loss of active shoulder range of motion. J Shoulder Elb Surg. 2014;23:1195–202.
19. Rhee YG, Cho NS, Song JH, Park JG, Kim TY. Volumetric evaluation of the rotator cuff musculature in massive rotator cuff tears with pseudoparalysis. J Shoulder Elb Surg. 2017;26(9):1520–6.

20. Mihata T, McGarry MH, Kahn T, Goldberg I, Neo M, Lee TQ. Biomechanical role of capsular continuity in superior capsule reconstruction for irreparable tears of the supraspinatus tendon. Am J Sports Med. 2016;44:1423–30.
21. Burkhart SS, Esch JC, Jolson RS. The rotator crescent and rotator cable: an anatomic description of the shoulder's "suspension bridge". Arthroscopy. 1993;9:611–6.
22. Hamada K, Yamanaka K, Uchiyama Y, Mikasa T, Mikasa M. A radiographic classification of massive rotator cuff tear arthritis. Clin Orthop Relat Res. 2011;469:2452–60.
23. Fuchs B, Weishaupt D, Zanetti M, Hodler J, Gerber C. Fatty degeneration of the muscles of the rotator cuff: assessment by computed tomography versus magnetic resonance imaging. J Shoulder Elb Surg. 1999;8:599–605.
24. Patte D. Classification of rotator cuff lesions. Clin Orthop Relat Res. 1990;254:81–6.
25. Drake GN, O'Connor DP, Edwards TB. Indications for reverse total shoulder arthroplasty in rotator cuff disease. Clin Orthop Relat Res. 2010;468(6):1526–33.
26. Field LD, Dines DM, Zabinski SJ, Warren RF. Hemiarthroplasty of the shoulder for rotator cuff arthropathy. J Shoulder Elb Surg. 1997;6:18–23.
27. Burks RT, Tashjian RZ. Should we have a better definition of pseudoparalysis in patients with rotator cuff tears? Arthroscopy. 2017;33:2281–3.
28. Lädermann A, Denard PJ, Collin P. Massive rotator cuff tears: definition and treatment. Int Orthop. 2015;39(12):2403–14.
29. Sellers TR, Abdelfattah A, Frankle MA. Massive rotator cuff tear: when to consider reverse shoulder arthroplasty. Curr Rev Musculoskelet Med. 2018;11(1):131–40.

12 Rotator Cuff Tear Arthropathy: Where Are the Limits for Repair?

Emmanouil Antonogiannakis
and Grigorios Avramidis

12.1 Introduction

The term "cuff tear arthropathy" was first described by Neer in 1983 [1] as a "peculiar lesion of the glenohumeral joint because of the unique anatomy of the rotator cuff." Neer claimed that both biological and mechanical factors were responsible for the development of cuff tear arthropathy. A massive rotator cuff tear is the initial event causing inactivity and disuse of the shoulder, leakage of the synovial fluid, and loss of normal joint pressure as well as instability of the shoulder. Its consequences are atrophy of the glenohumeral cartilage, disused osteoporosis of the humeral head, and superior migration of the humeral head that primarily impinges on acromion. Subsequently, the humeral head erodes the acromion, the acromioclavicular joint, and eventually the glenoid. Finally, the soft, atrophic humeral head collapses and forms the complete syndrome of cuff tear arthropathy with complete joint destruction.

12.2 Rotator Cuff Arthropathy Classification

Characteristic radiographic changes accompany the progression of massive rotator cuff tears (MRCT) to cuff tear arthropathy and have been reported since Codman's study in 1934 [2]. These changes include the narrowing of the acromiohumeral interval (AHI) and degenerative changes of the humeral head and the tuberosities, the acromion, the acromioclavicular joint, and the glenoid [1, 3]. Based on these radiographic changes, Hamada et al. [3] proposed five stages of massive rotator cuff tears (Figs. 12.1, 12.2, 12.3, 12.4 and 12.5) [4]. In stage I, the AHI was more than

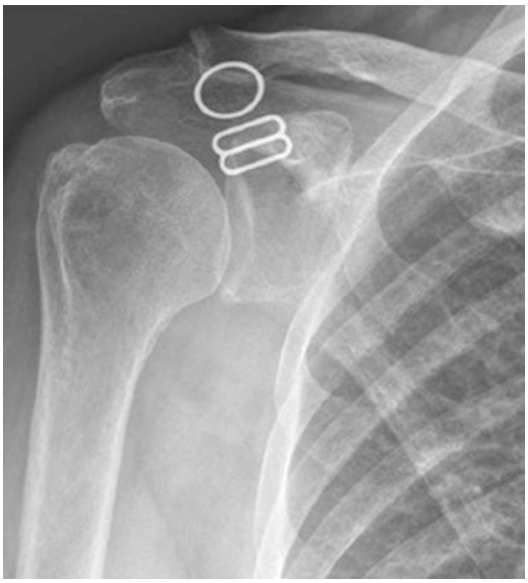


Fig. 12.1 Hamada classification grade 1 is characterized by an acromiohumeral interval more than 6 mm. (Reproduced from Brolin et al. [4])

E. Antonogiannakis (✉) · G. Avramidis
3rd Orthopaedic Department, Hygeia General Hospital, Athens, Greece

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_12

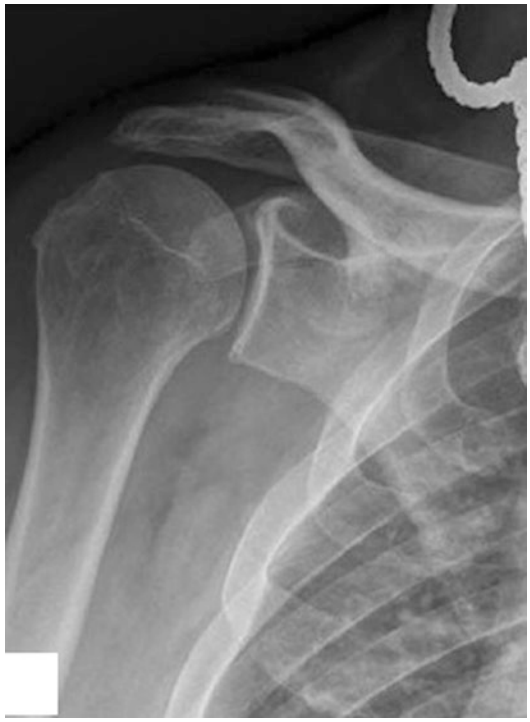


Fig. 12.2 Hamada classification grade 2 shows an acromiohumeral interval less than 5 mm. (Reproduced from Brolin et al. [4])

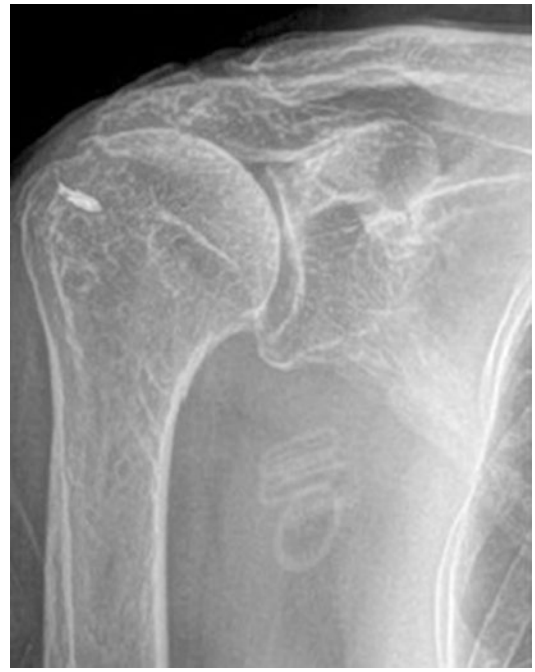


Fig. 12.4 Hamada classification grade 4 shows the addition of glenohumeral narrowing. (Reproduced from Brolin et al. [4])

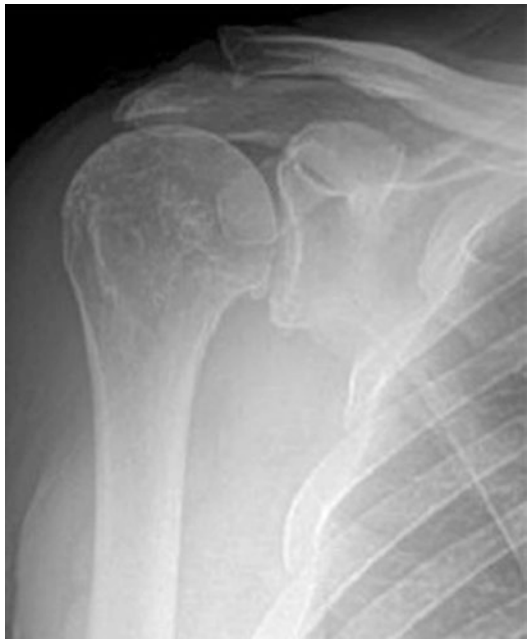


Fig. 12.3 Hamada classification grade 3 shows the acromial acetabulization. (Reproduced from Brolin et al. [4])

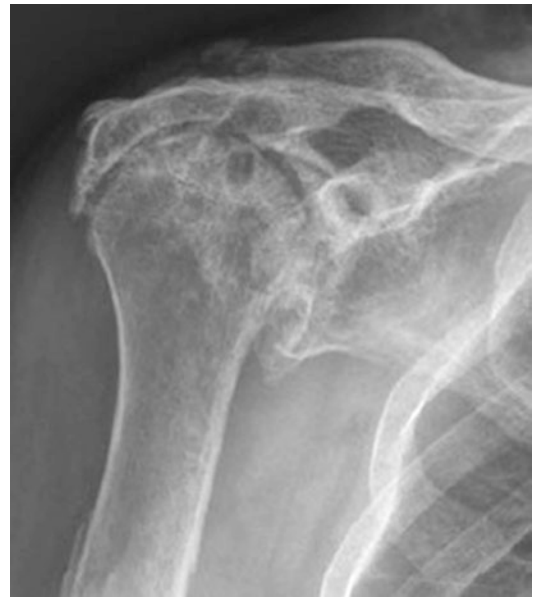


Fig. 12.5 Hamada classification grade 5 is characterized by the development of humeral head collapse. (Reproduced from Brolin et al. [4])

6 mm while it was 5 mm or less in stage 2. In stage 3, acetabulization of the acromion undersurface (excavating deformity or deformity formed by the excessive spur along the coracoacromial ligament) was added to the stage 2 features. In stage 4, narrowing of the glenohumeral joint was added to the stage 3 characteristics. Stage 5 comprised instances of humeral-head collapse. The last stages included radiological changes to the glenohumeral joint generally referred to as rotator tear arthropathy. In 2005, Walch et al. divided the Hamada grade 4 classification into two subtypes. In grade 4A, they noted the narrowing of the glenohumeral joint without subacromial acetabulization and in grade 4B, they observed the narrowing of the glenohumeral joint in the setting of subacromial acetabulization [5].

Although the Hamada classification includes glenohumeral joint changes, it does not address morphological erosions in the glenoid. These erosions are classified into four types by Sirveaux et al. [6]. Type E0 has proximal migration of the humeral head without glenoid erosion. Type E1 has concentric erosion, type E2 has erosion of the superior part of the glenoid, and type E3 has erosion that extends to the inferior portions of the glenoid.

The Seebauer classification defines four groups distinguished by the degree of superior migration of humeral head from the center of rotation and the extent of instability (Fig. 12.6) [7]. The classification is based on the biomechanics of the joint to aid in the decision-making of the implant type and the goals of reconstruction during the late stages of cuff tear arthropathy [8].

12.3 Searching for the Limits

12.3.1 Treatment Options

Historically, cuff tear arthropathy has been difficult to treat. Furthermore, it is especially difficult to set the limit for repair of the massive rotator cuff tear with or without radiographic changes in order to address the pathology and try to avoid the development of this condition. Treatment options besides conservative treatment include arthroscopic repair with debridement, subacromial decompression and biceps tenotomy, partial or complete rotator cuff repair, tendon transfer, various grafting and tendon augmentation techniques, superior capsular reconstruction, and reverse shoulder arthroplasty (RSA). While rotator cuff arthropathy at the end stage is an undisputable indication of reverse shoulder arthroplasty, the optimal treatment for patients with MRCT in the absence of arthritis or with early arthritic changes remains controversial and at the same time set up the limit between the repair and the arthroplasty. On the contrary, when a massive rotator cuff is reparable without evidence of glenohumeral arthritis, it is a clear indication for repair.

12.3.2 Tendon Reparability and Healing Potential

Therefore, one of the critical points is the tendon reparability and there are several factors that contribute to a successful rotator cuff repair. These factors include the patient's age and health, the size and chronicity of the tear, the retraction and fatty infiltration of the remaining cuff tissue as well as the surgeon's skills and experience [9–14]. Additionally, several factors such as poor tendon quality and patient's comorbidities can affect healing potential even in cases where anatomic repair is achieved [15–18]. However, in cases of chronicity, a complete anatomic repair may not be possible due to severe retraction and poor tendon quality [19]. Patient's health and the presence of comorbidities such as smoking and diabetes may lead to poor potential for healing after repair. Such patients can be good candidates for reverse shoulder arthroplasty especially if they are older than 65 years old [16–18]. Patient's age is one of the most important factors considered while making decisions for repair or arthroplasty. While multiple reports in literature have shown RSA to be a reliable procedure with good outcomes in patients more than 65 years of age [20–23], being younger seems to be a risk factor for lower satisfaction rates [20] and poor functional improvement after RSA in the specific setting of MRCT without arthritis in patients aged <60 [24].

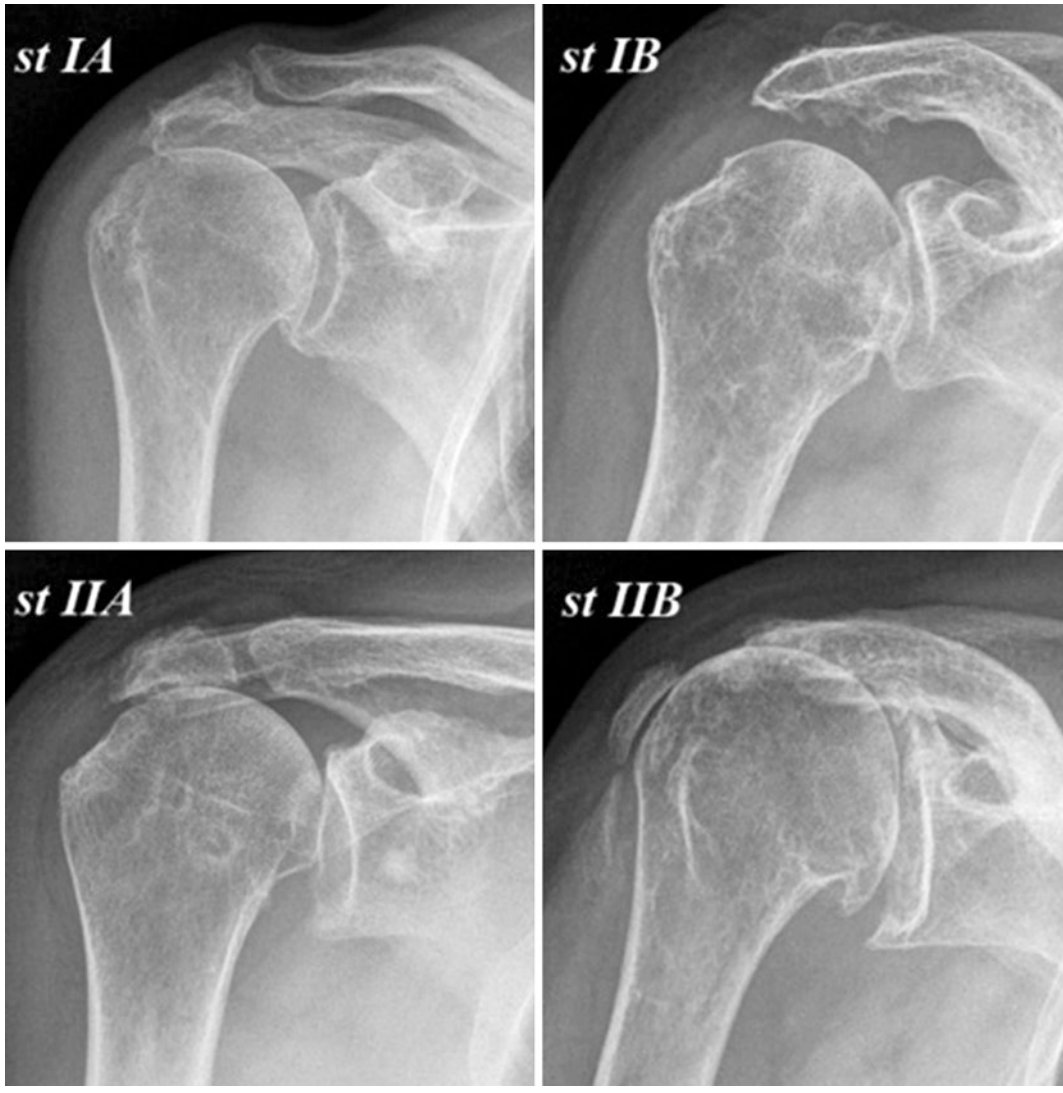


Fig. 12.6 Seebauer classification of rotator cuff arthropathy. Type IA is characterized as centered and stable. Type IB is characterized as centered and medialized. Type IIA is characterized as decentered, limited, and stable. Type IIB is characterized as decentered and unstable. (Reproduced from Domiziano Coppacchioli et al. [7])

12.3.3 Complete or Partial Repair

Henry et al. performed a meta-analysis of 954 patients with chronic MRCT with a mean age of 63, who underwent either complete (81%) or partial (19%) arthroscopic rotator cuff repair. The results of this systematic review demonstrate that either complete or partial arthroscopic repair is associated with an improvement in pain, range of motion, and functional outcome scores. However, on imaging, the retear rate of 79% is considered high. Moreover, reasonable short-term results are reported in literature with partial repair and in the absence of healing [10, 25, 26]. Thus, a chronic MRCT even in elderly patients in whom the probability of achieving complete anatomic repair and healing are not promising may not be considered a limitation to attempt an RCT repair. Other authors demonstrated that the presence of a retear negatively affected the clinical outcomes following rotator cuff repair

and refute the widely held belief that patients typically perform well regardless of the repair integrity following rotator cuff repair [27].

12.3.4 Superior Capsule Reconstruction

Patients in whom rotator cuff is irreparable without advanced cuff arthropathy, partial repair may be an option [28]. However, alternative modern treatments include superior capsule reconstruction that was originally described by Mihata et al. in 2013 [29] and similar, recently described, techniques such as superior capsule reconstruction with autograft such as fascia lata [30], semitendinosus [31] and biceps tendons [32], or synthetic graft [33]. Significant improvements have been noticed over preoperative levels in the short-term follow-up regardless of whether autografts or allograft were used. However, structural repair failures seem to be more common in allografts in comparison to autografts with negative effects on the final clinical results [34–36]. Favorable short-term outcomes have been reported after superior capsule reconstruction [34–39] and should be an available technique to our armamentarium in order to expand the limits of repair.

12.3.5 Reverse Shoulder Arthroplasty and Limitations

Patients with a massive irreparable rotator cuff tear without glenohumeral arthritis can be considered as candidates for a reverse shoulder arthroplasty with improvement in the range of motion, functional scores and in reduction of pain. However, the reported complication rates range between 17% and 20% [24, 40]. A systematic review of the literature by Petrillo et al. evaluated 408 reverse shoulder arthroplasties in 396 patients that were all performed for either rotator cuff tear arthropathy or massive, irreparable rotator cuff tear. Statistically significant improvement was observed in all clinical outcomes according to scores and improved range of motion, but they also found an overall complication rate of 17.4%, resulting in a revision rate of 7.3% [41].

High preoperative functionality is a poor prognostic factor for treatment with arthroplasty especially in patients with high simple shoulder test score (SST > 7) and high ASES [24, 42]. Werner et al. observed a large group of patients who underwent RSA with baseline and minimum 2-year follow-up outcome data to evaluate the association of patient-related factors with poor postoperative improvement after RSA. They reported that surrogates for better preoperative function, such as a higher baseline ASES score and intact rotator cuff at the time of surgery, correlated with poor postoperative improvement after RSA. Furthermore, male sex, depression, and the total number of medical comorbidities also correlated with poor postoperative improvement [42]. Hartzler et al. found that young age (<60 years), preoperative upper extremity neurologic dysfunction, and high preoperative function (SST > 7) were independent risk factors for poor functional improvement after RSA [24]. Finally, Boileau et al. reported that RSA in patients with active anterior elevation greater than 90° preoperatively risks lower patient satisfaction and a reduction in active anterior elevation postoperatively [43].

12.3.6 Revision Surgery

Previous failed rotator cuff surgeries can limit revision rotator cuff repair surgery due to lack of good functional outcomes after 6 months, high retear rates, increased pain with daily activities, lower activity level, and decreased overall satisfaction 2 years postoperatively compared to primary cuff repair surgeries [44]. Additionally, a revision surgery can reverse pesudoparalysis to less than 50% of patients with massive rotator cuff tears [45]. However, a previous repair attempt is not a negative prognostic factor in terms of outcomes and survivor rates for reverse shoulder arthroplasty [46]. Severe atrophy and fatty infiltration and in general poor tissue quality lead to poor results after an attempt for repair [47–49]. Severe instability

in patients with massive rotator cuff tear appear as anterosuperior escape in clinical examination while the patients trying to abduct the arm could indicate reverse arthroplasty even in the absence of arthritis in radiographs [16].

12.3.7 Pseudoparalysis

Pseudoparalysis should not be considered as an unwalkable limit for repair. Initial arthroscopic rotator cuff repair and superior capsule reconstruction have been shown to be effective in patients with pseudoparalysis. More specifically, Mihata et al. [50] published a case series of patients with irreparable rotator cuff tears who underwent arthroscopic SCR with fascia lata autografts and reported that pseudoparalysis was reversed in 96% of patients with preoperative moderate pseudoparalysis and 93% with preoperative severe pseudoparalysis. Denard et al. concluded that arthroscopic rotator cuff repair of MRCT with advanced mobilization techniques can lead to reverse of preoperative pseudoparalysis in 90% of patients who have not had previous surgery. However, in the setting of a revision arthroscopic rotator cuff repair and pseudoparalysis, only 43% of patients regained forward flexion above 90° [45]. Moreover, Oh et al. reported recovery from pseudoparalysis after rotator cuff repair in 76% of patients while postoperative function and cuff healing were not observed to be different according to the presence of pseudoparalysis. They also concluded that due to possible complications and longevity of RSA, rotator cuff repair should be the first-line treatment option for large to massive tears regardless of the presence of pseudoparalysis, and reparability should be confirmed intraoperatively and not judged solely based on preoperative criteria [51]. On the other hand, other authors mentioned that a patient being considered for RSA should have a painful, irreparable rotator cuff tear and evidence of pseudoparalysis with active forward elevation less than 90° [40, 52].

12.3.8 Repair Is a More Cost-Effective Initial Treatment

Makhni et al. [53] analyzed the cost effectiveness of RSA versus arthroscopic rotator cuff repair in patients over 65 years with symptomatic large or massive rotator cuff tear without arthropathy and found that arthroscopic rotator cuff repair despite high rates of tendon retearing can be a more cost-effective initial treatment strategy when compared to primary RSA and when assuming no detrimental impact of previous surgery on outcomes after arthroplasty. Due to similar clinical outcomes in patients after rotator cuff repair (with or without subsequent retear) as well as RSA, rotator cuff repair represented a more cost-effective option because of the higher cost of implants and in-patient hospital stay with RSA. The same analysis found that only under high progression rates from symptomatic retear to end-stage CTA (and in the base case of 68.5% retear likelihood after repair) was RSA, a cost-effective primary treatment option. More specifically, RSA is only more cost-effective than rotator cuff repair if nine of ten (approximately 90%) patients who retear after repair progress to CTA. Finally, they also found that in patients with retear after rotator cuff repair, RSA is the preferred treatment modality at ASES if they are aged 55–69 or lower.

12.4 Conclusion

The limits for repair in young, healthy patients with MRCT can be expanded in the absence of arthritis. The best candidates are patients with reparable tendons and good healing potential without comorbidities and with high preoperative function and active anterior elevation greater than 90°. However, pseudoparalysis should not be considered as an unwalkable limit for repair. Previous failed rotator cuff surgeries and severe anterosuperior instability can set limitations for

repair. Superior capsular reconstruction may be a good option for patients without arthritis with irreparable MRCT and those who suffer from pseudoparalysis and isolated anterosuperior escape. Despite the good outcomes after RSA, we must set a limitation against its wide use especially in patients without arthropathy due to potential complications and implant survivability and less cost-effectiveness. Each treatment must be individualized for each patient. Future studies should closely examine the long-term functional outcomes after both arthroscopic repair and RSA for MRCT and early stages of rotator cuff arthropathy.

Conflict of Interest Emmanouil Antonogiannakis and Grigorios Avramidis declare that they have no conflict of interest.

References

1. Neer CS II, Craig EV, Fukuda H. Cuff-tear arthropathy. J Bone Joint Surg Am. 1983;65(9):1232–44. https://doi.org/10.2106/00004623-198365090-00003.
2. Codman EA. The shoulder: rupture of the supraspinatus tendon and other lesions in or about the subacromial bursa. Boston: Thomas Todd; 1934.
3. Hamada K, Fukuda H, Mikasa M, Kobayashi Y. Roentgenographic findings in massive rotator cuff tears. A long-term observation. Clin Orthop Relat Res. 1990;(254):92–6.
4. Brolin TJ, Updegrove GF, Horneff JG. Classifications in brief: Hamada classification of massive rotator cuff tears. Clin Orthop Relat Res. 2017;475(11):2819–23. https://doi.org/10.1007/s11999-017-5340-7.
5. Walch G, Edwards B, Boulahia A, Nove-Josserand L, Neyton L, Szabo I. Arthroscopic tenotomy of the long head of the biceps in the treatment of rotator cuff tears: clinical and radiographic results of 307 cases. J Shoulder Elb Surg. 2005;14(3):238–46. https://doi.org/10.1016/j.jse.2004.07.008.
6. Sirveaux F, Favard L, Oudet D, Huquet D, Walch G, Mole D. Grammont inverted total shoulder arthroplasty in the treatment of glenohumeral osteoarthritis with massive rupture of the cuff. Results of a multicentre study of 80 shoulders. J Bone Joint Surg Br. 2004;86(3):388–95.
7. Coppacchioli D, Ponte G, Mangano T. CTA: instrumental evaluation. In: Gumina S, Grassi FA, Paladini P, editors. Reverse shoulder arthroplasty. Cham: Springer; 2018. p. 101–14.
8. Visotsky JL, Basamania C, Seebauer L, Rockwood CA, Jensen KL. Cuff tear arthropathy: pathogenesis, classification, and algorithm for treatment. J Bone Joint Surg Am. 2004;86(Suppl 2):35–40.
9. Boileau P, Brassart N, Watkinson DJ, Carles M, Hatzidakis AM, Krishnan SG. Arthroscopic repair of full-thickness tears of the supraspinatus: does the tendon really heal? J Bone Joint Surg Am. 2005;87(6):1229–40.
10. Chung SW, Kim JY, Kim MH, Kim SH, Oh JH. Arthroscopic repair of massive rotator cuff tears: outcome and analysis of factors associated with healing failure or poor postoperative function. Am J Sports Med. 2013;41(7):1674–83. https://doi.org/10.1177/0363546513485719.
11. Goutallier D, Postel JM, Gleyze P, Leguilloux P, Van Driessche S. Influence of cuff muscle fatty degeneration on anatomic and functional outcomes after simple suture of full-thickness tears. J Shoulder Elb Surg. 2003;12(6):550–4.
12. Meyer DC, Wieser K, Farshad M, Gerber C. Retraction of supraspinatus muscle and tendon as predictors of success of rotator cuff repair. Am J Sports Med. 2012;40(10):2242–7. https://doi.org/10.1177/0363546512457587.
13. Rhee YG, Cho NS, Yoo JH. Clinical outcome and repair integrity after rotator cuff repair in patients older than 70 years versus patients younger than 70 years. Arthroscopy. 2014;30(5):546–54. https://doi.org/10.1016/j.arthro.2014.02.006.
14. Wu XL, Briggs L, Murrell GA. Intraoperative determinants of rotator cuff repair integrity: an analysis of 500 consecutive repairs. Am J Sports Med. 2012;40(12):2771–6. https://doi.org/10.1177/0363546512462677.
15. Mall NA, Tanaka MJ, Choi LS, Paletta GA Jr. Factors affecting rotator cuff healing. J Bone Joint Surg Am. 2014;96(9):778–88.
16. Harreld KL, Puskas BL, Frankle M. Massive rotator cuff tears without arthropathy: when to consider reverse shoulder arthroplasty. J Bone Joint Surg Am. 2011;93(10):973–84.
17. Mallon WJ, Misamore G, Snead DS, Denton P. The impact of preoperative smoking habits on the results of rotator cuff repair. J Shoulder Elb Surg. 2004;13(2):129–32. https://doi.org/10.1016/j.jse.2003.11.002.
18. Tashjian RZ, Hollins AM, Kim HM, Teefey SA, Middleton WD, Steger-May K, et al. Factors affecting healing rates after arthroscopic double-row rotator cuff repair. Am J Sports Med. 2010;38(12):2435–42. https://doi.org/10.1177/0363546510382835.
19. Meyer DC, Farshad M, Amacker NA, Gerber C, Wieser K. Quantitative analysis of muscle and tendon retraction in chronic rotator cuff tears. Am J Sports Med. 2012;40(3):606–10. https://doi.org/10.1177/0363546511429778.

20. Muh SJ, Streit JJ, Wanner JP, Lenarz CJ, Shishani Y, Rowland DY, et al. Early follow-up of reverse total shoulder arthroplasty in patients sixty years of age or younger. J Bone Joint Surg Am. 2013;95(20):1877–83. https://doi.org/10.2106/JBJS.L.10005.
21. Ek ET, Neukom L, Catanzaro S, Gerber C. Reverse total shoulder arthroplasty for massive irreparable rotator cuff tears in patients younger than 65 years old: results after five to fifteen years. J Shoulder Elb Surg. 2013;22(9):1199–208. https://doi.org/10.1016/j.jse.2012.11.016.
22. Samuelsen BT, Wagner ER, Houdek MT, Elhassan BT, Sanchez-Sotelo J, Cofield R, et al. Primary reverse shoulder arthroplasty in patients aged 65 years or younger. J Shoulder Elb Surg. 2017;26(1):e13–7. https://doi.org/10.1016/j.jse.2016.05.026.
23. Sershon RA, Van Thiel GS, Lin EC, McGill KC, Cole BJ, Verma NN, et al. Clinical outcomes of reverse total shoulder arthroplasty in patients aged younger than 60 years. J Shoulder Elb Surg. 2014;23(3):395–400. https://doi.org/10.1016/j.jse.2013.07.047.
24. Hartzler RU, Steen BM, Hussey MM, Cusick MC, Cottrell BJ, Clark RE, et al. Reverse shoulder arthroplasty for massive rotator cuff tear: risk factors for poor functional improvement. J Shoulder Elb Surg. 2015;24(11):1698–706. https://doi.org/10.1016/j.jse.2015.04.015.
25. Holtby R, Razmjou H. Relationship between clinical and surgical findings and reparability of large and massive rotator cuff tears: a longitudinal study. BMC Musculoskelet Disord. 2014;15(1):180. https://doi.org/10.1186/1471-2474-15-180.
26. Namdari S, Donegan RP, Chamberlain AM, Galatz LM, Yamaguchi K, Keener JD. Factors affecting outcome after structural failure of repaired rotator cuff tears. J Bone Joint Surg Am. 2014;96(2):99–105. https://doi.org/10.2106/JBJS.M.00551.
27. Kim HM, Caldwell JM, Buza JA, Fink LA, Ahmad CS, Bigliani LU, et al. Factors affecting satisfaction and shoulder function in patients with a recurrent rotator cuff tear. J Bone Joint Surg Am. 2014;96(2):106–12. https://doi.org/10.2106/JBJS.L.01649.
28. Burkhart SS, Nottage WM, Ogilvie-Harris DJ, Kohn HS, Pachelli A. Partial repair of irreparable rotator cuff tears. Arthroscopy. 1994;10(4):363–70.
29. Mihata T, McGarry MH, Pirolo JM, Kinoshita M, Lee TQ. Superior capsule reconstruction to restore superior stability in irreparable rotator cuff tears: a biomechanical cadaveric study. Am J Sports Med. 2012;40:2248–55. https://doi.org/10.1177/0363546512456195.
30. de Campos Azevedo CI, Ângelo ACLPG, Vinga S. Arthroscopic superior capsular reconstruction with a minimally invasive harvested fascia lata autograft produces good clinical results. Orthop J Sports Med. 2018;6(11):2325967118808242.
31. Rosales-Varo AP, Zafra M, García-Espona MA, Flores-Ruiz MA, Roda O. Superior capsular reconstruction of irreparable rotator cuff tear using autologous hamstring graft. Rev Esp Cir Ortop Traumatol. 2019;63(1):1–6. https://doi.org/10.1016/j.recote.2018.12.006.
32. Veen EJD, Stevens M, Diercks RL. Biceps autograft augmentation for rotator cuff repair: a systematic review. Arthroscopy. 2018;34(4):1297–305. https://doi.org/10.1016/j.arthro.2017.10.044.
33. Hirahara AM, Adams CR. Arthroscopic superior capsular reconstruction for treatment of massive irreparable rotator cuff tears. Arthrosc Tech. 2015;4(6):e637–41. https://doi.org/10.1016/j.eats.2015.07.006.
34. Denard PJ, Brady PC, Adams CR, Tokish JM, Burkhart SS. Preliminary results of arthroscopic superior capsule reconstruction with dermal allograft. Arthroscopy. 2018;34(1):93–9. https://doi.org/10.1016/j.arthro.2017.08.265.
35. Hirahara AM, Andersen WJ, Panero AJ. Superior capsular reconstruction: clinical outcomes after minimum 2-year follow-up. Am J Orthop. 2017;46(6):266–78.
36. Mihata T, Lee TQ, Watanabe C, Fukunishi K, Ohue M, Tsujimura T, et al. Clinical results of arthroscopic superior capsule reconstruction for irreparable rotator cuff tears. Arthroscopy. 2013;29(3):459–70.
37. Gupta AK, Hug K, Berkoff DJ, Boggess BR, Gavigan M, Malley PC, et al. Dermal tissue allograft for the repair of massive irreparable rotator cuff tears. Am J Sports Med. 2012;40(1):141–7. https://doi.org/10.1177/0363546511422795.
38. Mihata T, McGarry MH, Pirolo JM, Kinoshita M, Lee TQ. Superior capsule reconstruction to restore superior stability in irreparable rotator cuff tears: a biomechanical study. Am J Sports Med. 2012;40(10):2248–55. https://doi.org/10.1177/0363546512456195.
39. Plachel F, Klatte-Schulz F, Minkus M, Böhm E, Moroder P, Scheibel M. Biological allograft healing after superior capsule reconstruction. J Shoulder Elb Surg. 2018;27(12):e387–92. https://doi.org/10.1016/j.jse.2018.08.029.
40. Mulieri P, Dunning P, Klein S, Pupello D, Frankle M. Reverse shoulder arthroplasty for the treatment of irreparable rotator cuff tear without glenohumeral arthritis. J Bone Joint Surg Am. 2010;92(15):2544–56. https://doi.org/10.2106/JBJS.I.00912.
41. Petrillo S, Longo UG, Papalia R, Denaro V. Reverse total shoulder arthroplasty for massive irreparable rotator cuff tears and cuff tear arthropathy: a systematic review. Musculoskelet Surg. 2017;101(2):105–12. https://doi.org/10.1007/s12306-017-0474-z.
42. Werner BC, Wong AC, Mahony GT, Craig EV, Dines DM, Warren RF, et al. Causes of poor postoperative improvement after reverse total shoulder arthroplasty. J Shoulder Elb Surg. 2016;25(8):e217–22. https://doi.org/10.1016/j.jse.2016.01.002.
43. Boileau P, Gonzalez JF, Chuinard C, Bicknell R, Walch G. Reverse total shoulder arthroplasty after failed rotator cuff surgery. J Shoulder Elb Surg. 2009;18(4):600–6. https://doi.org/10.1016/j.jse.2009.03.011.
44. Shamsudin A, Lam PH, Peters K, Rubenis I, Hackett L, Murrell GA. Revision versus primary arthroscopic

rotator cuff repair: a 2-year analysis of outcomes in 360 patients. Am J Sports Med. 2015;43(3):557–64. https://doi.org/10.1177/0363546514560729.

45. Denard PJ, Ladermann A, Jiwani AZ, Burkhart SS. Functional outcome after arthroscopic repair of massive rotator cuff tears in individuals with pseudoparalysis. Arthroscopy. 2012;28(9):1214–9. https://doi.org/10.1016/j.arthro.2012.02.026.
46. Sadoghi P, Vavken P, Leithner A, Hochreiter J, Weber G, Pietschmann MF, et al. Impact of previous rotator cuff repair on the outcome of reverse shoulder arthroplasty. J Shoulder Elb Surg. 2011;20(7):1138–46. https://doi.org/10.1016/j.jse.2011.01.013.
47. Gladstone JN, Bishop JY, Lo IK, Flatow EL. Fatty infiltration and atrophy of the rotator cuff do not improve after rotator cuff repair and correlate with poor functional outcome. Am J Sports Med. 2007;35(5):719–28. https://doi.org/10.1177/0363546506297539.
48. Shen PH, Lien SB, Shen HC, Lee CH, Wu SS, Lin LC. Long-term functional outcomes after repair of rotator cuff tears correlated with atrophy of the supraspinatus muscles on magnetic resonance images. J Shoulder Elb Surg. 2008;17(Suppl 1):1S–7S. https://doi.org/10.1016/j.jse.2007.04.014.
49. Yoo JC, Ahn JH, Koh KH, Lim KS. Rotator cuff integrity after arthroscopic repair for large tears with less-than-optimal footprint coverage. Arthroscopy. 2009;25(10):1093–100. https://doi.org/10.1016/j.arthro.2009.07.010.
50. Mihata T, Lee TQ, Hasegawa A, Kawakami T, Fukunishi K, Fujisawa Y, Itami Y, Ohue M, Neo M. Arthroscopic superior capsule reconstruction can eliminate pseudoparalysis in patients with irreparable rotator cuff tears. Am J Sports Med. 2018;46(11):2707–16. https://doi.org/10.1177/0363546518786489.
51. Oh JH, Kim SH, Shin SH, et al. Outcome of rotator cuff repair in large-to-massive tear with pseudoparalysis: a comparative study with propensity score matching. Am J Sports Med. 2011;39(7):1413–20. https://doi.org/10.1177/0363546511399865.
52. Harreld KL, Puskas BL, Frankle M. Massive rotator cuff tears without arthropathy: when to consider reverse shoulder arthroplasty. Instr Course Lect. 2012;61:143–56.
53. Makhni EC, Swart E, Steinhaus ME, Mather RC III, Levine WN, Bach BR Jr, et al. Cost-effectiveness of reverse total shoulder arthroplasty versus arthroscopic rotator cuff repair for symptomatic large and massive rotator cuff tears. Arthroscopy. 2016;32(9):1771–80. https://doi.org/10.1016/j.arthro.2016.01.063.

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

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_13

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

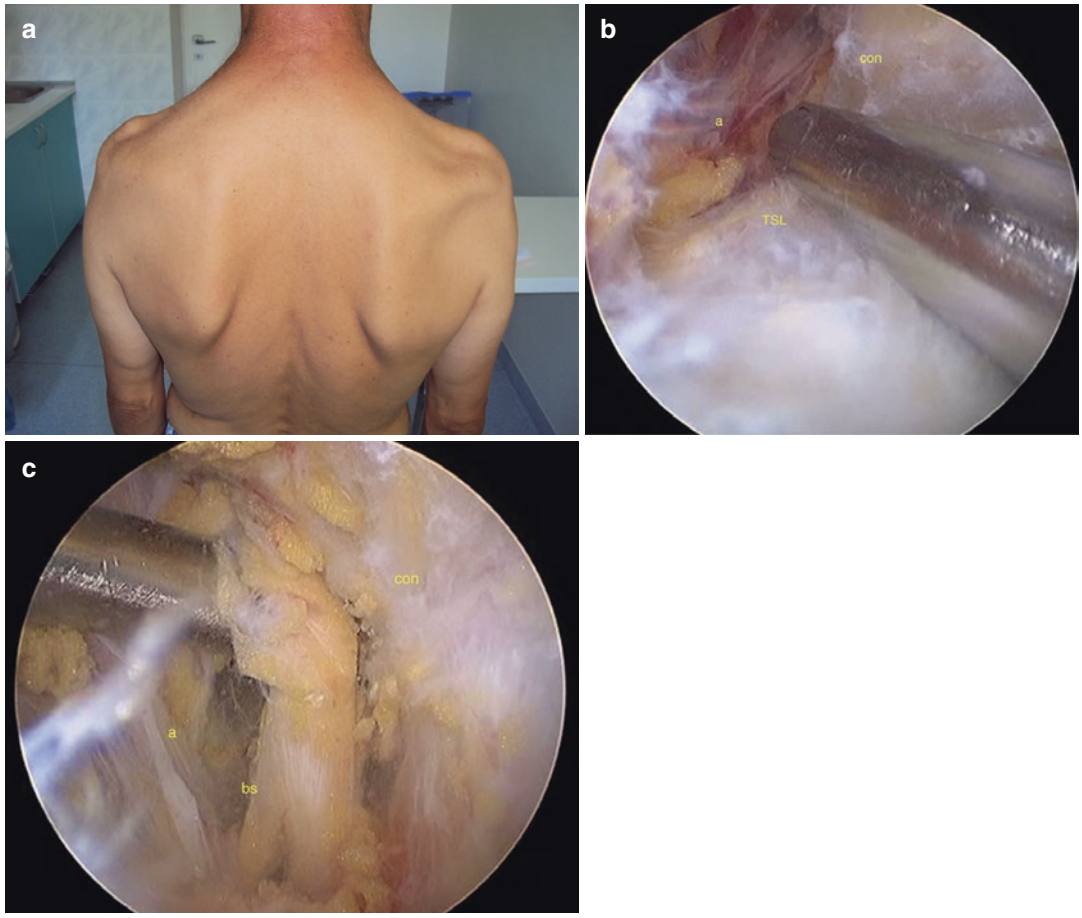


Fig. 13.1 The SSN pathology at the suprascapular notch. (**a**) The SSN disturbances in a patient with massive rotator cuff lesion. Clinical image of the supraspinatus and the infraspinatus muscles atrophy is the same as in the SSN compression at the suprascapular notch. (**b**) Arthroscopic view of 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)

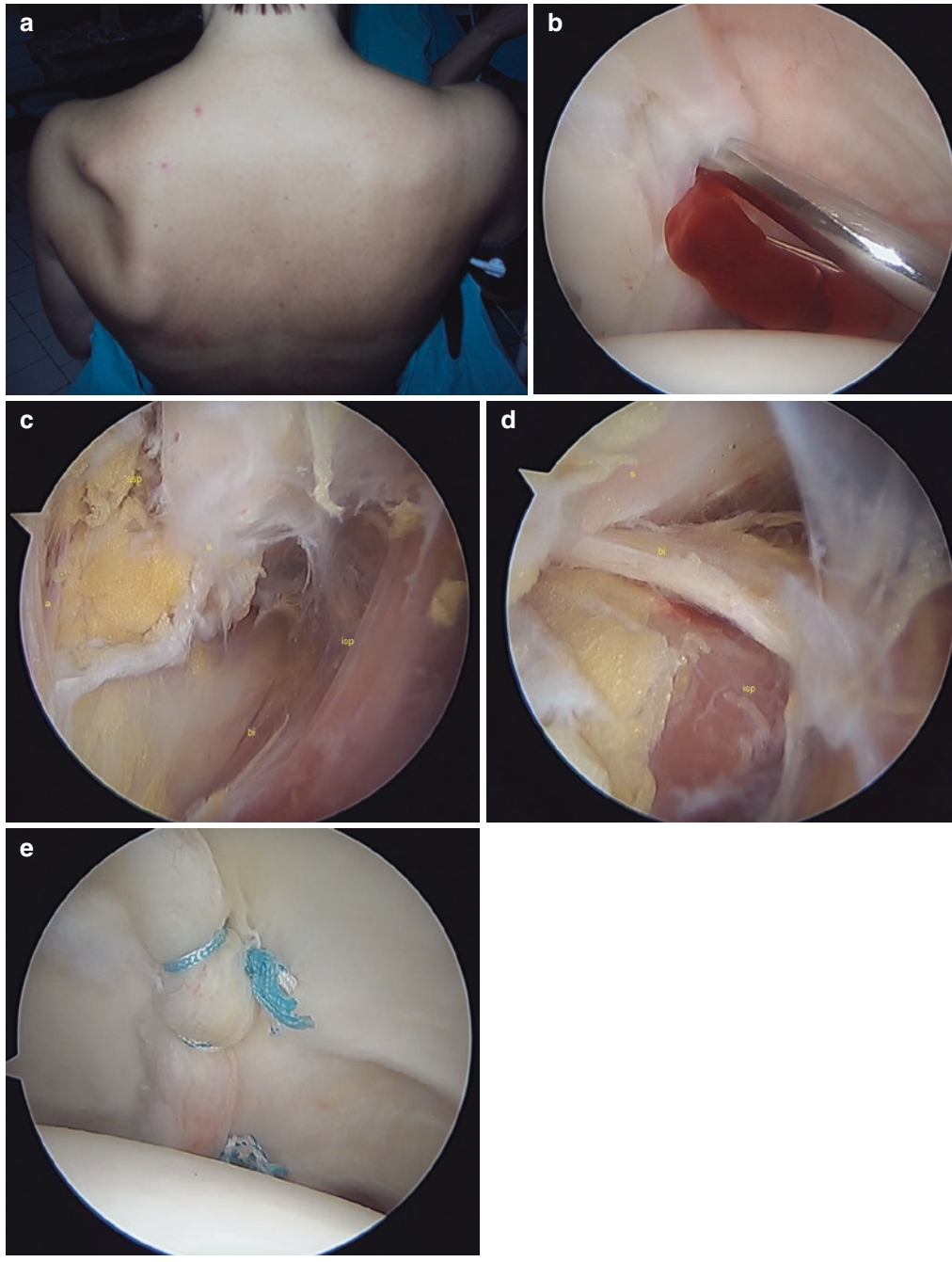


Fig. 13.2 The SSN pathology at the spinoglenoid notch. (**a**) The infraspinatus insufficiency—clinical image of the patient with a spinoglenoid cyst. (**b**) Arthroscopic intraarticular view, left shoulder, beach chair position, arthroscope in the anterolateral portal. Spinoglenoid cyst evacuation, 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.

References

1. Momaya AM, Kwapisz A, Choate WS, Kissenberth MJ, Tolan SJ, Lonergan KT, Hawkins RJ, Tokish JM. Clinical outcomes of suprascapular nerve decompression: a systematic review. J Shoulder Elb Surg. 2018;27:172–80.
2. Tsikouris GD, Bolia IK, Vlaserou P, Odantzis N, Angelis K, Psychogios V. Shoulder arthroscopy with versus without suprascapular nerve release: clinical outcomes and return to sport rate in elite overhead athletes. Arthroscopy. 2018;34:2552–7.
3. Denard PJ. Editorial commentary: Suprascapular neuropathy in overhead athletes: to release or not to release? Arthroscopy. 2018;34:2558–9.
4. Massimini DF, Singh A, Wells JH, Li G, Warner JJP. Suprascapular nerve anatomy during shoulder motion: a cadaveric proof of concept study with implications for neurogenic shoulder pain. J Shoulder Elb Surg. 2013;22:463–70.
5. Albritton MJ, Graham RD, Richards RS II, Basamania CJ. An anatomic study of the effects on the suprascapular nerve due to retraction of the supraspinatus muscle after a rotator cuff tear. J Shoulder Elb Surg. 2003;12:497–500.
6. Polguj M, Sibinski M, Grzegorzewski A, Grzelak P, Majos A, Topol M. Variation in morphology of suprascapular notch as a factor of suprascapular nerve entrapment. Int Orthop. 2013;37:2185–92.
7. Cummins CA, Messer TM, Schafer MF. Infraspinatus muscle atrophy in professional baseball players. Am J Sports Med. 2004;32:116–20.
8. Savoie FH III, Zunkiewicz M, Field LD, Replogle WH, O'Brien MJ. A comparison of functional outcomes in patients undergoing revision arthroscopic repair of massive rotator cuff tears with and without arthroscopic suprascapular nerve release. Open Access J Sport Med. 2016;7:129–34.
9. Shi LL, Boykin RE, Lin A, Warner JJP. Association of suprascapular neuropathy with rotator cuff tendon tears and fatty degeneration. J Shoulder Elb Surg. 2014;23:339–46.
10. Oizumi N, Suenaga N, Funakoshi T, Yamaguchi H, Minami A. Recovery of sensory disturbance after arthroscopic decompression of the suprascapular nerve. J Shoulder Elb Surg. 2012;21:759–64.
11. Plancher KD, Petterson SC. Posterior shoulder pain and arthroscopic decompression of the suprascapular nerve at the spinoglenoid notch. Oper Tech Sports Med. 2014;22:73–87.
12. Dörrien. Ueber Lähmung des N. suprascapularis. [About paralysis of the suprascapular nerve]. Dtsch Med Wochenschr. 1908;34:1345–6.
13. Kopell HP, Thompson WA. Pain and the frozen shoulder. Surg Gynecol Obstet. 1959;109:92–6.
14. Aiello I, Serra G, Traina GC, Tugnoli V. Entrapment of the suprascapular nerve at the spinoglenoid notch. Ann Neurol. 1982;12:314–6.

15. Mallon WJ, Bronec PR, Spinner RJ, Levin LS. Suprascapular neuropathy after distal clavicle excision. Clin Orthop Relat Res. 1996;329:207–11.
16. Edeland HG, Zachrisson BE. Fracture of the scapular notch associated with lesion of the suprascapular nerve. Acta Orthop Scand. 1975;46:758–63.
17. Parsonage MJ, Turner JWA. Neuralgic amyotrophy; the shoulder-girdle syndrome. Lancet (London, England). 1948;1:973–8.
18. Feinberg JH, Radecki J. Parsonage-turner syndrome. HSS J. 2010;6:199–205.
19. Leclere LE, Shi LL, Lin A, Yannopoulos P, Higgins LD, Warner JJP. Complete fatty infiltration of intact rotator cuffs caused by suprascapular neuropathy. Arthroscopy. 2014;30:639–44.
20. Plancher KD, Johnston JC, Peterson RK, Hawkins RJ. The dimensions of the rotator interval. J Shoulder Elb Surg. 2005;14:620–5.
21. Kong BY, Kim SH, Kim DH, Joung HY, Jang YH, Oh JH. Suprascapular neuropathy in massive rotator cuff tears with severe fatty degeneration in the infraspinatus muscle. Bone Joint J. 2016;98-B:1505–9.
22. Warner JP, Krushell RJ, Masquelet A, Gerber C. Anatomy and relationships of the suprascapular nerve: anatomical constraints to mobilization of the supraspinatus and infraspinatus muscles in the management of massive rotator-cuff tears. J Bone Joint Surg Am. 1992;74:36–45.
23. Greiner A, Golser K, Wambacher M, Kralinger F, Sperner G. The course of the suprascapular nerve in the supraspinatus fossa and its vulnerability in muscle advancement. J Shoulder Elb Surg. 2003;12:256–9.
24. Lafosse L, Piper K, Lanz U. Arthroscopic suprascapular nerve release: indications and technique. J Shoulder Elb Surg. 2011;20:S9–13.
25. Post M, Mayer J. Suprascapular nerve entrapment. Diagnosis and treatment. Clin Orthop Relat Res. 1987;(223):126–36.
26. Nardin RA, Rutkove SB, Raynor EM. Diagnostic accuracy of electrodiagnostic testing in the evaluation of weakness. Muscle Nerve. 2002;26:201–5.
27. Lafosse L, Tomasi A, Corbett S, Baier G, Willems K, Gobezie R. Arthroscopic release of suprascapular nerve entrapment at the suprascapular notch: technique and preliminary results. Arthroscopy. 2007;23:34–42.
28. Tubbs RS, Smyth MD, Salter G, Oakes WJ. Anomalous traversement of the suprascapular artery through the suprascapular notch: a possible mechanism for undiagnosed shoulder pain? Med Sci Monit. 2003;9:BR116–9.
29. Bhatia S, Chalmers PN, Yanke AB, Romeo AA, Verma NN. Arthroscopic suprascapular nerve decompression: transarticular and subacromial approach. Arthrosc Tech. 2012;1:e187–92.
30. Ghodadra N, Nho SJ, Verma NN, Reiff S, Piasecki DP, Provencher MT, Romeo AA. Arthroscopic decompression of the suprascapular nerve at the spinoglenoid notch and suprascapular notch through the subacromial space. Arthroscopy. 2009;25:439. https://doi.org/10.1016/j.arthro.2008.10.024.
31. Shah AA, Butler RB, Sung S-Y, Wells JH, Higgins LD, Warner JJP. Clinical outcomes of suprascapular nerve decompression. J Shoulder Elb Surg. 2011;20:975–82.
32. Garcia Junior JC, Paccola AMF, Tonoli C, Zabeu JLA, Garcia JPM. Arthroscopic release of the suprascapular nerve: surgical technique and evaluation of clinical cases. Rev Bras Ortop. 2011;46:403–7.
33. Kim DH, Murovic JA, Tiel RL, Kline DG. Management and outcomes of 42 surgical suprascapular nerve injuries and entrapments. Neurosurgery. 2005;57:120–7.
34. Kim D-S, Park H-K, Park J-H, Yoon W. Ganglion cyst of the spinoglenoid notch: comparison between SLAP repair alone and SLAP repair with cyst decompression. J Shoulder Elb Surg. 2012;21:1456–63.
35. Pillai G, Baynes JR, Gladstone J, Flatow EL. Greater strength increase with cyst decompression and SLAP repair than SLAP repair alone. Clin Orthop Relat Res. 2011;469:1056–60.
36. Costouros JG, Porramatikul M, Lie DT, Warner JJP. Reversal of suprascapular neuropathy following arthroscopic repair of massive supraspinatus and infraspinatus rotator cuff tears. Arthroscopy. 2007;23:1152–61.
37. Aramberri M. Arthroscopic treatment of the shoulder cuff tears. Barcelona: Autonomic University in Barcelona; 2011.
38. Lajtai G, Pfirrmann CWA, Aitzetmuller G, Pirkl C, Gerber C, Jost B. The shoulders of professional beach volleyball players: high prevalence of infraspinatus muscle atrophy. Am J Sports Med. 2009;37:1375–83.
39. Lajtai G, Wieser K, Ofner M, Raimann G, Aitzetmuller G, Jost B. Electromyography and nerve conduction velocity for the evaluation of the infraspinatus muscle and the suprascapular nerve in professional beach volleyball players. Am J Sports Med. 2012;40:2303–8.

14 Traumatic Cuff Tears: The Relevance of Timing

Francesco Franceschi, Pietro Gregori, and Edoardo Franceschetti

14.1 Epidemiology

An annual incidence of acute full-thickness rotator cuff tears (FTRCTs) is estimated to be 25 per 100,000 population aged 40–75 years old [1]. Furthermore, acute FTRCTs are common in the male population following simple falls [1]. While the overall average of FTRCTs is around the 60s, acute cuff tears due to a traumatic event can occur in younger ages, finding its mean age around 34 (Fig. 14.1) [2]. It is also suspected that there is a high risk of missed diagnosis, which could alter data collected until now. Most frequent injury pattern is a direct trauma, such as fall, often onto an outstretched arm [3].

14.2 Clinical Features

Patients with no signs of fractures or dislocations are often discharged from the emergency department with no further investigation. Only much later, the patient will be referred to a specialist if the disability and pain continue. Usually previously asymptomatic patients identify a traumatic incident leading to a sudden onset of symptoms such as severe pain, immediate loss of strength, and functional impairment of the shoulder. Missed diagnosis is also common because small lesions tend to be more painful than FTRCTs and pain seems to be related more to the degree of bursitis than to the degree of tearing [4].

14.3 Physical Examination

Traumatic lesions of the rotator cuff may often be missed on the first clinical examination because of minor physical findings dominated by pain. A detailed history and a proper clinical examination improve the early diagnosis of traumatic rotator cuff tears [5]. The clinical examination includes the assessment of passive and active range of motion, strength of the rotator cuff muscles, ability or inability to hold the arm in desired position (lag sign), and additional assessment of the subacromial impingement, acromioclavicular joint, and biceps tendon (Table 14.1). Additionally, since the pain following a traumatic event could influence the test performing, a subacromial injection of anesthetic (10 mL of 1% lidocaine) could be administrated to discriminate true positive lag signs from false positives due to sorrow.

F. Franceschi · P. Gregori · E. Franceschetti (✉)
Università Campus Bio-Medico di Roma,
Roma (IT), Italy
e-mail: f.franceschi@unicampus.it;
e.franceschetti@unicampus.it

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_14

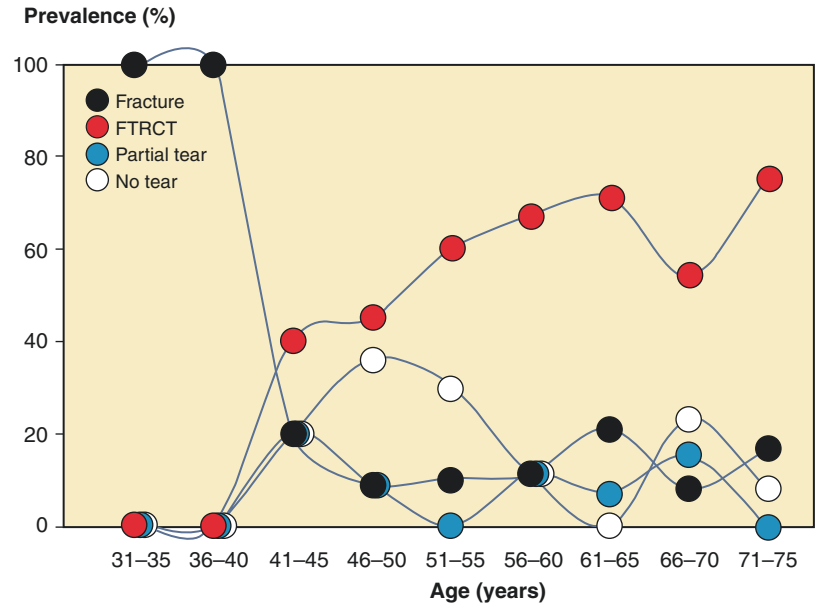


Fig. 14.1 Prevalence of MRI-verified lesions related to patient age

Table 14.1 Most accurate clinical test performed in a patient with full-thickness rotator cuff tear

Passive and active range of movement	Abduction in the scapular plane Forward flexion Internal and external rotation at 0° and 90° of abduction
Strength tests	Jobe test Test of external–internal rotation Lift off test
Lag signs	External rotation lag sign Infraspinatus drop test Drop-arm test Internal rotation lag sign
Impingement signs	Neer test Hawkins Painful arc
Tests of the acromioclavicular joint	Tenderness of the joint Cross-body test
Biceps tendon tests	Yergason and speed

14.4 Imaging

Magnetic resonance imaging has previously been considered the most precise diagnostic tool for chronic cases of FTRCTs, but during the last decade due to further improvements in equipment and technique, similar results have been shown with sonography, with sensitivity and specificity ranging from 80 to 100% for both modalities [6]. Fatty infiltration and muscle atrophy are the most used patterns to evaluate prognosis, and clinical reports suggest that fatty degeneration and muscle atrophy seen with delayed surgery are related to lower postoperative scores, both UCLA and Constant score [7]. Acute FTRCTs often occur on a pre-injured tendon. The presence of fatty infiltration within the muscles of rotator cuff classified according to Goutallier gives us precious information about the type and chronicity of the injury (acute event on a pre-injury tendon versus acute event on a healthy tendon) and thus the long-term prognosis (Fig. 14.2).

14.5 Treatment Options

14.5.1 Nonoperative Treatement

There is support in the literature for the nonoperative management of rotator cuff tears. Bokor et al. reported that patients presenting for nonoperative treatment within 3 months of their injury had satisfactory outcomes [8]. Successful nonoperative management has been associated with the presence of satisfactory motion and strength at the initiation of treatment. Unfortunately, Itoi and Tabata have demonstrated that the outcome of nonoperative management deteriorates over time,

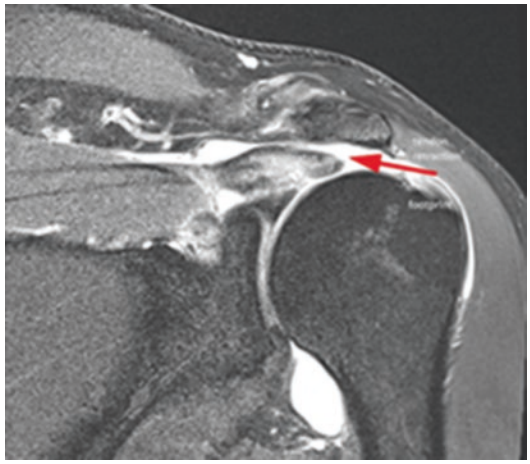


Fig. 14.2 MRI imaging of an acute FTRCT

and Goldberg et al. concluded that the overall response of shoulder function to nonoperative intervention was poor [9, 10].

14.5.2 Operative Treatement

It is well known how frictional attrition of the torn rotator cuff and musculotendinous tissues retraction are minimal immediately after injury [1]. Also scarring about the shoulder is not present, making early repair easier and more secure. Operative treatment is considered to be the first line of treatment in these types of lesions nowadays (Fig. 14.3). There is a trend suggesting that earlier time to surgery may be linked to better

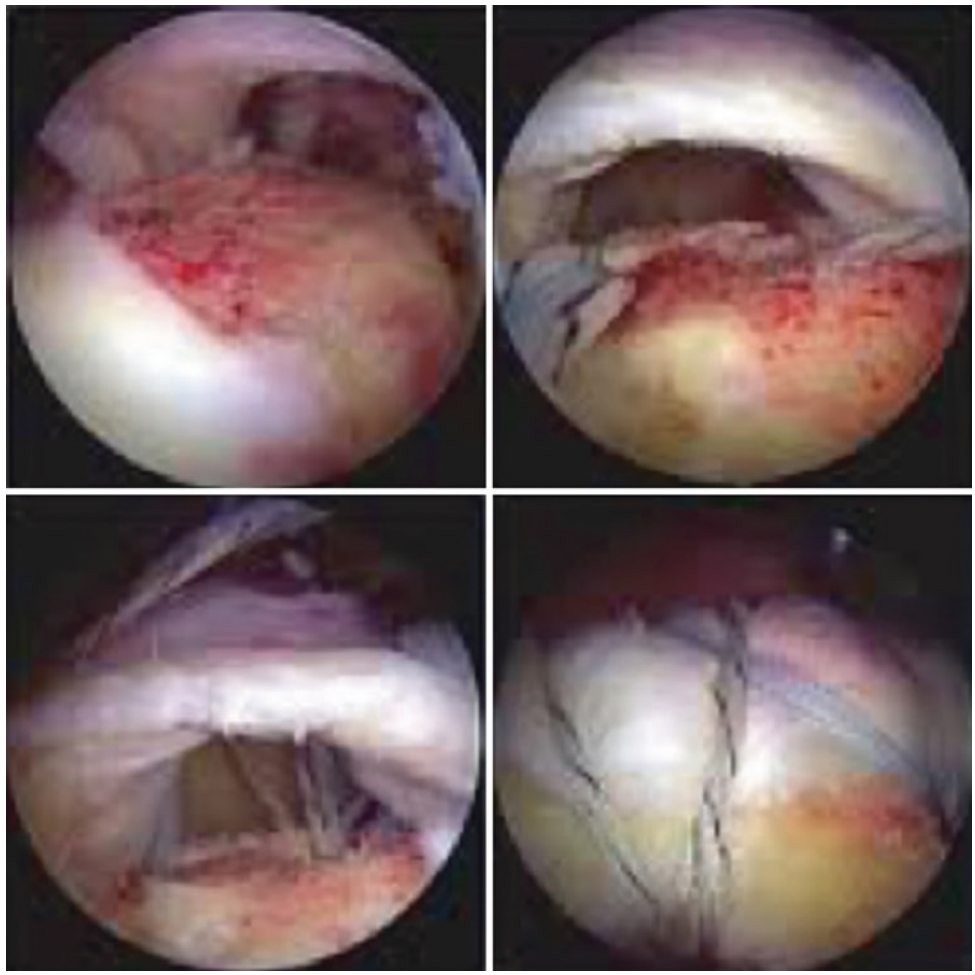


Fig. 14.3 Arthroscopic images of an acute FTRCT double row repair

clinical results regarding Constant scores and range of motion [11]. While an agreement on precise timing for surgery has not been outlined yet, several studies support this theory [11–14].

Gerber et al. reported higher improvement in outcomes in the 13 patients who underwent surgery within 20 months of injury than the 3 patients whose surgeries were delayed more than 36 months after the injury [14]. A study by Petersen and Murphy noted that when compared with those performed longer than 16 weeks after injury, repairs performed prior to 16 weeks from injury were associated with significantly improved active elevation (140° vs. 100°), ASES score (81 vs. 65), and UCLA score [15]. The most comprehensive study, comparing outcomes between early and delayed repair, was conducted by Bassett and Cofield who determined that tendons repaired within 3 weeks had significantly better forward elevation and showed a trend toward better strength in both abduction and external rotation than those repaired after 3 weeks [12]. Additional support for this results has been published by Hantes et al., who reported significantly higher mean postoperative Constant (82) and UCLA (31) scores in the acute repair group (<3 weeks) than in the delayed repair group (70 and 26, respectively) [13]. Even if the precise guidelines for the timing of surgical treatment have not been defined yet, there are some clear data as to how delayed surgery could affect subjective and objective patient's outcomes. Following this, it is undeniable that timely surgery is strictly recommended.

14.6 Summary

An agreement on relevance of timing on successful rotator cuff repair has not been completely defined yet; however, several studies suggest that earlier time to surgery may be linked to better clinical results. Immediately after traumatic tear, rotator cuff tendon retraction is minimal. Additionally, scarring about the shoulder is not present, making early repair easier and more secure. Important prognostic factors for the surgical treatment of these injuries are fatty degeneration and muscle atrophy of the relevant muscles as well. Clinical reports suggest that these changes seen with delayed surgery are related to lower postoperative scores. It is unlikely to see important grade of fatty degeneration and muscle atrophy after the injury. These changes might occur on a pre-injured tendon, thus giving us precious information about the type and chronicity of the injury like acute event on a pre-injury tendon. This observation is therefore important for the long-term prognosis.

References

1. Aagaard KE, Lunsjö K, Frobell R. Early repair of trauma-related full-thickness rotator cuff tears does not eliminate the problem of healing failure. Bone Jt J. 2019;101B:603–9.
2. Braune C, von Eisenhart-Rothe R, Welsch F, Teufel M, Jaeger A. Mid-term results and quantitative comparison of postoperative shoulder function in traumatic and non-traumatic rotator cuff tears. Arch Orthop Trauma Surg. 2003;123:419–24.
3. Namdari S, Henn RF 3rd, Green A. Traumatic anterosuperior rotator cuff tears: the outcome of open surgical repair. J Bone Joint Surg Am. 2008;90:1906–13.
4. Fukuda H. Partial-thickness rotator cuff tears: a modern view on Codman's classic. J Shoulder Elbow Surg. 2000;9:163–8.
5. Sørensen AK, Bak K, Krarup AL, Thune CH, Nygaard M, Jørgensen U, Sloth C, Torp-Pedersen S. Acute rotator cuff tear: do we miss the early diagnosis? A prospective study showing a high incidence of rotator cuff tears after shoulder trauma. J Shoulder Elbow Surg. 2007;16:174–80.
6. Dinnes J, Loveman E, McIntyre L, Waugh N. The effectiveness of diagnostic tests for the assessment of shoulder pain due to soft tissue disorders: a systematic review. Health Technol Assess. 2003;7:iii, 1-166.
7. Warner JJ, Parsons IM 4th. Latissimus dorsi tendon transfer: a comparative analysis of primary and salvage reconstruction of massive, irreparable rotator cuff tears. J Shoulder Elbow Surg. 2001;10:514–21.
8. Bokor DJ, Hawkins RJ, Huckell GH, Angelo RL, Schickendantz MS. Results of nonoperative management of full-thickness tears of the rotator cuff. Clin Orthop Relat Res. 1993;294:103–10.
9. Itoi E, Tabata S. Conservative treatment of rotator cuff tears. Clin Orthop Relat Res. 1992;275:165–73.
10. Goldberg BA, Nowinski RJ, Matsen FA III. Outcome of nonoperative management of full-thickness rotator cuff tears. Clin Orthop Relat Res. 2001;(382):99–107.

11. Mukovozov I, Byun S, Farrokhyar F, Wong I. Time to surgery in acute rotator cuff tear. Bone Jt Res. 2013;2:122–8.
12. Bassett RW, Cofield RH. Acute tears of the rotator cuff. The timing of surgical repair. Clin Orthop Relat Res. 1983;175:18–24.
13. Hantes ME, Karidakis GK, Vlychou M, Varitimidis S, Dailiana Z, Malizos KN. A comparison of early versus delayed repair of traumatic rotator cuff tears. Knee Surg Sports Traumatol Arthrosc. 2011;19:1766–70.
14. Gerber C, Hersche O, Farron A. Isolated rupture of the subscapularis tendon. J Bone Joint Surg Am. 1996;78:1015–23.
15. Petersen SA, Murphy TP. The timing of rotator cuff repair for the restoration of function. J Shoulder Elbow Surg. 2011;20:62–8.

15 Critical Shoulder Angle: Does Lateral Acromioplasty Have a Role in Preventing Re-rupture?

Geoffroy Nourissat, Anselme Billaud, and Theo Kavakelis

15.1 Introduction

The anatomy of the acromion is variable, and since Neer's description, it has been considered as one of the key factors for shoulder pain and rotator cuff tears, as stated in the theory of extrinsic subacromial impingement [1]. For several years, subacromial decompression by partial resection of the acromion has been suggested and performed [2, 3], albeit clinical studies were unable to demonstrate the benefits of this procedure [4–6]. More recently, experimental and descriptive studies reported that a large acromion is correlated with high loads on the supraspinatus (SSP) tendon during abduction and that resection of the lateral part of the acromion substantially decreases the load on the SSP [7]. Therefore, it can be hypothesized that resection of a too large acromion could prevent rotator cuff tear or re-tears after repair.

15.2 Acromion: The History of a Guilty Bone

The first author who considered acromion as responsible for rotator cuff disorders was Armstrong, in 1949, who suggested that the supraspinatus syndrome results from the compression of the tendons of the cuff under the anterior part the acromion [1]. This theory was confirmed by Neer, who stated that rotator cuff tears resulted from the compression, or impingement, of the soft tissues between the humeral head and the coracoacromial arch [2]. Bigliani et al. suggested that a hooked (type-III) acromion might be a predisposing factor for rotator cuff tears; other authors reported that a flatter slope of the acromion or a decreased lateral acromion angle might reduce the subacromial space and increase the pressure on the rotator cuff tendons, thus predisposing them to degenerative tears [3]. Nyffeler et al. first reported the possible correlation between a large acromion and the presence of a rotator cuff tear [8]. They emphasized the role of the width of the acromion as a cause of shoulder pain and disorders. Other authors confirmed this hypothesis [9]. Although acromial morphology was analyzed in the pathogenesis of rotator cuff disease, some authors believe that the shape of the acromion is the result of rotator cuff disease [10].

Moor et al. [11] first described the critical shoulder angle (CSA) as the angle between the

G. Nourissat (✉)
Clinique Maussins Nollet, Paris, France

A. Billaud
Clinique du Sport, Merignac, France

T. Kavakelis
Hôpital Lariboisière—AP-HP, Paris, France

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_15

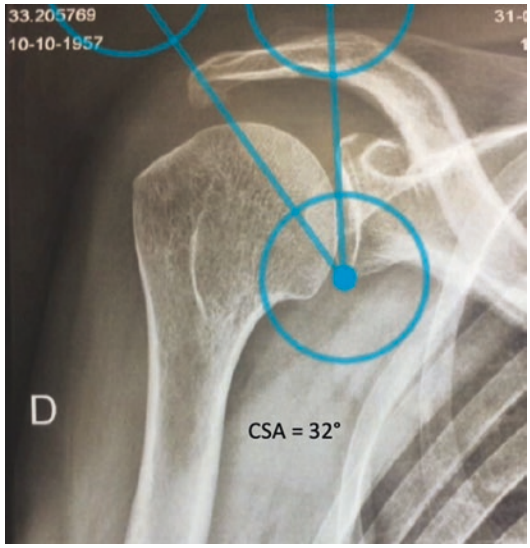


Fig. 15.1 Measurement of the CSA on a true AP view of the shoulder. Space between glenoid and humeral head is clearly seen

plane of the glenoid fossa and a line connecting the inferior glenoid rim with the most lateral extension of the acromion on a true antero-posterior (AP) radiograph (Fig. 15.1). The authors described 3 grades of CSA: In grade 1 the CSA is <30° and exposes mostly to osteoarthritis; in grade 2 the CSA is between 30° and 35° and is considered normal; in grade 3 the CSA is >35° and is mostly correlated with rotator cuff tears. Some authors hypothesized that the reduction of the CSA can prevent rotator cuff tear in symptomatic shoulders with intact cuff; moreover, reducing the CSA would protect cuff repair from mechanical stress and thus prevent re-tears [12–14].

15.3 Surgical Treatment

Several authors reported anatomical, radiological, and clinical studies on technical and safety issues of arthroscopic correction of CSA [8–10]. Arthroscopic resection of 6 mm of the lateral acromion can be done without damaging the deltoid [9]. This allows mean reduction of the CSA of 3.6°. Unfortunately, with a regular true AP X-ray view, there is a chance of error in the measurement of CSA of 2° [9]. Radiological data reported inconstant location of the most lateral aspect of the acromion seen on a true AP X-ray view. This location is called the critical acromion point (CAP). Some authors considered that the CAP is in the posterolateral aspect of the acromion, while others reported a more anterolateral position. Recent studies have shown that mean CAP is 21% ± 10% of the acromial anterior-posterior length from the anterolateral corner [10].

15.4 Literature Review

Recently published retrospective studies have reported that large acromion, with CSA superior to 38°, is related to rotator cuff re-tear. Garcia et al. [12] reported on 76 patients that underwent rotator cuff repair; preoperative average CSA was significantly lower (p = 0.001) for healed cuff group (34.3° ± 2.9°) than for non-healed cuff group (38.6° ± 3.5°). If CSA was >38°, the odds ratio of having a re-tear was 14.8 [8]. Li et al. [13] found also a correlation with re-tear after rotator cuff repair in patients with CSA superior or inferior to 38° (re-tear rate 15% and 0, respectively). Scheiderer et al. [15] reported on 57 patients that underwent rotator cuff repair; mean CSA for the re-tear group (37° ± 4°) was significantly higher (p = 0.014) than that in the intact group (35° ± 3°). If the CSA was >38°, the odds ratio of having a re-tear was 3.78 [15]. It is surprising to note that those authors did not find any other predisposing factor for re-tear.

As reported in several studies, patient age, tear size, and fatty degeneration of the supraspinatus are independent risk factors for a rotator cuff re-tear [14]. Wu et al. [16] found in 500 surgical cases that the initial tear size is the best predictor, followed by patient age; and Le et al. [17] evaluated preoperative and intraoperative factors for 1000 consecutive rotator cuff repairs and found that the rotator cuff tear size (tear dimensions, tear size area, and tear thickness) was strongly associated with re-tear 6 months after surgery.

It is possible to consider that CSA>38° could also be an independent risk factor for rotator cuff re-tear, but prospective studies must demonstrate

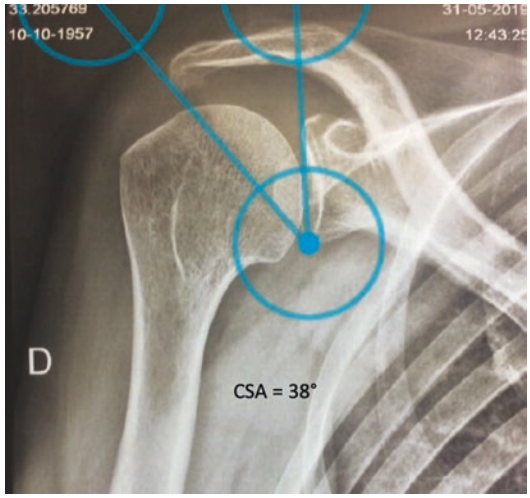


Fig. 15.2 Measurement of the CSA on a true AP view of the shoulder. The lateral osteophyte is known as a factor for cuff tear and influences the measurement of the CSA

it. Kim et al. [18] analyzed CSA and cuff tear in a retrospective study on 323 patients having MRI and X-rays. They identified patients with subacromial spurs or osteophytes, as these findings are associated to rotator cuff tears independently of acromion size. They found rotator cuff tears in all patients, independently of the value of CSA, and observed more correlation with acromial osteophytes than with the CSA value. The authors pointed out that acromial osteophyte is a main source of modification of the measurement of the CSA (Fig. 15.2) and concluded that correlation between a large acromion and a cuff tear should be related to osteophytes that jeopardize the measurement of the CSA.

Gerber et al. [9] reported that removing 6 mm of lateral acromion is safe and decreases the CSA from 3.6°. To date, it is unclear if removing lateral acromion to decrease the CSA under 35° is effective and safe. Kaiser et al. [19], in a cadaveric study, reported that it is necessary to perform a lateral acromioplasty to correctly decrease the CSA. Conversely, Billaud et al. [20] in a retrospective study of 90 patients showed that conventional anterior acromioplasty decreases the CSA from 2.9°. Thus, doing an anterolateral acromioplasty would be as efficient as doing a lateral in reducing the angle [10].

Some literature reviews [21, 22] analyzed the benefit of anterolateral acromioplasty on re-tear rate. They included level I and level II studies. No studies demonstrated the benefit of acromioplasty on preventing re-tear. Average reduction of CSA was 2.9°. However, there was no difference in re-tear rates between patients who underwent lateral acromioplasty and those who did not.

15.5 Conclusion

The concept of CSA is clever. Increasing the pulling strength of the deltoid is responsible for the compression of the humeral head against the acromion and increases the risk of tear of the supraspinatus. It is logic to believe that reducing the CSA could prevent re-tear after cuff repair, but there is currently not enough data to demonstrate that the acromioplasty, lateral or anterolateral, has any positive effect.

References

1. Armstrong JR. Excision of the acromion in treatment of the supraspinatus syndrome. Report of ninety-five excisions. J Bone Joint Surg Br. 1949;31:436–42.
2. Neer CS 2nd. Anterior acromioplasty for the chronic impingement syndrome in the shoulder: a preliminary report. J Bone Joint Surg Am. 1972;54:41–50.
3. Bigliani LU, Morrison DS, April EW. The morphology of the acromion and its relationship to rotator cuff tears. Orthop Trans. 1986;10:216.
4. Milano G, Grasso A, Salvatore M, Zarelli D, Deriu L, Fabbriciani C. Arthroscopic rotator cuff repair with and without subacromial decompression: a prospective randomized study. Arthroscopy. 2007;23(1):81–8.
5. MacDonald P, McRae S, Leiter J, Mascarenhas R, Lapner P. Arthroscopic rotator cuff repair with and without acromioplasty in the treatment of full-thickness rotator cuff tears: a multicenter, randomized controlled trial. J Bone Joint Surg Am. 2011;93(21):1953–60.
6. Abrams GD, Gupta AK, Hussey KE, Tetteh ES, Karas V, Bach BR Jr, Cole BJ, Romeo AA, Verma NN. Arthroscopic repair of full-thickness rotator cuff tears with and without acromioplasty: randomized prospective trial with 2-year follow-up. Am J Sports Med. 2014;42(6):1296–303.
7. Gerber C, Snedeker JG, Baumgartner D, Viehöfer AF. Supraspinatus tendon load during abduction is dependent on the size of the critical shoulder angle: a

biomechanical analysis. J Orthop Res. 2014;32(7):952–7. https://doi.org/10.1002/jor.22621.
8. Nyffeler RW, Werner CM, Sukthankar A, Schmid MR, Gerber C. Association of a large lateral extension of the acromion with rotator cuff tears. J Bone Joint Surg Am. 2006;88(4):800–5.
9. Gerber C, Catanzaro S, Betz M, Ernstbrunner L. Arthroscopic correction of the critical shoulder angle through lateral acromioplasty: a safe adjunct to rotator cuff repair. Arthroscopy. 2018;34(3):771–80.
10. Karns MR, Jacxsens M, Uffmann WJ, Todd DC, Henninger HB, Burks RT. The critical acromial point: the anatomic location of the lateral acromion in the critical shoulder angle. J Shoulder Elbow Surg. 2018;27(1):151–9.
11. Moor BK, Bouaicha S, Rothenfluh DA, Sukthankar A, Gerber C. Is there an association between the individual anatomy of the scapula and the development of rotator cuff tears or osteoarthritis of the glenohumeral joint?: a radiological study of the critical shoulder angle. J Bone Joint Br. 2013;95:935–41.
12. Garcia GH, Liu JN, Degen RM, Johnson CC, Wong AC, Dines DM, Gulotta LV, Dines JS. Higher critical shoulder angle increases the risk of retear after rotator cuff repair. J Shoulder Elbow Surg. 2017;26(2):241–5. https://doi.org/10.1016/j.jse.2016.07.009.
13. Li H, Chen Y, Chen J, Hua Y, Chen S. Large critical shoulder angle has higher risk of tendon retear after arthroscopic rotator cuff repair. Am J Sports Med. 2018;46(8):1892–900.
14. Lee M, Chen JY, Liow MHL, Chong HC, Chang P, Lie D. Critical shoulder angle and acromial index do not influence 24-month functional outcome after arthroscopic rotator cuff repair. Am J Sports Med. 2017;45(13):2989–94.
15. Scheiderer B, Imhoff FB, Johnson JD, Aglio J, Cote MP, Beitzel K, Imhoff AB, Arciero RA, Mazzocca AD, Morikawa D. Higher critical shoulder angle and acromion index are associated with increased retear risk after isolated supraspinatus tendon repair at short-term follow up. Arthroscopy. 2018;34(10):2748–54.
16. Wu XL, Briggs L, Murrell GA. Intraoperative determinants of rotator cuff repair integrity: an analysis of 500 consecutive repairs. Am J Sports Med. 2012;40(12):2771–6.
17. Le BT, Wu XL, Lam PH, Murrell GA. Factors predicting rotator cuff retears: an analysis of 1000 consecutive rotator cuff repairs. Am J Sports Med. 2014;42(5):1134–42.
18. Kim JH, Min YK, Gwak HC, Kim CW, Lee CR, Lee SJ. Rotator cuff tear incidence association with critical shoulder angle and subacromial osteophytes. J Shoulder Elbow Surg. 2019;28(3):470–5.
19. Kaiser D, Bachmann E, Gerber C, Meyer DC. Influence of the site of acromioplasty on reduction of the critical shoulder angle (CSA)—an anatomical study. BMC Musculoskelet Disord. 2018;19(1):371.
20. Billaud A, Cruz-Ferreira E, Pesquer L, Abadie P, Carlier Y, Flurin PH. Does the critical shoulder angle decrease after anterior acromioplasty? Arch Orthop Trauma Surg. 2019;139(8):1125–32.
21. Song L, Miao L, Zhang P, Wang WL. Does concomitant acromioplasty facilitate arthroscopic repair of full-thickness rotator cuff tears? A meta-analysis with trial sequential analysis of randomized controlled trials. Springerplus. 2016;5(1):685. eCollection 2016. https://doi.org/10.1186/s40064-016-2311-5.
22. Cheng C, Chen B, Xu H, Zhang Z, Xu W. Efficacy of concomitant acromioplasty in the treatment of rotator cuff tears: a systematic review and meta-analysis. PLoS One. 2018;13(11):e0207306 . eCollection 2018. https://doi.org/10.1371/journal.pone.0207306.

16 Shoulder Injections: Options, Ultrasound Assistance, Evidences

Madalena Pimenta, Adriana Moreira, and Nuno Sampaio Gomes

The shoulder is a major joint, frequently the site of multiple injuries and inflammatory conditions that ultimately may need a diagnostic or therapeutic injection. Joint injection is normally considered after other therapeutic interventions such as conservative treatment, oral medication, or physical therapy, as well as activity-modification, have been tried.

Indications for glenohumeral (GH) joint injection include ArthroMRI, treatment of osteoarthritis, hydrodistention in adhesive capsulitis and infiltrations in inflammatory conditions such as rheumatoid arthritis or various types of tendinopathies.

For the acromioclavicular joint, injections may be used for the diagnosis and treatment of osteoarthritis (OA) and distal clavicular osteolysis.

Subacromial injections are useful for a range of conditions including inflammatory conditions such as bursitis, impingement syndrome, and rotator cuff (RC) tendinosis. Scapulothoracic injections are reserved for the inflammation of the local bursa, namely, in cases of snapping scapula.

As all things in life, especially in medicine, proper technique is mandatory, as well as the choice and quantity of pharmaceuticals. Appropriate follow-up is also essential for effective outcomes.

16.1 Technique

The GH joint can be injected from an anterior, posterior, or superior approach. The anterior and posterior approaches are used more often. In the anterior approach (Fig. 16.1), the joint is most easily accessible with the patient sitting or lying, with the arm resting comfortably at the side, and the shoulder being externally rotated. Palpation of the head of the humerus, the coracoid process, and the acromion are essential landmarks to perform this injection without image guidance.

Sonography offers an accurate alternative to fluoroscopy for the injection of the shoulder joint. Sonographic guidance avoids the use of both ionizing radiation and iodinated contrast material and is generally faster than fluoroscopically guided injection. Both anterior and posterior sonographic approaches to GH joint injection have previously been reported [1, 2].

In our experience, we find the posterior approach (Figs. 2 and 3) safer, as it avoids any potential risk that may exist with an anterior

M. Pimenta (✉)
Radiology Department, Centro Hospitalar Universitário de São João, Porto, Portugal

A. Moreira
Radiology Department, Centro Hospitalar Universitário de São João, Porto, Portugal

Faculty of Medicine, Porto University, Porto, Portugal

N. Sampaio Gomes
Orthopaedics Department, Hospital Forças Armadas, Porto, Portugal

Hospital da Luz Porto, Arrábida, Póvoa, Portugal

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_16

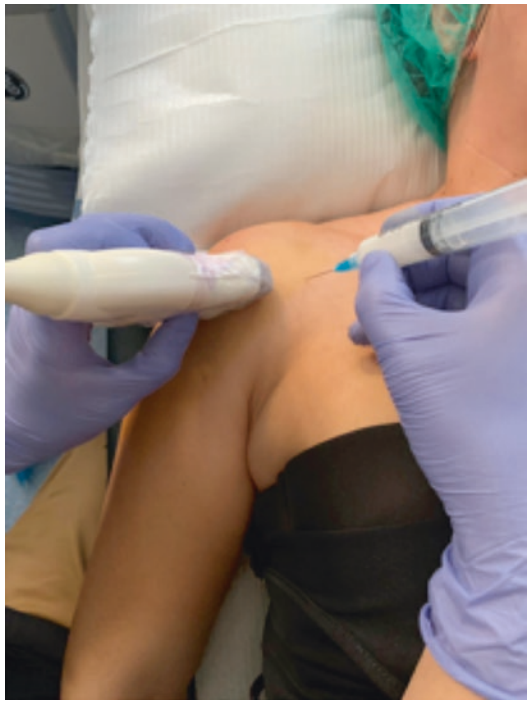


Fig. 16.1 Anterior approach for ultrasound-guided shoulder injections. The needle is advanced perpendicular to the medial edge of the humeral head, penetrating the subscapular tendon. If one hits the cartilage of the humeral head, the needle should be pulled back 1 or 2 mm, slightly angled by about 15% and then advanced tangentially to the head into the joint with the bevel of the needle facing into the joint

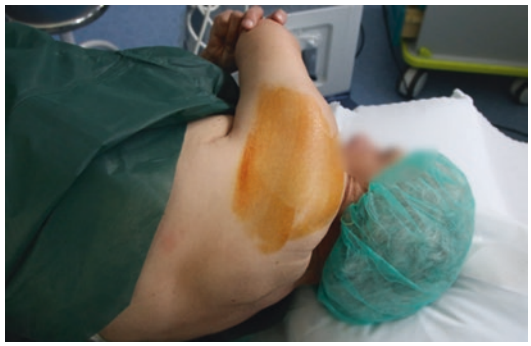
approach, for accidental puncture or injection of the major axillary neurovascular structures. It is also more comfortable to the patient. Ultrasound guidance is used as it gives a real-time needle position and avoids radiation exposure. The real-time visualization of the direction of needle passage, recognition of the typical feeling as the needle tip passes through the joint capsule, followed by the lack of resistance to injection, and the absence of any localized fluid pooling at the posterior joint line during early injection grant success to the procedure.

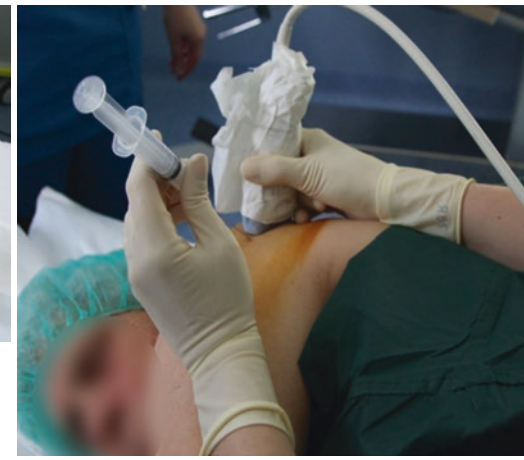
16.2 Therapeutical Options

It is important for the administering physician to understand the properties of the various pharmaceutical preparations available, as there is considerable variability in their effectiveness, duration of action, and potential for severe adverse effects.

16.2.1 Corticosteroids

Corticosteroids and local anesthetics are common parenteral medications in our practice, which can be administered in combination, either





Figs. 2 and 3 Posterior approach for ultrasound-guided shoulder injections. The patient is either lying obliquely prone on the contralateral shoulder or sitting upright with the back to the physician and the ipsilateral hand on the contralateral shoulder. The needle is inserted, from lateral to medial, parallel to the long axis of the transducer and advanced under US control into the joint between the humeral head and the posterior glenoid labrum

in the same syringe or separately during the same procedure.

Common indications include joint, bursal, paralabral cyst, and ganglion conditions [3].

Corticosteroids are predominantly administered because they are proven anti-inflammatory agents that provide medium-term relief of symptoms [4–6]. The common synthetic corticosteroids used in procedures are derivatives of prednisolone which is an analogue of cortisol, having anti-inflammatory potencies per dose unit somewhat greater than that of cortisol. Methylprednisolone is the methyl derivative of prednisolone, whereas betamethasone, dexamethasone, and triamcinolone are all fluorinated derivatives of prednisolone. Each corticosteroid formulation has a different potency.

Local anesthetics can provide not only immediate relief to the patient but also possible diagnostic feedback in the elucidation of the source of pain [7, 8].

The main concerns are the introduction of sepsis into a joint during the procedure and the exacerbation of sepsis already present within a joint. Contraindications to the use of steroid injections (SI) in shoulder disorders are local or intraarticular infection; and, because corticosteroids inhibit bone healing, an intraarticular fracture at the time of injection is a relative contraindication, as in markedly unstable joints or in those with severe juxta-articular osteoporosis [9–11]. Bacteremia or conditions likely to cause bacteremia (e.g., bacterial endocarditis, pneumonia) are generally regarded as contraindications to local SI [12]. Diabetes and treatment with anti-coagulant or anti-aggregation drugs are relative contraindications. Local corticosteroid injection can be administered using a delayed-release form of steroid and reducing the dose to 50% and the patient requested to check blood sugar every day for a week after the injection.

If the International Normalized Ratio (INR) values are adequate, local corticosteroid injection is generally well tolerated [13].

The established adverse effects associated with SI are post-injection "flare," local tissue atrophy, tendon rupture, cartilage damage, and flushing and increased blood glucose level.

The most common adverse effect is post-injection flare, which is a local increase in inflammation that develops within hours and can last 2–3 days. Local tissue necrosis, calcification, and tendon rupture have been associated with extra-articular injections, especially with the corticosteroid formulation triamcinolone hexacetonide [14].

Over the past several decades, there has been concern about the risk of articular hyaline cartilage damage with intra-articular injections. Results from large series confirm that cartilage loss can occur after repeated SI; however, it is thought that the risk is generally low, with 0.7–3% of patients who have received multiple injections developing substantial cartilage loss [15]. Additionally, corticosteroids may theoretically help preserve cartilage in the setting of active synovitis. Overall, many investigators believe this benefit outweighs the potential harm [16].

16.3 Biologic Approach of the Healing Response

Tendons have limited regeneration ability [17]. Many patients diagnosed with RC tendinopathy, with supraspinatus partial thickness tendon tears and tendinosis, are refractory to standard conventional non-operative care, and may have been on rehabilitation for long periods of time. It is also known that many factors affect healing after RC repair: tear characteristics, soft-tissue structural problems, repair technique and implants, or patient factors.

The interest in the biology of the healing response has been growing, and the biologic approach aims to optimize soft-tissue healing to improve clinical outcomes [18]. The biologic factors recently studied to enhance soft-tissue healing and regeneration have mainly focused on growth factors, stem cells, and platelet-rich plasma (PRP).

16.3.1 Platelet-Rich Plasma (PRP)

One biologic approach to RC disease utilizes PRP to restrain the inflammatory response and increase tendon-bone healing with growth factors.

Growth factors are molecules involved in the modulation of cell growth during the signal cascade of inflammation, and their influence is primordial in the inflammatory phase of tendon healing [19]. These growth factors are produced in great majority by fibroblast and inflammatory cells such as leukocytes and platelets [20]. During the inflammatory and repair phase of tendon healing, platelets aggregate at the site of soft-tissue injury and release a substantial quantity of growth factors, causing cell migration and differentiation at the site of injury [21].

One of the different classification systems to describe the final PRP concentrate broadly divides them into pure PRP (P-PRP) with a low content of leukocytes, leukocyte-rich PRP (L-PRP) with a high content of leukocytes, pure platelet-rich fibrin (P-PRF), and leukocyte-rich platelet-rich fibrin (L-PRF), with a high content of leukocytes and a high-density fibrin network [22].

PRP is easy to collect from blood and is, therefore, one of the most commonly used biological aids in RC repair [20].

There is a lack of high-level evidence from randomized clinical trials which have assessed the efficacy of platelet-rich plasma in treating ligament and tendon injuries.

A prospective randomized, double-blinded, clinical trial that compared the therapeutic effects of platelet-rich plasma injection with those of dry needling on shoulder pain and dysfunction in patients with RC disease concluded that platelet-rich plasma injections provided more significant pain relief and improved arm function, but not range of motion of the shoulder, in patients with supraspinatus tendon lesions (tendinosis or partial tear of less than 1.0 cm, but not a complete tear) when compared to dry needling [23].

Sham et al. conducted a prospective randomized controlled study to evaluate the results of subacromial injection of PRP versus SI therapy in 40 patients with symptomatic partial RC tears, in a study with a level II evidence [24]. They concluded that subacromial autologous platelet-rich plasma (PRP) injection for the treatment of a partial supraspinatus tendon tear is comparable to the standard SI. Additionally, more favorable clinical results were noticed at 3 months, although no statistically significant improvement in the outcome measures could be demonstrated at 6 months after injection [24].

It is our belief that the subacromial PRP injection could be a quite good alternative to SI, especially in patients with a contraindication to SI.

In recent years, the clinical application of PRP and PRF in association with RC repair has also increased [20].

Hurley et al. recently published a systematic review of randomized controlled trials in the literature to ascertain whether PRP or PRF improved patient outcomes in arthroscopic RC repair [25]. The most important finding from this study was that PRP had clinical benefits in improving tendon healing rates in tears of all sizes (including tears >3 cm), pain levels, and functional outcomes in RC repair. In contrast, PRF had no beneficial effect on tendon healing or clinical outcomes. This meta-analysis found that pain levels were significantly lower in the immediate postoperative period, a month after surgery, and at final follow-up when PRP was used compared to a control. PRF had no benefit in any single study in terms of tendon healing, tendon vascularity, or functional outcome [25].

Despite these results, there are limitations in recommending routine PRP in arthroscopic RC repair, as there is still uncertainty in the composition of PRP. With an assortment of PRP preparations, there are differences in the platelet count, leukocyte count, and growth factor concentration that vary depending on the patient characteristics and preparation kits used. The optimal dosage and timing intervals of injections also remain areas of concern, as they may affect the postoperative course and there is no literature to support any injection protocol.

16.3.2 Amniotic Membrane (AM)–Derived Products

Mesenchymal stem cells (MSCs), or multipotent progenitor cells, have been an important area of

investigation due to their ability to differentiate into various cell types in mesenchymal tissues and to secrete a large number of bioactive macromolecules important in the tissue regenerative process [26].

As the number of MSCs in marrow tissues is limited, researchers are ceaselessly investigating substitute sources of MSCs, including placental-derived tissues, such as the AM, amniotic fluid (AF), umbilical cord, and umbilical cord blood [27].

The efficacy and safety of amniotic tissues have been demonstrated through years of use in other medical subspecialties. However, the existing literature describing their use in orthopedic sports medicine is limited.

A recent systematic review identified 20 animal studies and 7 human studies reporting the use of placenta-derived cells and placental tissue allografts for orthopedic sports medicine indications [28]. One of the human studies was conducted by Gellhorn and Han, who performed a prospective case series of consecutive patients being treated for arthritis or tendinopathy with an ultrasound-guided injection of dehydrated human amniotic/chorionic membrane (dHACM) [29]. Forty patients were included in the final analysis, including 3 GH joints that received intra-articular injections. Treated tendons included, among others, the supraspinatus tendon. Despite a transient increase in pain at the injection site that lasted an average of 2 days, notable improvement of pain and function at 1, 2, and 3 months of follow-up were reported [30].

Despite the proven safety of human AM-derived products, the efficacy and mechanisms by which they exert a therapeutic benefit are unknown and higher-quality clinical trials to further elucidate their specific applications, therapeutic benefit, and cost-effectiveness are needed.

Nonetheless, they represent a wide variety of promising tissues and cell populations in the field of sports medicine and, as this area of orthobiologics is advancing, it will be important for clinicians to understand the individual products.

16.4 Hyaluronic Acid

Hyaluronic acid (HA) is naturally present in synovial fluid and highly concentrated at the articular cartilage surface. In high concentrations, HA increases the viscosity and elasticity of synovial fluid, allowing it to act not only as a lubricant but also as a shock absorber in synovial joints. Its effects protect the cartilage against shear and compressive forces [30].

In the natural history of OA, the concentration of HA decreases. The original logic behind viscosupplementation was restoration of the viscoelasticity of synovial fluid. However, it is also thought to augment the flow of synovial fluid and inhibit degradation of endogenous HA. This should lead to an overall decrease in joint pain and increase in function [31].

Some large-scale randomized controlled trials exist that support the use of viscosupplementation in GH OA.

Blaine and colleagues [30] randomized 660 patients with persistent shoulder pain due to GH OA, RC tears, and/or adhesive capsulitis and concluded that patients with OA had borderline significant improvements in the visual analogue scale (VAS) at 13 weeks and clearly significant pain reduction at 26 weeks.

The randomized controlled trial conducted by Kwon and colleagues enrolled 300 patients with GH OA and evaluated primary outcomes measured by the VAS over 26 weeks, concluding that there was a statistically significant VAS difference between the groups, favoring the group treated with HA over the control group [32].

Zhang and colleagues conducted a systematic review on the use of viscosupplementation in a cohort of patients with GH OA and found a significant reduction in pain at 3 months and 6 months for patients receiving intraarticular HA injections and also improved functional outcomes at every follow-up time point across all included studies [33].

Literature also reports a low rate of local reaction to HA, including pain at the injection site, effusion, and painful flares.

Regarding patients with RC tendinopathy and tears, the evidence is more scarce.

Frizziero et al. demonstrated that in patients with chronic non-calcific RC tendinopathy there was a significant improvement in pain and function and a significant reduction in shoulder disability until 3 months of follow-up [34]. However, in a recent meta-analysis by Lin et al., the clinical benefit resulting from the subacromial HA injection for RC tendinopathy was inconclusive [35].

Honda and colleagues examined the in-vitro effects of HA on RC healing after RC repair, particularly on the chondroid tissue at the repaired site, and demonstrated that HA accelerated tendon-to-bone healing in a RC tear model, enhancing the biomechanical strength and increasing chondroid formation at the repaired site [36].

In summary, intra-articular HA injection is safe and improves pain for patients with GH OA. Further randomized controlled trials are necessary to identify optimal dosing and route of administration and to test the efficacy of HA injections in cases of RC tendinopathy.

16.5 Prolotherapy

Prolotherapy is a type of regenerative injection therapy that uses an irritating agent, most commonly hypertonic dextrose solution, that is injected over several sessions into multiple sites of painful tendon and ligament insertions near the bone [37].

The precise mechanism of prolotherapy remains unclear, but it is thought that the injection of an irritant solution at painful ligament and tendon insertions stimulates local healing through proliferation of scar tissue [38].

Although most of the literature is limited to the treatment of knee OA, some evidence in RC disease exists. In a retrospective case-control study with a 1-year follow-up conducted by Lee and colleagues, prolotherapy improved pain, disability, isometric strength, and range of motion in patients with chronic refractory RC disease [39].

Bertrand et al., in a double-blind randomized-controlled trial, reported that hypertonic dextrose injection improved long-term pain, which improved patient satisfaction [40].

As stated above, multiple injection sites with many intervention sessions are used in prolotherapy. In contrast with traditional prolotherapy, Lin et al. carried out a double-blind randomized-controlled trial in which they applied hypertonic dextrose injections on the enthesis portion of the supraspinatus tendon with ultrasound guidance in patients with tendinopathy. Although they found relief from pain and disability for up to 2 weeks after intervention, the improvement in pain in function did not sustain for 6 weeks [41].

To the best of the authors' knowledge, there are no comparative studies of prolotherapy with other injection therapies in the setting of RC disease in the literature.

Definitive determination of the clinical utility of dextrose prolotherapy will require improved understanding of its exact mechanism of action as well as additional and larger clinical trials with more complete functional assessment tools.

16.6 Natural Medications

There is little evidence about the efficacy of natural medications in the setting of RC diseases.

Traumeel (Tr14) injection solution is a well-tolerated natural combination medicine. Although the exact mechanism of action of Tr14 injection solution is still to be understood, an "in vitro" study of Porozov et al. indicated that Tr14 inhibits the secretion of pro-inflammatory cytokines, such as IL-1β, CXCL8, and TNF-α in resting, as well as activated immune cells [42]. It was suggested that Tr14 injection solution could act by speeding up the healing process, instead of blocking the development of edema from the beginning [43].

Pilat et al. demonstrated Tr14 to reduce the exercise-induced inflammatory response of the innate immune system [44].

To the best of the author's knowledge, there is only one study protocol of a randomized, controlled trial aiming to assess the efficacy and safety of Tr14 in patients with RC syndrome and bursitis treated with Tr14 injection solution injections versus SI and versus placebo [45]. However, its results have not yet been published.

Ozone (O3) therapy raises the pain threshold as it works based on stimulating antinociceptive apparatus mediated by serotonin and endogenous opioids and it has applications in different specialties of medicine and dentistry [46–50].

O3 therapy is also a common theme among many literature reports in musculoskeletal disorders and has been used to treat many pathologies regarding the muscles, tendons, and joints [46].

Anecdotal reports of pain reduction and function recovery in cases of severe shoulder pain exist [51]. However, randomized controlled trials are lacking regarding O3 application in RC disorders.

It is hoped that further investigation and clinical trials will assist in providing more evidence concerning the efficacy and safety of natural medications.

16.7 Suprascapular Nerve Block

The suprascapular nerve (SSN) originates from the ventral rami of the fifth and sixth cervical nerve roots and enters the supraspinous fossa via the suprascapular notch underneath the superior transverse scapular ligament. The suprascapular artery and vein pass above this ligament [52].

The SSN sends two branches shortly after passing through the suprascapular notch, providing sensory supply to the superior part of the shoulder, and is the dominant motor supply to the supraspinatus and infraspinatus [53].

The suprascapular nerve block (SSNB) has proven to be an effective form of pain relief in patients with a broad range of shoulder pathologies [54].

The ideal site to perform SSNB with US is at the floor of the supraspinatus fossa, between the suprascapular notch and the spinoglenoid notch [55]. At this site, the SSN is covered by the fascia of supraspinatus in a natural compartment, which will contain the spread of the local anesthetic or injectate. When the needle is placed near the SSN, several methods of nerve blockade have been published. The commonly used methods include local anesthetic, steroids, pulsed RF, and chemical neurolysis. These may be used alone or in combination [54].

Among the pathologies in which the SNNB has proven to be effective are the adhesive capsulitis of the shoulder and the painful hemiplegic shoulder [56–58].

Full-thickness rotator cuff tears cause traction and tension on the SSN, which has been shown to increase with tear size and to be dynamic with a range of movements [59]. Suprascapular neuropathy is a severe clinical manifestation of this and is associated with large rotator cuff tears [60].

A recent randomized controlled trial compared SSNBs with SAs (subacromial injections), by administering 9 mL of 1% ropivacaine and 1 mL of betamethasone in both groups, in the nonoperative management of rotator cuff tears and concluded that SSNBs was superior to SAs in restoring function and reducing pain over a 12-week period, particularly full-thickness tears [53].

Given the evidence, SSNB can be an alternative to an SA for nonoperative management of rotator cuff tears.

16.8 Ultrasound-Guided Needling and Lavage (Barbotage)

Rotator cuff calcific tendonitis (RCCT) is a self-limiting disorder characterized by deposition of calcium salts in RC muscles. The presenting

symptom often is pain associated with activity persisting for months with spontaneous regression in most of the cases [61]. However, its symptoms can be severe and persistent.

The initial line of management of RCCT is usually nonoperative. It includes symptomatic management using systematic NSAIDs in acute phase, physical therapy using cold or heat, and manual therapy with exercises improving a range of movements.

Barbotage is a minimally invasive procedure that has become increasingly popular and involves image-guided irrigation of calcium deposits followed by aspiration.

Because this modality of treatment is ultrasound (US)-guided, it is radiation free and cost effective.

The procedure is done by using an 18, 20 G needle with a syringe filled with lidocaine, and an entry is made into the deposit (Fig. 16.4). Some amount of lidocaine is injected into it, and the calcium dissolved re-enters the syringe passively (Fig. 16.5). Further procedure can be continued with saline, and the puncture and aspiration can be done with 2 different needles [62].

There are several reports available with good mid- and long-term results of barbotage [63–65].

de Witte et al. published the outcomes comparing US-guided barbotage combined with SI in the subacromial bursa versus isolated SAIC

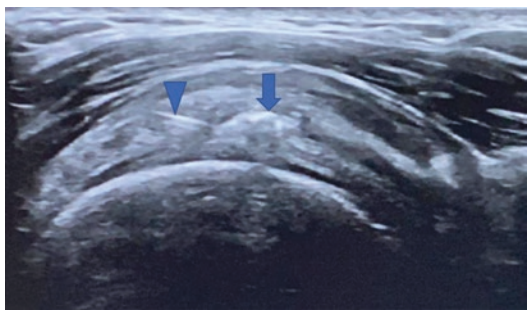


Fig. 16.4 Sagittal image of an ultrasound exam: note the needle as a linear image (arrowhead) and a heterotopic calcification (arrow)

Fig. 16.5 Syringes filled with calcium hydroxyapatite after Barbotage

in patients with RCCT, after 1-year and 5-year follow-up. They reported an improvement in clinical and radiographic status after 1-year follow-up in patients with symptomatic RCCT that is nonresponsive to conservative treatment. However, results of barbotage were significantly better in terms of more resorption and higher clinical scores at follow-up. Their 5-year follow-up randomized controlled trial showed no statistically significant differences in the clinical and radiological mid-term outcomes of US-guided barbotage combined with an SAIC, compared with an isolated US-guided SAIC in patients with RCCT. There was, however, a statistically significant and clinically relevant improvement in both groups in terms of clinical scores and radiological resorption rates compared with baseline [66, 67].

Therefore, barbotage can be a great option in patients with persisting symptoms of RCCT and no signs of spontaneous resorption over time (Fig. 16.6).

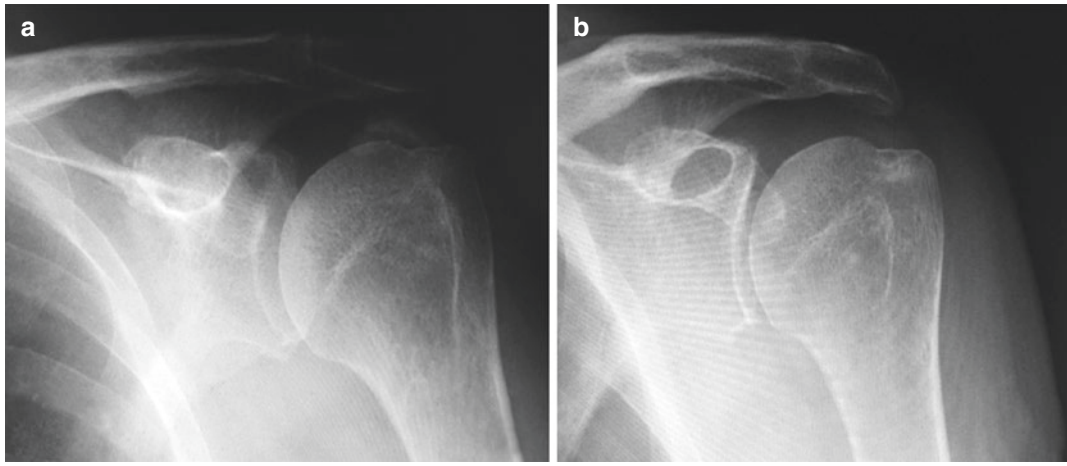


Fig. 16.6 AP view of Shoulder radiography: Calcific tendonitis (**a**) before and (**b**) after Barbotage

16.9 Conclusion

Shoulder injections are useful tools to address a numerous amount of clinical conditions, and orthobiologics are a very promising and powerful weapon that is gaining popularity among doctors that use those to treat their patients.

However, there is still an important lack of evidence concerning their role in musculoskeletal pathologies, and it is the physician's obligation to be judicious when deciding for a specific treatment for their patients.

A current widespread usage of orthobiologics by several health care professionals, doctors, and non-doctors has led to, in some countries like the USA, very restrictive measures for their marketing and strict regulatory requirements for their application.

PRPs, for instance, have shown some benefit in knee arthritis, lateral epicondylitis, and ulnar collateral ligament injury, but inconsistent or minimal benefits in rotator cuff repair [68]; and the optimal concentration of leucocytes and ideal kind and stage of injuries for PRP application are still unknown.

On the other hand, corticosteroids are still the cornerstone of injection therapy, in spite of its debatable efficacy in terms of pain relief, improvement in range of motion, and return of shoulder function [37].

Besides, it is clear that the accuracy of the administration is very important for the results [37] and ultrasound guidance is, in fact, one of the few evidences available today [69, 70] regarding injection therapies in the shoulder.

References

1. Valls R, Melloni P. Sonographic guidance of needle position for MR arthrography of the shoulder. Am J Roentgenol. 1997;169:845–7.
2. Cicak N, Matasovic T, Bajraktarevic T. Ultrasonic guidance of needle placement for shoulder arthrography. J Ultrasound Med. 1992;11:135–7.
3. Louis LJ. Musculoskeletal ultrasound intervention: principles and advances. Radiol Clin N Am. 2008;46:515–33. vi
4. Robinson P, Keenan AM, Conaghan PG. Clinical effectiveness and dose response of image-guided intra-articular corticosteroid injection for hip osteoarthritis. Rheumatology (Oxford). 2007;46:285–91.

5. Ravaud P, Moulinier L, Giraudeau B, et al. Effects of joint lavage and steroid injection in patients with osteoarthritis of the knee: results of a multicenter, randomized, controlled trial. Arthritis Rheum. 1999;42:475–82.
6. Hochberg MC, Perlmutter DL, Hudson JI, Altman RD. Preferences in the management of osteoarthritis of the hip and knee: results of a survey of community-based rheumatologists in the United States. Arthritis Care Res. 1996;9:170–6.
7. Crawford RW, Gie GA, Ling RS, Murray DW. Diagnostic value of intra-articular anaesthetic in primary osteoarthritis of the hip. J Bone Joint Surg Br. 1998;80:279–81.
8. Khoury NJ, el-Khoury GY, Saltzman CL, Brandser EA. Intraarticular foot and ankle injections to identify source of pain before arthrodesis. Am J Roentgenol. 1996;167:669–73.
9. Kondo T, Kitazawa R, Yamaguchi A, Kitazawa S. Dexamethasone promotes osteoclastogenesis by inhibiting osteoprotegerin through multiple levels. J Cell Biochem. 2008;103:335–45.
10. Aspenberg P. Drugs and fracture repair. Acta Orthop. 2005;76:741–8.
11. Pountos I, Georgouli T, Blokhuis TJ, Pape HC, Giannoudis PV. Pharmacological agents and impairment of fracture healing: what is the evidence? Injury. 2008;39:384–94.
12. van de Putte LBA, Furst DE, Williams HJ, van Riel PLCM, editors. Therapy of systemic rheumatic disorders. New York: Marcel Dekker; 1997.
13. Molini L, Mariacher S, Bianchi S. US guided corticosteroid injection into the subacromial-subdeltoid bursa: technique and approach. J Ultrasound. 2012;15(1):61–8.
14. MacMahon PJ, Eustace SJ, Kavanagh EC. Injectable corticosteroid and local anesthetic preparations: a review for radiologists. Radiology. 2009;252(3):647–61.
15. Balch HW, Gibson JM, El-Ghobarey AF, Bain LS, Lynch MP. Repeated corticosteroid injections into knee joints. Rheumatol Rehabil. 1977;16:137–40.
16. Ostergaard M, Halberg P. Intra-articular costicosteroids in arthritic disease: a guide to treatment. BioDrugs. 1998;9:95–103.
17. Ho JO, Sawadkar P, Mudera V. A review on the use of cell therapy in the treatment of tendon disease and injuries. J Tissue Eng. 2014;18(5):204–13.
18. LaPrade RF, Dragoo JL, Koh JL, Murray IR, Geeslin AG, Chu CR. AAOS research symposium updates and consensus: biologic treatment of orthopaedic injuries. J Am Acad Orthop Surg. 2016;24(7):e62–78.
19. Randelli P, Randelli F, Ragone V, et al. Regenerative medicine in rotator cuff injuries. Biomed Res Int. 2014;2014:129515.
20. McMahon PJ, editor. Rotator cuff injuries. Cham: Springer International Publishing AG; 2018.
21. Gamradt SC, et al. Platelet rich plasma in rotator cuff repair. Tech Orthop. 2007;22(1):26–33.
22. Dohan Ehrenfest DM, Rasmusson L, Albrektsson T. Classification of platelet concentrates: from pure platelet- rich plasma (P-PRP) to leucocyte- and platelet-rich fibrin (L-PRF). Trends Biotechnol. 2009;27(3):158–67.
23. Rha D, Park G-Y, Kim Y-K, Kim MT, Lee SC. Comparison of the therapeutic effects of ultrasound-guided platelet-rich plasma injection and dry needling in rotator cuff disease: a randomized controlled trial. Clin Rehabil. 2013;27(2):113–22.
24. Shams A, El-Sayed M, Gamal O, et al. Eur J Orthop Surg Traumatol. 2016;26:837.
25. Hurley ET, Fat DL, Moran CJ, Mullett H. The efficacy of platelet-rich plasma and platelet-rich fibrin in arthroscopic rotator cuff repair: a meta-analysis of randomized controlled trials. Am J Sports Med. 2019;47(3):753–61.
26. Caplan AI. Adult mesenchymal stem cells for tissue engineering versus regenerative medicine. J Cell Physiol. 2007;213(2):341–7.
27. Baghaban Eslaminejad M, Jahangir S. Amniotic fluid stem cells and their application in cell-based tissue regeneration. Int J Fertil Steril. 2012;6(3):147–56.
28. Duerr RA, Ackermann J, Gomoll AH. Amniotic-derived treatments and formulations. Clin Sports Med. 2019;38(1):45–59.
29. Gellhorn AC, Han A. The use of dehydrated human amnion/chorion membrane allograft injection for the treatment of tendinopathy or arthritis: a case series involving 40 patients. PM R. 2017;9(12):1236–43.
30. Blaine T, Moskowitz R, Udell J, et al. Treatment of persistent shoulder pain with sodium hyaluronate: a randomized, controlled trial. J Bone Joint Surg Am. 2008;90(5):970–9.
31. Jong BY, Goel DP. Biologic options for glenohumeral arthritis. Clin Sports Med. 2018;37:537–48.
32. Kwon YW, Eisenberg G, Zuckerman JD. Sodium hyaluronate for the treatment of chronic shoulder pain associated with glenohumeral osteoarthritis: a multicenter, randomized, double-blind, placebo-controlled trial. J Shoulder Elb Surg. 2013;22(5):584–94.
33. Zhang B, Thayaparan A, Horner N, Bedi A, Alolabi B, Khan M. Outcomes of hyaluronic acid injections for glenohumeral osteoarthritis: a systematic review and meta-analysis. J Shoulder Elb Surg. 2018;28(3):596–606. pii: S1058-2746(18)30701-8.
34. Frizziero A, Vittadini F, Barazzuol M, et al. Extracorporeal shockwaves therapy versus hyaluronic acid injection for the treatment of painful non-calcific rotator cuff tendinopathies: preliminary. J Sports Med Phys Fitness. 2017;57(9):1162–8.
35. Lin MT, Chiang CF, Wu CH, Huang YT, Tu YK, Wang TG. Comparative effectiveness of injection therapies in rotator cuff tendinopathy: a systematic review, pairwise and network meta-analysis of randomized controlled trials. Arch Phys Med Rehabil. 2019;100(2):336–349.e15.
36. Honda H, Gotoh M, Kanazawa T, Ohzono H, Nakamura H, Ohta K, Nakamura K-i, et al. Hyaluronic acid accelerates tendon-to-bone healing after rotator cuff repair. Am J Sports Med. 2017;45(14):3322–30.

37. Lin KM, Wang D, Dines JS. Injection therapies for rotator cuff disease. Orthop Clin North Am. 2018;49(2):231–9.
38. Rabago D, Patterson JJ, Mundt M, et al. Dextrose prolotherapy for knee osteoarthritis: a randomized controlled trial. Ann Fam Med. 2013;11(3):229–37.
39. Lee DH, Kwack KS, Rah UW, et al. Prolotherapy for refractory rotator cuff disease: retrospective case control study of 1-year follow-up. Arch Phys Med Rehabil. 2015;96(11):2027–32.
40. Bertrand H, Reeves KD, Bennett CJ, et al. Dextrose prolotherapy versus control injections in painful rotator cuff tendinopathy. Arch Phys Med Rehabil. 2016;97(1):17–25.
41. Lin CL, Huang CC, Huang SW. Effects of hypertonic dextrose injection on chronic supraspinatus tendinopathy of the shoulder: randomized placebo-controlled trial. Eur J Phys Rehabil Med. 2019;55(4):480–7.
42. Porozov S, Cahalon L, Weiser M, Branski D, Lider O, Oberbaum M. Inhibition of IL-1β and TNF-α secretion from resting and activated human immunocytes by the homeopathic medication Traumeel® S. Clin Dev Immunol. 2004;11(2):143–9.
43. Lussignoli S, Bertani S, Metelmann H, Bellavite P, Conforti A. Effect of Traumeel S®, a homeopathic formulation, on blood-induced inflammation in rats. Complement Ther Med. 1999;7:225–30.
44. Pilat C, Frech T, Wagner A, Krüger K, Hillebrecht A, Pons-Kühnemann J, Scheibelhut C, Bödeker R-H, Mooren F-C. Exploring effects of a natural combination medicine on exercise induced inflammatory immune response: a double-blind RCT. Scand J Med Sci Sports. 2015;25(4):534–42.
45. Vanden Bossche L, Vanderstraeten G. A multicenter, double-blind, randomized, placebo-controlled trial protocol to assess Traumeel injection vs dexamethasone injection in rotator cuff syndrome: the TRAumeel in ROtator cuff syndrome (TRARO) study protocol. BMC Musculoskelet Disord. 2015;16(1):8.
46. Seyam O, Smith NL, Reid I, Gandhi J, Jiang W, Khan SA. Clinical utility of ozone therapy for musculoskeletal disorders. Med Gas Res. 2018;8(3):103–10.
47. Martínez-Sánchez G, Al-Dalain SM, Menéndez S, Re L, Giuliani A, Candelario-Jalil E, et al. Therapeutic efficacy of ozone in patients with diabetic foot. Eur J Pharmacol. 2005;523:151–61.
48. Bocci V. Ozone as Janus: this controversial gas can be either toxic or medically useful. Mediat Inflamm. 2004;3-11(18):13.
49. Valacchi G, Fortino V, Bocci V. The dual action of ozone on the skin. Br J Dermatol. 2005;153: 1096–100.
50. Nogales CG, Ferrari PH, Kantorovich EO, Lage-Marques JL. Ozone therapy in medicine and dentistry. J Contemp Dent Pract. 2008;1:75–84.
51. Benvenuti P. Oxygen-ozone treatment of the knee, shoulder and hip: a personal experience. Rivista Italiana di Ossigeno-Ozonoterapia. 2006;5:135–44.
52. Bigliani LU, Dalsey RM, McCann PD, April EW. An anatomical study of the suprascapular nerve. Arthroscopy. 1990;6:301Y305.
53. Coory JA, Parr AF, Wilkinson MP, Gupta A. Efficacy of suprascapular nerve block compared with subacromial injection: a randomized controlled trial in patients with rotator cuff tears. J Shoulder Elb Surg. 2019;28(3):430–6.
54. Chan CW, Peng PW. Suprascapular nerve block: a narrative review. Reg Anesth Pain Med. 2011;36:358–73.
55. Peng PWH, Wiley MJ, Liang J, Bellingham GA. Ultrasound-guided suprascapular nerve block: a correlation with fluoroscopic and cadaveric findings. Can J Anesth. 2010;57(2):143–8.
56. Ozkan K, Ozcekic AN, Sarar S, Cift H, Ozkan FU, Unay K. Suprascapular nerve block for the treatment of frozen shoulder. Saudi J Anaesth. 2012;6(1):52–5.
57. Klc Z, MB Filiz TC, Toraman NF. Addition of suprascapular nerve block to a physical therapy program produces an extra benefit to adhesive capsulitis: a randomized controlled trial. Am J Phys Med Rehabil. 2015;94(10 Suppl 1):912–20.
58. Aydın T, Şen Eİ, Yardımcı MY, Kesiktaş FN, Öneş K, Paker N. Efficacy of ultrasound-guided suprascapular nerve block treatment in patients with painful hemiplegic shoulder. Neurol Sci. 2019;40(5):985–91.
59. Albritton MJ, Graham RD, Richards RS 2nd, Basamania CJ. An anatomic study of the effects on the suprascapular nerve due to retraction of the supraspinatus muscle after a rotator cuff tear. J Shoulder Elb Surg. 2003;12:497–500.
60. Boykin RE, Friedman DJ, Zimmer ZR, Oaklander AL, Higgins LD, Warner JJ. Suprascapular neuropathy in a shoulder referral practice. J Shoulder Elb Surg. 2011;20:983–8.
61. Umamahesvaran B, Sambandam SN, Mounasamy V, Gokulakrishnan PP, Ashraf M. Calcifying tendinitis of shoulder: a concise review. J Orthop. 2018;15(3):776–82.
62. Del Cura JL. Ultrasound-guided therapeutic procedures in the musculoskeletal system. Curr Probl Diagn Radiol. 2008;37(5):203–18.
63. Del Cura JL, Torre I, Zabala R, Legorburu A. Sonographically guided percutaneous needle lavage in calcific tendinitis of the shoulder: short and long-term results. Am J Roentgenol. 2007;189:W128–34.
64. Koester MC, Dunn WR, Kuhn JE, Spindler KP. The efficacy of subacromial corticosteroid injection in the treatment of rotator cuff disease: a systematic review. J Am Acad Orthop Surg. 2007;15:3–11.
65. Serafini G, Sconfienza LM, Lacelli F, Silvestri E, Aliprandi A, Sardanelli F. Rotator cuff calcific tendonitis: short-term and 10-year outcomes after two-needle US-guided percutaneous treatment. Nonrandomized controlled trial. Radiology. 2009;252:157–64.
66. de Witte PB, Selten JW, Navas A, Nagels J, Visser CPJ, Nelissen RGHH, Reijnierse M. Calcific tendinitis of the rotator cuff: a randomized controlled trial of ultrasound-guided needling and lavage versus subacromial corticosteroids. Am J Sports Med. 2013;41(7):1665–73.

67. de Witte PB, Kolk A, Overes F, Nelissen RGHH, Reijnierse M. Rotator cuff calcific tendinitis: ultrasound-guided needling and lavage versus subacromial corticosteroids: five-year outcomes of a randomized controlled trial. Am J Sports Med. 2017;45(14):3305–14.
68. Milano G, Sánchez M, Jo CH, et al. Platelet-rich plasma in orthopaedic sports medicine: state of the art. J ISAKOS. 2019;4:188–95.
69. Daniels EW, Cole D, Jacobs B, Phillips SF. Existing evidence on ultrasound-guided injections in sports medicine. Orthop J Sports Med. 2018;6(2):2325967118756576.
70. Aly AR, Rajasekaran S, Ashworth N. Ultrasound-guided shoulder girdle injections are more accurate and more effective than landmark-guided injections: a systematic review and meta-analysis. Br J Sports Med. 2015;49(16):1042–9.

Nonoperative Treatment: The Role of Rehabilitation

17

López-González Luis, Tiso Giovanni, Gomes Joana, and Aramberri Mikel

17.1 Rehabilitation of Massive Rotator Cuff Tears

Treatment for rotator cuff tendon diseases ranges from conservative treatment (including exercise, electrotherapy, manual therapy, injection therapy, hydrotherapy, taping…) to different surgical strategies: simple debridement, partial or complete tendon repair with or without tissue substitutes, synovectomies, biceps tenotomies, or tendon transfers [1–3]. So far, several systematic reviews have compared the effectiveness of operative management to nonoperative treatment (NOT), reaching ambiguous conclusions [4–6]: whereas some evidence supports surgical options [6, 7], others have equated them with conservative treatments [2, 8].

Not all patients meet all criteria to be eligible for surgical repair [9] in the context of massive rotator cuff tears (RCT), especially those who remain asymptomatic [10] and are often identified accidentally [11]. Reasons for non-eligibility include comorbidities that contraindicate surgery and tears considered upon evaluation to be irreparable [9]. Additionally, surgery has been thought to be less successful in elderly population when RCT have retracted medially to the glenoid rim and are massive in size (>5 cm) [2, 12].

In these landscapes, several studies [9, 13–17] have observed that conservative treatments can produce improvements in terms of pain relief and motion and disability enhancements, although they are still few in number. Among the conservative treatments considered, exercise combined with injection therapy and pharmacological management has been by far the most popular. These studies have widely shown relevant benefits with conservative treatment for its capacity to (1) improve motion (especially forward elevation, internal and external rotations), (2) strengthen muscle power, and (3) reduce pain in elderly patients with low activity levels and/or patients unsuitable for surgery due to severe comorbidities [18]. This reveals that the best primary treatment option should be chosen to palliate symptom worsening and in some cases NOT may be considered to improve activities of daily living [9, 13, 18, 19] (Table 17.1).

It is hopeful that both exercise and physical therapy have been shown to be the most viable

Table 17.1 Indications for rehabilitation in nonoperative treatment of massive rotator cuff tears

– Patients without significant pain
– Elderly patients with low activity level
– Patients with a functional range of motion of the shoulder
– Patients who have progressively improved with a multimodal physical treatment
– Contraindications for surgery

L.-G. Luis · T. Giovanni · A. Mikel (✉)
Alai Sport Medicine Clinic, Madrid, Spain
e-mail: luisfisio@centroalai.es; aramberri@centroalai.es

G. Joana
Department of Physical Medicine and Rehabilitation, Hospital Sra da Oliveira - Guimarães, Guimarães, Portugal

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_17

alternative option to surgery [2, 5, 20], but it has to be noted that researchers have found great difficulties to synthesize and standardize robust evidence-based rehabilitation programs, because most of them have been developed in observational non-randomized studies [2, 21, 22]. Some of these researches will be displayed throughout the present chapter.

Although the lack of high-quality studies has been highlighted, one of the latest systematic reviews [23] on exercise therapy in massive nonoperative RCTs (2018) has remarked that, in general, there is enough consistent evidence confirming that exercise is an effective treatment in these patients, with a Grade B recommendation.

One of the most relevant and current studies supporting physical therapy was a prospective multicenter research designed by the French Arthroscopic Society and carried out in 12 different centers [9]. NOT methods included analgesics, anti-inflammatory drugs, rehabilitation, and subacromial corticosteroid injections. Patients were followed up for 3, 6, and 12 months after the start of NOT. ROM, Visual Analogue Scale (VAS), Constant Score, and Subjective Shoulder Value (SSV) were assessed. Out of 71 eligible patients, only three failed to complete the whole NOT program and partial RCT repairs were performed. After 12 months, the remaining 68 presented improvements in mean Constant scores (40.7 ± 17.0 [range 9–75] to 57.1 ± 15.3 [range 20–86]) and in mean weighted Constant score (54.9% ± 22.7% [range 13–103] to 76.8% ± 0.2% [range 31–120]). Also, improvements in mean SSV (39% ± 15.8 [range: 0%–80%] to 65.2% ± 15.8 [range 20%–99%]) and ROM (especially forward elevation: improved from 112.2° ± 45.1 [range 20°-180°] to 137.4° ± 33.1° [range 60°–180°]) were observed.

This study supports the short-term usefulness of NOT in patients with irreparable massive RCTs regardless of the site of the initial tear, which showed no correlation with final functional outcomes or final ROM at 12 months. However, it must be taken into account that both the mean Constant scores and active forward elevation were significantly improved after 3–6 months of follow-up but, on the other hand, neither of them improved significantly between 6 and 12 months. Although it lacked a control group and a longer follow-up, it is the only one to provide data for function and motion range recovery during NOT and concludes that surgery treatment must be considered only in those patients with no or insufficient improvements after 6 months of conservative treatment.

Neri et al. [1] also described, in 2009, that nonoperative management of massive RCTs should only be reserved for those patients whose symptoms did not involve significant pain, because improved function may be achieved with activity modification, judicious use of steroid injections, and physical therapy (focused on anterior deltoid training, reeducation of muscle recruitment, coordination of co-contraction, maintenance of motion or periscapular strengthening).

Most massive tears tend to be classified, according to the location of the tear, as anterosuperior or postero-superior, each with different incidence, clinical presentation, examination findings, and prognosis [1]. Classically, the contraction of the deltoid has been thought to promote the humeral head stabilization beneath the coracoacromial arch, modifying the center of rotation of the humerus in this situation [11]. However, current literature has observed that the deltoid may even play a major role in the prevention of upward migration of the humeral head in shoulders with large RCT [24]. In this research, Gagey et al. examined the orientation of resultant forces along the vertical axis beyond the acromioclavicular joint and in 19/23 shoulders it was noted that the resultant vector was oriented downward [24].

Burkhart [25] radiographically evaluated 12 shoulders with massive, irreparable RCTs and described 3 patterns of glenohumeral kinematics based on fluoroscopy: stable, unstable, and captured fulcrum. Those patients belonging to the first group maintained a stable glenohumeral fulcrum and, therefore, a stable kinematics during elevation. In the second group, an unstable glenohumeral fulcrum led to anterior and superior translation of humeral head in active elevation. In the third group, although the incapacity to keep the humeral head centered in the glenoid cavity was noticeable, the elevation was performed at the undersurface fulcrum of the acromion.

With proper training, he found that the force couples around the joint could be maintained in patients with isolated tears of the supraspinatus, which relatively preserved shoulder function. However, if the tear extended also into the anterior (i.e., subscapularis involvement) or posterior (i.e., posterior infraspinatus or teres minor) cuff tendons, the force balances around the joint were disturbed and could lead to unstable kinematics and loss of function. Burkhart's "suspension bridge" concept argued that function could be maintained, even in the presence of a large tear, if the force balancing about the joint was preserved [25].

Hence, when put into practice, Anterior Deltoid Reeducation (ADR) has been proposed as an alternative treatment to compensate the altered biomechanics in shoulders with RCTs. Although it has been shown to be helpful in the short term, specifically in the debilitated elderly population, little is known about the durability of its benefits and effects on functional outcomes [19]. Levy et al. [19] enrolled elderly patients with debilitated or pseudoparalytic shoulders presenting with anterosuperior RCTs. They followed an ADR program and reported that 82% of patients succeeded in terms of pain, important ROM improvements (mean forward elevation improved from 40° to 160°), and perceived function (measured with Constant Scale). However, their follow-up was only considered for the first 9 months.

The application of protocols based on isolated ADR has proven to be effective ($p < 0.005$) on patients with massive RCT also at long term [26], but only a 40% of success was reported in this case. This protocol consisted on a home-based program applied for a period of 3 months in elderly patients who were followed up during 24 months and assessed for pain, ROM, strength, SSV, and American Shoulder and Elbow Surgeons (ASES) score. According to the results of this study, the success achieved following this ADR program was not statistically dependent on any of the ADR factors analyzed except for ROM: those patients with a forward flexion of less than 50° at the beginning of the ADR program reached to an unsuccessful outcome at 2 years compared to those with a forward flexion of 50° or more ($p < 0.022$).

From our point of view, ADR programs conform to standards in shoulder rehabilitation of massive RCTs, but not as a unique and independent treatment approach.

So much so that, studies such as the one carried out by Collin et al. [13] have reported improvements in a prospective study of 45 patients with irreparable massive RCTs with pseudo-paralysis by using a multimodal specific rehabilitation program. This protocol aimed to reduce pain and scapulo-thoracic dyskinesia, correct faulty humeral head centering, strengthen scapular stabilizers, and restore proprioception. Excellent outcomes were observed in those patients with postero-superior tears (supraspinatus and infraspinatus), but no improvements were seen in those with complete antero-superior tears (subscapularis and supraspinatus tears). Unlike Ainsworth [2, 27, 28] or Levy [19], Collin supports the idea that isolated strengthening of Anterior Deltoid may overstate the anterior decentering of the humerus as well as the isolated eccentric work on humerus depressors (latissimus dorsi and pectoralis major), as other studies have stated [29]. For these reasons, other kind of training approaches considering scapula, voluntary control of humeral head movements, or proprioception may be the key in conjunction to ADR programs, whose effectiveness have been undoubtedly demonstrated.

In this sense, later biomechanical studies have proven that compensatory increases in the deltoid force are required to preserve shoulder function in patients with massive RCTs but also the remaining rotator cuff is essential to improve kinematics in this context [26]. In the presence of a massive RCT, stable glenohumeral abduction without excessive superior humeral head translation requires significantly higher forces in the remaining intact portion of the rotator cuff. These force increases are within the physiologic range of rotator cuff muscles for 6-cm tears and most 7-cm tears [26]. Increases in deltoid force requirements occur in early abduction; however, greater relative increases are required from the rotator cuff, especially in the presence of larger rotator cuff tears [11].

Therefore, while rehabilitation of the deltoid is indeed important, these results highlight the relatively greater importance of rehabilitation of the rotator cuff muscles to prevent superior humeral head translation and subacromial impingement. Indeed, emphasis on rotator cuff strengthening has become a mainstay of NOT [11, 30].

Steenbrick et al. [31] evaluated the electromyograpic muscle activation pattern in 8 patients suffering a massive rotator cuff tear pre- and post-lidocaine injection. Before the injection, they observed a different activation pattern of the adductor muscles (pectoralis major/latissimus dorsi/teres major) comparing to normal shoulders. They noticed a contraction of the humeral head depressors in order to avoid humeral head impingement and pain. After the lidocaine injection, pain disappears and the adductor abnormal pattern becomes normal.

According to this previous biomechanical issue, we believe that the adductor muscles also should be activated in the presence of massive rotator cuff tears, in order to stabilize the humeral head and avoid superior migration as much as possible. Therefore, a specific rehabilitation protocol should include isolated or combined exercises for improving the adductor muscles group.

In 2009, Ainsworth et al. [27] compared the outcomes between elderly patients with massive RCTs whose physiotherapy management included conventional modalities (ultrasound, advice, encouragement, and analgesia) vs. those whose management had the addition of a specific exercise program. A total of 60 patients were recruited and followed up for 12 months and their assessment included: Oxford Shoulder Score (OSS) for shoulder disability, SF-36 for pain and goniometry for range of motion (ROM). Both groups experienced improvements at medium term (12 months) in all variables studied (perhaps because of the fact that increased knowledge about their shoulder condition encouraged them to use the arm without fear of worsening their tears), but those who followed the specific exercise program found greater and faster results at short and short-medium term. Within this study, two conclusions can be drawn with no difficulties: multimodal treatments are probably more effective in reducing pain and improving shoulder function and quality of life in these patients as long as they focus on a physical therapy adapted to the patient condition.

Patient education is fundamental for the patient to understand what the matter is with their shoulder, and they should be taught that pain in the shoulder does not always correlate with worsening harm.

17.2 The Role of Latissimus Dorsi in the Rehabilitations of Massive Rotator Cuff Tears

Scapular dyskinesia (SD) is an alteration associated with shoulder pathologies, producing an abnormal dynamic scapular control. It can be caused by fatigue, neurologic dysfunction, weakness of the periscapular muscles, and intraarticular glenohumeral pathologies such as subacromial impingement and massive rotator cuff tears [31]. Scapular stability based on the periscapular muscles strengthening could improve the ROM, decrease the acromiohumeral contact, and reduce pain.

The stability of glenohumeral joint is mainly given by the rotator cuff and the periscapular muscles. However, in massive RCT the superior subluxation of the humeral head occurs due to the strength of the deltoid muscle. Some authors showed that the periscapular muscles such as latissimus dorsi (LD) and pectoralis major (PM) have an important role in avoiding the superior migration of the humeral head [32]. Halder et al. [33] found, in their biomechanical study, that the depression of the humeral head was most effectively achieved by the latissimus dorsi and the teres major. The activations of these muscles increased after massive rotator cuff tear, showing that LD is the most effective depressor of the humeral head. Hawkes et al. [34] evaluated in

an electromyographic (EMG) study the shoulder muscles activations after massive rotator cuff tear. EMG signal amplitude was significantly higher for the biceps, trapezius-serratus anterior, latissimus dorsi, and teres major. The author concluded that activation of LD is an attempt to compensate the destabilizing forces of the deltoid in massive RCT.

Lee et al. [35] examined the biomechanics of the massive RCT and the role of LD/PM muscles in a cadaveric model, including measurement of kinematics, acromiohumeral contact pressure (migrations of the humeral head), and glenohumeral joint forces. Acromiohumeral contact pressures were undetectable when the LD/PM were loaded but increased significantly after LD/PM unloading, concluding that in massive RCT the LD and PM are effective to improve glenohumeral kinematics, reduce acromiohumeral pressure, and could delay the progression of the cuff tear.

Often, a general program of rehabilitation exercises addressed to strengthen the periscapular muscles is recommended for the NOT of the massive RCT. However, taking into account the aforementioned studies, exercise rehabilitation program must focus also on the LD and PM strengthening to reduce pain, delay the progression of cuff tear arthropathy, and improve shoulder function.

17.3 Exercises Protocol

1. **Mixed Exercises:** Combining both scapular corrections and shoulder movements. Examples:
   - **Wall side with a towel** (especially for serratus anterior deficits): The patient is asked to hold a towel in her hand and place it on a wall with her elbow flexed 90°. Then she is asked to slide the towel diagonally (scapular protaction movement −30°) until the elbow is completely extended. 3 series (s) × 8–12 repetitions (r) (Fig. 17.1).
   - **Frontal elevation with resisted external rotation** (especially for rotator cuff, rhomboids, and medium trapezius deficits): The patient is asked to keep her elbows stuck to her trunk and flexed 90°. Then she is asked to take a band (low resistance) with her hands and externally rotate 15° with each shoulder. From this position, a shoulder frontal elevation up to

Fig. 17.1 Wall side with a towel. Especially for serratus anterior deficits

90° is carried out while holding the band tension. 3s × 8-12r (Fig. 17.2).

- **Frontal elevation with resisted adduction** (especially for latissimus dorsi deficits): The patient is asked to take a band starting from 30° of shoulder abduction and move her arm close to her trunk with her elbow extended. Then she is asked to carry out a frontal elevation up to 90° while holding the band tension. 3s × 8-12r (Fig. 17.3).

2. **Scapulothoratic Exercices:** Aimed to correct scapular dyskinesia by recruiting hypoactive muscles and lengthening hyperactive muscles in the dysfunctional movement. Examples:
   - **Serratus punch:** The Patient standing with her shoulder in 90° of flexion and elbow extended, from a scapular retraction position. The patient is asked to carry out a scapular protraction with the elbow extended against the resistance of a band tied around her back. Exercise specific for serratus anterior when pectoralis minor is hyperactive. 3s × 20r (Fig. 17.4).
   - **Scapular retraction:** The Patient standing with her arms relaxed along his body and elbows extended. She is asked to hold the ends of a band (medium resistance) tied to a fix bar in front of her and to extend her arms to place them closed to her greater trochanters. The movement is completed with a scapular retraction, when the patient tries to join the medial border of both scapulas. Exercise specific for medium trapezius and rhomboids

Fig. 17.2 Frontal elevation with resisted external rotation. Especially for rotator cuff, rhomboids and medium trapezius deficits

Fig. 17.3 Frontal elevation with resisted adduction. Especially for latissimus dorsi deficits

Fig. 17.4 Serratus Punch. Exercise specific for serratus anterior when pectoralis minor is hyperactive

when pectoralis minor and superior trapezius remain hyperactive. 3s × 20r (Fig. 17.5).

- **Horizontal abduction with external rotation:** The patient in prone (also, it could be done with a light dumbbell 0.5–1 kg if the patient can do it), starting from an external rotation (thumbs to the ceiling) and 90–120° of shoulder flexion with elbow extended. She is asked to perform a horizontal abduction up to trunk plane. 3s × 20r (Fig. 17.6).

3. **Anterior Deltoid and Rotator Cuff Exercises**: Exercises based on Torbay Protocol.
   - **Exercise 1:** The patient in supine, with arm extended (also, it could be done with a light dumbbell 0.5–1 kg). She is asked to, firstly, flex the elbow up to 90°. Then she is asked to flex the shoulder toward

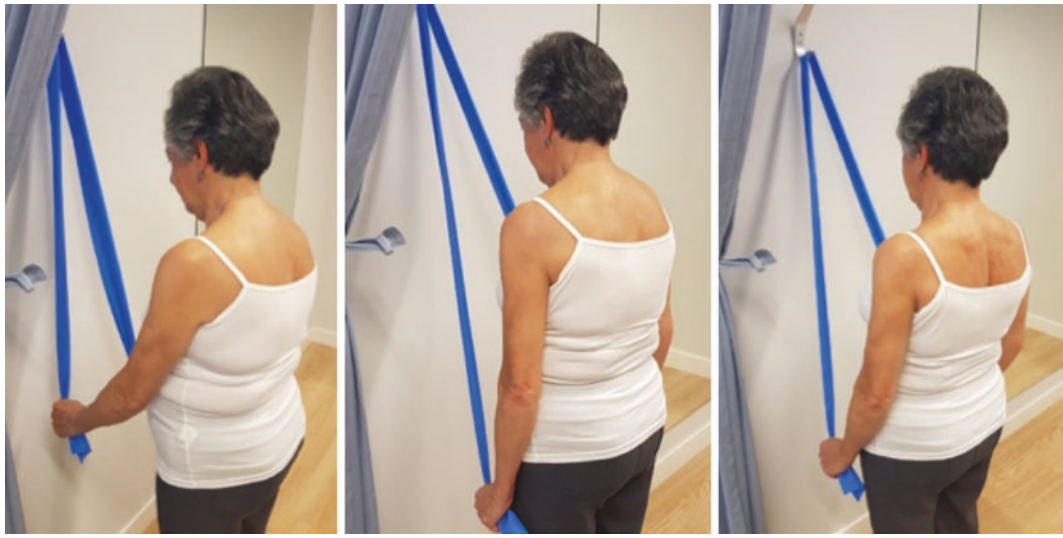


Fig. 17.5 Scapular Retraction. Exercise specific for medium trapezius and rhomboids when pectoralis minor and superior trapezius remain hyperactive

Fig. 17.6 Horizontal Abduction with external rotation

her head with the elbow flexed, and in this position, she extends the elbow toward the ceiling. Finally, the patient slowly swings the arm up and down before coming back to the starting position following the opposite way. 3s × 8-12r (Fig. 17.7).

- **Exercise 2:** The patient in front of a wall with a towel on the affected hand. The patient is asked to slide it up along the wall, with the aid of the opposite hand to reach as far as possible without pain. 3s × 8-12r.
- **Exercise 3:** The patient in lateral decubitus with the elbow next to the body and a light dumbbell begins to do shoulder external rotation to strengthen the remaining rotator cuff (Fig. 17.8).
- **Exercise 4:** The patient standing with the arm next to the body with 90° flexion of the elbow; the subject takes the band and begins to do shoulder internal rotation in

Fig. 17.7 Strengthening the anterior deltoid

order to strengthen the subscapularis tendon (Fig. 17.9).

17.3.1 Patient Education

Patients should be given a thorough explanation of what has happened to their shoulder and why their function is impaired. Time should be spent re-assuring the patient that whilst pain in the shoulder does not always correlate with harm, there is little to be gained by using the shoulder when pain increases. Patients should also be aware of the goals of the rehabilitation program because no progress will be made if the patient fails to engage with the process. Realistic and achievable goals should be set.

Fig. 17.8 Shoulder external rotation to strengthening the remaining rotator cuff

Fig. 17.9 Shoulder internal rotation to strengthening the subscapularis tendon

References

1. Neri BR, Chan KW, Kwon YW. Management of massive and irreparable rotator cuff tears. J Shoulder Elbow Surg. 2009;18(5):808–18.
2. Ainsworth R, Lewis JS. Exercise therapy for the conservative management of full-thickness tears of the rotator cuff: a systematic review. Br J Sports Med. 2007;41:200–10.
3. Nho SJ, Delos D, Yadav H, Pensak M, Romeo AA, Warren RF, MacGillivray JD. Biomechanical and biologic augmentation for the treatment of massive rotator cuff tears. Am J Sports Med. 2010;38(3):619–29.
4. Downie BK, Miller BS. Treatment of rotator cuff tears in older individuals: a systematic review. J Shoulder Elbow Surg. 2012;21(9):1255–61. [PubMed].
5. Ryosa A, Laimi K, Aarimaa V, Lehtimaki K, Kukkonen J, Saltychev M. Surgery or conservative treatment for rotator cuff tear: a meta-analysis [with consumer summary]. Disabil Rehabil. 2016;39(14):1357–63.
6. Moosmayer S, Lund G, Seljom U, et al. Tendon repair compared with physiotherapy in the treatment of rotator cuff tears: a randomized controlled study in 103 cases with a five-year follow-up. J Bone Joint Surg Am. 2014;96(18):1504–14. [PubMed].
7. Lambers Heerspink FO, van Raay J, Koorevaar RCT, et al. Comparing surgical repair with conservative treatment for degenerative rotator cuff tears: a randomized controlled trial. J Shoulder Elbow Surg. 2015;24(8):1274–81.
8. Kukkonen J, Joukainen A, Lehtinen J, et al. Treatment of nontraumatic rotator cuff tears: a randomized controlled trial with two years of clinical and imaging follow-up. J Bone Joint Surg Am. 2015;97(21):1729–37.
9. Agout C, Berhouet J, Spiry C, et al. Functional outcomes after non-operative treatment of irreparable massive rotator cuff tears: prospective multicenter study in 68 patients. Orthop Traumatol Surg Res. 2018;104(8S):S189–92.
10. DePalma AF, White JB, Callery G. Degenerative lesions of the shoulder joint at various age groups which are compatible with good function. Instr Course Lect. 1950;7:168–80.
11. Hansen ML, Otis JC, Johnson JS, Cordasco FA, Craig EV, Warren RF. Biomechanics of massive rotator cuff tears: implications for treatment. JBJS. 2008;90(2):316–25.
12. Gazielly DF, Gleyze P, Montagnon C. Functional and anatomical results after rotator cuff repair. Clin Orthop Relat Res. 1994;(304):43–53.
13. Collin PG, Gain S, Nguyen Huu F, Lädermann A. Is rehabilitation effective in massive rotator cuff tears? Orthop Traumatol Surg Res. 2015;101:S203–5.
14. Zingg PO, Jost B, Sukthankar A, Buhler M, Pfirrmann CWA, Gerber C. Clinical and structural outcomes of nonoperative management of massive rotator cuff tears. J Bone Joint Surg Am. 2007;89:1928–34.
15. Bokor DJ, Hawkins RJ, Huckell GH, Angelo RL, Schickendantz MS. Results of nonoperative management of full-thickness tears of the rotator cuff. Clin Orthop Relat Res. 1993;294:103–10.
16. Itoi E, Tabata S. Conservative treatment of rotator cuff tears. Clin Orthop Relat Res. 1992;275:165–73.
17. Kuhn JE, Dunn WR, Sanders R, An Q, Baumgarten KM, Bishop JY, Brophy RH, Carey JL, Holloway BG, Jones GL, Ma CB, Marx RG, EC MC, Poddar SK, Smith MV, Spencer EE, Vidal AF, Wolf BR, Wright RW, MOON Shoulder Group. Effectiveness of physical therapy in treating atraumatic full-thickness rotator cuff tears: a multicenter prospective cohort study. J Shoulder Elbow Surg. 2013;22(10):1371–9.
18. Oh JH, Park MS, Rhee SM. Treatment strategy for irreparable rotator cuff tears. Clin Orthop Surg. 2018;10(2):119–34.
19. Levy O, Mullett H, Roberts S, Copeland S. The role of anterior deltoid reeducation in patients with massive irreparable degenerative rotator cuff tears. J Shoulder Elbow Surg. 2008;17(6):863–70.
20. Edwards P, Ebert J, Joss B, Bhabra G, Ackland T, Wang A. Exercise rehabilitation in the non-operative management of rotator cuff tears: a review of the literature. Int J Sports Phys Ther. 2016;11(2):279–301.
21. Huisstede BM, Koes BW, Gebremariam L, Keijsers E, Verhaar JA. Current evidence for effectiveness of interventions to treat rotator cuff tears. Man Ther. 2011;16(3):217–30. [PubMed].
22. Seida JC, le Blanc C, Schouten JR, et al. Systematic review: nonoperative and operative treatments for rotator cuff tears [with consumer summary]. Ann Intern Med. 2010;153(4):246–55. [PubMed].
23. Jeanfavre M, Husted S, Leff G. Exercise therapy in the non-operative treatment of full-thickness rotator cuff tears: a systematic review. Int J Sports Phys Ther. 2018;13(3):335–78.
24. Gagey O, Hue E. Mechanics of the deltoid muscle. A new approach. Clin Orthop. 2000;375:250–7.
25. Burkhart SS. Fluoroscopic comparison of kinematic patterns in massive rotator cuff tears. A suspension bridge model. Clin Orthop Relat Res. 1992;(284):144–52.
26. Yian EH, Sodl JF, Dionysian E, Schneeberger AG. Anterior deltoid reeducation for irreparable rotator cuff tears revisited. J Shoulder Elbow Surg. 2017;26(9):1562–5.
27. Ainsworth R, Lewis J, Conboy V. A prospective randomized placebo controlled clinical trial of a rehabilitation programme for patients with a diagnosis of massive rotator cuff tears of the shoulder. Shoulder Elb. 2009;1:55–60.

28. Ainsworth R, Dziedzic K, Hiller L, et al. A prospective double blind placebo controlled randomized trial of ultrasound in the physiotherapy treatment of shoulder pain. Rheumatology (Oxford). 2007;46:815–20.
29. Koubaa S, Ben Salah FZ, Lebib S, et al. Conservative management of full-thickness rotator cuff tears. A prospective study of 24 patients. Ann Readapt Med Phys. 2006;49(2):62–7.
30. Jobe FW, Moynes DR. Delineation of diagnostic criteria and a rehabilitation program for rotator cuff injuries. Am J Sports Med. 1982;10:336–9.
31. Burn MB, McCulloch PC, Lintner DM, Liberman SR, Harris JD. Prevalence of scapular Dyskinesis in overhead and nonoverhead athletes: a systematic review. Orthop Sport Med. 2016;4(2):1–8. https://doi.org/10.1177/2325967115627608.
32. Oh JH, Jun BJ, McGarry MH, Lee TQ. Does a critical rotator cuff tear stage exist?: a biomechanical study of rotator cuff tear progression in human cadaver shoulders. J Bone Joint Surg Am. 2011;93:2100–9. https://doi.org/10.2106/jbjs.j.00032.
33. Halder AM, Zhao KD, O'Driscoll SW, Morrey BF, An KN. Dynamic contributions to superior shoulder stability. J Orthop Res. 2001;19:206–12.
34. Hawkes DH, Alizadehkhaiyat O, Kemp GJ, Fisher AC, Roebuck MM, Frostick SP. Shoulder muscle activation and coordination in patients with a massive rotator cuff tear: an electromyographic study. J Orthop Res. 2012;30:1140–6.
35. Campbell ST, Ecklund KJ, Chu EH, McGarry MH, Gupta R, Lee TQ. The role of pectoralis major and latissimus dorsi muscles in a biomechanical model of massive rotator cuff tear. J Shoulder Elbow Surg. 2014;23(8):1136–42.

18 Patient Expectation in the Treatment of Rotator Cuff Tears: What Is Its Role?

Roger Hackney, Emma Pollard, and Paul Cowling

18.1 Introduction

Greater patient involvement in discussion and decision-making in managing their care is demanded by patients and governing bodies alike, worldwide. Proper informed consent is a legal necessity. Patient involvement and understanding leads to a patient having a realistic expectation of outcome. This is based on the information supplied by the surgeon, but increasingly from the information gained from other sources such as the internet and lay media. These latter sources are not regulated and may not be entirely correct factually.

Aside from avoiding medico-legal challenges, does a better understanding improve the patient's perception of their care and outcomes?

18.2 Literature Review

There is very little in the literature regarding patient expectations of orthopaedic conditions and interventions in general, let alone of large and massive rotator cuff tears. Zywiel et al. [1] performed a systematic literature review to investigate how to measure expectations in shoulder surgery. They found that although some validated expectation instruments have been developed, there is little evidence of testing or validation. More recently, Swarup et al. [2] investigated patient satisfaction around elective orthopaedic surgery and found that pre-operative patient expectations are associated with post-operative patient outcomes and satisfaction. They also concluded there are very few validated measures of patient satisfaction in orthopaedic surgery.

There are few research studies investigating patient expectations from shoulder surgery. Work has been done in the Unites States, primarily at the Hospital for Special Surgery, New York, to investigate patient expectations of shoulder surgery [3]. They devised a 'patient-derived shoulder surgery expectation survey' after interviewing 509 patients with varying shoulder problems, including a large proportion of patients with a 'complete rotator cuff tear'. In their interviews, they found that 48% of patients expected complete pain relief from any operative intervention for their shoulder disorder. This scoring system was to be used prior to any shoulder surgery. The authors recommend it can be used to learn about a patient's perspective of surgery and provide the treating surgeon with a template to guide a discussion about how realistic their goals are. In validating this scoring measure, Henn III et al.

R. Hackney (✉)
Spire Hospital, Leeds, UK
e-mail: roger.hackney@nhs.net

E. Pollard
Nuffield Hospital, Leeds, UK

P. Cowling
The General Infirmary, University of Leeds, Leeds, UK
e-mail: cowling@doctors.org.uk

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_18

[4] found that younger patients had higher expectations of total shoulder replacement.

When looking specifically at patient expectations of rotator cuff treatment, most studies examine this with surgical management in mind. Tashjian et al. [5] investigated patient satisfaction with rotator cuff repair surgery. In this study, a self-assessment questionnaire was used to assess patient expectations: Musculoskeletal Outcomes Data Evaluation and Management System (MODEMS) [6]. This is an instrument for collecting musculoskeletal outcome data using several questionnaires, including questions around comorbidities, work status and education level. Six questions concerning how likely a patient expected a certain change in function or activity following treatment were asked, including relief from symptoms, ability to perform everyday household tasks, improved sleep, return to usual job, participation in recreational activities and prevention of future disability. They found that if the pre-operative patient expectations could be met, post-operatively, patients had higher satisfaction. Higher pre-operative and post-operative met expectations led to higher patient satisfaction.

The same authors used the MODEMS score to assess 125 patients undergoing primary repair of a chronic rotator cuff tear [7]. They found that patients overall had high expectations regarding surgical management of rotator cuff tears, and greater than 85% expected surgery was 'very likely' or 'extremely likely' to improve each parameter asked. They found that the greater the pre-operative patient expectations, the better the post-operative performance of standard functional outcome scores (DASH, SST, SF-36), and that even when controlling results for age, gender, smoking, comorbidities and a host of cuff tear specific factors, greater expectations were a significant independent predictor of a better performance at 1 year following rotator cuff repair.

The MODEMS was also used in a study into the effect of expectations and concerns around rotator cuff repair [8]. They found that on the whole, the patient demonstrated high levels of expectations for surgical treatment of a rotator cuff tear. Their cohort's main expectation of their post-operative function was 'relief from symptoms', followed by 'prevent further disability', 'to perform everyday household tasks', 'to participate in recreational activities', 'sleep more comfortably', and 'to return to usual job'.

The patients with the highest expectations (the top 33% in expectation score) demonstrated significantly different factors to patients with lesser expectations: higher expectations were found in patients who were employed, had high levels of information pre-operatively, where the information was provided by their surgeon, and had a poorer pre-operative Constant Score. Interestingly, those patients with higher expectations demonstrated significant improvement from their baseline functional scores (SST, Constant score, SF-36 score). In this study, patients were also asked questions regarding 64 different pre-operative 'concerns'. The authors found no difference in clinical outcomes in patients with high pre-operative concerns compared to these classified as of low concern.

Few studies investigated patient expectations related to physiotherapy for rotator cuff tears. Chester et al. [9] used a cohort of 1030 patients with shoulder pain to identify baseline patient and clinical characteristics that led to a better outcome after commencing physiotherapy. They found that patient expectation of a 'complete recovery' compared to 'slight improvement' as a result of physiotherapy was a factor associated with better outcomes.

It therefore seems that, although there is little in the literature regarding patient expectations of the treatment of rotator cuff tears, often patient expectations can be high, and can be influenced by employment, pre-operative information provided by their surgeon and their own pre-operative shoulder function. However, patients with greater expectations of the treatment of their rotator cuff often record better functional outcomes after the intervention [10].

18.2.1 Patient Expectations When Managed with Physiotherapy

The role of physiotherapy in the management of large to massive rotator cuff tears is well docu-

mented [11–14]. Massive cuff tears compromise the normal biomechanics of the shoulder. Re-education of the deltoid muscle is key in optimising the function of the deltoid along with the remaining uninjured rotator cuff muscles and other parascapular muscles, in order to maintain overhead function [15–17].

For patients who present with massive cuff tears and pseudoparalysis, treatment is likely to include the anterior deltoid program [11], along with posture correction, activity modification and strengthening of the other non-injured muscles of the rotator cuff and shoulder complex.

Several studies have shown a correlation between pre-operative expectations and outcomes of orthopaedic surgery [7, 18–20]. Sociodemographic patient factors such as age, marital status, employment status, co-morbidities and level of education have also been shown to influence these outcomes [1, 21–24] and recently have also been linked to outcomes of non-operative treatment of rotator cuff tears [25, 26]. In the study by Jain et al. [25], baseline questionnaires were used to assess function such as the shoulder pain and disability index (SPADI), fear avoidance beliefs questionnaire (FABQ) and mental health (MHI-5), which is a component of the Short Form health survey (SP 36). Outcomes were measured at 3-, 6- and 18-month intervals using the SPADI, and results showed better outcomes for those who were married, with lower alcohol consumption, had shorter duration of symptoms, in light/non-manual work and those with a college level of education and above. Such characteristics may be an indication of patient compliance and pain coping strategies.

Boorman et al. [26] used the rotator cuff quality-of-life index (RC-QOL), which also looked at baseline demographic characteristics such as sex, length of symptoms, age and whether they were smokers or non-smokers. In addition, hand dominance and strength/range of motion (ROM) were recorded. Arm dominance may affect the QOL for those with symptoms on their dominant side, as would have made functional tasks more challenging. Despite this possible confounding factor, there is evidence that the results support the use of such questionnaires in helping to predict outcomes and optimising clinical decisions when choosing how to manage massive rotator cuff tears non-operatively.

It is acknowledged that expectations are an integral part of the psychosocial make-up of each individual [27]. Addressing each patient on an individual basis in terms of pain and dysfunction, with awareness and consideration of the above sociodemographic and psychosocial characteristics may therefore be key to a successful outcome.

The first patient/therapist consultation is paramount in the rehabilitation process. It is important that the patients are educated regarding their diagnosis and the role of physiotherapy. With the increase in often-poor quality, but readily accessible, information via the internet [28], the first contact is key to educating the patient concerning their diagnosis and the treatment options available to them. It allows patients to discuss their expectations of physiotherapy, to express any concerns, including any risks and benefits of their treatment and helps them to engage with their treatment plan, as failure to do so may result in poorer outcomes from poor compliance. This is the opportunity to set realistic goals and treatment plans.

Education is key. For those with inoperable rotator cuffs tears, it is important that the patient understands the role of the rotator cuff and the fact that massive cuff tears can comprise the normal biomechanics of the shoulder. This allows patients to understand why they may present with reduced function and pain and thus why specific exercises are chosen in hope to improve these symptoms. Understanding that the aim of physiotherapy is not to ‘heal’ the damaged tendon but instead to re-educate and train other muscles of the shoulder to compensate for the cuff tear, in the hope of reducing pain and restoring the function of the upper limb.

In addition to strengthening exercises, other treatments may include postural correction in order to obtain optimum positioning of the humeral head prior to exercise, proprioceptive rehabilitation, ROM exercises and modification of activities of daily living. Other adjuncts such as pain relief, use of heat and manual treatment may also be used [11, 12, 17, 29].

Patients need to expect to partake in regular home exercise–based program along with regular visits to the physiotherapist for guidance and progression of their rehabilitation program. Successful outcomes require commitment and compliance from the individuals, and they must be informed that progress does not happen overnight but can take weeks/months of hard work.

Studies have shown successful outcomes in the conservative management of rotator cuff tears when exercise programs are carried out regularly over a 3-month period. However, some suggest that a longer period of 6–9 months may be required [12, 14, 17, 29].

Across physiotherapy, there appears to be no 'gold standard' to the amount of repetitions, sets or how often the program is carried out. Studies report that the frequency of home exercise program is often varied [29, 30].

There are many factors that may dictate how many repetitions, sets and how frequent a home exercise program should be carried out and is very much chosen on an individual basis. Consideration of pain, irritability and patient ability are all taken into account.

18.3 Implications for Personal Practice

The literature review suggests that patients who have better expectation of outcome perform better in follow-up studies, but in the days of informed consent and patient involvement in decision making, it is crucial to set realistic targets and provide a realistic expectation of outcome. This is especially important in the management of large to massive tears where outcomes are not always as good as the surgeon would wish for. If the surgeon and patient have widely different expectation of outcome, the disappointed party is usually the patient, and none of us wish for unhappy patients. Younger patients will often have researched the internet and come to clinic with some fixed ideas, not always based on evidence and fact.

Patients with large and massive tears have a wide variety of presentations, from mild discomfort and weakness, to a flail or pseudoparalytic shoulder with awful pain and loss of function. Management is adjusted according to the patient's individual needs and the surgeon's own particular preferences in management.

In the UK, patients often present to secondary care having already undergone a course of physiotherapy. Unfortunately, this is frequently of dubious quality from a non-specialist physiotherapist. They may have undergone basic investigation to identify the tear of the rotator cuff.

Once the individual patient's levels of pain and loss of function have been assessed, further investigation is usually required. It is the practice of the corresponding author to draw a picture for the patient to explain the anatomy and function of the rotator cuff, what happens when a tear occurs and what the options are for anyone with a large to massive cuff tear. The pros and cons of rehabilitation, the risks (with regard to increased risk of any subsequent joint replacement) and benefits of steroid injection are all detailed. It is explained that the programme offered by a specialist physiotherapist is not the same as that from a non-specialist and that some very satisfactory outcomes can be obtained with physiotherapy alone. It may take 2–3 months, however. All of the surgical options are detailed, to give the patient an overall picture of what is available for their care. These are divided into simple arthroscopic surgery, potential repair of the rotator cuff and possible augmentation with a patch, and on to reverse total shoulder replacement. The outcomes of these various options are provided.

Even with simple debridement perhaps including biceps surgery, the short to medium may be quite good, and about two-thirds of patients do satisfactorily, according to the literature.

If an arthroscopic repair is achievable, patients will undergo a period of 4 weeks' immobilisation in a sling, but they are warned about the lengthy period of rehabilitation and the risk of re-rupture. I do believe it is important to fully inform patients of the time it takes to recover.

The patch augmentation provides better outcomes and reduced risk of recurrence, but for very large tears, open surgery is employed, which carries with it a higher risk of infection com-

pared with arthroscopic surgery. Severe muscle wasting may not recover or take over a year to regain power with overhead activity. The patient is shown the sagittal MRI scan to demonstrate the amount of wasting/fatty infiltration.

When discussing other management options that are not fully researched or proven such as balloon arthroplasty and superior capsular reconstruction, the surgeon should describe a summary of published literature and be honest with regard to the evidence, benefits and risks.

All patients need to know the time in sling and time off work/driving/lifting. They are told that strengthening work does not start for 10–12 weeks so that manual workers understand the reason for the time off lifting.

Where patients have osteoarthritis and a reverse total shoulder is an option, then full counselling of the advantages and disadvantages is provided.

This information is all provided on a sheet for the patient to take home with them, as it is well established that patients retain less than half the information provided in a clinic setting.

Rotator cuff repair can be extremely painful. An audit of my patients showed a tendency for larger tears to be more painful. The audit led to an improvement in practice and a fuller explanation from the anaesthetist as to how the pain will be managed. My patients undergo day case surgery with a general anaesthetic and interscalene nerve block. The addition of 3.3 mg dexamethasone to the block increases the duration of the block by 50%, with many patients experiencing pain relief for well over 24 h. Patients are then supplied with the World Health Organisation analgesic ladder of analgesia to use post-operatively. The interscalene block should last until the post-operative morning. Patients are encouraged to take paracetomol and dihydrocodeine 6 hourly with a non-steroidal starting the morning after surgery even if full resolution of the interscalene block has not yet occurred. Oromorph is used for rescue pain relief only if required. An audit revealed that only 10% required this, prior to using dexamethasone this was 40%. Using this regimen has significantly improved patient satisfaction rates.

18.4 Conclusion

Patients in the modern era are encouraged to be more involved in the decision-making process. In order to facilitate this, the patient should be fully informed of the benefits and risks of any management options offered and the alternatives. Once the patient has been fully informed and understands the path ahead, the result will be improved outcome and the happier and more satisfied patient.

References

1. Zywiel MG, Mahomed A, Gandhi R, Perruccio AV, Mahomed NN. Measuring expectations in orthopaedic surgery: a systematic review. Clin Orthop Relat Res. 2013;471:3446–56.
2. Swarup I, Henn CM, Gulotta LV, Henn RF III. Patient expectations and satisfaction in orthopaedic surgery: a review of the literature. J Clin Orthopaed Trauma. 2019;10(4):755–60.
3. Mancuso CA, Altchek DW, Craig EV, Jones EC, Robbins L, Warren RF, Williams-Russo P. Patients' expectations of shoulder surgery. J Shoulder Elb Surg. 2002;6:541–9.
4. Henn IIIRF, Ghomrawi H, Rutledge JR, Mazumdar M, Mancuso CA, Marx RG. Preoperative patient expectations of total shoulder arthroplasty. J Bone Joint Surg Am. 2011;93:2110–5.
5. Tashjian RZ, Bradley MP, Tocci S, Rey J, Henn RF, Green A. Factors influencing patient satisfaction after rotator cuff repair. J Shoulder Elb Surg. 2007;16:752–8.
6. American Academy of Orthopaedic Surgeons. Musculoskeletal outcomes data evaluation and management system. Rosemont, IL: American Academy of Orthopaedic Surgeons; 1997.
7. Henn IIIRF, Kang L, Tashjian RZ, Green A. Patients' preoperative expectations predict the outcome of rotator cuff repair. J Bone Joint Surg Am. 2007;89(9):1913–9.
8. Oh JH, Yoon JP, Kim JY, Kim SH. Effect of expectations and concerns in rotator cuff disorders and correlations with preoperative patient characteristics. J Shoulder Elb Surg. 2012;21:715–21.
9. Chester R, Jerosch-Herold C, Lewis J, Shepstone L. Psychological factors are associated with the outcome of physiotherapy for people with shoulder pain: a multicentre longitudinal cohort study. Br J Sports Med. 2018;52(4):269–75.
10. Chung SW, Park JS, Kim SH, Shin SH, Oh JH. Quality of life after arthroscopic rotator cuff repair evaluation using SF-36 and an analysis of affecting clinical factors. Am J Sports Med. 2012;40(3):631–9.

11. Ainsworth R. Physiotherapy rehabilitation in patients with massive, irreparable rotator cuff tears. Musculoskeletal Care. 2006;4(3):140–51.
12. Ainsworth R, Lewis J. Exercise therapy for conservative management of full thickness tears of the rotator cuff: a systematic review. Br J Sports Med. 2007;41:200–10.
13. Jeanfavre M, Husted S, Leff G. Exercise therapy in the non-operative treatment of full-thickness rotator cuff tears: a systematic review. Int J Sports Phys Ther. 2018;13:335–78.
14. Edwards P, Ebert J, Joss B, Bhabra G, Ackland T, Wang A. Exercise rehabilitation in the non-operative management of rotator cuff tears: a review in the literature. Int J Sports Phys Therapy. 2016;11(2):279–301.
15. Burkhart SS. Fluroscopic comparison of kinematic patterns in massive rotator cuff tears. A suspension bridge model. Clin Orthop Relat Res. 1992;(284):144–52.
16. Levy O, Mullett H, Roberts S, Copeland S. The role of anterior deltoid re-education in patients with massive irreparable cuff tears. J Shoulder Elb Surg. 2008;17:863–70.
17. Thes A, Hardy P, Bak K. Decision making in massive rotator cuff tear. Knee Surg Tramatol Arthrosc. 2015;23:449–59.
18. Mahomed NN, Liang MH, Cook EF, Daltroy LH, Fortin PR, Fossel AH. The importance of patient expectations in predicting functional outcomes after total joint arthroplasty. J Rheumatol. 2002;29:1273–9.
19. Mondloch M, Cole D, Frank J. Does how you do depend on how you think you'll do? A systematic review of the evidence or a relation between patients recovery expectations and health outcomes. Can Med Assoc J. 2001;165(2):174–8.
20. Barron C, Klaber MJ. Patient expectations of physiotherapy: definitions, concepts and theories. Physiother Theory Pract. 2007;23(1):37–46.
21. Chester R, Jerosch-Herold C, Lewis J, Shepstone L. Psychological factors are associated with the outcome of physiotherapy for people with shoulder pain: a multicentre longitudinal study. Br J Sports Med. 2018;52(4):269–75.
22. Warth RJ, Briggs KK, Dornan GJ, Horan MP, Millett PJ. Patient expectations before arthroscopic shoulder surgery: correlation with patients' reasons for seeking treatment. J Shoulder Elb Surg. 2013;22:1676–81.
23. Mancuso C, Altchek D, Craig E, Jones E, Robbins L, Warren R, Williams-Russo P. Patient expectations of shoulder surgery. J Shoulder Elb Surg. 2002;11:541–9.
24. Pogorzelski J, et al. Postoperative outcomes—are we asking the right questions? Shoulder arthroscopy patient quality of life correlates with joint-specific outcome and is predicted on patient expectation. Arthroscopy. 2017;33(10):1786–7.
25. Jain N, Ayers G, Fan R, Kuhn J, Baumgarten K, Matzkin E, Higgins L. Predictors of pain and functional outcomes after the nonoperative treatment of rotator cuff tears. J Shoulder Elb Surg. 2018;27(8):1393–400.
26. Boorman R, More K, Hollinshead R, Wiley J, Brett K, Mohtadi N, Nelson A, Lo I, Bryant D. The rotator cuff quality of life index predicts the outcome of nonoperative treatment of patients with a chronic rotator cuff tear. J Bone Joint Surg. 2014;96:1883–8.
27. Chartered Society of Physiotherapy. Core standards of physiotherapy practice. London: Chartered Society of Physiotherapy; 2005.
28. Dalton D, Kelly E, Molony D. Availability of accessible and high-qaulity information on the internet for patients regarding the diagnosis and management of rotator cuff tears. J Shoulder Elb Surg. 2015;24:135–40.
29. Levy O. Deltoid re-education program for massive cuff tears. Oper Tech Orthopaed. 2018;28:1790190.
30. Seida JC, LeBlanc C, Schouten JR, Mousavi SS, Hartling L, Vandermeer B, Tjosvold L, Sheps DM. Systematic review: nonoperative and operative treatments for rotator cuff tears. Ann Intern Med. 2010;153:246–55.

19 Treatment of Massive Irreparable Cuff Tears: Decision Making Process

Alessandra Scaini, Andrea G. Salvi, and Giuseppe Milano

19.1 Introduction

Massive irreparable rotator cuff tears (MIRCTs) are defined as rotator cuff tears (RCTs) that cannot be repaired with adequate mobilization and advanced arthroscopic techniques, because of their inability to reach the anatomic footprint on the humeral head [1–5].

Massive RCTs are not synonymous with irreparable, as all irreparable lesion are massive, but the opposite is not true. Massive tears are defined as defects involving two or more tendons or measuring >5 cm; Burkhart observed that 85% of massive lesions are at least reparable. Although repair can be achieved, failure rate of massive tears is high, ranging between 25 and 94% [6–9].

The identification of true irreparable tears is a challenge, and often diagnosis is confirmed only during arthroscopy after an unsuccessful attempt to mobilize and advance the torn tendons to the footprint. Nevertheless, irreparability is usually due to the size of the lesion, retraction, fatty degeneration, and muscle atrophy, and therefore, diagnostic criteria are proposed based on imaging features [2, 10]. Applying that reasoning, criteria to describe MIRCTs are in general defect size >3 cm, advanced fatty infiltration (stage ≥3 of Goutallier's classification), reduced acromiohumeral distance (AHD), and significant tendon retraction (stage >3 of Patte's classification) with muscular atrophy and poor tissue quality [4, 11, 12].

From this perspective, many authors [13–16] tried to correlate specific preoperative magnetic resonance (MR) findings with reparability of RCTs (Table 19.1). Kim et al. [13] proposed a quantitative scoring system (Reparability Index) to predict if the tear is reparable.

Table 19.1 Preoperative radiographic risk factors for irreparability of RCTs

	Sugihara [13]	Yoo [14]	Dwyer [15]	Kim [16]
Tear size (length)	> 4 cm	> 3.1 cm		Lateral >4.2 cm AP > 3.7 cm
Fatty infiltration (Goutallier classification)	Severe	SS > 4 IS > 3	Severe	>3
Tendon retraction (Patte classification)			>3	>3
Superior humeral migration			Yes	

A. Scaini · A. G. Salvi · G. Milano (✉)
Department of Bone and Joint Surgery, Spedali Civili, University of Brescia, Brescia, Italy

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_19

Besides tear characteristics, history and clinical exam are crucial to identify patient-related factors for reparability. For example, traumatic versus atraumatic (or acute versus chronic) onset can drive us to a correct assessment, because traumatic tears may be more likely to be reparable since they are not associated with significant atrophy, tendon retraction, and fatty degeneration [10, 17]. Age and activity level are also relevant to define the patient's demand, which is relevant to choose correct treatment [3, 10, 17, 18].

Over the years, many treatments have been proposed with good results, the real challenge being to choose the most adequate treatment for each patient. At this aim, some authors proposed treatment algorithms [3, 10, 17] (Fig. 19.1). Unfortunately, no clear evidence exists on the efficacy of different surgical options to address MIRCTs, and further studies are necessary to determine specific indications based on patient and anatomical features and to assess safety, efficacy, and adequacy of treatments.

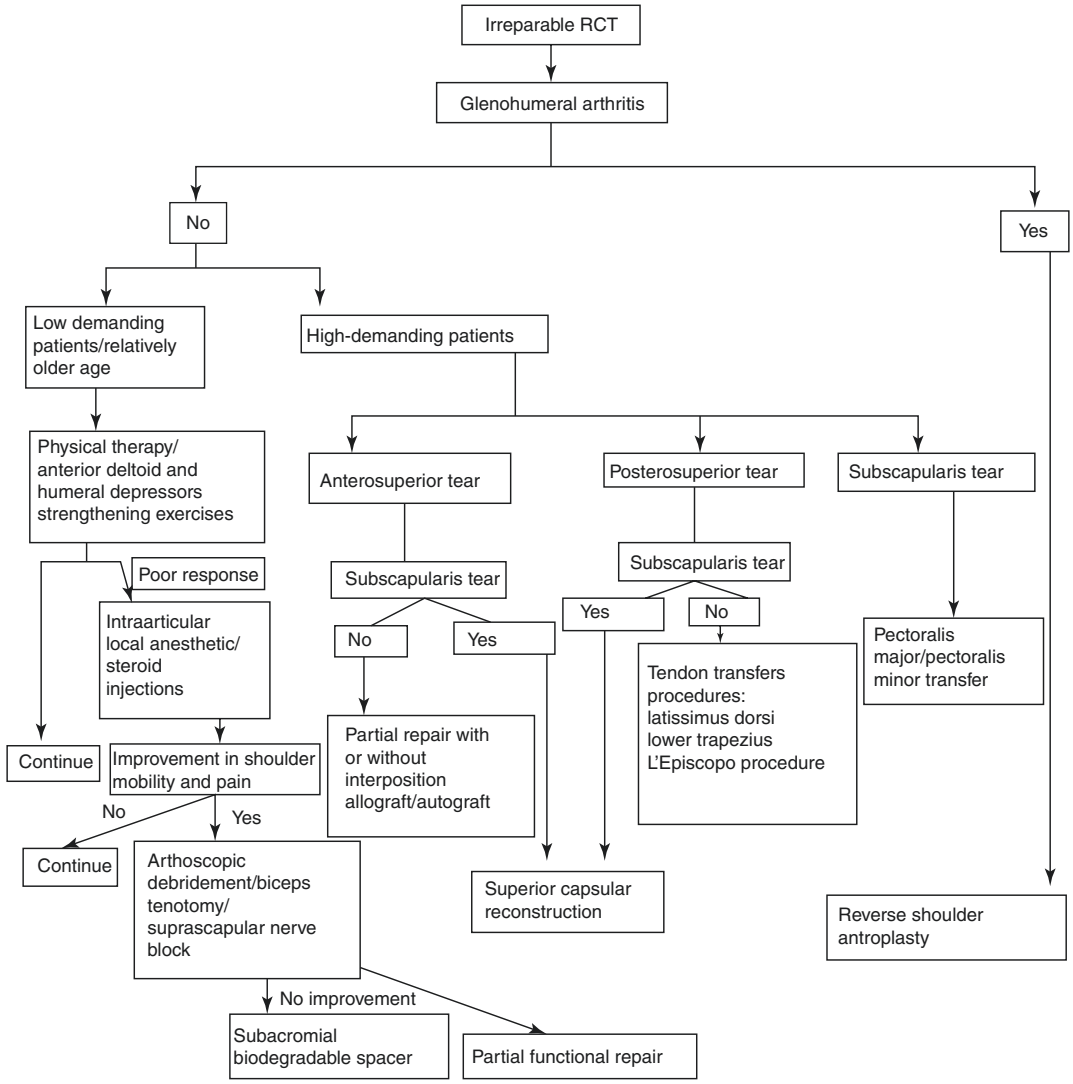


Fig. 19.1 Treatment algorithm proposed by Novi et al. [3]

19.2 Nonoperative Treatment

Nonoperative management is based on activity modification, steroid injections, and physical therapy, with a focus on rehabilitation of the anterior deltoid, which is paramount for the overhead activities [19, 20].

The recruitment should be mainly based on the patient's features. According to Dunn et al. [21], some specific patient-related factors can be used as predictors of treatment failure and need for surgery, such as low expectations of physical therapy, high activity level, and smoking. Conversely, anatomic characteristics of RCTs seem not to be correlated to the conservative treatment outcomes.

If conservative management does not provide satisfactory results, surgery should be considered over a period of 6 months in chronic tears and 6 to 8 weeks in acute scenario [17].

19.3 Isolated Biceps Tenotomy

The long head of the biceps tendon (LHBT) pathology is almost always present in an RCT scenario. The improvement of symptoms after a spontaneous rupture of the biceps tendon in the patient with chronic RCTs suggested that the isolated management of LHBT pathology can be enough. To achieve this result, both tenotomy and tenodesis can be performed; tenotomy drawbacks are loss of biceps strength (about 20% in forearm supination and elbow flexion), popeye's deformity, and risk of superior migration of the humeral head.

Some authors showed an improvement in Constant score without significant decrease in AHD (average 1.1 mm) or progression to glenohumeral osteoarthritis (OA); however, they contraindicated this procedure in case of pseudoparalysis or severe cuff tear arthropathy (CTA) [22, 23]. On the other hand, according to Klinger et al. [24], there is no difference in the outcomes by adding LHBT tenotomy to a debridement procedure in patients with MIRCT.

19.4 Suprascapular Nerve Release

Few studies suggested that in massive posterosuperior RCTs there could be a suprascapular nerve (SNN) neuropathy due to tendon retraction. Diagnosis is based on physical examination and positive electromyography (EMG) signs [25]. Considering this, some authors proposed the release of the SNN at the scapular notch; by the way the real impact on outcome of this procedure is controversial. Some authors found an improvement in pain and function in their case series [26, 27], while Lafosse et al. did not observe differences in comparison with cuff repair alone [28].

19.5 Arthroscopic Debridement

Proposed in 1995 by Rockwood, the original technique included tendon debridement, subacromial decompression with anterior acromioplasty, and resection of coracoacromial (CA) ligament. Currently there is no consensus about resection of CA ligament, which can be apparently related to superior humeral head migration [1, 29, 30]. Debridement should be proposed after the failure of conservative treatment in the patient with a MIRCT without a high-degree OA [30]. Moreover, since the goal of the procedure is just to relieve pain and not to regain shoulder movements, it is best suitable for elderly patients with low functional demand, albeit it does not slow progression of osteoarthritis [17, 31].

Outcome studies showed some improvement in functional and subjective scores probably related to the analgesic effect of the procedure. As far as the effect on the range of motion (ROM), it is sure that pain reduction allows regaining some degree of motion without a real enhancement in shoulder function [18, 32, 33].

Several preoperative factors were analyzed to understand who can really benefit of the procedure. Tear size, age, and Goutallier's stage seem to not be related to outcomes, whereas the reduction of the AHD and the presence of a subscapularis (SSC) tear could have a negative

impact. There is no consensus about the effect of preoperative OA, working activity, and gender [24, 32–36].

19.6 Tuberoplasty and Reverse Arthroscopic Subacromial Decompression

Since the important role played by the coracoacromial arch and coracoacromial ligament in anterior-superior stability of the humeral head, Fenlin et al. [37] developed a procedure called tuberoplasty. The objective of this technique was to make more congruent the acromiohumeral articulation by means of an open resection of the exostosis of the greater tuberosity; in their case series, they reported 95% satisfactory results in 19 patients [37].

The term "reverse arthroscopic subacromial decompression" (RASD) was introduced by Scheibel et al. [38]. In their procedure, the authors performed an arthroscopic debridement of the subacromial space and arthroscopic tuberoplasty, with or without biceps tenotomy. Both Scheibel et al. [38] and Verhelst et al. [39] reported good mid-term results with respect to pain relief, functional recovery, and patient satisfaction following RASD. They observed a decrease in the AHD postoperatively, probably due to biceps tenotomy.

Lee et al. [40] presented satisfactory results in patients who underwent acromioplasty and tuberoplasty procedure.

By the way, none of the studies could state that tuberoplasty was responsible by itself of good results recorded, as it was combined with bursectomy and treatment of the biceps tendon. For this reason, the procedure is seldom used in clinical practice.

19.7 Partial Repair

Partial repair is a technique first described by Burkhart in 1994 to restore the transverse force couple and a stable fulcrum for the glenohumeral joint. This technique includes a repair of the infraspinatus (IS) (at least the inferior half) and the SSC tendon, creating a "suspension bridge," while leaving the irreparable supraspinatus (SS) unrepaired [41]. For this assumption, partial repair is indicated in patients with reparable IS and SSC and irreparable SS, without glenohumeral OA (Hamada stage <2) and no improvement after conservative management (Fig. 19.2). Partial repair is not indicated in patients with deltoid deficiency [17, 18, 42, 43].

Many studies [42, 44–47] showed significant functional improvement with a significant increase in forward flexion, external rotation, and

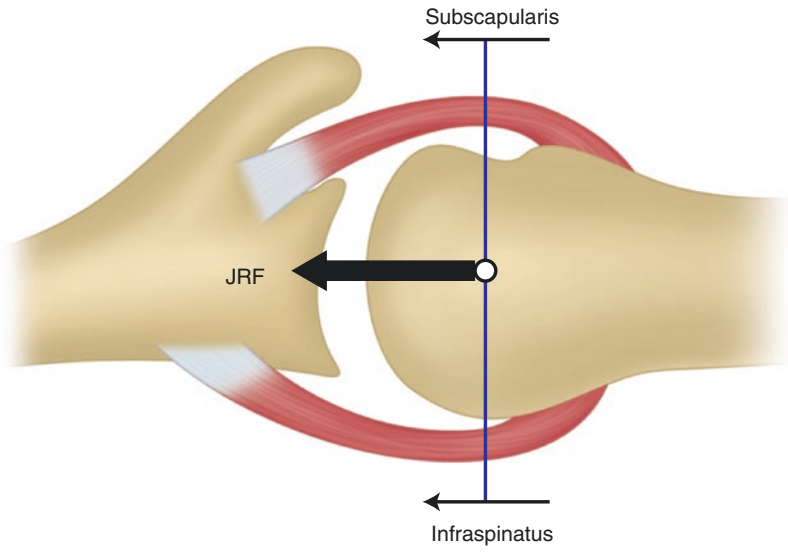


Fig. 19.2 Partial repair is used to restore the transverse force couple and a stable fulcrum for the glenohumeral joint. This technique includes a repair of the infraspinatus (at least the inferior half) and the subscapularis tendon, creating a "suspension bridge," while leaving the irreparable supraspinatus unrepaired (JRF: joint reaction force)

internal rotation, albeit no difference in forward flexion and external rotation was reported in two studies [42, 44, 47].

There is no consensus about factors affecting the outcome of the procedure. Galasso et al. [42, 44, 47] found that demographic factors like male gender and young age were associated with a greater improvement in strength in abduction, external rotation, and internal rotation, albeit other authors found no association between age and gender and clinical outcome [44, 45, 47, 48]. Preoperative duration of symptoms had no effect on clinical outcome; no association was reported with diabetes, smoking status, and AHD, while there was a greater improvement in patients with preoperative night pain, high VAS score, and low ASES score [44, 48]. Shon et al. [45] observed that fatty infiltration of teres minor (TM) was the only preoperative factor associated with poor outcome.

Despite promising clinical outcome, there is a high failure rate after this procedure (41.6%), and most of these patients turn into reverse total shoulder arthroplasty (RTSA) [42, 44, 47, 48].

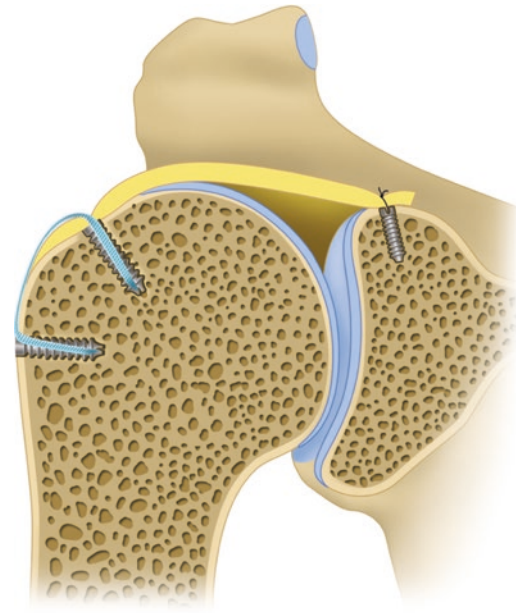


Fig. 19.3 Superior capsule reconstruction creates a static restraint against upper migration of the humeral head. The graft is secured to the superior glenoid and the greater tuberosity, with additional sutures between the patch graft and the residual rotator cuff tendon

19.8 Superior Capsule Reconstruction

Superior capsule reconstruction (SCR) consists of a reconstruction of the superior glenohumeral joint capsule to avoid the superior migration of the humeral head often associated with MIRCTs. Superior capsule reconstruction was first described by Mihata in 2012 [49–51]. In the original technique, the static restraint was secured suturing a fascia lata autograft to the superior glenoid and the greater tuberosity, with additional sutures between the patch graft and the residual rotator cuff tendon [49, 52] (Fig. 19.3). In the last few years, several modified procedures using acellular dermal allograft (Fig. 19.4) or proximal long head of biceps (Fig. 19.5) instead of fascia lata autograft have been proposed [51–54].

Indications for SCR are patients with massive contracted tear of the superior cuff, superior migration of the humeral head, intact or reparable SSC, intact TM, and no severe CTA (Hamada stage ≤3); fixed high riding humeral head is a

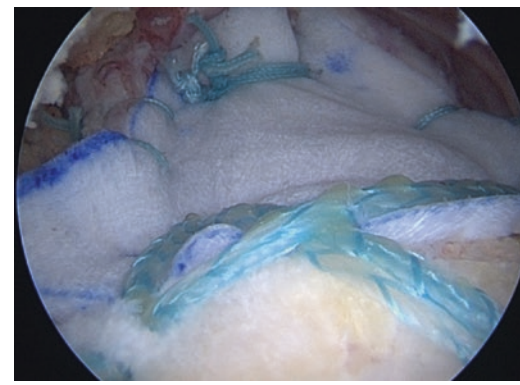


Fig. 19.4 Superior capsule reconstruction with extracellular dermal matrix

contraindication only if the humeral head cannot be reduced during the surgery [52, 55].

Clinical reports on case series showed an improvement in elevation, external and internal rotation, subjective and functional scores, and an increase of AHD. Furthermore, no progression of OA is described in a significant number of patients after SCR [52–54, 56].

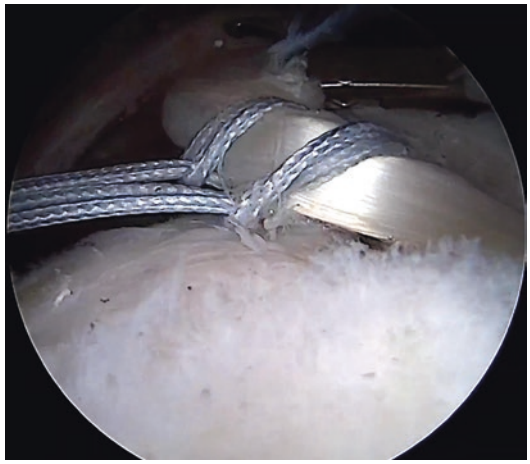


Fig. 19.5 Superior capsule reconstruction with the proximal stump of the long head of the biceps tendon

In the current literature, failure rate is between 16.7% and 36.1%, and revision surgery occurs in 18.6% of the patients where the most are conversion to RTSA [18, 52, 53, 57]. Predictive factors for re-tear are poor posterior remnant tissue, which cannot provide continuity with the graft and inadequate postoperative AHD improvement [57].

19.9 Graft Interposition

Bridging graft interposed in the gap between irreparable rotator cuff tendons and the anatomic footprint was proposed by Post in 1985 [58]; and this technique actually includes various graft options, such as synthetic grafts [59, 60], xenografts (like porcine acellular dermal matrix) [61], different sources of autografts (i.e., fascia lata or iliotibial band with a Gerdy's tubercle bone block) [62, 63], and allografts (i.e., fascia lata or human dermal allograft) [46, 64, 65].

An ideal patient for this surgery should be the one with symptomatic irreparable tears after failed conservative treatment without OA [61, 66]. Severe muscle atrophy and fatty infiltration is a relative contraindication because of its irreversible condition that does not favor tendon healing [61, 66].

All patch grafts showed a significant improvement in pain and ROM, even if most of the studies are small case series with short follow-up.

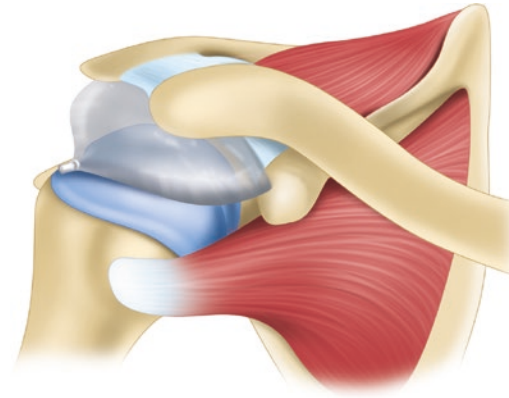


Fig. 19.6 Balloon spacer arthroplasty consists of implanting a biodegradable balloon inflated with saline solution in the subacromial space

Synthetic grafts seem to be more susceptible to re-tear [18, 66].

19.10 Balloon Spacer Arthroplasty

Balloon spacer arthroplasty consists of implanting a biodegradable balloon inflated with saline solution in the subacromial space (Fig. 19.6). This is a simple and minimally invasive procedure that reduces friction and allows smooth glide of the joint, enabling an intense painless rehabilitation to restore ROM and effective action of the deltoid muscle. The balloon dissolves over a period of 12 to 18 months [67, 68].

Good outcomes are reported from many studies in terms of pain relief and subjective and functional scores, while describing a non-significant AHD reduction [69–72]. There is a moderate–strong correlation between preoperative ROM and subjective general satisfaction after the procedure [18, 72]. Failure rate of this procedure varies from 3 to 8.3% [68, 69, 71, 72].

19.11 Muscle Transfer

Many transfers have been proposed in the past years to restore shoulder strength and kinematics. Specific muscle transfers are indicated depending on the type of lost function, which is strictly linked to the site of the lesion. Correct indications and

patient selection are crucial. These procedures are indicated in a young or high-demand patient with impairment related to weakness and loss of movement, with minimal glenohumeral OA [5, 73, 74].

19.11.1 Posterior-Superior Tears

Posterior-superior tears are the most common tears; different kinds of muscle transfer have been proposed in the literature.

19.11.1.1 Latissimus Dorsi

This transfer is usually performed for SS and IS tears to restore external rotation while elevation is provided by residual deltoid function. The procedure, first described by Gerber in 1988, consists in relocation of the distal insertion of this muscle from the intertubercular groove to the superolateral humeral head; this change of the LD function into an external rotator creates a head-depressing moment that allows more effective action of the deltoid muscle in elevation and prevents cranial migration of the humeral head on attempted flexion or abduction [75] (Fig. 19.7).

Several studies supported a gain in pain, mobility, function, and strength, with an increase of functional and subjective scores and a high satisfaction rate but also showed a mild progression of humeral escape and glenohumeral OA [76–79]. Gerber et al. [77], in a long-term (10 years) follow-up study, estimated this progression limited to one stage in Hamada classification with no influence on the clinical results.

Despite the original technique being an open procedure, nowadays more and more surgeons are performing it by arthroscopic assistance, with good outcome; and some authors described no progression of OA in comparison with the open procedure [79–84].

Latissimus dorsi transfer (LDT) is relatively contraindicated in patients with SSC tear > grade 2 according to Lafosse's classification (the lesion must be reparable), severe CTA (Hamada stage ≥4). Deltoid dysfunction, atrophy or fatty degeneration of TM, deltoid and SSC, large critical shoulder angle, and pseudoparalysis could negatively affect the outcome. The outcome is strictly related also to the compliance of the patient with physical therapy [17, 76–78, 83, 85].

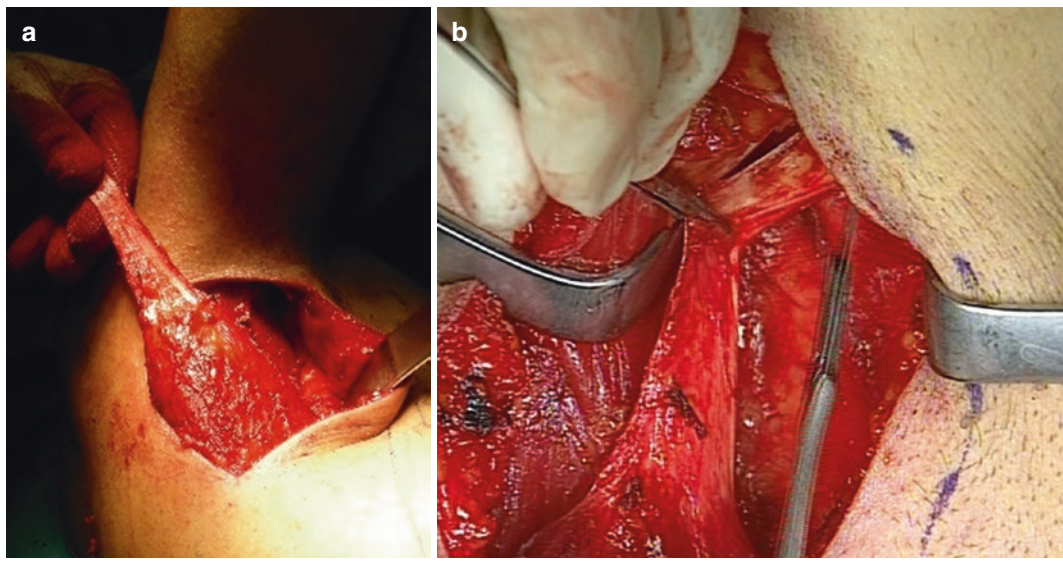


Fig. 19.7 (**a**, **b**) Latissimus dorsi (LD) transfer is usually performed for supraspinatus and infraspinatus tears to restore external rotation and consists in relocation of the distal insertion of this muscle from the intertubercular groove to the superolateral humeral head. This change of the LD function into an external rotator creates a head-depressing moment that allows more effective action of the deltoid muscle in elevation and prevents cranial migration of the humeral head on attempted flexion or abduction

Failure rate is moderate with a peaking up to 38% described by Kany et al. [76, 77, 82–84], while other studies reported lower failure rate [76, 77, 82–84].

19.11.1.2 Lower Trapezius

The technique describes the transfer of the lower portion of the trapezius, which is then fixed to the humeral head to restore the function of an irreparable posterior-superior rotator cuff tear. Elhassan et al. also described a modified arthroscopically assisted technique, and recently some papers suggest an augmentation with Achilles tendon allograft [86–88].

Biomechanical evidence also suggests that lower trapezius may restore shoulder kinematics better than latissimus dorsi transfer [89].

19.11.1.3 L'Episcopo Procedure

The technique was originally described in 1934 to treat obstetrical brachial plexus palsy. L'Episcopo procedure always includes a combined teres major and LD transfer, and a modified technique was proposed by Hebermeyer et al. [90] to restore external rotation in patients with MIRCTs. Boileau et al. [91] published another variation of the technique through a delto-pectoral approach during RTSA. However, more studies are necessary to confirm its efficacy in the treatment of MIRCTs.

19.11.2 Anterior-Superior Tears

Anterosuperior tears are less frequent but are a surgical challenge because of the frequent delay in diagnosis; generally, they include SSC and SS tear with a loss of internal rotation and in some cases a relative anterior instability of the glenohumeral joint.

19.11.2.1 Pectoralis Major

To change the function of this muscle into an internal rotator, the pectoralis major (PM) is routed behind the conjoined tendon and then fixed to the lesser tuberosity. This procedure is indicated in irreparable SSC tears (Goutallier grade > 3) with intact posterior-superior cuff, minimal OA, functioning deltoid, and high-demand patients [92].

Studies suggested good results with good improvement in pain and internal rotation and high grade of satisfaction, even if this procedure does not arrest the progression of CTA [93–96].

19.11.2.2 Pectoralis Minor

This procedure was first described by Wirth and Rockwood in 1997 [95, 96] and provides a reconstruction in patients with irreparable SSC tendons by transferring the pectoralis minor to the lesser tuberosity. Paladini et al. [95, 96] described a variation with a coracoid block attached to the pectoralis minor. The technique provided significant improvements in ROM, pain, and satisfaction [95, 96].

19.11.2.3 Latissimus Dorsi and Superior Trapezius

Mun et al. [97] proposed a transfer of LD for irreparable SSC tears, because of their similar direction, achieving a restoration of shoulder function and pain relief. Goutallier also performed a reconstruction of SSC with superior trapezius transfer with poor outcome, so this option does not appear recommendable [98].

19.12 Reverse Total Shoulder Arthroplasty

The term "cuff tear arthopathy" (CTA) was introduced by Neer in 1983 [99–102]. Reverse total shoulder arthroplasty (RTSA) has a main indication in CTA with good results in restoring active elevation and relieving pain [99].

Indeed, RTSA is the first choice to improve elevation in patients with a MIRCT, true pseudoparalysis, and glenohumeral OA, for whom arthroscopic procedures or tendon transfers are not suitable (Fig. 19.8). Even though the indications for RTSA have been broadened to manage a number of pathologies with average good results, the best outcomes with this procedure are achieved in patients with CTA [91, 103].

A deficit in active external rotation due to tears of the TM (with clinically positive hornblower's sign) is an obstacle to reach satisfactory shoulder function after a TRSA alone. In these

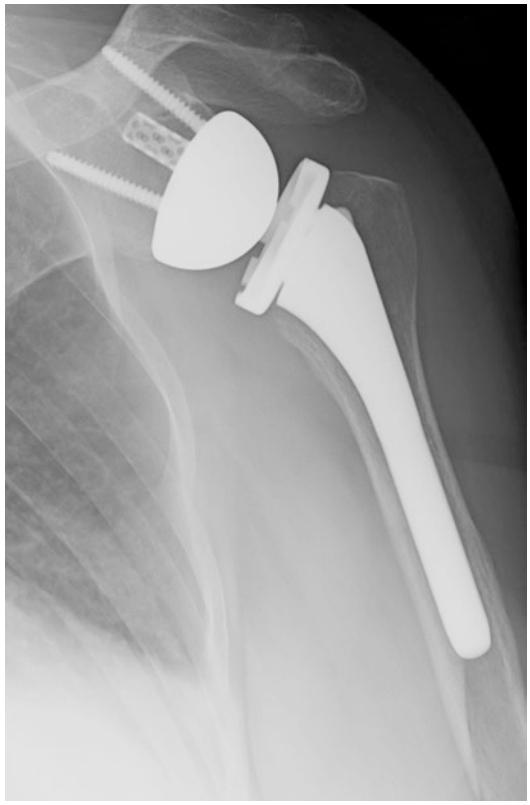


Fig. 19.8 Reverse total shoulder arthroplasty is the first choice to improve elevation in patients with a massive irreparable rotator cuff tear, true pseudoparalysis, and glenohumeral osteoarthritis, for whom arthroscopic procedures or tendon transfers are not suitable

patients, a LDT combined with RTSA can overcome the problem of the active external rotation deficit [104].

The importance of properly selecting the patients that undergo this procedure has been highlighted several times in the literature. Hartzer et al. found poor outcomes in patients who underwent RTSA for irreparable rotator cuff tear and were younger than 60 years, with high preoperative function or neurologic dysfunction [102]. In the same way, Boileau et al. observed a high rate of dissatisfaction in a subgroup of patients with preoperative active elevation over 90° [105, 106].

Even though the RTSA seems to be the gold standard for the treatment of elderly patients with a MIRCT with pseudoparalysis complicated by severe glenohumeral OA, the complication rates have been reported between 20 and 50% [107]. These complications are represented by mechanical failure, acromion fractures, infections, and heterotopic ossification [105, 106]. Moreover, another matter of concern is the longevity of these implants, with revision rates of 10–33% and increasing complications rates with each revisions [105, 106].

References

1. Rockwood CA, Williams GR, Burkhead WZ. Debridement of degenerative, irreparable lesions of the rotator cuff. J Bone Joint Surg Ser A. 1995;77(6):857–66.
2. Merolla G, et al. Tendon transfer for irreparable rotator cuff tears: indications and surgical rationale. Muscles Ligaments Tendons J. 2014;4(4):425–32.
3. Novi M, Kumar A, Paladini P, Porcellini G, Merolla G. Irreparable rotator cuff tears: challenges and solutions. Orthop Res Rev. 2018;10:93–103.
4. Khair MM, Gulotta L v. Treatment of irreparable rotator cuff tears. Curr Rev Musculoskelet Med. 2011;4(4):208–13.
5. Dines DM, Moynihan DP, Dines JS, McCann P. Irreparable rotator cuff tears: what to do and when to do it; the surgeon's dilemma. Instr Course Lect. 2007;56:13–22.
6. Cofield RH, Parvizi J, Hoffmeyer PJ, Lanzer WL, Ilstrup DM, Rowland CM. Surgical repair of chronic rotator cuff tears. A prospective long-term study. J Bone Joint Surg Am. 2001;83(1):71–7.
7. Gerber C, Fuchs B, Hodler J. The results of repair of massive tears of the rotator cuff. J Bone Joint Surg Ser A. 2000;82(4):505–15.
8. Kim SJ, Kim SH, Lee SK, Seo JW, Chun YM. Arthroscopic repair of massive contracted rotator cuff tears: aggressive release with anterior and posterior interval slides do not improve cuff healing and integrity. J Bone Joint Surg Ser A. 2013;95(16):1482–8.
9. Galatz LM, Ball CM, Teefey SA, Middleton WD, Yamaguchi K. The outcome and repair integrity of completely arthroscopically repaired large and massive rotator cuff tears. J Bone Joint Surg Ser A. 2004;86(2):219–24.
10. Cvetanovich GL, Waterman BR, Verma NN, Romeo AA. Management of the irreparable rotator cuff tear. J Am Acad Orthop Surg. 2019;27(24):909–17.
11. Kim SJ, Lee IS, Kim SH, Lee WY, Chun YM. Arthroscopic partial repair of irreparable large to massive rotator cuff tears. Am J Sports Med. 2012;28(6):761–8.
12. Virk MS, Nicholson GP, Romeo AA. Irreparable rotator Cuff tears without arthritis treated with reverse total shoulder arthroplasty. Open Orthop J. 2018;10(1):296–308.

13. Kim S-J, Park J-S, Lee K-H, Lee B-G. The development of a quantitative scoring system to predict whether a large-to-massive rotator cuff tear can be arthroscopically repaired. Bone Joint J. 2016;98-B(12):1656–61.
14. Kim JY, Park JS, Rhee YG. Can preoperative magnetic resonance imaging predict the reparability of massive rotator Cuff tears? Am J Sports Med. 2017;45(7):1654–63.
15. Yoo JC, Ahn JH, Yang JH, Koh KH, Choi SH, Yoon YC. Correlation of arthroscopic Repairability of large to massive rotator Cuff tears with preoperative magnetic resonance imaging scans. Arthroscopy. 2009;25(6):573–82.
16. Sheean AJ, et al. Preoperative radiographic risk factors for incomplete arthroscopic supraspinatus tendon repair in massive rotator Cuff tears. Arthroscopy. 2018;34(4):1121–7.
17. Thès A, Hardy P, Bak K. Decision-making in massive rotator cuff tear. Knee Surg Sports Traumatol Arthrosc. 2015;23(2):449–59.
18. Carver TJ, Kraeutler MJ, Smith JR, Bravman JT, McCarty EC. Nonarthroplasty surgical treatment options for massive, irreparable rotator cuff tears. Orthop J Sports Med. 2018;6(11):2325967118805385.
19. Zingg PO, Jost B, Sukthankar A, Buhler M, Pfirrmann CWA, Gerber C. Clinical and structural outcomes of nonoperative management of massive rotator cuff tears. J Bone Joint Surg Ser A. 2007;89(9):1928–34.
20. Levy O, Mullett H, Roberts S, Copeland S. The role of anterior deltoid reeducation in patients with massive irreparable degenerative rotator cuff tears. J Shoulder Elbow Surg. 2008;17(6):863–70.
21. Dunn WR, et al. 2013 Neer award: predictors of failure of nonoperative treatment of chronic, symptomatic, full-thickness rotator cuff tears. J Shoulder Elbow Surg. 2016;25(8):1303–11.
22. Walch G, Edwards TB, Boulahia A, Nové-Josserand L, Neyton L, Szabo I. Arthroscopic tenotomy of the long head of the biceps in the treatment of rotator cuff tears: clinical and radiographic results of 307 cases. J Shoulder Elbow Surg. 2005;14(3):238–46.
23. Boileau P, Baqué F, Valerio L, Ahrens P, Chuinard C, Trojani C. Isolated arthroscopic biceps tenotomy or tenodesis improves symptoms in patients with massive irreparable rotator cuff tears. J Bone Joint Surg Ser A. 2007;89(4):747–57.
24. Klinger HM, Steckel H, Ernstberger T, Baums MH. Arthroscopic debridement of massive rotator cuff tears: negative prognostic factors. Arch Orthop Trauma Surg. 2005;125(4):261–6.
25. Kong BY, Kim SH, Kim DH, Joung HY, Jang YH, Oh JH. Suprascapular neuropathy in massive rotator cuff tears with severe fatty degeneration in the infraspinatus muscle. Bone Joint J. 2016;98-B(11):1505–9.
26. Mallon WJ, Wilson RJ, Basamania CJ. The association of suprascapular neuropathy with massive rotator cuff tears: a preliminary report. J Shoulder Elbow Surg. 2006;15(4):395–8.
27. Costouros JG, Porramatikul M, Lie DT, Warner JJP. Reversal of suprascapular neuropathy following arthroscopic repair of massive supraspinatus and infraspinatus rotator cuff tears. Arthroscopy. 2007;23(11):1152–61.
28. Lafosse L, Piper K, Lanz U. Arthroscopic suprascapular nerve release: indications and technique. J Shoulder Elbow Surg. 2011;20(2 Suppl):S9–13.
29. Gartsman GM. Massive, irreparable tears of the rotator cuff: results of operative debridement and subacromial decompression. J Bone Joint Surg Ser A. 1997;79(5):715–21.
30. Hawi N, et al. Arthroscopic debridement for irreparable rotator Cuff tears. Open Orthop J. 2016;10(1):324–9.
31. Gerber C, Wirth SH, Farshad M. Treatment options for massive rotator cuff tears. J Shoulder Elbow Surg. 2011;20(2 Suppl):S20–9.
32. Franceschi F, Papalia R, Vasta S, Leonardi F, Maffulli N, Denaro V. Surgical management of irreparable rotator cuff tears. Knee Surg Sports Traumatol Arthrosc. 2015;23(2):494–501.
33. Berth A, Neumann W, Awiszus F, Pap G. Massive rotator cuff tears: functional outcome after debridement or arthroscopic partial repair. J Orthop Traumatol. 2010;11(1):13–20.
34. Veado MA d C, Rodrigues AU. Functional evaluation of patients who have undergone arthroscopic debridement to treat massive and irreparable tears of the rotator cuff. Rev Bras Ortoped. 2010;45(5):426–31.
35. Heuberer PR, et al. Arthroscopic management of massive rotator cuff tears: an evaluation of debridement, complete, and partial repair with and without force couple restoration. Knee Surg Sports Traumatol Arthrosc. 2016;24(12):3828–37.
36. Liem D, Lengers N, Dedy N, Poetzl W, Steinbeck J, Marquardt B. Arthroscopic debridement of massive irreparable rotator Cuff tears. Arthroscopy. 2008;24(7):743–8.
37. Fenlin JM, Chase JM, Rushton SA, Frieman BG. Tuberoplasty: creation of an acromiohumeral articulation—a treatment option for massive, irreparable rotator cuff tears. J Shoulder Elbow Surg. 2002;11(2):136–42.
38. Scheibel M, Lichtenberg S, Habermeyer P. Reversed arthroscopic subacromial decompression for massive rotator cuff tears. J Shoulder Elbow Surg. 2004;13(3):272–8.
39. Verhelst L, Vandekerckhove PJ, Sergeant G, Liekens K, van Hoonacker P, Berghs B. Reversed arthroscopic subacromial decompression for symptomatic irreparable rotator cuff tears: mid-term follow-up results in 34 shoulders. J Shoulder Elbow Surg. 2010;19(4):601–8.
40. Lee BG, Cho NS, Rhee YG. Results of arthroscopic decompression and tuberoplasty for irreparable massive rotator cuff tears. Arthroscopy. 2011;27(10):1341–50.
41. Burkhart SS, Nottage WM, Ogilvie-Harris DJ, Kohn HS, Pachelli A. Partial repair of irreparable rotator cuff tears. Arthroscopy. 1994;10(4):363–70.

42. Cuff DJ, Pupello DR, Santoni BG. Partial rotator cuff repair and biceps tenotomy for the treatment of patients with massive cuff tears and retained overhead elevation: midterm outcomes with a minimum 5 years of follow-up. J Shoulder Elbow Surg. 2016;25(11):1803–9.
43. Castricini R, et al. Arthroscopic partial repair of irreparable, massive rotator Cuff tears. Arthrosc Tech. 2017;6(1):e143–7.
44. Duralde XA, Bair B. Massive rotator cuff tears: the result of partial rotator cuff repair. J Shoulder Elbow Surg. 2005;14(2):121–7.
45. Shon MS, Koh KH, Lim TK, Kim WJ, Kim KC, Yoo JC. Arthroscopic partial repair of irreparable rotator Cuff tears: preoperative factors associated with outcome deterioration over 2 years. Am J Sports Med. 2015;43(8):1965–75.
46. Pandey R, Tafazal S, Shyamsundar S, Modi A, Singh HP. Outcome of partial repair of massive rotator cuff tears with and without human tissue allograft bridging repair. Shoulder Elbow. 2017;9(1):23–30.
47. Galasso O, et al. Quality of life and functional results of arthroscopic partial repair of irreparable rotator Cuff tears. Arthroscopy. 2017;33(2):261–8.
48. Chen KH, Chiang ER, Wang HY, Ma HL. Arthroscopic partial repair of irreparable rotator Cuff tears: factors related to greater degree of clinical improvement at 2 years of follow-up. Arthroscopy. 2017;33(11):1949–55.
49. Mihata T, McGarry MH, Pirolo JM, Kinoshita M, Lee TQ. Superior capsule reconstruction to restore superior stability in irreparable rotator cuff tears: a biomechanical cadaveric study. Am J Sports Med. 2012;40(10):2248–55.
50. Petri M, Greenspoon JA, Millett PJ. Arthroscopic superior capsule reconstruction for irreparable rotator Cuff tears. Arthrosc Tech. 2015;4(6):e751–5.
51. Burkhart SS, Denard PJ, Adams CR, Brady PC, Hartzler RU. Arthroscopic superior capsular reconstruction for massive irreparable rotator Cuff repair. Arthrosc Tech. 2016;5(6):e1407–18.
52. Mihata T, et al. Clinical results of arthroscopic superior capsule reconstruction for irreparable rotator cuff tears. Arthroscopy. 2013;29(3):459–70.
53. Denard PJ, Brady PC, Adams CR, Tokish JM, Burkhart SS. Preliminary results of arthroscopic superior capsule reconstruction with dermal allograft. Arthroscopy. 2018;34(1):93–9.
54. Chillemi C, Mantovani M, Gigante A. Superior capsular reconstruction of the shoulder: the ABC (Arthroscopic Biceps Chillemi) technique. Eur J Orthop Surg Traumatol. 2018;28(6):1215–23.
55. Wall KC, Toth AP, Garrigues GE. How to use a graft in irreparable rotator cuff tears: a literature review update of interposition and superior capsule reconstruction techniques. Curr Rev Musculoskelet Med. 2018;11(1):122–30.
56. Pennington WT, Bartz BA, Pauli JM, Walker CE, Schmidt W. Arthroscopic superior capsular reconstruction with Acellular dermal allograft for the treatment of massive irreparable rotator Cuff tears: short-term clinical outcomes and the radiographic parameter of superior capsular distance. Arthroscopy. 2018;34(6):1764–73.
57. Lee SJ, Min YK. Can inadequate acromiohumeral distance improvement and poor posterior remnant tissue be the predictive factors of re-tear? Preliminary outcomes of arthroscopic superior capsular reconstruction. Knee Surg Sports Traumatol Arthrosc. 2018;26(7):2205–13.
58. Post M. Rotator cuff repair with carbon filament. A preliminary report of five cases. Clin Orthop Relat Res. 1985;196:154–8.
59. Audenaert E, VanNuffel J, Schepens A, Verhelst M, Verdonk R. Reconstruction of massive rotator cuff lesions with a synthetic interposition graft: a prospective study of 41 patients. Knee Surg Sports Traumatol Arthrosc. 2006;14(4):360–4.
60. Ranebo MC, Björnsson Hallgren HC, Norlin R, Adolfsson LE. Long-term clinical and radiographic outcome of rotator cuff repair with a synthetic interposition graft: a consecutive case series with 17 to 20 years of follow-up. J Shoulder Elbow Surg. 2018;27(9):1622–8.
61. Neumann JA, et al. Interposition dermal matrix Xenografts: a successful alternative to traditional treatment of massive rotator Cuff tears. Am J Sports Med. 2017;45(6):1261–8.
62. Mori D, Funakoshi N, Yamashita F, Wakabayashi T. Effect of fatty degeneration of the infraspinatus on the efficacy of arthroscopic patch autograft procedure for large to massive rotator cuff tears. Am J Sports Med. 2015;43(5):1108–17.
63. Mihara S, Fujita T, Ono T, Inoue H, Kisimoto T. Rotator cuff repair using an original iliotibial ligament with a bone block patch: preliminary results with a 24-month follow-up period. J Shoulder Elbow Surg. 2016;25(7):1155–62.
64. Gupta AK, et al. Dermal tissue allograft for the repair of massive irreparable rotator cuff tears. Am J Sports Med. 2012;40(1):141–7.
65. Dimitrios V, Athanasios P, Eleni A, Xenofon P, George F, John F. Results of reconstruction of massive irreparable rotator cuff tears using a fascia lata allograft. Indian J Orthopaed. 2015;49(3):304–11.
66. Lewington MR, Ferguson DP, Smith TD, Burks R, Coady C, Wong IHB. Graft utilization in the bridging reconstruction of irreparable rotator cuff tears: a systematic review. Am J Sports Med. 2017;45(13):3149–57.
67. Savarese E, Romeo R. New solution for massive, irreparable rotator Cuff tears: the subacromial 'biodegradable spacer'. Arthrosc Tech. 2012;1(1):e69–74.
68. Gervasi E, Cautero E, Dekel A. Fluoroscopy-guided implantation of subacromial 'Biodegradable Spacer' using local anesthesia in patients with irreparable rotator cuff tear. Arthrosc Tech. 2014;3(4):e455–8.
69. Senekovic V, Poberaj B, Kovacic L, Mikek M, Adar E, Dekel A. Prospective clinical study of a novel

biodegradable sub-acromial spacer in treatment of massive irreparable rotator cuff tears. Eur J Orthop Surg Traumatol. 2013;23(3):311–6.
70. Piekaar RSM, Bouman ICE, van Kampen PM, van Eijk F, Huijsmans PE. Early promising outcome following arthroscopic implantation of the subacromial balloon spacer for treating massive rotator cuff tear. Musculoskelet Surg. 2018;102(3):247–55.
71. Deranlot J, et al. Arthroscopic subacromial spacer implantation in patients with massive irreparable rotator Cuff tears: clinical and radiographic results of 39 retrospectives cases. Arthroscopy. 2017;33(9):1639–44.
72. Prat D, Tenenbaum S, Pritsch M, Oran A, Vogel G. Sub-acromial balloon spacer for irreparable rotator cuff tears: is it an appropriate salvage procedure? J Orthop Surg. 2018;26(2):2309499018770887.
73. Bedi A, Dines J, Warren RF, Dines DM. Massive tears of the rotator cuff. J Bone Joint Surg Ser A. 2010;92(9):1894–908.
74. Warner JJ. Management of massive irreparable rotator cuff tears: the role of tendon transfer. Instr Course Lect. 2001;50:63–71.
75. Gerber C, Vinh TS, Hertel R, Hess CW. Latissimus dorsi transfer for the treatment of massive tears of the rotator cuff. A preliminary report. Clin Orthop Relat Res. 1988;232:51–61.
76. Namdari S, Voleti P, Baldwin K, Glaser D, Huffman GR. Latissimus dorsi tendon transfer for irreparable rotator cuff tears: a systematic review. J Bone Joint Surg Ser A. 2012;94(10):891–8.
77. Gerber C, Rahm SA, Catanzaro S, Farshad M, Moor BK. Latissimus dorsi tendon transfer for treatment of irreparable posterosuperior rotator cuff tears: long-term results at a minimum follow-up of ten years. J Bone Joint Surg Ser A. 2013;95(21):1920–6.
78. El-Azab HM, Rott O, Irlenbusch U. Long-term follow-up after latissimus dorsi transfer for irreparable posterosuperior rotator cuff tears. J Bone Joint Surg Am. 2015;97(6):462–9.
79. Petriccioli D, Bertone C, Marchi G. Recovery of active external rotation and elevation in young active men with irreparable posterosuperior rotator cuff tear using arthroscopically assisted latissimus dorsi transfer. J Shoulder Elbow Surg. 2016;25(9):e265–75.
80. Castricini R, et al. Functional status and failed rotator cuff repair predict outcomes after arthroscopic-assisted latissimus dorsi transfer for irreparable massive rotator cuff tears. J Shoulder Elbow Surg. 2016;25(4):658–65.
81. Castricini R, et al. Arthroscopic-assisted latissimus dorsi transfer for the management of irreparable rotator cuff tears: short-term results. J Bone Joint Surg Am. 2014;96(14):e119.
82. Kany J, Grimberg J, Amaravathi RS, Sekaran P, Scorpie D, Werthel JD. Arthroscopically-assisted Latissimus Dorsi transfer for irreparable rotator Cuff insufficiency: modes of failure and clinical correlation. Arthroscopy. 2018;34(4):1139–50.
83. Grimberg J, Kany J, Valenti P, Amaravathi R, Ramalingam AT. Arthroscopic-assisted latissimus dorsi tendon transfer for irreparable posterosuperior cuff tears. Arthroscopy. 2015;31(4):599–607.e1.
84. Kanatlı U, et al. Arthroscopic-assisted Latissimus Dorsi tendon transfer for massive, irreparable rotator Cuff tears: technique and short-term follow-up of patients with Pseudoparalysis. Arthroscopy. 2017;33(5):929–37.
85. Anastasopoulos PP, Alexiadis G, Spyridonos S, Fandridis E. Latissimus Dorsi transfer in posterior irreparable rotator Cuff tears. Open Orthop J. 2017;11(1):77–94.
86. Elhassan BT, Wagner ER, Werthel JD. Outcome of lower trapezius transfer to reconstruct massive irreparable posterior-superior rotator cuff tear. J Shoulder Elbow Surg. 2016;25(8):1346–53.
87. Elhassan BT, Alentorn-Geli E, Assenmacher AT, Wagner ER. Arthroscopic-assisted lower trapezius tendon transfer for massive irreparable posterior-superior rotator Cuff tears: surgical technique. Arthrosc Tech. 2016;5(5):e981–8.
88. Stoll LE, Codding JL. Lower trapezius tendon transfer for massive irreparable rotator Cuff tears. Orthop Clin North Am. 2019;50(3):375–82.
89. Omid R, Heckmann N, Wang L, McGarry MH, Vangsness CT, Lee TQ. Biomechanical comparison between the trapezius transfer and latissimus transfer for irreparable posterosuperior rotator cuff tears. J Shoulder Elbow Surg. 2015;24(10):1635–43.
90. Gerhardt C, Lehmann L, Lichtenberg S, Magosch P, Habermeyer P. Modified l'episcopo tendon transfers for irreparable rotator cuff tears: 5-year followup. Clin Orthop Relat Res. 2010;468(6):1572–7.
91. Boileau P, Chuinard C, Roussanne Y, Neyton L, Trojani C. Modified latissimus dorsi and teres major transfer through a single delto-pectoral approach for external rotation deficit of the shoulder: as an isolated procedure or with a reverse arthroplasty. J Shoulder Elbow Surg. 2007;16(6):671–82.
92. Neer CS, Craig E v, Fukuda H. Cuff-tear arthropathy. J Bone Joint Surg Ser A. 1983;65(9):1232–44.
93. Elhassan B, Ozbaydar M, Massimini D, Diller D, Higgins L, Warner JJP. Transfer of pectoralis major for the treatment of irreparable tears of subscapularis: does it work? J Bone Joint Surg. 2008;90(8):1059–65.
94. Shin JJ, Saccomanno MF, Cole BJ, Romeo AA, Nicholson GP, Verma NN. Pectoralis major transfer for treatment of irreparable subscapularis tear: a systematic review. Knee Surg Sports Traumatol Arthrosc. 2016;24(6):1951–60.
95. Wirth MA, Rockwood CA. Operative treatment of irreparable rupture of the subscapularis. J Bone Joint Surg Ser A. 1997;79(5):722–31.
96. Paladini P, Campi F, Merolla G, Pellegrini A, Porcellini G. Pectoralis minor tendon transfer for irreparable anterosuperior cuff tears. J Shoulder Elbow Surg. 2013;22(6):e1–5.

97. Mun SW, Kim JY, Yi SH, Baek CH. Latissimus dorsi transfer for irreparable subscapularis tendon tears. J Shoulder Elbow Surg. 2018;27(6):1057–64.
98. Goutallier D, de Abreu L, Postel JM, le Guilloux P, Radier C, Zilber S. Is the trapezius transfer a useful treatment option for irreparable tears of the subscapularis? Orthop Traumatol Surg Res. 2011;97(7):719–25.
99. Wall B, Nové-Josserand L, O'Connor DP, Edwards TB, Walch G. Reverse total shoulder arthroplasty: A review of results according to etiology. J Bone Joint Surg Ser A. 2007;89(7):1476–85.
100. Al-Hadithy N, Domos P, Sewell MD, Pandit R. Reverse shoulder arthroplasty in 41 patients with cuff tear arthropathy with a mean follow-up period of 5 years. J Shoulder Elbow Surg. 2014;23(11):1662–8.
101. Ek ETH, Neukom L, Catanzaro S, Gerber C. Reverse total shoulder arthroplasty for massive irreparable rotator cuff tears in patients younger than 65 years old: results after five to fifteen years. J Shoulder Elbow Surg. 2013;22(9):1199–208.
102. Boileau P, Gonzalez J-F, Chuinard C, Bicknell R, Walch G. Reverse total shoulder arthroplasty after failed rotator cuff surgery. J Shoulder Elbow Surg. 2009;18:600–6.
103. Boughebri O, Kilinc A, Valenti P. Reverse shoulder arthroplasty combined with a latissimus dorsi and teres major transfer for a deficit of both active elevation and external rotation. Results of 15 cases with a minimum of 2-year follow-up. Orthop Traumatol Surg Res. 2013;99(2):131–7.
104. Hartzler RU, et al. Reverse shoulder arthroplasty for massive rotator cuff tear: risk factors for poor functional improvement. J Shoulder Elbow Surg. 2015;24(11):1698–706.
105. Mulieri P, Dunning P, Klein S, Pupello D, Frankle M. Reverse shoulder arthroplasty for the treatment of irreparable rotator cuff tear without glenohumeral arthritis. J Bone Joint Surg Ser A. 2010;92(15):2544–56.
106. Gerber C, Canonica S, Catanzaro S, Ernstbrunner L. Longitudinal observational study of reverse total shoulder arthroplasty for irreparable rotator cuff dysfunction: results after 15 years. J Shoulder Elbow Surg. 2018;27(5):831–8.
107. Petrillo S, Longo UG, Papalia R, Denaro V. Reverse shoulder arthroplasty for massive irreparable rotator cuff tears and cuff tear arthropathy: a systematic review. Musculoskelet Surg. 2017;101(2):105–12.

Part III

Surgical Techniques

Partial Repair

20

Gonzalo Samitier and Emilio Calvo

20.1 Introduction

Partial repair for massive rotator cuff tears was first described by Burkhart et al. in 1994 [1]. The goal of repairing infraspinatus and subscapularis tendons to their footprint while leaving the irreparable supraspinatus unrepaired creates force coupling of the deltoid and repaired rotator cuff tendons to allow for effective elevation of the arm. Although the supraspinatus is left unrepaired, complete coverage of the humeral head was considered unnecessary because the biomechanics of the shoulder were restored. This technique was originally described as an open procedure, although several recent articles [2, 3] using the same principles have allowed surgeons to perform partial tendon repairs arthroscopically; this procedure has been considered in patients with irreparable supraspinatus tendon and reparable infraspinatus and subscapularis with no glenohumeral osteoarthritis who continue to have pain and dysfunction after conservative management [3, 4].

G. Samitier (✉)
Instituto Cugat, Hospital Quironsalud Barcelona, Barcelona, Spain
e-mail: gsamitier@sportrauma.com

E. Calvo
Department of Orthopaedic Surgery and Traumatology, IIS-Fundación Jiménez Díaz, Universidad Autónoma, Madrid, Spain
e-mail: ECalvo@fjd.es

Frequently, by using mobilization techniques, we are able to reach near anatomic repairs for most of large-to-massive tears, minimizing the exposure of the humeral head. A double-row repair for the infraspinatus portion of the repair combined with side-to-side and single-row repair techniques for the most anterior cuff increases the footprint contact and potentially contributes to improved healing rates, and provides greater humeral head coverage and better long-term results, as reported in previously published studies that targeted only for partial repair [5, 6].

Indeed, there is a recent controversy defining the conceptual and clinical differences between partial repair vs. incomplete repair; as many articles have indistinctly used these terms, a recent article from Yoo et al. [7] tried to clarify the issue and defined partial repair as the construct that leaves a substantial unrepaired defect that entails moderate to extensive exposure of the humeral head (>10 mm) after surgery, unlike incomplete repair (<10 mm). Duralde and Bair [8] reported in their series on partial repair that the mean size of the residual humeral head uncovered defect was 1.7 cm. Burkhart [9] and Kim et al. [10] reported a mean residual defect of 29 mm and 12 mm with partial repair, respectively. This controversy is relevant as recent publications have shown that partial repair does not always guarantee satisfactory clinical outcomes in the mid- and long term despite initial encouraging results [5]. Yoo et al. [7] compared both repairs and evidenced more

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_20

satisfactory clinical outcomes when arthroscopic incomplete repair was achieved in contrast with partial repair for large-to-massive rotator cuff tears.

20.2 Indications to Partial Repair

A complete medical history of the patient and a full physical exam with an adequate imaging study are important to build a convenient preoperative plan. In revision cases, the main factors related to failure should also be examined: history of trauma, complete reports about previous surgical procedures, smoking habits, and whether early aggressive rehabilitation contributed to structural failure. Rotator cuff repair is an elective surgery that requires careful preoperative evaluation and discussion of treatment expectations, risks and benefits. Overall, complete repairs of rotator cuff tears have been shown to lead to good-to-excellent outcomes in most patients, with significant improvement in the mean scores on self-assessed questionnaires; however, multiple factors including age, gender, smoking habit, larger tear size, poor tendon quality, severe fatty infiltration (Goutallier grade >3) [11, 12], advanced rotator cuff tear arthropathy (Hamada stage >3) [13, 14], workers' compensation status, and healing potential of the rotator cuff repair have been shown to be associated with less favorable outcomes after rotator cuff repair [15, 16]. The patient must be advised of potential surgery- and anesthesia-related issues; postoperative timing should be discussed as well as the recovery process after rotator cuff repair, especially in regard to motion and strength, as this is usually slow and occasionally cannot be reversed back to normal [17].

For relatively low-demand older patients with chronic symptomatic tears that never tried conservative measures, or those who remain asymptomatic, we reserve initial 8–12 weeks of nonsurgical treatment regardless of the size of the tear and as long as they do not have pseudoparalysis; for relatively young patients with complete tears, we do not delay consultation for surgery if symptoms and/or limitations are present [18, 19]. For large tears in acute traumatic setting, we will offer surgery primarily in most cases as cuff tears tend to progress rapidly over time, becoming more difficult to repair and showing irreversible changes.

Our non-operative approach consists in guided physical therapy to keep a strong force couple. It is very common for these patients to have one or two subacromial corticosteroid injections along the process trying to reduce the inflammatory response and pain if that benefits the rehabilitation process. We also favor conservative treatment for symptomatic low-demand elderly population, patients who are not willing to have surgery, and/or patients medically inadequate.

For irreparable large-to-massive rotator cuff tears, we first consider incomplete repair. If only a partial repair is achievable, we may complement our partial repair with superior capsular reconstruction (SCR) given the promising preliminary results observed with this combination [20–22]. Occasionally, after partial repair, if flexibility of the tissues allows for side-to-side repair, we may consider to approximate both lateral ends of the infraspinatus and subscapularis tendons instead of doing a SCR.

We assessed large-to-massive tears according to the classification system described by Post et al. [23] (small, <1 cm; medium, 1–3 cm; large, 3–5 cm; and massive, >5 cm). The decision about the repair type is made often intraoperatively after mobilizing the torn tendons as bursectomy and bursal release from the acromion and deltoid, complete coracohumeral ligament release from the base of the coracoid, complete detachment of the superior capsule from the glenoid, and occasionally using an anterior interval slide.

In our partial or incomplete tendon repairs, especially for the infraspinatus tendon, we favor double-row anatomic partial repair because of the numerous biomechanical studies demonstrating improved tendon-to-bone contact, increased footprint coverage, decreased gap formation, and increased mechanical strength with double-row configurations [24–28]. Single-row or side-to-side repair techniques are reserved for the most anterior aspect of the full-thickness tear areas.

Our double-row consists in a transosseous equivalent (TOE) double-row suture bridge technique often with medial row tying. Described by Park et al. in 2006 [29], we favor medial row knot tying over knotless repairs especially in delaminated tears where anatomic independent layer repair is intended using the lasso-loop technique [30]. Previous studies have shown better biomechanical properties after medial knot tying performed as part of a TOE suture bridge construct compared with all-knotless constructs [31–34]; nevertheless, knotless repairs have also shown clinical success in the past [35, 36]. We may utilize a suture/tape speed bridge configuration, without medial knot tying for mid-size complete crescent-type tears [37] (Fig. 20.1).

Type 2 re-tears, at the level of the muscle tendon unit, with medial-row knot tying seem to be an increasing finding in recent studies [38, 39]; in order to avoid this complication, we tend to separate the thread couples when piercing the cuff and avoid over-tensioning and tying knots distal to the musculotendinous junction [40].

For those relatively young patients with very retracted tears including infraspinatus and teres minor tendons, grade III–IV of fatty infiltration, and less than mild osteoarthritic changes (Hamada stage <3), we may consider tendon transfer first instance, preferably lower trapezius transfer, which demonstrated better abduction and external rotation moment arms when transferred to the infraspinatus insertion instead of latissimus dorsi transfer [41].

For persistently symptomatic massive irreparable tears in elderly patients with or without signs of rotator cuff arthropathy and/or pseudoparalysis, we opt for reverse total shoulder arthroplasty, given the predictable good outcomes in regard to pain and functionality [42].

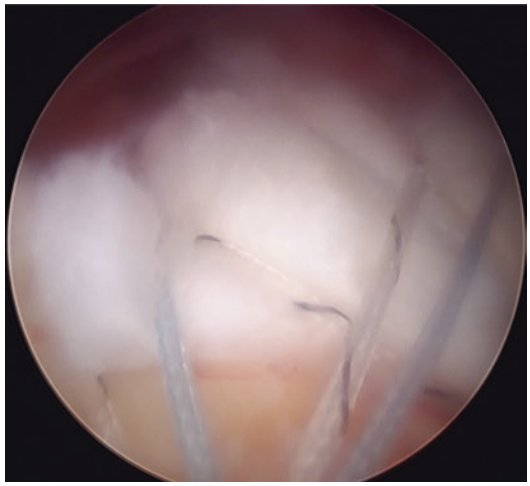


Fig. 20.1 Intraoperative arthroscopic view from the lateral portal of the rotator cuff (white area) partially repaired using a double-row trans-osseous equivalent (TOE) suture-bridge technique with medial row tying

20.3 Surgical Technique

Anesthesia is carried out following a standardized protocol based on a single-shot interscalenic blockade under ultrasound control (L-bupivacaine 0.5% 30–40 mL plus epinephrine) combined with general anesthesia (propofol 2–2.5 mg/kg iv and alfentanil 20–150 μg/kg iv initially, plus 15 μg/kg bolus, and maintenance with sevofluorane). Isolated regional anesthesia is reserved for those with increased risk of complications with general anesthesia. Antibiotic prophylaxis (2-g cefazolin or 1 g vancomycin as an alternative for patients with beta-lactam allergy) is administered 30 min before surgery. Rotator cuff tear repair can be performed either on the lateral decubitus or on beach-chair positions (BCP) with the arm forward flexed and 3–4 kg of traction (Fig. 20.2). We find beach position more ergonomic and easier to work in the subacromial space; the surgeon stands in front of the shoulder facing the shoulder positioned in its anatomic position, left and right hands can be used to insert instruments alternatively through anterior or posterior portals, and the arm can be moved easily, facilitating the rotator cuff repair.

Controlled hypotension and muscular relaxation are desirable as it may allow for better visualization and decreased blood loss; it does reduce the operative time and secondarily patient safety but also can affect the quality of the repair. Because of the risk for neurological ischemic events, caution should be exercised with hypotensive anesthesia in the BCP; elderly, hypertension with poor control, BMI>34, diabetes mellitus, obstructive

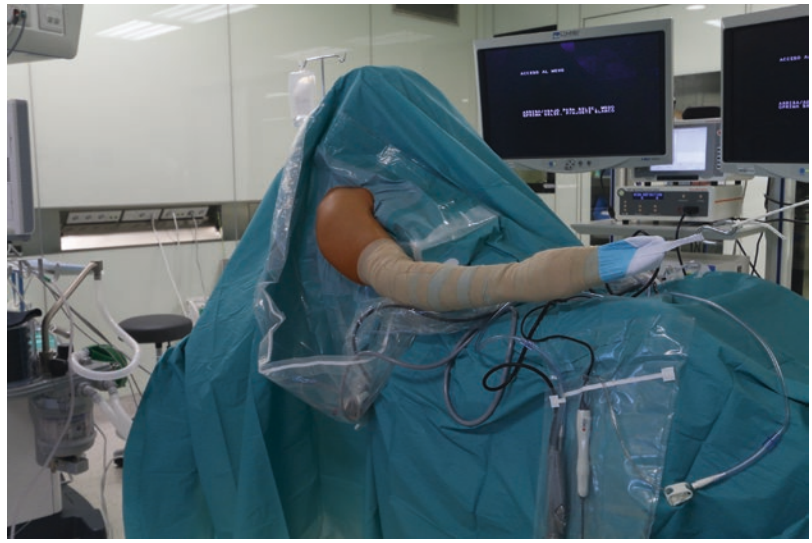


Fig. 20.2 Beach-chair positioned (BCP) patient with the right arm forward flexed 45° and 3–4 kg of traction using a pulley mechanism

sleep apnea, and previous history of stroke are considered high risk factors [43]. We maximize patient safety using routinely near-infrared spectroscopy (NIRS), which provides a non-invasive continuous assessment of the cerebral perfusion.

For fluid management, we use an automated pump system with dual, pressure and volume, control. (FMS®; DePuy, Mitek, Raynham, Massachusetts). The pump is usually set up initially to start at 70 mmHg.

20.3.1 Posterosuperior Partial Repair

Although numerous descriptions were made on where portals for rotator cuff reconstruction should be precisely located, we recommend establishing liberally as many portals as needed after testing the appropriate position and direction of the portal with a spinal needle. A precise anatomic knowledge is necessary to avoid injuries of neurovascular structures. Typically, three to six arthroscopic portals are established. These portals are placed posteriorly, posterolaterally, laterally, anterolaterally, and anteroinferiorly. For anchor insertion, a more medialized lateral portal close to the lateral edge of the acromion is often necessary to have an adequate entry angle. The anterior portal, lateral to the coracoid process, is often used to repair subscapularis tears. The Neviaser portal, medial and posterior to the acromioclavicular joint, can be helpful for suture passing when the tear is not accessible from anterior or posterior portals, but in the beach chair position, the patient's head can restrict the movement of instruments. The authors do not routinely use cannulas for arthroscopic rotator cuff repair.

In our practice, we only perform subacromial decompression if signs of subacromial impingement are identified or when extra room is needed to proceed with the rotator cuff repair; In these scenarios, the antero-lateral acromion is identified, the coracoacromial ligament is resected, and a 4.5 mm burr is used to perform the acromioplasty. When incomplete or partial repair is predicted because of the irreparability of the tendons, we are very cautious not to disrupt entirely the coraco-acromial arch but only any antero-lateral acromial osteophyte if present, preserving the most superior aspect of the coraco-acromial ligament attached. If any intervention to the long head of the biceps tendon is necessary (either tenotomy or tenodesis), it is performed prior to the rotator cuff tear repair to prevent any interference with the reconstruction; we do not include the biceps tendon in the repair.

After an initial visualization from the posterior portal, the camera is moved to a more lateral portal for better visualization and definition of the tear configuration; the retraction degree

of the tear can then be tested using arthroscopic forceps from accessory anterior or posterior portals. It is very important to understand the tear configuration and tendon mobility to plan the repair. While crescent-shaped tears are repaired to the footprint, reducing the tendon medial to lateral, in L-shaped and reverse L-shaped tears, it is very important to identify the apex of the tear that should be reduced to the corresponding edge of the footprint. At this point, tendon releases are performed; for larger immobile rotator cuff tears, the objective is to achieve a tension-free repair of the rotator cuff, so the tendons move easily in line with the direction of the retraction to the footprint; a proper release includes resection of the coracohumeral and superior glenohumeral ligaments, resection of the rotator interval to the base of the coracoid, and a release between the undersurface of the rotator cuff and the glenoid neck. An interval slide technique between supraspinatus and infraspinatus, as suggested by Burkhart et al. [44], can be additionally performed if needed; our experience with the rotator interval slide technique is limited, but we failed to find any benefit or increased tendon excursion, like other authors [45]. It is also important to not separate the anterior attachment of the supraspinatus tendon to the coracohumeral ligament and the subscapularis (the so-called "comma sign"), as this compromises the strength of the distal tendon repair. Anterior repair in continuity enormously helps in the reduction of the supraspinatus once the subscapularis is in place; thus we rarely perform anterior interval slide. Traction sutures may also be helpful in exposing adhesions to the rotator cuff during the release, managing tendons, and relieving tension during the knot tying. Medialization of the footprint can be necessary in cases with severe retraction of supraspinatus and/or infraspinatus.

Once adequate release has been achieved, it is important to recheck the tension and confirm the viability to perform a double-row repair. The greater tuberosity is gently decorticated with a burr or shaver. In most double-row constructs, two double-threaded 4.75- or 5.5-mm suture anchors (Healicoil Regenesorb®; Smith&Nephew, Andover, MA, USA) are placed medially at the junction of the articular surface and the greater tuberosity. Pilot holes are then performed using a punch or a tap, depending on the quality of the bone. Typically, anchors are placed from an accessory more medialized lateral portal in order to achieve an adequate insertion angle of 45°. The posterolateral and anterolateral portals are utilized to pierce the tendon using a self-retrieving hook device (Cleverhook®; Depuy Mitek, Raynham, MA, USA) in a horizontal mattress pattern depending on the configuration and size of the tear. In some cases, a suture passer grasp (Firstpass suture passer®; Smith&Nephew) or a shuttle relay device (Suturelasso®; Arthrex, Naples, FL, USA) is preferable to minimize tissue damage during the suture passage. When all the sutures from each anchor are passed through the rotator cuff, they should be clamped together outside of the skin to optimize suture management. The suture pairs coming from the same anchor are grasped together before proceeding to the knot tying of the medial row; sutures from each anchor are preserved without cutting and used to link them to the lateral row. The best portal to insert our anterior and posterior lateral row anchors is usually the anterolateral portal. One limb from each single knot tied medially is retrieved and loaded into the eyelet of a 5.5-mm knotless anchor (Multifix S®; Smith&Nephew). While some tension is applied to the threads, the anchor is placed just lateral to the bursal rotator cuff footprint on the greater tuberosity. The remaining limbs are gathered, and a second anchor is loaded and placed in a similar fashion to complete the TOE repair. Cortical bone in the lateral aspect of the footprint is usually weak; therefore burying the anchors in this area is not recommended.

Occasionally, for small and medium-size non-delaminated tears, we may load single tape-suture in two medial-row knotless anchors in order to reproduce the double-row TOE-type configuration as described initially by Park et al. [46].

When a delaminated rotator cuff tear is present, we often use the technique of the lasso-loop stitch for the deep layer, as described by Lafosse et al. (Fig. 20.3), bringing it down effectively to the native medial footprint [30]. It is impor-

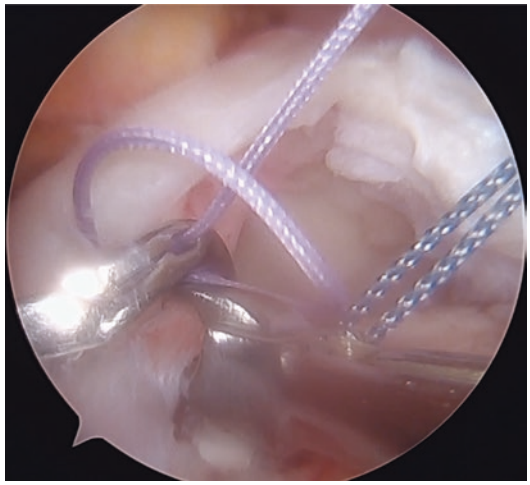


Fig. 20.3 Intraoperative arthroscopic view from the lateral portal of the rotator cuff delaminated tear. On the left, the retrieval hook (Cleverhook® Depuy Mitek) was passed through the lateral edge of the deep layer of the infraspinatus posterior tear and one of the sutures (purple) was partially retrieved, leaving a loop so the same thread can be again passed through the loop to complete the lasso-loop stitch

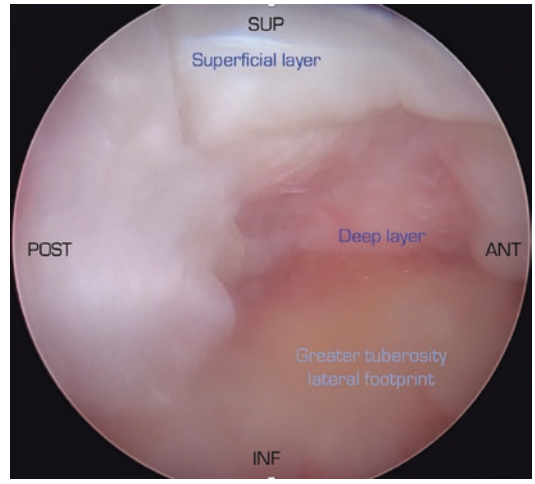


Fig. 20.4 Intraoperative arthroscopic view from the lateral portal of the partially repaired rotator cuff deep layer using the lasso-loop technique, bringing down effectively the deep cuff layer to the native medial footprint

tant to ensure that both the superficial and deep tendon possess appropriate mobility for anatomic repair by pulling each layer independently. In order to perform the lasso-loop stitch, the retrieval hook is passed through the lateral edge of the deep layer of the tear and one suture is partially retrieved, making a loop, then the curved tip of the device must enter through the loop and retrieve the free end of the same suture, forming the lasso-loop pulling system. The free end of the lasso-loop is then passed through the superficial layer as well. The other thread from the anchor is passed in a conventional manner through both layers of the tear. The lasso-loop technique allows, by pulling from the non-loop thread, restoring anatomically the deep layer to the footprint (Fig. 20.4).

Commonly the posterior cuff covers most of the posterior greater tuberosity original footprint; however, in massive irreparable cuff tears frequently the anterior margin of the cuff fails to cover the entire footprint and remains medialized; thus, a partial repair is performed at this point. When the antero-superior cuff is very difficult to mobilize, several side-to-side stitches can approximate the edge of the tendon and then a single-row repair of the anterior supraspinatus and subscapularis can be performed using a modified Mason-Allen suture technique. Some tips and tricks to achieve a satisfactory repair are reported in Table 20.1.

20.3.2 Anterosuperior Partial Repair

Double-row fixation of the subscapularis is challenging because of the small anterior space overlying the subscapularis. Whereas the subacromial space allows freedom of movement, the limited subcoracoid space makes visualization, instrument manipulation, and knot tying more difficult. Denard et al. first described the technique for double-row subscapularis repair [47]. The same principles described for posterosuperior rotator cuff repair are followed for subscapularis repair. For type I to III subscapularis tears (Lafosse classification) [48], a single-row configuration with one or two 4.75 mm double-loaded anchors seems sufficient; we reserve double-row repair for the largest subscapularis tendon tears, types IV and V [48].

The patient is placed in the BCP, and the camera is kept in the posterior portal to proceed with

Table 20.1 Tips, tricks, and pitfalls in arthroscopic partial and incomplete repair of massive and irreparable rotator cuff tears

– Use additional portals as needed for suture management, cuff release or anchor placement
– For visualization, avoid excessive water extravasation and turbulence, plugging temporarily the portals with a needle cup or an urinary catheter plug
– Identify the tear pattern and do work in your cuff release as described
– Use temporary traction sutures to improve your release and facilitate suture passing
– Medialize the footprint if needed using a motorized burr
– Be aware that most of the larger rotator cuff tears are delaminated and include the inferior layer in your repair
– Do not bury the anchors in the lateral row; cortical bone is weaker in this area
– Use the adequate tap or punch depending on the quality of the bone and be aware of the presence of previous bone cysts in the humeral head
– If you are not using cannulas, always shuttle the involved two sutures out together and take out any other suture in that portal before tying
– During TOE repairs, separate enough the threads' couples and avoid over-tensioning the medial row knot tying
– Do not tie knots through the musculotendinous junction

the repair from the intraarticular site in most cases; for the types IV and V and most retracted tears with multiple adhesions, we may move the camera to a more lateral or anterolateral portal in order to improve visualization when performing the extensive circumferential release of the subscapularis required for these tears. Because most tears begin at the upper articular surface, a bare lesser tuberosity footprint is indicative of a tear. Sometimes the footprint is difficult to identify and placing the arm in abduction and internal rotation helps to visualize the subscapularis footprint; also pushing back the shoulder from the arm will expose the superior and deep insertion of the subscapularis tendon.

For the medial row, anchors are inserted at the border between the cartilage and bone through an anterior portal lateral to the coracoid process. Medialization of the footprint can be necessary in cases with severe subscapularis retraction. Sutures are retrieved through the accessory anterolateral portal, and then a suture-passing device passed through the anterior portal pierce the tendon anteriorly to retrieve the sutures sequentially. Once the sutures are tied, the suture ends from the medial row are used to feed an additional knotless anchor, accomplishing the lateral-row fixation.

20.4 Postoperative Care

We perform most of our cuff repairs as an outpatient procedure except for those patients who are not medically suitable. A brachial plexus block is performed as previously described, which results in a great analgesic effect for at least 10 h. Before discharge, patients are instructed to start taking oral medication at home regularly from about 6 h after surgery (whilst the block is still working). Postoperative analgesia after discharge consists of an oral non-steroidal anti-inflammatory (NSAID) agent (ibuprofen, 600 mg/8 h) combined with acetaminophen 1000 mg/8 h. Patients are instructed to receive oral tramadol 50 mg/12 h as a rescue medication. After 48 h from surgery, patients are recommended to reduce doses as soon as the pain subsides; cold therapy commences 3 h after surgery and is used 15 min at a time every 2 h to control pain and swelling. NSAIDs intake is limited postoperatively because of the potential side effects and the known adverse impact on tissue healing and bone metabolism [49, 50].

The postoperative rehabilitation program is critical for success after partial or incomplete repair of massive rotator cuff tears. There is no agreement about the best timing to start rehabilitation postoperatively; we delay physical therapy for these complex repairs until the sixth week even if a complete repair has been achieved. Parson et al. found that range of motion (ROM) restriction did not predispose to stiffness at 1 year in this group of patients [51].

At 6 weeks, we initiate supervised passive motion until full ROM is achieved. From 8 to 12 weeks, a progression from active-assisted to active motion is allowed. At 3 months, ROM is unrestricted and patients are allowed

to perform daily activities with minimal caution. Proper strengthening-resisted exercises begin at 3–4 months depending on the quality of the repair and the patient.

20.5 Literature Review

Several studies reported clinical outcomes after partial repair of massive irreparable rotator cuff tears [4, 5, 10, 13, 14, 52]. All studies found a statistically significant improvement in functional outcome scores compared with preoperative values although some have also shown a relatively high failure rate [4, 5, 8, 14, 52].

Shon et al. [5] evaluated patients at 1 and 2 years after partial repair; the mean VAS, American Shoulder and Elbow Surgeons (ASES), and Simple Shoulder Test (SST) scores significantly improved ($p < 0.003$) from pre-operatively to 1-year follow-up. However, despite this initial improvement, the number of patients reporting that they were dissatisfied with the procedure increased from 1-year (6%) to >2-year follow-up (32%). Additionally, the VAS score was significantly worse at >2-year follow-up (3.2) compared with 1-year follow-up (2.1) ($p = 0.039$).

Chen et al. [14] found that failure rate of partial repair was 41.6%.

The rate of complications other than failure or the need for revision is low with partial repair of massive irreparable tears and is reported to be 4% [8]. Three studies evaluated ROM in patients undergoing partial repair of massive irreparable rotator cuff tears [4, 8, 52]. Duralde and Bair [8] found an average increase in both forward flexion (114° to 154°) and external rotation (44° to 54°) over an average follow-up of 43 months. Cuff et al. [4] found no significant changes in forward flexion (–14°; $p = 0.07$), external rotation (1°; $p > 0.99$), or internal rotation (–4°; $p > 0.99$) from baseline conditions; however, only patients with preoperative forward flexion >120° were included in this study. Galasso et al. [52] analyzed ROM of operated shoulders in comparison with the contralateral shoulder; no significant difference was found between the two shoulders with regard to forward flexion or external rotation, although patients showed an average loss of 10° in abduction ($p < 0.001$) and significant loss in internal rotation ($p < 0.001$) in the affected shoulder compared with the contralateral side at an average follow-up of 7 years.

Yoo et al. [7] reported clinical results and structural integrity of patients with a minimum 2-year follow-up comparing arthroscopic incomplete repair vs. partial repair for large–to-massive rotator cuff tears. They evaluated retrospectively 65 patients; 45 patients underwent arthroscopic incomplete (hybrid) repair, and 20 patients received partial repair for large-to-massive cuff tears. Comparisons of the preoperative values with follow-up results of hybrid incomplete repair showed significant improvement ($p = 0.001$) in the mean pain VAS score (from 5.56 to 0.93), mean function VAS score (from 4.77 to 8.59), ASES score (from 44.89 to 84.67), and Constant score (from 44.27 to 73.46). Most ROM measures showed some improvement compared with preoperative ROM at the last follow-up (2 years). However, there was no significant difference. Re-tear occurred in 9 patients (20%) in the hybrid-incomplete repair group. Most of the postoperative clinical outcomes showed excellent results with incomplete repair compared with partial repair.

20.6 Conclusions

For irreparable rotator cuff tears, achieving a balanced pair force with partial or incomplete repair can reach good enough results for many patients in the mid-term and does not preclude further surgery; by using tendon mobilization techniques, medializing the anterior portion of the repair and using double/single-row and side-to-side repair techniques, we can achieve close to complete humeral head coverage in most cases and improved healing rates.

References

1. Burkhart SS, Nottage WM, Ogilvie-Harris DJ, Kohn HS, Pachelli A. Partial repair of irreparable rotator cuff tears. Arthroscopy. 1994;10(4):363–70.
2. Burkhart SS. Arthroscopic treatment of massive rotator cuff tears. Clin Orthop Relat Res. 2001;390:107–18.
3. Castricini R, Galasso O, Riccelli DA, Familiari F, De Benedetto M, Orlando N, et al. Arthroscopic partial repair of irreparable, massive rotator cuff tears. Arthrosc Tech. 2017;6(1):e143–e7.
4. Cuff DJ, Pupello DR, Santoni BG. Partial rotator cuff repair and biceps tenotomy for the treatment of patients with massive cuff tears and retained overhead elevation: midterm outcomes with a minimum 5 years of follow-up. J Shoulder Elbow Surg. 2016;25(11):1803–9.
5. Shon MS, Koh KH, Lim TK, Kim WJ, Kim KC, Yoo JC. Arthroscopic partial repair of irreparable rotator cuff tears: preoperative factors associated with outcome deterioration over 2 years. Am J Sports Med. 2015;43(8):1965–75.
6. Heuberer PR, Kolblinger R, Buchleitner S, Pauzenberger L, Laky B, Auffarth A, et al. Arthroscopic management of massive rotator cuff tears: an evaluation of debridement, complete, and partial repair with and without force couple restoration. Knee Surg Sports Traumatol Arthrosc. 2016;24(12):3828–37.
7. Jeong JY, Yoon YC, Lee SM, Yoo JC. Arthroscopic incomplete repair using a "hybrid technique" for large to massive rotator cuff tears: clinical results and structural integrity. Arthroscopy. 2018;34(7):2063–73.
8. Duralde XA, Bair B. Massive rotator cuff tears: the result of partial rotator cuff repair. J Shoulder Elbow Surg. 2005;14(2):121–7.
9. Burkhart SS. Reconciling the paradox of rotator cuff repair versus debridement: a unified biomechanical rationale for the treatment of rotator cuff tears. Arthroscopy. 1994;10(1):4–19.
10. Kim SJ, Lee IS, Kim SH, Lee WY, Chun YM. Arthroscopic partial repair of irreparable large to massive rotator cuff tears. Arthroscopy. 2012;28(6):761–8.
11. Kissenberth MJ, Rulewicz GJ, Hamilton SC, Bruch HE, Hawkins RJ. A positive tangent sign predicts the repairability of rotator cuff tears. J Shoulder Elbow Surg. 2014;23(7):1023–7.
12. Sheean AJ, Hartzler RU, Denard PJ, Ladermann A, Sanders TG, Zlatkin MB, et al. Preoperative radiographic risk factors for incomplete arthroscopic supraspinatus tendon repair in massive rotator cuff tears. Arthroscopy. 2018;34(4):1121–7.
13. Braun S, Steadman JR, Rodkey WG, Briggs KK. Microfracture and specific rehabilitation for treating osteoarthritis of the knee. Indications, surgical technique, and rehabilitation protocol. Z Rheumatol. 2009;68(10):811–8.
14. Chen KH, Chiang ER, Wang HY, Ma HL. Arthroscopic partial repair of irreparable rotator cuff tears: factors related to greater degree of clinical improvement at 2 years of follow-up. Arthroscopy. 2017;33(11):1949–55.
15. Henn RF 3rd, Kang L, Tashjian RZ, Green A. Patients' preoperative expectations predict the outcome of rotator cuff repair. J Bone Joint Surg Am. 2007;89(9):1913–9.
16. Lee YS, Jeong JY, Park CD, Kang SG, Yoo JC. Evaluation of the risk factors for a rotator cuff retear after repair surgery. Am J Sports Med. 2017;45(8):1755–61.
17. Romeo AA, Hang DW, Bach BR Jr, Shott S. Repair of full thickness rotator cuff tears. Gender, age, and other factors affecting outcome. Clin Orthop Relat Res. 1999;367:243–55.
18. Goldberg BA, Nowinski RJ, Matsen FA 3rd. Outcome of nonoperative management of full-thickness rotator cuff tears. Clin Orthop Relat Res. 2001;(382):99–107.
19. Itoi E, Tabata S. Conservative treatment of rotator cuff tears. Clin Orthop Relat Res. 1992;275:165–73.
20. Varvitsiotis D, Papaspiliopoulos A, Antipa E, Papacharalampous X, Flevarakis G, Feroussis J. Results of reconstruction of massive irreparable rotator cuff tears using a fascia lata allograft. Indian J Orthop. 2015;49(3):304–11.
21. Mihata T, Lee TQ, Watanabe C, Fukunishi K, Ohue M, Tsujimura T, et al. Clinical results of arthroscopic superior capsule reconstruction for irreparable rotator cuff tears. Arthroscopy. 2013;29(3):459–70.
22. Denard PJ, Brady PC, Adams CR, Tokish JM, Burkhart SS. Preliminary results of arthroscopic superior capsule reconstruction with dermal allograft. Arthroscopy. 2018;34(1):93–9.
23. Post M, Silver R, Singh M. Rotator cuff tear. Diagnosis and treatment. Clin Orthop Relat Res. 1983;173:78–91.
24. Kim DH, Elattrache NS, Tibone JE, Jun BJ, DeLaMora SN, Kvitne RS, et al. Biomechanical comparison of a single-row versus double-row suture anchor technique for rotator cuff repair. Am J Sports Med. 2006;34(3):407–14.
25. Lorbach O, Kieb M, Raber F, Busch LC, Kohn D, Pape D. Comparable biomechanical results for a modified single-row rotator cuff reconstruction using triple-loaded suture anchors versus a suture-bridging double-row repair. Arthroscopy. 2012;28(2):178–87.
26. Ma CB, Comerford L, Wilson J, Puttlitz CM. Biomechanical evaluation of arthroscopic rotator cuff repairs: double-row compared with single-row fixation. J Bone Joint Surg Am. 2006;88(2):403–10.

27. Ma HL, Chiang ER, Wu HT, Hung SC, Wang ST, Liu CL, et al. Clinical outcome and imaging of arthroscopic single-row and double-row rotator cuff repair: a prospective randomized trial. Arthroscopy. 2012;28(1):16–24.
28. Mazzocca AD, Millett PJ, Guanche CA, Santangelo SA, Arciero RA. Arthroscopic single-row versus double-row suture anchor rotator cuff repair. Am J Sports Med. 2005;33(12):1861–8.
29. Park MC, Elattrache NS, Ahmad CS, Tibone JE. "Transosseous-equivalent" rotator cuff repair technique. Arthroscopy. 2006;22(12):1360 e1–5.
30. Lafosse L, Brzoska R, Toussaint B, Gobezie R. The outcome and structural integrity of arthroscopic rotator cuff repair with use of the double-row suture anchor technique. Surgical technique. J Bone Joint Surg Am. 2008;90(Suppl 2 Pt 2):275–86.
31. Mall NA, Lee AS, Chahal J, Van Thiel GS, Romeo AA, Verma NN, et al. Transosseous-equivalent rotator cuff repair: a systematic review on the biomechanical importance of tying the medial row. Arthroscopy. 2013;29(2):377–86.
32. Anderl W, Heuberer PR, Laky B, Kriegleder B, Reihsner R, Eberhardsteiner J. Superiority of bridging techniques with medial fixation on initial strength. Knee Surg Sports Traumatol Arthrosc. 2012;20(12):2559–66.
33. Pauly S, Fiebig D, Kieser B, Albrecht B, Schill A, Scheibel M. Biomechanical comparison of four double-row speed-bridging rotator cuff repair techniques with or without medial or lateral row enhancement. Knee Surg Sports Traumatol Arthrosc. 2011;19(12):2090–7.
34. Park MC, Peterson A, Patton J, McGarry MH, Park CJ, Lee TQ. Biomechanical effects of a 2 suture-pass medial inter-implant mattress on transosseous-equivalent rotator cuff repair and considerations for a "technical efficiency ratio". J Shoulder Elbow Surg. 2014;23(3):361–8.
35. Millett PJ, Warth RJ. Posterosuperior rotator cuff tears: classification, pattern recognition, and treatment. J Am Acad Orthop Surg. 2014;22(8):521–34.
36. Vaishnav S, Millett PJ. Arthroscopic rotator cuff repair: scientific rationale, surgical technique, and early clinical and functional results of a knotless self-reinforcing double-row rotator cuff repair system. J Shoulder Elbow Surg. 2010;19(2 Suppl):83–90.
37. Millett PJ, Espinoza C, Horan MP, Ho CP, Warth RJ, Dornan GJ, et al. Predictors of outcomes after arthroscopic transosseous equivalent rotator cuff repair in 155 cases: a propensity score weighted analysis of knotted and knotless self-reinforcing repair techniques at a minimum of 2 years. Arch Orthop Trauma Surg. 2017;137(10):1399–408.
38. Cho NS, Lee BG, Rhee YG. Arthroscopic rotator cuff repair using a suture bridge technique: is the repair integrity actually maintained? Am J Sports Med. 2011;39(10):2108–16.
39. Cho NS, Yi JW, Lee BG, Rhee YG. Retear patterns after arthroscopic rotator cuff repair: single-row versus suture bridge technique. Am J Sports Med. 2010;38(4):664–71.
40. Virk MS, Bruce B, Hussey KE, Thomas JM, Luthringer TA, Shewman EF, et al. Biomechanical performance of medial row suture placement relative to the musculotendinous junction in transosseous equivalent suture bridge double-row rotator cuff repair. Arthroscopy. 2017;33(2):242–50.
41. Reddy A, Gulotta LV, Chen X, Castagna A, Dines DM, Warren RF, et al. Biomechanics of lower trapezius and latissimus dorsi transfers in rotator cuff-deficient shoulders. J Shoulder Elbow Surg. 2019;28(7):1257–64.
42. Samitier G, Alentorn-Geli E, Torrens C, Wright TW. Reverse shoulder arthroplasty. Part 1: systematic review of clinical and functional outcomes. Int J Shoulder Surg. 2015;9(1):24–31.
43. Papadonikolakis A, Wiesler ER, Olympio MA, Poehling GG. Avoiding catastrophic complications of stroke and death related to shoulder surgery in the sitting position. Arthroscopy. 2008;24(4):481–2.
44. Lo IK, Burkhart SS. Arthroscopic repair of massive, contracted, immobile rotator cuff tears using single and double interval slides: technique and preliminary results. Arthroscopy. 2004;20(1):22–33.
45. Kim SJ, Kim SH, Lee SK, Seo JW, Chun YM. Arthroscopic repair of massive contracted rotator cuff tears: aggressive release with anterior and posterior interval slides do not improve cuff healing and integrity. J Bone Joint Surg Am. 2013;95(16):1482–8.
46. Park MC, ElAttrache NS, Tibone JE, Ahmad CS, Jun BJ, Lee TQ, Part I. Footprint contact characteristics for a transosseous-equivalent rotator cuff repair technique compared with a double-row repair technique. J Shoulder Elbow Surg. 2007;16(4):461–8.
47. Denard PJ, Ladermann A, Burkhart SS. Double-row fixation of upper subscapularis tears with a single suture anchor. Arthroscopy. 2011;27(8):1142–9.
48. Lafosse L, Lanz U, Saintmard B, Campens C. Arthroscopic repair of subscapularis tear: Surgical technique and results. Orthop Traumatol Surg Res. 2010;96(8 Suppl):S99–108.
49. Chechik O, Dolkart O, Mozes G, Rak O, Alhajajra F, Maman E. Timing matters: NSAIDs interfere with the late proliferation stage of a repaired rotator cuff tendon healing in rats. Arch Orthop Trauma Surg. 2014;134(4):515–20.
50. Chen MR, Dragoo JL. The effect of nonsteroidal anti-inflammatory drugs on tissue healing. Knee Surg Sports Traumatol Arthrosc. 2013;21(3):540–9.
51. Parsons BO, Gruson KI, Chen DD, Harrison AK, Gladstone J, Flatow EL. Does slower rehabilitation after arthroscopic rotator cuff repair lead to long-term stiffness? J Shoulder Elbow Surg. 2010;19(7):1034–9.
52. Galasso O, Riccelli DA, De Gori M, De Benedetto M, Orlando N, Gasparini G, et al. Quality of life and functional results of arthroscopic partial repair of irreparable rotator cuff tears. Arthroscopy. 2017;33(2):261–8.

Patch Graft Augmentation

21

Florian Hess, Daniel Smolen, and Jan Leuzinger

21.1 Introduction

Surgical management of large symptomatic rotator cuff (RC) tears is a challenge. Large tears are associated with a high failure rate, even in the absence of osteoarthritis or fatty infiltration. The primary goals of surgery are complete rotator cuff reconstruction and anatomic preservation. The outcome is largely dependent on (1) the size and type of the tear, (2) the duration of the condition—chronic cases can lead to diminished soft tissue quality and a higher degree of tendon retraction—and (3) patient's age and comorbidities. According to reports in the medical literature, the recurrence rate is high and varies among studies [1–5]. A successful outcome (structural integrity) depends on bone fixation strength and bone and tendon quality [6–8]. Despite advances in techniques and repair technology (e.g., anchor type, use of tapes, double- or triple-row repair techniques), healing rates have remained relatively stable over time [9].

Debridement or margin convergence techniques [10, 11] can lead to acceptable short-term results, especially in low-demand patients such as the elderly. However, long-term clinical outcomes are less favorable [12–15]. Extra-anatomical tendon transfers, such as pectoralis major transfer [16], latissimus dorsi transfer [17], or deltoid muscle flaps [18], are often used to treat tendon defects and irreparable tears. Although tendon transfers are viable treatment options, especially in younger patients [19, 20], they are often associated with higher complication rates [21].

21.2 Rotator Cuff Augmentation

Augmentation most often consists of patches of either biologic or synthetic origin [22]. Biologic patches have the inherent advantage of being degradable and are considered more biocompatible [23]. However, their main disadvantage is that they provide only short-term reinforcement of the construct. The relatively low mechanical properties, as well as the undefined degradation rate and the variations in biocompatibility depending on the source of the graft (autogeneic, allogeneic, or xenogeneic), have often led to uneven loading or complete failure of the construct. Although failure can be attributed to all the reasons mentioned before, inflammation can be caused by either an immunologic response to the respective graft or the degradation process itself.

In the 1990s and early 2000s, synthetic patches to reinforce the tendon were introduced

F. Hess (✉)
Department of Orthopaedic Surgery and Traumatology, Cantonal Hospital Frauenfeld, Frauenfeld, Switzerland
e-mail: florian.hess@stgag.ch

D. Smolen · J. Leuzinger
Etzel Clinic, Pfaeffikon, Switzerland
e-mail: leuzinger@etzelclinic.ch

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_21

to the market [24–27]. In vitro and animal testing of these patches showed promising results, which included favorable soft tissue incorporation and foreign body reaction. However, results from initial clinical trials were poor. Similarly, discouraging results were reported when using the first xenograft made from porcine small intestine submucosa [28–30].

Nevertheless, different patch types are currently available on the market. To date, the limited empirical evidence that has been gathered was from clinical trials with small sample sizes, and comparative studies are lacking.

21.2.1 Autografts

Although the medical literature includes case reports of rotator cuff repairs using an autograft, such as fascia lata or Achilles tendon, larger trials assessing long-term clinical outcome of those procedures have not yet been conducted [24]. Other autografts (e.g., a periosteal flap, the transplanted long head of the biceps, or a free coracoacromial ligament graft) do not naturally cause adverse reactions and can positively influence re-tear rates. These types of autografts were used to treat large or massive rotator cuff tears in only a small number of patients. These were done mostly as interposition instead of augmentation. Furthermore, the lack of a control group made comparisons difficult.

21.2.2 Allografts

Allografts, such as freeze-dried RC allografts [31, 32] and fascia lata allografts [33], have shown good clinical and radiological outcomes, when assessed.

Several companies offer acellular human dermis for repairing rotator cuff tears (ArthroFLEX®, Arthrex, LifeNet Health, Virginia Beach, VA, USA; Allopatch HD®, MTF Sports Medicine, Edison, NJ, USA; GraftJacket® Wright Medical Technology). Most evidence-based results originate from studies using the GraftJacket®. Acellular human skin consists of collagen (types I, III, IV, and VII), elastin, chondroitin sulfate, proteoglycans, and fibroblast growth factor, and the grafts are obtained from tissue banks [34, 35]. The tissue is rendered *acellular* because the dermal and epidermal cells are completely removed during processing. However, the vascular channels are preserved to allow rapid infiltration of new fibroblasts, and the vascular tissue is kept minimizing host inflammatory response [35, 36].

Several studies reported improvements in clinical and functional outcomes when using human dermal grafts for augmentation of the rotator cuff [37–43].

Barber et al. [37] conducted a prospective randomized controlled trial comparing GraftJacket® for augmentation ($n = 22$) and repair only ($n = 20$) in patients with chronic two-tendon tears. At a mean follow-up period of 24 months, American Shoulder and Elbow Surgeons (ASES) shoulder score and Constant-Murley score (CMS) improved in the two groups, although both scores were significantly better in the augmented group. The repairs were intact in 85% of the augmented group and 40% in the non-augmented group.

Bond et al. [38] radiologically assessed full incorporation of the GraftJacket® in >70% of their patients.

Gupta et al. [39] reported similar results from a study with 24 patients. Overall, the results of using allografts for the augmentation of large rotator cuff tears have been promising. Improvements in clinical and functional results, including a high rate of ingrowth and repair integrity, were significantly better than results of the non-augmented groups [44, 45].

21.2.3 Xenografts

Extracellular matrix for augmentation or interposition of a rotator cuff tear is one viable treatment option, and several products are currently available on the market. Initially, the only type of graft was from porcine small intestine submucosa (SIS) (Restore Orthobiologic Implant; DePuy, Warsaw, IN, USA) and consisted of ten non-cross-linked layers of up to 1-mm-thick tissue [35]. Earlier studies found high failure rates with this patch,

which resulted in recommendations to discontinue its use [28–30]. Since Iannotti et al. [28] reported less favorable outcomes in patients who received the porcine SIS compared to the control group without augmentation, the authors discouraged further use of this method. One possible explanation for these poor outcomes was that early resorption of the graft may have led to weakness of the construct and then failure [46, 47]. Nevertheless, these suboptimal results from the porcine small intestine submucosa prompted its discontinuation in Europe and discouragement of further use by the American Academy of Orthopaedic Surgeons (AAOS) [28, 30, 48].

The new generation of porcine xenografts is mostly porcine dermal grafts (Zimmer Collagen Repair Patch, Zimmer, Warsaw, IN, USA; Conexa, Wright Medical, Arlington, IN, USA; Permacol, Medtronic, Mansfield, MA, USA), which are thicker than the original tissue. Others are made of bovine pericardium (Tutopatch, Tutogen Medical GmbH, Neunkirchen am Brand, Germany) or fetal bovine dermis (Tissue Mend, Stryker Orthopedics, Mahwah, NJ, USA). Recent findings gathered from studies using bovine dermal graft for augmentation and interposition of rotator cuff tears are inconclusive. While some authors reported promising results [49], others discouraged the use of xenografts [50].

21.2.4 Synthetic Patches

Since biologic patches have low mechanical properties and synthetic patches can permanently reinforce the construct, irrespective of tissue ingrowth, the synthetic options have been successfully introduced into the market [22, 23]. Besides their superior mechanical strength, they also exhibit better control of chemical and physical properties.

A large variety of synthetic patches for augmentation/interposition of rotator cuff tears currently exist. Most consist of polyethylene terephthalate (Poly-Tape, Neoligaments, Leeds, UK; LARS Ligament, LARS, Arc-sur-Tille, Burgundy, France; Mersilene mesh, Ethicon, Inc., Somerville, NJ, USA). Others are made of poly-L-lactic acid (X-Repair, Synthasome, San Diego, CA, USA), carbon fiber tow (Integraft, Hexcel Medical, Dublin, CA, USA), polytetrafluoroethylene (Teflon, Dupont Company, Wilmington, DE, USA), or polypropylene (Repol Angimesh, ANGIOLOGICA BM Srl, Pavia, Italy) [35, 51].

The authors' preferred patch is the Pitch-Patch (IST, Innovative Shoulder Technology AG, Cham, Switzerland) (Fig. 21.1), a polyester patch with a shape that adapts to the anatomy and has reinforced borders and prepared suture holes designed for augmentation of rotator cuff tendons [52]. The pullout strength (300 N) of this patch was designed and tested to meet the specific requirements for treating large RC tears. Polyethylene terephthalate, the most common thermoplastic polymer resin of the polyester family, is used to make fibers for clothing and containers for liquids and foods. It is also used in medical devices, such as suture material. The chemical formula is $(C_{10}H_8O_4)_n$, and single polyethylene terephthalate fibers can be further processed and woven into shape. In a study carried out by researchers at our institution [52], we were able to show histologically complete soft tissue integration without foreign body reaction (Fig. 21.2).

According to the literature, synthetic patches are effective in reconstructing large or massive rotator cuff tears and in restoring good functionality [25, 53–55].

Fig. 21.1 The Pitch-Patch provides seven predefined holes and measures 2 × 3 cm. To attach the patch medially, there are three holes; laterally, there are two holes; and anteriorly and posteriorly, there is one additional hole on each side

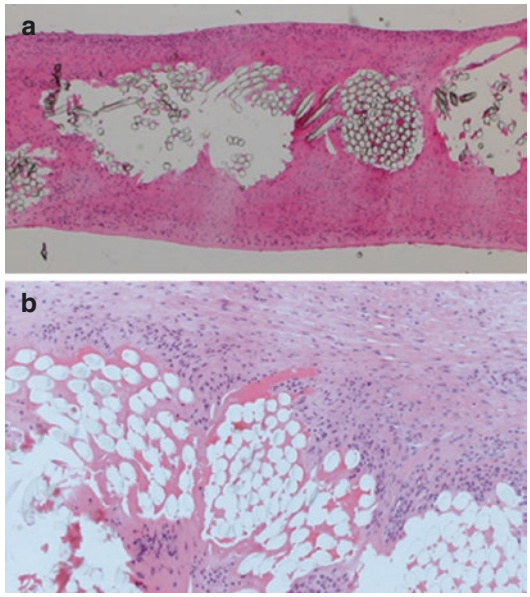


Fig. 21.2 Standard hematoxylin and eosin histology of an explanted patch in a patient who underwent revision for symptomatic crepitus. At 100× magnification (**a**), complete integration of the synthetic polyester patch is evident (white areas around patch fibers are due to fixation artifacts). Signs of foreign body reaction or tissue rejection are not detectable (**b**)

In 1986, Ozaki et al. [25] presented the first study of large rotator cuff tears and augmentation with a polyester graft and Teflon. Unfortunately, no standardized scoring system to evaluate the clinical outcomes was used. Although the authors found favorable results, the use of carbon fiber was eventually stopped due to reactive synovitis and fragmentation [26, 27]. Significant improvements in clinical and functional outcomes were obtained when using the LARS ligament in 17 patients, with confirmed intact tendon in 15 patients after the 36-month follow-up [55].

Ciampi et al. [56] compared clinical and functional outcomes in patients who underwent repair of massive posterosuperior rotator cuff tears treated with the following: (1) augmented with polypropylene patch, (2) non-augmented, or (3) Tutopatch®. During the 3-year follow-up visits, patients with a synthetic patch had significantly better functional outcomes than the non-augmented or Tutopatch® groups.

The potential disadvantage of synthetic patches is comparable to the presence of a foreign body, which continually has the potential to lead to a chronic immune response or reaction, making them prone to infection [57].

21.3 Authors' Preferred Technique

Several techniques to augment the cuff, which are safe and reproducible, are currently being used. According to recently published literature, synthetic patches are superior to xenografts or allografts [44, 45]. However, clear guidelines on the use of these patches are lacking. The conditions under which a patient would clearly benefit from an augmented cuff remain unclear. Nevertheless, indications for augmentation are most likely (1) poor tendon tissue quality, (2) high tension on the repaired cuff, (3) partial repairs, or (4) cover tissue loss of the tendon due to the chronic lesion. The following section describes the authors' preferred technique using a synthetic patch.

21.3.1 Surgical Technique

With the patient in the beach chair position, we perform the arthroscopic procedure under general anesthesia, which includes administration of an informal scalenus nerve block. After washing and draping, a standard viewing portal is placed at the dorsal soft spot. Further portals may be used and should not be limited to lateral, dorsolateral, anterolateral, or anterior portals. Intra-articular pathologies, such as tears/instability of the long head of the biceps and subscapularis tendon tears, are usually handled first. In addition, synovectomy and labral debridement are performed, when necessary. After a thorough bursectomy, the rupture of the supraspinatus is fully visualized and then measured.

After debridement of the footprint, we perform a double-row repair for the supraspinatus. If the infraspinatus presents with a complete rupture, it is addressed in the same way. Two double-loaded anchors are placed at the bone-cartilage interface into the previously debrided footprint. If a par-

tial infraspinatus tear is present, we use a posterior anchor at the debrided footprint. In elderly patients with known osteoporosis or reduced bone density, we use two threaded anchors (5.5-mm V-Lox PEEK CF Screw-In Suture Anchors, Parcus Medical, Sarasota, USA). If good bone quality is found in younger patients, all-suture anchors (RC-Y-Knot-Anchor, Conmed, Utica, New York, USA) are used for the medial row. All sutures are then passed through the tendon using a suture passer or directly knotted. Thereafter, the suture bridge is completed as usual using two Plug-n-Twist anchors (IST Innovative Shoulder Technology AG, Cham, Switzerland) at the lower facet of the greater tubercle.

To insert the polyester patch (Pitch-Patch), a flexible percutaneous cannula (Pass-Port Button Cannula; Arthrex, Naples, FL, USA) is inserted into the lateral portal. The four corners of the patch and its potential position are determined intraoperatively by using a measurement tool. If placed correctly, the patch should cover the tendon-bone intersection and most of the suture bridge repair. The Pitch-Patch provides seven predefined holes. To attach the patch medially, there are three holes; for lateral attachment, there are two holes; and for anterior and posterior attachment, there is one additional hole on each side. To attach the patch medially, three sutures (FiberWire 2.0, Arthrex, Naples, FL, USA) are passed through the musculotendinous junction, anterior, median, and posterior of the width of the supraspinatus tendon (Fig. 21.3). One end of each suture is individually shuttled through its respective portal—anteromedial, posteromedial, and anterosupero-lateral.

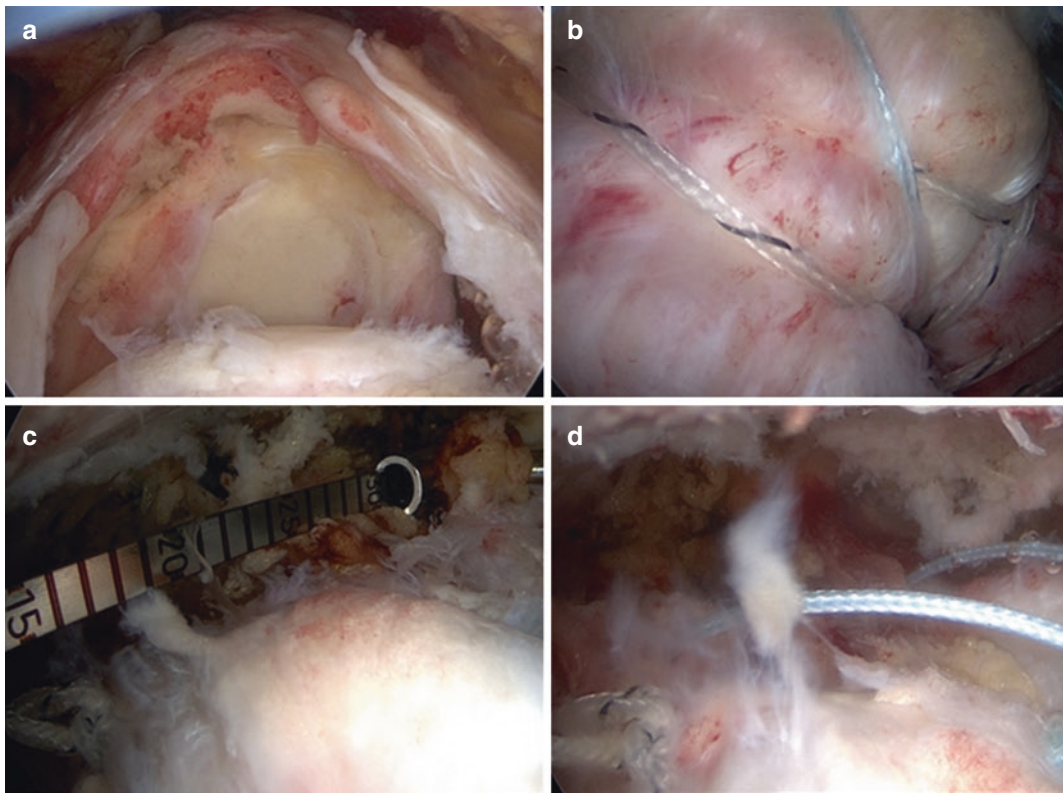


Fig. 21.3 Example of a traumatic complete supraspinatus rupture 6 weeks prior to surgery in a 53-year-old woman (**a**). The cuff was repaired in a double-row technique (**b**). Due to high tension, the decision was made to perform an additional patch augmentation. The correct placement of the medial three fixation sutures was predefined by a measurement tool (**c**). Three fibers were passed through at the corresponding point to prepare the medial patch fixation (**d**)

The corresponding end is gathered through the PassPort cannula (Arthrex) into the lateral portal. It is critical to avoid interfering in soft tissue bridges to allow free passage of the sutures. The three ends are then brought through the three medial holes of the patch and knotted. After the patch is "armed" medially, it is folded lengthwise in a "parachute" manner and inserted into the PassPort cannula. Simultaneously, equal traction is applied on the three ends of the sutures at the respective portals (anteromedial, anterosuperolateral, posteromedial) to correctly place the patch, covering the tendon-bone intersection and most of the suture-bridge repair.

At this point, the arthroscope is placed in the posterolateral portal. All three sutures can now be knotted and the patch spread at the defined position. To attach the path laterally, the fibers of the lateral row of anchors may be used to complete fixation if they were placed in the correct distance or two all-suture anchors (RC-Y-Knot) are inserted at the predefined anterolateral and posterolateral place, according to the predefined distance between the two patch holes. If the anchor is armed with two sutures, one may be removed and the other passed through the corresponding hole with a suture passer. When tying the knots, the tension on the patch may now be determined. As the final step, the intermediate anterior and posterior holes can be used to complete the attachment of the patch (Fig. 21.4).

21.3.2 Results

We recently published clinical, functional, and radiological findings using a synthetic patch

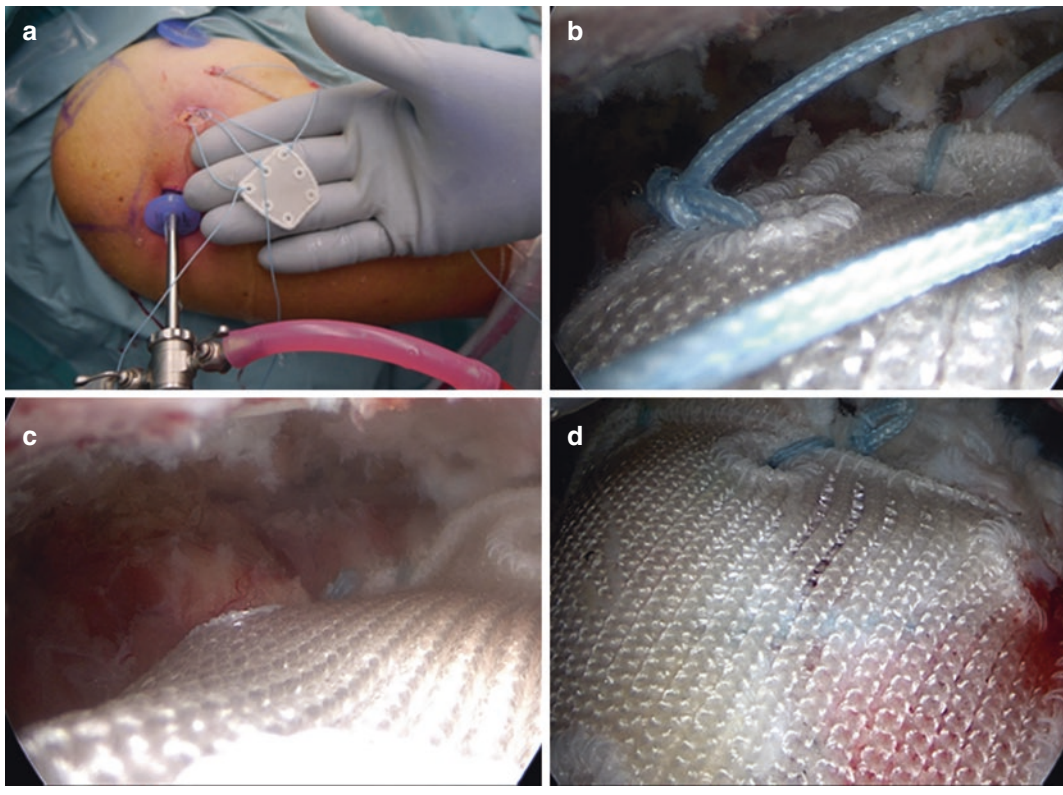


Fig. 21.4 One end of each fiber placed for the medial fixation is passed out through the lateral (or anterolateral) portal where the patch may be fixed by using the predefined holes (**a**). The patch may be pushed through the hole by using a clamp, and the other ends of the three medial fibers may be used to bring the patch in the right position (**b**, **c**). For the lateral patch, fixation fibers of the lateral row of anchors may be used to complete fixation if they were placed in the correct distance according to the patch size (**d**)

for rotator cuff augmentation in a series of 50 patients [52]. The integrity of tendons was analyzed using MR, computed tomography (CT), and ultrasounds. These assessments revealed improved healing when compared to recently published data [58, 59].

All 50 patients benefited from the procedure, with significant improvements observed in the Constant-Murley and Subjective Shoulder Value (SSV) ($p < 0.0001$) scores. Re-tear rate was 14%, which was comparable to standard non-augmented RC repairs. Only one revision was performed because of a re-rupture, whereas most revisions were due to frozen shoulders and arthrofibrosis ($n = 6$, 12%). When comparing intact tendons with re-ruptures, as determined at MR, the patients with intact tendons had significantly better SSV and Constant-Murley scores. Particularly, patients with intact tendons had significantly greater strength according to the Constant-Murley score. However, patient-reported pain levels were not significantly different. Statistical analyses revealed that the greater the retraction grade (according to Patte), the more likely a re-rupture would occur ($p < 0.001$). This was also the case for tendons with increased fatty degeneration, according to Goutallier. Re-ruptures increased as the grade of fatty infiltration increased, although this was not statistically significant.

21.4 Conclusions

The potential benefits of using patches in rotator cuff repairs are not well known. Evidence-based recommendations indicating which rotator cuff conditions have better outcomes and lower rates of re-tears are lacking. Furthermore, the use of different outcome scores makes heterogenic results difficult to compare. Nevertheless, a review of the current literature reveals some trends concerning the benefits of additional patch augmentation [44, 45, 60].

A recent meta-analysis by Bailey et al. [44] reported clinical and functional improvements when using patches as augmentation as well as interposition. Regarding the outcome measures, authors report significantly higher ASES [37, 61, 62] and Constant-Murley scores [37, 42, 55, 63–65] when using patch augmentation or interposition compared to the rotator cuff repair alone, whereas the UCLA scores did not improve significantly [37, 56, 62, 66]. Regarding the clinical and functional outcomes of the different patches, the autograft had the best results when assessed using the ASES and UCLA scores, and allograft had superior results for postoperative pain measured by the visual analogue score (VAS) [44]. Xenografts had the worst results when using the UCLA and Constant-Murley scores [45]. The synthetic grafts seem to have the most favorable results when comparing the Constant-Murley score and postoperative forward flexion.

References

1. Boileau P, et al. Arthroscopic repair of full-thickness tears of the supraspinatus: does the tendon really heal? J Bone Joint Surg Am. 2005;87(6):1229–40.
2. Cole BJ, et al. Arthroscopic rotator cuff repair: prospective functional outcome and repair integrity at minimum 2-year follow-up. J Shoulder Elbow Surg. 2007;16(5):579–85.
3. Kim HM, et al. Factors affecting satisfaction and shoulder function in patients with a recurrent rotator cuff tear. J Bone Joint Surg Am. 2014;96(2):106–12.
4. Papadopoulos P, et al. Functional outcome and structural integrity following mini-open repair of large and massive rotator cuff tears: a 3–5 year follow-up study. J Shoulder Elbow Surg. 2011;20(1):131–7.
5. Rhee YG, Cho NS, Yoo JH. Clinical outcome and repair integrity after rotator cuff repair in patients older than 70 years versus patients younger than 70 years. Arthroscopy. 2014;30(5):546–54.
6. Berdusco R, et al. Arthroscopic repair of massive, contracted, immobile tears using interval slides: clinical and MRI structural follow-up. Knee Surg Sports Traumatol Arthrosc. 2015;23(2):502–7.
7. Goutallier D, et al. Fatty muscle degeneration in cuff ruptures. Pre- and postoperative evaluation by CT scan. Clin Orthop Relat Res. 1994;304:78–83.
8. Sugihara T, et al. Prediction of primary reparability of massive tears of the rotator cuff on preoperative magnetic resonance imaging. J Shoulder Elbow Surg. 2003;12(3):222–5.
9. Randelli P, et al. Regenerative medicine in rotator cuff injuries. Biomed Res Int. 2014;2014:129515.
10. Gartsman GM. Massive, irreparable tears of the rotator cuff. Results of operative debridement and subacromial decompression. J Bone Joint Surg Am. 1997;79(5):715–21.

11. Rockwood CA Jr, Williams GR Jr, Burkhead WZ Jr. Debridement of degenerative, irreparable lesions of the rotator cuff. J Bone Joint Surg Am. 1995;77(6):857–66.
12. Kempf JF, et al. A multicenter study of 210 rotator cuff tears treated by arthroscopic acromioplasty. Arthroscopy. 1999;15(1):56–66.
13. Liem D, et al. Arthroscopic debridement of massive irreparable rotator cuff tears. Arthroscopy. 2008;24(7):743–8.
14. Walch G, et al. Arthroscopic tenotomy of the long head of the biceps in the treatment of rotator cuff tears: clinical and radiographic results of 307 cases. J Shoulder Elbow Surg. 2005;14(3):238–46.
15. Zvijac JE, Levy HJ, Lemak LJ. Arthroscopic subacromial decompression in the treatment of full thickness rotator cuff tears: a 3- to 6-year follow-up. Arthroscopy. 1994;10(5):518–23.
16. Resch H, et al. Transfer of the pectoralis major muscle for the treatment of irreparable rupture of the subscapularis tendon. J Bone Joint Surg Am. 2000;82(3):372–82.
17. Gerber C, et al. Latissimus dorsi tendon transfer for treatment of irreparable posterosuperior rotator cuff tears: long-term results at a minimum follow-up of ten years. J Bone Joint Surg Am. 2013;95(21): 1920–6.
18. Gille J, et al. Deltoid muscular flap transfer for the treatment of irreparable rotator cuff tears. Orthop Rev (Pavia). 2009;1(2):e15.
19. Gerber C. Latissimus dorsi transfer for the treatment of irreparable tears of the rotator cuff. Clin Orthop Relat Res. 1992;275:152–60.
20. Gerber C, Maquieira G, Espinosa N. Latissimus dorsi transfer for the treatment of irreparable rotator cuff tears. J Bone Joint Surg Am. 2006;88(1):113–20.
21. Memon M, et al. Arthroscopic-assisted latissimus dorsi tendon transfer for massive rotator cuff tears: a systematic review. Orthop J Sports Med. 2018;6(6):2325967118777735.
22. Papalia R, et al. Augmentation techniques for rotator cuff repair. Br Med Bull. 2013;105:107–38.
23. Longo UG, et al. Tendon augmentation grafts: a systematic review. Br Med Bull. 2010;94:165–88.
24. Comley AS, Krishnan J. Donor site morbidity after quadriceps tendon harvest for rotator cuff repair. Aust N Z J Surg. 1999;69(11):808–10.
25. Ozaki J, et al. Reconstruction of chronic massive rotator cuff tears with synthetic materials. Clin Orthop Relat Res. 1986;202:173–83.
26. Post M. Rotator cuff repair with carbon filament. A preliminary report of five cases. Clin Orthop Relat Res. 1985;196:154–8.
27. Visuri T, Kiviluoto O, Eskelin M. Carbon fiber for repair of the rotator cuff. A 4-year follow-up of 14 cases. Acta Orthop Scand. 1991;62(4):356–9.
28. Iannotti JP, et al. Porcine small intestine submucosa augmentation of surgical repair of chronic two-tendon rotator cuff tears. A randomized, controlled trial. J Bone Joint Surg Am. 2006;88(6):1238–44.
29. Sclamberg SG, et al. Six-month magnetic resonance imaging follow-up of large and massive rotator cuff repairs reinforced with porcine small intestinal submucosa. J Shoulder Elbow Surg. 2004;13(5):538–41.
30. Walton JR, et al. Restore orthobiologic implant: not recommended for augmentation of rotator cuff repairs. J Bone Joint Surg Am. 2007;89(4):786–91.
31. Nasca RJ. The use of freeze-dried allografts in the management of global rotator cuff tears. Clin Orthop Relat Res. 1988;228:218–26.
32. Neviaser JS, Neviaser RJ, Neviaser TJ. The repair of chronic massive ruptures of the rotator cuff of the shoulder by use of a freeze-dried rotator cuff. J Bone Joint Surg Am. 1978;60(5):681–4.
33. Ito J, Morioka T. Surgical treatment for large and massive tears of the rotator cuff. Int Orthop. 2003;27(4):228–31.
34. Derwin KA, et al. Commercial extracellular matrix scaffolds for rotator cuff tendon repair. Biomechanical, biochemical, and cellular properties. J Bone Joint Surg Am. 2006;88(12):2665–72.
35. Karuppaiah K, Sinha J. Scaffolds in the management of massive rotator cuff tears: current concepts and literature review. EFORT Open Rev. 2019;4(9):557–66.
36. Coons DA, Alan Barber F. Tendon graft substitutes-rotator cuff patches. Sports Med Arthrosc Rev. 2006;14(3):185–90.
37. Barber FA, et al. A prospective, randomized evaluation of acellular human dermal matrix augmentation for arthroscopic rotator cuff repair. Arthroscopy. 2012;28(1):8–15.
38. Bond JL, et al. Arthroscopic replacement of massive, irreparable rotator cuff tears using a GraftJacket allograft: technique and preliminary results. Arthroscopy. 2008;24(4):403–9. e1
39. Gupta AK, et al. Dermal tissue allograft for the repair of massive irreparable rotator cuff tears. Am J Sports Med. 2012;40(1):141–7.
40. Pandey R, et al. Outcome of partial repair of massive rotator cuff tears with and without human tissue allograft bridging repair. Shoulder Elbow. 2017;9(1):23–30.
41. Gupta AK, et al. Arthroscopic distal tibial allograft augmentation for posterior shoulder instability with glenoid bone loss. Arthrosc Tech. 2013;2(4):e405–11.
42. Rotini R, et al. Human dermal matrix scaffold augmentation for large and massive rotator cuff repairs: preliminary clinical and MRI results at 1-year follow-up. Musculoskelet Surg. 2011;95(Suppl 1):S13–23.
43. Wong I, Burns J, Snyder S. Arthroscopic GraftJacket repair of rotator cuff tears. J Shoulder Elbow Surg. 2010;19(2 Suppl):104–9.
44. Bailey JR, et al. Rotator cuff matrix augmentation and interposition: a systematic review and meta-analysis. Am J Sports Med. 2019;47(6):1496–506.
45. Steinhaus ME, et al. Outcomes after patch use in rotator cuff repair. Arthroscopy. 2016;32(8):1676–90.
46. Kluger R, et al. Long-term survivorship of rotator cuff repairs using ultrasound and magnetic resonance imaging analysis. Am J Sports Med. 2011;39(10):2071–81.

47. Nicholson GP, et al. Evaluation of a cross-linked acellular porcine dermal patch for rotator cuff repair augmentation in an ovine model. J Shoulder Elbow Surg. 2007;16(5 Suppl):S184–90.
48. Malcarney HL, Bonar F, Murrell GA. Early inflammatory reaction after rotator cuff repair with a porcine small intestine submucosal implant: a report of 4 cases. Am J Sports Med. 2005;33(6):907–11.
49. Avanzi P, et al. Prospective randomized controlled trial for patch augmentation in rotator cuff repair: 24-month outcomes. J Shoulder Elbow Surg. 2019;28(10):1918–27.
50. Maillot C, et al. Surgical repair of large-to-massive rotator cuff tears seems to be a better option than patch augmentation or debridement and biceps tenotomy: a prospective comparative study. J Shoulder Elbow Surg. 2018;27(9):1545–52.
51. Shepherd HM, Lam PH, Murrell GA. Synthetic patch rotator cuff repair: a 10-year follow-up. Shoulder Elbow. 2014;6(1):35–9.
52. Smolen D, et al. Application of a new polyester patch in arthroscopic massive rotator cuff repair-a prospective cohort study. J Shoulder Elbow Surg. 2019;29(1):e11–21.
53. Cole BJ, et al. Biocompatibility of a polymer patch for rotator cuff repair. Knee Surg Sports Traumatol Arthrosc. 2007;15(5):632–7.
54. Encalada-Diaz I, et al. Rotator cuff repair augmentation using a novel polycarbonate polyurethane patch: preliminary results at 12 months' follow-up. J Shoulder Elbow Surg. 2011;20(5):788–94.
55. Nada AN, et al. Treatment of massive rotator-cuff tears with a polyester ligament (Dacron) augmentation: clinical outcome. J Bone Joint Surg Br. 2010;92(10):1397–402.
56. Ciampi P, et al. The benefit of synthetic versus biological patch augmentation in the repair of posterosuperior massive rotator cuff tears: a 3-year follow-up study. Am J Sports Med. 2014;42(5):1169–75.
57. Chen J, et al. Scaffolds for tendon and ligament repair: review of the efficacy of commercial products. Expert Rev Med Devices. 2009;6(1):61–73.
58. Henry P, et al. Arthroscopic repair for chronic massive rotator cuff tears: a systematic review. Arthroscopy. 2015;31(12):2472–80.
59. Shimokobe H, et al. Risk factors for retear of large/massive rotator cuff tears after arthroscopic surgery: an analysis of tearing patterns. J Orthop Surg Res. 2017;12(1):140.
60. Ferguson DP, et al. Graft utilization in the augmentation of large-to-massive rotator cuff repairs: a systematic review. Am J Sports Med. 2016;44(11):2984–92.
61. Gilot GJ, et al. Outcome of large to massive rotator cuff tears repaired with and without extracellular matrix augmentation: a prospective comparative study. Arthroscopy. 2015;31(8):1459–65.
62. Mori D, Funakoshi N, Yamashita F. Arthroscopic surgery of irreparable large or massive rotator cuff tears with low-grade fatty degeneration of the infraspinatus: patch autograft procedure versus partial repair procedure. Arthroscopy. 2013;29(12):1911–21.
63. Audenaert E, et al. Reconstruction of massive rotator cuff lesions with a synthetic interposition graft: a prospective study of 41 patients. Knee Surg Sports Traumatol Arthrosc. 2006;14(4):360–4.
64. Badhe SP, et al. An assessment of porcine dermal xenograft as an augmentation graft in the treatment of extensive rotator cuff tears. J Shoulder Elbow Surg. 2008;17(1 Suppl):35S–9S.
65. Giannotti S, et al. Study of the porcine dermal collagen repair patch in morpho-functional recovery of the rotator cuff after minimum follow-up of 2.5 years. Surg Technol Int. 2014;24:348–52.
66. Vitali M, et al. Employment of synthetic patch with augmentation of the long head of the biceps tendon in irreparable lesions of the rotator cuff: our technique applied to 60 patients. Tech Hand Up Extrem Surg. 2015;19(1):32–9.

Superior Capsule Reconstruction

22

Lukas N. Muench, Bastian Scheiderer, and Andreas B. Imhoff

22.1 Introduction

The treatment of irreparable massive rotator cuff tears remains a challenge, particularly in young, active patients without glenohumeral osteoarthritis [1, 2]. Numerous surgical management options exist without clear evidence-based guidelines, including debridement with long head of the biceps tenotomy or tenodesis, partial repair, tendon transfer, subacromial balloon spacer, or reverse total shoulder arthroplasty (rTSA) [1, 3]. Patients may develop pain from subacromial impingement, limitation of active shoulder range of motion (ROM), and muscle weakness. This is due to abnormal superior humeral head translation and consequential narrowing of the subacromial space [4–8]. To elevate the arm in patients with tears of the rotator cuff, greater forces are required by both the deltoid and the intact muscle-tendon units of the rotator cuff [9, 10].

In 2013, superior capsule reconstruction (SCR) with fascia lata autograft was proposed as a joint-preserving treatment for irreparable posterosuperior rotator cuff tears [11]. This technique was utilized for patients with a low-grade cuff tear arthropathy and could even be used in cases of severe elevation dysfunction (pseudoparalysis) [2, 12, 13].

SCR is an arthroscopic surgical procedure to reverse superior humeral head migration and restore the muscle balance of the force couples in a cuff-deficient shoulder, without requiring repair of the torn supraspinatus tendons [1, 2, 11, 12]. SCR can either be performed with a fascia lata autograft, acellular dermal allograft or xenograft, or the long head of the biceps tendon [1, 11, 14, 15]. In order to reconstruct the superior capsule of the glenohumeral joint, the graft is attached medially to the superior glenoid and laterally to the tendon footprint at the greater tuberosity [16].

22.2 Relevant Anatomy

Dynamic stabilization of the glenohumeral joint is primarily ensured by a synergism of the deltoid and rotator cuff muscles, which are responsible for maintenance of the force couples to maintain humeral head-centered motion [2, 4]. The superior capsule is an important static stabilizer to prevent superior translation of the humeral head [2]. As a thin, fibrous structure, it spans across the glenohumeral joint space between the greater tuberosity and the superior glenoid, being continuous with

L. N. Muench
Department of Orthopaedic Sports Medicine, Technical University of Munich, Munich, Germany

Department of Orthopaedic Surgery, University of Connecticut, Farmington, CT, USA

B. Scheiderer · A. B. Imhoff (✉)
Department of Orthopaedic Sports Medicine, Technical University of Munich, Munich, Germany
e-mail: bastian.scheiderer@tum.de; imhoff@tum.de

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_22

the rotator cable and overlaid by the supraspinatus and infraspinatus tendons [2, 16, 17]. At the lateral insertion, the superior capsule occupies 30–61% of the greater tuberosity and may have a larger humeral footprint than the supraspinatus [2].

Current literature has described various measures of thickness of the superior capsule, ranging from 0.4 mm up to 9.1 mm at the attachment of the greater tuberosity [2, 18]. Moreover, the superior capsule seems to be an inconsistent structure, constituted by a confluence of various ligaments and being continuous in only 27% of the cases [19].

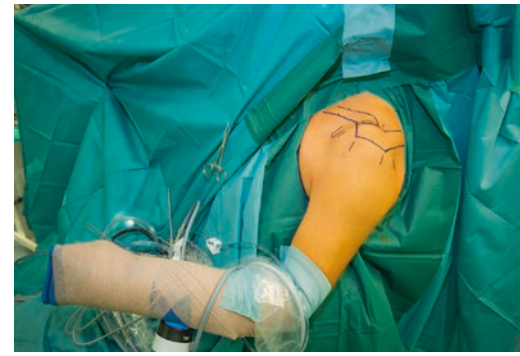


Fig. 22.1 Patient in beach-chair position with holding device after draping and drawing of anatomical landmarks and arthroscopic portals

22.3 Indications and Contraindications

Indications for SCR include symptomatic irreparable massive tears of the posterosuperior rotator cuff in active patients with cuff arthropathy (Hamada grades 1–3) and full function of the deltoid muscle [2, 20].

Relative contraindications for SCR are severe arthropathy with glenohumeral osteoarthritis (Hamada grades 4 and 5), irreparable concomitant subscapularis and/or teres minor tears, and deltoid dysfunction [20]. Moreover, cervical/brachial/axillary nerve palsy or ongoing infections are contraindications [20]. It has been proposed that patients with complete loss of active elevation (pseudoparalysis) or anterior-superior escape of the humeral head may not regain motion with SCR and should be indicated for rTSA [21].

22.4 Arthroscopic Superior Capsule Reconstruction Using a Porcine Acellular Dermal Patch

The surgery is performed under general anesthesia with an additional interscalene nerve catheter. The patient is positioned in the beach-chair position with the operative arm placed on a holder device. Examination of the shoulder is performed, followed by standard preparation and draping (Fig. 22.1).

A diagnostic arthroscopy of the glenohumeral joint is performed through a standard posterior viewing portal. Attention should be directed to the subscapularis and its tendon integrity. Next, an anterior working portal is created through the rotator interval, and a cannula is inserted to facilitate instrumentation. For synovectomy and adhesiolysis, a radiofrequency cautery device is used. The long head of the biceps is then tenotomized or tenodesed according to the surgeon's preference.

An anterolateral and posterolateral portal is created, followed by an extensive subacromial bursectomy and decompression allowing for visualization of the rotator cuff tear. The torn rotator cuff tendons should be evaluated for tissue quality and mobility. If the tear is categorized as irreparable, an SCR is performed using a porcine acellular dermal patch.

The greater tuberosity and the superior glenoid are prepared using an arthroscopic shaver to enhance graft-to-bone healing. A Neviaser portal is established 1 cm medial to the medial border of the acromion, followed by insertion of a cannula for drilling of the first glenoid anchor (Fig. 22.2). The size of the defect in the sagittal and coronal planes is measured in 45° of abduction to determine the patch size used for the reconstruction. The first all-suture anchor (FiberTak; Arthrex, Naples, FL, USA) is inserted at the 12 o'clock position of the glenoid, just medial to the labrum, and the suture limbs are shuttled out through the anterolateral portal.

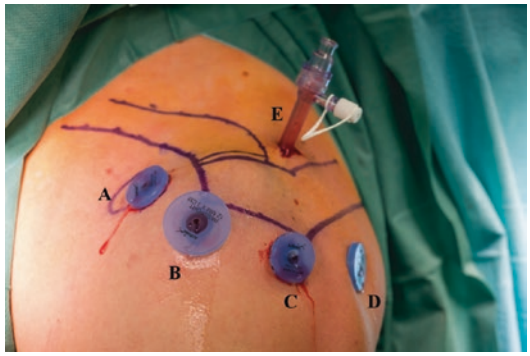


Fig. 22.2 Arthroscopic portals (*A* anterior, *B* anterolateral, *C* posterolateral, *D* posterior, *E* Neviaser) in relation to the anatomical landmarks

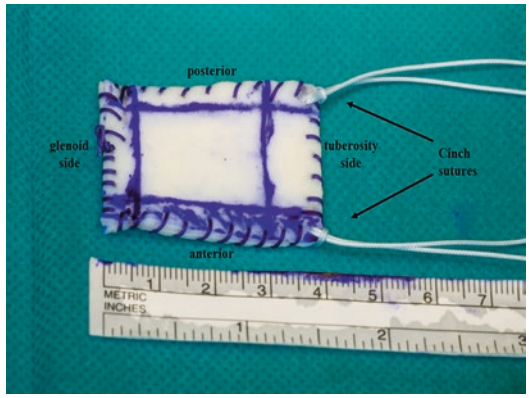


Fig. 22.3 Porcine acellular dermal patch with marked rims and additional cinch sutures as it would be prepared for a reconstruction in a left shoulder

A 3-mm-thick porcine acellular dermal patch (DX reinforcement matrix; Arthrex) is prepared on the side table according to the arthroscopic measurements of the defect. Graft size is extended 10 mm on the tuberosity side and 5 mm on the anterior, posterior, and glenoid side. Two congruent grafts (1.5 mm thick) of the same size are required. An absorbable suture is used to suture the two grafts congruently together. Additional cinch sutures (FiberLink; Arthrex) are then placed through the anterolateral and posterolateral site of the patch, allowing for better control, spanning, and tensioning of the graft (Fig. 22.3).

Ex vivo, suture limbs of the first glenoid anchor are shuttled through the midportion of the medial side of the patch using a suture passing instrument. While visualizing over the posterior portal, the patch is then introduced into the shoulder with an arthroscopic knot pusher through the anterolateral portal (Fig. 22.4). With the cinch sutures, the graft is unfolded and spanned by retrieving the suture limbs anterolaterally and posterolaterally through percutaneous portals.

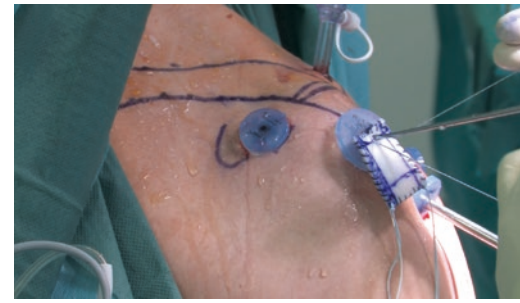


Fig. 22.4 The patch is introduced into the shoulder with an arthroscopic knot pusher through the anterolateral portal

The suture limbs of the 12 o'clock anchor are then tied to secure the graft to the superior glenoid. Using the anterior and posterior portals, two additional all-suture anchors (FiberTak) are inserted at the 2 o'clock and 10 o'clock glenoid position just medial to the labrum. The glenoid fixation is completed by passing the suture limbs through the corresponding anteromedial and posteromedial site of the graft and tying them (Fig. 22.5).

Next, the lateral fixation at the greater tuberosity is performed using a knotless double-row technique. An anchor (4.75-mm BioComposite SwiveLock; Arthrex) loaded with FiberTape is then inserted at the anteromedial portion of the humeral footprint, followed by a posteromedial anchor. The preloaded FiberTapes from these anchors are retrieved and passed through the marked lateral rim of the patch using the suture passer.

Subsequently, the anterior anchor of the lateral row is placed, connecting one FiberTape limb of each of the anteromedial and posteromedial anchors. The humeral fixation is completed by placing the posterolateral anchor with the

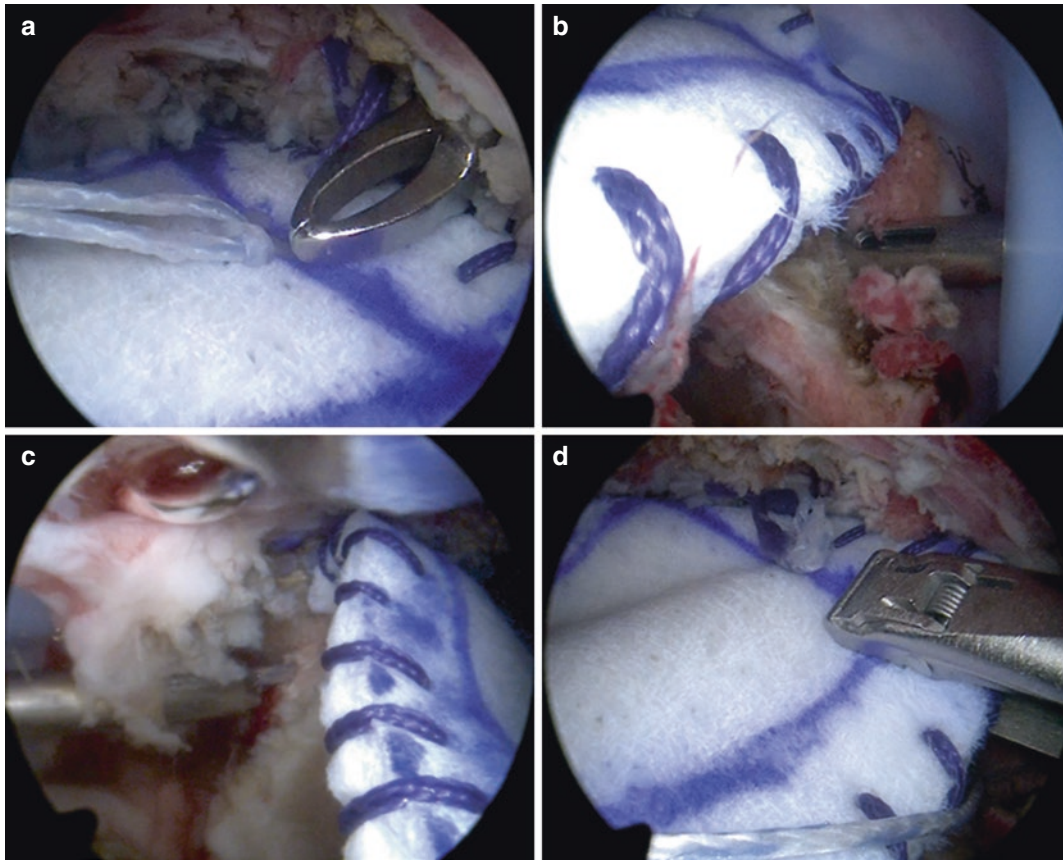


Fig. 22.5 Securing the graft to the superior glenoid. (**a**) The suture strands of the first glenoid anchor placed at 12 o'clock position are tied via the Neviaser portal. (**b**) The second glenoid anchor is inserted at the 2 o'clock position and (**c**) the third anchor at the 10 o'clock position. (**d**) Representatively, the suture limbs are passed through the posteromedial site of the patch

remaining FiberTape limbs of the anteromedial and posteromedial anchors. The teres minor or the remaining infraspinatus is then connected to the graft using two side-to-side sutures. The final reconstruction is visualized through the anterolateral and posterolateral portals and evaluated for integrity and stability of the glenoidal and humeral fixation (Fig. 22.6). Finally, the arthroscopic portals are closed.

22.4.1 Pearls and Pitfalls

It is highly recommended to measure the size of the torn rotator cuff tendon accurately prior to graft preparation and defect reconstruction. The edges of the patch should be marked with a sterile pen, leaving enough rim to cover the defect. Following the authors' technique, graft size should be extended 10 mm on the tuberosity side and 5 mm on the anterior, posterior, and glenoid side.

As with most arthroscopic surgery, suture management is essential for functioning graft passage. Additional cinch sutures should be passed through the anterolateral and posterolateral portion of the patch and retrieved anterolaterally and posterolaterally through percutaneous portals. These sutures facilitate introducing the graft into the shoulder and allow for easy unfolding, spanning, and tensioning of the graft.

During fixation of the graft at the superior glenoid, a spinal needle may be used to assess trajectory before the anchor is finally inserted.

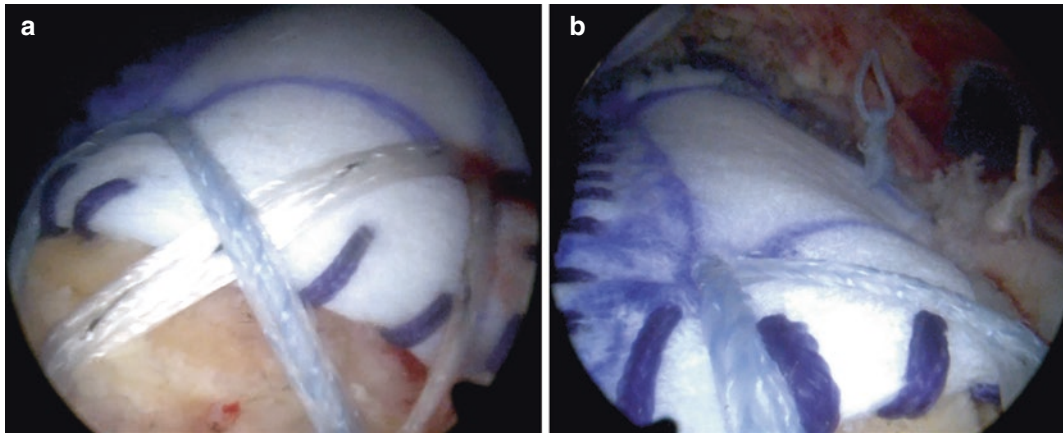


Fig. 22.6 (**a**) Humeral fixation of the patch to the greater tuberosity. (**b**) Visualization of the final reconstruction

Moreover, the glenoid may vary in terms of bone quality, thus requiring careful placement and evaluation of pullout resistance.

22.4.2 Postoperative Care

The standard rehabilitation protocol is characterized by four phases beginning on postoperative day one. The patient is immobilized in a sling with an abduction pillow for the first 6 weeks (Phase 1), in order to ensure protection of the surgical repair by minimizing stress placed on the graft. As passive glenohumeral ROM should be restricted as much as possible, maintaining mobility of accessory joints (cervical spine, elbow, wrist, and hand) as well as scapular retraction and depression is important.

After 6 weeks, the patient should begin with full restoration of passive and active glenohumeral ROM as well as muscular endurance of the rotator cuff (Phase 2). This phase aims to reestablish a normal scapulohumeral rhythm and to allow for return to light functional activities of daily living. Introduced at 10–12 weeks postoperatively, Phase 3 includes improvement of muscular strength, restoration of functional ROM, and return to functional activities of a higher level. Finally, Phase 4 aims to further advance strength and return to activity by focusing on closed chain, proprioceptive, and plyometric exercises according to the patients' functional deficits.

22.5 Results

Limited articles have reported on clinical and radiological results of SCR [1]. In their original clinical study of 23 patients, Mihata et al. showed an increase in mean active elevation from 84° to 148° and external rotation from 26° to 40° using a fascia lata autograft at a mean follow-up of 34.1 months [11]. The mean ASES score improved significantly from 23.5 to 92.9, with 83.3% of the patients demonstrating an intact graft, without progression of muscle atrophy or osteoarthritis confirmed by MRI [11]. A follow-up study including 92 patients confirmed these convincing outcomes by reporting a mean ASES score of 93.3 and 92% intact grafts during the period of 5–8 years postoperatively [22]. This series also suggested that graft healing may affect clinical outcomes, as subjects who have healed had higher ASES scores and better forward elevation [22].

When using a dermal allograft patch, Denard et al. reported an increase in forward flexion from 130° to 158° and external rotation from 36° to 45° postoperatively, along with a mean improvement in ASES score from 43.6 to 77.5 [23]. Only 45% of the grafts demonstrated complete healing confirmed by MRI after a minimum follow-up of 1 year [23]. However, only in 74.6% of the patients was the postoperative outcome considered a success, with 18.6% of the patients

undergoing revision surgery [23]. In a series of 86 patients, Pennington et al. showed a decrease in pain and recovered shoulder function with a significant improvement of strength and range of motion at a minimum follow-up of 1 year [24]. Besides, their radiographic analysis revealed a consistent decrease in superior capsular distance and increase of the acromiohumeral interval [24].

Mihata et al. demonstrated that even in patients with preoperative pseudoparalysis, SCR could restore superior glenohumeral stability and improve shoulder function as long as no postoperative graft tear was observed [13]. Pseudoparalysis was reversed in 96% of the patients with preoperative moderate pseudoparalysis and in 93% with preoperative severe pseudoparalysis [13]. Additionally, graft healing after arthroscopic SCR was not affected by the presence of pseudoparalysis [13]. Burkhart et al. reported that profound pseudoparalysis was reversed in 90% of cases in patients without concomitant glenohumeral arthritis [12].

22.6 Conclusions

Arthroscopic SCR remains a promising option for patients with massive irreparable rotator cuff tears, who present with low-grade osteoarthritis. SCR appears to be a viable, non-prosthetic alternative to restore superior glenohumeral stability and shoulder function, even in patients with severe pseudoparalysis [2, 12, 13].

Careful patient selection and accurate surgical technique are essential to achieve satisfying clinical outcomes. However, long-term data, from multiple authors and with the use of different graft types, are still pending. True randomized trials comparing debridement with biceps treatment, partial repair, and SCR are needed but will be hard to execute. As well, SCR does not seem to burn any bridges to potential future revision procedures, such as conversion to rTSA [2].

References

1. Ladermann A, Collin P, Athwal GS, Scheibel M, Zumstein MA, Nourissat G. Current concepts in the primary management of irreparable posterosuperior rotator cuff tears without arthritis. EFORT Open Rev. 2018;3(5):200–9.
2. Sethi P, Franco WG. The role of superior capsule reconstruction in rotator cuff tears. Orthop Clin North Am. 2018;49(1):93–101.
3. Greenspoon JA, Petri M, Warth RJ, Millett PJ. Massive rotator cuff tears: pathomechanics, current treatment options, and clinical outcomes. J Shoulder Elb Surg. 2015;24(9):1493–505.
4. Burkhart SS. Fluoroscopic comparison of kinematic patterns in massive rotator cuff tears. A suspension bridge model. Clin Orthop Relat Res. 1992;284:144–52.
5. Ishihara Y, Mihata T, Tamboli M, et al. Role of the superior shoulder capsule in passive stability of the glenohumeral joint. J Shoulder Elb Surg. 2014;23(5):642–8.
6. Nove-Josserand L, Edwards TB, O'Connor DP, Walch G. The acromiohumeral and coracohumeral intervals are abnormal in rotator cuff tears with muscular fatty degeneration. Clin Orthop Relat Res. 2005;433:90–6.
7. Paletta GA, Warner JJ, Warren RF, Deutsch A, Altchek DW. Shoulder kinematics with two-plane x-ray evaluation in patients with anterior instability or rotator cuff tearing. J Shoulder Elbow Surg. 1997;6(6):516–27.
8. Thompson WO, Debski RE, Boardman N, et al. A biomechanical analysis of rotator cuff deficiency in a cadaveric model. Am J Sports Med. 1996;24(3):286–92.
9. Dyrna F, Kumar NS, Obopilwe E, et al. Relationship between deltoid and rotator cuff muscles during dynamic shoulder abduction: a biomechanical study of rotator cuff tear progression. Am J Sports Med. 2018;46(8):1919–26.
10. Hansen ML, Otis JC, Johnson JS, Cordasco FA, Craig EV, Warren RF. Biomechanics of massive rotator cuff tears: implications for treatment. J Bone Joint Surg Am. 2008;90(2):316–25.
11. Mihata T, Lee TQ, Watanabe C, et al. Clinical results of arthroscopic superior capsule reconstruction for irreparable rotator cuff tears. Arthroscopy. 2013;29(3):459–70.
12. Burkhart SS, Hartzler RU. Superior capsular reconstruction reverses profound pseudoparalysis in patients with irreparable rotator cuff tears and minimal or no glenohumeral arthritis. Arthroscopy. 2018;35(1):22–8.

13. Mihata T, Lee TQ, Hasegawa A, et al. Arthroscopic superior capsule reconstruction can eliminate pseudoparalysis in patients with irreparable rotator cuff tears. Am J Sports Med. 2018;46(11):2707–16.
14. Boutsiadis A, Chen S, Jiang C, Lenoir H, Delsol P, Barth J. Long head of the biceps as a suitable available local tissue autograft for superior capsular reconstruction: "the Chinese way". Arthrosc Tech. 2017;6(5):e1559–66.
15. Burkhart SS, Denard PJ, Adams CR, Brady PC, Hartzler RU. Arthroscopic superior capsular reconstruction for massive irreparable rotator cuff repair. Arthrosc Tech. 2016;5(6):e1407–18.
16. Mihata T, McGarry MH, Kahn T, Goldberg I, Neo M, Lee TQ. Biomechanical effect of thickness and tension of fascia lata graft on glenohumeral stability for superior capsule reconstruction in irreparable supraspinatus tears. Arthroscopy. 2016;32(3):418–26.
17. Adams CR, DeMartino AM, Rego G, Denard PJ, Burkhart SS. The rotator cuff and the superior capsule: why we need both. Arthroscopy. 2016;32(12):2628–37.
18. Nimura A, Kato A, Yamaguchi K, et al. The superior capsule of the shoulder joint complements the insertion of the rotator cuff. J Shoulder Elbow Surg. 2012;21(7):867–72.
19. Pouliart N, Somers K, Eid S, Gagey O. Variations in the superior capsuloligamentous complex and description of a new ligament. J Shoulder Elbow Surg. 2007;16(6):821–36.
20. Altintas B, Higgins B, Anderson N, Millett PJ. Superior capsule reconstruction for the treatment of irreparable rotator cuff tears. Oper Tech Orthop. 2018;28(4):226–31.
21. Tokish JM, Alexander TC, Kissenberth MJ, Hawkins RJ. Pseudoparalysis: a systematic review of term definitions, treatment approaches, and outcomes of management techniques. J Shoulder Elbow Surg. 2017;26(6):e177–87.
22. Mihata T, Lee TQ, Itami Y, Hasegawa A, Ohue M, Neo M. Arthroscopic superior capsule reconstruction for irreparable rotator cuff tears. Orthop J Sports Med. 2016;4(3_Suppl3).
23. Denard PJ, Brady PC, Adams CR, Tokish JM, Burkhart SS. Preliminary results of arthroscopic superior capsule reconstruction with dermal allograft. Arthroscopy. 2018;34(1):93–9.
24. Pennington WT, Bartz BA, Pauli JM, Walker CE, Schmidt W. Arthroscopic superior capsular reconstruction with acellular dermal allograft for the treatment of massive irreparable rotator cuff tears: short-term clinical outcomes and the radiographic parameter of superior capsular distance. Arthroscopy. 2018;34(6):1764–73.

23 Posterosuperior Massive Irreparable Rotator Cuff Tears: The Biceps Autograft

Achilleas Boutsiadis, John Swan, and Johannes Barth

23.1 Introduction

The treatment of massive rotator cuff tears remains challenging, since a number of irreversible factors like tendon retraction, muscle atrophy, fatty degeneration, and even ultrastructural changes of the muscle architecture predispose to high failure rates after direct repair [1–3]. Therefore, several non-arthroplasty therapeutic options with joint preservation have been proposed for the management of symptomatic massive rotator tears. These could be simple biceps tenotomy in older patients [4], partial repair in order to restore shoulder couple forces [5], interposition or augmentation patches [6, 7], several tendon transfers [8], and the implantation of a balloon-shaped biodegradable spacer in the subacromial space [9]. The international literature has not proven the superiority of any of these techniques, and in some cases, it has shown mixed results including considerable risk of complications [10].

In 2013, Mihata et al. proposed that patients with massive irreparable rotator cuff tears have a loss of superior glenohumeral stability due to a defect of the superior capsule. Therefore, the authors described an arthroscopic superior capsular reconstruction (ASCR) using a fascia lata autograft. They reported the technique to be reliable and successful in restoring superior glenohumeral stability and showed a postoperative increase in the acromiohumeral distance with promising clinical results [11]. Since then, in order to simplify the technique and eliminate donor site morbidity, several authors described alternative ASCR using different autografts or acellular dermal allograft [12–15].

Based on the principles of superior capsular reconstruction, a modified technique using the long head biceps tendon autograft (LHBT) has been described. In this technique, the tendon's insertion on the glenoid is left intact, while it is tenotomized laterally and fixed with a suture anchor onto the humeral greater tuberosity, thus preventing possible superior head migration [16]. This chapter describes the current indications, the surgical technique, and the tips and tricks in performing ASCR using the long head of biceps tendon autograft.

23.2 Indications and Contraindications

The possible indications for ASCR with the LHBT autograft are massive posterosuperior rotator cuff tears, with stage 2 cuff tear arthropathy (Hamada classification) and stage 3

A. Boutsiadis
Department of Orthopedic Surgery, 401 Military Hospital of Athens, Athens, Greece

J. Swan · J. Barth (✉)
Department of Orthopaedic Surgery, Centre Osteoarticulaire des Cèdres, Grenoble, France

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_23

supraspinatus tendon retraction (Patte classification). The muscle fatty infiltration of the supraspinatus may be grade 3 or 4 (Goutallier classification). However, the infraspinatus and the possible subscapularis tears should be reparable, and their fatty infiltration should be grade ≤3. All possible indications and contraindications are presented in Table 23.1.

23.3 Surgical Technique

23.3.1 Patient Positioning, Initial Evaluation, and Subscapularis Repair

The patient is routinely placed in the beach-chair position under general anesthesia with an interscalene nerve block performed prior to commencement of surgery. The osseous landmarks of the acromion, the clavicle, and the coracoid are identified and marked on the skin prior to arthroscopy. Four to six arthroscopic portals are created as required: posterior, posterolateral, lateral, anterolateral, anterior, and Neviaser portals. Using a 30° arthroscope placed in the standard posterior viewing portal, an intra-articular evaluation of the joint is first performed. After initial assessment confirms a massive rotator cuff tear and the presence of a LHBT of good tissue, the anterior and anterolateral portals are established with the aid of a spinal needle. We should mention that minimal fraying or degeneration of the LHBT is not a contraindication. The anterolateral portal is placed at the level of the lateral projection of the clavicle, about 2 cm below the level of the lateral acromion, through the rotator cuff tear, just above the greater tuberosity.

In cases where a reparable subscapularis lesion exists, this is initially repaired in order to restore the important stabilizing force couple in the axial plane. According to the authors' preferences, the "inside the box" repair technique is performed while also preserving the superior glenohumeral/coracohumeral ligament complex ("comma sign") [17]. This technique is performed with the arthroscope positioned intra-articularly in the posterior portal while any adhesions of the subscapularis tendon are released, and the lesser tuberosity is prepared. Depending on the size of the lesion, the subscapularis tendon is repaired onto the lesser tuberosity by using a single- or double-row repair. In all cases, the LHBT is preserved and care is made not to damage it throughout the subscapularis repair.

Table 23.1 Possible indications and contraindications of ASCR with the long head biceps autograft

Indications
Massive posterosuperior rotator cuff tears
Mild cuff tear arthropathy (Hamada stages 1–3)
Stage 3 (Patte classification) supraspinatus tendon retraction
Supraspinatus muscle fatty infiltration grade 3 or 4 (Goutallier classification)
Full function of the deltoid muscle
Partially or fully reparable coexisting infraspinatus and/or subscapularis tendon tears
Intra-articular presence of a LHBT even with partial fraying or degeneration
Repairable (complete or partially) tears of the infraspinatus and/or subscapularis
Fatty infiltration of the infraspinatus and/or subscapularis ≤3 (Goutallier classification)
Contraindications
Severe arthropathy with glenohumeral osteoarthritis (Hamada stage 4 or 5)
Irreparable subscapularis or teres minor lesions
Brachial plexus or axillary nerve pathology
Infection
Excessive preoperative stiffness
Revision cases with the absence of the LHBT

23.3.2 Superior Capsular Reconstruction with the Long Head of the Biceps

The arthroscope is introduced into the subacromial space, and an additional posterolateral portal is established. With the aid of a motorized shaver and a radio-frequency electrocautery device (Super TurboVac 90; ArthroCare, Austin, TX, USA), extensive bursectomy and release of tendon adhesions is performed. It is of great importance to recognize the limits of the supraspinatus and infraspinatus tendons by identifying the spine of the scapula in the subacromial space.

Tendons' quality, stiffness, and degree of retraction are evaluated, and their irreparability is confirmed (Fig. 23.1). Thereafter, an acromioplasty is performed with a 5.5-mm motorized burr in order to remove any anterior or lateral acromial osteophytes. However, the coracoacromial ligament is preserved, in order to protect the humeral head from anterosuperior migration. Additionally, the authors' preferred method includes preoperative measurement of the critical shoulder angle, and in cases >35° a lateral acromioplasty is performed. The upper surface of the greater tuberosity is also prepared using the burr in order to create a bleeding cancellous bone bed to enhance tendon-to-bone healing.

The 30° arthroscope is kept in the posterolateral portal, and a flexible PassPort cannula (Arthrex, Naples, FL, USA) is placed through the anterolateral portal in order to facilitate suture management. A 5.5-mm triple-loaded suture anchor (Bio-Corkscrew FT; Arthrex) is inserted at the midpoint of the humeral greater tuberosity. The arm is placed in both 40° of flexion and abduction. Thereafter, with the aid of a needle suture passer, all three sutures are passed individually through the intact LHBT using a "lasso-loop" configuration (Fig. 23.2). In order to obtain enough tension of the tendon, the first lasso-loop suture should be placed at the middle distance between the glenoid and the greater tuberosity. Using a radio-frequency cautery device, the LHBT is dissected and tenotomized in the middle of the bicipital groove. The distal part of the tendon is left free without any additional tenodesis. The proximal part of the tendon is transferred and securely fixed onto the footprint of the supraspinatus tendon by tying the already passed sutures. The sutures are not cut and retrieved from the

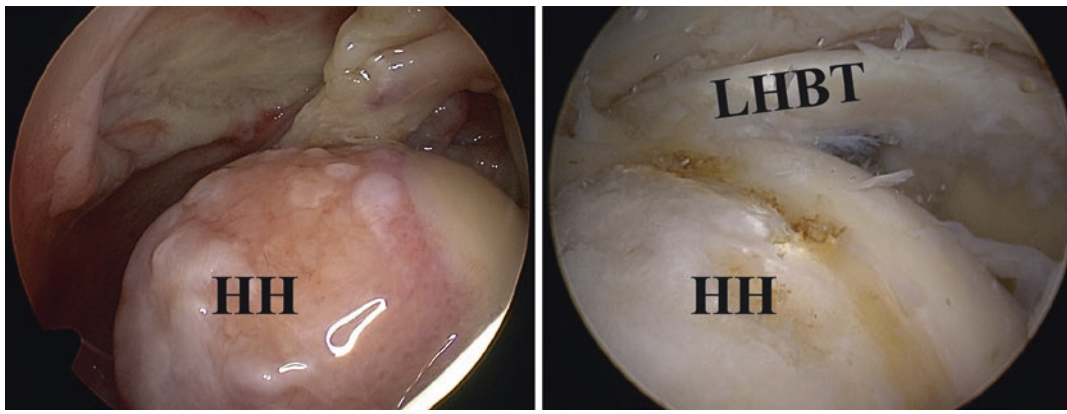


Fig. 23.1 Figure showing a massive rotator cuff tear with the long head of the biceps available for superior capsular reconstruction

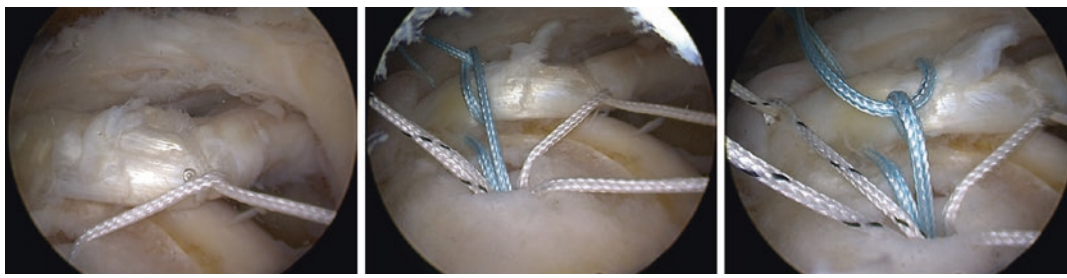


Fig. 23.2 The three sutures of the central anchor are passed sequentially through the intact LHBT using a "lasso-loop" configuration

anterior working portal. Therefore, the biceps tendon, which is natively fixed on the glenoid, acts as an autograft for the superior capsular reconstruction and as an interposition graft for the fixation of the supraspinatus (Fig. 23.3).

Depending on the degeneration and retraction of the remaining infraspinatus tendon posteriorly, a "tension-free" complete or partial repair is attempted. For the infraspinatus fixation, several fixation techniques can be utilized, depending on tendon excursion and quality. These include a classic double-row technique with two additional triple-loaded suture anchors, or a transosseous-equivalent configuration technique with four additional posterior suture anchors, or, finally, a simple lateral row technique with a combination of a suture tape and a knotless anchor (Fig. 23.4).

Once the posterior cuff is fixed and the "length of the infraspinatus tendon is restored," it is possible to perform a side-to-side repair with the LHBT (Fig. 23.5). Additionally, the retracted supraspinatus tendon can be fixed without tension, sometimes in a more significantly medial position, onto the LHBT also. This is done using the sutures that were previously used for the LHBT fixation by sequentially retrieving these from the anterior portal and moving them to the anterolateral portal. Therefore, as mentioned above, the LHBT could act also as an interposition graft for the repair of the supraspinatus tendon. By using the needle punch suture passer (Arthrex), they are passed through the infraspinatus and the retracted supraspinatus providing an additional side-to-side, "tension-free" marginal repair of the rotator cuff integrating the LHBT also. All the tendon defects can be covered, resulting to a "watertight-like" repair. Finally, intra-articular evaluation of the construct is performed (Fig. 23.6).

23.4 Discussion

Despite the numerous techniques and modifications of ASCR described, there is limited data in published outcomes. Mihata et al. reported significant improvement in range of motion (forward flexion from 84° to 148°, external rotation from 26° to 40°) and consequently functional score improvement (ASES = 92.9 points; JOA = 92.6 points; UCLA = 32.4 points). They

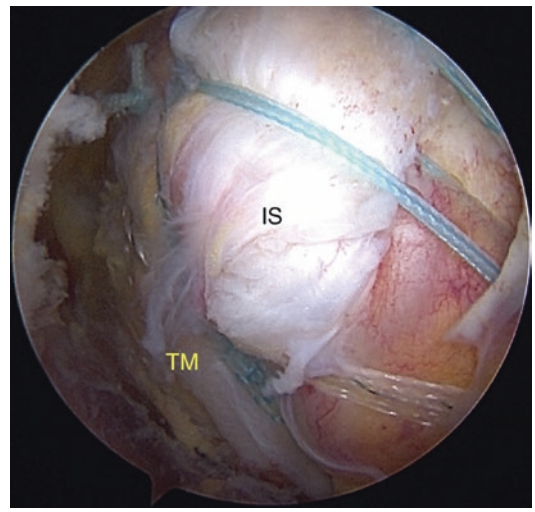


Fig. 23.4 Fixation of the posterior cuff (*IS* infraspinatus, *TM* teres minor) using transosseous equivalent technique

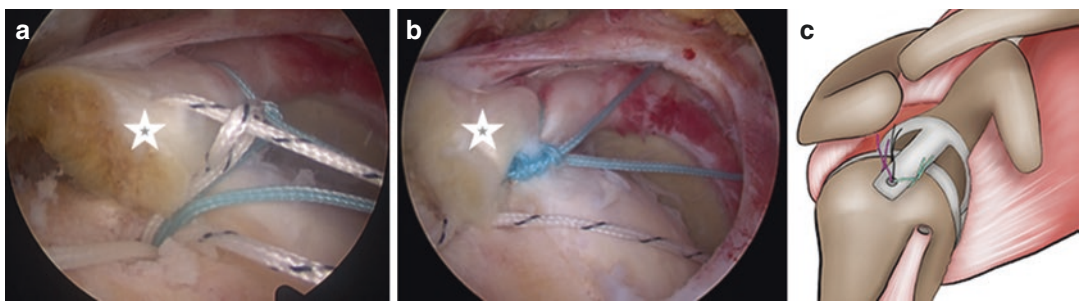


Fig. 23.3 The distal part of the LHBT (asterisk) is cut (**a**), and the proximal part is transferred and secured onto the footprint of the supraspinatus tendon (**b**). Art design showing the fixed LHBT onto the great tuberosity (**c**)

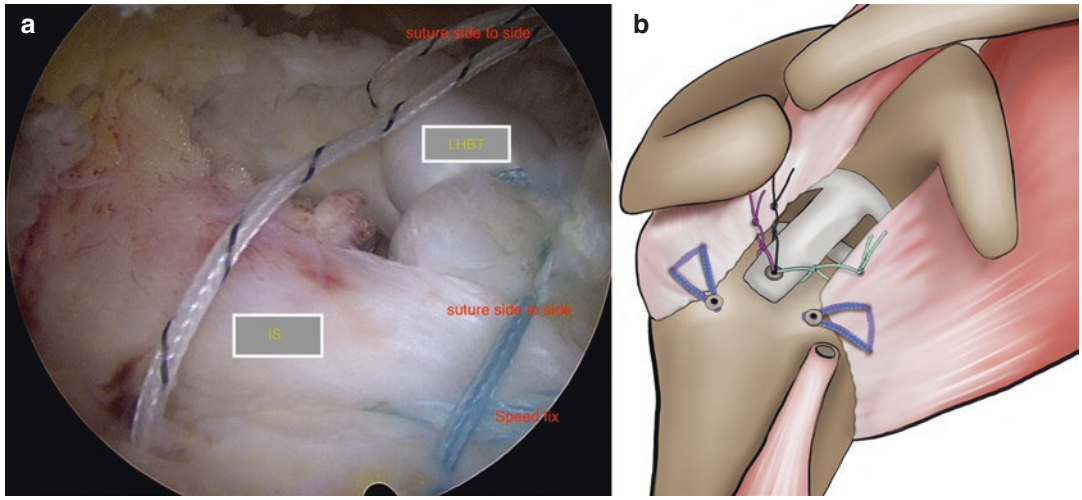


Fig. 23.5 (**a**) Side-to-side repair of the LHBT with the posterosuperior rotator cuff (*IS* infraspinatus). (**b**) Art design showing the final construct after superior capsular reconstruction with the long head biceps tendon

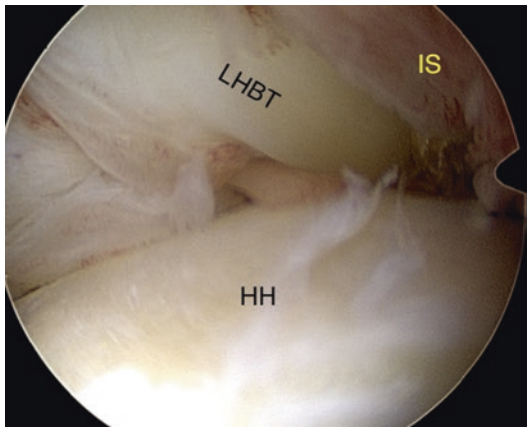


Fig. 23.6 Intra-articular view after superior capsular reconstruction with the long head biceps tendon (LHBT) (*HH* humeral head)

also reported an increase of the acromiohumeral distance from 4.6 ± 2.2 mm preoperatively to 8.7 ± 2.6 mm postoperatively. Finally, they reported a failure rate of 17.7%. Further studies using dermal allograft showed promising clinical outcomes with significant improvement in both clinical and functional outcomes after 2 years follow-up. However, there is a lack of precise radiological evaluation regarding the integrity of the construct at final evaluation [18–20].

Our unpublished data of the superior capsular reconstruction using the LHBT also showed significant clinical improvement and promising results. Theoretically, the use of the LHBT as an autograft for superior capsular reconstruction could potentially increase postoperative pain. However, no increased pain or stiffness was observed.

Interestingly, a recent cadaveric biomechanical study showed that superior capsular reconstruction with the LHBT is a feasible procedure and biomechanically equivalent to or even stronger than superior capsular reconstruction using a fascia lata autograft. This study reported that a LHBT autograft required 393.2% ± 87.9% of the force for superior humeral migration in the massive rotator cuff tear condition, while a fascia lata autograft required only 194.0% ± 21.8% of force for superior humeral head migration [21].

23.5 Conclusions

Superior capsular reconstruction with the LHBT autograft is a reproducible technique that can be performed with the least technical demands and possibly lower overall cost and results in a good final biomechanical construct. However,

prospective studies in large cohort populations with long-term follow-up are necessary to establish the effectiveness of this technique.

References

1. Barth J, Fotiadis E, Barthelemy R, Genna S, Saffarini M. Ultrasonic evaluation of the repair integrity can predict functional outcomes after arthroscopic double-row rotator cuff repair. Knee Surg Sports Traumatol Arthrosc. 2015;23:376–85.
2. Ditsios K, Boutsiadis A, Kapoukranidou D, Chatzisotiriou A, Kalpidis I, Albani M, Christodoulou A. Chronic massive rotator cuff tear in rats: in vivo evaluation of muscle force and three-dimensional histologic analysis. J Shoulder Elbow Surg. 2014;23:1822–30.
3. Kim I-B, Kim M-W. Risk factors for retear after arthroscopic repair of full-thickness rotator cuff tears using the suture bridge technique: classification system. Arthroscopy. 2016;32:2191–200.
4. Walch G, Edwards TB, Boulahia A, Nové-Josserand L, Neyton L, Szabo I. Arthroscopic tenotomy of the long head of the biceps in the treatment of rotator cuff tears: clinical and radiographic results of 307 cases. J Shoulder Elbow Surg. 2005;14:238–46.
5. Galasso O, Riccelli DA, De Gori M, De Benedetto M, Orlando N, Gasparini G, Castricini R. Quality of life and functional results of arthroscopic partial repair of irreparable rotator cuff tears. Arthroscopy. 2017;33(2):261–8.
6. Petri M, Warth RJ, Horan MP, Greenspoon JA, Millett PJ. Outcomes after open revision repair of massive rotator cuff tears with biologic patch augmentation. Arthroscopy. 2016;32:1752–60.
7. Steinhaus ME, Makhni EC, Cole BJ, Romeo AA, Verma NN. Outcomes after patch use in rotator cuff repair. Arthroscopy. 2016;32:1676–90.
8. Valenti P. Joint-preserving treatment options for irreparable rotator cuff tears. Orthopade. 2018;47:103–12.
9. Moon AS, Patel HA, Ithurburn MP, Brabston EW, Ponce BA, Momaya AM. Subacromial spacer implantation for the treatment of massive irreparable rotator cuff tears: a systematic review. Arthroscopy. 2019;35:607–14.
10. Lädermann A, Collin P, Athwal GS, Scheibel M, Zumstein MA, Nourissat G. Current concepts in the primary management of irreparable posterosuperior rotator cuff tears without arthritis. EFORT Open Rev. 2018;3:200–9.
11. Mihata T, Lee TQ, Watanabe C, Fukunishi K, Ohue M, Tsujimura T, Kinoshita M. Clinical results of arthroscopic superior capsule reconstruction for irreparable rotator cuff tears. Arthroscopy. 2013;29:459–70.
12. Laskovski JR, Boyd JA, Peterson EE, Abrams JS. Simplified technique for superior capsular reconstruction using an acellular dermal allograft. Arthrosc Tech. 2018;7:e1089–95.
13. Makovicka JL, Patel KA, Tokish JM. Superior capsular reconstruction with the addition of an acromial acellular dermal allograft spacer. Arthrosc Tech. 2018;7:e1181–90.
14. Petri M, Greenspoon JA, Millett PJ. Arthroscopic superior capsule reconstruction for irreparable rotator cuff tears. Arthrosc Tech. 2015;4:e751–5.
15. Rosales-Varo AP, Zafra M, García-Espona MA, Flores-Ruiz MA, Roda O. Reconstrucción de la cápsula superior en las roturas irreparables del manguito mediante injerto autógeno de isquiotibiales. Revista Española de Cirugía Ortopédica y Traumatología. 2019;63:1–6.
16. Boutsiadis A, Chen S, Jiang C, Lenoir H, Delsol P, Barth J. Long head of the biceps as a suitable available local tissue autograft for superior capsular reconstruction: “the Chinese way”. Arthrosc Tech. 2017;6:e1559–66.
17. Burkhart SS, Brady PC. Arthroscopic subscapularis repair: surgical tips and pearls A to Z. Arthroscopy. 2006;22:1014–27.
18. Denard PJ, Brady PC, Adams CR, Tokish JM, Burkhart SS. Preliminary results of arthroscopic superior capsule reconstruction with dermal allograft. Arthroscopy. 2018;34:93–9.
19. Hirahara AM, Andersen WJ, Panero AJ. Superior capsular reconstruction: clinical outcomes after minimum 2-year follow-up. Am J Orthop. 2017;46:266–78.
20. Pennington WT, Bartz BA, Pauli JM, Walker CE, Schmidt W. Arthroscopic superior capsular reconstruction with acellular dermal allograft for the treatment of massive irreparable rotator cuff tears: short-term clinical outcomes and the radiographic parameter of superior capsular distance. Arthroscopy. 2018;34:1764–73.
21. El-shaar R, Soin S, Nicandri G, Maloney M, Voloshin I. Superior capsular reconstruction with a long head of the biceps tendon autograft: a cadaveric study. Orthop J Sports Med. 2018;6:232596711878536.

24 Tendon Transfer for Posterosuperior Cuff: Latissimus Dorsi Transfer

Frank Martetschläger

24.1 Introduction

Transfer of the tendon of latissimus dorsi muscle (LDT) for the treatment of irreparable posterosuperior tears was introduced by Gerber et al. in 1988 [1]. Adopted from the field of surgery for brachial plexus palsy, this technique was introduced in order to provide containment of the humeral head inside the shoulder joint, to regain control of external rotation, and finally to improve joint kinematics.

In the beginning, two incisions were used for this technique of latissimus dorsi transfer: a dorsal approach for mobilization and harvesting of the latissimus dorsi muscle flap and a transacromial approach for accessing the greater tuberosity and tendon reattachment. Since then, several different techniques have been introduced, and long-term follow-up studies have shown a reliable value of the procedure in a strictly selected patient cohort.

24.2 Indications and Contraindications for Surgery

A latissimus dorsi transfer surgery can be indicated in patients with irreparable posterosuperior rotator cuff tears and combined weakness of abduction and external rotation strength and fatty muscle atrophy greater than stage 2 according to Goutallier. An intact or at least reparable subscapularis tendon is mandatory, and the acromiohumeral distance should measure at least 5–7 mm. Furthermore, the procedure is mainly indicated in a younger and very active highly demanding patient cohort with a high level of postoperative compliance. Contraindications for LDT include an irreparable subscapularis tear, rotator cuff arthropathy greater stage 2 according to Hamada, and an axillary nerve palsy or deltoid muscle deficiency.

24.3 Surgical Planning and Patient Positioning

The patient is placed in lateral decubitus position. When performing a single incision technique, an arthroscopy should be performed in order to examine and treat the long head of the biceps tendon. In case of a double incision technique, the biceps tendon can be evaluated and treated through the deltoid-splitting approach. Surgical

F. Martetschläger (✉)
German Center for Shoulder Surgery, ATOS Clinic, Munich, Germany

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_24

planning includes the choice of the desired surgical technique. According to the present literature, several different single and double incision techniques can be used. In this chapter, one single and one double incision technique are described.

24.4 Surgical Techniques

Several different techniques have been described over the years. The main difference between techniques is whether a single incision technique or double incision technique is performed. For the double incision technique, the LD is mobilized and harvested through a posterior approach, then shuttled to the anterior aspect of the shoulder, and reattached to the greater tuberosity through a separate anterolateral deltoid-splitting approach [1].

As for the single incision technique, the muscle and tendon portion is mobilized through the posterior incision and attached through the same approach to the greater tuberosity [2]. Both techniques are described in the following section.

24.5 The Double Incision Technique

24.5.1 Step 1: Deltoid-Splitting Approach and Footprint Preparation

A deltoid-splitting approach is made at the anterolateral side of the humerus to access and expose the greater tuberosity. The torn and degenerated cuff tendons can also be visualized and debrided via this approach.

24.5.2 Step 2: Harvesting the Latissimus Dorsi Tendon

The second curved incision is made from approximately 3 cm above the axillary fold running along the lateral boarder of the latissimus dorsi. Via blunt dissection, the latissimus dorsi is released from superficial adhesions and separated, and the boarders of latissimus dorsi and teres major are identified, which is facilitated by a fatty demarcation between these two muscles. Next, the two muscles are separated and mobilized. Then the insertion of the tendon on the humerus is identified at the inner border of the bicipital groove. The shoulder is positioned in abduction and maximal internal rotation at this point in order to achieve the maximum length possible. By sharp dissection, the LD tendon is released from the bone. The flat and thin tendon is armed by traction sutures on both sides.

24.5.3 Step 3: Establishment of the Subdeltoid Tunnel and Transfer of the Tendon to the Greater Tuberosity

By blunt dissection, a tunnel is established between the posterior deltoid muscle and the triceps. Care has to be taken in order not to injure the axillary nerve. By the use of a clamp, a shuttle suture can be placed within the created tunnel. Next, by the use of the shuttle suture, the LD tendon can be transferred through the created tunnel to the greater tuberosity site for reattachment.

24.5.4 Step 4: Fixation of the Latissimus Dorsi onto the Greater Tuberosity

Like for any rotator cuff repair, several fixation methods are possible here. The author prefers a suture anchor double-row repair in order to achieve a flat compression of the flat tendon to the footprint on the greater tuberosity. The main steps are shown in Fig. 24.1a–c.

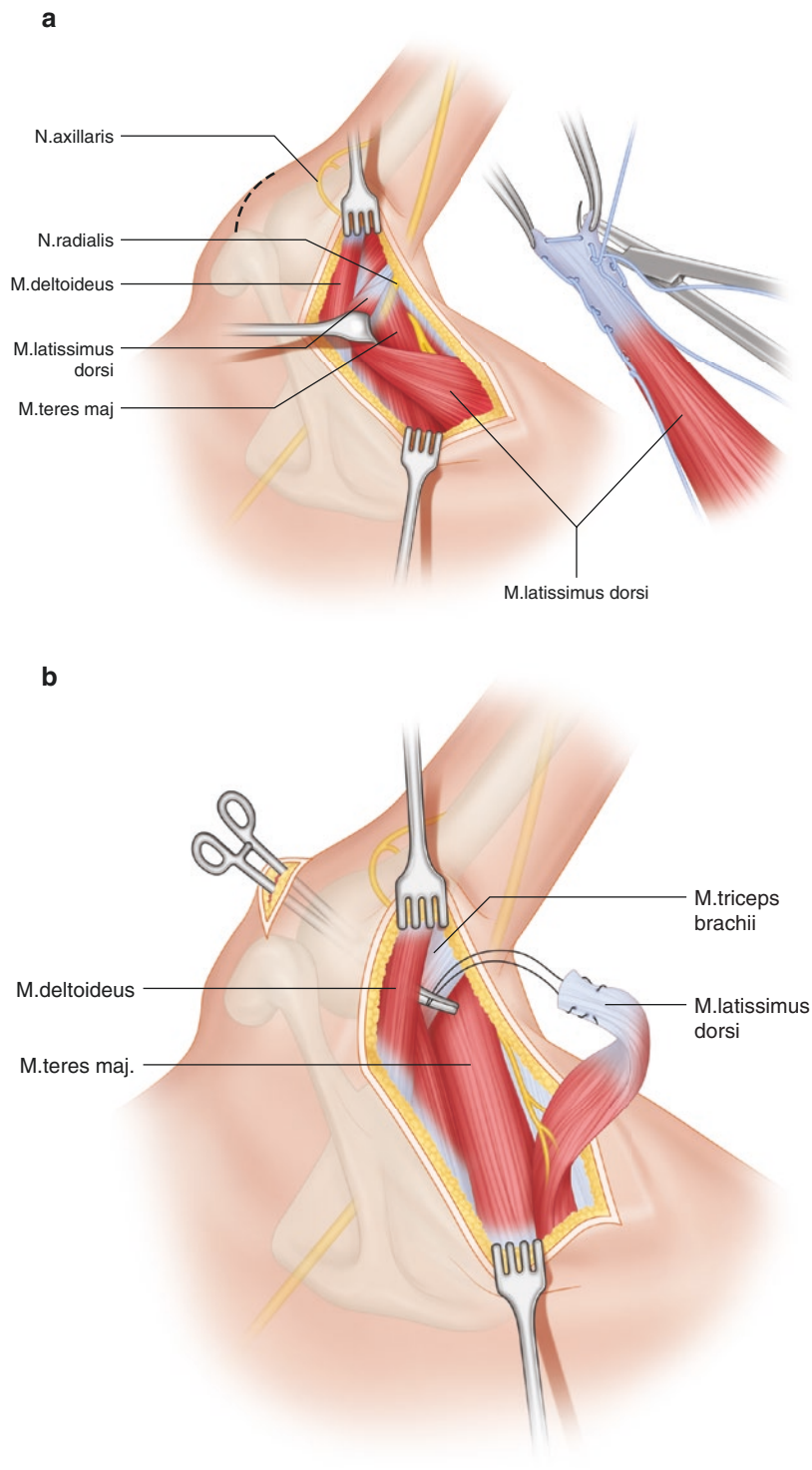
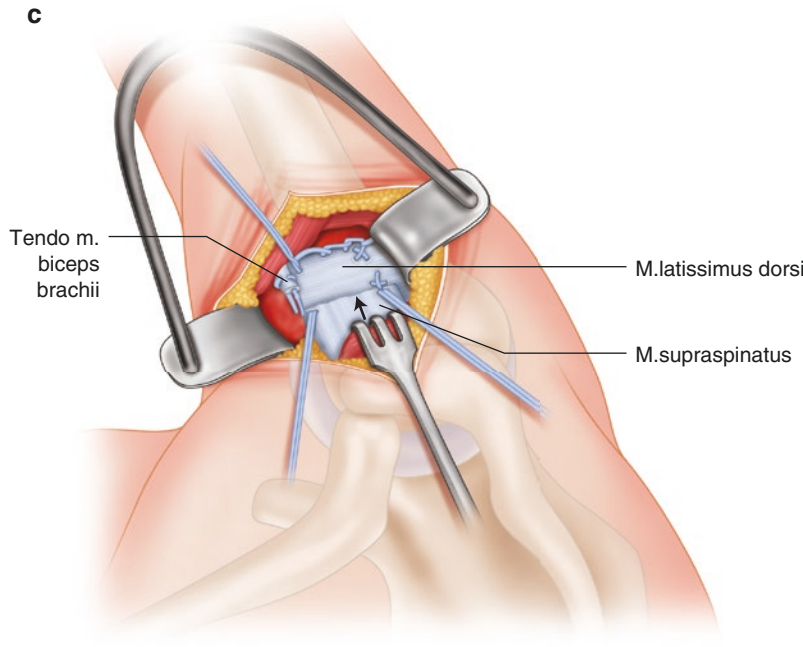


Fig. 24.1 (**a**) Posterior approach with approximity to nerves and surrounding muscles. The suturing of the harvested tendon is also shown. (**b**) Using a clamp, a tunnel is established between the posterior deltoid and triceps. Next, the tendon is shuttled onto the greater tuberosity site. (**c**) Final result after reattachment of the tendon onto the greater tuberosity through a deltoid-splitting approach

Fig. 24.1 (continued)



24.6 The Single Incision Technique According to Habermeyer

24.6.1 Step 1: Surgical Approach

An incision is made from the posterior portal to the axillary pouch (Fig. 24.2). Latissimus dorsi and teres major are then identified and mobilized.

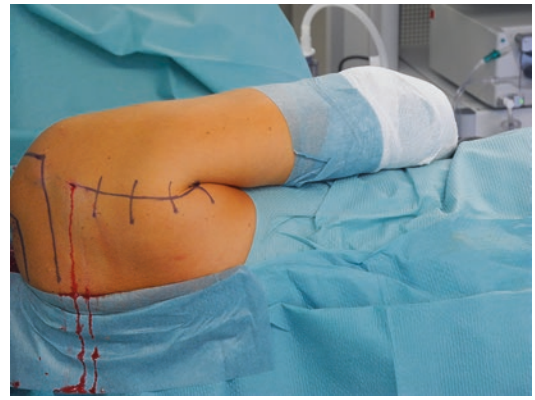


Fig. 24.2 The patient is placed in the lateral decubitus position. An incision is made from the posterior viewing portal toward the axillary pouch

24.6.2 Step 2: Latissimus Dorsi Tendon Preparation and Release

The latissimus dorsi is then separated from teres major proximally to its insertion site and then carefully detached from the shaft of the humerus in maximum internal rotation. Traction sutures are used to arm the tendon (Figs. 24.3 and 24.4). Care must be taken to avoid damage to the radial nerve. The thoracodorsal vessels and nerve are identified.

24.6.3 Step 3: Footprint Preparation and Anchor Placement

With the arm in 90° of flexion and maximum external rotation, the back of the greater tuberosity is identified, and the posterosuperior rotator cuff and glenohumeral joint are evaluated.

An area 3 cm wide on the posterosuperior aspect of the greater tuberosity was prepared, and three titanium corkscrew suture anchors (Arthrex Inc., Naples, Florida) armed with two pairs of No. 2 FiberWire were inserted 1 cm apart from each other (Fig. 24.5).

24.6.4 Step 4: Transposition and Reattachment

The tendon of latissimus dorsi is then transposed to the prepared bony bed at the site of the insertion of infraspinatus and attached using the sutures from the anchors (Fig. 24.6) with Mason-Allen stitches or a double-row construct. Finally, stability of the reconstruction and the vitality of the muscle flap are controlled (Fig. 24.7).

Postoperative Rehabilitation. The patients are immobilized for 48 h in a sling and then in an abduction pillow for 6 weeks. In this period, passive movement is restricted to 30° of abduction, 30° of flexion, 60° of internal rotation, and 0° of external rotation. From weeks 4 to 6, 60° of abduction, 90° of flexion, 60° of

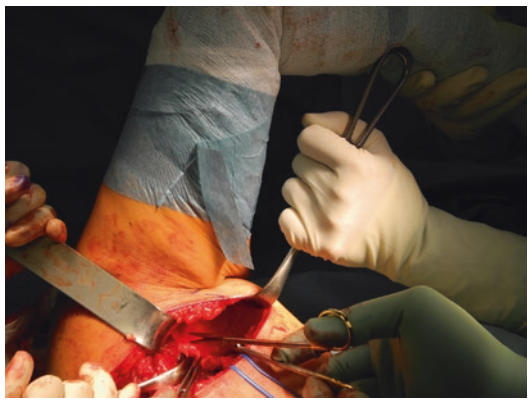


Fig. 24.3 Next, the latissimus and teres major muscles are identified and mobilized. The tendon needs to be released sharply from the bone using scissors or knife

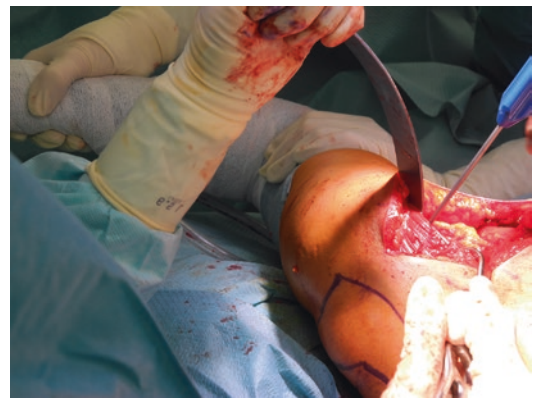


Fig. 24.5 Then, the posterosuperior aspect of the greater tuberosity is prepared, and three suture anchors are inserted 1 cm apart from each other

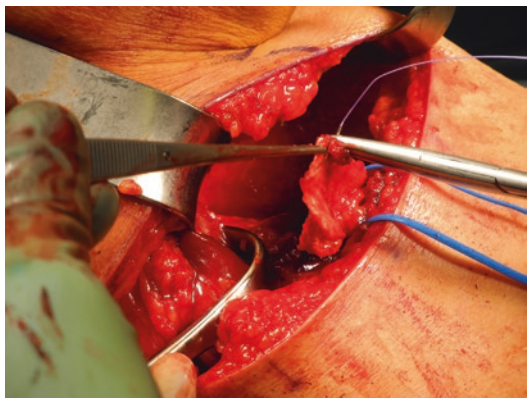


Fig. 24.4 After mobilization of the muscle, traction sutures are placed through the tendon

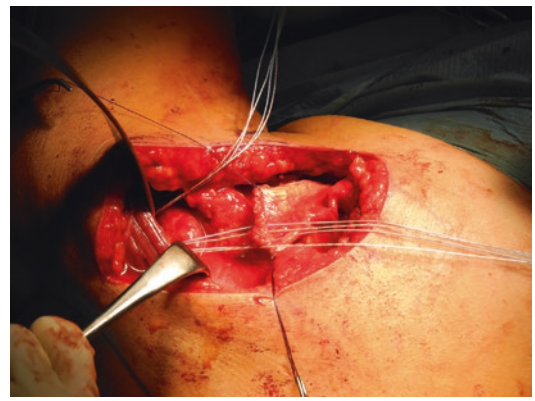


Fig. 24.6 In external rotation of the arm, the sutures are then stitched through the tendon, and the tendon is fixed onto the greater tuberosity

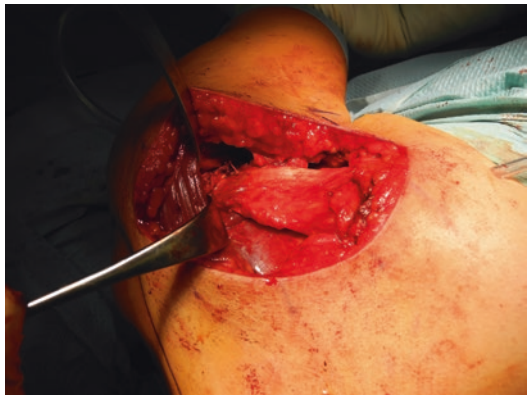


Fig. 24.7 After knotting the sutures and placing a final knotless anchor for double-row repair, the final result can be checked

internal rotation, and 0° of external rotation are allowed, and after 6 weeks free range of movement is exercised. Strengthening exercises are started after 8 weeks when a full passive range of movement is established.

24.7 Predictive Factors and Outcomes

Several factors have been identified to negatively influence the postoperative outcome following LDT:

- Insufficient subscapularis function
- Release of the deltoid muscle
- Fatty atrophy of the teres minor
- Previous surgical interventions (discussed controversially) [3–5]

The following table gives some data regarding outcomes that can be expected following the single and double incision technique for LDT.

	Double incision technique Gerber et al. (2013) [4]	Double incision technique El-Azab et al. 2015 [3]	Single incision technique Habermeyer et al. 2006 [2]
No. of patients	46	93	31
Age	56	56	62
Follow-up (months)	147	112	45
Constant score preop. (P)	56	44	43
Constant score postop. (P)	80	71	70
Pain preop. (P)	7 (15P = no pain)	8 (VAS: 0P = no pain)	8 (15P = no pain)
Pain postop. (P)	13	2	14
Strength preop. (kg)	1.2	1.6	2.1
Strength postop. (kg)	2.0	3.4	1.9
Flexion preop.	118	86	131
Flexion postop.	132	134	170
Abduction preop.	112	89	118
Abduction postop.	123	127	163
ERO preop.	18	18	26
ERO postop.	33	29	23
AHD pre-/postop.	Decreased by 2.5 mm	Decreased by 1.0 mm	Constant
Glenohumeral OA pre-/postop.	Increased	Increased	Increased

Outcomes following LDT procedure
AHD acromiohumeral distance, *OA* osteoarthritis

24.8 Summary

Latissimus dorsi transfer is a good option for a selected group of patients with irreparable posterosuperior rotator cuff tears. If patients are selected carefully, this procedure can provide a predictable improvement of pain and shoulder function.

In contrast to modern techniques like the "superior capsule reconstruction" or "subacromial balloon spacer implantations," this procedure has already shown to reliably better patients' situation in the long-term follow-up.

References

1. Gerber C, Vinh TS, Hertel R, Hess CW. Latissimus dorsi transfer for the treatment of massive tears of the rotator cuff. A preliminary report. Clin Orthop Relat Res. 1988;232:51–61.
2. Habermeyer P, Magosch P, Rudolph T, Lichtenberg S, Liem D. Transfer of the tendon of latissimus dorsi for the treatment of massive tears of the rotator cuff: a new single-incision technique. J Bone Joint Surg Br. 2006;88(2):208–12.
3. El-Azab HM, Rott O, Irlenbusch U. Long-term follow-up after latissimus dorsi transfer for irreparable posterosuperior rotator cuff tears. J Bone Joint Surg Am. 2015;97(6):462–9.
4. Gerber C, Rahm SA, Catanzaro S, Farshad M, Moor BK. Latissimus dorsi tendon transfer for treatment of irreparable posterosuperior rotator cuff tears: long-term results at a minimum follow-up of ten years. J Bone Joint Surg Am. 2013;95(21):1920–6.
5. Irlenbusch U, Bracht M, Gansen HK, Lorenz U, Thiel J. Latissimus dorsi transfer for irreparable rotator cuff tears: a longitudinal study. J Shoulder Elb Surg. 2008;17(4):527–34.

25 Tendon Transfer for Posterosuperior Cuff: Lower Trapezius Transfer

Gia Rodriguez-Vaquero, Gonzalo Samitier, and Emilio Calvo

25.1 Introduction

Irreparable rotator cuff tears can be especially difficult to manage in young population.

A massive cuff tear is considered irreparable when a direct repair of native tendon to the greater tuberosity cannot be achieved despite an exhaustive release of the remaining tissue [1, 2]. Other criteria for the reparability are rotator cuffs involving two or more tendons that are retracted up to the glenoid level and show relevant fatty infiltration at least stage 3 [3, 4], as well as proximal migration of the humeral head [5].

Results after surgical repair of massive rotator cuff tears have reported high failure rates. Shamsudin et al. [6] reported failure rates of massive posterosuperior rotator cuff repair ranging from 21% in primary repair to 40% in revision rotator cuff repair. Although aggressive anterior and posterior interval releases have been proposed to repair massive posterosuperior rotator cuff tears, Kim et al. have reported also rates of failure up to 91% using these techniques [7].

Many surgical techniques have been proposed for irreparable rotator cuff tears when a complete repair cannot be achieved, including cuff debridement, partial repair, or biceps tenotomy [8]. Reverse total shoulder arthroplasty has predictable good outcomes and pain relief and good function in case of cuff tear arthropathy and irreparable rotator cuff tears with pseudoparalysis [9] and is regarded as an excellent option, especially in elderly patients.

When there is not advanced glenohumeral osteoarthritis in young patients, surgeons must be reluctant to offer reverse shoulder replacement because of durability issues. In these situations, tendon transfers may offer a solution.

Gerber described in 1988 [2] the latissimus dorsi transfer for irreparable rotator cuff tears. Subsequent studies with medium- and long-term follow-up of these transfers report good pain relief and improvements of shoulder motion [10–14].

In case of fatty infiltration grade 3 or higher, osteoarthritis, subscapularis insufficiency, or preoperative forward elevation <90° results are less predictable [15–17]. When subscapularis or deltoid insufficiency is present, latissimus dorsi transferred for posterosuperior rotator cuff may cause inferior humeral head subluxation due to the vertical force vector developed by the transferred tendon [16]. Latissimus dorsi transferred

G. Rodriguez-Vaquero
Department of Orthopaedic Surgery and Traumatology, Hospital General de Villalba, Madrid, Spain
e-mail: gia.rodriguez@quironsalud.es

G. Samitier · E. Calvo (✉)
Department of Orthopaedic Surgery and Traumatology, Hospital General de Villalba, Fundacion Jimenez Diaz, Madrid, Spain
e-mail: ecalvo@fjd.es

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_25

to the greater tuberosity works as external rotator and humeral head depressor whenever the subscapularis and deltoid muscles are intact compensating the missing infraspinatus function [18].

Hartzler et al. [19] evaluated different types of tendon transfer to restore external rotation of the shoulder. These authors found that the lower trapezius transfer (LTT) resulted in superior external rotation moment arm in adduction compared with latissimus dorsi transfer. Omid et al. [8] found in other cadaveric study superior results for restoration of shoulder external rotation with LTT but also improvement of glenohumeral kinematics and restoration of anteroposterior force couple balance, compared with latissimus dorsi transfer.

Trapezius muscle is divided into superior, inserted in acromion and superior lateral spine; middle, inserted in superior scapular spine surface; and lower portion, inserted in the inferomedial scapular spine. It is innervated by the spinal accessory nerve (cranial nerve XI) [20]. During the dissection for the graft, pedicle is situated along the underside of the muscle between 2.3 and 5.8 mm (average 3.25 cm) medial to the distal insertion of the lower portion of the trapezius in the scapular spine [21]. It is possible to transfer the lower portion of the trapezius directly without the use of tendon grafts; however, there is a high risk of spinal accessory nerve traction injury as demonstrated in a cadaveric study by Gracitelli el al. [22].

Elhassan and Bertelli first described LTT to restore external rotation in patients with brachial plexus palsy [18, 23]. Biomechanical and clinical studies support the use of this muscle to mainly restore external rotation when the posterosuperior cuff is deficient or irreparable [8, 18, 19, 24, 25].

Arthroscopically assisted lower trapezius transfer was first described in 2016 by Elhassan et al. [3]. This transfer has become a good option of treatment of irreparable posterosuperior cuff tears in young people with no significant glenohumeral osteoarthritis changes.

Table 25.1 Indications for lower trapezius transfer

Irreparable posterosuperior rotator cuff tear
• Refractory shoulder pain without stiffness
• Loss of external rotation
• Fatty infiltration (Goutallier 3 (27))
Young and active patient
Glenohumeral arthrosis Hamada grade 3 or less (32)

25.2 Indications

The ideal candidate for LTT is a relatively young patient with an irreparable posterosuperior rotator cuff tear with refractory shoulder pain to conservative or other surgical treatments like partial repair and debridement and the absence of external rotation. The shoulder should not be stiff, and a flexion of at least 60° should be preserved [25]. Fatty infiltration is not a contraindication for LTT, but major arthritic changes (superior to Hamada grade 3) should not be present [26] (Table 25.1).

25.3 Preoperative Assessment

25.3.1 Physical Exam

Most patients with massive rotator cuff tears have pain in the deltoid region irradiated to the lateral area of the shoulder; function loss, reduced strength, and crepitation are frequent findings also in these patients; night pain is characteristic and interferes with their ability to sleep; pain also typically worsens with overhead activities or when trying to lift objects. Physical examination should include an evaluation for muscle atrophy and scapular dyskinesia, passive and active range of motion of the affected and unaffected shoulders, and provocative maneuvers to rule out different shoulder pathologies, cervical spine problems, or neurovascular compressive syndromes.

Specific maneuvers help to assess the affected tendons and their degree of incompetence. The insufficient infraspinatus will manifest as an important loss of external rotation strength in adduction and lag or dropping sign in case of

massive posterosuperior cuff tear. Lag sign or dropping sign and hornblower sign may orient us to a massive posterosuperior cuff tear.

25.3.2 Imaging

Plain radiographs (anteroposterior view, axial and lateral scapula Y view) allow to evaluate articular changes like acromial changes (shape, acetabulization, os acromiale), proximally migrated or decentered humeral head, tuberosity sclerotic changes, and cysts or signs of cuff tear arthropathy. Magnetic resonance imaging (MRI) is the gold standard test choice to evaluate bony and soft tissue structure of the shoulder. MRI allows measuring the cuff tear, fatty infiltration, length of the tendon, level of retraction, and cartilage and bone changes [4]. Computed tomography (CT) scan can be used too to characterize cuff tear and fatty infiltration.

The Goutallier grading system was first recognized using computed tomography [27]; nowadays, it is most easily assessed on MRI non-fat-saturated oblique-sagittal T1 sequences which have superior fat-to-muscle contrast [4]. Using intra-articular injection of contrast (CT arthrogram) will enhance evaluation of the tear.

25.4 Surgical Technique

25.4.1 Positioning and Preparation

Anesthesia is carried out following a standardized protocol based on a single-shot interscalene blockade under ultrasound control (L-bupivacaine 0.5% 30–40 ml plus epinephrine) combined with general anesthesia (propofol 2–2.5 mg/kg iv and alfentanil 20–150 μg/kg iv initially, plus 15 μg/kg bolus, and maintenance with sevoflurane). Antibiotic prophylaxis (2-g cefazolin or 1-g vancomycin as alternative for patients with b-lactam allergy) is administered 30 min before surgery.

The patient can be placed in lateral decubitus or beach chair position. The lateral decubitus position is more commonly preferred for the open technique as described by Elhassan et al. in 2014 [28]. Beach chair position is the option of choice for the arthroscopic-assisted technique. A Betaclassic mobile OR Maquet® table or equivalent with a head holder system allows full access to the posterior aspect of the scapula facilitating the graft harvesting. The arm is placed in a pneumatic arm holder allowing movement during the surgery. The greater trochanter must be aligned with the break in the operating table to allow hip flexion preventing sciatic nerve compression, and the torso must be kept in neutral position using straps to prevent any lateralization of the patient during the procedure. It is also recommended to keep the head centered maintaining a neutral position of the neck with no rotation. This setup allows the surgeon to stand in front or behind the shoulder alternatively moving around easily the arm depending on the surgery stage that is being carried out. It is also important to adequately pad patient's heels, hands, and forearms.

The operative extremity is prescrubbed with chlorhexidine solution and drapped conveniently. It is important to leave the entire ipsilateral half of the back uncovered until midline. At the conclusion, the surgical team should change gloves and conduct a final preincision timeout.

During the arthroscopic time, controlled hypotension and muscular relaxation are desirable as it may allow better visualization, decrease blood loss, and reduce operative time which secondarily can affect the quality of the repair and patient safety. Because of the risk for neurological ischemic events, caution should be exercised with hypotensive anesthesia in the beach chair position. Elderly patients, hypertensive patients with poor control, those with body mass index superior to 34, patients with diabetes mellitus, those with obstructive sleep apnea, and patients with previous history of stroke or cardiac events are considered high-risk population [29]. We maximize patient safety using routinely near-infrared spectroscopy (NIRS), which provides a noninvasive continuous assessment of cerebral perfusion.

For fluid management, we use an automated pump system with dual pressure and volume control (FMS®; DePuy, Mitek, Raynham, Massachusetts). We usually set up the pump to start at 80/90 mmHg.

25.4.2 Surgical Technique

We first delineate on the skin the medial border of the scapula and the lower trapezius insertion site on the spine of the scapula. It is recommended also to mark the osseous eminences of the shoulder and the arthroscopic portals.

For graft harvesting, we can either follow a vertical skin incision, 5–8 cm in length, approximately 1 cm medial to the medial border of the scapula starting from the upper-medial border, or a 5-cm transverse incision just inferior to the scapular spine from 2 cm medial to 3 cm lateral to the medial border of the spine of the scapula. After the skin incision and subcutaneous dissection, we will find a triangular fat area corresponding with the lateral border of the trapezius; dissecting this area medially and laterally will expose the tendon from the deep fascial tissue. The optimal method to identify the lower trapezius tendon is to "hook" it with the surgeon's index finger laterally beneath the trapezius, freeing the tendon from deep adhesions. There is a triangular bony region at the junction of the medial border of the scapula and the scapular spine free from tendon insertion (Fig. 25.1). Once the lower trapezius footprint is identified and isolated, we will be able to detach easily the trapezius off from its insertion in the scapular spine bony region. The shape of the footprint is triangular, and the length of the tendinous portion is about 49 mm [21]. Then we continue the dissection medially along the upper border of the tendon following the interval between the middle and lower trapezius with the goal of getting adequate release and mobilization of the tendon. The spinal accessory nerve lies within the fascial layer, underneath of the trapezius; thus, deep dissection should be performed with caution. Identifying the nerve is not mandatory, but it is advised if there is not enough excursion of the tendon in order to detect over-

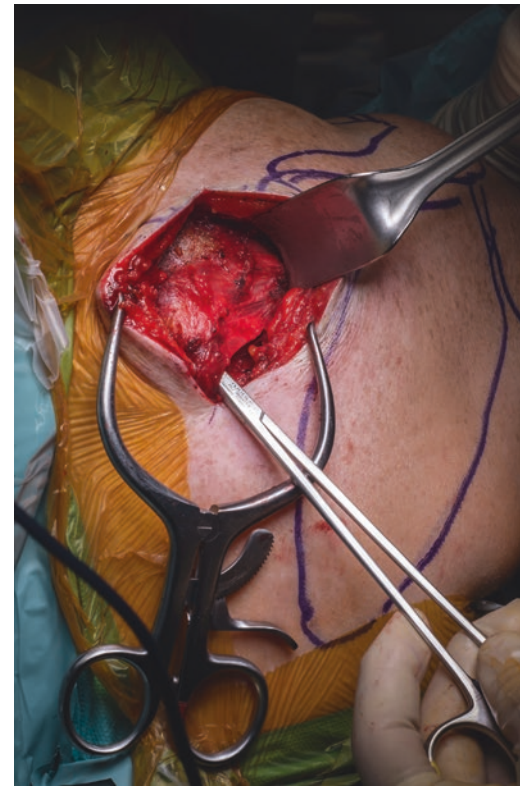


Fig. 25.1 Tendon identification for harvesting. The inferior border of the lower trapezius is identified at its insertion on the spine of the scapula and elevated with a forceps

tensioning. Removing the edge of the spine of the scapula can be helpful to avoid impingement between the graft and the accessory nerve at the level of the medial border of the spine.

Two #2 high-strength Orthocord® sutures (DePuy Synthes, Warsaw, IN) in a Krakow configuration at superior and inferior part of the tendon are used and are left inside the incision to prevent damage of the graft (Fig. 25.2).

The next step is the allograft preparation. It can be performed simultaneously while the lower trapezius is harvested. An Achilles tendon allograft without the osseous calcaneus portion is the graft of choice for this purpose. Again, two #2 high-strength Orthocord® sutures (DePuy Synthes, Warsaw, IN) in a Krakow configuration are used to prepare the thick and narrow end of the allograft. It is recommended using different colors for better identification of the sutures dur-

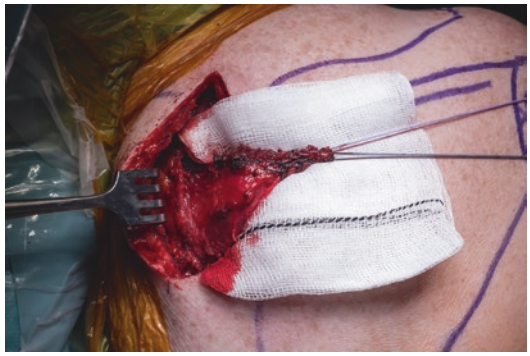


Fig. 25.2 Lower trapezius harvested. Two #2 high-strength sutures are passed through the superior and inferior edges of the tendon to facilitate manipulation

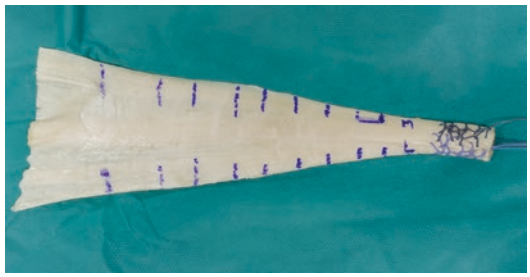


Fig. 25.3 Achilles tendon allograft. High-strength sutures are passed to prepare the narrow end. One suture has also been placed at the thin expanded side of the allograft. Medial and lateral borders are also marked

ing the arthroscopic time as well as marking the dorsal and frontal aspect of the graft. One suture is placed at the thin expanded side of the allograft to avoid lateral migration during the passage and fixation of the graft but also to facilitate suturing to the lower trapezius portion by creating some tension from pulling once the allograft is fixed in the humeral head (Fig. 25.3).

Then, the arthroscopic step is started. The main portals needed for this procedure are a posterior portal for visualization and additional anterolateral and lateral portals for instrumentation. Additional portals can be established if necessary. The scope is inserted through the posterior portal for visualization of the tuberosity and the remaining cuff, while the other portals are used initially for bursectomy, to prepare the tuberosity and to perform additional technical gestures as needed, depending on the findings. The supraspinatus footprint debridement must cause bleeding of the subchondral bone to enhance graft healing. We also need to create a passing track for the allograft underneath the infraspinatus fascia. The anterolateral portal is placed just 1 cm distal to the edge of the anterior corner of the acromion. From this portal, we introduce in the subacromial space a long grasping clamp; then, under the opened infraspinatus fascia, the clamp is directed toward the harvesting wound. A moist packing gauze can be used to enlarge the passing area for the allograft. Once the medial wound was achieved with our clamp, we gather the sutures placed in the thick end of the allograft to pull them out through the anterolateral portal. Before the definitive fixation of the graft over the greater tuberosity, we must check that there is an optimal glissade of the graft pulling from it back and forth using our prearranged sutures in both ends (Fig. 25.4).

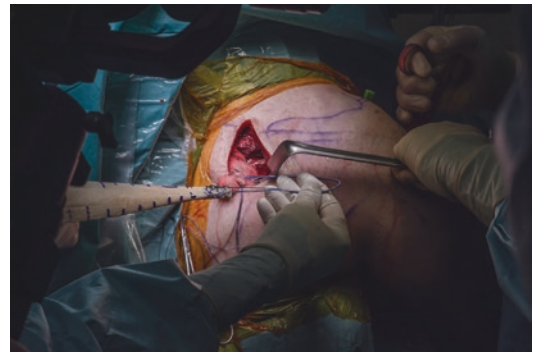


Fig. 25.4 Allograft orientation

Allograft attachment with suture anchors to the tuberosity is first carried out. The allograft must be visualized into the joint looking for the dorsal mark which indicates that our graft is not flipped. Two 5.5 mm Healix Advance KnotlessTM anchors (DePuy Mitek Sports Medicine, Raynham, MA) are used, one for each Krakow suture, and buried anteromedial and anterolateral in the footprint area of the greater tuberosity. For extra fixation, one or two HealixTM Advance 5.5-mm double-threaded anchors (DePuy Synthes, Warsaw, IN) are recommended as medial row anchors; the sutures are passed through the allograft using

a Cleverhook instrument (DePuy Mitek Sports Medicine) or any other direct or indirect suture passer device. The extra suture of the anchor can be used to get additional fixation of the allograft to the remnant of the native rotator cuff. It is important to remind your assistant to hold some tension in the opposite side of the allograft during suture passing and knotting to avoid fixation in a twisted position (Fig. 25.5).

When the intra-articular allograft fixation is finished, adequate allograft excursion must be checked with several cycles of shoulder external and internal rotations holding the free part of the allograft increasing the tension (Fig. 25.6).

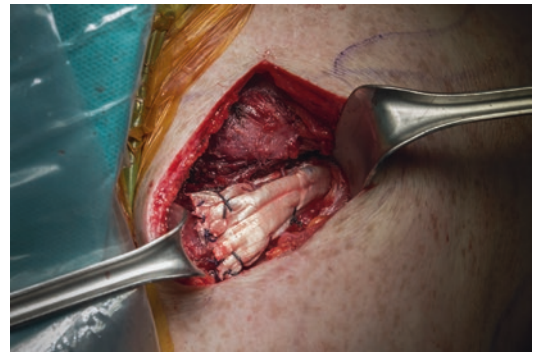


Fig. 25.5 Allograft in position. The lateral thin end has been passed into the subacromial space, while the medial wide end is sutured on the lower trapezius end

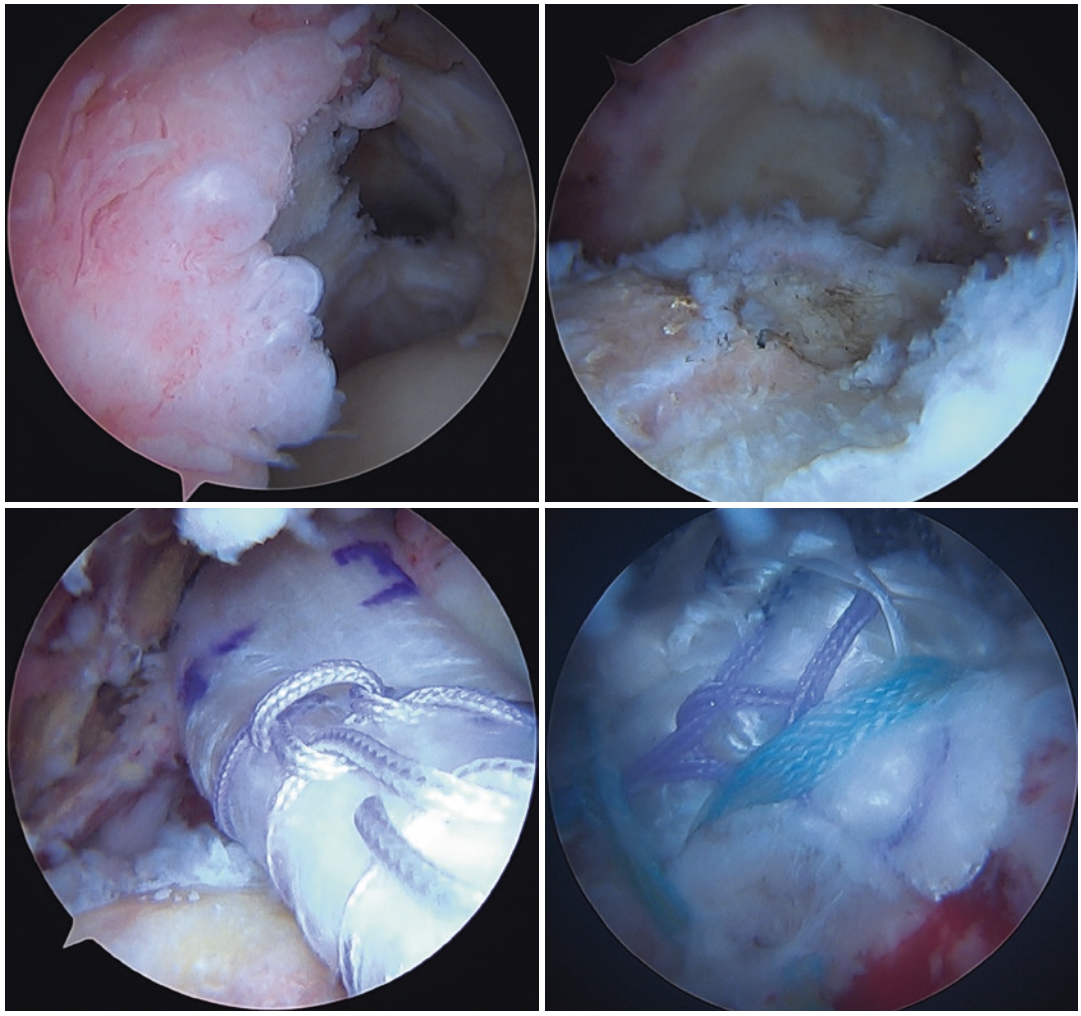


Fig. 25.6 Achilles tendon allograft passing into the join from posterior to anterior pulling from anterosuperior portal. Medial and lateral borders indicate the adecuate orientation of the allograft not twisted

The last step is the attachment of the Achilles allograft to the lower trapezius. Using the arm holder, the arm is placed in maximal external rotation with some flexion and no abduction. In this position, the Krakow sutures that we prepared at the beginning of the surgery are passed with a free needle laterally through the allograft. It is recommended to reinforce the fixation with some free sutures medially removing the remaining allograft.

Arthroscopic portals are closed using 3-0 Monocryl® suture (Ethicon, Johnson & Johnson, Somerville, NJ); the open wound is closed in layers using 0 and 2-0 Vicryl, and a running 3-0 Monocryl stitch is used for skin closure with no drain; the wound is covered with sterile dressing, and the patient's arm is placed in a brace with an anti-rotatory pillow.

25.5 Postoperative Management and Follow-Up

The postoperative rehabilitation period begins after 6 weeks of immobilization in a brace with a pillow avoiding internal rotation. We only allow removal of the brace during this period for bath and flexion-extension exercises of the elbow every day. After 6 weeks, the patient starts proper physical therapy including progression from passive to active assisted motion and finally unassisted active motion around 12 weeks. Physical therapy should include special training of the transfer for its new function. External rotation strengthening exercises with elastic bands begin at 16 weeks. Unrestricted activity is allowed after 6 months from surgery. Standard shoulder AP and axial radiographic views are recommended at 3–6 and 12 months to detect any precocious off-center humeral head change from previous X-ray [3].

25.6 Results

Based on the midterm results as shown from Elhassan et al., on average, the vast majority of patients who undergo lower trapezius transfer with Achilles tendon allograft for massive irreparable posterosuperior rotator cuff tears experience significant improvement of pain, external rotation, shoulder flexion, and abduction, although better motion is observed in patients with preoperative flexion over 60° [25].

Elhassan et al. published in 2016 the outcomes of LTT for this indication with a minimum of 2 years follow-up. The study included 33 patients who underwent open transfer of the lower trapezius through an osteotomy of the acromion to reconstruct patients with persistent symptomatic posterosuperior rotator cuff massive tears. Eleven patients had no prior surgeries, but the remaining 22 patients had undergone an average of two prior surgeries [25].

At an average follow-up of 47 months (range, 24–73 months), 32 of the 33 patients had significant improvement in pain levels ($P < 0.01$) and shoulder range of motion, with an average forward flexion (FF) of 120° (range, 80–150°) (average preoperative FF 70°), abduction (ABD) 90° (range, 60–140°) (average preoperative ABD 40°), and 50° of external rotation (ER) (range, 20–70°; $P < 0.01$) (average preoperative ER 20°). Postoperative internal rotation (IR) was maintained when compared to the preoperative examination.

Regarding the results in clinical scores, the mean SSV improved from 54% preoperatively to 78% postoperatively ($P < 0.01$), and the mean DASH score improved from 52 ± 19 to 18 ± 10 ($P < 0.01$). At clinical examination, palpation of the transferred lower trapezius demonstrated active muscle contraction during shoulder external rotation.

Of note, when the eight patients who had flexion/abduction of less than 60° preoperatively were compared with the 25 patients who had more motion, the latter group had more significant improvement of motion. Shoulder external rotation motion and strength improved in all patients regardless of the extent of the preoperative loss of motion, with grade 4 or higher muscle strength achieved in all patients.

When radiographs were evaluated for arthritic changes, the authors noticed a mild increase in joint narrowing in patients who did not have

full correction of the proximal migration of the humeral head; however, none of these patients showed signs of progressive arthritis on radiographs at the final outcome. In addition, interestingly, the authors did not find a correlation between the extent of correction of the proximal migration of the humeral head and the outcome of the tendon transfer reconstruction [25].

Valenti and Werthel recently published a variation of the original technique of LLT extended with a semitendinosus tendon and fixed to the insertion of the infraspinatus via arthroscopy. They included 14 patients with a mean follow-up of 24 months (range: 12–36 months). The semitendinosus graft is introduced at the level of the insertion of the infraspinatus into an anteroposterior bone tunnel and locked with a ZipTight device for fixation. Mean active forward flexion improved from 150° to 160°, external rotation with the arm at the side improved from −20° to 24°, and external rotation with the arm at 90° of abduction improved from −10° to 40°. The mean Constant–Murley score improved from 35 to 60. Mean VAS decreased from 7 to 2 (visual analogue scale, 0–10), and mean SSV improved from 40% to 70% ($P < 0.01$). Both the lag sign and hornblower sign were negative after this transfer.

25.7 Complications

From the experience in patients with brachial plexus injury and paralytic shoulder, when LT was performed as single-tendon transfer, complications from surgery were unusual and generally not serious. Elhassan in 2014, from a total of 111 patients with this diagnosis, reported seroma in patients with no drain (11 patients) and worsening postoperative pain in patients who experienced deafferentation pain from the brachial plexus injury (23 patients) [28]. Most of the complications they encountered in this group of patients with single-tendon transfer were related to the postoperative custom-made brace as skin irritation and soreness related to pressure from the brace, which can lead to intolerance and poor compliance.

Elhassan et al. reported complications on the aforementioned study of 33 patients with open LTT and found also seroma formation in four patients, who were managed by observation with no sequelae. One patient sustained a fall during his first month of rehabilitation and lost some of the gains; ultrasound imaging of the lower trapezius showed some redundancy in the Achilles tendon with external rotation, indicating stretch injury of the transfer. There was one infection in this series requiring debridement and later shoulder fusion [26]. Acromion osteotomy healed radiographically in 25 of the 33 patients, but, clinically, there was no difference in the examination results between patients whose osteotomy had healed and those whose osteotomy did not heal radiographically, and this did not change at the last follow-up evaluation.

Valenti et al. reported two patients with hematoma localized on the harvest site, and one of them had deep infection requiring open debridement and oral antibiotics, but both had good outcomes.

The arthroscopic approach is associated with faster short-term recovery, reduced infection rate, and less complications related to the open technique because of the transacromial approach needed which increases the risk of acromial malunion/nonunion and deltoid insufficiency [3].

25.8 Summary

Arthroscopic transfer of the lower trapezius using Achilles tendon allograft to reconstruct massive irreparable posterosuperior rotator cuff tear leads to good outcomes in most patients, especially those with preoperative flexion over 60°. Longer follow-up is required to confirm the durability of the transfer. Prospective randomized studies comparing the LTT with other therapeutic options as the latissimus dorsi transfer or combined SCR in the long term will further help to elucidate the difference between the two transfers and other therapeutic options.

References

1. Valenti P. Joint-preserving treatment options for irreparable rotator cuff tears. Orthopade. 2018;47(2):103–12.
2. Gerber C, Vinh TS, Hertel R, Hess CW. Latissimus dorsi transfer for the treatment of massive tears of the rotator cuff. A preliminary report. Clin Orthop Relat Res. 1988;232:51–61.
3. Elhassan BT, Alentorn-Geli E, Assenmacher AT, Wagner ER. Arthroscopic-assisted lower trapezius tendon transfer for massive irreparable posterior-superior rotator cuff tears: surgical technique. Arthrosc Tech. 2016;5(5):e981–e8.
4. Goutallier D, Postel JM, Bernageau J, Lavau L, Voisin MC. Fatty muscle degeneration in cuff ruptures. Pre- and postoperative evaluation by CT scan. Clin Orthop Relat Res. 1994;304:78–83.
5. Keener JD, Wei AS, Kim HM, Steger-May K, Yamaguchi K. Proximal humeral migration in shoulders with symptomatic and asymptomatic rotator cuff tears. J Bone Joint Surg Am. 2009;91(6):1405–13.
6. Shamsudin A, Lam PH, Peters K, Rubenis I, Hackett L, Murrell GAC. Revision versus primary arthroscopic rotator cuff repair: a 2-year analysis of outcomes in 360 patients. Am J Sports Med. 2015;43(3):557–64.
7. Kim S-J, Kim S-H, Lee S-K, Seo J-W, Chun Y-M. Arthroscopic repair of massive contracted rotator cuff tears: aggressive release with anterior and posterior interval slides do not improve cuff healing and integrity. J Bone Joint Surg Am. 2013;95(16):1482–8.
8. Omid R, Heckmann N, Wang L, McGarry MH, Vangsness CT Jr, Lee TQ. Biomechanical comparison between the trapezius transfer and latissimus transfer for irreparable posterosuperior rotator cuff tears. J Shoulder Elb Surg. 2015;24(10):1635–43.
9. Drake GN, O'Connor DP, Edwards TB. Indications for reverse total shoulder arthroplasty in rotator cuff disease. Clin Orthop Relat Res. 2010;468(6):1526–33.
10. Elhassan B, Bishop AT, Hartzler RU, Shin AY, Spinner RJ. Tendon transfer options about the shoulder in patients with brachial plexus injury. J Bone Joint Surg Am. 2012;94(15):1391–8.
11. Axe JM. Tendon transfers for irreparable rotator cuff tears: an update. EFORT Open Rev. 2016;1(1):18–24.
12. Moursy M, Forstner R, Koller H, Resch H, Tauber M. Latissimus dorsi tendon transfer for irreparable rotator cuff tears: a modified technique to improve tendon transfer integrity. J Bone Joint Surg Am. 2009;91(8):1924–31.
13. Gerber C. Latissimus dorsi transfer for the treatment of irreparable tears of the rotator cuff. Clin Orthop Relat Res. 1992;275:152–60.
14. Namdari S, Voleti P, Baldwin K, Glaser D, Huffman GR. Latissimus dorsi tendon transfer for irreparable rotator cuff tears: a systematic review. J Bone Joint Surg Am. 2012;94(10):891–8.
15. Costouros JG, Espinosa N, Schmid MR, Gerber C. Teres minor integrity predicts outcome of latissimus dorsi tendon transfer for irreparable rotator cuff tears. J Shoulder Elb Surg. 2007;16(6):727–34.
16. Gerber C, Maquieira G, Espinosa N. Latissimus dorsi transfer for the treatment of irreparable rotator cuff tears. J Bone Joint Surg Am. 2006;88(1):113–20.
17. Iannotti JP, Hennigan S, Herzog R, Kella S, Kelley M, Leggin B, et al. Latissimus dorsi tendon transfer for irreparable posterosuperior rotator cuff tears. Factors affecting outcome. J Bone Joint Surg Am. 2006;88(2):342–8.
18. Elhassan B, Bishop A, Shin A, Spinner R. Shoulder tendon transfer options for adult patients with brachial plexus injury. J Hand Surg Am. 2010;35(7):1211–9.
19. Hartzler RU, Barlow JD, An KN, Elhassan BT. Biomechanical effectiveness of different types of tendon transfers to the shoulder for external rotation. J Shoulder Elb Surg. 2012;21(10):1370–6.
20. Crowe MM, Elhassan BT. Scapular and shoulder girdle muscular anatomy: its role in periscapular tendon transfers. J Hand Surg Am. 2016;41(2):306–14.
21. Omid R, Cavallero MJ, Granholm D, Villacis DC, Yi AM. Surgical anatomy of the lower trapezius tendon transfer. J Shoulder Elb Surg. 2015;24(9):1353–8.
22. Gracitelli MEC, Assunção JH, Malavolta EA, Sakane DT, de RMR, Ferreira Neto AA. Trapezius muscle transfer for external shoulder rotation: anatomical study. Acta Ortop Bras. 2014;22(6):304–7.
23. Bertelli JA. Lengthening of subscapularis and transfer of the lower trapezius in the correction of recurrent internal rotation contracture following obstetric brachial plexus palsy. J Bone Joint Surg Br. 2009;91(7):943–8.
24. Bertelli JA. Upper and lower trapezius muscle transfer to restore shoulder abduction and external rotation in longstanding upper type palsies of the brachial plexus in adults. Microsurgery. 2010;31(4):263–7.
25. Elhassan BT, Wagner ER, Werthel JD. Outcome of lower trapezius transfer to reconstruct massive irreparable posterior-superior rotator cuff tear. J Shoulder Elb Surg. 2016;25(8):1346–53.
26. Hamada K, Yamanaka K, Uchiyama Y, Mikasa T, Mikasa M. A radiographic classification of massive rotator cuff tear arthritis. Clin Orthop Relat Res. 2011;469(9):2452–60.
27. Fuchs B, Weishaupt D, Zanetti M, Hodler J, Gerber C. Fatty degeneration of the muscles of the rotator cuff: assessment by computed tomography versus magnetic resonance imaging. J Shoulder Elb Surg. 1999;8(6):599–605.
28. Elhassan B. Lower trapezius transfer for shoulder external rotation in patients with paralytic shoulder. J Hand Surg Am. 2014;39(3):556–62.
29. Papadonikolakis A, Wiesler ER, Olympio MA, Poehling GG. Avoiding catastrophic complications of stroke and death related to shoulder surgery in the sitting position. Arthroscopy. 2008;24(4):481–2.

26 Tendon Transfer for Anterosuperior Cuff: The Pectoralis Major Transfer

Jean Kany

26.1 Introduction

Posterosuperior rotator cuff tears are among the most common shoulder disorders. Repairing them is frequently performed [1] by surgeons. On the contrary, ruptures of the subscapularis tendon, isolated or combined with a supraspinatus tear, are even less common [2, 3]. When the muscle belly has a fatty infiltration beyond stage 3 of Goutallier [4], when the tendon is too short to be pulled and sutured onto the lesser tuberosity even with a 60° of shoulder abduction and after proper surgical release, after a failed previous surgical repair (re-tear) or in case of anterosuperior escape of the humeral head, the subscapular rupture can prove to be massive and irreparable.

Many treatment options exist for patients with irreparable subscapularis tears, starting with nonsurgical treatments [5]. Physical therapy usually focuses on pain management, followed by deltoid reconditioning [6] and strengthening of any remaining cuff and peri-scapular muscles [7]. Oral nonsteroidal anti-inflammatory drugs and subacromial corticosteroid injections can also be used [5], but after failure of these medical treatments, surgical options should be considered. Unfortunately, there is no consensus on the surgical treatment of irreparable tears in the absence of glenohumeral arthritis.

Boileau el al. [8] and Walch et al. [9] after tenotomy of the long head of the biceps alone in elderly patients as well as Burkhart et al. [10] after partial cuff repair using the *margin convergence* technique showed acceptable results for posterosuperior cuff deficiency but not for anterosuperior deficiency. Other different types of operations have been published, including rotator cuff debridement (with or without suprascapular nerve release) [11, 12], partial rotator cuff repair [13, 14], tendon transfers [15, 16], superior capsular reconstruction [17, 18], and even reverse shoulder arthroplasty [19]. Superior capsular reconstruction might represent a proper option as a joint-preserving technique for younger patients, but no report could be found concerning the use of SCR in subscapularis deficiency. Reverse shoulder arthroplasty, in turn, is probably neither the only one nor the best option for younger active patients, as the longevity of these implants in this specific population is yet unknown and also due to the fact that this technique could still be used following possible failures of other types of surgery.

In such a situation, the transfer of the pectoralis major tendon can be performed [20–25]. Different techniques have been described. The entire pectoralis major tendon [24] or only the clavicular [26] or the sternal head [27] can be harvested, and the transfer may be rerouted either above or underneath the conjoint tendon.

J. Kany (✉)
Clinique de l'Union, Ramsay, Saint Jean, France
e-mail: jean.kany@epaule-toulouse.com

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_26

The purpose of this article is to review the literature about the pectoralis major tendon transfer for the treatment of irreparable anterosuperior rotator cuff tear.

26.2 Surgical Technique

The surgery is performed in a beach chair position under general anesthesia with an interscalene block. A distalized deltopectoral approach (5–7 cm) allows to assess anterosuperior cuff and to harvest the pectoralis major tendon. The cephalic vein is retracted laterally with the anterior fibers of the deltoid, while the conjoint tendon is retracted medially. The pectoralis major is exposed at the level of the humerus, in front of the long head of the biceps, which is a precise anatomical landmark. The upper and lower borders of the PM tendon are identified at the insertion on the humerus as well as both heads of the muscle (Fig. 26.1). A biceps tenodesis is performed if still present at the upper part of PM tendon into the bicipital groove. Neither acromioplasty nor section of the coracoacromial ligament is performed. With the arm in abduction, medial rotation, and forward flexion, the scar tissue over the lesser tuberosity is resected, and the stump of the remnant native subscapularis retracted under the conjoint tendon is identified and grasped with two nonabsorbable sutures. It is essential to recognize the musculocutaneous and the axillary nerves correctly in order to prevent any damage (Fig. 26.2) [28, 29] after the transfer rerouting. The musculocutaneous nerve should not only be palpated but also visualized to check that there is an adequate space for passage superficial to the nerve. The distance between the coracoid process and the main trunk of the musculocutaneous nerve as it enters the coracobrachialis muscle averages 6.1 cm, but its proximal branch averages 4.4 cm [30]. Adhesions are released and scar tissues are resected from the lesser tuberosity. The remaining subscapularis tendon (if present) is released from the glenoid. When possible, a combined partial repair of subscapularis tendon is performed. The anterior humeral circumflex vessels landmark the distal edge of the pectoralis major tendon and are cauterized. Splitting of both the clavicular and the sternocostal portions is performed through the natural interval separating them medially from proximal to distal. The sternocostal portion twists around the clavicular one and is more deeply inserted: its tendon (the pos-

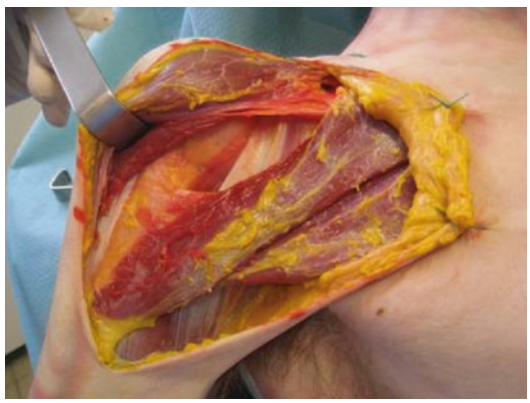


Fig. 26.1 (courtesy Valenti P): Right shoulder. Cadaveric view of the clavicular and sternal portions of the pectoralis major tendon

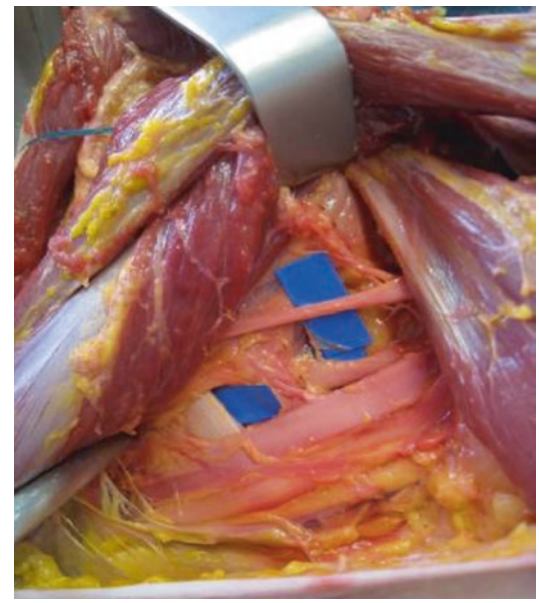


Fig. 26.2 (courtesy Valenti P): Right shoulder. Cadaveric view. Musculocutaneous and axillary nerves. The distance between the coracoid process and the main trunk of the musculocutaneous nerve as it enters the coracobrachialis muscle averages 6.1 cm, but its proximal branch averages 4.4 cm

terior lamina) is larger and stronger (6 cm length, 2.5 cm breadth) than the clavicular portion one (the anterior lamina). On the contrary, the clavicular tendinous fibers are more superficial, thinner, and shorter: they insert distally. Each portion can be harvested separately since two independent neurovascular pedicles supply for the pectoralis major: the lateral pectoral pedicle (12.5 cm from humeral insertion) and the medial pectoral pedicle (8.5 cm from humeral insertion and 2 cm from the inferior border) (Fig. 26.3). Depending on the chosen technique, one head of the tendon (or both) can be detached from the humerus with periosteum to reinforce the tendon [30]. The harvested tendon is tagged with a nonabsorbable suture and then passed either posteriorly looking for a "pulley effect" with the transfer, under the conjoint tendon (in front of the musculocutaneous nerve), or above (Fig. 26.4). The area of fixation onto the upper part of the lesser tuberosity is decorticated, and the harvested pectoralis major tendon is fixed "flat" onto the refreshed-bone surface area either transosseously (with two 2.7-mm tunnels) or with suture anchors (Fig. 26.5a, b). The anterior border of the supraspinatus tendon (when present) is sutured to the proximal border of the pectoralis major tendon, allowing a closure of the "new" rotator interval. When the supraspinatus tendon is torn, it is repaired (if possible) after release. In the specific cases of irreparable combined supraspinatus and subscapularis tendon rupture, the pectoralis major transfer can cover the anterior part of the greater tuberosity despite a short excursion. The tendon is sutured with the arm placed in a 0° to 30° external rotation position and forward flexion. The fixation is tested in rotations and retropulsion. Drainage is useless. Patients are immobilized in a sling with the arm at the side in a neutral position for 4 weeks. Personal pendulum exercises are to be started the day following surgery. Hydrotherapy is started after stitch removal if possible. Passive external rotation over 30° is allowed after

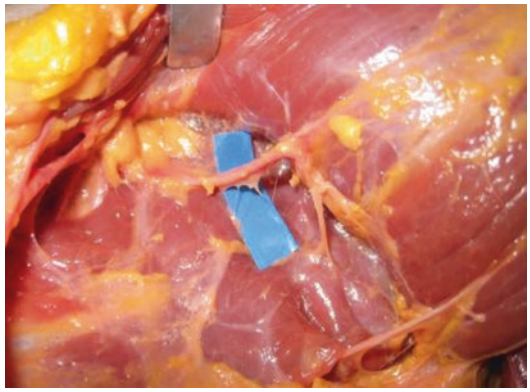


Fig. 26.3 (courtesy Valenti P): Right shoulder. Cadaveric view of the two independent pedicules which supply the pectoralis major: the lateral pectoral pedicle (12.5 cm from humeral insertion) and the medial pectoral pedicle (8.5 cm from humeral insertion and 2 cm from the inferior border)

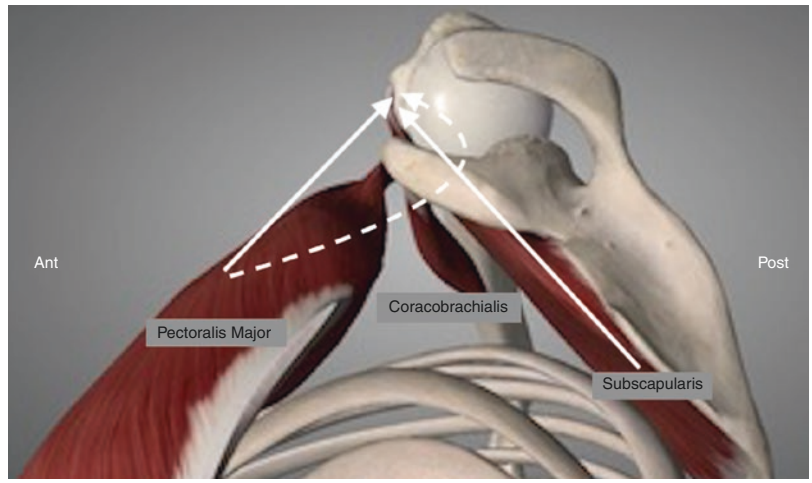


Fig. 26.4 Right shoulder. Superior view. The draw shows that the line of pull from the pectoralis major does not replicate the line of pull from the subscapularis. It has been shown that the rerouting underneath the conjoint tendon results in a better biomechanical effect than a transfer above the conjoint tendon

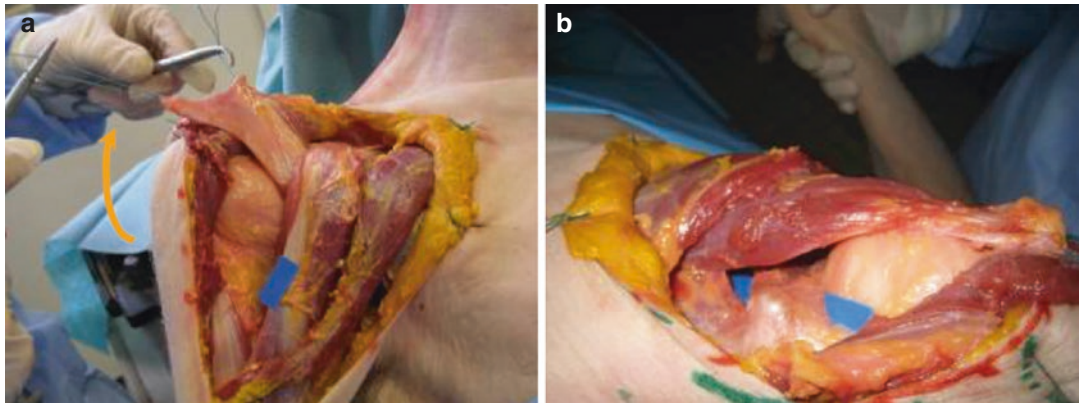


Fig. 26.5 [**a** and **b** (courtesy Valenti P)]: Right shoulder. Cadaveric view. (**a**) (left picture—anterior view): The pectoralis major (sternocostal portion) is rerouted underneath (behind) the conjoint tendon. (**b**) (right picture—superior view): The pectoralis major (sternocostal portion) is rerouted above (in front of) the conjoint tendon

4 weeks followed by an active rehabilitation after 6 weeks. Strengthening exercises are delayed but will be advised after 3 months.

26.3 Discussion

Anterosuperior rotator cuff tears and isolated subscapularis tears are less common than the isolated superior or posterosuperior ones [2, 3]. Nonetheless, large isolated tears of the subscapularis, or tears of its proximal half that progress so much that they will involve the supraspinatus, usually result in an anterosuperior escape of the humeral head that precludes proper active elevation of the shoulder. When this occurs, it is thought to be due to an imbalance of the anteroposterior couple of forces implied by the rotator cuff to stabilize the joint's center of rotation [16, 31, 32]. Contributing to the patient's dysfunction and pain is the concomitant lesion to the biceps pulley that leads to instability of its tendon. That happens because the anterior fibers of the supraspinatus and the proximal fibers of the subscapularis are major components of the proximal humerus' transverse ligament.

Several tendinous transfers for irreparable anterosuperior rotator cuff tears have been studied, including the ones from the pectoralis major, pectoralis minor, and latissimus dorsi (with or without the teres major). In 1997, Wirth and Rockwood [20] originally described and published the pectoralis major tendon transfer anterior to the conjoint tendon. In the year 2000, Resch et al. [21] modified this technique, transferring only the superior two-thirds of the tendon under the conjoint tendon. In the following years, two other modifications were proposed and the results thereof published. Klepps et al. advocated transferring the entire pectoralis major tendon posterior to the conjoint tendon [29], while Warner et al. would only transfer its inferior (sternal head) portion, which was kept posterior to the clavicular portion and anterior to the conjoint tendon [33].

Gerber et al. [34] proposed to perform a double tendon transfer in patients: they transferred through an extended deltopectoral approach the teres major to the inferior part of the lesser tuberosity and the sternal portion of the pectoralis major to the superior part of the lesser tuberosity. There was no significant difference in terms of pain, mobility, and strength between a single and a double transfer in 11 and nine patients.

It has been reported that, from a biomechanical point of view, the sternal head is superior to restore internal rotation [27]. Moreover, the harvested tendon can be rerouted to the lesser tuberosity underneath [20, 24, 35] or over [20, 33, 36] the conjoint tendon. It has been shown that the rerouting underneath the conjoint tendon results in a better biomechanical effect than a transfer

above the conjoint tendon (Fig. 26.4). Regarding those differences, keeping the transfer posterior to the conjoint tendon results in greater loss of external rotation (approximately 25° of postoperative external rotation loss). Nevertheless, no technique has so far proved its clinical superiority (Fig. 26.5).

The results published by Elhassan et al. [33] in the 2008 study are very similar to the findings of other authors and can be used to summarize the findings from different pectoralis major transfer techniques. They retrospectively analyzed a subgroup of 11 patients with irreparable subscapularis tears operated by the technique previously described by Warner et al. [16] with a mean follow-up of 57 months (range, 44–82). The mean pain score decreased from 7.9 (6 to 10) to 4.2 (0 to 10) ($p = 0.01$). The mean constant score improved from 28.7 (20 to 42) to 52.3 (24 to 78) ($p = 0.01$). The belly-press sign remained positive in all patients, and none had a normal lift-off test.

The functional outcomes are unpredictable because the line of pull generated by all the aforementioned techniques differs too much from the original subscapularis' line of pull, i.e., the pectoralis major origin is anterior to the thorax, while the subscapularis origin is posterior (Fig. 26.4). Indeed, Kany et al. [37] found that regardless of which part of the pectoralis major muscle or position to the conjoint tendon is used, the lines of pull between the transferred tendon and the original subscapularis are almost at a 90° angle to one another. Similarly, Elhassan et al. [38] supposed that changing the vector of the transferred PM (posterior to the conjoint tendon) would not change the direction of its line of action, especially because in internal rotation the lesser tuberosity becomes very medial, and therefore the conjoint tendon would not be able to properly function as a pulley.

Nevertheless, Moroder [39] has shown that pectoralis major tendon transfer resulted in a significant clinical improvement, especially with regard to pain and internal rotation, which was maintained 10 years after surgery. Despite long-term radiographic progression of cuff arthropathy, patient satisfaction remained high over the time, with only a minimal requirement for revision with reverse shoulder arthroplasty. The complication rate was low, but care must be taken to prevent nerve injury when performing pectoralis major tendon transfer.

This is the reason why, up to now, alternative transfers such as latissimus dorsi (which originates posteriorly to the thoracic wall and reproduces the subscapularis' original line of pull) are currently suggested and proposed but are still in infancy [37, 38, 40].

26.4 Conclusion

Irreparable rotator cuff tears in active young patients are still challenging conditions to treat. In case of irreparable isolated rupture of the subscapularis tendon or combined with the supraspinatus tear, a transfer of pectoralis major permits a long-term pain relief improving function but fails to increase the strength of the shoulder. Different techniques have been developed, but none of them has yet proved to be the best one. Other better biomechanical transfers have been recently proposed, but the pectoralis major transfer remains the gold standard regarding long-term follow-up.

References

1. Tashjian RZ. Epidemiology, natural history, and indications for treatment of rotator cuff tears. Clin Sports Med. 2012;31:589–604.
2. Gerber C, Krushell RJ. Isolated rupture of the tendon of the subscapularis muscle. Clinical features in 16 cases. J Bone Joint Surg Br. 1991;73:389–94.
3. Gerber C, Hersche O, Farron A. Isolated rupture of the subscapularis tendon. J Bone Joint Surg Am. 1996;78:1015–23.
4. Goutallier D, Postel JM, Bernageau J, Lavau L, Voisin MC. Fatty muscle degeneration in cuff ruptures. Pre- and postoperative evaluation by CT scan. Clin Orthop. 1994;304:78–83.
5. Bokor DJ, Hawkins RJ, Huckell GH, Angelo RL, Schickendantz MS. Results of nonoperative management of full-thickness tears of the rotator cuff. Clin Orthop. 1993;294:103–10.
6. Levy O, Mullett H, Roberts S, Copeland S. The role of anterior deltoid reeducation in patients with massive irreparable degenerative rotator cuff tears. J Shoulder Elb Surg. 2008;17:863–70.

7. Hansen ML, Otis JC, Johnson JS, Cordasco FA, Craig EV, Warren RF. Biomechanics of massive rotator cuff tears: implications for treatment. J Bone Joint Surg Am. 2008;90:316–25.
8. Boileau P, Baqué F, Valerio L, Ahrens P, Chuinard C, Trojani C. Isolated arthroscopic biceps tenotomy or tenodesis improves symptoms in patients with massive irreparable rotator cuff tears. J Bone Joint Surg Am. 2007;89:747–57.
9. Walch G, Edwards TB, Boulahia A, Nové-Josserand L, Neyton L, Szabo I. Arthroscopic tenotomy of the long head of the biceps in the treatment of rotator cuff tears: clinical and radiographic results of 307 cases. J Shoulder Elb Surg. 2005;14:238–46.
10. Burkhart SS, Athanasiou KA, Wirth MA. Margin convergence: a method of reducing strain in massive rotator cuff tears. Arthroscopy. 1996;12:335–8.
11. Gartsman GM. Massive, irreparable tears of the rotator cuff. Results of operative debridement and subacromial decompression. J Bone Joint Surg Am. 1997;79:715–21.
12. Kim S-J, Lee I-S, Kim S-H, Lee W-Y, Chun Y-M. Arthroscopic partial repair of irreparable large to massive rotator cuff tears. Arthroscopy. 2012;28:761–8.
13. Iagulli ND, Field LD, Hobgood ER, Ramsey JR, Savoie FH. Comparison of partial versus complete arthroscopic repair of massive rotator cuff tears. Am J Sports Med. 2012;40:1022–6.
14. Berth A, Neumann W, Awiszus F, Pap G. Massive rotator cuff tears: functional outcome after debridement or arthroscopic partial repair. J Orthop Traumatol. 2010;11:13–20.
15. Gerber C. Latissimus dorsi transfer for the treatment of irreparable tears of the rotator cuff. Clin Orthop. 1992;(275):152–60.
16. Warner JJ. Management of massive irreparable rotator cuff tears: the role of tendon transfer. Instr Course Lect. 2001;50:63–71.
17. Mihata T, Lee TQ, Watanabe C, Fukunishi K, Ohue M, Tsujimura T, et al. Clinical results of arthroscopic superior capsule reconstruction for irreparable rotator cuff tears. Arthroscopy. 2013;29:459–70.
18. Mihata T, Watanabe C, Fukunishi K, Ohue M, Tsujimura T, Kinoshita M. Arthroscopic superior capsular reconstruction restores shoulder stability and function in patients with irreparable rotator cuff tears: a prospective study (SS-15). Arthroscopy. 2011;27:e36–7.
19. Mulieri P, Dunning P, Klein S, Pupello D, Frankle M. Reverse shoulder arthroplasty for the treatment of irreparable rotator cuff tear without glenohumeral arthritis. J Bone Joint Surg Am. 2010;92:2544–56.
20. Wirth MA, Rockwood CA Jr. Operative treatment of irreparable rupture of the subscapularis. J Bone Joint Surg Am. 1997;79:722–31.
21. Resch H, Povacz P, Ritter E, Matschi W. Transfer of the pectoralis major muscle for the treatment of irreparable rupture of the subscapularis tendon. J Bone Joint Surg Am. 2000;82:372–82.
22. Galatz LM, Connor PM, Calfee RP, Hsu JC, Yamaguchi K. Pectoralis major transfer for anterior-superior subluxation massive rotator cuff insufficiency. J Shoulder Elb Surg. 2003;12:1–5.
23. Jost B, Puskas GJ, Lustenberger A, Gerber C. Outcome of pectoralis major transfer for the treatment of irreparable subscapularis tears. J Bone Joint Surg Am. 2003;85:1944–51.
24. Klepps SJ, Goldfarb C, Flatow E, Galatz LM, Yamaguchi K. Anatomic evaluation of the subcoracoid pectoralis major transfer in human cadavers. J Shoulder Elb Surg. 2001;10:453–9.
25. Aldridge JM III, Atkinson TS, Mallon WJ, Durham B. Combined pectoralis major and latissimus dorsi tendon transfer for massive rotator cuff deficiency. J Shoulder Elb Surg. 2004;13:621–9.
26. Chaffai MA, Mansat M. Anatomic bases for the reconstruction of a musculotendinous flap derived from the pectoralis major muscle. Surg Radiol Anat. 1988;10:273–82.
27. Jennings GJ, Keereweer S, Buijze GA, De Beer J, DuToit D. Transfer of segmentally split pectoralis major for the treatment of irreparable rupture of the subscapularis tendon. J Shoulder Elb Surg. 2007;16:837–42.
28. Owens BD, Nelson BJ, Taylor DC. Acute brachial plexus compression after pectoralis major transfer for subscapularis insufficiency. Am J Sports Med. 2008;36:173–5. Epub 2007 Jul 19
29. Klepps SJ, Galatz LM. The treatment of anterior-superior instability with the pectoralis major muscle transfer. Oper Tech Orthop. 2003;13:228–34.
30. Valenti P, Boughebri O, Moraiti C, Dib C, Maqdes A, Amouyel T, Kany J. Transfer of the clavicular or sternocostal portion of the pectoralis major muscle for irreparable tears of the subscapularis. Technique and clinical results. Int Orthop. 2015;39:477–83.
31. Collin P, Matsumura N, Lädermann A, Denard PJ, Walch G. Relationship between massive chronic rotator cuff tear pattern and loss of active shoulder range of motion. J Shoulder Elb Surg. 2014;23:1195–202.
32. Costouros JG, Espinosa N, Schmid MR, Gerber C. Teres minor integrity predicts outcome of latissimus dorsi tendon transfer for irreparable rotator cuff tears. J Shoulder Elb Surg. 2007;16:727–34. Epub 2007 Nov 5
33. Elhassan B, Ozbaydar M, Massimini D, Diller D, Higgins L, Warner JJ. Transfer of pectoralis major for the treatment of irreparable tears of subscapularis: does it work? J Bone Joint Surg Br. 2008;90:1059–65.
34. Gerber A, Clavert P, Millet PJ, Holovacs TF, Warner JJP. Split pectoralis major and teres major tendon transfers for reconstruction of irreparable tears of the subscapularis. Tech Shoulder Elb Surg. 2004;5:5–12.
35. Konrad GG, Sudkamp NP, Kreuz PC, Jolly JT, McMahon PJ, Debski RE. Pectoralis major tendon transfers above or underneath the conjoint tendon in subscapularis-deficient shoulders. An in vitro biomechanical analysis. J Bone Joint Surg Am. 2007;89:2477–84.

36. Vidil A, Augereau B. Transfer of the clavicular portion of the pectoralis major muscle in the treatment of irreparable tears of the subscapularis muscle. Rev Chir Orthop. 2000;86:835–43.
37. Kany J, Guinand R, Croutzet P, Valenti P, Werthel JD, Grimberg J. Arthroscopic-assisted latissimus dorsi transfer for subscapularis deficiency. Eur J Orthop Surg Traumatol. 2016;26:329–34.
38. Elhassan B, Christensen TJ, Wagner ER. Feasibility of latissimus and teres major transfer to reconstruct irreparable subscapularis tendon tear: an anatomic study. J Shoulder Elb Surg. 2014;23:492–9.
39. Moroder P, Schulz E, Mitterer M, Plachel F, Resch H, Lederer S. Long-term outcome after pectoralis major transfer for irreparable anterosuperior rotator cuff tears. J Bone Joint Surg Am. 2017;99:239–45. https://doi.org/10.2106/JBJS.16.00485.
40. Mun SW, Kim JY, Yi SH, Baek CH. Latissimus dorsi transfer for irreparable subscapularis tendon tears. J Shoulder Elb Surg. 2018;27:1057–64. Epub 2018 Feb 3

27 Tendon Transfer for Anterosuperior Cuff: Latissimus Dorsi Transfer

Viktoras Jermolajevas

27.1 Introduction

Rupture of subscapularis (SUBS) tendon leads to loss of active internal rotation, horizontal imbalance, and pain. Isolated ruptures are rare and consists of about 13% of all SUBS ruptures [1]. Extended ruptures are more common; an associated rupture of supraspinatus leads to vertical imbalance and proximal migration of humeral head. Long-lasting ruptures are difficult to treat due to bad tendon mobility, muscle fatty degeneration, and atrophy. Direct tendon repair is sometimes not possible, and even if repair would be accomplished, a SUBS muscle will not generate enough contraction power to centralize a humeral head. Several options for irreparable subscapularis tears are described. Pectoralis major or minor transfers for irreparable SUBS were common, and recently latissimus dorsi tendon (LDT) and teres major transfers gain popularity. The anterior LD tendon transfer appears to replicate the line of subscapularis pullout more anatomically and follows basic rules of tendon transfers. The idea of latissimus dorsi anterior transfer (aLDT) was published by B. Elhassan in 2014 [2]. This cadaveric study shows that the aLDT provides a more anatomic transfer option compared to the pectoralis major tendon transfer for subscapularis insufficiency. The risk of nerve compression was found to be low when transferring the latissimus dorsi alone to the lesser tuberosity. This aLDT transfer was done open in 17 patients with not yet published clinical results [3]. Clinical study with 1 year follow-up done by S. W. Mun recently showed good clinical results. 24 patients operated by open surgery, and graft fixation with two knotless anchors and transosseous sutures on lesser tuberosity were performed [4]. J. Kany developed arthroscopic-assisted technique and published preliminary results of five patients in 2016 [5]. Our technique follows the same rules of above mentioned open and arthroscopic-assisted techniques but in full arthroscopic manner.

27.2 Indications

Indication for anterior latissimus dorsi transfer is irreparable total or 2/3 of SUBS tendon tear, with Goutallier grade III or IV fatty infiltration [6] (Fig. 27.1), which could be classified as Lafosse type V SUBS rupture [7]. Any kind of infection or arthritis more than Hamada stage III is a contraindication to do this surgery [8].

V. Jermolajevas (✉)
UAB "Ortogida", Klaipėda, Lithuania
e-mail: jermola@ortogida.lt

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_27

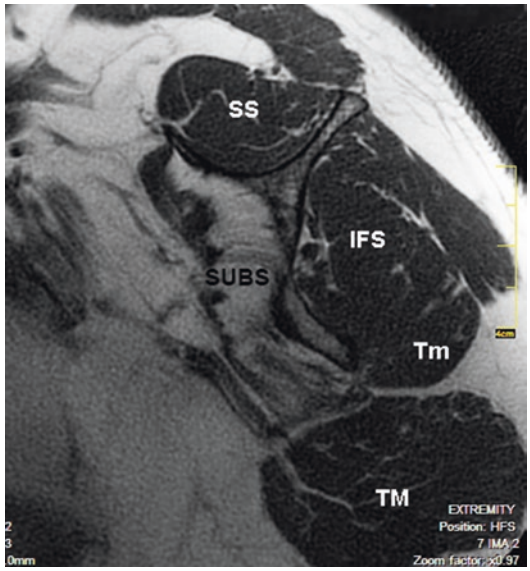


Fig. 27.1 Fatty infiltration grade IV of subscapularis muscle while other muscles are well preserved (left shoulder, T1 sagittal view). (*SUBS* subscapularis, *SS* supraspinatus, *IFS* infraspinatus, *Tm* teres minor, *TM* teres major muscles)

27.3 Technique

The patient is operated on in the beach-chair position. The shoulder is draped in the same fashion as for conventional shoulder surgery, leaving at least 6 cm of free space below the axillary fold. Axilla and optionally top of the shoulder are covered with plastic adhesive drape to reduce possible suture contamination with *Corynebacterium* (*Propionibacterium*) *acnes* [9]. Routine arthroscopy portals comprise the posterior (P), anterosuperior (AS), posterolateral (PL), and anterolateral (AL) portals, with an additional suprapectoral portal (sP) for arthroscopic LDT release (Fig. 27.2). Simple arm traction (3–5 kg with a nonelastic tape) is used to slightly distract the subacromial space as in regular rotator cuff repair. An additional suspension band is placed around the elbow to maintain the arm in overhead abduction during the axillar LDT release, extrusion, and suturing.

The operation can be divided into four steps.

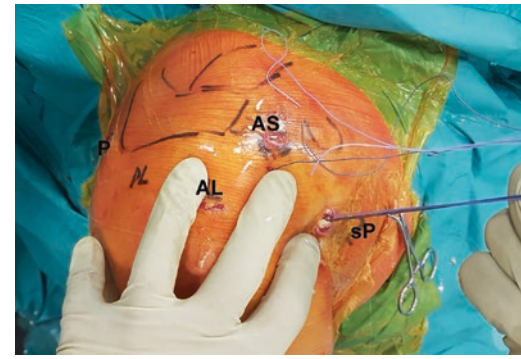


Fig. 27.2 Arthroscopic portals (right shoulder with patient in beach-chair position). Latissimus dorsi tendon with sutures is visible through suprapectoral portal (sP) (*P* posterior, *PL* posterolateral, *AL* anterolateral, *AS* anterosuperior portals)

27.3.1 SUBS Tendon Release and Partial Cuff Repair

The arthroscopy begins from posterior (P) portal. Standard AS and AL portals are created, and SUBS tendon is inspected. In case of isolated SUBS rupture, the tendon is retracted to glenoid rim, and conjoined tendon with coracoid is clearly visualized. If SUBS ruptured together with SS, a "comma sign" should be founed to define superior border of SUBS [10]. Resection of rotator interval is performed with care to preserve an intact junction of SS and SUBS. After initial mobilization, viewing portal is changed to AS portal. Looking distally, following the long.

head of the biceps tendon in its groove. Shaving is performed through the AL portal, until the superior border of the pectoralis major tendon (PM) is clearly visible. Medial to the PM insertion, the anterior humeral circumflex vessels ("three sisters") are identified, marking the inferior border of the subscapularis tendon. Just underneath, the superior border of the LDT is visible. At this point, the suprapectoral portal (sP), just above the PM, is established. Skin incision for this portal is made just above anterior angle of axillar crease.

Viewing portal is changed to AL, and complete mobilization of SUBS tendon through sP portal is done. To obtain the tension-free repair, it is necessary to perform superior, posterior, and anterior releases. The superior part is released from any adhesions as far as to the medial side of coracoid base. Posteriorly to SUBS, any capsule adhesions are resected. Anteriorly, subcoracoid bursa and space between SUBS and conjoined tendon are opened. The anterior release is more dangerous. The anterior side of the SUBS often adheres to the conjoined tendon and coracobrachialis muscle and requires to be released. Axillar nerve (Ax), radial nerve (Rn), and SUBS small neurovascular structures should be identified and protected.

Final SUBS tendon assessment is made. The definitive decision to perform latissimus dorsi (LD) transfer or not is done. Factors for decision are tendon mobility and thickness, fatty infiltration defined in T1 sagittal view of MRI. At this stage, if technically possible, SUBS repair at least partially should be done.

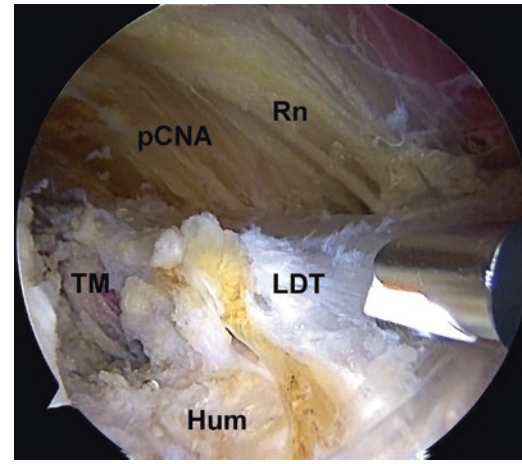


Fig. 27.3 Arthroscopic view from anterosuperior portal (right shoulder, patient in beach-chair position). VAPR in the suprapectoral portal. Superior-lateral edge of latissimus dorsi tendon is released (*Hum* humerus, *TM* teres major, *LDT* latissimus dorsi tendon, *pCNA* posteriors cutaneous nerve of arm, *Rn* radial nerve)

27.3.2 LDT Proximal Release

This release is similar to technique described in arthroscopic-assisted latissimus dorsi transfer for posterior cuff deficiency [11].

Visualization: camera is in AS portal, working portal- suprapectoral (sP). The release of 1 cm of the superior part of the PM tendon is performed. This allows the surgeon to bluntly prepare some space between three structures: the conjoined tendon anteriorly, the PM laterally, and the LDT posteriorly. Shaving is performed inferiorly to the circumflex vessels (three sisters) until complete exposure of the LDT fibers, running medial to lateral, is achieved. The limit for inferior dissection is radial nerve (Rn) and 1 cm medially—posterior cutaneous nerve of the arm (pCNA). They found inferiorly, crossing the borders of the LDT and TM tendon 3–4 cm medial to their humeral insertion (Fig. 27.3). More medial and inferior, the band of tissue representing the remnant of the dorsoepitrochlearis brachii of apes is present. It connects LD tendon and long head of triceps. This tissue band is mandatory to release but is not always possible through sP portal. Tendon dissection is continued as far medially as the arthroscope and shaver length allow. Then, distal release from the humerus at the crista tuberculi minoris is performed. The tendon is reflected medially for easier grasping later. A standard urinary catheter (Foley) inserted from the sP portal is left in this location; its balloon is inflated for future LDT passage.

27.3.3 LDT Final Release and Tendon Passage

The arm is removed from traction and is fully abducted. Traction band holds the arm in the overhead position using a solid anesthesiology frame around the patient's head for fixation. Two axillar portals to complete LDT release are created: medial axillar portal (mAx), in the middle

of axilla and 3 cm below axillar crease, and posterior axillar portal (pAx), in the posterior corner at the same level. Scope introduces into pAx portal. Latissimus dorsi tendon and its distal end is identified and grasped with Kocher or grasped from mAx portal and extruded. The tendon edges are then grasped with two clamps. VAPR electrode (Mitek) is introduced in the same mAx portal, and final release of tissue band to long head of triceps and subcutaneous tissue is continued (Fig. 27.4).

Tendon release facilitates the placement of two pairs of Krackow stitches along the medial and lateral border using two different colored sutures. The LD muscle is released proximally as full muscle belly, and neurovascular bundle is visualized. Care must be taken during anterior muscle belly release because the neurovascular bundle is anterior and 6–8 cm proximal to the musculotendinous junction (Fig. 27.5).

The Foley catheter balloon is extracted from the mAx portal. Sutures from the LD are attached to the catheter that serves as a shuttle relay. The LDT is then passed into the anterior space with the catheter pulled out through the sP portal. It is important to control the sutures to avoid twisting the tendon and define which one is medial or lateral. Final release of LD belly if necessary is done from subcutaneous tissue, until LD tendon edge is visible in the sP portal (Fig. 27.2). The axillary portals are closed in a standard manner. The arm is released from the overhead position and is left in a position of flexion with 3–5 kg of traction as in the beginning of surgery.

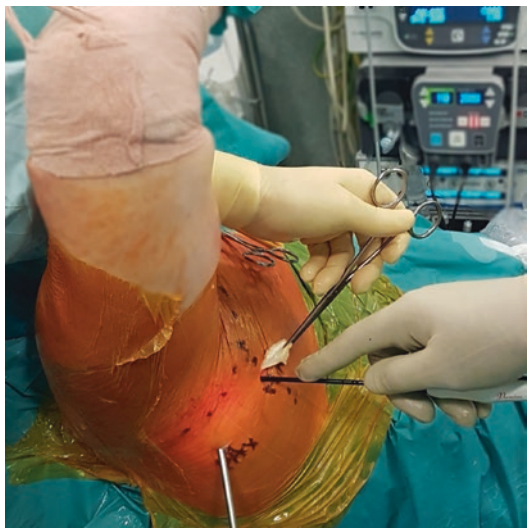


Fig. 27.4 LDT final release through axillary portals (right shoulder, patient in beach-chair position, arm in ABD position). Arthroscope in posterior axillary portal, VAPR, and the end of latissimus dorsi tendon in medial axillary portal

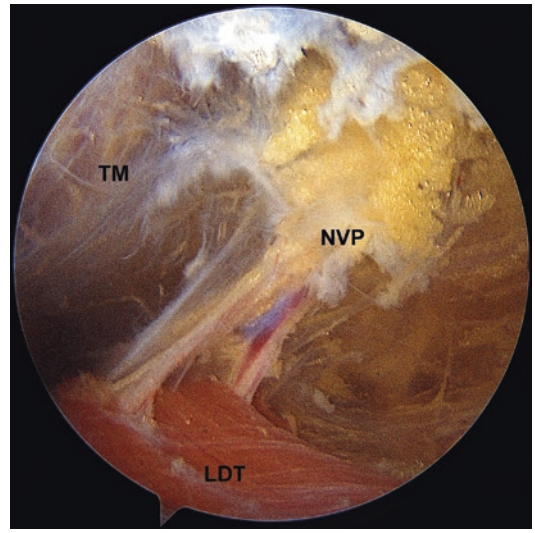


Fig. 27.5 Arthroscopic view from posterior axillary portal (right shoulder, patient in beach-chair position, arm in ABD position) (*TM* teres major, *LDT* latissimus dorsi muscle belly, *NVP* neurovascular pedicle of thoracodorsal vessels and nerve)

27.3.4 LDT Fixation to Lesser Tuberosity

Viewing through sP portal, working in AL, AS portals. Sutures withdraw from AS portal. Lateral pair of sutures is kept in tension, while medial sutures are used for fixation. If release was done properly, it is not difficult to fix medial sutures above the bicipital groove with Healix Advance Knotless (Mitek) or Versalok (Mitek) anchors. After fixing medial pair of sutures, tension from lateral pair of sutures is released. Slight internal rotation will bring greater tuberosity closer to AS portal. The second anchor is used for fixation on anterior edge of greater tuberosity. Axillar nerve should be inspected, and contact with latissimus

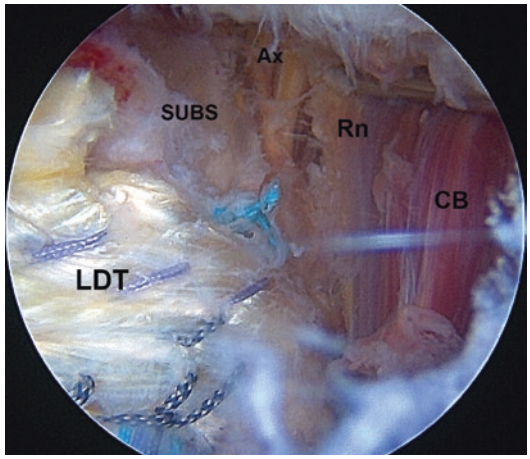


Fig. 27.6 Final view through suprapectoral portal of anteriorly transposed latissimus dorsi tendon (right shoulder, patient in beach-chair position). Subscapularis remnant tendon behind LDT is visible. Note relation and distance of transfer to axillary nerve (*LDT* latissimus dorsi tendon, *SUBS* remnant of subscapularis tendon, *Ax* axillary nerve, *Rn* radial nerve, *CB* coracobrachialis muscle)

dorsi should be documented. The personal experience: more lateral and shifted on greater tuberosity fixation points had less possibility of axillar nerve contact and less probability of compression with internal rotation and abduction of the arm (Fig. 27.6).

27.4 Postoperative Management

The shoulder was placed in a brace with the arm at the belly in an internal rotation position for 6 weeks postoperatively. Only elbow and wrist exercises are allowed. Passive assisted exercises were started at 6 weeks after surgery. Gradual return to daily activities, any arm movement without pain, is allowed. Weight lifting up to 5 kg or support of body weight is allowed after 3 months. Gentle strengthening exercises were started and progressed slowly during the next 3 months. Return to full activity, heavy manual working, is allowed 4–6 months after surgery.

References

1. Lanz U, Fullick R, Bongiorno V, Saintmard B, Campens C, Lafosse L. Arthroscopic repair of large subscapularis tendon tears: 2- to 4-year clinical and radiographic outcomes. Arthroscopy. 2013;29:1471–8.
2. Elhassan B, Christensen TJ, Wagner ER. Feasibility of latissimus and teres major transfer to reconstruct irreparable subscapularis tendon tear: an anatomic study. J Shoulder Elb Surg. 2014;23:492–9.
3. Elhassan BT. Feasibility of latissimus and teres major transfer to reconstruct irreparable subscapularis tendon tear: an anatomic study. J Shoulder Elb Surg. 2015;24:e102–3.
4. Mun SW, Kim JY, Yi SH, Baek CH. Latissimus dorsi transfer for irreparable subscapularis tendon tears. J Shoulder Elb Surg. 2018;27:1057–64.
5. Kany J, Guinand R, Croutzet P, Valenti P, Werthel JD, Grimberg J. Arthroscopic-assisted latissimus dorsi transfer for subscapularis deficiency. Eur J Orthop Surg Traumatol. 2016;26:329–34.
6. Goutallier D, Postel JM, Bernageau J, Lavau L, Voisin MC. Fatty muscle degeneration in cuff ruptures. Pre- and postoperative evaluation by CT scan. Clin Orthop. 1994:78–83.
7. Lafosse L, Lanz U, Saintmard B, Campens C. Arthroscopic repair of subscapularis tear: surgical technique and results. Orthop Traumatol Surg Res. 2010;96:S99–S108.
8. Hamada K, Fukuda H, Mikasa M, Kobayashi Y. Roentgenographic findings in massive rotator cuff tears. A long-term observation. Clin Orthop. 1990:92–6.
9. Yamakado K. Propionibacterium acnes suture contamination in arthroscopic rotator cuff repair: a prospective randomized study. Arthroscopy. 2018;34:1151–5.
10. Lo IKY, Burkhart SS. The comma sign: an arthroscopic guide to the torn subscapularis tendon. Arthroscopy. 2003;19:334–7.
11. Jermolajevas V, Kordasiewicz B. Arthroscopically assisted latissimus dorsi tendon transfer in beach-chair position. Arthrosc Tech. 2015;4:e359.

Subacromial Spacer

28

Ladislav Kovačič and Vladimir Senekovič

28.1 Introduction

Implantation of the biodegradable spacer in the subacromial space in patients with massive irreparable rotator cuff tear is one of the valuable newest treatment options among the possibilities for surgical management, which arose in the last years in the clinical practice [1, 2]. Management of those patients is frequently individualized according to their clinical presentation and specific needs. Patients expect from surgical procedure reduction in pain and improvement in range of motion (ROM). Those with predominant pain problem are especially good candidates for the presented surgical treatment. The procedure is simple, safe, and less invasive with minimal complication rate as it was shown in latest systematic review, which synthesized and reported early clinical and radiographic outcomes associated with subacromial spacer use in patient with massive irreparable rotator cuff tears [3].

L. Kovačič (✉)
Department of Traumatology, University Medical Centre Ljubljana, Ljubljana, Slovenia
e-mail: ladislav.kovacic@kclj.si

V. Senekovič
Arbormea, Ljubljana, Slovenia
e-mail: vladimir.senekovic@arbormea.com

28.2 Biomechanical Background

Following the biomechanics of force couples that results in resultant movement without resultant force, the acting mechanism of the subacromial spacer could be explained. Namely, the deltoid and supraspinatus muscles act as the coronal force couple, compressing the humeral head to the glenoid in abduction, thus creating stable fulcrum allowing motion of the humerus around the glenoid. On the contrary, the rotator cuff disruption compromises concavity compression and alters glenohumeral load structure and direction [4, 5]. The pathomechanics involved with the tear typically results in superior humeral head migration. To restore normal kinematics in the patients with posterosuperior tear, greater forces are required by both the deltoid and the intact muscle units of the rotator cuff to achieve stable joint during abduction and to prevent superior subluxation of the humeral head [6]. Increase in muscle strain of those muscles may be the reason for shoulder pain. Furthermore, increased forces required to maintain the center of rotation contribute to the anterior and posterior tear propagation, particularly if the remaining tendons are of poor quality [7]. Additionally, with initiation of abduction and subsequent superior migration of the humeral head, increased impingement between the greater tuberosity and undersurface of the acromion can occur [8, 9]. Subacromial friction during shoulder abduction can be additional reason for shoulder pain.

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_28

The implantation of a spacer in the subacromial space should help to restore force couples of the glenohumeral joint in coronal plane, to maintain humeral head in the stable position during dynamic movement, and to decrease subacromial friction. These are biomechanical grounds of subacromial spacer effect in the case of rotator cuff tears leading to pain reduction and improvement in shoulder function by lowering the humeral head, reducing the forces required to achieve abduction, and facilitating humeral gliding against acromion [2, 10]. On the contrary, the subacromial spacer cannot influence the force couples in transverse plane; thus, subscapularis tendon and teres minor should be preserved or repaired.

28.3 Implant and Arthroscopic Surgical Procedure

The subacromial spacer is made of a copolymer of poly(L-lactide-co-ε-caprolactone) that is biodegradable and totally dissipates within up to 12 months. The unique cold molding technology enables manufacturing in the shape of balloon, suitable to be implanted between the acromion and the humeral head. The device attempts to restore painless shoulder biomechanics by lowering the humeral head, stabilizing the center of rotation, and decreasing subacromial friction [10].

Standard shoulder arthroscopy in a beach-chair or lateral decubitus position is performed. After debridement and bursectomy, the rotator cuff is assessed for reparability. The tendon mobility to the footprint region and quality of the tendon are assessed. Once deemed irreparable, the correct size of the subacromial spacer is selected measuring the distance from the lateral border of the greater tuberosity to approximately 1 cm medial to the glenoid apex (Fig. 28.1a). If the long head of biceps is still present, biceps tenotomy is advised. Smoothening of the undersurface of the acromion is advisable as well, but coracoacromial arch should be preserved. The rolled-up spacer

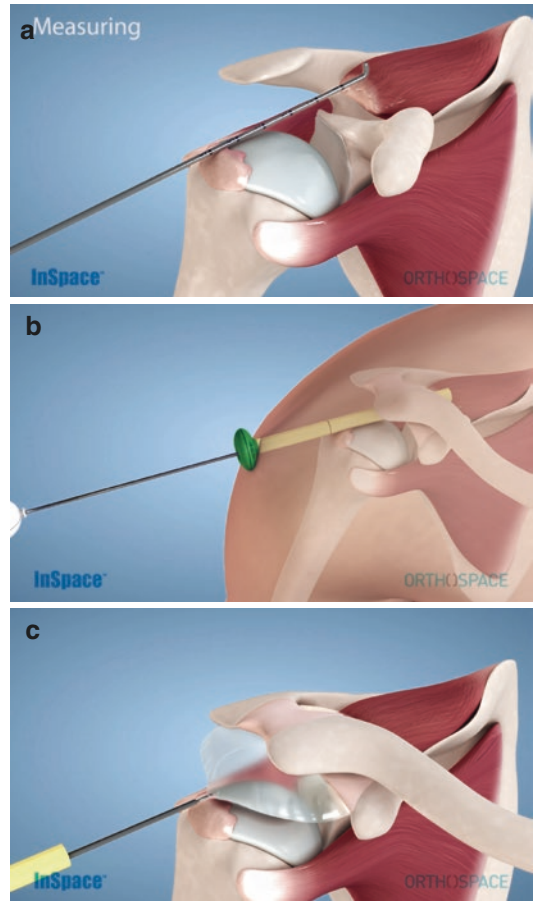


Fig. 28.1 Schematic illustration of the subacromial biodegradable spacer (InSpace™) implantation (with permission of Orthospace company). (**a**) Measurement of the distance from lateral border of the greater tuberosity to approximately 1 cm medially to the glenoid apex, which will allow appropriate implant size selection. (**b**) Insertion of the InSpace™ spacer rolled up inside the introducer through the lateral portal. (**c**) Inflation of the balloon-shaped spacer with saline and deployment in the subacromial space

of correct size is then inserted through the lateral portal and inflated with a sterile saline to the recommended volume depending on the selected implant size (Fig. 28.1b). The appropriate inflation volume is being adjusted by inducing passive full range of shoulder movements prior of spacer sealing. Once the volume and position are optimized, the device is sealed utilizing the mechanism, which is part of the deployment system (Fig. 28.1c).

28.4 Clinical Outcomes

To the end of 2018, several scientific reports are available in the literature describing the results of patients with massive irreparable rotator cuff tear treated by subacromial spacer implantation [2, 10–18]. These studies report the mean preoperative and postoperative Constant scores as 22,5–44,8 preoperatively and 51,4–69,5 postoperatively at a minimum of 12 months follow-up (Table 28.1). In detail, significant decrease in pain, increase in range of motion, and increased ability to perform activities of daily living are reported. On the other hand, there is limited improvement in strength of abduction. Results in available literature and surgeons' experience show that results for subacromial spacer improve over time. In the study with the longest follow-up, all the parameters of the Constant score improved over 5 years period (Fig. 28.2) [10]. The overall reported results are thus comparable to the other treatment options in patients with this kind of pathology [19]. Interestingly, patients included in the subacromial spacer studies usually have more significant shoulder function impairment compared to patients treated by other techniques as has been shown by lower Constant score preoperatively.

The influence of simultaneous surgical acts as debridement, subacromial decompression, acromioplasty, and surgical treatment of biceps tendon together with subacromial spacer implantation is frequently discussed. Fluoroscopy-guided implantation of the spacer without performing surgical decompression or addressing biceps tendon shows similar results of improvement in Constant score and its subcategories like in the studies where the spacer was implanted during arthroscopic procedure [12]. Adding to this, arthroscopic debridement of the joint is usually minimal, furthermore limited rather than complete bursectomy is advised and performed in order to avoid subacromial spacer migration in the postoperative period. Six studies describe the status of the long head of biceps tendon (LHB) [2, 11, 13–16]. Intraoperatively, the LHB was intact in 69,8% and ruptured in 30,2% of patients. The results of the studies show that preoperative status of LHB did not affect the postoperative outcome measured by Constant score. Furthermore, the study that compares subacromial spacer implantation with and without biceps tenotomy reported no difference among subgroups of the patients [14].

Inconsistent outcome of the subacromial spacer implantation was published in one study [17]. Authors reported improvement for more than 10 points in Constant score only in 6 out of 15 patients. At 2 years follow-up, Constant score was improved from 35,0 to 53,5. However, 9 out of 15 patients showed no improvement in 2 years follow-up period, and 5 of them have been revised to reverse shoulder arthroplasty. This is somehow different to the previous experience.

Table 28.1 Published studies until early 2018 about subacromial spacer in massive rotator cuff tears

Study	No. of patients included	Male/female ratio	Age	Constant score preop	Constant score at follow-up	Follow-up
Senekovic [2]	20	11 m/9f	70,5 (54–85)	33,4	65,4	3 y
Gervasi [12]	15	7 m/8f	74,6 (±6,5)	31,9	62	24 m
Senekovic [10]	20	11 m/9f	70,5 (54–85)	33,4	67,4	5 y
Holschen [13]	12	6 m/6f	62,4	36,8	69,5	22 m
Maman [14]	42	na	na	36	67	12 m
Piekaar [15]	44	25 m/19f	66 (63,7–68,3)	37,1	60,2	12 m
Deranlot [11]	37	15 m/22f	69,8 (53–84)	44,8	76	3 y
Ricci [16]	30	na	na	39,9	65,4	12 m
Ruiz Iban [17]	15	4 m/11f	69,4 (60–80)	35,0	51,4	24 m
Yallapragada [18]	14	10 m 4f	76,2 (70–85)	22,5	51,4	12,6 m

na none available, *m* months, *y* years

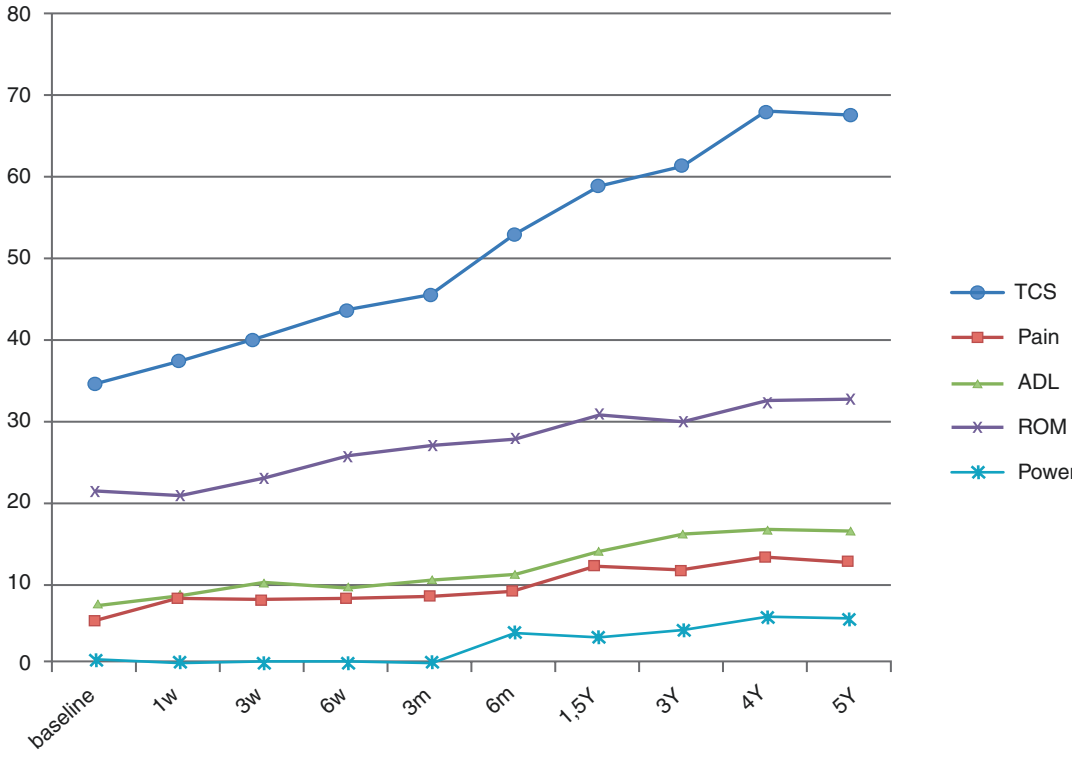


Fig. 28.2 Clinical results following biodegradable spacer insertion presenting total Constant score (TCS) and variables of the TCS: pain, activity of daily living (ADL), range of motion (ROM), and power of abduction. Change in mean scores from baseline to 5 years postoperatively is presented as reported in study with the longest follow-up [10]

The reported failure rate and overall complication rate have been minimal in the previous studies. In systematic review including 6 studies, only in 6 out of 200 patients (3%) unsatisfactory outcome or complication was detected [3]. One patient required revision surgery for spacer migration, three patients were converted to reverse total shoulder arthroplasty, and two patients had no improvement in total Constant score.

28.5 Future Directions

Subacromial spacer was first used as a main or single treatment option for patients with irreparable massive rotator cuff tear. The use of the implant was shown to be simple and safe. It is quick procedure in the hand of shoulder surgeon, which does not preclude any additional or further surgical treatment. Recently, the spacer is frequently used as additional or supportive therapy in partial or complete repair of massive rotator cuff tears [20]. While in rotator cuff torn condition, there is significant increase in superior humeral head migration, subacromial spacer can restore the humeral head position similar to the intact condition [21]. Additionally, biomechanical study shows that spacer can restore also functional abduction force [21]. In this setting, implantation of the spacer over the rotator cuff repair could be protective for the repaired construct, especially in the patients where the tendon quality is not as we wish.

Several properties of the device still need to be elucidated. It is uncertain why the positive effect continues beyond the time of spacer degradation period [10]. The possible reason is that spacer helps the patient in the immediate postoperative period to rehabilitate the shoulder and to train the appropriate muscles to establish sufficient efficiency for satisfactory shoulder function. Further research is needed to answer this question.

28.6 Summary

Treatment of the patients with massive rotator cuff tears is based on patient factors and associated pathology. The decision-making process includes personal experience and scientific data. A thorough knowledge of existing treatment options and indications is crucial to achieve best outcomes for the patients. There is a wide variety of patients in terms of rotator cuff tear pattern, functional impairment, and reparability. The subgroup of patients with massive irreparable rotator cuff tear and predominant symptom of pain but relatively preserved active elevation, meaning that there is not pseudoparalytic shoulder, are the best candidates for treatment option with biodegradable subacromial spacer. Good clinical results can be expected in patients with preserved force couple in transverse plane in the absence of shoulder osteoarthritis. The spacer in subacromial space will only influence the force couple in coronal plane by lowering the humeral head, facilitating humeral gliding against the acromion, and reducing subacromial friction during shoulder abduction. The procedure is simple, safe, reliable, and less invasive with minimal complication rate comparing to some other treatment options.

References

1. Savarese E, Romeo R. New solution for massive, irreparable rotator cuff tears: the subacromial "biodegradable spacer". Arthrosc Tech. 2012;1:e69–74. https://doi.org/10.1016/j.eats.2012.02.002.
2. Senekovic V, Poberaj B, Kovacic L, Mikek M, Adar E, Dekel A. Prospective clinical study of a novel biodegradable sub-acromial spacer in treatment of massive irreparable rotator cuff tears. Eur J Orthop Surg Traumatol. 2013;23:311–6. https://doi.org/10.1007/s00590-012-0981-4.
3. Moon AS, Patel HA, Ithurburn MP, Brabston EW, Ponce BA, Momaya AM. Subacromial spacer implantation for the treatment of massive irreparable rotator cuff tears: a systematic review. Arthroscopy. 2019;35:607–14. https://doi.org/10.1016/j.arthro.2018.08.006.
4. Kedgley AE, MacKenzie GA, Ferreira LM, Johnson JA, Faber KJ. In vitro kinematics of the shoulder following rotator cuff injury. Clin Biomech. 2007;22:1068–73.
5. Parsons IM, Apreleva M, Fu FH, Woo SL. The effect of rotator cuff tears on reaction forces at the glenohumeral joint. J Orthop Res. 2002;20:439–46. https://doi.org/10.1016/S0736-0266(01)00137-1.
6. Hansen ML, Otis JC, Johnson JS, Cordasco FA, Craig EV, Warren RF. Biomechanics of massive rotator cuff tears: implications for treatment. J Bone Joint Surg Am. 2008;90:316–25. https://doi.org/10.2106/JBJS.F.00880.
7. Greenspoon JA, Petri M, Warth RJ, Millett PJ. Massive rotator cuff tears: pathomechanics, current treatment options, and clinical outcomes. J Shoulder Elb Surg. 2015;24:1493–505. https://doi.org/10.1016/j.jse.2015.04.005.
8. Nam D, Maak TG, Raphael BS, Kepler CK, Cross MB, Warren RF. Rotator cuff tear arthropathy: evaluation, diagnosis, and treatment. J Bone Joint Surg Am. 2012;94:e34. https://doi.org/10.2106/JBJS.K.00746.
9. Su WR, Budoff JE, Luo ZP. The effect of anterosuperior rotator cuff tears on glenohumeral translation. Arthroscopy. 2009;25:282–9. https://doi.org/10.1016/j.arthro.2008.10.005.
10. Senekovic V, Poberaj B, Kovacic L, Mikek M, Adar E, Markovitz E, Maman E, Dekel A. The biodegradable spacer as a novel treatment modality for massive rotator cuff tears: a prospective study with 5-year follow-up. Arch Orthop Trauma Surg. 2017;137:95–103. https://doi.org/10.1007/s00402-016-2603-9.
11. Deranlot J, Herisson O, Nourissat G, Zbili D, Werthel JD, Vigan M, Bruchou F. Arthroscopic subacromial spacer implantation in patients with massive irreparable rotator cuff tears: clinical and radiographic results of 39 retrospectives cases. Arthroscopy. 2017;33:1639–44. https://doi.org/10.1016/j.arthro.2017.03.029.
12. Gervasi E, Maman E, Dekel A, Cautero E. Fluoroscopy-guided biodegradable spacer implantation using local anesthesia: safety and efficacy study in patients with massive rotator cuff tears. Musculoskelet Surg. 2016;100(Suppl 1):S19–24. https://doi.org/10.1007/s12.306-016-0433-0.
13. Holschen M, Brand F, Agneskirchner JD. Subacromial spacer implantation for massive rotator cuff tears: clinical outcome of arthroscopically treated patients. Obere Extrem. 2017;12:38–45. https://doi.org/10.1007/s11678-016-0386-9.
14. Maman E, Safran O, Beyth S, Mozes G, Dekel A, Michael B, Chechik O, Adar E. Biceps tenotomy does not affect the functional outcomes of patients treated with spacer implantation due to massive irreparable rotator cuff tears. Open Orthop J. 2017;11:1577–84. https://doi.org/10.2174/1874325001711011577.
15. Piekaar RSM, Bouman ICE, van Kampen PM, van Eijk F, Huijsmans PE. Early promising outcome following arthroscopic implantation of the subacromial balloon spacer for treating massive rotator cuff tear. Musculoskelet Surg. 2018;102:247–55. https://doi.org/10.1007/s12306-017-0525-5.
16. Ricci M, Vecchini E, Bonfante E, Micheloni GM, Berti M, Schenal G, Zanetti G, Sambugaro E, Maluta T,

Magnan B. A clinical and radiological study of biodegradable subacromial spacer in the treatment of massive irreparable rotator cuff tears. Acta Biomed. 2017;88:75–80. https://doi.org/10.23750/abm.v88i4-S.6797.

17. Ruiz Ibán MA, Lorente Moreno R, Ruiz Díaz R, Álvarez Sciamanna R, Paniagua Gonzalez A, Lorente Gómez A, Díaz Heredia J. The absorbable subacromial spacer for irreparable posterosuperior cuff tears has inconsistent results. Knee Surg Sports Traumatol Arthrosc. 2018;26:3848–54. https://doi.org/10.1007/s00167-018-5083-3.
18. Yallapragada RK, Apostolopoulos A, Katsougrakis I, Selvan TP. The use of a subacromial spacer-inspace balloon in managing patients with irreparable rotator cuff tears. J Orthop. 2018;15:862–8. https://doi.org/10.1016/j.jor.2018.08.004.
19. Lädermann A, Collin P, Athwal GS, Scheibel M, Zumstein MA, Nourissat G. Current concepts in the primary management of irreparable posterosuperior rotator cuff tears without arthritis. EFORT Open Rev. 2018;3:200–9. https://doi.org/10.1302/2058-5241.3.180002.
20. Szöllösy G, Rosso C, Fogerty S, Petkin K, Lafosse L. Subacromial spacer placement for protection of rotator cuff repair. Arthrosc Tech. 2014;3:e605–9. https://doi.org/10.1016/j.eats.2014.06.017.
21. Singh S, Reeves J, Langohr GDG, Johnson JA, Athwal GS. The subacromial balloon spacer versus superior capsular reconstruction in the treatment of irreparable rotator cuff tears: a biomechanical assessment. Arthroscopy. 2019;35:382–9. https://doi.org/10.1016/j.arthro.2018.09.016.

29 Reverse Total Shoulder Arthroplasty

Bruno Toussaint, Agathe Kling, and Jérôme Bahurel

29.1 Introduction

Massive ruptures are a chapter in the treatment of rotator cuff tears that has always posed a delicate problem. As muscle transfers have made it possible to treat massive rotator cuff tears in young patients, reverse total shoulder arthroplasty (rTSA) brought a relatively simple solution for the elderly patients.

Reverse total shoulder arthroplasty is more reliable than anatomical prostheses with large heads or with a suitable profile. Results of rTSA improve and appear more and more promising over years.

More than massive ruptures, rTSA addresses irreparable rotator cuff tears. There are different situations: massive irreparable rotator cuff tears (MIRCTs) with or without pseudoparalytic aspect and cuff arthropathy with eccentric osteoarthritis (OA) [1–6] (Fig. 29.1).

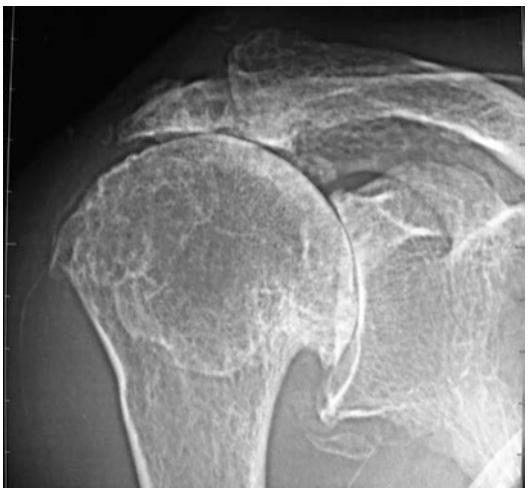


Fig. 29.1 Radiographic appearance of cuff tear arthropathy

29.1.1 What Are the Criteria for Defining Irreparable Rotator Cuff Tears?

The reducibility of the tendons, appreciable preoperatively, is a subjective assessment. Before surgery, muscle quality can be evaluated on the coronal sections at the computed tomography (CT) scan or at the magnetic resonance imaging (MRI), especially fatty infiltration of muscles.

Function is also important, because massive rotator cuff tears are not always with pseudoparalysis. Without pseudoparalysis, rTSA indication must be discussed.

B. Toussaint (✉)
Centre Teomera, Le Grand Saconnex, Geneva, Switzerland

A. Kling · J. Bahurel
Clinique Générale Annecy, Annecy, France

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_29

29.1.2 Does Cuff Arthropathy Always Need an Arthroplasty to Solve the Symptoms?

The indications for rTSA evolved since the modifications made by Grammont [7]. Indeed, the principles laid down by Grammont have made it possible to improve the functional results of the rTSA in a spectacular way.

A very important element is the age of the patients. In the beginning, the age of patients had to be greater than 75 years to minimize the consequences of the unknown outcomes of rTSA.

The first studies showed implant survivorship that dropped after 8 years [6]. Several studies have gradually shown that implant survivorship lengthened beyond 10 years. More recent works show a better longevity of rTSA. This improvement depends on the technical innovation in implant designs and better positioning of the glenoid component. This broadened indication for implanting an rTSA.

Today, it is reasonably possible to implant rTSA in patients under the age of 65 to treat rotator cuff tear. Other studies confirmed that rTSA results are better in the treatment of rotator cuff tear than in other indications. In complex cases, rTSA can be combined with muscle transfer to restore external rotation.

29.1.3 Preoperative Assessment

As with any prosthesis, preoperative evaluation of bone stock is essential. The glenoid side is crucial, as it is important to restore the glenohumeral joint line to meet Grammont's recommendation to place the center of the glenosphere at the *paleoglenoid* surface [7].

It is important to assess the orientation of the glenoid in the frontal and sagittal plane.

Walch et al. [1] classified the glenoid morphology into three types based on the CT scan findings out of 113 patients (Fig. 29.2).

Type A (59%): The humeral head is centered, and the resultant strengths are equally distributed against the surface of the glenoid. Glenoid retroversion average was 11.5°. The erosion may be minor (type A1, 43%) or major (type A2, 16%) marked by a central erosion that leads to a centered glenoid cupula. In advanced cases, the humeral head protrudes into the glenoid cavity.

Type B (32%): The humeral head is subluxated posteriorly, and the distributed loads are

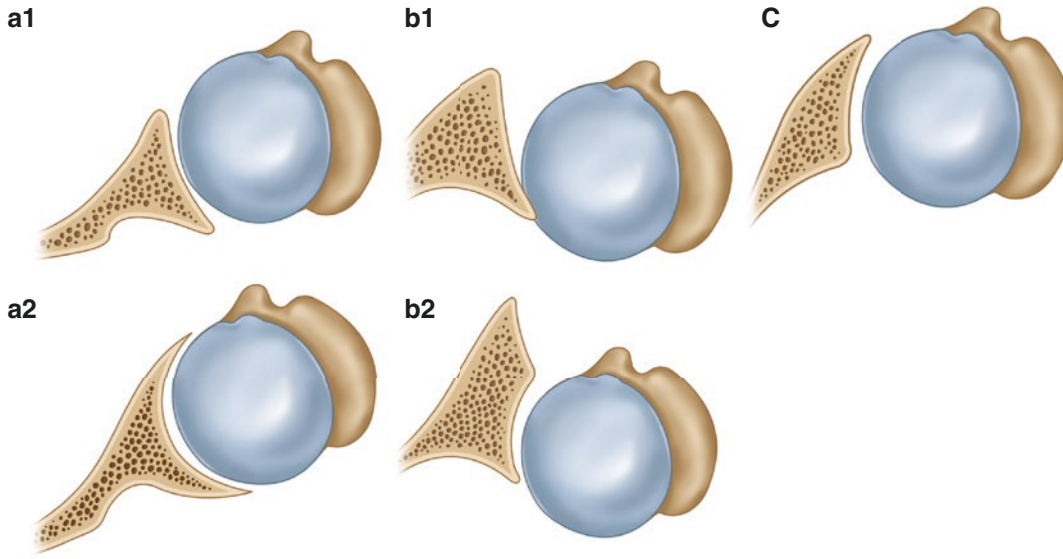


Fig. 29.2 Classification of glenoid morphology in osteoarthritis in the axial plane, according to Walch et al. [1]

asymmetric. The CT scan reveals numerous anatomic changes, more pronounced on the posterior margin of the glenoid. The retroversion average was 18°. Two subgroups were identified: B1 (17%) shows narrowing of the posterior joint space, subchondral sclerosis, and osteophytes; B2 (15%) demonstrates a posterior cupula that gives an unusual biconcave aspect of the glenoid. In type B2, there is an excessive retroversion of the glenoid, but the value of the retroversion does not explain the biconcavity of the glenoid.

Type C (9%): This type of glenoid morphology is defined by a glenoid retroversion of more than 25°, regardless of the erosion. The average retroversion was 35.7°.

Habermeyer et al. [2] classified vertical glenoid morphology in centered osteoarthritis. In this assessment, the coracoid baseline is considered reproducible because the AP view is taken into a standardized standing position of the patient, so that inferior border of the X-ray film is parallel to the bottom and the lateral base of the coracoid does not change with the rotation of the scapula (Fig. 29.3). Type 0 represents normal glenoids; the coracoid baseline and the glenoid line run parallel. Both lines intersect below the inferior glenoid rim in type 1 glenoids. In type 2 glenoids, the coracoid baseline and the glenoid line intersect between the inferior glenoid rim and the center of the glenoid. In type 3 glenoids, the lines intersect above the coracoid base (Fig. 29.4).

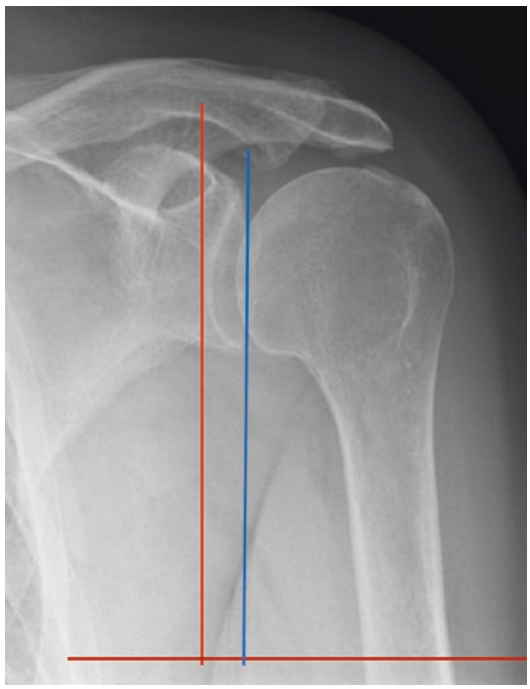


Fig. 29.3 Measurement of glenoid inclination according to the method described by Habermeyer et al. [2]

Classification of osteoarthritis with massive rotator cuff tears according to Favard et al. [3] considers three groups. Group 1 is characterized by upward migration of the humeral head, superior glenohumeral joint space narrowing, a change in the shape of the acromion due to the imprint of the humeral head, and subacromial arthritis. Group 2 is characterized by central glenohumeral joint space narrowing and with little alteration in the shape of the acromion, which does not have a humeral head imprint. Group 3 is characterized by signs of bony destruction in the form of lysis of either the head or the acromion. The bony elements not affected by the lysis do not undergo any modification in their shape, for example, the greater tuberosity is not eroded, and the acromion does not have humeral head imprint. Glenohumeral joint space narrowing is either minimal or absent.

Analysis of cuff arthropathy and failed treatment has led to a biomechanical classification of cuff tear arthropathy. Visotsky et al. [4] recognized four distinct groups based on the biomechanics and clinical outcomes of arthroplasty. The four types are distinguished by the degree of superior migration from the center of rotation and amount of instability of the center of rotation. This classification has proposed benefits in surgical decision-making for optimal implant type, goals of reconstruction, and outcomes.

Roentgenographic grades of cuff tear arthropathy were proposed by Hamada et al. [5]. These were mainly based on the acromiohumeral interval (AHI), which has been considered a sensitive indicator for the full-thickness cuff tears. Five grades were classified: grade 1, the AHI is more than 6 mm; grade 2, the AHI is 5 mm or less; grade 3, acetabularization is added to the grade 2 characteristics; grade 4, narrowing of the glenohumeral joint is added to the grade 3; and grade 5, humeral head collapse.

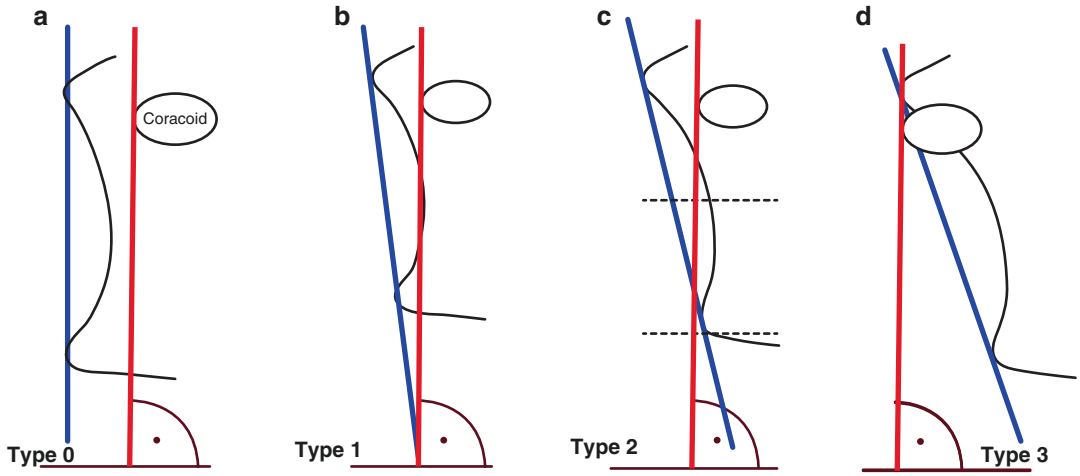


Fig. 29.4 Classification of glenoid inclination according to Habermeyer et al. [2]. (**a**) In type 0, the coracoid base line (red) and the glenoid line (blue) run parallel (the brown line represents the inferior border of the radiograph). (**b**) In type 1, the coracoid base line and the glenoid line intersect below the inferior glenoid rim. (**c**) In type 2, the coracoid base line and the glenoid line intersect between the inferior glenoid rim and the center of the glenoid. (**d**) In type 3, the coracoid base line and the glenoid line intersect above the coracoid base

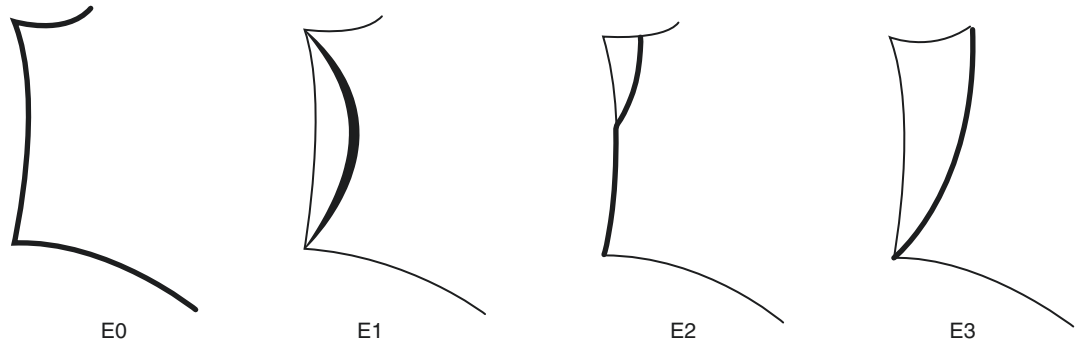


Fig. 29.5 Classification of glenoid erosion in osteoarthritis with massive rupture of the cuff according to Sirveaux et al. [6]

Sirveaux et al. [6] defined four types of glenoid erosion. In type E0, the humeral head migrated upward without erosion of the glenoid. Type E1 was defined by a concentric erosion of the glenoid. In type E2, there was an erosion of the superior of the glenoid, and in type E3 the erosion extended to the inferior part of the glenoid (Fig. 29.5).

The preoperative radiographic study, defining the possible bone reconstruction, influences approach and the technique of setting up the rTSA. The study of glenoid morphology is the key point of preoperative planning because the glenoid fixation is the weak link in the rTSA. It makes possible to evaluate the need for a bone graft and to calculate the size of the graft. Computer-aided 3-D planning allows to optimize the positioning of the glenoid component and to find the *paleoglenoid* and to calculate the exact shape of the necessary graft [8–10]. Customized glenoid component is an interesting option for filling bone loss in glenoid reconstructions [11].

29.2 Surgical Approach

Two approaches can be used, deltopectoral or anterosuperior. Each of them has advantages and disadvantages.

The deltopectoral approach requires dislocation of the humeral head to expose the articular surfaces and mainly the glenoid surface. This dislocation causes tension in the axillary nerve and may cause elongation with paresis of the axillary nerve.

For good glenoid exposure, the partial release of the tendon of the pectoralis major muscle is sometimes required in addition to the complete section of the subscapularis muscle. Repair of the subscapularis muscle is not always possible due to humeral lateralization and longer humerus. In these circumstances, the risk of prosthesis dislocation is significantly increased. However, deltopectoral approach allows an easy extension of the approach if it is necessary to expose the humeral shaft in case of intraoperative fracture. The front edge of the glenoid is also easier for glenoid reconstructions in case of anterior bone loss. In the event of an extensive approach to the shoulder, clavicle osteotomy is an option to protect the anterior fibers of the deltoid muscle.

The anterosuperior approach allows a direct view of the glenoid surface, with a good exposure of the B2 glenoid. The inferior tilt is more difficult to adjust. The violation of the deltoid muscle fibers is the most negative point of this approach. On the other hand, the absence of dislocation of the glenohumeral joint protects the nerves from the risk of elongation and paresis [12].

29.3 Implant Design

The choice of the implant is the main important step of the procedure. According to Grammont [7], the proximity of the center of rotation at the bone-prosthesis interface on the glenoid side solved the problem of the fixation on the scapula, and the 155° neck-shaft angle ensured better stability of the prosthesis. These improvements extended the implant survivorship. Unfortunately, notching observed with this generation of rTSA was surely one of the main causes of the degradation of the results in the longer term.

In the continuation of the work of Grammont [7], numerous studies contributed to evolve the design of rTSA. Several studies showed that implant lateralization maintains the moment arm of the deltoid muscle forces and ensures optimal muscle strength. Frankle et al. [13] showed that lateralization of the center of rotation, located within the glenoid implant and no longer at the bone interface, allows an improvement of mobility without altering the glenoid bone fixation.

Nevertheless, the ideal amount of global lateralization and the ideal contribution from the glenoid or from the humerus remain unknown. It probably varies depending on patient anatomy, quality and quantity of any remaining cuff, deltoid quality, and the amount of distalization of the humerus (arm and deltoid lengthening) [14].

Hamilton et al. [15] described three different combinations: a medial glenoid center of rotation with a medialized humerus, meaning that the location of the humeral shaft is the closest to the scapula of all three designs; a lateralized glenoid center of rotation and a medialized humerus, so that the position of the humerus is more lateral than the Grammont-style design; and a lateralized humerus—as a result, the humerus is positioned further lateral than the previous two designs (Fig. 29.6). According to the authors, the latter design increases the efficiency of the posterior deltoid compared to the other two designs.

Werthel et al. [14] described that mean insert lateralization is +11 mm (range, 7.3–17.7 mm) in onlay implants versus +2.2 mm (range, 1.9–5.5 mm) in inlay implants. Humeral insert offset varies from −3.5 to 14.2 mm for all designs. The average range of lateral offset is 10.8 mm. The range of global lateral offset that is possible to obtain with one given implant varies from 3.3 to 20.9 mm.

The choice of the glenosphere's diameter is an important point. Changes in the shape of the glenosphere did not affect function or longevity of the rTSA. Some authors recommend the maximum size (42 mm) to achieve the maximum lateralization of the humerus and prevention of notching. However, its placement is sometimes

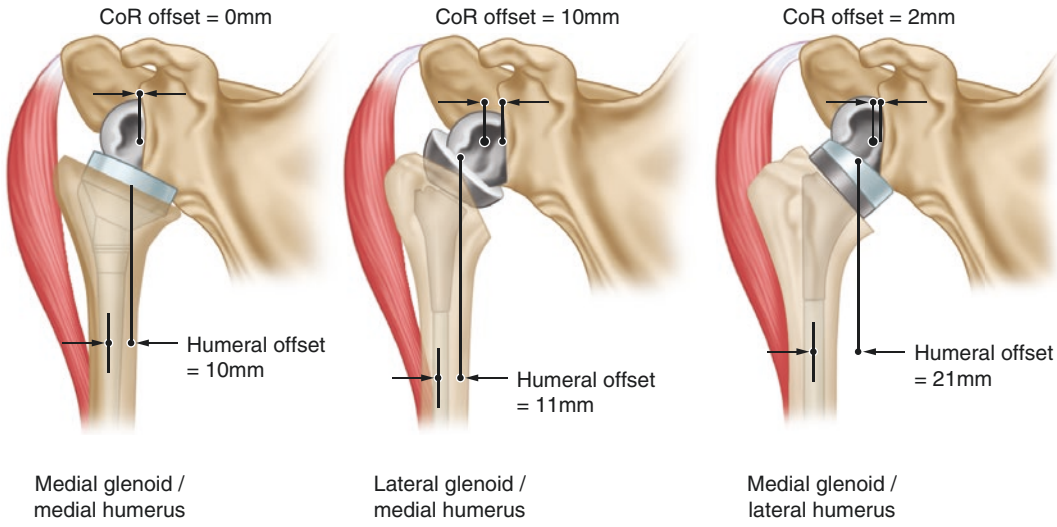


Fig. 29.6 Combinations of glenoid and humeral lateralization according to Hamilton et al. [15]

more difficult than a smaller-diameter glenosphere [15–20].

The design of the neck-shaft inclination angle has recently evolved. Initially, Grammont considered the 155° angle to be the best angle to provide stability to the prosthesis. This idea is questionable, and studies have shown that angles of 135° and 145° only partially increase the risk of dislocation, but they could have a beneficial effect to prevent the risk of notching and allow lateralization of the humeral component [20–25].

Humeral lateralization is also influenced by stem design and onlay versus inlay design [26–29]. The concept of modularity and conversion of total shoulder arthroplasty (TSA) into rTSA without removing the stem of the prosthesis has led to changes in the shape of the humeral component.

Currently, onlay design models are more numerous than inlay design. The onlay position of the humeral trail theoretically increases the humeral offset and the arm moment of the deltoid muscle and improves mobility and muscle strength [22, 30]. Berthouet et al. [30] conducted an experimental study about the effects of the humeral tray component positioning for onlay rTSA design and observed that humeral tray offset may not have as large effect on RTSA function as other fixation or design factors, such as inferior glenoid placement, size of glenosphere, and obliquity of humeral osteotomy, but should be considered because it can affect the overall function of the prosthesis [31–35].

Although polyethylene wearing increases the glenoid bone resorption and makes the glenoid bone fixation weaker, notching studies did not provide a reliable solution for the reduction of polyethylene wearing. Indeed, all biomechanical studies are done with fixed scapula, albeit the scapula is mobile on the chest during shoulder movements. This bias makes these studies difficult to correlate with clinical outcomes.

The lengthening of the humerus shaft is an element of stability of the RTSA. It can be planned preoperatively. It can be appreciated during surgery by the tension of the conjoint tendon more than by the tension of the fibers of the deltoid muscle. Excessive lengthening can lead to complications, such as acromion fracture or axillary nerve injury that decrease mobility and strength.

Levy et al. [36] reported on stemless rTSA with very promising results, though with short follow-up. Stemless design is encouraging because of the preservation of bone stock and

decreased difficulties to treat humeral shaft fractures. Moreover, it makes humeral component revision easier. However, in case of implant loosening, degradation of results is faster.

For the more recently designed short-stem prostheses, there is still insufficient follow-up time to demonstrate their superiority. But as stemless rTSA they are promising, because the humeral component complication rate is low.

Finally, while many authors initially recommended cementing the humeral component to prevent the prosthesis from sinking into the humeral shaft, currently all the prostheses are cementless with a press-fit fixation.

29.3.1 Muscle Transfer in Reverse Total Shoulder Arthroplasty

A particular condition corresponds to the treatment of massive ruptures with total loss of external rotation. Assessment of the total loss of the external rotation is assessed by positivity for the hornblower's sign in abduction and external rotation and the external rotation lag sign with the elbow at the body. Clinical exam is complemented by the evaluation of muscle atrophy and fatty infiltration of the teres minor muscle on coronal sections of MRI or CT arthrography [37, 38].

Reverse total shoulder arthroplasty cannot restore external rotation in the absence of the teres minor muscle. Palliative surgery of the sequelae of obstetric paralysis of the brachial plexus has improved the results of reverse total shoulder arthroplasty in these indications.

L'Episcopo described in 1934 the transfer of the teres major with tenotomy of the subscapularis muscle and the latissimus dorsi muscle to improve the external rotation of the arm. In 1939, he modified this technique by transferring the muscles latissimus dorsi and teres major by a double approach [39, 40]. The same double transfer technique was described in parallel by Merle d'Aubigné using a single deltopectoral approach [41] (Fig. 29.7). These techniques have been associated with rTSA to restore external rotation [42].

The technique using a single approach is the easiest to achieve. The deltopectoral approach is extended with a complete detachment of the pectoralis major to expose the muscles latissimus dorsi and teres major. With this approach, the separation of the two tendons could be sometimes difficult.

The reinsertion of the two tendons is made on the same zone. The use of buttons allows a very strong fixation and allows immediate mobilization of the arm after surgery. Fixation of the pectoralis major tendon is very important to maintain the active internal rotation and stability of the rTSA. Recovery of external rotation is slow and effective after recovery of the range of motion.

29.3.2 Results

Despite a high survival rate and good long-term clinical results of rTSA [43–47], outcomes deteriorated over time when compared with medium-term results. The cause of this decrease is probably related to patient aging coupled with bone erosion and/or deltoid impairment over time.

Ernstbrunner et al. [48], in a systematic review, reported eight studies with a total of 365 shoulders and a mean follow-up of 9.5 years (range, 5–20 years). They concluded that long-term results of rTSA in MIRCTs show significant improvement of overhead function and objective and subjective outcome scores up to 20 years after surgery. Shoulder function and outcome scores also showed no significant deterioration between 5 and 20 years of follow-up.

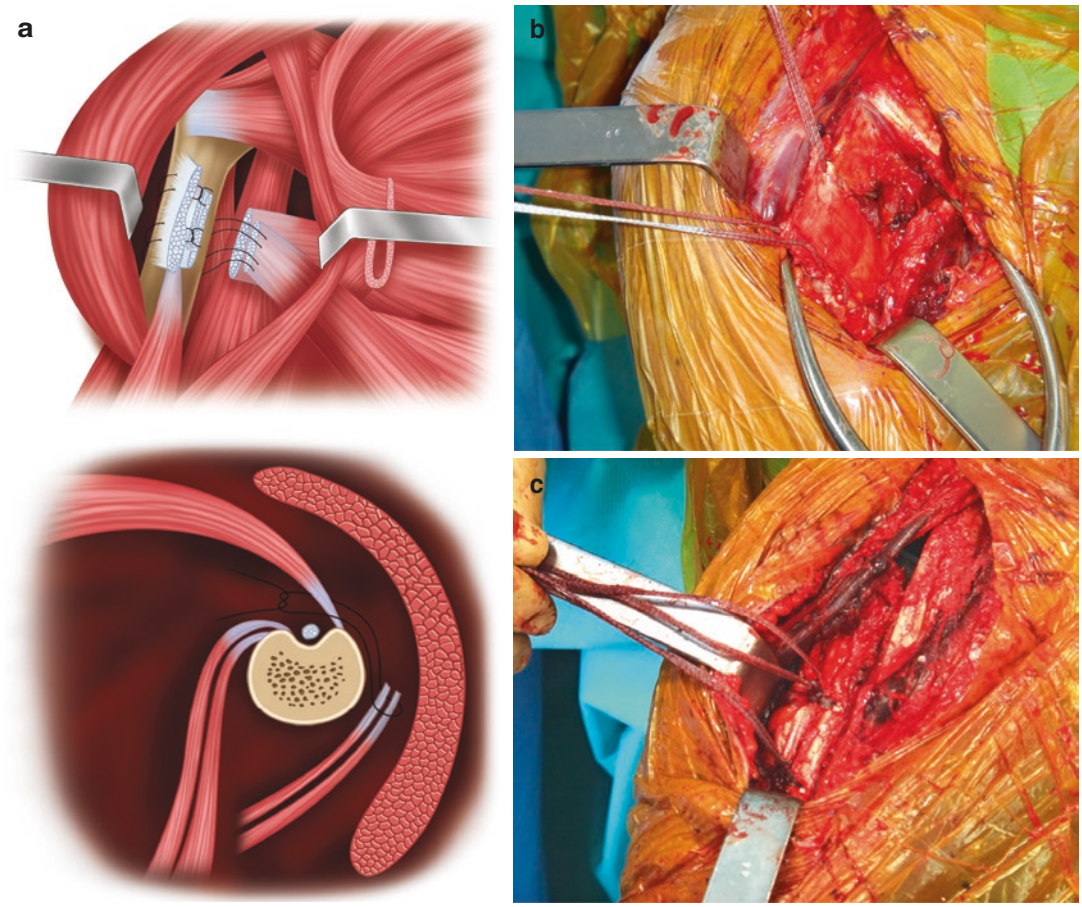


Fig. 29.7 (**a**–**c**) Latissimus dorsi transfer according to Merle d'Aubigné [41]

29.4 Conclusions

Numerous clinical and biomechanical studies have enabled the concepts of the reverse prosthesis to be developed and their design gradually modified. From inlay, the design of the RTSA has evolved toward an onlay design. Today, onlay prostheses are more numerous than inlay prostheses without providing an improvement greater than the glenosphere diameter increase or glenoid implant positioning. Modification of the neck-shaft angle from 155° to 145° or 135° seems to decrease the risk of notching. Humeral lateralization seems also useful to restore deltoid muscle arm and improve the functional results. The use of 3-D CT preoperative planning and patient-specific instrumentation has been demonstrated to improve the accuracy of glenoid implant placement in difficult cases of implantation. Future improvements will make rTSA even more reliable and will provide better patient outcomes.

References

1. Walch G, Badet R, Boulahia A, Khoury A. Morphologic study of the glenoid in primary glenohumeral osteoarthritis. J Arthroplast. 1999;14(6):756–60.
2. Habermeyer P, Magosch P, Luz V, Lichtenberg S. Three-dimensional glenoid deformity in patients with osteoarthritis: a radiographic analysis. J Bone Joint Surg Am. 2006;88(6):1301–7.
3. Favard L, Lautmann S, Clement P. Osteoarthritis with massive rotator cuff tear: the limitation of its current definitions. In: Gazielly D, Gleyze P, Thomas T, editors. The cuff. Paris: Elsevier; 1997. p. 261–5.

4. Visotsky JL, Basamania C, Seebauer L, Rockwood CA, Jensen KL. Cuff tear arthropathy: pathogenesis, classification, and algorithm for treatment. J Bone Joint Surg Am. 2004;86-A Suppl 2: 35–40.
5. Hamada K, Yamanaka K, Uchiyama Y, Mikasa T, Mikasa M. A radiographic classification of massive rotator cuff tear arthritis. Clin Orthop Relat Res. 2011;469:2452–60. https://doi.org/10.1007/s11999-011-1896-9.
6. Sirveaux F, Favard L, Oudet D, Huquet D, Walch G, Mole D. Grammont inverted total shoulder arthroplasty in the treatment of glenohumeral osteoarthritis with massive rupture of the cuff. Results of a multicentre study of 80 shoulders. J. Bone Joint Surg Br. 2004;86(3):388–95.
7. Grammont P, Laffay JP, Deries X. Etude et réalisation d'une nouvelle prothèse d'épaule. Rhumatologie. 1987;39:407–18.
8. Van Haver A, Heylen S, Vuylsteke K, Declercq G, Verborgt O. Reliability analysis of glenoid component inclination measurements on postoperative radiographs and computed tomography-based 3D models in total and reversed shoulder arthroplasty patients. J Shoulder Elb Surg. 2016;25:632–40.
9. Wylie JD, Tashjian RZ. Planning software and patient-specific instruments in shoulder arthroplasty. Curr Rev Musculoskelet Med. 2016;9:1–9.
10. Iannotti JP, Walker K, Rodriguez E, Patterson TS, Jun BJ, Ricchetti ET. Accuracy of 3-dimensional planning, implant templating, and patient-specific instrumentation in anatomic total shoulder arthroplasty. J Bone Joint Surg Am. 2019;101:446–57.
11. Ivaldo N, Mangano T, Caione G, Rossoni M, Ligas A. Customized tantalum-augmented reverse shoulder arthroplasty for glenoid bone defect and excessive medialization: description of the technique. Musculoskelet Surg. 2016;100(Suppl 1):S13–8.
12. Lenoir H, Dagneaux L, Canovas F, Waitzenegger T, Pham TT, Chammas M. Nerve stress during reverse total shoulder arthroplasty: a cadaveric study. J Shoulder Elb Surg. 2017;26:323–30.
13. Mark F, Steven S, Derek P, Arif S, Mark M, Matthew V. The reverse shoulder prosthesis for glenohumeral arthritis associated with severe rotator cuff deficiency a minimum two years follow-up study of sixty patients. J Bone Joint Surg Am. 2005;87-A(8):1697–705.
14. Werthel JD, Walch G, Vegehan E, Deransart P, Sanchez-Sotelo J, Valenti P. Lateralization in reverse shoulder arthroplasty: a descriptive analysis of different implants in current practice. Int Orthop. 2019;43(10):2349–60.
15. Hamilton MA, Diep P, Roche C, Flurin PH, Wright TW, Zuckerman JD, Routman H. Effect of reverse shoulder design philosophy on muscle moment arms. J Orthop Res. 2015;33(4):605–13.
16. Langohr GD, Willing R, Medley JB, Athwal GS, Johnson JA. Contact mechanics of reverse total shoulder arthroplasty during abduction: the effect of neck-shaft angle, humeral cup depth, and glenosphere diameter. J Shoulder Elb Surg. 2016;25:589–97.
17. Oh JH, Shin S-J, McGarry MH, Scott JH, Heckmann N, Lee TQ. Biomechanical effects of humeral neck-shaft angle and subscapularis integrity in reverse total shoulder arthroplasty. J Shoulder Elb Surg. 2014;23:1091–8.
18. Brent M, Mahure SA, Roche CP, Zuckerman JD. Impact of glenosphere size on clinical outcomes after reverse total shoulder arthroplasty: an analysis of 297 shoulders. J Shoulder Elb Surg. 2016;25:763–71.
19. North LR, Hetzler MA, Pickell M, Bryant JT, Deluzio KJ, Bicknell RT. Effect of implant geometry on range of motion in reverse shoulder arthroplasty assessed using glenohumeral separation distance. J Shoulder Elb Surg. 2015;24:1359–66.
20. Lawrence C, Williams GR, Namdari S. Influence of glenosphere design on outcomes and complications of reverse arthroplasty: a systematic review. Clin Orthop Surg. 2016;8:288–97.
21. Greiner S, Schmidt C, Herrmann S, Pauly S, Perka C. Clinical performance of lateralized versus non-lateralized reverse shoulder arthroplasty: a prospective randomized study. J Shoulder Elb Surg. 2015;24:1397–404.
22. Ackland DC, Patel M, Knox D. Prosthesis design and placement in reverse total shoulder arthroplasty. J Orthop Surg Res. 2015;10:101–9.
23. Chaea SW, Leeb J, Hanc SH, Kim S-Y. Inferior tilt fixation of the glenoid component in reverse total shoulder arthroplasty: a biomechanical study. Orthop Traumatol Surg Res. 2015;101:421–5.
24. Fortun CM, Streit JJ, Horton SA, Muh SJ, Gillespie RJ, Gobezie R. Scapular neck length and implications for reverse total shoulder arthroplasty: an anatomic study of 442 cadaveric specimens. Int J Shoulder Surg. 2015;9(2):38–42.
25. Giles JW, Langohr GDG, Johnson JA, Athwal GS. Implant design variations in reverse total shoulder arthroplasty influence the required deltoid force and resultant joint load. Clin Orthop Relat Res. 2015;473:3615–26.
26. Walker DR, Kinney AL, Wright TW, Banks SA. How sensitive is the deltoid moment arm to humeral offset changes with reverse total shoulder arthroplasty? J Shoulder Elb Surg. 2016;25:998–1004.
27. Fischer C, Krammera D, Hug A, Weber M-A, Kauczor H-U, Krix M, Bruckner T, Kunz P, Schmidmaier G, Zeifang F. Dynamic contrast-enhanced ultrasound and elastography assess deltoid muscle integrity after reverse shoulder arthroplasty. J Shoulder Elb Surg. 2017;26:108–17.
28. Feeley BT, Zhang AL, Barry JJ, Shin E, Ho J, Tabaraee E, Benjamin C. Decreased scapular notching with lateralization and inferior baseplate placement in reverse shoulder arthroplasty with high humeral inclination. Int J Shoulder Surg. 2014;8(3):65–71.
29. Mathews S, Burkhard M, Serrano N, Link K, Häusler M, Frater N, Franke I, Bischofberger H, Buck FM, Gascho D, Thali M, Serowy S, Müller-Gerbl M, Harper G, Qureshi F, Böni T, Bloch H-R, Ullrich O,

Rühli F-J, Eppler E. Glenoid morphology in light of anatomical and reverse total shoulder arthroplasty: a dissection- and 3D-CT-based study in male and female body donors. BMC Musculoskelet Disord. 2017;18(9):1373–4.
30. Berhouet J, Kontaxis A, Gulotta LV, Craig E, Warren R, Dines J, Dines D. Effects of the humeral tray component positioning for onlay reverse shoulder arthroplasty design: a biomechanical analysis. J Shoulder Elb Surg. 2015;24:569–77.
31. Nyffeler RW, Werner CML, Gerber C. Biomechanical relevance of glenoid component positioning in the reverse Delta III total shoulder prosthesis. J Shoulder Elb Surg. 2005;14:524–8.
32. Smith T, Bäunker A, Krämer M, Hurschler C, Kaufmann M, Pastor MF, Wellmann M. Biomechanical evaluation of inferior scapula notching of reverse shoulder arthroplasty depending on implant configuration and scapula neck anatomy. Int J Shoulder Surg. 2015;9(4):103–9.
33. Carpenter S, Pinkas D, Newton MD, Kurdziel MD, Baker KC, Michael Wiater J. Wear rates of retentive versus nonretentive reverse total shoulder arthroplasty liners in an in vitro wear simulation. J Shoulder Elb Surg. 2015;24:1372–9.
34. Teeter MG, Dawson MT, Athwal GS. Inter and intrasystem size variability of reverse shoulder arthroplasty polyethylene inserts. Int J Shoulder Surg. 2016;10(1):10–4.
35. Berhouet J, Garaud P, Favard L. Evaluation of the role of glenosphere design and humeral component retroversion in avoiding scapular notching during reverse shoulder arthroplasty. J Shoulder Elb Surg. 2014;23(2):151–8.
36. Levy O, Narvani A, Hous N, Abraham R, Relwani J, Pradhan R, Bruguera J, Sforza G, Atoun E. Reverse shoulder arthroplasty with a cementless short metaphyseal humeral implant without a stem: clinical and radiologic outcomes in prospective 2- to 7-year follow-up study. J Shoulder Elb Surg. 2016;25:1362–70.
37. Gregory N. Drake, Daniel P. O'Connor' T. Bradley Edwards Indications for reverse total shoulder arthroplasty in rotator cuff disease. Clin Orthop Relat Res (2010) 468 : 1526–1533.
38. Hartzler RU, Steen BM, Hussey MM, Cusick MC, Cottrell BJ, Clark RE, Frankle MA. Reverse shoulder arthroplasty for massive rotator cuff tear: risk factors for poor functional improvement. J Shoulder Elb Surg. 2015;24:1698–706.
39. L'Episcopo JB. Restoration of muscle balance in the treatment of obstetrical paralysis. N Y State J Med. 1939:357–63.
40. L'Episcopo JB. Tendon transplantation in obstetrical paralysis. Am J Surg. 1934;25:122–5.
41. Merle d'Aubigné R. Symposium on reconstructive surgery of paralised upper limb. Proc Royal Soc Med. 1949;42:831–5.
42. Shi LL, Cahill KE, Ek ET, Tompson JD, Higgins LD, Warner JJP. Latissimus dorsi and teres major transfer with reverse shoulder arthroplasty restores active motion and reduces pain for posterosuperior cuff dysfunction. Clin Orthop Relat Res. 2015;473:3212–7.
43. Guery J, Favard L, Sirveaux F, Oudet D, Mole D, Walch G. Reverse total shoulder arthroplasty survivorship analysis of eighty replacements followed for five to ten years. J Bone Joint Surg Am. 2006;88-A(8):1742–7.
44. Melis B, DeFranco M, Lädermann A, Molé D, Favard L, Nérot C, Maynou C, Walch G. An evaluation of the radiological changes around the Grammont reverse geometry shoulder arthroplasty after eight to 12 years. J Bone Joint Surg. 2011;93-B(9):1240–6.
45. Bacle G, Nové-Josserand L, Garaud P, Walch G. Long-term outcomes of reverse total shoulder arthroplasty a follow-up of a previous study. J Bone Joint Surg Am. 2017;99-A(6):454–61.
46. Kempton LB, Ankerson E, Wiater JM. A complication-based learning curve from 200 reverse shoulder arthroplasties. Clin Orthop Relat Res. 2011;469(9):2496–504.
47. Samitier G, Alentorn-Geli E, Torrens C, Wright TW. Reverse shoulder arthroplasty. Part 1: systematic review of clinical and functional outcomes. Int J Shoulder Surg. 2015;9(1):24–31.
48. Ernstbrunner L, Andronic O, Grubhofer F, Camenzind RS, Wieser K, Gerber C. Long-term results of reverse total shoulder arthroplasty for rotator cuff dysfunction: a systematic review of longitudinal outcomes. J Shoulder Elb Surg. 2019;28(4):774–81.

30 Regional Blocks and Opioid-Sparing Anesthesia: Helping the Surgeon and with Patients' Satisfaction

Clara Lobo and Nuno Sampaio Gomes

30.1 Introduction

Arthroscopic shoulder surgery is one of the most common orthopedic procedures [1, 2] routinely performed in ambulatory setting, despite the association with severe postoperative pain [3, 4]. *S*houlder surgeries are among the top 100 most painful procedures: shoulder joint replacement as the 12th most painful, followed by open reconstruction shoulder ligaments, partial shoulder joint replacement, and arthroscopic shoulder surgery with and without ligament repair as 21st, 22nd, 55th, and 100th place in the list [4].

More complex ambulatory surgery has been made possible due to advances of technology and surgical technique allied to innovations of the anesthetic plan. Pain management with minimal side effects is crucial for successful ambulatory surgery and recovery [1]. Traditionally, pain management was based on high-dose opioids, invaliding a fast recovery and associated with postoperative nausea and vomiting (PONV), respiratory depression, sedation, and prolonged length of stay (LOS). The best profile technique includes regional anesthesia (RA) alone or in combination with general anesthesia (GA). When properly performed and managed, upper extremity blocks do not delay operating room time, do improve patient recovery and satisfaction, and do decrease hospital discharge time, unanticipated hospital admission, and added costs [5]. The advantages of these analgesic techniques over traditional oral narcotics have led to their rapid acceptance as a standard of care, at many institutions, as the base of the pyramid [6] of multimodal analgesia (MMA) (Fig. 30.1).

Defining the best anesthetic technique requires knowledge of the type of surgery, patient positioning, medical background, expectations, and preferences.

30.2 Considerations for the Attending Anesthesiologist

30.2.1 Preoperative

Less invasive arthroscopic procedures and RA techniques are expanding the indications and benefits of surgery to sicker patients. The procedures can be performed in patients who are awake, sedated (conscious or unconscious), or under endotracheal intubation or supraglottic airway GA.

C. Lobo (✉)
Hospital das Forças Armadas, Porto, Portugal

N. Sampaio Gomes
CH Póvoa Varzim Vila do Conde, Porto, Portugal

Hospital das Forças Armadas, Porto, Portugal

Hospital da Luz, Porto, Portugal

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_30

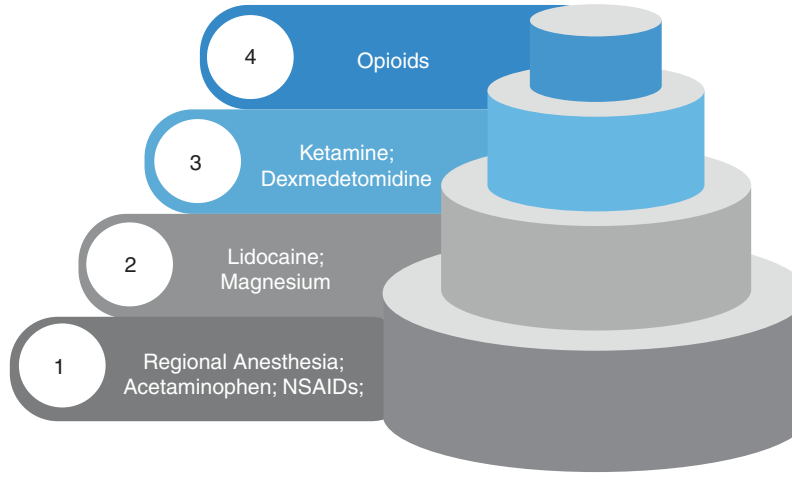


Fig. 30.1 Multimodal analgesia pyramid. RA—regional analgesia

Table 30.1 Patient physical status (ASA) classification, according to the American Society of Anesthesiologists

ASA	Definition	Examples
1	A normal healthy patient	Healthy (minimal alcohol use and no smoking)
2	A patient with mild systemic disease	Mild disease with minimal disability (pregnancy, BMI 30–40, well-controlled DM/HTN, mild lung disease, social alcohol use, smoker)
3	A patient with severe systemic disease	One or more moderate to severe diseases (poorly controlled DM/HTN, COPD, BMI ≥ 40, active hepatitis, alcohol dependence or abuse, implanted pacemaker, moderate reduction of ejection fraction, ESRD undergoing regularly scheduled dialysis, >3 months history of MI, CVA, TIA, or CAD/stents)
4	A patient with severe systemic disease that is a constant threat to life	Examples include (not limited to): <3 months MI, CVA, TIA, or CAD/stents, ongoing cardiac ischemia or severe valve dysfunction, severe reduction of ejection fraction, sepsis, DIC, ARD, or ESRD not undergoing regularly scheduled dialysis
5	A moribund patient, not expected to survive without the operation	Examples include (not limited to): ruptured abdominal/thoracic aneurysm, massive trauma, intracranial bleed with mass effect, ischemic bowel in the face of significant cardiac pathology, or multiple organ/system dysfunction
6	A declared brain-dead patient whose organs are being removed for donor purposes	

ARD acute respiratory distress, *BMI* body mass index, *CAD* coronary artery disease, *COPD* chronic obstructive pulmonary disease, *CVA* cerebrovascular accident, *DIC* disseminated intravascular coagulation, *DM* diabetes mellitus, *ESRD* end-stage renal disease, *HTN* hypertension, *MI* myocardial infarction, *TIA* transient ischemic attack

[a]The addition of "E" means emergency surgery: when delay in treatment of the patient would lead to a significant increase in the threat to life or body part

Tailoring anesthesia involves the evaluation of the patient health status (ASA) [7] (Table 30.1) and medication to the type of surgical procedure and postoperative care planned.

Zhao et al. [8] using a cohort of 124,860 patients calculated a risk stratification index (RSI) and recognized several risks associated with unexpected overstay (OS) admission and emergency department (ED) transfer in outpatient shoulder arthroscopic surgery: arrhythmia, COPD, obesity, diabetes, neurologic disease with function impairment, GA requirement, advanced age (>80 years old), and MO (BMI > 40). Patients with RSI scores of ≤2 were eligible to a freestanding surgical center, while those with higher RSI scores were transferred to a unit prepared with ED and OS. Predictors for increased LOS after shoulder arthroplasty are obesity,

> 65 years, female sex, diabetes, and reverse shoulder arthroplasty [9].

Identification of patients at high risk of deterioration from phrenic nerve palsy (PNP) is essential. These patients are often the same group who would benefit from the avoidance of perioperative opioids and GA. Choosing RA techniques that spare phrenic nerve involvement (see below) and cautious planning for possible postoperative respiratory support in a high dependency unit are mandatory for high-risk patients [10]. Using CPAP helps to overcome imminent respiratory distress [11].

For awake procedures, a careful patient selection is mandatory. Preparation should start before the day of surgery, and all details should be discussed, as well as the plan "B" in case of RA failure, the close proximity of the surgery and drapes to the patients' face promoting a sense of claustrophobia, the numb limb for long periods of time, Horner's syndrome, hoarseness, and mild dyspnea. Patients should give their informed consent for anesthesia, meaning that a discussion of risks, complications, benefits, and alternatives related to the anesthesia care occurred between him and the anesthesiologist. It is recommended that some form of written documentation of the consent process should exist and signed by the patient, but it is not mandatory [12].

30.2.2 Perioperative

RA, GA, or a combination of both needs an anesthesia perioperative evaluation and standard monitoring [13, 14] (Fig. 30.2).

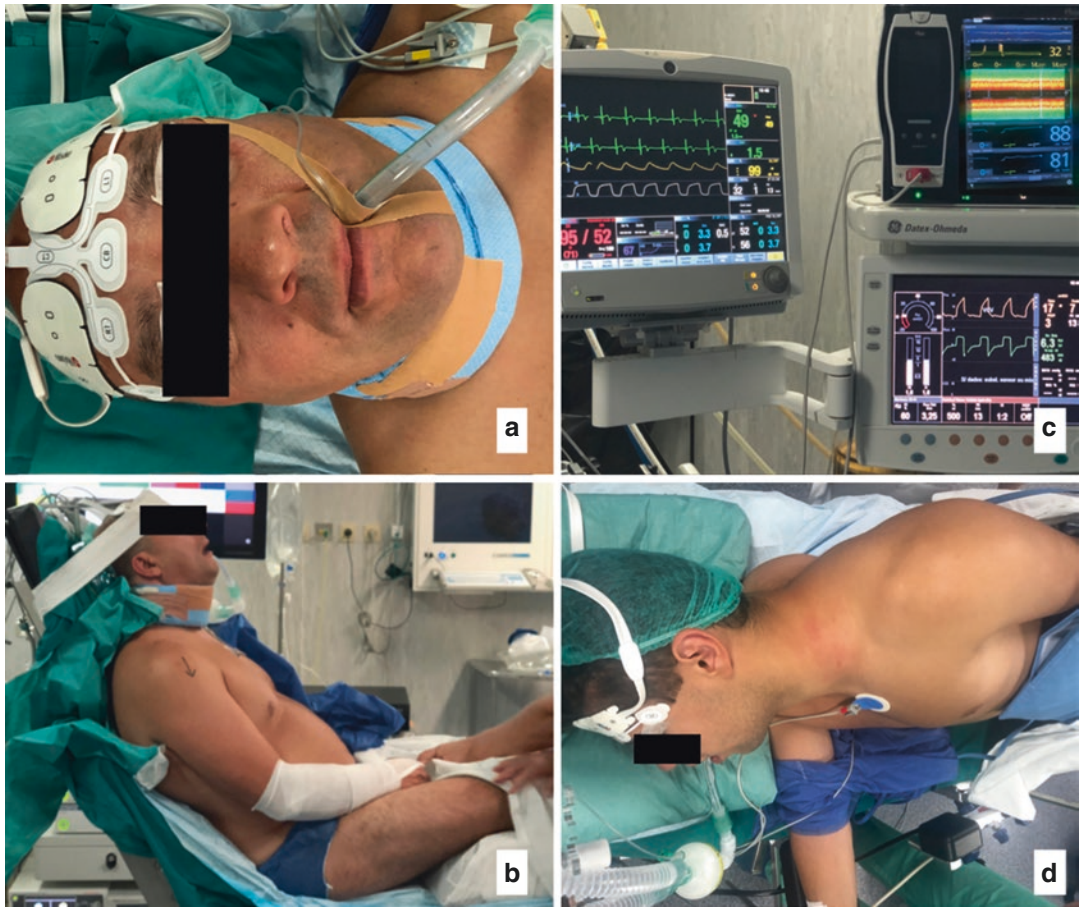


Fig. 30.2 Patient monitorization (**a** and **c**) and positioned in sitting or beach chair position (**b**) and lateral position (**d**)

Safe practice of RA recommends to gently and frequently aspirate before injecting LA—epinephrine can be of use to signal IV LA injection or as a means to reduce LA absorption rate—and use nerve localization techniques as peripheral nerve stimulation (PNS) and/or ultrasound guidance (USG). The presence of a motor response with PNS $\leq$ 0.2 mA is highly specific of intraneural needle placement. Using injection pressure monitor helps to identify intrafascicular needle placement if injection pressure is >15 psi. USG reduces time of block performance, number of needle passes, and doses of local anesthetics (LA), avoids vascular puncture, controls LA spread, exhibits better postoperative analgesia compared to PNS, and shows less inflammatory response [10, 15, 16]. Out-of-plane vs. in-plane techniques remain controversial and dependent on the anesthesiologist preference. Injection pressure monitoring can objectively be determined by appropriate monitors, but its use is debatable [17].

The combination of RA and sedation has several potential advantages like avoidance of airway manipulation, positive pressure lung ventilation and cardiovascular complications associated with GA, reduced incidence of PONV, patient engagement in treatment and care (if possible to see the arthroscopy live in the monitor), reduced time in OR turnover, faster recovery, and return to diet and medication postoperatively [10, 18]. Performing PNB under sedation or GA does not enhance the risk of major complications [19], but multiple skin punctures and bloody taps in anesthetized patients should be avoided. Nevertheless, RA performance in patients under GA or deep sedation should be executed by experienced hands.

Fluid irrigation used for arthroscopy can cause cervical edema and tracheal compression or laryngeal edema, often recognized during tracheal extubation. Risk factors associated with these life-threatening events are prolonged surgery (>2 h), lateral decubitus position, high pump pressures, and subacromial procedures [20].

30.2.3 Patient Positioning

Shoulder surgery can be performed in the beach chair (sitting) position (BCP) or lateral decubitus position (Fig. 30.2). Several factors favor the use of the BCP because it facilitates the setup and conversion to an open approach, offers a better visualization of intra-articular structures and orientation, and is associated with less brachial plexus traction damage [21]. Although rare, risks associated with BCP are stroke, coma, hemiparesis, quadriplegia and awareness, spinal cord ischemia, and visual loss associated with lower levels of blood pressure, cardiac output, and cerebral perfusion pressure (CPP) [21, 22]. BCP causes significant cerebral hemodynamic changes that if not detected and left untreated may cause a stroke and postoperative neurologic deficit [23].

Maintaining adequate cerebral blood perfusion (CBP) and oxygenation ($rScO_2$) should be the main objectives of the attending anesthesiologist. The blood pressure cuff should be placed in the contra-lateral upper arm, closest to the brain, for more accurate measurement compared to lower limb placement. Indirect MAP in the brain is lower (in average 30–40 mmHg) than the value determined in the upper arm and can be calculated using the formula: *height difference between blood pressure cuff and external meatus (cm)* × *0.76 mmHg*. If an arterial line is placed, the blood pressure transducer must be at the external meatus level, for accuracy [24].

There are different ways to monitor cerebral perfusion: near-infrared spectroscopy (NIRS), electroencephalography, transcranial color-coded duplex of cerebral arterial blood flow (TCCD), and jugular vein venous oxygenation determination ($SjVO_2$). NIRS devices are most commonly used and measure $rScO_2$ values noninvasively. NIRS has limitations [21, 25] and must be interpreted with care because contamination by extracranial blood and intra- and extracranial blood distribution are influenced by modifications in position, ventilation, and vasoconstrictors and contamination by nonheme tissue chromophores

such as bilirubin is also possible; only measures a small portion of brain tissue oxygenation—the electrodes are placed at the frontal bone (Fig. 30.2), measuring the frontal lobe oxygenation dependent of the anterior cerebral artery; it cannot identify the cause of desaturation, and, finally, a critical deoxygenation value is still unknown. $SjVO_2$ [21] is an invasive method that measures unilateral oxygenation (left or right); extracranial contamination occurs as CBP drops, and catheter dislodgement is possible with head movement and is a global cerebral oxygenation value that provides no information considering focal ischemia. TCCD allows direct visualization of the arteries of the circle of Willis and midbrain through a transtemporal window, providing basic information as blood flow velocity and pulsatility index [23]. Highly operator dependent, TCCD requires perfect knowledge of cerebrovascular anatomy and uses blood flow velocity as a surrogate for cerebral blood flow (CBF). Some patients lack an adequate acoustic window, which is limited to large basal arteries, and specific cortical regional changes may not be detected. Proper positioning of transcranial Doppler ultrasound probes may be difficult in the BCP [26]. Electroencephalography [21] requires a specific technician for interpretation and several channels for collecting appropriate information and is influenced by anesthetic drugs, electrolyte imbalances, hypothermia, and elevated ICP.

Variables associated with BCP that influence [21] cerebral oxygenation ($rScO_2$) are displayed in Table 30.2 and Fig. 30.3.

Vasopressors (phenylephrine, arginine vasopressin) increase MAP but have an inconstant effect on $rScO_2$ values, which may reflect changes in CBF, vasoconstriction of extracerebral vessels, or a combination of both factors [21, 27, 28]. Hypercapnia seems to blunt the negative impact of phenylephrine cerebral oxygenation, and hypocapnia intensifies that effect [28]. Ephedrine is recommended to treat hypotension [27], instead of phenylephrine. Severity and duration of cerebral desaturation events (CDEs) determine the occurrence of neurologic injury. There are reports that 15–25% reductions in $rScO_2$ are associated with cognitive dysfunction in cardiac surgery, hospital LOS in abdominal surgery, and higher level of brain injury markers after liver transplantation. In cardiac surgical patients, CDEs < 50 min were not associated with cognitive decline [21]. In a recent review, reported incidences of CDEs may vary between 18%, 35%, and 45%, and the timing of desaturation occurred 8 minutes after BCP positioning [21]. In patients under GA, the level of CDEs from supine to BCP or to lateral decubitus position ranged between 78% to 58% [29] and 72% to 55% [30], and its incidence was 80% and 57%, respectively. There was a small difference in volunteers, and the incidence was 6% [30]. RA with sedation reduces significantly the incidence of regional brain desaturation [21].

It is important to note that the incidence of adverse events is only as precise as the level of

Table 30.2 Variables associated with BCP that influence cerebral oxygenation

Cardiac output	Level of patient volemia Patient medical history Effects of general anesthetics (vasodilation) Blood pooling in lower extremities BCP position angle—linear decline in $rScO_2$ was observed as the BCP changed from 0° to 80° Positive pressure ventilation Vasopressor effects [27, 28]
Hypotension	Lower MAPs impair CO_2 reactivity Lower limit of autoregulation is difficult to determine in the particular patient
Variations of arterial CO_2 concentration	Higher arterial CO_2 concentration increases CBF, but CO_2 levels >50 mmHg disrupt cerebral autoregulation
Head rotation	The head should be in a neutral position
Cerebral vasculature disorders	Abnormal anatomy [89] or disease states with patients with high risk of ischemic events [22]; there is a consistent reduction in middle cerebral artery blood velocity

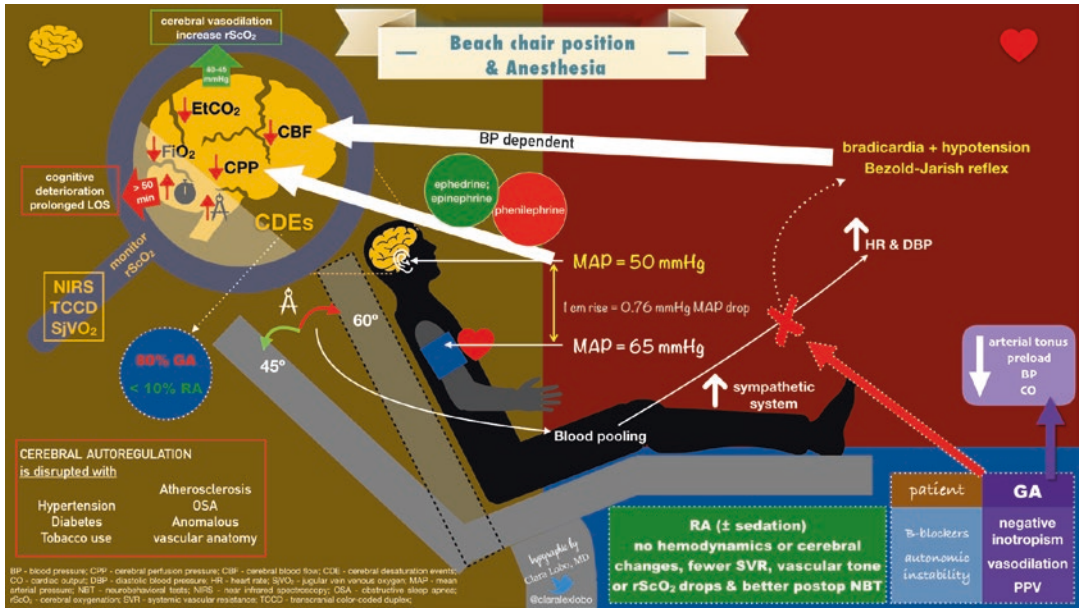


Fig. 30.3 Infographics explaining physiologic changes associated with BCP. Reprinted with permission from Clara Lobo Drawings

surveillance, reconnaissance, data recording, and reporting. Assessing all patients postoperatively can reveal other unreported and more subtle degrees of neurologic damage. The timing for that evaluation is to be determined.

30.2.4 Postoperative Analgesia

MMA combines non-opioid medications targeting different pain receptors to better manage and control pain (Fig. 30.1, Table 30.3), reducing opioid drug use and their complications [6, 31, 32]. The best MMA regimen remains unknown, but perioperative analgesic protocols should associate multiple analgesic modalities [31] for a better outcome. Regional analgesia is one of the components of MMA and cornerstone in opioid-tolerant patients [6]. The PROSPECT group published recommendations for pain management after arthroscopic rotator cuff repair [33] (ARCR; Table 30.4).

Shoulder procedures associated with mild to moderate pain may be managed with a single-injection nerve block, but rarely the block exceeds 18 h, including the use of most common adjuvants as clonidine, dexamethasone, or dexmedetomidine [34] (Table 30.5). Adjuvants are useful to improve block quality, onset, duration, and spread and prolong analgesia [35]. Drawbacks are prolonged motor block, delaying rehabilitation program, sedation, hemodynamic instability, the potential risk of off-label use, neurotoxicity, and medication errors while combining different types of drugs [35–37]. Studies point a slight prolongation of action with perineural compared to systemic administration [35, 38]. Systemic dexmedetomidine has a prolonged analgesic effect compared to dexamethasone [39] and clonidine [40]. Perineural dexamethasone is superior to dexmedetomidine without the risks of hypotension or sedation [41] and is administered perineurally and does not increase glycemia significantly [42]. Coadministration of IV dexamethasone (0.11 mg/kg) with dexmedetomidine (1.0 μg/kg) significantly prolonged analgesia after single-shot ISB for arthroscopic shoulder surgery [42].

Major shoulder procedures associated with intense long-lasting pain (e.g., arthroscopic rotator cuff repair, ARCR; total shoulder arthro-

Table 30.3 Drug categories used as part of multimodal analgesia regimens

Drug category	Mechanism of action	Recommended dose	Comment
Acetaminophen or paracetamol	CNS: COX-3 inhibitor Periphery: Weak COX-1 and COX-2 inhibitor	1000 mg q 6–8 h (max 4000 mg/d)	WHO approved for mild to severe pain management No antiplatelet, gastric, renal, or cardiovascular effects
NSAIDs	Inhibits COX-2 pathway, prostaglandin synthesis inhibition	Ibuprofen 400–600 mg q 4–8 h (max 2400 mg/d) Naproxen 220 mg q 8 h (max 660 mg/d) Celecoxib 200 mg q 12 h (max 400 mg/d)	Decrease opioid consumption and VAS scores Concerns of increased risk of cardiovascular events and delayed bone healing
Gabapentinoids	Reduce calcium entry on presynaptic nerves Decrease the release of excitatory neurotransmitters into the synaptic cleft Reduce neural postsynaptic transmission of pain	Gabapentin 300 mg q 8 h (max 3600 mg/d) Pregabalin 75 mg q 24 h (max 600 mg/d)	Anxiolytics, improve sleep quality, reduce perioperative delirium and postoperative pain chronicity Elderly patients are very sensitive, especially if combined with opioids
Alpha 2-agonists	Block the hyperpolarization-activated cation current and cause α2-selective vasoconstriction in the nerve	Clonidine IV or perineural dose—50–250 μg Dexmedetomidine IV dose—1–2 μg/kg; perineural dose—50–60 μg	Hypotension, bradycardia, and prolonged sedation (dexmedetomidine effects resolve in 2 h after terminating the infusion) are common and dose dependent Clonidine has neurotoxic properties when associated with ropivacaine and in diabetic rats [35]
Ketamine	*N*-methyl-*D*-aspartate antagonist	0.5 mg/kg IV bolus followed by 0.15–0.25 mg/kg/h	Opioid-sparing effect, better analgesia, reduced postoperative chronic pain incidence, and suitable for opioid-tolerant patients
Dexamethasone	Glucocorticosteroid	0.11–0.2 mg/kg IV (max 8 mg) or 4 mg (perineural – ceiling dose of 4 mg [90]	Reduced postoperative pain and opioid consumption Blood glucose levels not significantly increased if administered PN Used as nausea/vomiting prophylaxis (0.1 mg/kg), IV
Magnesium sulfate	Regulation of calcium influx and *N*-methyl-*D*-aspartate antagonist	40–50 mg/kg IV during induction, followed by 24 h 10–15 mg/kg/h infusion	Reduced both early, late postoperative pain and opioid consumption

plasty, TSA) have better functional outcomes and analgesia with a continuous block [34, 43] compared to GA [20]. Advantages of continuous peripheral nerve block (CPNB) include continuous analgesia, faster rehabilitation, reduced use and side effects of opioids, improved patient satisfaction, and faster hospital discharge [18, 44]. Placing a CPNB is more complex than the single-shot technique with potential serious complications and, obviously, not suited to the occasional practitioner.

30.3 Essential Anatomy for Anesthesiologists

A profound knowledge of shoulder innervation is mandatory. Major nerves involved in shoulder innervation are branches of the BP and SCP terminal branches—supraclavicular nerves [10]. Glenohumeral joint (GHJ) and acromioclavicular joint (ACJ) have typical innervation patterns [45] (Table 30.6 and Fig. 30.4), although anatomical variations are common [5].

The *cervical plexus* originates from the anterior rami of C1–C4 roots and their communicating rami. The cutaneous, muscular, and communicating branches are localized between, anteromedially, the prevertebral muscles and, posteriorly, the muscles anchored to the posterior cervical transverse process tubercle. Blocking the SCP (Fig. 30.5b) can potentially involve the phrenic nerve, but decreasing LA volume lowers that risk [46].

The *brachial plexus* (BP) is a neuronal network of ventral rami of C5–T1 roots, with possible contributions from C4 or T1. Typically, C5 and C6 unite to form the superior trunk (ST), close to the medial border of the middle scalene muscle (MSM); the C7 root becomes the middle trunk, and the inferior trunk results from C8 and T1 union, as they pass between the anterior (ASM) and MSM—the interscalene groove. Close to the first rib, trunks divide and reorganize as lateral, medial, and posterior cords at the level of the second part of the axillary artery [47]. More distally, the cords split into the terminal branches of the BP. Ultrasonography helps to visualize anatomical variants [5, 48] (solitary trunk, postfixed plexus, or abnormal C5 and C6 relation toward

Table 30.4 Prospect group recommendations for arthroscopic rotator cuff repair

Timing	Intervention		Grade
Pre- and intraoperative	Paracetamol or acetaminophen		D
	COX-2-specific inhibitor		D
	Dexamethasone IV		B
	Regional analgesia	CISB	A
		SS-ISB	A
		SSN (± AxN)	B
Postoperative	Paracetamol or acetaminophen		D
	NSAIDs or COX-2 inhibitors		D
	Opioid on demand (for breakthrough pain)		B

Grades of recommendation: Grade A, based on >2 studies or a single, large, well-designed study RCT, level of evidence (LoE) 1; Grade B, extrapolation from 1 procedure-specific, LoE 1; Grade C, based on non-systematic review, cohort study, case study; LoE 3; Grade D, based on clinical practice or expert opinion; LoE 4

Table 30.6 Roots, plexus, and nerves involved in shoulder joint and surrounding skin innervation

Brachial plexus (BP)	Suprascapular N (SSN)	Upper trunk; C5–C6
	Axillary N (AxN)	Posterior cord; C5–C6
	Subscapularis N (NS)	
	Lateral pectoral N (LPN)	Lateral cord; C5–C6
Superficial cervical plexus	Supraclavicular N	C3–C4
Intercostobrachial N		T1–T2
Joint		*Nerves*
GHJ		SSN, NS AxN
ACJ		LPN and acromial branch from SSN

N nerve, *GHJ* glenohumeral joint, *ACJ* acromioclavicular joint

Table 30.5 Perineural (pn) or systemic (IV) adjuvants to RA

	Alfa 2-agonists		
	Clonidine	Dexmedetomidine	Dexamethasone
Dose	pn – 150 µg	pn – 50–60 µg IV – 1–2 µg/kg	pn – 4 mg (max) IV – 0.1–0.2 mg/kg (max 8 mg)
Analgesia duration (h)	2 (pn)	5 (pn, IV)	6 (pn—short- to intermediate-acting LA) 8 (pn—long-acting LA) 3 (IV)
Sensory block duration (h)	1	4 (pn, IV)	7 (pn)
Motor block duration (h)	2 (pn)	3 (pn)	4 (pn)

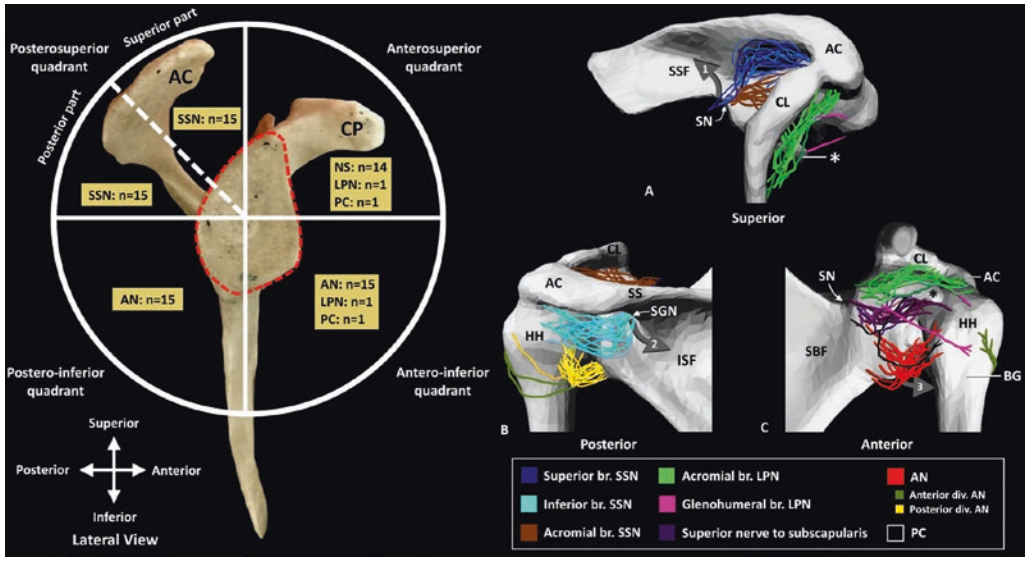


Fig. 30.4 Innervation patters of glenohumeral and acromioclavicular joints. *AC* acromion process, *ACJ* acromioclavicular joint, *AN* axillary nerve, *BG* bicipital groove, *br* branch, *CL* clavicle, *CP* coracoid process, *div* division, *GHJ* glenohumeral joint, *HH* humeral head, *ISF* infraspinous fossa, *LPN* lateral pectoral nerve, *SBF* subscapular fossa, *SGN* spinoglenoid notch, *SN* suprascapular notch, *SS* spine of scapula, *SSF* supraspinous fossa, *SSN* suprascapular nerve; ∗, coracoid process. Reprinted with permission from Philip Peng Educational Series

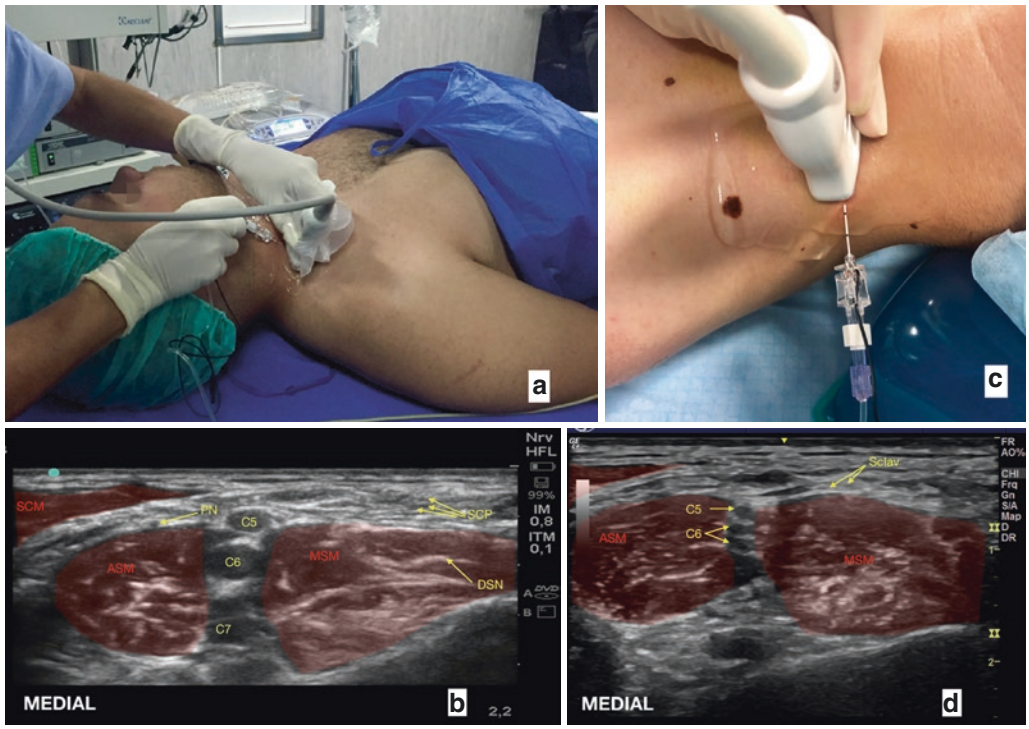


Fig. 30.5 (**a**, **b**) Interscalene block position and sonoanatomy; (**c**, **d**) Superior trunk block positioning and sonoanatomy. *ASM* anterior scalene muscle, *MSM* middle scalene muscle, *SCM* sternocleidomastoid muscle, *C5* C5 root, *C6* C6 root, *C7* C7 root, *DSC* dorsoscapular nerve, *PN* phrenic nerve, *SCP* superficial cervical plexus, *Sclav* supraclavicular nerves

the ASM not residing within the interscalene groove) and warrants a successful block.

The *phrenic nerve's* (PN) main origin is C4 ventral ramus, with contributions from C3 and C5; an accessory phrenic nerve may arise from roots C5–C6. At the C5 and C6 levels, PN and BP are close to each other (mean distance of 0.18 cm), and, as they move distally, their distance increases (mean distance is 1.08 cm, 3 cm down C5–C6) [49]. Several arteries have an intimate relation with the neural structures of the BP at the interscalene (90%) and supraclavicular (86%) areas [50]. Their recognition is important as landmarks (e.g., supraclavicular artery) or as structures to avoid (e.g., vertebral artery). *Vertebral artery* is anterior to the nerve roots and medial to the ASM, crossing the vertebral foramen of cervical transverse processes. Subcutaneously, the **external jugular vein** can cross the roots/trunks of the BP at the level of C6 as it overlies, although inconsistently, the interscalene groove [5]. The divisions of the BP (i.e., bundle of grapes) run posterior and superficial to the **subclavian artery** at the supraclavicular area [51]. **Posterior cervical artery**, a branch of the costocervical artery, courses posterior, between C7 and C8 roots, and the **transverse cervical artery**, a branch of the thyrocervical trunk, is close to, or even lie between, the trunks or divisions of the BP [52].

Dorsoscapular and suprascapular arteries are commonly close to the supraclavicular plexus, often coursing directly through it [50]. **Cervical transverse processes (TP) C6 and C7** are different from the others: TP C6 has a prominent anterior tubercle (Chassaignac tubercle), C6 nerve root is found posterolateral to it in transverse view emerging between the anterior and posterior tubercles, with a typical *fishmouth* appearance on ultrasound image, and TP C7 has a prominent posterior tubercle and absent or rudimentary anterior tubercle [53].

30.4 General Anesthesia

The patient in the BCP under GA has several factors influencing CBF and rScO2 (Table 30.2). Anesthetic agents influence cerebral metabolism and hemodynamics differently; some of them preserve CBF and cerebral metabolic rate better than others. Sevoflurane-nitrous oxide has a better margin of safety against impaired cerebral oxygenation compared propofol-remifentanil [54]. In another study, ventilation strategy was more determinant in $rScO_2$ than the anesthetic choice, as no difference was detected between patients randomized to receive desflurane or total intravenous anesthesia with propofol [55].

Furthermore, patients experiencing CDEs, under GA, had a worse performance on all neurobehavioral tests 24 h after surgery compared to patients without CDE, as reported by Aguirre et al. [56].

30.5 Regional Techniques

Regional techniques can be used alone or in combination with (conscious or deep) sedation or GA (Table 30.7) [57].

RA has many advantages and, according to the current thinking, should feature on MMA [6] (Fig. 30.1).

For this review, the author CL describes briefly RA techniques, emphasizing the positive and negative aspects of each one and suggesting dose regimens based on her clinical practice. LA selection and best dosing (volume and concentration) for each RA approach are still to be determined. Detailed RA techniques can be reviewed elsewhere [10].

30.5.1 Interscalene Block

Interscalene block is the gold standard and cost-effective technique for shoulder procedures [58, 59]. However, as any paravertebral approach, has risk of serious complications and should be performed by experienced practitioners [3], using PNS and/or USG (Fig. 30.5). It is important to identify and avoid the dorsoscapular and long thoracic nerves when needling through MSM (Fig. 30.5b).

Table 30.7 Summary of regional techniques for shoulder surgery

	RA technique	Aim	Patient positioning	Equipment	Dosing	Advantages	Disadvantages
Awake surgery	ISB	C5–C6 roots	Supine, 30° elevation, slight contralateral head rotation	12–15 Hz transducer; short beveled needles, <50 mm	20–10 mL	Superior intra- and postoperative analgesia; good for awake surgery; allows continuous technique	PN involvement and hemidiaphragmatic paralysis; vascular puncture and pneumothorax risk; catheter dislodgement occurs frequently; motor weakness of upper limb
	ST block	Superior trunk			10–15 mL	Similar to ISB; less PNP	Vascular puncture and pneumothorax risk
	SCB	Brachial plexus	Supine, 30° elevation or a pillow under the ipsilateral shoulder, slight contralateral head rotation		20–30 mL	Similar to ISB	Phrenic nerve involvement and hemidiaphragmatic paralysis (less than ISB); vascular puncture and pneumothorax risks; upper limb motor weakness
Combined with general anesthesia	Shoulder block	SSN block + AxN block	Supine, siting, or lateral position	High−/low-frequency US transducer considering patient *habitus*; short beveled needle, 50–100 mm	10–15 mL/nerve	Good post-op analgesia for shoulder arthroscopy (partial block compared to the other techniques); no upper limb motor weakness associated with higher patient satisfaction; no PNP	Requires two separate punctures; higher pain scores and morphine consumption compared to ISB
	ESB	T2/C7 transverse process	Sitting or lateral decubitus		20 mL	Seems to have good analgesia and no motor weakness	Small number of case reports

30.5.1.1 Continuous Versus Single-Shot ISB

For long-lasting severe pain, continuous block (CISB) is the better choice [33, 34]. Placing the LA through the catheter to induce the block is a simple method of confirm the functionality of catheter and plexus block [34] and limiting advancement of a non-stimulating CISB—no more than 1–2 cm past the needle tip is recommended. Catheter fixation is difficult due to the mobile nature of the surrounding area, superficial placement, local leakage, and adjacent hair follicles, making dislodgment easy. Strategies to manage catheters include topical medical cyanoacrylate at the catheter entry site, catheter tunneling, and the use of a simple epidural catheter securing device combined with a clear occlusive dressing and nonwoven fabric [3]. The best LA agent, volume, and concentration for interscalene infusions remain unknown. Usually, low-concentration infusion is preferable to avoid dense motor block, for example, ropivacaine 0.1–0.2%, 5–8 mL/h, by a portable infusion pump. If using a PCRA CISB, basal infusion can be optimized with a bolus of 5–8 mL every 30–60 min.

30.5.1.2 Adverse Effects and Complications

ISB is associated with a 100% phrenic nerve (PN) involvement with consequent hemidiaphragmatic paralysis. Mild dyspnea is the most common symptom (7%) [6], but respiratory compromise and respiratory support [11] may be needed. PN dysfunction incidence has been estimated to be 1:2000 cases, and it is mainly associated with local inflammation [60]. Transient postoperative neurological symptoms that are ISB-related are relatively common (8–14%) at day 10 and decrease to 2–4% 1 month later, and neurological sequelae lasting more than 6 months are exceedingly rare [3, 10]. Hypotension and bradycardia (Bezold-Jarisch reflex) are frequent and occur around 30 min after the block; hoarseness (4%) and Horner's syndrome (7–50%) are minor events but can be distressing to the unwarned patient [10, 48]. Pneumothorax and local anesthetic systemic toxicity (LAST), due to absorption or intravascular injection, are rare events. ISB prevents patient's neurologic function assessment after the surgical procedure [61]. There have been some case reports of catastrophic events associated with ISB [62]. Perineural catheters are associated with a high percentage of inadequate analgesia, wrong catheter placement and secondary block failure, dislodgement, knotting, migration, obstruction, leakage, bacterial colonization and risk of infection, dyspnea, lower lobe collapse, malfunction of the infusion device, myotoxicity, and LAST [20].

30.5.1.3 Strategies to Avoid Phrenic Nerve Block

Reducing LA dose and performing more distal blocks are important strategies [63] to avoid phrenic nerve paralysis (PNP). LA volume of 10 mL reduces the incidence of PNP to 13–60% [64–66], and further reduction to ≤5 mL has an even better positive impact (33–45%) [65, 67]. There are reports of no PN involvement [15, 68] using 5 mL of LA at the posterior border of the superior trunk (ST).

Superior trunk (ST) block is performed proximal to the point where the suprascapular nerve dives deep to the omohyoid muscle and avoids PN involvment [69] (Fig. 30.5c, d). The transverse cervical artery may lie across and superficial to the ST at this level and its presence should always be excluded before needle insertion, using the color Doppler tool. Placing LA above the MSM when removing the needle blocks the supraclavicular nerves of the SCP (Fig. 30.5d). For more details, follow the link https://youtu.be/938F1O90S34.

Supraclavicular block (SCB) may be an alternative with less risk of neurological symptoms and same success rate [18, 48]. However, PNP incidence is similar to ISB, and there is the risk of missing a more proximally departing suprascapular nerve [10]. A recent review found similar analgesia, morphine consumption, and less adverse events compared to ISB [70].

Shoulder block—SSN and AxN blocks—combined with GA is suitable for intra- and postoperative analgesia [33, 58]. The main advantages are no motor block, no PNP, higher patient satisfaction [71], and suitable if ISB is contraindicated or failed (as a rescue block). The main disadvantages are the need for two separate nerve block punctures and

incomplete blockade of all shoulder nerves [3]. SSN block improved pain control after shoulder arthroscopy, with minimal complications [18], compared to no nerve block but is less efficacious when compared to ISB [72]. Proximal SSN block involves placing the LA under the omohyoid muscle at the sub-omohyoid space, tracking it as it leaves C5 root (Fig. 30.6a). Distally, place the transducer at the suprascapular notch or across the supraspinous fossa [73] (Fig. 30.6b). As for the AxN, it can be reached anteriorly or posteriorly (Fig. 30.6c). The advantage of the anterior approach is to, theoretically, include more consistently the AxN articular branches and a better pain control [74]. The posterior AxN approach reaches the nerve at the quadrangular space (Fig. 30.6c, c1) [73].

30.5.2 Superficial Cervical Plexus Block/Supraclavicular Nerve Block

The SCP (Fig. 30.5b) can be an attractive technique to numb the skin over the shoulder and/or place an interscalene catheter [3].

30.5.3 Other Regional Anesthesia Techniques

Liposomal bupivacaine (LB) consists of multivesicular liposomes containing bupivacaine that prolong the block duration of action and delay bupivacaine peak plasma concentration due to a slow and controlled release from the liposomes [75]. LB can be used to block peripheral nerves and/or local infiltration by the surgeon. Several authors [76–79] found similar pain scores between standard ISB and LB infiltration, although ISB is better within 4 h after TSA [76], while LB infiltration group experienced significantly reduced rate of complications and hospital LOS after TSA. Associating LB infiltration with standard ISB resulted in better pain scores and reduced opioid for ARCR [80]. Reports of similar early block characteristics but significantly longer-lasting analgesia with the ISB when LB was added to bupivacaine HCl, led to the food and Drug Administration Agency approval of LB for interscalene analgesia for major shoulder procedures, in 2018 [81].

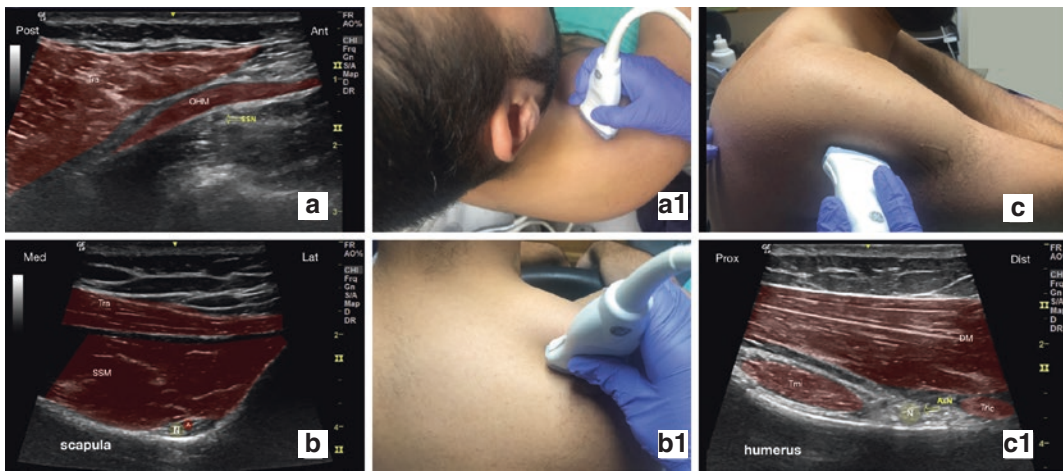


Fig. 30.6 Shoulder block: **a** and **a1**. SSN block anterior approach; patient is in supine or sitting position, and transducer is coronal oblique at the suprascapular fossa. Identify C5 root and track it caudally, looking for the SSN to branch with a latero-posterior trajectory, under the omohyoid muscle. **b** and **b1**. Posterior approach to the SSNB. Place the transducer along the long axis of the supraspinatus muscle (SSM) deep to the trapezius muscle (Tra) and look for the SSN (N) on the floor of the lateral aspect of the supraspinous fossa. **c** and **c1**. Posterior approach of the axillary nerve (N). The patient is in sitting, lateral decubitus, or supine position with upper limb adducted with the hand on the contralateral shoulder. The transducer is in plane with the humerus. Move the transducer cephalad; the AxN is close to the circumflex artery, under the deltoid muscle (DM) and inferior border of teres minor muscle (Tmi). *Tric* triceps muscle

Subacromial/intra-articular infiltration analgesia is no longer recommended as a result of effectiveness ambiguity, a rise in popularity of peripheral nerve blockade and several reports of catastrophic glenohumeral chondrolysis in healthy young patients (all received high/prolonged doses of intra-articular bupivacaine) [3, 33].

Erector spinae block (ESP) can be used for intra- and postoperative analgesia of shoulder arthroscopic [82] surgery, shoulder disarticulation [83], or TSA [84]. Aiming the TP of T2 or C7 posterior tubercle, the LA spreads toward the nerve roots of the BP and intercostal nerves, preferentially blocking C fibers allowing a motor-sparing block [82, 84]. There is a risk of vertebral artery injury or PNP, if aiming to the TP C7 [83].

30.5.4 Avoiding Wrong-Sided Block

Campaigns like "Stop Before You Block" [85] introduced a time-out before puncturing the patient, identifying the side to be blocked and preventing blocking the wrong side. Nevertheless, the number of wrong-sided blocks did not decrease as expected. Preoperative involvement of both surgeon and anesthesiologist in the identification of the limb to be blocked while in the block room or preoperative area could have an important role in reducing this complication incidence.

30.5.5 Discharge Criteria and Limb Protection

The odds of bypassing the post-anesthesia care unit (PACU) is higher for patients using RA compared to GA [34].

Patients discharged home with an insensate limb or a continuous block should receive clear, simple, easy-to-understand instructions, know when and how to contact the anesthesia staff member, and feel comfortable with the treatment plan [86]. Patient and caregiver should receive oral and written instructions on how to protect the numb limb, recognize possible complications, when to expect the block to recede and are strongly advised to take the systemic pain medication as prescribed before block resolution (usually the block wears off suddenly) [20]. If using catheters, explain how to stop the pump temporarily in case of excessive numbness, keep the catheter area clean, and observe signs of infection or leakage. Pain team should call daily for the duration of therapy.

Inpatients should be referred to the pain unit [34] for follow-up, evaluation, and therapy adjustments.

30.5.6 Patient Satisfaction and Regional Anesthesia

The discomfort and pain associated with placing a block can be improved with sedation [19] or music [87]. Patient satisfaction is proportional to the information received, interaction with the anesthesiologist, and comfort/painless block technique [88].

30.6 Main Strategies for a Good Outcome After Shoulder Surgery

30.6.1 Position

Avoid BCP if possible. Lateral decubitus position has less negative impact in hemodynamics and cerebral oximetry.

Use the lower degree of BCP elevation possible.

The head should be immobilized in a neutral position (no flexion, extension, rotation, or twist).

30.6.2 Respiratory

Avoid hyperventilation during GA as there is a linear relationship between $ETCO_2$ and $rScO_2$. In diabetic patients, this relation is attenuated. However, hypoventilation can be harmful because hypercapnia worsens events of focal

cerebral ischemia as it potentiates intracerebral steal.

If possible, avoid positive pressure ventilation with GA (prefer spontaneous ventilation).

Use high O_2 inspired fraction, especially in patients with basic low levels of $rScO_2$.

30.6.3 Cardiovascular

Keep blood pressure close to baseline levels and use the contralateral upper arm to place the blood pressure cuff.

Ephedrine should be the drug of choice to treat hypotension and avoid cerebral desaturation.

Use intermittent lower limb compressive devices to avoid blood pooling and reduce hypotension.

30.6.4 Neurologic

Cerebral saturation level monitors (e.g., NIRS) can provide precious information and an opportunity to identify and treat cerebral ischemia before a catastrophic permanent event occurs.

Sudden low BIS levels and/or burst suppression might suggest cerebral ischemia.

Middle cerebral artery flow velocity can dynamically monitor cerebral perfusion.

30.6.5 Type of Anesthesia

Regional anesthesia with sedation has a better safety profile, as it decreases the cardiovascular consequences of BCP and associated adverse effects.

Regional anesthesia should play a major role on MMA regimens, as long as there are no contraindications.

ISB is the gold standard for shoulder procedures as anesthesia and analgesia and recommended for ARCR. However, there are other techniques, associated with good analgesic control, patient satisfaction, and less complications as shoulder block and superior trunk block.

General anesthesia is associated with higher incidence of CDEs, especially with positive pressure ventilation (and hypocapnia) and worse performance on all neurobehavioral tests 24 h after surgery.

Ventilation strategy is more important for maintaining $rScO_2$ than the anesthetic agents chosen. Hypercapnia is frequent with sedation, lowering the incidence of CDEs.

30.6.6 Postoperative Analgesia

Discuss with the patient and caregiver the anesthetic and analgesic care planned, especially when it involves awake surgery and/or ambulatory perineural catheters.

Provide adequate information and written instructions for the patient with an insensate limb.

Choose MMA regimens to reduce opioid consumption and better pain control.

Perineural adjuvants can reduce opioid requirements and better pain management. Perineural catheters are suitable and recommended for long-lasting severe pain procedures, as ARCR.

Strongly advise the patient to follow the MMA plan before the block wears off.

References

1. Warrender WJ, Syed UAM, Hammoud S, et al. Pain management after outpatient shoulder arthroscopy: a systematic review of randomized controlled trials. Am J Sports Med. 2017;45(7):1676–86. https://doi.org/10.1177/0363546516667906.
2. Uquillas CA, Capogna BM, Rossy WH, Mahure SA, Rokito AS. Postoperative pain control after arthroscopic rotator cuff repair. J Shoulder Elb Surg. 2016;25(7):1204–13. https://doi.org/10.1016/j.jse.2016.01.026.
3. Fredrickson MJ, Krishnan S, Chen CY. Postoperative analgesia for shoulder surgery: a critical appraisal and review of current techniques. Anaesthesia. 2010;65(6):608–24. https://doi.org/10.1111/j.1365-2044.2009.06231.x.
4. Gerbershagen HJ, Aduckathil S, van Wijck AJM, Peelen LM, Kalkman CJ, Meissner W. Pain intensity on the first day after surgery. Anesthesiology. 2013;118(4):934–44. https://doi.org/10.1097/ALN.0b013e31828866b3.

5. Neal JM, Gerancher JC, Hebl JR, et al. Upper extremity regional anesthesia: essentials of our current understanding, 2008. Reg Anesth Pain Med. 2009;34(2):134–70. https://doi.org/10.1097/AAP.0b013e31819624eb.
6. Koepke EJ, Manning EL, Miller TE, Ganesh A, Williams DGA, Manning MW. The rising tide of opioid use and abuse: the role of the anesthesiologist. Perioper Med. 2018;7(1) https://doi.org/10.1186/s13741-018-0097-4.
7. American Society of Anesthesiologists. ASA physical status classification system. https://www.asahq.org/standards-and-guidelines/asa-physical-status-classification-system. 2014.
8. Zhao J, Guo Z, Liu X, Glaser DL, Elkassabany NM, Liu J. The development and application of a risk stratification index system for outpatient shoulder arthroscopy patient management—a single academic center's experience. JSES Open Access. 2017;1(1):1–4. https://doi.org/10.1016/j.jses.2017.03.005.
9. Berman JE, Mata-Fink A, Kassam HF, Blaine TA, Kovacevic D. Predictors of length of stay and discharge disposition after shoulder arthroplasty. J Am Acad Orthop Surg. 2019;27(15):e696–701. https://doi.org/10.5435/jaaos-d-18-00244.
10. Hewson DW, Oldman M, Bedforth NM. Regional anaesthesia for shoulder surgery. BJA Educt. 2019:1–7. https://doi.org/10.1016/j.bjae.2018.12.004.
11. Dirzu DS. Hemidiaphragmatic paresis versus block failure: the effect of anesthetic volume reduction for supraclavicular plexus block. Reg Anesth Pain Med. 2019;0(0):rapm-2019-100413. https://doi.org/10.1136/rapm-2019-100413
12. Tierney S, Perlas A. Informed consent for regional anesthesia. Curr Opin Anaesthesiol. 2018;31(5):614–21. https://doi.org/10.1097/ACO.0000000000000635.
13. Anesthesia C of R. Statement on regional anesthesia committee. ASA Statements. 2017;(6):67–72.
14. Guidelines C of S and P. Basic standards for preanesthesia care. ASA Stand Guid. 2015;151:10–7. https://doi.org/10.1145/3132847.3132886.
15. Smith HM, Duncan CM, Hebl JR. Clinical utility of low-volume ultrasound-guided interscalene blockade. J Ultrasound Med. 2009;28(9):1251–8. https://doi.org/10.7863/jum.2009.28.9.1251.
16. Mejía-Terrazas GE, Ruíz-Suárez M, Vadillo-Ortega F, Franco y Bourland RE, López-Muñoz E. Effect of interscalene nerve block on the inflammatory response in shoulder surgery: a randomized trial. J Shoulder Elb Surg. 2019; https://doi.org/10.1016/j.jse.2019.02.030.
17. Gadsden J, McCally C, Hadzic A. Monitoring during peripheral nerve blockade. Curr Opin Anaesthesiol. 2010;23(5):656–61. https://doi.org/10.1097/ACO.0b013e32833d4f99.
18. Sripada R, Bowens C. Regional anesthesia procedures for shoulder and upper arm surgery upper extremity update—2005 to present. Int Anesthesiol Clin. 2012;50(1):26–46.
19. Kubulus C, Schmitt K, Albert N, et al. Awake, sedated or anaesthetised for regional anaesthesia block placements? Eur J Anaesthesiol. 2016;33:1. https://doi.org/10.1097/EJA.0000000000000495.
20. Sulaiman L, Macfarlane RJ, Waseem M. Current concepts in anaesthesia for shoulder surgery. Open Orthop J. 2013;3:323–8.
21. Murphy GS, Greenberg SB, Szokol JW. Safety of beach chair position shoulder surgery. Anesth Analg. 2019;129(1):101–18. https://doi.org/10.1213/ANE.0000000000004133.
22. Gilotra MN, Klein A, Elkassabany N, Kuntz AF, Huffman R, Glaser DL. Risk factors for cerebral desaturation events during shoulder surgery in the beach chair position. Arthrosc J Arthrosc Relat Surg. 2018;1:1–6. https://doi.org/10.1016/j.arthro.2018.10.123.
23. Cardim D, Robba C, Matta B, et al. Cerebrovascular assessment of patients undergoing shoulder surgery in beach chair position using a multiparameter transcranial Doppler approach. J Clin Monit Comput. 2019;33(4):615–25. https://doi.org/10.1007/s10877-018-0211-7.
24. Aguirre JA, Mohajeri MS, Rupnik B, et al. Early recognition of a transient ischemic attack utilizing cerebral oxygenation monitoring during shoulder surgery in the beach chair position. A A Pract. 2019;13(5):1. https://doi.org/10.1213/xaa.0000000000001027.
25. Orebaugh S, Palmeri S, Lin C, YaDeau J. Daring discourse: is nerve block with sedation the safest anesthetic for beach chair position? Reg Anesth Pain Med Pain Med. 2019;44(7):707–12. https://doi.org/10.1136/rapm-2018-100230.
26. Robba C, Goffi A, Geeraerts T, et al. Brain ultrasonography: methodology, basic and advanced principles and clinical applications. A narrative review. Intensive Care Med. 2019;45(7):913–27. https://doi.org/10.1007/s00134-019-05610-4.
27. Aliane J, Dualé C, Guesmi N, et al. Compared effects on cerebral oxygenation of ephedrine vs phenylephrine to treat hypotension during carotid endarterectomy. Clin Exp Pharmacol Physiol. 2017;44(7):739–48. https://doi.org/10.1111/1440-1681.12759.
28. Meng L, Gelb AW, Alexander BS, et al. Impact of phenylephrine administration on cerebral tissue oxygen saturation and blood volume is modulated by carbon dioxide in anaesthetized patients. Br J Anaesth. 2012;108(5):815–22. https://doi.org/10.1093/bja/aes023.
29. Moerman AT, De Hert SG, Jacobs TF, De Wilde LF, Wouters PF. Cerebral oxygen desaturation during beach chair position. Eur J Anaesthesiol. 2012;29(2). https://journals.lww.com/ejanaesthesiology/Fulltext/2012/02000/Cerebral_oxygen_desaturation_during_beach_chair.5.aspx
30. Meex I, Vundelinckx J, Buyse K, et al. Cerebral tissue oxygen saturation values in volunteers and patients in the lateral decubitus and beach chair positions: a prospective observational study. Can J

Anesth. 2016;63(5):537–43. https://doi.org/10.1007/s12630-016-0604-3.
31. Memtsoudis SG, Poeran J, Zubizarreta N, et al. Association of multimodal pain management strategies with perioperative outcomes and resource utilization. Anesthesiology. 2018;128(5):891–902.
32. Pitchon DN, Dayan AC, Schwenk ES, Baratta JL, Viscusi ER. Updates on multimodal analgesia for orthopedic surgery. Anesthesiol Clin. 2018;36(3):361–73. https://doi.org/10.1016/j.anclin.2018.05.001.
33. Toma O, Persoons B, Pogatzki-Zahn E, et al. PROSPECT guideline for rotator cuff repair surgery: systematic review and procedure-specific postoperative pain management recommendations. Anaesthesia. 2019:anae.14796. https://doi.org/10.1111/anae.14796
34. Grosh T, Elkassabany NM. Enhanced recovery after shoulder arthroplasty. Anesthesiol Clin. 2018;36(3):417–30. https://doi.org/10.1016/j.anclin.2018.04.006.
35. Desai N, Albrecht E, El-Boghdadly K. Perineural adjuncts for peripheral nerve block. BJA Educt. 2019;19(9):276–82. https://doi.org/10.1016/j.bjae.2019.05.001.
36. Kang R, Jeong JS, Yoo JC, et al. Effective dose of intravenous dexmedetomidine to prolong the analgesic duration of interscalene brachial plexus block: "a single-center, prospective, double-blind, randomized controlled trial". Reg Anesth Pain Med. 2018;43(5):488–95. https://doi.org/10.1097/AAP.0000000000000773.
37. Vorobeichik L, Brull R, Abdallah FW. Evidence basis for using perineural dexmedetomidine to enhance the quality of brachial plexus nerve blocks: a systematic review and meta-analysis of randomized controlled trials. Br J Anaesth. 2017;118(2):167–81. https://doi.org/10.1093/bja/aew411.
38. Andersen JH, Jaeger P, Grevstad U, et al. Systemic dexmedetomidine is not as efficient as perineural dexmedetomidine in prolonging an ulnar nerve block. Reg Anesth Pain Med. 2019;44(3):333–40. https://doi.org/10.1136/rapm-2018-100089.
39. Aliste J, Layera S, Bravo D, et al. Randomized comparison between perineural dexamethasone and dexmedetomidine for ultrasound-guided infraclavicular block. Reg Anesth Pain Med. 2019:rapm-2019-100680. https://doi.org/10.1136/rapm-2019-100680
40. El-Boghdadly K, Brull R, Sehmbi H, Abdallah FW. Perineural dexmedetomidine is more effective than clonidine when added to local anesthetic for supraclavicular brachial plexus block: a systematic review and meta-analysis. Anesth Analg. 2017;124(6). https://journals.lww.com/anesthesia-analgesia/Fulltext/2017/06000/Perineural_Dexmedetomidine_Is_More_Effective_Than.40.aspx
41. Albrecht E, Vorobeichik L, Jacot-Guillarmod A, Fournier N, Abdallah FW. Dexamethasone is superior to dexmedetomidine as a perineural adjunct for supraclavicular brachial plexus block: systematic review and indirect meta-analysis. Anesth Analg. 2019;128(3). https://journals.lww.com/anesthesia-analgesia/Fulltext/2019/03000/Dexamethasone_Is_Superior_to_Dexmedetomidine_as_a.21.aspx
42. Kang RA, Jeong JS, Yoo JC, et al. Improvement in postoperative pain control by combined use of intravenous dexamethasone with intravenous dexmedetomidine after interscalene brachial plexus block for arthroscopic shoulder surgery: a randomised controlled trial. Eur J Anaesthesiol. 2019;36(5). https://journals.lww.com/ejanaesthesiology/Fulltext/2019/05000/Improvement_in_postoperative_pain_control_by.7.aspx
43. Gurger M, Ozer AB. A comparison of continuous interscalene block versus general anesthesia alone on the functional outcomes of the patients undergoing arthroscopic rotator cuff repair. Eur J Orthop Surg Traumatol. 2019; https://doi.org/10.1007/s00590-019-02482-8.
44. Codding JL, Getz CL. Pain management strategies in shoulder arthroplasty. Orthop Clin North Am. 2018;49(1):81–91. https://doi.org/10.1016/j.ocl.2017.08.010.
45. Tran J, Peng PWH, Agur AMR. Anatomical study of the innervation of glenohumeral and acromioclavicular joint capsules: implications for image-guided intervention. Reg Anesth Pain Med. 2019:rapm-2018-100152. https://doi.org/10.1136/rapm-2018-100152
46. Musso D, Flohr-Madsen S, Meknas K, Wilsgaard T, Ytrebø LM, Klaastad Ø. A novel combination of peripheral nerve blocks for arthroscopic shoulder surgery. Acta Anaesthesiol Scand. 2017;61(9):1192–202. https://doi.org/10.1111/aas.12948.
47. Mian A, Chaudhry I, Huang R, Rizk E, Tubbs RS, Loukas M. Brachial plexus anesthesia: a review of the relevant anatomy, complications, and anatomical variations. Clin Anat. 2014;27(2012):210–21. https://doi.org/10.1002/ca.22254.
48. Halaszynski TM. Ultrasound brachial plexus anesthesia and analgesia for upper extremity surgery: essentials of our current understanding. Curr Opin Anaesthesiol. 2011;24(5):581–91. https://doi.org/10.1097/ACO.0b013e32834aca03.
49. Kessler J, Schafhalter-Zoppoth I, Gray AT. An ultrasound study of the phrenic nerve in the posterior cervical triangle: implications for the interscalene brachial plexus block. Reg Anesth Pain Med. 2008;33(6):545–50. https://doi.org/10.1136/rapm-00115550-200811000-00006.
50. Muhly WT, Orebaugh SL. Sonoanatomy of the vasculature at the supraclavicular and interscalene regions relevant for brachial plexus block. Acta Anaesthesiol Scand. 2011;55(10):1247–53. https://doi.org/10.1111/j.1399-6576.2011.02528.x.
51. van Geffen GJ, Moayeri N, Bruhn J, Scheffer GJ, Chan VW, Groen GJ. Correlation between ultrasound imaging, cross-sectional anatomy, and histology of the brachial plexus: a review. Reg Anesth

Pain Med. 2009;34(5):490–7. https://doi.org/10.1097/AAP.0b013e3181add8a3.
52. Orebaugh SL, B a W. Brachial plexus anatomy: normal and variant. Scientific World J. 2009;9:300–12. https://doi.org/10.1100/tsw.2009.39.
53. Soeding P, Eizenberg N. Review article: anatomical considerations for ultrasound guidance for regional anesthesia of the neck and upper limb. Can J Anaesth. 2009;56(7):518–33. https://doi.org/10.1007/s12630-009-9109-7.
54. Jeong H, Jeong S, Lim HJ, Lee J, Yoo KY. Cerebral oxygen saturation measured by near-infrared spectroscopy and jugular venous bulb oxygen saturation during arthroscopic shoulder surgery in beach chair position under sevoflurane-nitrous oxide or propofol-remifentanil anesthesia. Anesthesiology. 2012;116(5):1047–56. https://doi.org/10.1097/ALN.0b013e31825154d2.
55. Picton P, Dering A, Alexander A, et al. Influence of ventilation strategies and anesthetic techniques on regional cerebral oximetry in the beach chair position: a prospective interventional study with a randomized comparison of two anesthetics. Anesthesiology. 2015;123(4):765–74. https://doi.org/10.1097/ALN.0000000000000798.
56. Aguirre JA, Etzensperger F, Brada M, et al. The beach chair position for shoulder surgery in intravenous general anesthesia and controlled hypotension: impact on cerebral oxygenation, cerebral blood flow and neurobehavioral outcome. J Clin Anesth. 2018;2019(53):40–8. https://doi.org/10.1016/j.jclinane.2018.09.035.
57. Bosco L, Zhou C, Murdoch JAC, et al. Pre- or postoperative interscalene block and/or general anesthesia for arthroscopic shoulder surgery: a retrospective observational study. Can. J. Anaesth. 2017;64(10):1048–58. https://doi.org/10.1007/s12630-017-0937-6.
58. Bowens C, Sripada R. Regional blockade of the shoulder: approaches and outcomes. Anesthesiol Res Pract. 2012;2012:1–12. https://doi.org/10.1155/2012/971963.
59. Hughes MS, Matava MJ, Wright RW, Brophy RH, Smith MV. Interscalene brachial plexus block for arthroscopic shoulder surgery. A systematic review. J Bone Joint Surg Am. 2013;95(14):1318–24. https://doi.org/10.1016/S0021-9355(13)72333-6.
60. Hogan QH. Phrenic nerve function after interscalene block revisited: now, the long view. Anesthesiol J Am Soc Anesthesiol. 2013;119(2):250–2. https://doi.org/10.1097/ALN.0b013e31829c2f3a.
61. Gupta R. Think again about that block: commentary on an article by Surena Namdari, MD, MSc, et al.: "Randomized controlled trial of interscalene block compared with injectable liposomal bupivacaine in shoulder arthroplasty". J Bone Joint Surg Am. 2017;99(7):e35. https://doi.org/10.2106/JBJS.16.01584.
62. Benumof J. Permanent loss of cervical spinal cord function associated with interscalene block performed under general anesthesia. Anesthesiology. 2000;93:1541–4. http://journals.lww.com/anesthesiology/Abstract/2000/12000/Permanent_Loss_of_Cervical_Spinal_Cord_Function.33.aspx. Accessed 31 Aug 2012
63. Verelst P, van Zundert A. Respiratory impact of analgesic strategies for shoulder surgery. Reg Anesth Pain Med. 2013;38(1):50–3. https://doi.org/10.1097/AAP.0b013e318272195d.
64. Sinha SK, Abrams JH, Barnett JT, et al. Decreasing the local anesthetic volume from 20 to 10 ml for ultrasound-guided interscalene block at the cricoid level does not reduce the incidence of hemidiaphragmatic paresis. Reg Anesth Pain Med. 2011;36(1):17–20. https://doi.org/10.1097/AAP.0b013e3182030648.
65. Lee J-H, Cho S-H, Kim S-H, et al. Ropivacaine for ultrasound-guided interscalene block: 5 mL provides similar analgesia but less phrenic nerve paralysis than 10 mL. Can J Anesth. 2011;58(11):1001. https://doi.org/10.1007/s12630-011-9568-5.
66. Renes SH, Rettig HC, Gielen MJ, Wilder-Smith OH, van Geffen GJ. Ultrasound-guided low-dose interscalene brachial plexus block reduces the incidence of hemidiaphragmatic paresis. Reg Anesth Pain Med. 2009;34(5):498–502. https://doi.org/10.1097/AAP.0b013e3181b49256.
67. Riazi S, Carmichael N, Awad I, Holtby RM, McCartney CJL. Effect of local anaesthetic volume (20 vs 5 ml) on the efficacy and respiratory consequences of ultrasound-guided interscalene brachial plexus block. Br J Anaesth. 2008;101(4):549–56. https://doi.org/10.1093/bja/aen229.
68. Jack NTM, Renes SH, Bruhn J, van Geffen GJ. Phrenic nerve-sparing ultrasound-guided interscalene brachial plexus block in a patient with a contralateral pneumonectomy. Reg Anesth Pain Med. 2009;34(6):618. https://doi.org/10.1097/AAP.0b013e3181b4d0f1.
69. Burckett-St.Laurent D, Chan V, Chin KJ. Raffinement du bloc interscalénique échoguidé du plexus brachial: la voie de la branche supérieure. Can J Anesth. 2014;61(12):1098–102. https://doi.org/10.1007/s12630-014-0237-3.
70. Schubert A-K, Dinges H-C, Wulf H, Wiesmann T. Interscalene versus supraclavicular plexus block for the prevention of postoperative pain after shoulder surgery: a systematic review and meta-analysis. Eur J Anaesthesiol. 2019;36(6). https://journals.lww.com/ejanaesthesiology/Fulltext/2019/06000/Interscalene_versus_supraclavicular_plexus_block.7.aspx
71. Pani N, Routray SS, Pani S, Mallik S, Pattnaik S, Pradhan A. Post-operative analgesia for shoulder arthroscopic surgeries: a comparison between interscalene block and shoulder block. Indian J Anaesth. 2019;63(5):382–7. https://doi.org/10.4103/ija.IJA_65_19.
72. Hu T, Athwal G, Kay J, et al. Suprascapular nerve blockade for postoperative pain control after arthroscopic shoulder surgery: a systematic review and meta-analysis. Orthop J Sport Med. 2018;6(12):232596711881585. https://doi.org/10.1177/2325967118815859.

73. Dhir S, Sondekoppam RV, Sharma R, Ganapathy S, Athwal GS. A comparison of combined suprascapular and axillary nerve blocks to interscalene nerve block for analgesia in arthroscopic shoulder surgery: an equivalence study. Reg Anesth Pain Med. 2016;41(5):564–71. https://doi.org/10.1097/AAP.0000000000000436.
74. González-Arnay E, Jiménez-Sánchez L, García-Simón D, et al. Ultrasonography-guided anterior approach for axillary nerve blockade: an anatomical study. Clin Anat. 2019; https://doi.org/10.1002/ca.23394.
75. Grant GJ, Barenholz Y, Bolotin EM, et al. A novel liposomal bupivacaine formulation to produce ultralong-acting analgesia. Anesthesiology. 2004;101(1):133–7. https://doi.org/10.1097/01.sa.0000151238.77039.f4.
76. Sun H, Li S, Wang K, et al. Do liposomal bupivacaine infiltration and interscalene nerve block provide similar pain relief after total shoulder arthroplasty: a systematic review and meta-analysis. J Pain Res. 2018;11:1889–900. https://doi.org/10.2147/JPR.S177716.
77. Cao X, Pan F. Comparison of liposomal bupivacaine infiltration versus interscalene nerve block for pain control in total shoulder arthroplasty: a meta-analysis of randomized control trails. Medicine (Baltimore). 2017;96(39):e8079. https://doi.org/10.1097/MD.0000000000008079.
78. Yan Z, Chen Z, Ma C. Liposomal bupivacaine versus interscalene nerve block for pain control after shoulder arthroplasty: a meta-analysis. Medicine (Baltimore). 2017;96(27) https://doi.org/10.1097/MD.0000000000007226.
79. Wang K, xia ZH. Liposomal bupivacaine versus interscalene nerve block for pain control after total shoulder arthroplasty: a systematic review and meta-analysis. Int J Surg. 2017;46:61–70. https://doi.org/10.1016/j.ijsu.2017.08.569.
80. Sethi PM, Brameier D, Mandava NK, Miller SR. Liposomal bupivacaine reduces opiate consumption after rotator cuff repair in a randomized controlled trial. J Shoulder Elb Surg. 2019:1–9. https://doi.org/10.1016/j.jse.2019.01.008.
81. Balocco AL, Van Zundert PGE, Gan SS, Gan TJ, Hadzic A. Extended release bupivacaine formulations for postoperative analgesia: an update. Curr Opin Anaesthesiol. 2018;31(5):636–42. https://doi.org/10.1097/ACO.0000000000000648.
82. Selvi O, Tulgar S, Ozer Z. Case report presentation of ultrasound-guided erector spinae plane block in shoulder surgery: three patients and two different results. Cureus. 2018;10(11):e3538. https://doi.org/10.7759/cureus.3538.
83. Hamadnalla H, Elsharkawy H, Shimada T, Maheshwari K, Esa WAS, Tsui BCH. Cervical erector spinae plane block catheter for shoulder disarticulation surgery. Can J Anesth. 2019; https://doi.org/10.1007/s12630-019-01421-9.
84. Ma W, Sun L, Ngai L, et al. Motor-sparing high-thoracic erector spinae plane block for proximal humerus surgery and total shoulder arthroplasty surgery: clinical evidence for differential peripheral nerve block? Can J Anesth. 2019; https://doi.org/10.1007/s12630-019-01442-4.
85. French J, Bedforth N, Townsley P. Stop before you block campaign. 2011;2010:1–2. https://www.rcoa.ac.uk/sites/default/files/CSQ-PS-sbyb-supporting.pdf, http://www.rcoa.ac.uk/standards-of-clinical-practice/wrong-site-block.
86. Hunter OO, Kim TE, Mariano ER, Harrison TK. Care of the patient with a peripheral nerve block. J Perianesth Nurs. 2019;34(1):16–26. https://doi.org/10.1016/j.jopan.2018.01.006.
87. Graff V, Cai L, Badiola I, Elkassabany NM. Music versus midazolam during preoperative nerve block placements: a prospective randomized controlled study. Reg Anesth Pain Med. 2019;44(8):796–9. https://doi.org/10.1136/rapm-2018-100251.
88. Ironfield CM, Barrington MJ, Kluger R, Sites B. Are patients satisfied after peripheral nerve blockade? Results from an International Registry of Regional Anesthesia. Reg Anesth Pain Med. 2014;39(1):48–55. https://doi.org/10.1097/AAP.0000000000000038.
89. Drummond JC, Lee RR, Howell JP. Focal cerebral ischemia after surgery in the "beach chair" position: the role of a congenital variation of circle of Willis anatomy. Anesth Analg. 2012;114(6):1301–3. https://doi.org/10.1213/ANE.0b013e31823aca46.
90. Kirkham KR, Jacot-Guillarmod A, Albrecht E. Optimal dose of perineural dexamethasone to prolong analgesia after brachial plexus blockade: a systematic review and meta-analysis. Anesth Analg. 2018;126(1). https://journals.lww.com/anesthesia-analgesia/Fulltext/2018/01000/Optimal_Dose_of_Perineural_Dexamethasone_to.41.aspx

Part IV

Complex and Revision Problems

31 Traumatic Rotator Cuff Tears with Shoulder Stiffness

Carlos Maia Dias, João Sousa, and Tiago Paiva Marques

31.1 Traumatic Rotator Cuff Tears

Pure traumatic rotator cuff tears (RCT) seem to be rare but they have been described in the literature [1, 2] which has also shown us that the frequency of RCT increases with age [3, 4], so it seems consensual that even though trauma can have an important role in its aetiology, tendon degenerative changes predispose to it.

From the clinical point of view, degenerative/chronic rotator cuff tears can exist in asymptomatic patients and in the majority of cases they should be initially managed conservatively even when they progress to become symptomatic, which usually occurs in a few years [5]. The same attitude for the remaining RCT can be followed but acute on chronic (enlargement of a tendon tear after trauma or a different rotator cuff tendon tear in the setting of a pre-existent one) [6] or pure traumatic RCT can be very painful and debilitating [3, 7–9] and in a young and active population they may cause significant losses in patient wages and long periods of work absence [7, 8, 10].

Basset [6] in its classical 1985 paper showed that acute repair (3< weeks from injury) afforded the best opportunity for maximal recovery of shoulder function, although pain control and general satisfaction were achieved even if surgery was performed later. Other authors also showed that earlier repair was associated with better outcomes [11, 12].

31.1.1 Definition

Taking into account the clinical, social and legal implications some authors tried to discriminate some characteristic features of traumatic tears, finding that they usually affect males with an average age of 54.7 years and that a fall in an outstretched arm is the most common injury mechanism. Over 75% of cases report to medium to large tears [10], frequently involving the subscapularis and showing muscle edema (Fig. 31.1) [8, 10]. Tendon kinking (Fig. 31.2) also favours the acute setting while a positive tangent sign [13], muscle atrophy and moderate/severe fatty infiltration (>Stage 2) [14] were associated with chronic tears (Fig. 31.3).

31.2 The Problem

According to the previously mentioned literature, early surgical treatment for traumatic rotator cuff tears seems acceptable because surgical delay may cause irreversible tendon retraction

C. M. Dias (✉)
Hospital da Luz Lisboa / UCMA Fidelidade Lisboa / Hospital CUF Santarém, Lisboa, Portugal

J. Sousa
Centro Hospitalar do Oeste, Torres Vedras, Portugal

T. P. Marques
Hospital CUF Santarém, Santarém, Portugal

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_31

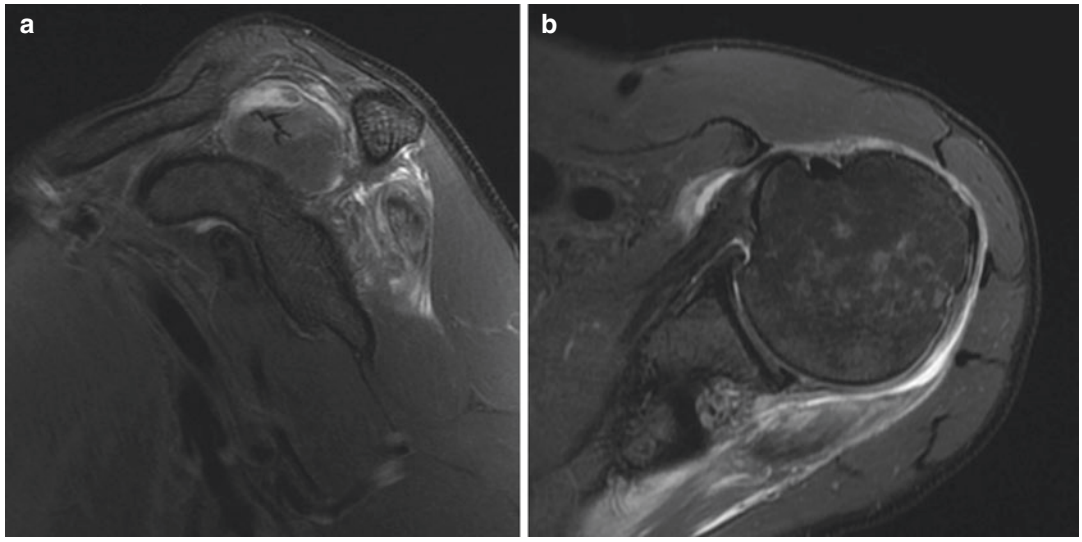


Fig. 31.1 (**a**, **b**) Infraspinatus muscle edema in an acute rupture (**a**) SAG T2; (**b**) AX T2

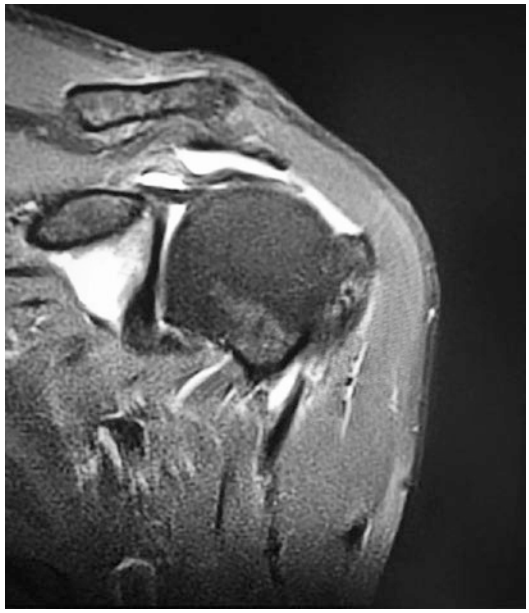


Fig. 31.2 Supraspinatus tendon kinking (COR T2)

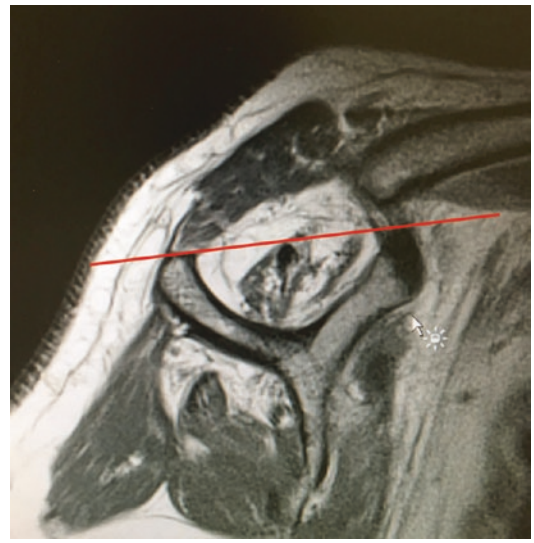


Fig. 31.3 Supraspinatus muscle atrophy, stage 3 fatty infiltration and positive tangent sign (SAG T1)

and tear enlargement as well as muscle atrophy, fatty infiltration and tissue thinning that can compromise its repair and definitive healing [3, 8, 15–17].

Unfortunately, rotator cuff tears often are associated with some degree of stiffness, which can also be very debilitating, and trauma seems to be one of the risk factors for this association [18] as well as Diabetes mellitus [9, 19, 20]. Tear pattern (bursal sided and full thickness) and direction (postero-superior) can also correlate with the appearance of concomitant stiffness [18].

This can occur between 10.9 and 41.7% of cases [21–24] and has been a classical reason for surgical delay in these patients because RCT repair is considered a shoulder-tightening proce-

dure with a postoperative rate of stiffness that can range from 4.9 to 32.7% [18, 25, 26] and there is data supporting preoperative stiffness as a risk factor for its development [20, 26, 27].

31.2.1 Defining Stiffness

When evaluating this difficult combination, one of the main problems lies in defining stiffness [28]. Different authors have different criteria (Table 31.1) and this may explain the discrepancy that is evident in the literature regarding pre- and postoperative stiffness rates in RCT. If reaching a definition is a problem then finding its causes and risk factors seems even harder, not to mention the fact that intra- and interrater variability in shoulder mobility assessment seems high [27].

Regarding its treatment, surgical intervention has not been shown to overcome conservative treatment and neither of them has shown to alter the natural history of the disease, so the mainstay of treatment is still initially conservative, including home-based exercises, physical therapy, oral medication (NSAID, steroids) and injections (steroids, HA), while capsular release and/or manipulation under anaesthesia (MUA) is reserved for those cases that don't respond adequately to conservative treatment [29]. This approach is in direct conflict with the previously mentioned one for acute traumatic RCT in which early surgical repair seems to offer better outcomes.

31.2.2 Treatment Paradox

Having had trouble defining stiffness and sometimes defining the type and chronicity of the tear, the surgeon may find difficult to establish the adequate treatment when both conditions occur at the same time.

In fact, treating RCT and stiffness simultaneously can be paradoxical in two features that are fundamental to establish a treatment plan: how and when to treat.

– **How**: RCT repair is generally considered a shoulder-tightening procedure and as previously mentioned, postoperative stiffness is not uncommon. Despite that fact, there seems to be some consensus that RCT repairs require a certain time of postoperative immobilization in order to potentiate tendon healing [30, 31] but this initially safer approach, while protecting tendon

Table 31.1 Definition of shoulder stiffness in different papers

Study	Definition
Seo et al. [18]	Restriction of active and passive motion of 100° of elevation or less, less than 50% of external rotation **and** internal rotation only to the sacrum
C.H. Cho et al. [24]	Passive forward flexion <120°, external rotation <30° **or** internal rotation at the back lower than L3
Chuang et al. [34]	Passive forward flexion <135° **and** external rotation <45° in 90° of abduction
Park et al. [9]	Passive forward flexion (FF) equal or less than 120° **and** ER at the side equal or less than 45°
Koorevar et al. [35]	Painful restriction of active and passive movement, with passive movement limited to <100° elevation, <30° external rotation **and** internal rotation limited to L5 or less **and** external rotation <45° in 90° of abduction
Kim and Jung [21]	Passive forward flexion equal or less than 120°, passive ER equal or less than 30° **or** passive IR equal or less than L3
Kim et al. [36]	Forward flexion of less than 100° (maximum of 150°), external rotation of less than 45° (maximum of 90°) **or** internal rotation of a level where the thumb reaches lower than the first lumbar spine junction (maximum of T7 level)
Tauro [20]	Exclusive evaluation of glenohumeral motion by stabilization of the scapulothoracic motion: Normal glenohumeral motion was 90° of forward flexion, external rotation, abduction and internal rotation Loss of motion in these four basic movements was identified and then added together to calculate total range of motion deficit (TROMD) If TROMD >25° and <70° there was moderate stiffness If TROMD >70° there was severe stiffness

repair and allowing lower retear rates, can induce, at least, transient postoperative stiffness [30–32] that can in fact be beneficial and protective for tendon healing [26, 33]. Most of the times this loss of motion is transitory but in some cases it can persist and be very frustrating for both the surgeon and the patient, occasionally requiring a second surgical procedure in order to regain adequate and functional ROM. This initial immobilization period seems to be completely incompatible with the treatment needed for stiffness that includes immediate and continuous mobilization that, as demonstrated, can preclude tendon healing [29–31].

- The mentioned paradox led surgeons to delay surgery for rotator cuff repair until full ROM was achieved with an adequate conservative approach for shoulder stiffness (staged procedure). Unfortunately a new treatment paradox appeared when the consequences of surgical delay in RCT became more evident. It was the *When* Paradox.
- **When**: As previously shown, delaying rotator cuff repair may create a local biological impairment for tendon healing and tear progression seems inevitable, so the ideal treatment for a symptomatic traumatic RCT would be surgical repair as soon as possible. The opposite is described for the treatment of stiffness as initial conservative treatment is still the mainstay of treatment [29].

The staged procedure, despite acceptable, rises several issues:

1. The cause for the stiffness may be the rotator cuff tear itself due to the pain, muscle contracture and weakness [18, 21, 36] which will not solve without adequate RCT treatment.
2. Patient compliance to a painful, long and demanding rehabilitation process seems to be quite low [22, 23].
3. There is no guarantee that conservative treatment for stiffness will be effective [36]. In fact, it seems that most ROM gains occur in scapulo-thoracic motion instead of glenohumeral [1] and if it fails, the surgeon may have partially jeopardized tendon healing.
4. Long preoperative duration of symptoms seems to be associated with worse functional outcomes if single-stage surgery is delayed (especially after 6 months of symptoms) [22].
5. Forceful mobilization has the risk of inducing intraarticular iatrogenic injuries [1, 34] and other conservative methods such as steroids with local anaesthetic usually used to treat shoulder stiffness can induce tenocyte cytotoxicity and apoptosis, besides decreasing tendon biomechanical properties [37].

Performing single-stage surgery (capsular release + MUA + rotator cuff repair) to address both conditions became progressively more acceptable, and recently Kim described that at least similar results could be achieved when comparing this approach with the staged approach concluding that surgical delay should be avoided as it didn't show any advantage [36].

Others also provided sufficient evidence that when comparing single-stage surgery for both conditions with simple RCT repair in non-stiff shoulders, similar results could be achieved although ROM recovery was slower [16, 20–22, 24, 34], which led the authors to conclude that this procedure is safe and perfectly acceptable.

It is important to state that capsular release has several clear advantages over isolated MUA, as it allows for a direct visual control of the capsular iatrogenic opening while controlling bleeding and hemarthrosis, that can themselves induce postoperative fibrosis and scarring, limiting the benefit of the capsulotomy performed. Also, direct capsular release can avoid some of the described MUA complications such as humeral fractures, glenohumeral dislocations, chondral injuries, rotator cuff tear enlargement/creation and labral or SLAP tears [1, 38, 39].

Having solved the mentioned treatment paradox, some questions still remain unanswered:

- What is the main cause of pain? Is it stiffness, the rotator cuff tear or other pathology?
- How do we define stiffness?
- What is the cause for stiffness?
- Should different degrees of stiffness induce different treatment strategies?

Answers to these questions can only be given with future investigation and for now, only general recommendations can be given.

31.3 Recommended Approach

In case of isolated stiffness (either idiopathic adhesive capsulitis or secondary nontraumatic stiffness), full-thickness RCT tears are rare [40], so treatment should focus in this condition which is beyond the focus of this chapter.

In case of trauma in which shoulder stiffness develops, one should always consider the presence of a rotator cuff tear [12, 41].

Because physical examination can be extremely difficult in this setting, MRI or arthro CT can be used to define the presence and characteristics of a rotator cuff tear.

If an acute tendon tear is found it seems adequate to offer immediate single-stage procedure for these patients, especially if they have severe stiffness or if they are diabetic [20]. In this scenario, examination under anaesthesia is mandatory because if stiffness disappears, capsular release and MUA should be avoided. The opposite can also happen if during the rotator cuff repair of a moderately stiff patient the surgeon finds a very thick and inflamed capsule, in which case a capsular release is recommended [20].

When performing single-stage surgery, two premises should be kept in mind: adequate cuff fixation and fast and safe recovery of passive ROM l. This approach allows the surgeon to address the treatment paradox in the safest way possible, trying to minimize the risk of both retear and post-operative stiffness. Immediate passive ROM exercises can be performed if adequate fixation is achieved, as stiff patients tend to have a lower retear rate [21]. In some cases in which cuff repair isn't fully satisfactory, the surgeon must bear in mind that postoperative stiffness is a complication that can be addressed later on with good results but a retear is a failure that can bring catastrophic functional results, so in these scenarios, the author recommendation is to use a more conservative postoperative protocol, delaying passive ROM exercises and subsequently all the other stages of rehabilitation.

Nonetheless, a small trial (< than 6 weeks) of physical therapy and oral medication can also be used in patients with traumatic RCT and stiffness in order to clarify the symptoms. If the patient starts to increase his range of motion, delaying surgery can be an option for as long as the benefit from the delay surpasses its risk, but finding the correct balance can be very difficult and subjective. Despite being impossible to predict the future, in this setting the surgeon should always consider the type and direction of the tear, number of affected tendons and muscular and tendon quality as well as the severity of stiffness when choosing to maintain or abort conservative treatment.

When performing a staged procedure we do not recommend subacromial or intraarticular injections because of their theoretical biological disadvantages for the tendon, aggravating its condition in an already difficult situation [37].

31.4 Single-Stage Surgical Procedure: Eight Key Surgical Steps

1. Evaluate patient's ROM under anesthesia
2. If joint entering through the posterior portal is difficult, perform subacromial bursectomy, move to the anterior part of the shoulder and find the coracoid. Posterior and lateral to it is the rotator interval that can and should be safely opened allowing direct access to the joint. At that point a switching stick can be introduced from anterior to posterior, creating a posterior portal that can be used for visualization
3. Open the rotator interval and debride it
4. Perform anterior capsule release using a radiofrequency device through an antero-superior portal and go as distal as possible from anterior
5. Move the scope to the antero-superior portal and perform a posterior and inferior capsule release using the radiofrequency in the posterior portal. Inferior access can be facilitated using a 7 o'clock posterior portal (Fig. 31.4)
6. Always keep the radiofrequency tip facing the glenoid and close to it, especially during inferior release (to avoid axillary nerve injury)
7. Perform manipulation under anesthesia after anterior, inferior and posterior capsular release
8. Perform rotator cuff repair and include biceps tenotomy/tenodesis (it is usually pathologic and if kept in place it can be a source of pain)

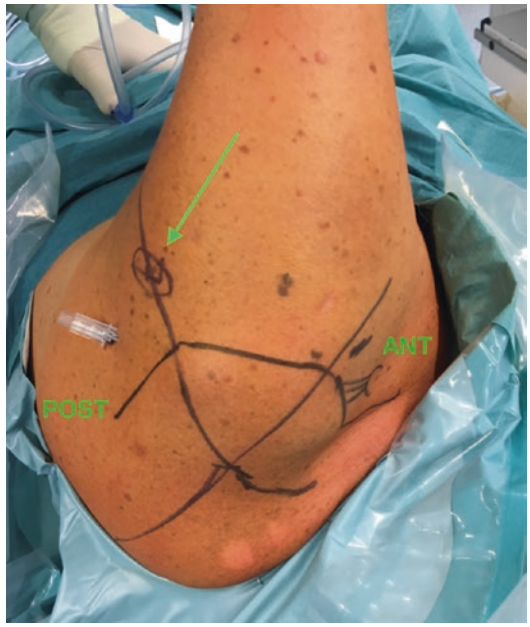


Fig. 31.4 7 o'clock portal position (green arrow)

31.5 Conclusion

Traumatic rotator cuff tears and stiffness can occur simultaneously. Although treating both conditions at the same time in a single-stage procedure can appear paradoxical, no major advantage in surgical delay has been shown in the literature, although further investigation is required to establish which degree of stiffness is neglectable and which should be addressed through a simultaneous capsular release and RCT repair.

References

1. Loew M, Heichel TO, Lehner B. Intraarticular lesions in primary frozen shoulder after manipulation under general anesthesia. J Shoulder Elb Surg. 2005;14:16–21. https://doi.org/10.1016/j.jse.2004.04.004.
2. Walcott ME, Daniels SD, Sinz NJ, et al. Traumatic full-thickness transtendinous rotator cuff tears: a case series. J Shoulder Elb Surg. 2017;26:62–7. https://doi.org/10.1016/j.jse.2016.04.023.
3. Yamaguchi K, Ditsios K, Middleton WD, et al. The demographic and morphological features of rotator cuff disease. J Bone Joint Surg Am. 2006;88:1699–704. https://doi.org/10.2106/jbjs.e.00835.
4. Milgrom C, Schaffler M, Gilbert S, Holsbeeck MV. Rotator-cuff changes in asymptomatic adults. The effect of age, hand dominance and gender. J Bone Joint Surg Br. 1995;77-B:296–8. https://doi.org/10.1302/0301-620x.77b2.7706351.
5. Mall NA, Kim HM, Keener JD. Symptomatic progression of asymptomatic rotator cuff tears: a prospective study of clinical and sonographic variables. J Bone Joint Surg Am. 2010;17:2623–33. https://doi.org/10.2106/JBJS.I.00506.
6. Bassett RW, Cofield RH. Acute tears of the rotator cuff. Clin Orthop Relat Res. 1983. https://doi.org/10.1097/00003086-198305000-00003.
7. Lazarides AL, Alentorn-Geli E, Choi JJ, et al. Rotator cuff tears in young patients: a different disease than rotator cuff tears in elderly patients. J Shoulder Elb Surg. 2015;24:1834–43. https://doi.org/10.1016/j.jse.2015.05.031.
8. Loew M, Magosch P, Lichtenberg S, et al. How to discriminate between acute traumatic and chronic degenerative rotator cuff lesions: an analysis of specific criteria on radiography and magnetic resonance imaging. J Shoulder Elb Surg. 2015;24:1685–93. https://doi.org/10.1016/j.jse.2015.06.005.
9. Park J-Y, Chung SW, Hassan Z, et al. Effect of capsular release in the treatment of shoulder stiffness concomitant with rotator cuff repair. Am J Sports Med. 2014;42:840–50. https://doi.org/10.1177/0363546513519326.
10. Mall NA, Lee AS, Chahal J, et al. An evidenced-based examination of the epidemiology and outcomes of traumatic rotator cuff tears. Arthroscopy. 2013;29:366–76. https://doi.org/10.1016/j.arthro.2012.06.024.
11. Gerber C, Hersche O, Farron A. Isolated rupture of the subscapularis tendon. Results of operative repair∗. J Bone Joint Surg Am. 1996;78:1015–23. https://doi.org/10.2106/00004623-199607000-00005.
12. Hantes ME, Karidakis GK, Vlychou M, et al. A comparison of early versus delayed repair of traumatic rotator cuff tears. Knee Surg Sports Traumatol Arthrosc. 2011;19:1766–70. https://doi.org/10.1007/s00167-011-1396-1.
13. Zanetti M, Gerber C, Hodler J. Quantitative assessment of the muscles of the rotator cuff with magnetic resonance imaging. Invest Radiol. 1998;33:163–70. https://doi.org/10.1097/00004424-199803000-00006.
14. Fuchs B, Weishaupt D, Zanetti M, et al. Fatty degeneration of the muscles of the rotator cuff: assessment by computed tomography versus magnetic resonance imaging. J Shoulder Elb Surg. 1999;8:599–605. https://doi.org/10.1016/s1058-2746(99)90097-6.
15. Melis B, Defranco MJ, Chuinard C, Walch G. Natural history of fatty infiltration and atrophy of the supraspinatus muscle in rotator cuff tears. Clin Orthop Relat Res. 2010;468:1498–505. https://doi.org/10.1007/s11999-009-1207-x.
16. Oh JH, Kim SH, Lee HK, et al. Moderate preoperative shoulder stiffness does not Alter the clinical outcome of rotator cuff repair with arthroscopic release and

manipulation. Arthroscopy. 2008;24:983–91. https://doi.org/10.1016/j.arthro.2008.06.007.
17. Goutallier D, Postel J-M, Gleyze P, et al. Influence of cuff muscle fatty degeneration on anatomic and functional outcomes after simple suture of full-thickness tears. J Shoulder Elb Surg. 2003;12:550–4. https://doi.org/10.1016/s1058-2746(03)00211-8.
18. Seo S-S, Choi J-S, An K-C, et al. The factors affecting stiffness occurring with rotator cuff tear. J Shoulder Elb Surg. 2012;21:304–9. https://doi.org/10.1016/j.jse.2011.04.011.
19. Cho NS, Rhee YG. Functional outcome of arthroscopic repair with concomitant manipulation in rotator cuff tears with stiff shoulder. Am J Sports Med. 2008;36:1323–9. https://doi.org/10.1177/0363546508314402.
20. Tauro JC. Stiffness and rotator cuff tears: incidence, arthroscopic findings, and treatment results. Arthroscopy. 2006;22:581–6. https://doi.org/10.1016/j.arthro.2006.03.004.
21. Kim I-B, Jung D-W. A rotator cuff tear concomitant with shoulder stiffness is associated with a lower retear rate after 1-stage arthroscopic surgery. Am J Sports Med. 2018;46:1909–18. https://doi.org/10.1177/0363546518768813.
22. Chen Y, Chen S, Qiao Y, et al. A long preoperative duration of symptoms is associated with worse functional outcomes after 1-stage arthroscopic treatment of rotator cuff tears with shoulder stiffness. Am J Sports Med. 2017;45:2336–44. https://doi.org/10.1177/0363546517707202.
23. Ho WP, Huang C-H, Chiu C-C. One-stage arthroscopic repair of rotator cuff tears with shoulder stiffness. Arthroscopy. 2013;29:1283–91.
24. Cho C-H, Jang H-K, Bae K-C, et al. Clinical outcomes of rotator cuff repair with arthroscopic capsular release and manipulation for rotator cuff tear with stiffness: a matched-pair comparative study between patients with and without stiffness. Arthroscopy. 2015;31:482–7. https://doi.org/10.1016/j.arthro.2014.09.002.
25. Vastamäki H, Vastamäki M. Postoperative stiff shoulder after open rotator cuff repair: a 3- to 20-year follow-up study. Scand J Surg. 2014;103:263–70. https://doi.org/10.1177/1457496913514383.
26. Huberty DP, Schoolfield JD, Brady PC, et al. Incidence and treatment of postoperative stiffness following arthroscopic rotator cuff repair. Arthroscopy. 2009;25:880–90. https://doi.org/10.1016/j.arthro.2009.01.018.
27. Trenerry K, Walton JR, Murrell GAC. Prevention of shoulder stiffness after rotator cuff repair. Clin Orthop Relat Res. 2005;(430):94–9. https://doi.org/10.1097/01.blo.0000137564.27841.27.
28. Audigé L, Blum R, Müller AM, et al. Complications following arthroscopic rotator cuff tear repair. Orthop J Sports Med. 2015;3:232596711558786. https://doi.org/10.1177/2325967115587861.
29. Yip M, Francis A-M, Roberts T, et al. Treatment of adhesive capsulitis of the shoulder. JBJS Rev. 2018;6(6):e5. https://doi.org/10.2106/jbjs.rvw.17.00165.
30. Denard PJ, Lädermann A, Burkhart SS. Prevention and management of stiffness after arthroscopic rotator cuff repair: systematic review and implications for rotator cuff healing. Arthroscopy. 2011;27:842–8. https://doi.org/10.1016/j.arthro.2011.01.013.
31. Thigpen CA, Shaffer MA, Gaunt BW, et al. The American Society of Shoulder and Elbow Therapists consensus statement on rehabilitation following arthroscopic rotator cuff repair. J Shoulder Elb Surg. 2016;25:521–35. https://doi.org/10.1016/j.jse.2015.12.018.
32. Arndt J, Clavert P, Mielcarek P, et al. Immediate passive motion versus immobilization after endoscopic supraspinatus tendon repair: a prospective randomized study. Orthop Traumatol Surg Res. 2012;98(6 Suppl):S131–8. https://doi.org/10.1016/j.otsr.2012.05.003.
33. Parsons BO, Gruson KI, Chen DD, et al. Does slower rehabilitation after arthroscopic rotator cuff repair lead to long-term stiffness? J Shoulder Elb Surg. 2010;19:1034–9. https://doi.org/10.1016/j.jse.2010.04.006.
34. Chuang T-Y, Ho W-P, Chen C-H, et al. Arthroscopic treatment of rotator cuff tears with shoulder stiffness. Am J Sports Med. 2012;40:2121–7. https://doi.org/10.1177/0363546512453296.
35. Koorevaar RCT, Riet EVT, Ipskamp M, Bulstra SK. Incidence and prognostic factors for postoperative frozen shoulder after shoulder surgery: a prospective cohort study. Arch Orthop Trauma Surg. 2017;137:293–301. https://doi.org/10.1007/s00402-016-2589-3.
36. Kim Y-S, Lee H-J, Park I, et al. Are delayed operations effective for patients with rotator cuff tears and concomitant stiffness? An analysis of immediate versus delayed surgery on outcomes. Arthroscopy. 2015;31:197–204. https://doi.org/10.1016/j.arthro.2014.08.014.
37. Honda H, Gotoh M, Kanazawa T, et al. Effects of lidocaine on torn rotator cuff tendons. J Orthop Res. 2016;34:1620–7. https://doi.org/10.1002/jor.23153.
38. Weber M, Prim J, Bugglin R, et al. Long-term follow up of patients with frozen shoulder after mobilization under anesthesia, with special reference to the rotator cuff. Clin Rheumatol. 1995;14:686–91. https://doi.org/10.1007/bf02207937.
39. Atoun E, Funk L, Copeland S, et al. The effect of shoulder manipulation on rotator cuff integrity. Acta Orthop Belg. 2013;79:255–9.
40. Ueda Y, Sugaya H, Takahashi N, et al. Rotator cuff lesions in patients with stiff shoulders. J Bone Joint Surg. 2015;97:1233–7. https://doi.org/10.2106/jbjs.n.00910.
41. Sørensen AK, Bak K, Krarup AL, et al. Acute rotator cuff tear: do we miss the early diagnosis? A prospective study showing a high incidence of rotator cuff tears after shoulder trauma. J Shoulder Elb Surg. 2007;16:174–80. https://doi.org/10.1016/j.jse.2006.06.010.

Failed Rotator Cuff Repair: Decision-Making Algorithm

32

Antonio Cartucho

32.1 Introduction

There are several factors that increase the risk of rotator cuff retear after surgical repair. Older age, larger tear size, thickness of the tear, greater muscle-tendon unit retraction and poor muscle quality have all seemed to negatively affect tendon healing [1–3]. Retear rates after surgical intervention for rotator cuff tear ranged from 20 to 94% [4–6]. Retears, defined as a fluid-filled gap on MRI, occur mainly between 6 and 26 weeks after arthroscopic rotator cuff repair (RCR) with only a few occurring after this period. The "critical" interval for tear recurrence is considered between the 12 and the 26 weeks after repair [7]. Tear recurrence can be related to various factors such as: poor patient selection, inadequate initial repair, biological failure to heal, inappropriate postoperative rehabilitation and poor patient compliance causing structural failure of the repair [8–10]. The retear is usually smaller than the original tear, and the structural failures may be well tolerated [11]. Although some patients with failure of healing exhibit poor outcomes and require revision surgery, others report pain relief and a return of function despite a lack of healing [12–15]. Nevertheless Namdari et al. found only 54% of good clinical results in patients with rotator cuff retear [16]. However, long-term outcome after structural failure of RCR has to be taken into consideration. Clinical outcome remains significantly improved over the preoperative state in terms of pain, function, strength and patient satisfaction over time. Overall, retears do not increase in size and the ones that are smaller than 400 mm^2 had the potential to heal [11]. This being said not all retears need to be approached and its clinical presentation may vary from a tolerable shoulder discomfort to a significant shoulder function impairment [11].

Management of failed RCRs must be considered as a clinical challenge. Decision on how to act must be supported on various factors: the degree of shoulder impairment, the reasons for structural failure, the type of structural failure and the tendon, bone and muscle conditions. Patient expectations must be assessed and controlled knowing that surgical results for cuff retear are inferior to the ones of the index procedure [16].

32.2 Causes of Failed Rotator Cuff Repair

Causes of failure of RCR are often multifactorial and include poor patient selection and diagnostic errors, surgical complications, technical errors at the initial repair, failure to heal, additional traumatic event, inappropriate postoperative rehabilitation and poor patient compliance [8–10]. Determining the causes of failure and

A. Cartucho (✉)
Hospital Cuf Descobertas, Lisbon, Portugal

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_32

correcting these factors, when possible, is important when someone deal with failed RCR.

32.2.1 Poor Patient Selection and Diagnostic Errors

Patient age and comorbidities, tear size, tendon quality and retraction, muscle atrophy and fatty degeneration have been associated to poor clinical results [2, 10, 17, 18]. Also, incomplete diagnosis can lead to poor results after RCR. Supraspinatus tears may be associated with other lesions at the rotator interval, long head of the biceps (LHB) or subscapularis tendons [19].

32.2.2 Surgical Complications

Complications associated with open and arthroscopic RCR include disruption of the deltoid origin, infection, foreign body reaction, stiffness and neurologic injury. Deltoid injury being rare at arthroscopic procedures may be due to inadequate deltoid reattachment to the acromion, in open procedures, leading to poor cosmetics and deltoid impairment.

The most common infection after RCR is *Cutibacterium acnes* (former *Propionibacterium acnes*), affecting 50–86% of postoperative infections. Surgical irrigation and debridement and intravenous antibiotics are necessary to eradicate the infection. Even when properly treated, postoperative infection often results in stiffness, adhesions, failure of the repaired tissue and continued pain [20, 21].

Foreign body reactions are associated mainly with bioabsorbable materials but also to rigid sutures and metallic anchors. Loose anchors or sutures, due to the synovial reaction, might mimic an infection. Imaging diagnosis is difficult and sometimes these reactions are only diagnosed intraoperatively [22, 23].

Stiffness is a relatively frequent complication after RCR with an incidence ranging from 5 to 10% [24, 25]. Risk factors are limited preoperative range of motion (ROM), diabetes, hypothyroidism, partial rotator cuff tear and calcifying tendonitis. Poor patient compliance to demanding postoperative rehabilitation may also contribute to the onset of shoulder stiffness [17]. Patient dissatisfaction after RCR might be due to restriction of passive ROM and/or pain due to capsule inflammatory reaction. This being said, corticoid intra-articular injections and physiotherapy might reduce pain and recover ROM without the need of a new surgery [26].

Neurological complications are rare and most of the times transient, mostly due to nerve traction during the surgical procedure. Direct nerve damage is very uncommon. Nerves at risk are mainly the suprascapular nerve, the musculocutaneous and the axillary nerve [27].

32.2.3 Inadequate Initial Repair

Rotator cuff tear is a complex pathology and tears might be partial or complete and involve one or more tendons. These tendons might be more or less mobile and the geometry of the rupture might differ between V, anterior L, posterior L and crescent like (Fig. 32.1) [28]. When the quality of the muscle is assessed, factors such as atrophy, retraction and fatty degeneration have been associated with poor prognosis [10, 18]. Inadequate assessment of all these factors may lead to an attempted repair prone to failure. Structural failure might be due to poor anchor positioning, non-

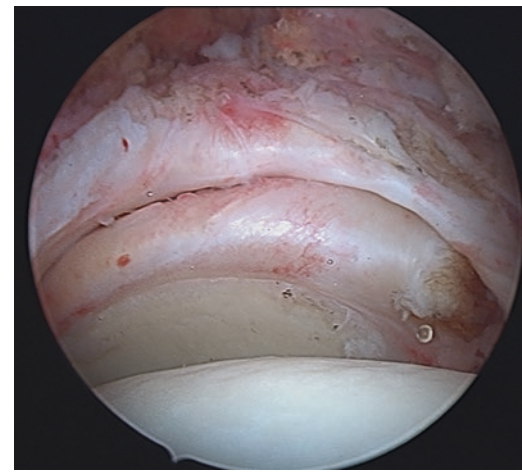


Fig. 32.1 Arthroscopic view on U-shaped retracted rotator cuff tear

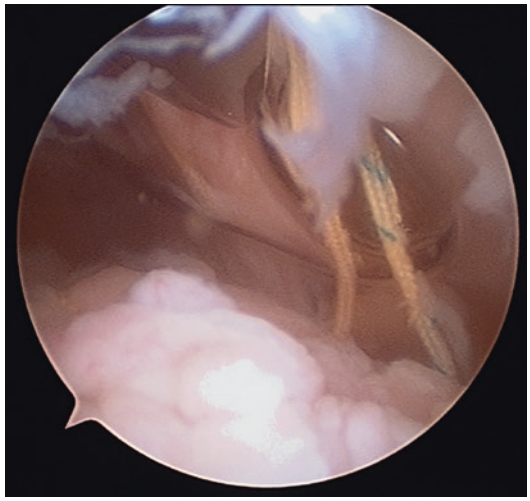


Fig. 32.2 Tendon to bone interface failure as seen in revision of rotator cuff repair

use of tension relieving suture techniques (side by side or ripstop-like) and tendon overtensioning [29]. The location of structural failure may vary from tendon to bone interface to the muscle-tendon interface (Fig. 32.2). This fact is important when considering a surgical review as the remaining tendon might not be long enough to permit a novel reattachment [30].

32.2.4 Failure to Heal

Failure to heal after surgical intervention for rotator cuff tear has ranged from 20 to 94%. The "critical" interval for tear recurrence is considered between the 12 and the 26 weeks after repair. Even in a well-performed repair the tendon may fail to heal. Factors associated with non-healing environment are poor rotator cuff and greater tuberosity vascularity and poor tendon and bone quality [14]. This last factor must be taken into consideration when choosing the revision RCR and the choice of a transosseous technique might be adequate [31, 32].

32.2.5 Traumatic Cause

Traumatic postoperative failure may occur before or after tendon healing due to direct trauma such as a fall or indirect trauma. Aggressive rehabilitation or poor patient compliance plays an important role at the early phase. Structural failure occurs at the suture-tissue interface as the suture cuts out through the tissue [30].

32.3 Diagnosis

Evaluation of the painful or weak shoulder after surgery for rotator cuff tear consists of a detailed history, physical examination and imaging. Clinical signs include persistent pain, weakness, loss of active ROM and sometimes stiffness that do not respond to appropriate postoperative physical therapy. The goal of imaging studies is to confirm the existence and site of the recurrent tear, the type of failure (in continuity, muscle-tendon or tendon to bone interface) and, if possible, its cause [26, 33]. MRI may not provide the same sensitivity in recurrent rotator cuff tear as in primary rotator cuff tear. Only 10% of reattached tendons generate a normal MRI signal. Thus, a common finding is the presence of an intermediate signal within the tendon indicating granulation tissue or of a low-intensity signal produced by fibrous tissue [34, 35]. These signal changes may persist for longer than 6 months, due to tissue remodelling, artefact, suture material and suture anchors. MRI in this setting has a reported accuracy of 70–90% [36, 37]. One must have these facts into consideration when considering a structural failure. Correlation with clinical aspects is of upmost importance and MRI with intra-articular contrast may be used to increase the sensitivity of detection of recurrent rotator cuff tears [38]. Postoperative cuff healing and integrity might be assessed using the Sugaya classification with the type IV and V describing a partial retear and a complete retear, respectively (Fig. 32.3) [39]. Ultrasound has a 91% sensitivity, 86% specificity and 89% accuracy regarding retear diagnosis with economic advantage but strongly relying on the operator [40]. CT arthrogram can also be used to aid in the identification of recurrent rotator cuff tears when neither ultrasound nor MRI is an option [41].

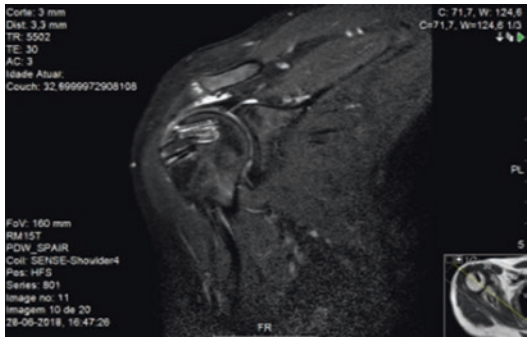


Fig. 32.3 Failed rotator cuff repair classified as Sugaya's type V—complete retear

32.4 Treatment Options

Addressing failed RCR numerous factors must be considered such as: patient age, comorbidities, patient expectations and functional demands, remaining rotator cuff tissue quality, tuberosity bone quality, muscle atrophy and fatty degeneration and the existence of glenohumeral arthritis [42, 43]. The options to address failed rotator cuff tears that have been repaired include non-operative management, revising the primary repair, tendon transfer, biological augmentation or use of tissue-engineered grafts for reconstruction, interposition techniques and reverse shoulder arthroplasty [44].

32.4.1 Non-operative Management

Non-operative treatment of failed RCR consists in applying a protocol of physical therapy, which includes stretching and muscle strengthening (the remaining portions of the rotator cuff, deltoid and, especially, the periscapular musculature, lower trapezius and rhomboids), nonsteroidal anti-inflammatory medications and occasional corticosteroid injection [17]. Although conservative treatment of rotator cuff tears has been extensively reported with good results in selected patients only a few series have been published regarding failed RCR [45, 46]. In these series good results were reported both in functional and subjective scores. Furthermore, these results were stable over time with minimum 2 years of follow-up [11, 16]. When considering non-operative treatment it is important to keep in mind the concept of functional and non-functional tear patterns. Active elevation was significantly decreased in patients with three tear patterns involved. Pseudoparalysis was described in 80% of the cases with supraspinatus and complete subscapularis tears and in 45% of the cases with tears involving the supraspinatus, infraspinatus and superior subscapularis. Loss of active external rotation was related to tears involving the infraspinatus and teres minor; loss of active internal rotation was related to tears of the subscapularis [47].

32.4.2 Surgical Options and Indications

Surgical options range from extensive debridement, revising the primary repair with complete or partial repair, tendon transfer, biological augmentation, use of tissue-engineered grafts for reconstruction and reverse shoulder arthroplasty.

Patient expectations should be assessed and the surgeon must be aware of the factors that can or cannot be changed when deciding on revision surgery. Risk factors for poorer postoperative results are patient age, female gender, surgery of the dominant arm, preoperative VAS greater than 5 and poor preoperative ROM ranging from 90° to 140° depending on the authors [9, 48–50]. On the other hand, acromio-humeral distance inferior to 7 mm and the impact of more than one previous surgery have not been proven to negatively affect the outcome [49, 50].

32.4.2.1 Arthroscopic Extensive Debridement and Subacromial Decompression

Painful failed RCR may be due to foreign body reaction due to reabsorbable materials, loose anchors or sutures causing irritation of the subacromial space. Arthroscopically addressing this factors the surgeon might address an acromial spur, assess and perform a tenotomy or tenode-

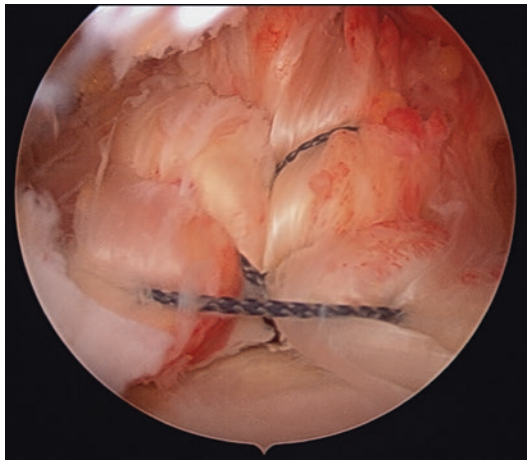


Fig. 32.4 Partial rotator cuff repair with side to side sutures—margin convergence repair

sis of the long head of the biceps and debride a degenerative labrum lesion [44, 51]. This procedure should be used in low-demand patients with pain as the predominant complaint.

32.4.2.2 Partial Repair

Based on the "suspension-bridge" principles and in the impossibility to fully repair the reteared cuff, a partial repair might be a good solution to diminish pain and to enhance shoulder performance (Fig. 32.4) [28]. Fatty infiltration of the teres minor has been described as a poor prognostic factor [52]. Studies comparing partial repair to debridement alone describe better results regarding pain and functional scores for the partial repair despite a retear rate of 54% [53, 54].

32.4.2.3 Anatomic Repair

When possible, as the results are consistently better in the healed tendon group patients, but dependable of patient conditions, tendon, muscle and bone quality, anatomic reconstruction of the rotator cuff retear should be performed. Mobilization of the tendons involved using anterior and posterior interval slide techniques, load-sharing suture techniques, double row or transosseous equivalent fixation methods have been associated with better healing results and consequently with better clinical results [6, 43, 55]. Results in the literature show a higher retear rate compared to the primary intervention but a consistent improvement in pain scores with a less predictable improvement in ROM gain [50]. In the revision setting, pseudoparalysis was only reversed in 43% of patients, with a low rate (54%) of patient satisfaction [43]. This being said pseudoparalysis in a failed RCR scenario might be better addressed with a reverse shoulder arthroplasty [56].

32.4.2.4 Graft Augmentation and Graft Interposition

Grafts may be used to improve stability to the repaired tendon and enhance healing or as a substitute of the part of the missing lateral tendon. They may be derived from allografts, xenografts and synthetic materials. The commercially available tendon augmentation grafts are from human dermis, porcine small intestinal submucosa, bovine dermis and porcine dermis, may or not be cross-linked, have different processing methods and differ also on the number of layers and thickness. The selection depends on the tissue of origin, graft processing, cross linking of the material, physical properties of the tissue and the surgeon selection based on previous experience [57].

Porcine submucosa subintestinal grafts were found to increase pain and lead to poorer tendon healing and may not be suitable for human RCR. These results are in contrast to the animal preclinical studies [58]. On the other hand, human dermis-derived grafts, used as scaffold augmentation or as a bridging construct for irreparable rotator cuff, were reported to improve clinical outcomes at follow-up, with low incidence of complications and no cases of graft rejection [59].

When comparing clinical and structural results of cuff reconstruction with or without graft augmentation, authors presented better results with the graft augmentation [60–62]. Nevertheless, Ladermann et al. in a recent systematic review found no scientific data to support any systematic associated augmentation technique to rotator cuff repair [63]. Recently, Cai et al. described a better healing rate but similar clinical outcome when comparing graft augmentation with repair alone [64].

Graft augmentation, if considered, must be used in selected patients aiming to reinforce the cuff reconstruction to promote tendon healing and permit earlier rehabilitation.

32.4.2.5 Superior Capsule Reconstruction

Superior capsule reconstruction was first described by Mihata et al. [65] to approach irreparable rotator cuff tears and consists on using fascia lata autograft and more recently allograft, in order to reconstruct the superior capsule, between the superior glenoid and the great tuberosity using suture anchors (Fig. 32.5). The purpose of this construct is to avoid superior humeral head migration when the deltoid is actively contracting [66]. Only preliminary results with short follow-up are available and the authors present improvement of functional scores, both using fascia lata and allograft [65, 67].

If these preliminary results pass the test of time, superior capsule reconstruction might be the choice for relatively young, motivated patients with high functional demands, suffering from a failed RCR, who are able to coop with a long rehabilitation time.

32.4.2.6 Tendon Transfers

The goal of tendon transfer is to achieve stable kinematic by restoring rotational strength and force coupling about the joint. The optimal indication for latissimus dorsi tendon transfer is an isolated loss off external rotation in a young patient with an irreparable infraspinatus tear and a teres minor deficiency [68]. The subscapularis tendon must be intact or have a repairable tear. Clinically these patients present a positive lag sign in external rotation and loss of active elevation [69]. The procedure might be performed arthroscopically assisted [70]. The transferred tendon partially correcting the active external rotation deficit enables recovery of daily activities and reducing functional disability. Nevertheless, latissimus dorsi transfer does not correct lack of strength, which remains significant and is the main sequelae reported by patients [71–74].

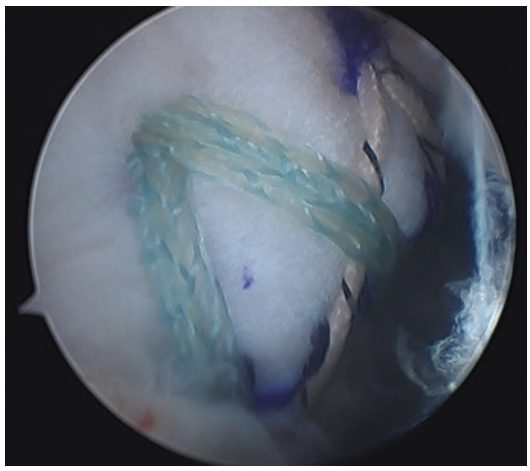


Fig. 32.5 Transosseous equivalent allograft fixation on greater tuberosity in superior capsular reconstruction

Irreparable ruptures of subscapularis tendon may be addressed by pectoralis major transfer leading to improvement of shoulder function, strength and pain relief [75]. More recently, the use of latissimus dorsi to address subscapularis irreparable ruptures has been described [76]. Preliminary results showed that belly press test results were negative for 18 of 24 patients, and the lift-off test results were negative for 16 of 20 patients. The authors concluded that latissimus dorsi transfer resulted in pain relief and restoration of shoulder range of motion and function [77].

32.4.2.7 Interposition Techniques

With the purpose to prevent impingement and promote humeral head centering during abduction thus producing a painless activation of the scapulohumeral musculature, some authors propose interposing a device in the subacromial space, which lowers the humeral head and provides an improved balance between the subscapularis anteriorly and the infraspinatus posteriorly, permitting better deltoid activation and compensation through the arc of motion. A biodegradable balloon meant for arthroscopic insertion into the subacromial space following bursa excision was used to achieve improvement in daily and nightly pain as well as ROM and ultimately strength in this preliminary prospective pilot study with 22 patients with symptomatic MRCT and a mean 3-year follow-up [78]. More recently the balloon has been also used to protect fragile repairs [79].

32.4.2.8 Reverse Shoulder Arthroplasty

Reverse shoulder arthroplasty (RSA) may be used in failed RCR with pseudoparalysis in the absence of glenohumeral arthritis when it is impossible to restore cuff function [80]. Complication rates are relatively high specially in patients younger than 60 years old [81]. Nevertheless, results after reverse total shoulder arthroplasty to address massive rotator cuff tear arthropathy with long follow-up are good, demonstrating pain relief and functional improvement [82]. These results may also be achieved after a failed RCR with a high rate of patient satisfaction [56].

Patients with irreparable unbalanced cuff tears with a concomitant teres minor insufficiency have a pseudoparalysis and lag sign in external rotation. Results of an isolated RSA are non-satisfactory as the patients continue to lack external rotation to perform daily living activities. An associated transfer of the latissimus dorsi using a modified L'Episcopo technique in order to restore active external rotation is proposed in this cases [83–85].

32.5 Conclusion

Retear or non-healing of rotator cuff tendons is rather frequent. Surgical treatment is rarely indicated as this condition is often well tolerated with marked clinical improvement in comparison with the preoperative state. In young, motivated patients in a post-traumatic scenario, a second repair must be considered taking into consideration tendon, muscle and bone status. The use of load-sharing techniques is recommended. Low-demand patients with pain as the major complaint may benefit from debridement and LHB tenotomy or tenodesis. Partial repair of the retear may produce good functional results if the "suspension cable" of the rotator cuff is restored. The use of biological enhancement products or devices, such as grafts, might be used in selected cases. Tendon transfer of the latissimus dorsi should be used in patients with isolated loss of active external rotation and this same tendon has recently been used to solve subscapularis deficiency. Interposition techniques such as superior capsule reconstruction and the subacromial balloon have only preliminary results but the first might be a solution for young patients where not even a partial repair is possible and the second might be used to protect a fragile repair. For patients with pseudoparalysis after a cuff repair, RSA should be the choice as being the only predictable way to restore ROM. If a lag sign in external rotation is present, a concomitant latissimus dorsi transfer is mandatory. A decision algorithm must be based on a balance taking into consideration all the facts presented in this chapter (Fig. 32.6).

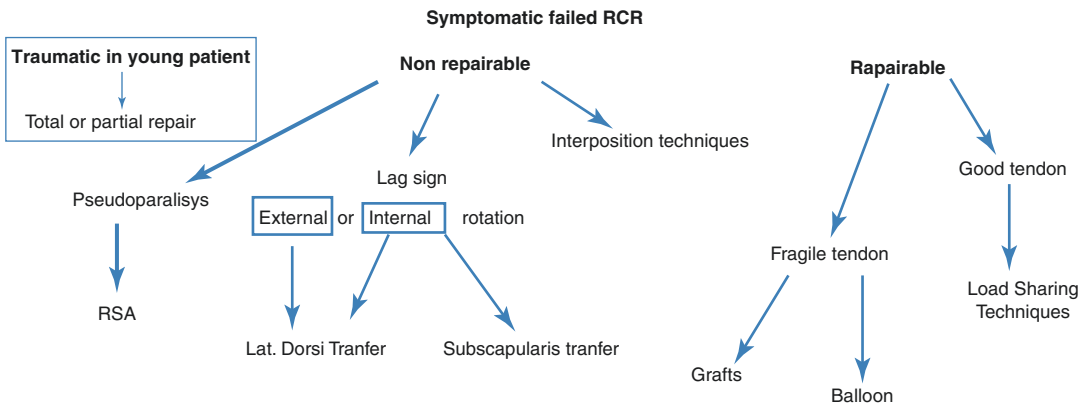


Fig. 32.6 Decision-making algorithm—management of failed rotator cuff repair

References

1. Galanopoulos I, Ilias A, Karliaftis K, Papadopoulos D, Ashwood N. The impact of re-tear on the clinical outcome after rotator cuff repair using open or arthroscopic techniques—a systematic review. Open Orthop J. 2017;11:95–107.
2. Abtahi AM, Granger EK, Tashjian RZ. Factors affecting healing after arthroscopic Rotator Cuff Repair. World J Orthop. 2015;6:211–20.
3. Cho NS, Lee BG, Rhee YG. Arthroscopic Rotator Cuff Repair using a suture bridge technique: is the repair integrity actually maintained? Am J Sports Med. 2011;39:2108–16.
4. Galatz LM, Ball CM, Teefey SA, Middleton WD, Yamaguchi K. The outcome and repair integrity of completely arthroscopically repaired large and massive rotator cuff tears. J Bone Joint Surg Am. 2004;86-A:219–24.
5. Neri BR, Chan KW, Kwon YW. Management of massive and irreparable rotator cuff tears. J Shoulder Elb Surg. 2009;18:808–18.
6. Tashjian RZ, Hollins AM, Kim HM, Teefey SA, Middleton WD, Steger-May K, Galatz LM, Yamaguchi K. Factors affecting healing rates after arthroscopic double-row rotator cuff repair. Am J Sports Med. 2010;38:2435–42.
7. Iannotti JP, Deutsch A, Green A, Rudicel S, Christensen J, Marraffino S, Rodeo S. Time to failure after rotator cuff repair: a prospective imaging study. J Bone Joint Surg Am. 2013;95:965–71.
8. Boileau P, Brassart N, Watkinson DJ, Carles M, Hatzidakis AM, Krishnan SG. Arthroscopic repair of full-thickness tears of the supraspinatus: does the tendon really heal? J Bone Joint Surg Am. 2005;87:1229–40.
9. Chung SW, Oh JH, Gong HS, Kim JY, Kim SH. Factors affecting rotator cuff healing after arthroscopic repair: osteoporosis as one of the independent risk factors. Am J Sports Med. 2011;39:2099–107.
10. Goutallier D, Postel JM, Bernageau J, Lavau L, Voisin MC. Fatty muscle degeneration in cuff ruptures. Pre- and postoperative evaluation by CT scan. Clin Orthop Relat Res. 1994;304:78–83.
11. Jost B, Zumstein M, Pfirrmann CW, Gerber C. Long-term outcome after structural failure of Rotator Cuff Repairs. J Bone Joint Surg Am. 2006;88:472–9.
12. Anderson K, Boothby M, Aschenbrener D, van Holsbeeck M. Outcome and structural integrity after arthroscopic Rotator Cuff Repair using 2 rows of fixation: minimum 2-year follow-up. Am J Sports Med. 2006;34:1899–905.
13. McCarron JA, Derwin KA, Bey MJ, Polster JM, Schils JP, Ricchetti ET, et al. Failure with continuity in rotator cuff repair "healing". Am J Sports Med. 2013;41:134–41.
14. Keener JD, Wei AS, Kim HM, Paxton ES, Teefey SA, Galatz LM, et al. Revision arthroscopic Rotator Cuff Repair: repair integrity and clinical outcome. J Bone Joint Surg Am. 2010;92:590–8.
15. Oh JH, Kim SH, Ji HM, Jo KH, Bin SW, Gong HS. Prognostic factors affecting anatomic outcome of Rotator Cuff Repair and correlation with functional outcome. Arthroscopy. 2009;25:30–9.
16. Namdari S, Donegan RP, Chamberlain AM, Galatz LM, Yamaguchi K, Keener JD. Factors affecting outcome after structural failure of repaired rotator cuff tears. J Bone Joint Surg Am. 2014;96:99–105.
17. George MS, Khazzam M. Current concepts review: revision rotator cuff repair. J Shoulder Elb Surg. 2012;21:431–40.
18. Thomazeau H, Rolland Y, Lucas C, Duval JM, Langlais F. Atrophy of the supraspinatus belly. Assessment by MRI in 55 patients with rotator cuff pathology. Acta Orthop Scand. 1996;67:264–8.
19. Walch G, Nove-Josserand L, Levigne C, Renaud E. Tears of the supraspinatus tendon associated with "hidden" lesions of the rotator interval. J Shoulder Elb Surg. 1994;3:353–60.
20. Athwal GS, Sperling JW, Rispoli DM, Cofield RH. Deep infection after rotator cuff repair. J Shoulder Elb Surg. 2007;16:306–11.
21. Kwon YW, Kalainov DM, Rose HA, Bisson LJ, Weiland AJ. Management of early deep infection after rotator cuff repair surgery. J Shoulder Elb Surg. 2005;14:1–5.
22. Chow JC, Gu Y. Material reaction to suture anchor. Arthroscopy. 2004;20:314–6.
23. Nusselt T, Freche S, Klinger HM, Baums MH. Intraosseous foreign body granuloma in rotator cuff repair with bioabsorbable suture anchor. Arch Orthop Trauma Surg. 2010;130:1037–40.
24. Brislin KJ, Field LD, Savoie FH 3rd. Complications after arthroscopic rotator cuff repair. Arthroscopy. 2007;23:124–8.
25. Huberty DP, Schoolfield JD, Brady PC, Vadala AP, Arrigoni P, Burkhart SS. Incidence and treatment of postoperative stiffness following arthroscopic rotator cuff repair. Arthroscopy. 2009;25:880–90.
26. McNamara WJ, Lam PH, Murrell GA. The relationship between shoulder stiffness and rotator cuff healing: a study of 1,533 consecutive arthroscopic rotator cuff repairs. J Bone Joint Surg Am. 2016;98:1879–89.
27. Rains DD, Rooke GA, Wahl CJ. Pathomechanisms and complications related to patient positioning and anesthesia during shoulder arthroscopy. Arthroscopy. 2011;27:532–41.
28. Burkhart SS, Esch JC, Jolson RS. The rotator crescent and rotator cable: an anatomic description of the shoulder's "suspension bridge". Arthroscopy. 1993;9:611–6.
29. Burkhart SS, Denard PJ, Konicek J, Hanypsiak BT. Biomechanical validation of load-sharing rip-stop fixation for the repair of tissue-deficient rotator cuff tears. Am J Sports Med. 2014;42:457–62.
30. Cummins CA, Murrell GA. Mode of failure for Rotator Cuff Repair with suture anchors identified at revision surgery. J Shoulder Elb Surg. 2003;12:128–33.

31. Mall NA, Tanaka MJ, Choi LS, Paletta GA Jr. Factors affecting rotator cuff healing. J Bone Joint Surg Am. 2014;96:778–88.
32. Pauly S, Kieser B, Schill A, Gerhardt C, Scheibel M. Biomechanical comparison of 4 double-row suture-bridging Rotator Cuff Repair techniques using different medial-row configurations. Arthroscopy. 2010;26:1281–8.
33. Mellado JM, Calmet J, Olona M, Ballabriga J, Camins A, Perez del Palomar L, Giné J. MR assessment of the repaired rotator cuff: prevalence, size, location, and clinical relevance of tendon rerupture. Eur Radiol. 2006;16:2186–96.
34. Khazzam M, Kuhn JE, Mulligan E, Abboud JA, Baumgarten KM, Brophy RH, et al. Magnetic resonance imaging identification of rotator cuff retears after repair: interobserver and intraobserver agreement. Am J Sports Med. 2012;40:1722–7.
35. Mohana-Borges AV, Chung CB, Resnick D. MR imaging and MR arthrography of the postoperative shoulder: spectrum of normal and abnormal findings. Radiographics. 2004;24:69–85.
36. Gaenslen ES, Satterlee CC, Hinson GW. Magnetic resonance imaging for evaluation of failed repairs of the rotator cuff. Relationship to operative findings. J Bone Joint Surg Am. 1996;78:1391–6.
37. Magee TH, Gaenslen ES, Seitz R, Hinson GA, Wetzel LH. MR imaging of the shoulder after surgery. AJR Am J Roentgenol. 1997;168:925–8.
38. de Jesus JO, Parker L, Frangos AJ, Nazarian LN. Accuracy of MRI, MR arthrography, and ultrasound in the diagnosis of rotator cuff tears: a meta-analysis. AJR Am J Roentgenol. 2009;192:1701–7.
39. Sugaya H, Maeda K, Matsuki K, Moriishi J. Repair integrity and functional outcome after arthroscopic double-row rotator cuff repair. A prospective outcome study. J Bone Joint Surg Am. 2007;89:953–60.
40. Prickett WD, Teefey SA, Galatz LM, Calfee RP, Middleton WD, Yamaguchi K. Accuracy of ultrasound imaging of the rotator cuff in shoulders that are painful postoperatively. J Bone Joint Surg Am. 2003;85-A:1084–9.
41. Nazarian LN, Jacobson JA, Benson CB, Bancroft LW, Bedi A, McShane JM, et al. Imaging algorithms for evaluating suspected rotator cuff disease: society of radiologists in ultrasound consensus conference statement. Radiology. 2013;267:589–95.
42. Strauss EJ, McCormack RA, Onyekwelu I, Rokito AS. Management of failed arthroscopic rotator cuff repair. J Am Acad Orthop Surg. 2012;20:301–9.
43. Denard PJ, Burkhart SS. Arthroscopic revision Rotator Cuff Repair. J Am Acad Orthop Surg. 2011;19:657–66.
44. Mannava S, Samborski SA, Kenney RJ, Maloney MD, Voloshin I. Options for failed rotator cuff repair. Sports Med Arthrosc Rev. 2018;26:134–8.
45. Bokor DJ, Hawkins RJ, Huckell GH, Angelo RL, Schickendantz MS. Results of nonoperative management of full-thickness tears of the rotator cuff. Clin Orthop Relat Res. 1993;294:103–10.
46. Goldberg BA, Nowinski RJ, Matsen FA 3rd. Outcome of nonoperative management of full-thickness rotator cuff tears. Clin Orthop Relat Res. 2001;(382):99–107.
47. Collin P, Matsumura N, Ladermann A, Denard PJ, Walch G. Relationship between massive chronic rotator cuff tear pattern and loss of active shoulder range of motion. J Shoulder Elb Surg. 2014;23:1195–202.
48. Chuang MJ, Jancosko J, Nottage WM. Clinical outcomes of single-row arthroscopic revision rotator cuff repair. Orthopedics. 2014;37:e692–8.
49. Ladermann A, Denard PJ, Burkhart SS. Midterm outcome of arthroscopic revision repair of massive and nonmassive rotator cuff tears. Arthroscopy. 2011;27:1620–7.
50. Piasecki DP, Verma NN, Nho SJ, Bhatia S, Boniquit N, Cole BJ, et al. Outcomes after arthroscopic revision rotator cuff repair. Am J Sports Med. 2010;38:40–6.
51. Gartsman GM. Massive, irreparable tears of the rotator cuff. Results of operative debridement and subacromial decompression. J Bone Joint Surg Am. 1997;79:715–21.
52. Shon MS, Koh KH, Lim TK, Kim WJ, Kim KC, Yoo JC. Arthroscopic partial repair of irreparable rotator cuff tears: preoperative factors associated with outcome deterioration over 2 Years. Am J Sports Med. 2015;43:1965–75.
53. Berth A, Neumann W, Awiszus F, Pap G. Massive rotator cuff tears: functional outcome after debridement or arthroscopic partial repair. J Orthop Traumatol. 2010;11:13–20.
54. Konig MA, Braunstein VA. Tendon repair leads to better long-term clinical outcome than debridement in massive rotator cuff tears. Open Orthop J. 2017;11:546–53.
55. Lo IK, Burkhart SS. Arthroscopic repair of massive, contracted, immobile rotator cuff tears using single and double interval slides: technique and preliminary results. Arthroscopy. 2004;20:22–33.
56. Boileau P, Gonzalez JF, Chuinard C, Bicknell R, Walch G. Reverse total shoulder arthroplasty after failed rotator cuff surgery. J Shoulder Elb Surg. 2009;18:600–6.
57. Cartucho A. Massive cuff tears. In: Randelli P, editor. Arthroscopy. Berlin: Springer; 2016. p. 597–608.
58. Iannotti JP, Codsi MJ, Kwon YW, Derwin K, Ciccone J, Brems JJ. Porcine small intestine submucosa augmentation of surgical repair of chronic two-tendon rotator cuff tears. A randomized, controlled trial. J Bone Joint Surg Am. 2006;88:1238–44.
59. Wong I, Burns J, Snyder S. Arthroscopic GraftJacket repair of rotator cuff tears. J Shoulder Elb Surg. 2010;19(2 Suppl):104–9.
60. Mori D, Funakoshi N, Yamashita F. Arthroscopic surgery of irreparable large or massive rotator cuff tears with low-grade fatty degeneration of the infraspinatus: patch autograft procedure versus partial repair procedure. Arthroscopy. 2013;29:1911–21.
61. Pandey R, Tafazal S, Shyamsundar S, Modi A, Singh HP. Outcome of partial repair of massive rotator cuff

tears with and without human tissue allograft bridging repair. Shoulder Elbow. 2017;9:23–30.
62. Gupta AK, Hug K, Berkoff DJ, Boggess BR, Gavigan M, Malley PC, et al. Dermal tissue allograft for the repair of massive irreparable rotator cuff tears. Am J Sports Med. 2012;40:141–7.
63. Ladermann A, Denard PJ, Burkhart SS. Management of failed rotator cuff repair: a systematic review. J ISAKOS. 2016;1:32–7.
64. Cai YZ, Zhang C, Jin RL, Shen T, Gu PC, Lin XJ, et al. Arthroscopic rotator cuff repair with graft augmentation of 3-Dimensional biological collagen for moderate to large tears: a randomized controlled study. Am J Sports Med. 2018;46:1424–31.
65. Mihata T, Lee TQ, Watanabe C, Fukunishi K, Ohue M, Tsujimura T, Kinoshita M. Clinical results of arthroscopic superior capsule reconstruction for irreparable rotator cuff tears. Arthroscopy. 2013;29:459–70.
66. Mihata T, McGarry MH, Kahn T, Goldberg I, Neo M, Lee TQ. Biomechanical role of capsular continuity in superior capsule reconstruction for irreparable tears of the supraspinatus tendon. Am J Sports Med. 2016;44:1423–30.
67. Denard PJ, Brady PC, Adams CR, Tokish JM, Burkhart SS. Preliminary results of arthroscopic superior capsule reconstruction with dermal allograft. Arthroscopy. 2018;34:93–9.
68. Gerber C. Latissimus dorsi transfer for the treatment of irreparable tears of the rotator cuff. Clin Orthop Relat Res. 1992;275:152–60.
69. Hertel R, Lambert SM, Ballmer FT. The deltoid extension lag sign for diagnosis and grading of axillary nerve palsy. J Shoulder Elb Surg. 1998;7:97–9.
70. Gervasi E, Causero A, Parodi PC, Raimondo D, Tancredi G. Arthroscopic latissimus dorsi transfer. Arthroscopy. 2007;23:1243.e1–4.
71. Nove-Josserand L, Costa P, Liotard JP, Safar JF, Walch G, Zilber S. Results of latissimus dorsi tendon transfer for irreparable cuff tears. Orthop Traumatol Surg Res. 2009;95:108–13.
72. Aoki M, Okamura K, Fukushima S, Takahashi T, Ogino T. Transfer of latissimus dorsi for irreparable rotator-cuff tears. J Bone Joint Surg Br. 1996;78:761–6.
73. Werner CM, Zingg PO, Lie D, Jacob HA, Gerber C. The biomechanical role of the subscapularis in latissimus dorsi transfer for the treatment of irreparable rotator cuff tears. J Shoulder Elb Surg. 2006;15:736–42.
74. Habermeyer P, Magosch P, Rudolph T, Lichtenberg S, Liem D. Transfer of the tendon of latissimus dorsi for the treatment of massive tears of the rotator cuff: a new single-incision technique. J Bone Joint Surg Br. 2006;88:208–12.
75. Shin JJ, Saccomanno MF, Cole BJ, Romeo AA, Nicholson GP, Verma NN. Pectoralis major transfer for treatment of irreparable subscapularis tear: a systematic review. Knee Surg Sports Traumatol Arthrosc. 2016;24:1951–60.
76. Kany J, Guinand R, Croutzet P, Valenti P, Werthel JD, Grimberg J. Arthroscopic-assisted latissimus dorsi transfer for subscapularis deficiency. Eur J Orthop Surg Traumatol. 2016;26:329–34.
77. Mun SW, Kim JY, Yi SH, Baek CH. Latissimus dorsi transfer for irreparable subscapularis tendon tears. J Shoulder Elb Surg. 2018;27:1057–64.
78. Senekovic V, Poberaj B, Kovacic L, Mikek M, Adar E, Dekel A. Prospective clinical study of a novel biodegradable sub-acromial spacer in treatment of massive irreparable rotator cuff tears. Eur J Orthop Surg Traumatol. 2013;23:311–6.
79. Szollosy G, Rosso C, Fogerty S, Petkin K, Lafosse L. Subacromial spacer placement for protection of Rotator Cuff Repair. Arthrosc Tech. 2014;3:e605–9.
80. Mulieri P, Dunning P, Klein S, Pupello D, Frankle M. Reverse shoulder arthroplasty for the treatment of irreparable rotator cuff tear without glenohumeral arthritis. J Bone Joint Surg Am. 2010;92:2544–56.
81. Ernstbrunner L, Suter A, Catanzaro S, Rahm S, Gerber C. Reverse total shoulder arthroplasty for massive, irreparable rotator cuff tears before the age of 60 years: long-term results. J Bone Joint Surg Am. 2017;99:1721–9.
82. Guery J, Favard L, Sirveaux F, Oudet D, Mole D, Walch G. Reverse total shoulder arthroplasty. Survivorship analysis of eighty replacements followed for five to ten years. J Bone Joint Surg Am. 2006;88:1742–7.
83. Boileau P, Chuinard C, Roussanne Y, Bicknell RT, Rochet N, Trojani C. Reverse shoulder arthroplasty combined with a modified latissimus dorsi and teres major tendon transfer for shoulder pseudoparalysis associated with dropping arm. Clin Orthop Relat Res. 2008;466:584–93.
84. Boileau P, Chuinard C, Roussanne Y, Neyton L, Trojani C. Modified latissimus dorsi and teres major transfer through a single delto-pectoral approach for external rotation deficit of the shoulder: as an isolated procedure or with a reverse arthroplasty. J Shoulder Elb Surg. 2007;16:671–82.
85. L'Épiscopo JB. Tendon transplantation in obstetrical paralysis. Am J Surg. 1934;25(1):122–5.

How to Avoid Complications in Tendon Transfers

33

Roman Brzoska, Hubert Laprus, and Viktoras Jermolajevas

33.1 Introduction

Tendon transfer is a well-documented procedure for treating irreparable rotator cuff tears. They provide good pain relief and improvement of shoulder function over a long-term follow-up period.

Thanks to tendon transfers, we can treat irreparable tears of either posterosuperior or anterosuperior rotator cuff. Currently, based on long-term follow-up studies it seems like the right candidates for this kind of procedure are relatively young patients with massive and irreparable tears and with preserved shoulder girdle muscles and preserved anteflexion. In addition, these procedures are also useful during reverse total shoulder arthroplasty (rTSA) in case of the teres minor tear or inefficiency as the last chance to regain external rotation. Unfortunately, these operations are surgically challenging, and the effects may not meet the expectations neither for patients nor for surgeons.

There is also a challenging issue to clarify the definition of a massive and irreparable cuff tear. Massive tears have been described as tears larger than 5 cm in diameter [1] or as tears involving two or three tendons [2]. Moreover, a massive tear may be evaluated as irreparable preoperatively with a combination of tendon retraction to the glenoid edge and muscular atrophy on magnetic resonance imaging (stage 3 according to Thomazeau's classification) [3] and/or a muscle fatty infiltration (stages 3 and 4 on computed tomography arthrography according to Goutallier's classification) [4]. A tear may also be evaluated as irreparable intraoperatively when after extensive subacromial bursectomy and periglenoidal capsulotomy the surgeon is not able to pull the supra- and/or the infraspinatus tendon to the edge of the greater tuberosity.

The purpose of the following chapter is to summarize current knowledge about the most commonly used tendon transfer procedures in massive, irreparable rotator cuff tears. Another objective is to provide valuable tips and tricks that could help to avoid complications and minimize unsatisfactory postoperative results.

33.2 Transfers for Irreparable Posterosuperior Rotator Cuff Tears

33.2.1 Latissimus Dorsi Transfer

Latissimus dorsi transfer (LDT) is the best known and documented of all muscle transfers used in shoulder surgery. Originally developed by Gerber

R. Brzoska · H. Laprus (✉)
St Luke's Hospital, Bielsko-Biala, Poland

V. Jermolajevas
Traumatology and Orthopedics Department, Klaipėda Regional Hospital, Klaipėda, Lithuania

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_33

in 1988 [5], it is used today with various modifications. The idea of this transfer is an attempt to recover external rotation in case of complete, irreparable posterosuperior rotator cuff tear by posterior force reconstitution. Another idea behind this transfer is an attempt to lower the humeral head and thus to reduce the risk of rotator cuff arthropathy by acting latissimus dorsi muscle as the humeral head depressor.

LDT was initially used mainly in young men engaged in manual works, but the latest analyses did not confirm that factors such as gender, dominant hand, or occupation have a significant impact on results [6]. Nonetheless, age is a contentious issue, as Ianotti et al. [7] found a significant influence of age and gender on results of LDT. They reported that four out of five dissatisfied patients were women with a mean age of 60. Eight of nine satisfied patients were men with a mean age of 52 [7].

There is also paucity of consensus about the influence of prior surgery on the outcome. Few authors proved that presence of prior rotator cuff repair has resulted in poorer patient outcomes [8–12]. On the other hand, Weening and Willems [13] as weel as Debeer and De Smet found no difference in final outcome between LDT as a primary or revision procedure, found no difference between primary and revision procedures [14]. This reported lack of difference might be since most of the preoperative revision procedures were arthroscopic with no deltoid detachment.

Subscapularis tendon tear is a clear risk factor for failure of LDT [9, 13, 15, 16]. Gerber et al. conducted a biomechanical study which showed that a LDT after a complete subscapularis tear was responsible for a great imbalance of the shoulder joint with anterior dislocation forces [9]. However, small tears of the upper third of the subscapularis completely repaired during surgery do not affect the outcome [8, 12, 17].

The role of the teres minor (Tm) in LDT was recently highlighted, but it is not clearly understood. Nové-Josserand et al. [12] found that patients with severe atrophy of Tm had lower function than patients with no or moderate Tm atrophy; however, patients with severe Tm atrophy had greater increase in Constant score than patients with no or moderate atrophy.

Pseudoparalytic shoulder was criteria of exclusion for many authors [9, 17, 18], but in a study conducted by Valenti et al. [17], patients with preoperative forward flexion lower than 80° had better increase of flexion than patients with preoperative anteflexion higher than 120°. However, in one study, in seven patients considered as pseudoparalytic, nonsignificant increase in forward flexion was gained [19].

Techniques for LDT are evolving. Habermeyer et al. described a single-incision approach that uses a more posterior attachment of the transfer into the humeral head [20]. Currently, arthroscopically assisted LTD is wide and results similar to those for traditional open technique [21]. Benefits of arthroscopic technique are related to reduced morbidity to the deltoid muscle and stronger resistance of the transferred tendon to traction due to fixation of the tendon in a bone tunnel.

The most common complications are hematoma (up to 14.3%) and secondary tendon graft rupture (up to 44%) [21].

33.2.2 Teres Major Transfer

Transfer of the latissimus dorsi is the most frequent procedure for posterosuperior massive rotator cuff tears. Teres major transfer (TMT) has been advocated as an alternative with underlined potentially favorable orientation of muscle fibers, like better resembling the infraspinatus muscle [22, 23]. Patients qualified for the TMT procedure usually have similar symptoms as with LDT, mainly the limitation of external rotation and forward flexion. What determines the quality of muscle transfer is the durability of clinical improvements. In the case of the LDT, we have several studies assessing the good function of the transfer after years. In the case of TMT, there are few studies with long-term follow-up [24].

Kolk et al. [25] in their study proved improvement in shoulder function and pain after TMT over the course of 10 years, and these results were comparable to long-term results of LDT. The authors stated that TMT transfer should

be a valid substitute for the infraspinatus muscle. Due to its orientation, TM should work as biological augmentation of the infraspinatus [25].

Indeed, it should be underlined that TMT is much more surgically demanding than LDT. Lower subscapular nerve innervates the TM in over 85% of cases, with the difference supplied by the thoracodorsal nerve. The vascular supply inserts directly into the muscle within 2 cm of the nearby nerves, in the middle of the muscle belly. Often shorter in length than local nerves, the vascular structures tend to be the limiting factor when performing a TMT [26]. What is more, the tendon is not as long as LD and detachment flush to the bone is necessary to avoid limitation of the tendon. Mansat et al. described the following recommendations for TMT: the patient should be under the age of 55 years with a proper understanding of the condition and treatment and an intact subscapularis and anterior supraspinatus cable [26, 27].

33.2.3 Lower Trapezius Transfer

Lower trapezius transfer (LTT) is a relatively novel transfer developed by Elhassan et al. initially for patients with brachial plexus palsy [28]. After first promising results, this technique has been recently implemented for patients with irreparable tear of the posterosuperior rotator cuff [29]. In this technique, a two-incision approach is performed as well. The first incision is similar to that used for LDT. The second incision is based 1 cm medial to the scapula. The lower trapezius attachment to the scapula is released and an Achilles allograft is used to augment for length. A subcutaneous tunnel is created from the medial incision to the lateral incision deep to the deltoid. The tendon is transferred to the footprint of the supraspinatus and upper part of the infraspinatus tendon.

In a cadaveric study, Omid et al. concluded that the LTT was superior to LDT for restoration of GH mechanics and joint reaction forces [30]. Origin of the lower trapezius is cranial to the origin of the LD, just medial to the infraspinatus fossa, causing the line of pull of the lower trapezius to be more like the infraspinatus tendon [29].

Subscapularis tear is not a contraindication to the transfer due to the ease of training the transfer, because the trapezius has been shown to contract during shoulder external rotation [31].

Elhassan et al. in their clinical study found that at an average follow-up of 47 months, 32 of the 33 patients who underwent LTT prolonged with Achilles tendon allograft experienced significant improvement of pain and external rotation and this improvement was higher in case of preserved forward flexion (>60°) [29].

33.3 Transfers for Irreparable Anterosuperior Rotator Cuff Tears

33.3.1 Pectoralis Major Transfer

The purpose of pectoralis major transfer (PMT) is to stand in for the subscapularis tear by exerting an internal rotation centering force. Wirth and Rockwood originally described the PMT in 1997 [32]. The PMT was performed anterior to the conjoined tendon and resulted in a high satisfaction rate at 5-year follow-up. Resch et al. [33] adapted this technique to transfer only the superior two-thirds of the tendon under the conjoined tendon. Subsequently, Warner transferred the inferior sternal head attachment under the clavicular head but anterior to the conjoined tendon to avoid injury to the musculocutaneous nerve [34].

Biomechanically, subcoracoid placement of the transfer is better, but there have been no comparative studies to date. Galatz et al. [35] demonstrated a subcoracoid PMT for irreparable rotator cuff tears with over 75% satisfaction at almost 18-month follow-up with improved ASES and VAS scores. Elhassan et al. [36] evaluated patients treated with Warner's technique and found that those with a preoperatively concentric humeral head had better outcomes. Other recommendations include: age less than 65 years (ideally <40), intact or repairable posterosuperior rotator cuff, and minimal glenohumeral osteoarthritis. It

was noted that if the subcoracoid transfer is successful, external rotation loss up to 25° can be expected owing to a tenodesis effect [33].

33.3.2 Latissimus Dorsi Transfer and Teres Major Transfer

The most commonly reported procedure for irreparable subscapularis tears is PMT. LDT and TMT have been frequently reported as a common transfer performed for patients with massive irreparable posterosuperior rotator cuff tears. Anatomic feasibility of LDT or TMT or both to reconstruct an irreparable tear of the subscapularis tendon was firstly performed by Elhassan et al. [37]. After cadaveric dissection of 20 specimens they concluded that transfer of the LD and TM tendons as isolated single transfers, together but as separate transfers, or as a combined transfer to different locations of the subscapularis tendon insertion (inferior, middle, and superior third) is feasible. They proved also that risk of nerve compression, including the axillary, radial, and musculocutaneous nerves, is very low in most of these types of transfers excluded transfer of the TM to the proximal third of the SS tendon which may increase risk of compression of the axillary nerve. Because of the long tendinous portion of the LD tendon compared with the TM tendon, transferring the LD tendon would be easier, with essentially no risk of nerve compression [37].

Clinical studies are needed to confirm the effectiveness of this transfer in reconstructing the function of subscapularis insufficiency.

33.4 Tendon Transfers for Reverse Total Shoulder Arthroplasty

In recent years, the indications for rTSA have been expanded and have become a therapeutic option for patients with a massive rotator cuff tear. Preoperative or postoperative external rotation deficits may increase the need for additional tendon transfers to recover activities of daily living and restore external rotation in case of positive external rotation lag sign. Gerber et al. [38] first reported benefits after rTSA with LDT for patients with external rotation deficits. In their study, survivorship of the transfer was excellent as well as function at midterm follow-up [38]. Subsequently, Boileau et al. [39] and Boughebri et al. [40] published good results with external rotation regaining by modified L'Episcopo procedures combined with rTSA. In their studies, modification was performed by single incision and results at short follow-up were really encouraging.

33.5 Tips and Tricks to Avoid Complications with Tendon Transfers

33.5.1 Anchor Pullout

The most common reason for anchor pullout is improper mobilization of the muscles and tendons. It concerns either muscle or tendon which is transferred during procedure and the deltoid muscle. Tendon must be mobilized as far as it's possible with part of the muscle if necessary. What is more, for a more feasible passage of the transferred tendon, a wide release of the subcutaneous tissue from deltoid muscle should be accomplished. This is particularly important in case of combined LDT/TMT, where TM has a rather short tendinous part.

The second most common reason of anchor pullout is due to poor quality of cancellous bone of the humeral head. The best tip in this case is to replace the anchors with buttons (Fig. 33.1). The second good tip is to secure the anchor by threads from previously used anchor (e.g., for partial repair of anterior rotator cuff repair). In such situation, the surgeon can use the sutures from anchors for subscapularis repair to lock and secure the anchor used for fixation of transfer.

33.5.2 Tendon to Bone Friction

This is a very uncommon but severe complication and results from misdiagnosed and uncor-

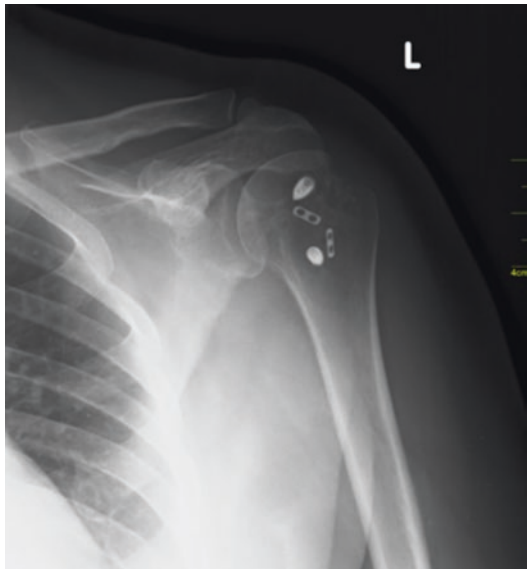


Fig. 33.1 Endobutton fixation for tendon transfer

rected anterior subluxation of the humeral head. This subluxation is the result of a tear and insufficiency of the subscapularis tendon that leads to shoulder balance impairment. It is crucial to identify it either in preoperative MRI or at the clinical examination. Anterior subluxation increases tendon-to-bone surface friction and finally leads to collapse of the tendon into the humeral head and loss of stability. The sequela of this condition is upper migration of the humeral head and subsequent loss of the external rotation and forward flexion.

33.5.3 Infections

The risk of infection is enrolled into tendon transfer, because of the approach, localization, and continuous exposition to skin bacteria. Furthermore, traditional LDT relies on graft harvesting from extended approach that even increases this exposition.

One of the best but surgical challenging solutions for reducing exposure time and risk of infection is to harvest the graft from an arthroscopic approach. For better visualization, additional anterior and anterolateral portals are created (Fig. 33.2). Afterward, graft can be prepared arthroscopically before fixation (Fig. 33.3). Second solution is to not forget about suture contaminations by bacteria from skin and washing the threads very often.

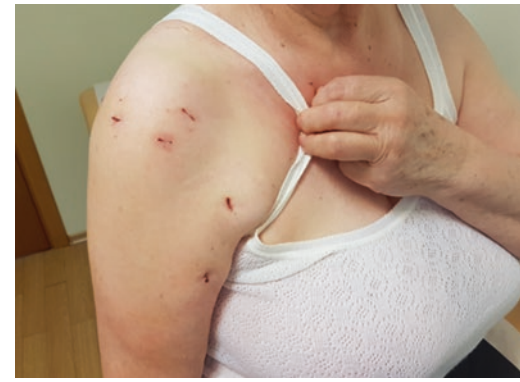


Fig. 33.2 Additional portals for LD arthroscopic harvesting. Suprapectoral portal is clearly visible

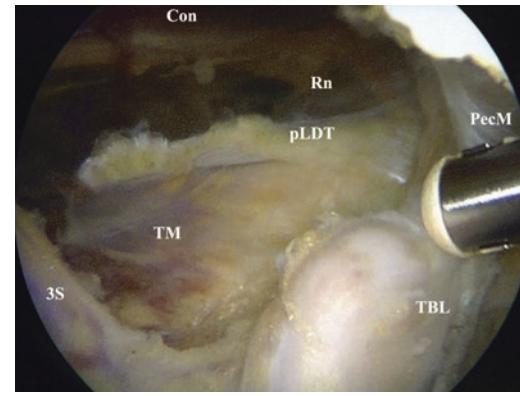


Fig. 33.3 Arthroscopic latissimus dorsi tendon harvesting. Anterior view from anterosuperior portal, looking inferiorly in plane of humerus (right shoulder with patient in beach chair position). An electrode is in the suprapectoral portal. The superior part of the latissimus dorsi tendon insertion has just been released, with the inferior part still attached. *Con* conjoined tendon, *PecM* pectoralis major tendon, *pLDT* partially released latissimus dorsi tendon, *Rn* radial nerve, *TBL* biceps longus tendon, *3S* 3 sisters [anterior circumflex vessels], *TM* teres major muscle

33.5.4 Neurological Complications

Axillary nerve tensioning is a complication that surgeons traditionally concerned pendant tendon

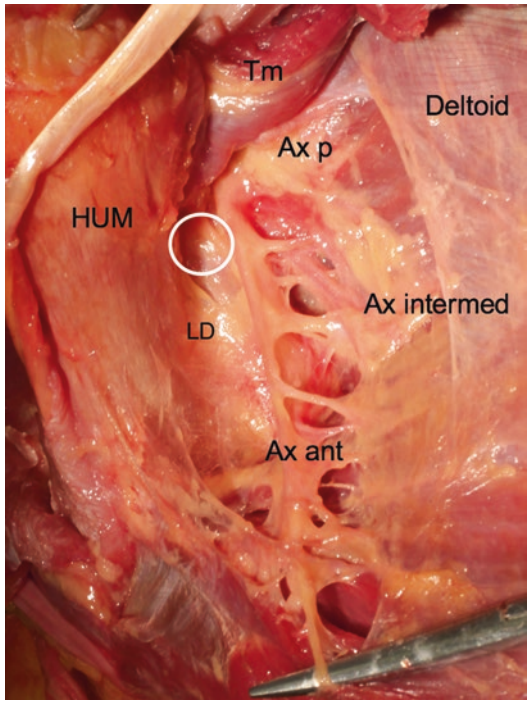


Fig. 33.4 Relationship between axillary nerve (Ax), muscles (*Tm* teres minor, *LD* latissimus dorsi), and deltoid muscle. Proper place for passage indicated in the circle

transfers, especially TMT and LDT. Teres major, because it is a bulky muscle with a small tendon, when transferred generally increases the risk of contact pressure between the transferred muscle and the axillary nerve. Some surgeons recommend deltoid muscle excision to reduce the tension, but we disagree with that and recommend rather focusing on identification of the proper place for tendon passage (Fig. 33.4). Releasing the nerve arthroscopically before harvesting the tendon can be also helpful.

References

1. Cofield RH. Rotator cuff disease of the shoulder. J Bone Joint Surg Am. 1985;67:974–9.
2. Gerber C, Fuchs B, Hodler J. The results of repair of massive tears of the rotator cuff. J Bone Joint Surg Am. 2000;82:505–15.
3. Thomazeau H, Boukobza E, Morcet N, Chaperon J, Langlais F. Prediction of rotator cuff repair results by magnetic resonance imaging. Clin Orthop Relat Res. 1997;344:275–83.
4. Goutallier D, Postel JM, Bernageau J, Lavau L, Voisin MC. Fatty muscle degeneration in cuff ruptures. Pre- and postoperative evaluation by CT scan. Clin Orthop Relat Res. 1994;304:78–83.
5. Gerber C, Vinh TS, Hertel R, Hess CW. Latissimus dorsi transfer for the treatment of massive tears of the rotator cuff. A preliminary report. Clin Orthop Relat Res. 1988;232:51–61.
6. Namdari S, Voleti P, Baldwin K, Glaser D, Hu Man GR. Latissimus dorsi tendon transfer for irreparable rotator cuff tears: a systematic review. J Bone Joint Surg Am. 2012;94-A:891–8.
7. Ianotti JP, Hennigan S, Herzog R, Kella S, Kelley M, Leggin B. Latissimus dorsi tendon transfer for irreparable posterosuperior rotator cuff tears. Factors affecting outcome. J Bone Joint Surg Am. 2006;88:342–8.
8. Warner JJ, Parsons IM IV. Latissimus dorsi tendon transfer: a comparative analysis of primary and salvage reconstruction of massive, irreparable rotator cuff tears. J Shoulder Elb Surg. 2001;10:514–21.
9. Gerber C, Maquieira G, Espinosa N. Latissimus dorsi transfer for the treatment of irreparable rotator cuff tears. J Bone Joint Surg Am. 2006;88-A:113–20.
10. Irlenbusch U, Bracht M, Gansen HK, Lorenz U, Thiel J. Latissimus dorsi transfer for irreparable rotator cuff tears: a longitudinal study. J Shoulder Elb Surg. 2008;17:527–34.
11. Moursy M, Forstner R, Koller H, Resch H, Tauber M. Latissimus dorsi tendon transfer for irreparable rotator cuff tears: a modified technique to improve tendon transfer integrity. J Bone Joint Surg Am. 2009;91-A:1924–31.
12. Nové-Josserand L, Costa P, Liotard JP, et al. Results of latissimus dorsi tendon transfer for irreparable cuff tears. Orthop Traumatol Surg Res. 2009;95:108–13.
13. Weening AA, Willems WJ. Latissimus dorsi transfer for treatment of irreparable rotator cuff tears. Int Orthop. 2010;34:1239–44.
14. Debeer P, De Smet L. Outcome of latissimus dorsi transfer for irreparable rotator cuff tears. Acta Orthop Belg. 2010;76:449–55.
15. Moursy M, Fortsner R, Loller H, Resch H, Tauber M. Latissimus dorsi transfer for irreparable rotator cuff tears: a modified technique to improve tendon transfer integrity. J Bone Joint Surg Am. 2009;91:1924–31.
16. Miniaci A, MacLeod M. Transfer of the latissimus dorsi muscle after failed repair of a massive tear of the rotator cuff. A 2–5 year review. J Bone Joint Surg Am. 1999;81:1120–7.
17. Valenti P, Kalouche I, Diaz LC, Kaouar A, Kilinc A. Results of latissimus dorsi tendon transfer in primary or salvage reconstruction of irreparable rotator cuff tears. Orthop Traumatol Surg Res. 2010;96:133–8.
18. Lichtenberg S, Magoch P, Habermeyer P. Are there advantages of the combined latissimus dorsi transfer according to L'Episcopo compared with the isolated latissimus dorsi transfer according to Herzberg after a mean follow-up of 6 years? A matched-pair analysis. J Shoulder Elb Surg. 2012;21:1499–507.

19. Pearsall AW, Madanagopal S, Karas SG. Transfer of the latissimus dorsi as a salvage procedure for failed debridement and attempted repair of massive rotator cuff tears. Orthopedics. 2007;30:943–9.
20. Habermeyer P, Magosch P, Rudolph T, Lichtenberg S, Liem D. Transfer of the tendon of latissimus dorsi for the treatment of massive tears of the rotator cu: a new single-incision technique. J Bone Joint Surg Br. 2006;88:208–12.
21. Grimberg J, Kany J. Latissimus dorsi tendon transfer for irreparable postero-superior cuff tears: current concepts, indications, and recent advances. Curr Rev Musculoskelet Med. 2014;7:22–32.
22. Henseler JF, Nagels J, van der Zwaal P, Nelissen RG. Teres major tendon transfer for patients with massive irreparable posterosuperior rotator cuff tears: short-term clinical results. Bone Joint J. 2013;95-B:523–9.
23. Magermans DJ, Chadwick EK, Veeger HE, van der Helm FC, Rozing PM. Bio-mechanical analysis of tendon transfers for massive rotator cuff tears. Clin Biomech (Bristol, Avon). 2004;19:350–7.
24. Kolk A, Henseler JF, Overes FJ, Nagels J, Nelissen RGHH. Teres major tendon transfer in the treatment of irreparable posterosuperior rotator cuff tears. Bone Joint J. 2018;100-B(3):309–17.
25. Celli L, Rovesta C, Marongiu MC, Manzieri S. Transplantation of teres major muscle for infraspinatus muscle in irreparable rotator cuff tears. J Shoulder Elb Surg. 1998;7:485–90.
26. Axe JM. Tendon transfers for irreparable rotator cuff tears: an update. EFORT Open Rev. 2016;1(1):18–24.
27. Mansat P, Dotziz A, Bellumore Y, Mansat M. Teres major flap: surgical anatomy, technique of harvesting, methods of fixation, postoperative management. In: Valenti P, editor. Tendon transfer for irreparable rotator cuff tear. Paris: Springer; 2011. p. 49–64.
28. Elhassan B. Lower trapezius transfer for shoulder external rotation in patients with paralytic shoulder. J Hand Surg Am. 2014;39:556–62.29.
29. Elhassan B, Wagner E, Werthel JD. Outcome of lower trapezius transfer to reconstruct massive irreparable posterior-superior rotator cuff tear. J Shoulder Elb Surg. 2016;25(8):1346–53.
30. Omid R, Heckmann N, Wang L, et al. Biomechanical comparison between the trapezius transfer and latissimus transfer for irreparable posterosuperior rotator cu tears. J Shoulder Elb Surg. 2015;24:1635–43.
31. Smith J, Padgett DJ, Dahm DL, Kaufman KR, Harrington SP, Morrow DA, et al. Electromyographic activity in the immobilized shoulder girdle musculature during contralateral upper limb movements. J Shoulder Elb Surg. 2004;13:583–8.
32. Wirth MA, Rockwood CA Jr. Operative treatment of irreparable rupture of the subscapularis. J Bone Joint Surg Am. 1997;79-A:722–31.
33. Resch H, Povacz P, Ritter E, Matschi W. Transfer of the pectoralis major muscle for the treatment of irreparable rupture of the subscapularis tendon. J Bone Joint Surg Am. 2000;82-A:372–82.
34. Warner JJ. Management of massive irreparable rotator cuff tears: the role of tendon transfer. Instr Course Lect. 2001;50:63–71.
35. Klepps S, Galatz LM, Yamaguchi K. Subcoracoid pectoralis major transfer: a salvage procedure for irreparable subscapularis deficiency. Tech Shoulder Elb Surg. 2001;2:85–91.
36. Elhassan B, Ozbaydar M, Massimini D, et al. Transfer of pectoralis major for the treatment of irreparable tears of subscapularis: does it work? J Bone Joint Surg Br. 2008;90-B:1059–65.
37. Elhassan B, Christensen TJ, Wagner ER. Feasibility of latissimus and teres major transfer to reconstruct irreparable subscapularis tendon tear: an anatomic study. J Shoulder Elb Surg. 2014;23:492–9.
38. Gerber C, Pennington SD, Lingenfelter EJ, Sukthankar A. Reverse Delta-III total shoulder replacement combined with latissimus dorsi transfer. A preliminary report. J Bone Joint Surg Am. 2007;89-A:940–7.
39. Boileau P, Chuinard C, Roussanne Y, Neyton L, Trojani C. Modified latissimus dorsi and teres major transfer through a single delto-pectoral approach for external rotation deficit of the shoulder: as an isolated procedure or with a reverse arthroplasty. J Shoulder Elb Surg. 2007;16:671–82.
40. Boughebri O, Kilinc A, Valenti P. Reverse shoulder arthroplasty combined with a latissimus dorsi and teres major transfer for a deficit of both active elevation and external rotation. Results of 15 cases with a minimum of 2-year follow-up. Orthop Traumatol Surg Res. 2013;99:131–7.

34 Management of Bone Loss in Rotator Cuff Tear Arthropathy

Maristella F. Saccomanno, Alessandra Scaini, and Giuseppe Milano

34.1 Introduction

The cuff tear arthropathy (CTA) was first described by Neer in the early 1980s [1] as "degenerative changes of the glenohumeral joint consequent to a massive rotator cuff tear" and further defined by Jensen in 1999 [2] as a disease characterized by three main findings: (a) massive rotator cuff tear associated with shoulder pain, muscle atrophy, and loss of motion; (b) degenerative changes in the glenohumeral joint; and (c) upward migration of the humeral head observable on x-rays in anteroposterior (AP) view.

Subsequent radiological classification aimed to define and correlate progressive stages of the disease and consequent treatment strategies [3, 4].

Interestingly, management of CTA has largely changed in the last decades in a way that probably nothing else did in orthopedics. At present, improved arthroscopic techniques and emerging technologies, such as superior capsule reconstruction, may provide a possible treatment solution for certain stages [5]. However, when degenerative changes and bone loss occur, reverse shoulder arthroplasty (RSA) does remain the best treatment option. As imaging tools, design, and biomechanical rationale of RSA and surgical techniques improved, there have been expanding options in augmentation techniques and baseplate fixation, which widens the opportunity to improve the functional outcomes even in the late stages of CTA.

The aim of the present chapter is to provide an overview on pathology, classification, and treatment of CTA with bone loss.

34.2 Pathogenesis

From an epidemiological standpoint CTA has been reported to be more common in women, in the 6th–7th decades, particularly involving the dominant shoulder [6]. Several risk factors have been identified: rotator cuff tear, rheumatoid arthritis, crystalline-induced arthropathy, and hemorrhagic shoulder (hemophiliacs/anticoagulants) [6]. Recently, Gumina et al. [7], based on the assumption that the instability consequent to massive cuff tear may worsen in patients with joint laxity and that joint laxity is notoriously more common in women, hypothesized that generalized joint laxity could be a risk factor for development of CTA. However, the authors finally showed no correlation at all between joint laxity and glenohumeral osteoarthritis.

M. F. Saccomanno
Department of Orthopaedics and Traumatology, Fondazione Policlinico Universitario A. Gemelli IRCCS, Rome, Italy

A. Scaini · G. Milano (✉)
Department of Bone and Joint Surgery, University of Brescia, Spedali Civili, Brescia, Italy

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_34

Two main etiopathogenetic theories for CTA have been developed: (a) crystal mediated and (b) rotator cuff tear mediated.

In 1981, Halverson et al. [8] proposed a crystal-mediated theory at the origin of CTA. They coined the term "Milwaukee shoulder syndrome" and hypothesized that the trigger point was an immunologic cascade activated by calcium phosphate-containing crystals in the synovial tissue. Subsequently, McCarty [9] showed that basic calcium phosphate crystal accumulation in the glenohumeral joint actually correlates with rotator cuff deficiency. Synovial cells phagocytize the crystals, releasing prostaglandins and proteases that destroy articular cartilage. A positive feedback cycle accelerates degeneration of the rotator cuff and biceps tendon, leading to glenohumeral joint degradation.

On the opposite, Neer et al. [1] hypothesized the rotator cuff theory, which involves both mechanical and nutritional factors. Rotator cuff tears are thought to produce at least two simultaneous negative effects:

- A muscle unbalance that, based on the force couple theory explained later on by Burkhart et al. [10], leads to the upward migration of the humeral head and consequently to glenoid and acromial wear as well as eccentric humeral head motion and premature wear of the articular cartilage in the areas of higher glenohumeral compression
- Loss of the watertight effect (loss of negative pressure normally existing inside the glenohumeral joint), which allows extravasation of the synovial fluid and, consequently, leads to an impaired delivery of nutrients to the articular surface, so the cartilage is poorly nourished and would easily become atrophied.

Furthermore, pain associated to cuff tear and degenerative changes makes the shoulder range of motion (ROM) rather limited, leading by time to disuse osteoporosis and collapse of the subchondral bone of the humeral head.

34.3 Clinical Features

Patients with CTA are typically elderly and usually describe classical symptoms and functional impairment related both to osteoarthritis and cuff disease. They have a history of progressively worsening pain, accompanied by limited shoulder motion and stiffness. These symptoms may be precipitated by an acute, traumatic event. Patients with a diagnosis of rheumatoid arthritis or of another inflammatory arthropathy may also present with polyarthralgia and a prior history of medical treatment for their systemic disease [11].

The physical examination always starts with a global inspection of both shoulders. Any difference between shoulders in muscle atrophy should be noticed. Swelling as well as clinical evidence of anterosuperior escape of the humeral head are not uncommon and indicate a grossly deficiency of subscapularis and supraspinatus tendons.

Both active and passive ROM are usually very limited by weakness, pain, and stiffness, but at varying degrees. Tests for evaluation of cuff integrity are positive both for pain and strength deficit.

Cervical spine disorders as well as complete deltoid deficiency and any sign of neurological disorders must be ruled out.

34.4 Imaging

Diagnosis of CTA is essentially clinical and radiographic, as standard x-rays in the AP and axillary views may demonstrate characteristic findings. Magnetic resonance (MR) could be helpful in evaluation of cuff tendons and muscle status. A computed tomography (CT) scan is mandatory for preoperative planning especially in the setting of bone loss.

34.4.1 X-Rays

A true AP and axillary views are enough. No specific views are required either for CTA diagnosis or for preoperative planning.

Pathognomonic radiographic signs of CTA are as follows:

- Superior migration of the humeral head, represented by decreased acromiohumeral distance
- Femoralization of the humeral head, which means erosion of the greater tuberosity
- Acetabularization of the acromion, represented by a thinning of the coracoacromial arch and superior glenoid erosion
- Posterior glenoid erosion
- Glenohumeral subluxation as a result of rotator cuff insufficiency
- Osteopenia in both the proximal aspect of the humerus and the glenoid

Joint space narrowing and osteophytes are common findings in CTA as well as in primary osteoarthritis (Fig. 34.1).

CTA has been classified on radiographic imaging according to Hamada [3] and Seebauer [4].

The Hamada classification [3] (Fig. 34.2) depicts the process of progressive superior migration of the humeral head:

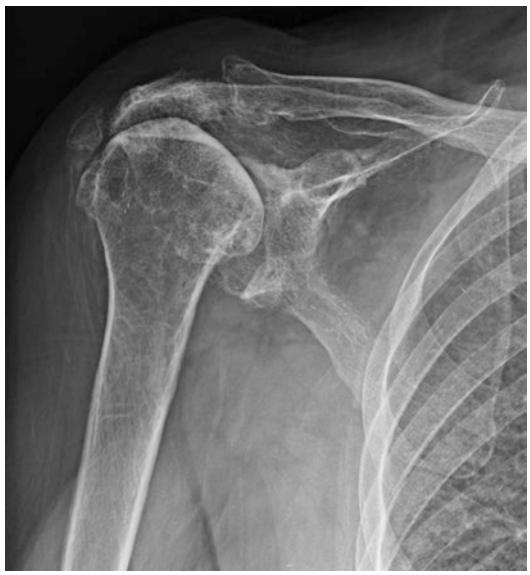


Fig. 34.1 Anteroposterior x-ray view of a right shoulder with some pathognomonic radiographic signs of cuff tear arthropathy (CTA)

- Stage 1, the acromiohumeral interval is >6 mm
- Stage 2, the acromiohumeral interval is <5 mm
- Stage 3, the acromiohumeral interval is <5 mm and acetabularization of the coracoacromial arch is present
- Stage 4, the glenohumeral joint is narrowed, either without acetabularization (stage 4a) or with acetabularization (stage 4b)
- Stage 5, humeral head osteonecrosis results in collapse

The Seebauer classification [4] is quite more complicated and therefore less widespread in clinical practice. It is a biomechanical description of CTA, in which each type is distinguished according to the amount of upward migration of the humeral head from the center of rotation and the amount of instability. The amount of decentralization seen on radiographs is dependent on "the extent of the rotator cuff tear, the integrity of the coracoacromial arch, and the degree and direction of the glenoid bone erosion" [4].

Plain radiographs have also been employed as a tool for preoperative planning. Several classifications have been proposed to assess glenoid wear [12–15]. As a matter of fact, it is important to highlight that bone loss is always multiplanar; therefore assessing glenoid wear means a comprehensive evaluation of glenoid version [12], inclination [13, 14], and medialization [15]. Glenoid version is usually evaluated on axillary view, whereas inclination can be evaluated on a true AP view and medialization has been classified on AP and axial views.

Nyffeler et al. [12], after comparing measurement of glenoid version on x-rays and CT scans, actually showed that glenoid retroversion can be overestimated on x-rays in up to 86% of cases; therefore up to now CT scan is the modality of choice for the estimate of glenoid version.

On the contrary, radiographic classification systems for glenoid inclination and medialization are still valid.

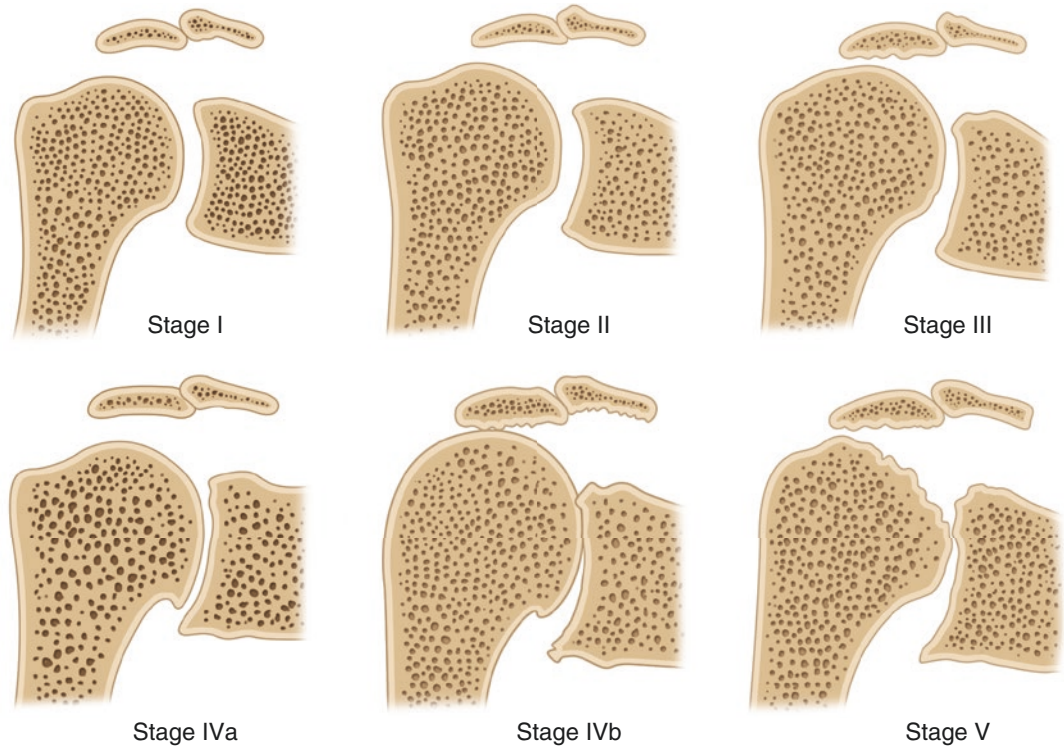


Fig. 34.2 Radiographic classification of CTA according to Hamada [2]

Anatomically, the angle of inclination of the glenoid is equivalent to the amount of glenoid tilt in the coronal plane and defines the position of the humeral head relative to the subacromial space. The normal glenoid tilt in the coronal plane has been reported to range from −8° to 15.8° (average, 4° to 5°) [16]. Two classification systems are available [13, 14].

Sirveaux et al. [14] defined four types of glenoid in order to describe the progression of superior erosion:

- Type E0, the head of the humerus migrated upward without erosion of the glenoid
- Type E1, concentric erosion of the glenoid
- Type E2, erosion of the superior part of the glenoid
- Type E3, erosion extended to the inferior part of the glenoid

Conversely, Habermeyer et al. [13] depicted the evolution of eccentric inferior glenoid wear. The glenoid inclination angle was measured with use of one line drawn along the superior and inferior glenoid rim (the glenoid line) and another line drawn along the lateral base of the coracoid process (the coracoid base line) from the superior glenoid rim perpendicular to the bottom margin of the radiograph. Four types of glenoid were identified:

- Type 0, normal glenoid, in which the coracoid base line and the glenoid line run parallel
- Type 1, the coracoid base line and the glenoid line intersect below the inferior glenoid rim
- Type 2, the coracoid base line and the glenoid line intersect between the inferior glenoid rim and the center of the glenoid
- Type 3, the coracoid base line and the glenoid line intersect above the coracoid base

Very high interobserver reliability was found by the authors [13].

Classification of glenoid medialization has been recently described by Kocsis et al. [15] on AP and axial views. Two anatomical reference

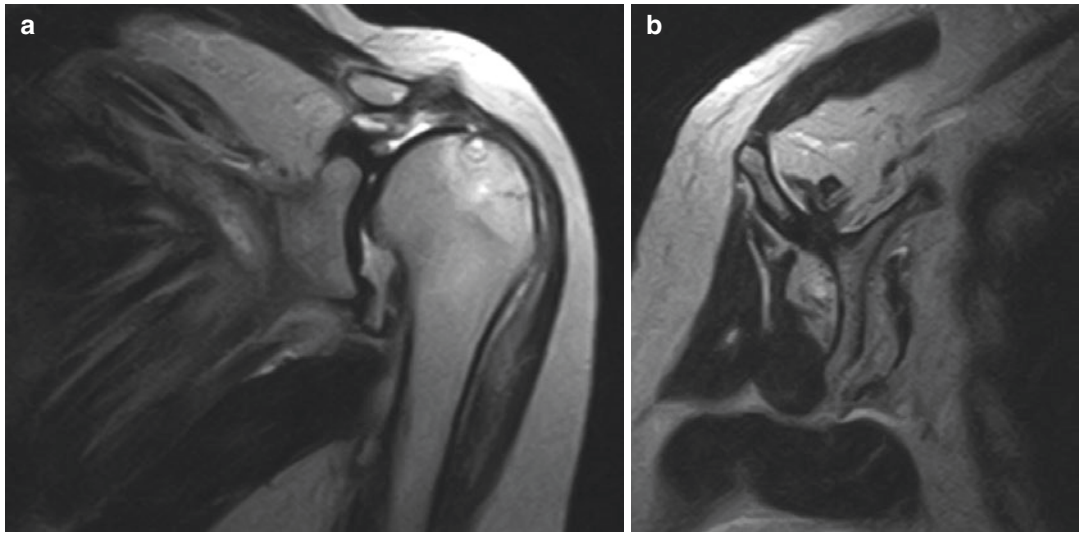


Fig. 34.3 MR is useful for assessing the extension of the rotator cuff tear (**a**) and, even more, muscle atrophy and fatty infiltration (**b**)

points were used to define limits of three zones: the most medial point of the spinoglenoid notch and the most lateral edge of the base of the coracoid. Three types have been recognized:

- Type 1: the most medial (or lowest) point of the intact glenoid articular surface is at the level of or lateral to the base of the coracoid (zone 1)
- Type 2: the most medial (or lowest) point of the intact glenoid articular surface falls between the base of the coracoid and the most medial point of the spinoglenoid notch (zone 2)
- Type 3: the most medial (or lowest) point of the glenoid articular surface reaches the level of the spinoglenoid notch or is medial to it (zone 3)

Excellent inter-method reliability, interobserver, and test-retest reliability were reported by the authors [15].

34.4.2 Magnetic Resonance

Although not essential for diagnosis, MR is useful for assessing the extension of the rotator cuff tear and, even more, muscle atrophy and fatty infiltration (Fig. 34.3). Recent studies showed that degree of rotator cuff muscle fatty infiltration is associated with glenoid type [17]. Moreover, Donohue et al. [18] showed that high-grade fatty infiltration of rotator cuff muscle is associated to increased pathologic glenoid retroversion and increased joint-line medialization.

34.4.3 Computed Tomography

CT scan evaluation is paramount for the preoperative planning. It provides accurate visualization and quantification of glenoid bone stock as well as detecting competence of the coracoacromial arch and/or eventual presence of an acromial stress fracture.

As already mentioned, CT scan is up to now considered the gold standard for definition of glenoid version. Unfortunately, assumptions about how much of the measured glenoid version is physiologic and how much is pathologic in any one patient are quite complicated due to the fact that native glenoid version has been reported to vary over a 25° range from −14° (retroversion) to +12° (anteversion) [16, 19].

Walch et al. [20] first developed a classification system to describe glenoid version in cases

of primary glenohumeral osteoarthritis by using two-dimensional (2D) CT scan. It includes five categories of glenoid patterns:

- A1, centered humeral head, minor erosion
- A2, centered humeral head, major central glenoid erosion
- B1, posterior subluxated head, no bony erosion
- B2 posterior subluxated head, posterior erosion with biconcavity of the glenoid
- C, dysplastic glenoid with at least 25° of retroversion regardless of erosion

Recently, the original Walch's classification system was modified by adding new glenoid subtypes [21, 22]. Bercik et al. [21] added the following subtypes (Fig. 34.4):

- B3, monoconcave glenoid and posteriorly worn, with at least 15° of retroversion or at least 70% posterior humeral head subluxation, or both
- D, glenoid with any level of anteversion or with humeral head subluxation of less than 40% (i.e., anterior subluxation)
- A more precise definition of the A2 glenoid: "cupula" describes a glenoid in which a line drawn from the anterior to posterior rims of the native glenoid transects the humeral head

Intra- and interobserver reliability were also successfully proved [21].

Davis et al. [22] described the C2 glenoid: a glenoid with greater than 25% of retroversion in addition to posterior subluxation of the humeral head with respect to the glenoid face (Fig. 34.5).

In both studies glenoid was evaluated by using three-dimensional (3D) CT scan reconstructions. It has been proven that 3D CT reconstructions portray glenoid version more reliably than 2D CT because 3D reconstructions allow reorientation of the scapula as a free body [19, 23–26] (Fig. 34.6).

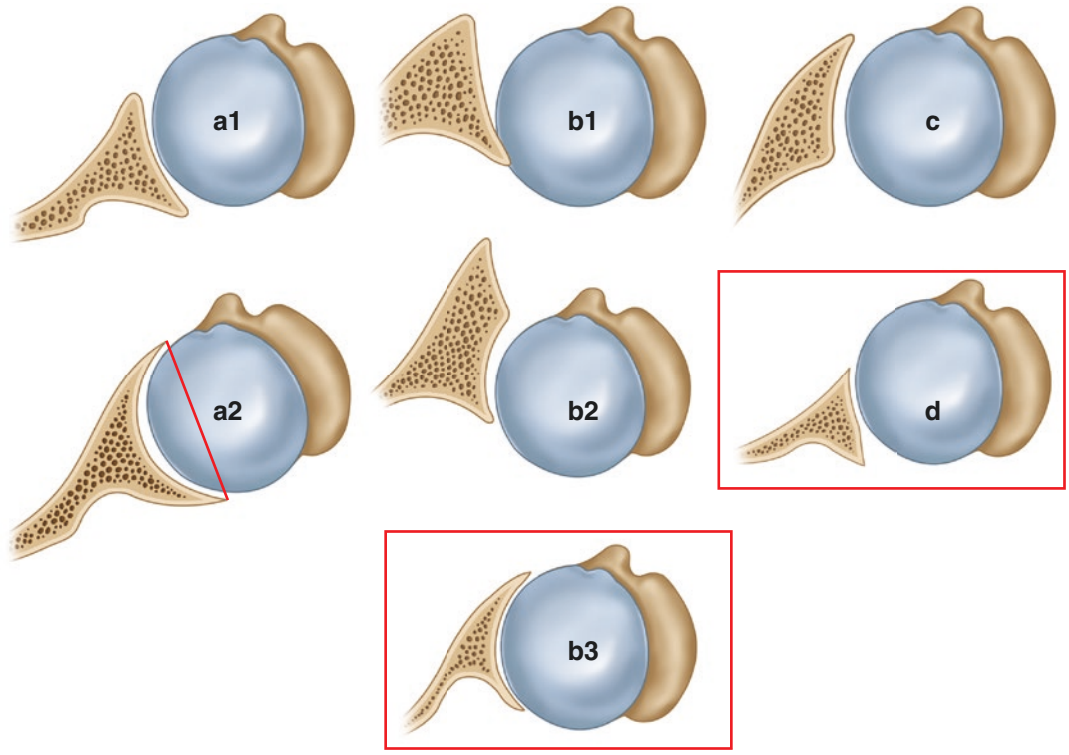


Fig. 34.4 Classification of glenoid version according to Walch et al. [20] modified by Bercik et al. [21]

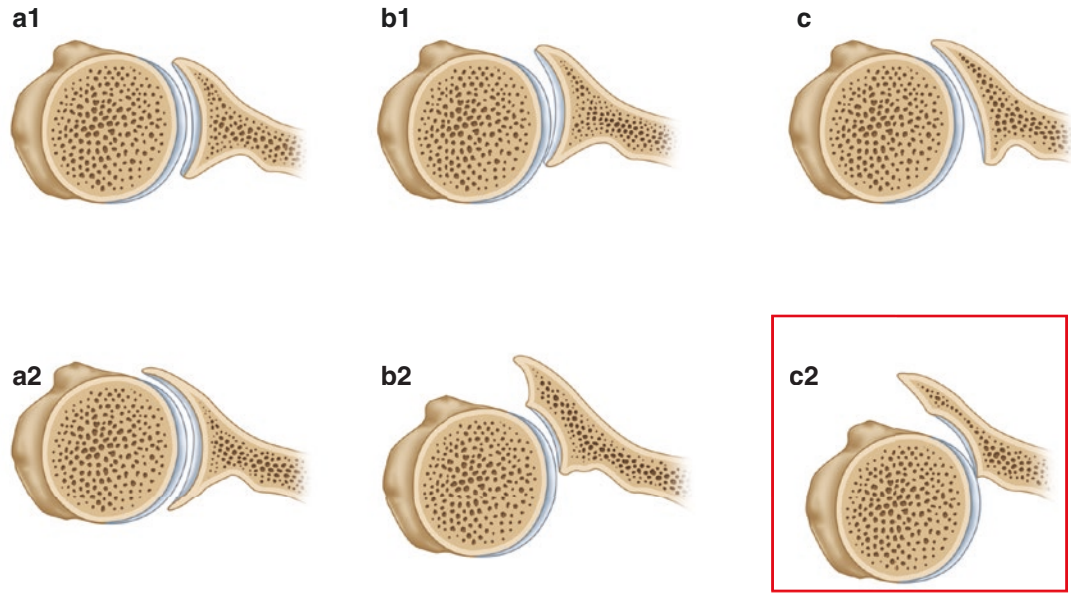


Fig. 34.5 Classification of glenoid version according to Walch et al. [20] modified by Davis et al. [22]

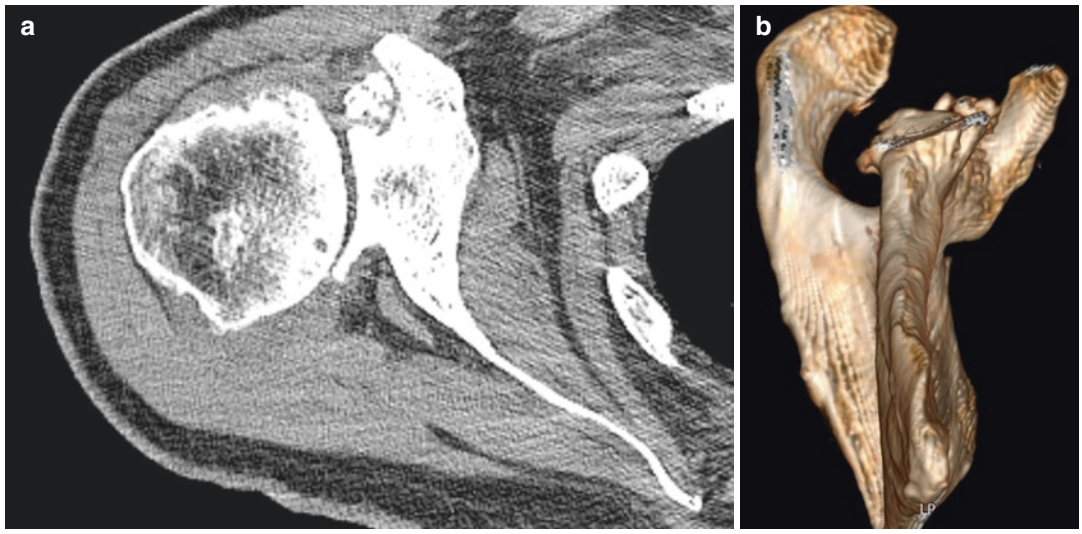


Fig. 34.6 2D CT reconstructions (**a**) are less reliable than 3D CT reconstructions (**b**) in estimating the true glenoid version

Advancement in 3D CT reconstruction software and awareness of the wide range of anatomic variations in glenoid version led to define a new 3D glenoid vault model [27]. The internal architecture of the glenoid vault was found to have a reproducible triangular morphology, defined by the endosteal surfaces of the vault. This technique has been first applied to the contralateral, normal glenoid as a template for initial model orientation [27], but subsequently it has been shown that when placed in the best-fit position, the vault model could be used to estimate the physiologic glenoid version in an individual with severe glenoid disease, independent of knowledge of the contralateral glenoid version [28, 29]. Besides the glenoid vault model, several

commercial software able to quantify volume, severity, and morphology of glenoid bone loss, with or without the assistance of patient-specific instrumentation (PSI), have been recently developed in order to improve surgeon's ability to place the glenoid implant in the desired location or to understand preoperatively when a standard implant cannot be used [30–33].

34.5 Addressing Glenoid Wear in CTA

Managing severe glenoid bone loss in CTA poses a unique surgical challenge. Historically, these patients were treated with hemiarthroplasty avoiding glenoid implantation. However, clinical studies showed uncertain pain relief and poor functional outcomes [34, 35]. Therefore, up to now, RSA is the best and only treatment option in stage IVb and V CTA according to Hamada's classification [3]. Moreover, a recent systematic review analyzed the indications to RSA across the world, questioning that, although the number of RSA performed has steadily increased over the past 20 years, there is a lack information about the similarities and differences in surgical indications in various parts of the world [36]. The authors found out that CTA is the most common indication to RSA across North America, Europe, Asia, and Australia accounting for 52% of all RSA implanted [36]. Shoulder arthroplasty is one of the fastest growing fields in orthopedic surgery. The goal of glenoid implantation is to correct the glenoid version and use the glenoid vault anatomy to maximize fixation and minimize medialization [29]. Based on size and morphology of glenoid wear, different strategies have been developed.

34.5.1 Asymmetric Reaming

Eccentric reaming prior to glenoid component insertion is a common technique used to improve excessive glenoid retroversion. From a technical standpoint it is quite easy to perform, requiring only attention to the direction of the reamer in order to avoid worsening of the defect. Cannulated reaming systems allow placement of a guide pin to assess planned version correction before reaming.

Indeed, it has been shown that aggressive reaming can reduce the subchondral bone available for implant support, medialize the joint line, and allow cortical perforation of the polyethylene implant [37]. Studies that have attempted to define the limits of eccentric reaming in order to minimize the removal of subchondral bone while maximizing version correction showed that correction of 10° resulted in a significant decrease in anteroposterior glenoid diameter and correction of 15° of retroversion led to either implant peg penetration or inadequate bone support, which means high risk of implant loosening [38, 39]. Although biomechanical studies showed no micromotion when at least 50% of the baseplate is supported by glenoid bone [40, 41], based on clinical studies it is safer to limit eccentric reaming to mild defects with no more than 10°–15° of glenoid retroversion [42].

34.5.2 Bone Grafting

Bone grafting provides a biologic solution in cases of severe bone loss that do not guarantee secure seating of a glenoid component and that are not amenable to adequate correction of glenoid version by standard methods, such as asymmetric reaming or small changes in glenoid or humeral component version.

Indications for bone grafting, based on the previously described radiological features, can be summarized as follows:

- >15° of retroversion (B2-B3-C-C2 glenoid) [21, 22]
- Superior tilt (E3 glenoid) [14]
- Excessive medialization (Type 2-3) [15]
- Loss of depth: 10–15 mm (axial CT) [33]

Basing treatment on bone loss classifications allows meaningful evaluation of surgical options [43].

Theoretically, advantages of bone grafting in the setting of glenoid wear include: preservation

of available glenoid bone stock, maintenance of a quite normal joint line that avoids altered joint kinematics secondary to shortening of the glenoid vault, and a permanent restorative solution by biological osseous integration. On the other hand, concerns have also been raised, due to the risk of nonunion, resorption, fixation failure, or subsidence [42, 44]. Moreover, differently from an eccentric reaming, bone grafting is a technically demanding procedure.

Multiple graft sources have been proposed, including humeral head autograft [45, 46], iliac crest autograft [43, 47], cancellous autograft [48, 49], cancellous allograft [50], femoral neck allograft [48], and femoral head allograft [51, 52].

In 2011, Boileau et al. [45] popularized a standardized technique, which required a specific instrumentation for graft harvesting, preparation, and implantation, called "bony increased offset reverse shoulder arthroplasty" (BIO-RSA; Wright Med Group, Memphis, TN, USA). Recently, the BIO-RSA technique has been updated by introducing the angled BIO-RSA, an asymmetric BIO-RSA which adds more flexibility in managing multiplanar defects by using a trapezoidal bone graft in order to correct not only version and medialization but also the superior tilt [53], based on the assumption that uncorrected superior glenoid erosion (E2, E3 glenoid) [14] can lead to superior tilt of the baseplate which can result in increased scapular impingement, instability, inferior scapular notching, and medial polyethylene wear [54, 55]. At the same time, several companies designed their own instrumentation for symmetrical and asymmetrical bone grafting (Fig. 34.7).

Bateman et al. [48], in order to maximize integration and stability, also proposed a hybrid graft glenoid reconstruction by using a peripherally seated cortical femoral neck allograft acting as a sleeve bushing to provide a stable ring under compression in which to house impacted cancellous autograft centrally for early incorporation and ingrowth.

Applying the principles of BIO-RSA (symmetric and asymmetric), it is authors' preference to use distal tibial allograft as a bone graft source, when the autologous humeral head is not available (e.g., osteoporosis, humeral head collapse, revision cases) (Fig. 34.8). Distal tibial allograft has been recently introduced as a viable treatment option for glenoid bone loss in anterior and posterior shoulder instability [56, 57]. Main advantages

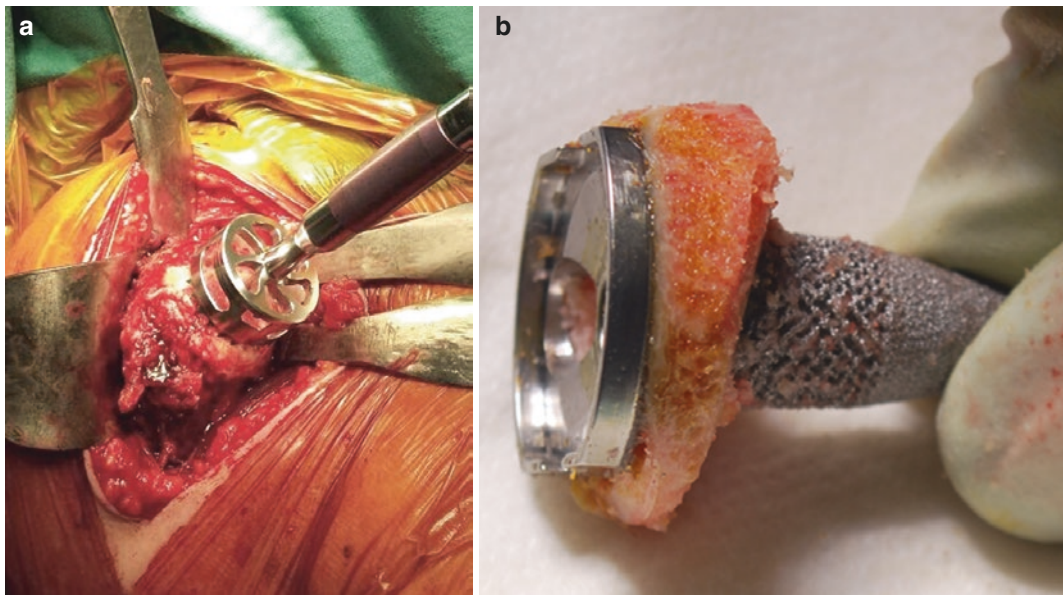


Fig. 34.7 Instrumentation for bone grafting from the humeral head (**a**). Asymmetrical bone graft (**b**)

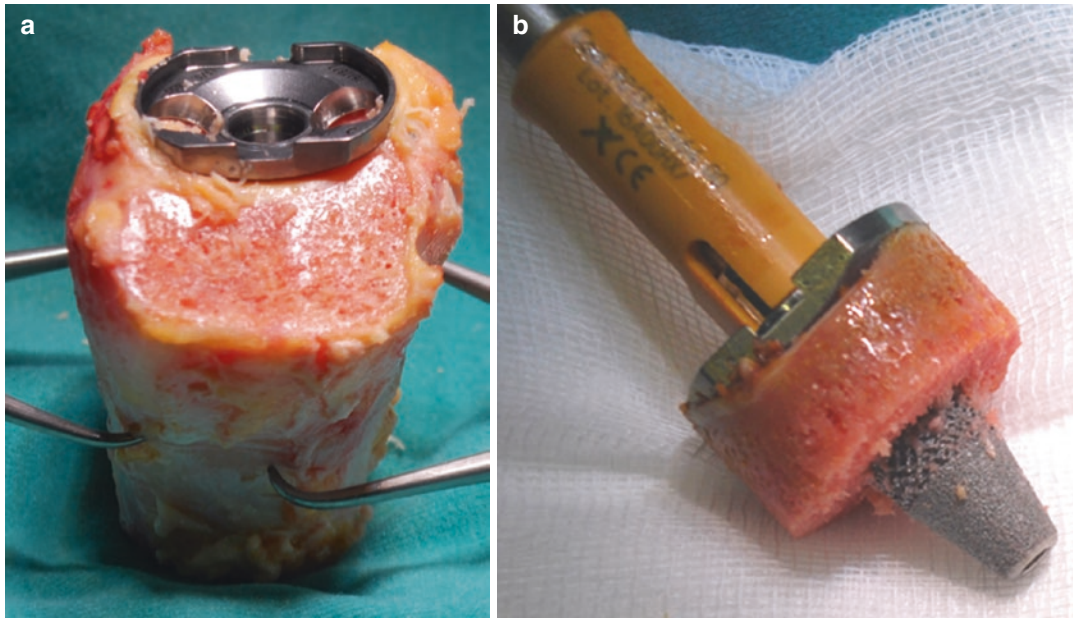


Fig. 34.8 Distal tibia allograft for treating large glenoid bone defects (**a**, **b**)

over other bone grafts are mainly related to the radius of curvature of the lateral aspect of the distal tibia, which resembles that of the native glenoid, thus providing a more anatomical reconstruction. Besides, the graft contains a cartilaginous layer, so the subchondral bone is thick and dense and acts as adequate support for baseplate fixation [58].

However, very large glenoid defects still present a great challenge. Although rare in primary cases, even when a meticulous preoperative planning has been carried out, intraoperative findings can somehow leave the surgeon some unpleasant surprises and make him/her wondering if a two-stage procedure could be worth it. Therefore, a further classification system guiding intraoperative decision-making for the management of the glenoid defects, regardless its primary etiology, has been proposed [59]. The authors aimed to provide a sort of "golden rules" for a successful single-stage procedure which attempts to get: restoration of the 3D glenoid anatomy, stable reconstruction of the bone defect, and secure fixation of the glenoid baseplate. Based on Antuna's classification [60], the defects were broadly classified as centric and eccentric. Centric contained defects were further subclassified as follows:

- C1: Shallow (depth <50% of AP glenoid diameter)
- C2: Deep (depth >50% of AP glenoid diameter + stable vault)
- C3: Cavitary (C2 + unstable vault)
- C4: Destructive (significant destruction of the glenoid and vault)

Eccentric uncontained defects were subclassified, based on size and location. Size was assessed as follows:

- E1: small or shallow
- E2: medium (<30% of the glenoid bone stock)
- E3: large (30–60% of the glenoid bone stock)
- E4: massive (>60% of the glenoid bone stock)

Location was broadly defined as: anterior (A), posterior (P), inferior (I), superior (S), and further subcategorized based on the direction in which the defect extended (e.g., anterior-inferior, Ai).

The authors suggested a single-stage implantation only when the so-called 50% rule could be met: (1) a minimum of 30–50% of the baseplate or the baseplate bone graft composite which should be resting on the native glenoid vault, (2) 50% of

central peg in native scapula, and (3) minimum of two opposite locking screws in native scapula. Therefore, when C3 and C4 as well as E3 and E4 defects are encountered, a two-stage procedure should be considered as it is more stable and safer.

A recent systematic review summarized the results of glenoid bone grafting in RSA [61]. Eleven studies were included. The pooled union rate of glenoid bone graft was 95%, but it reached 97% when an autograft bone was preferred and 100% if a concentric defect was addressed. On the other hand, eccentric grafts also showed high, but lower union rates compared to concentric defects. They showed an overall union rate of 92%, which reached 94% when using autograft bone. The overall pooled mean complication rate was 18%. Of these, 8% were considered major complications, such as glenoid component failure, migration, and loosening, whereas 10% were considered minor complications, mainly related to imaging findings, such as: heterotopic ossifications or proximal humeral bone resorption without loosening.

However, although pooled results showed by Paul et al. [61] seem encouraging, it must be taken into account that included studies were mainly retrospective cohort studies; therefore optimal graft source and technique for placement and stabilization remain controversial, since different grafting techniques, implants, and uncontrolled confounding patient-related variables were pooled all together.

34.5.3 Augmented Baseplate

New prosthetic solutions to glenoid bone loss have been proposed to overcome concerns raised about previously described options. However, similarly to bone grafting, augmented glenoid baseplate implantation is a technically demanding procedure that requires precise creation of a glenoid bone bed to seat the augmented component in order to avoid micromotion and risk of loosening [42].

Literature is still lacking on this topic, even if encouraging results in very small case series have been reported [62–65]. Different designs with various degrees of version and thickness have been described, such as: wedged glenoid, usable with or without bone grafting, which allows multiplanar correction of glenoid wear [65], or a customized porous tantalum augment in order to improve lateralization [62].

Finite element studies comparing bone grafting versus augmented baseplate implantation showed that bony lateralization increases stress and displacement to a greater degree than prosthetic lateralization [66, 67]. Particularly, Denard et al. [66] showed that bony lateralization is not advisable if more than 5 mm is required. Clinical studies are needed.

34.5.4 Custom-Made Implants

Custom-made implants should be considered a salvage option in CTA or in revision after failed RSA with severe bone loss.

First examples were CAD/CAM (computer-assisted design/computer-assisted manufacture) shoulder replacement resembling a total hip prosthesis [68–70]. Subsequently, more suitable designs, helped by PSI technology, have been proposed to treat massive glenoid defects [71] (Fig. 34.9).

However, further studies are needed before drawing any conclusion on actual results.

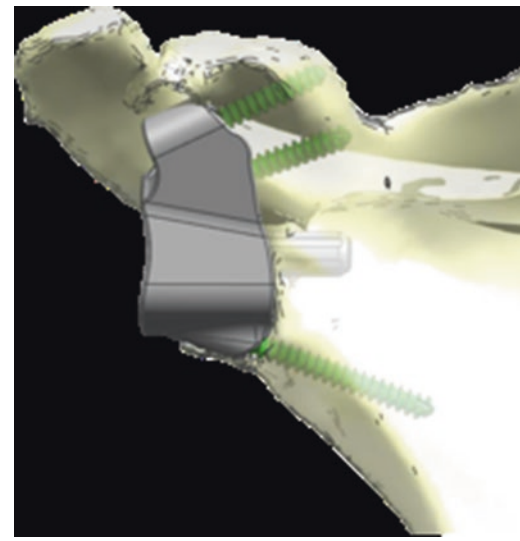


Fig. 34.9 Custom-made implants should be considered a salvage option in CTA or in revision after failed RSA with severe bone loss

References

1. Neer CS, Craig EV, Fukuda H. Cuff-tear arthropathy. J Bone Joint Surg Am. 1983;65:1232–44.
2. Jensen KL, Williams GR, Russell IJ, Rockwood CA. Rotator cuff tear arthropathy. J Bone Joint Surg Am. 1999;81:1312–24.
3. Hamada K, Fukuda H, Mikasa M, Kobayashi Y. Roentgenographic findings in massive rotator cuff tears. A long-term observation. Clin Orthop. 1990;(254):92–6.
4. Visotsky JL, Basamania C, Seebauer L, et al. Cuff tear arthropathy: pathogenesis, classification, and algorithm for treatment. J Bone Joint Surg Am. 2004;86-A(Suppl 2):35–40.
5. Rugg CM, Gallo RA, Craig EV, Feeley BT. The pathogenesis and management of cuff tear arthropathy. J Shoulder Elb Surg. 2018;27:2271–83. https://doi.org/10.1016/j.jse.2018.07.020.
6. Aumiller WD, Kleuser TM. Diagnosis and treatment of cuff tear arthropathy. JAAPA. 2015;28:33–8. https://doi.org/10.1097/01.JAA.0000469435.44701.ce.
7. Gumina S, Castagna A, Candela V, et al. Aetiopathogenesis of cuff-tear arthropathy: could juvenile joint laxity be considered a predisposing factor? Int Orthop. 2018;42:1113–7. https://doi.org/10.1007/s00264-017-3718-5.
8. Halverson PB, Cheung HS, McCarty DJ, et al. "Milwaukee shoulder"—association of microspheroids containing hydroxyapatite crystals, active collagenase, and neutral protease with rotator cuff defects. II. Synovial fluid studies. Arthritis Rheum. 1981;24:474–83.
9. McCarty DJ. Milwaukee shoulder syndrome. Trans Am Clin Climatol Assoc. 1991;102:271–83; discussion 283–4.
10. Burkhart SS, Nottage WM, Ogilvie-Harris DJ, et al. Partial repair of irreparable rotator cuff tears. Arthroscopy. 1994;10:363–70.
11. Nam D, Maak TG, Raphael BS, et al. Rotator cuff tear arthropathy: evaluation, diagnosis, and treatment: AAOS exhibit selection. J Bone Joint Surg Am. 2012;94:e34. https://doi.org/10.2106/JBJS.K.00746.
12. Nyffeler RW, Jost B, Pfirrmann CWA, Gerber C. Measurement of glenoid version: conventional radiographs versus computed tomography scans. J Shoulder Elb Surg. 2003;12:493–6. https://doi.org/10.1016/S1058274603001812.
13. Habermeyer P, Magosch P, Luz V, Lichtenberg S. Three-dimensional glenoid deformity in patients with osteoarthritis: a radiographic analysis. J Bone Joint Surg Am. 2006;88:1301–7. https://doi.org/10.2106/JBJS.E.00622.
14. Sirveaux F, Favard L, Oudet D, et al. Grammont inverted total shoulder arthroplasty in the treatment of glenohumeral osteoarthritis with massive rupture of the cuff. Results of a multicentre study of 80 shoulders. J Bone Joint Surg Br. 2004;86:388–95.
15. Kocsis G, Thyagarajan DS, Fairbairn KJ, Wallace WA. A new classification of glenoid bone loss to help plan the implantation of a glenoid component before revision arthroplasty of the shoulder. Bone Joint J. 2016;98-B:374–80. https://doi.org/10.1302/0301-620X.98B3.36664.
16. Churchill RS, Brems JJ, Kotschi H. Glenoid size, inclination, and version: an anatomic study. J Shoulder Elb Surg. 2001;10:327–32. https://doi.org/10.1067/mse.2001.115269.
17. Walker KE, Simcock XC, Jun BJ, et al. Progression of glenoid morphology in glenohumeral osteoarthritis. J Bone Joint Surg Am. 2018;100:49–56. https://doi.org/10.2106/JBJS.17.00064.
18. Donohue KW, Ricchetti ET, Ho JC, Iannotti JP. The association between rotator cuff muscle fatty infiltration and glenoid morphology in glenohumeral osteoarthritis. J Bone Joint Surg Am. 2018;100:381–7. https://doi.org/10.2106/JBJS.17.00232.
19. Scalise JJ, Codsi MJ, Bryan J, et al. The influence of three-dimensional computed tomography images of the shoulder in preoperative planning for total shoulder arthroplasty. J Bone Joint Surg Am. 2008;90:2438–45. https://doi.org/10.2106/JBJS.G.01341.
20. Walch G, Badet R, Boulahia A, Khoury A. Morphologic study of the glenoid in primary glenohumeral osteoarthritis. J Arthroplast. 1999;14:756–60.
21. Bercik MJ, Kruse K, Yalizis M, et al. A modification to the Walch classification of the glenoid in primary glenohumeral osteoarthritis using three-dimensional imaging. J Shoulder Elb Surg. 2016;25:1601–6. https://doi.org/10.1016/j.jse.2016.03.010.
22. Davis DE, Acevedo D, Williams A, Williams G. Total shoulder arthroplasty using an inlay miniglenoid component for glenoid deficiency: a 2-year follow-up of 9 shoulders in 7 patients. J Shoulder Elb Surg. 2016;25:1354–61. https://doi.org/10.1016/j.jse.2015.12.010.
23. Budge MD, Lewis GS, Schaefer E, et al. Comparison of standard two-dimensional and three-dimensional corrected glenoid version measurements. J Shoulder Elb Surg. 2011;20:577–83. https://doi.org/10.1016/j.jse.2010.11.003.
24. Kwon YW, Powell KA, Yum JK, et al. Use of three-dimensional computed tomography for the analysis of the glenoid anatomy. J Shoulder Elb Surg. 2005;14:85–90. https://doi.org/10.1016/j.jse.2004.04.011.
25. Daggett M, Werner B, Gauci MO, et al. Comparison of glenoid inclination angle using different clinical imaging modalities. J Shoulder Elb Surg. 2016;25:180–5. https://doi.org/10.1016/j.jse.2015.07.001.
26. Werner BS, Hudek R, Burkhart KJ, Gohlke F. The influence of three-dimensional planning on decision-making in total shoulder arthroplasty. J Shoulder Elb Surg. 2017;26:1477–83. https://doi.org/10.1016/j.jse.2017.01.006.
27. Codsi MJ, Bennetts C, Gordiev K, et al. Normal glenoid vault anatomy and validation of a novel glenoid

implant shape. J Shoulder Elb Surg. 2008;17:471–8. https://doi.org/10.1016/j.jse.2007.08.010.
28. Scalise JJ, Codsi MJ, Bryan J, Iannotti JP. The three-dimensional glenoid vault model can estimate normal glenoid version in osteoarthritis. J Shoulder Elb Surg. 2008;17:487–91. https://doi.org/10.1016/j.jse.2007.09.006.
29. Ganapathi A, McCarron JA, Chen X, Iannotti JP. Predicting normal glenoid version from the pathologic scapula: a comparison of 4 methods in 2- and 3-dimensional models. J Shoulder Elb Surg. 2011;20:234–44. https://doi.org/10.1016/j.jse.2010.05.024.
30. Iannotti JP, Weiner S, Rodriguez E, et al. Three-dimensional imaging and templating improve glenoid implant positioning. J Bone Joint Surg Am. 2015;97:651–8. https://doi.org/10.2106/JBJS.N.00493.
31. Lombardo DJ, Khan J, Prey B, et al. Quantitative assessment and characterization of glenoid bone loss in a spectrum of patients with glenohumeral osteoarthritis. Musculoskelet Surg. 2016;100:179–85. https://doi.org/10.1007/s12306-016-0406-3.
32. Nowak DD, Bahu MJ, Gardner TR, et al. Simulation of surgical glenoid resurfacing using three-dimensional computed tomography of the arthritic glenohumeral joint: the amount of glenoid retroversion that can be corrected. J Shoulder Elb Surg. 2009;18:680–8. https://doi.org/10.1016/j.jse.2009.03.019.
33. Sabesan V, Callanan M, Sharma V, Iannotti JP. Correction of acquired glenoid bone loss in osteoarthritis with a standard versus an augmented glenoid component. J Shoulder Elb Surg. 2014;23:964–73. https://doi.org/10.1016/j.jse.2013.09.019.
34. Lynch JR, Clinton JM, Dewing CB, et al. Treatment of osseous defects associated with anterior shoulder instability. J Shoulder Elb Surg. 2009;18:317–28. https://doi.org/10.1016/j.jse.2008.10.013.
35. Levine WN, Djurasovic M, Glasson JM, et al. Hemiarthroplasty for glenohumeral osteoarthritis: results correlated to degree of glenoid wear. J Shoulder Elb Surg. 1997;6:449–54.
36. Erickson BJ, Bohl DD, Cole BJ, et al. Reverse total shoulder arthroplasty: indications and techniques across the world. Am J Orthop (Belle Mead NJ). 2018;47. https://doi.org/10.12788/ajo.2018.0079.
37. Walch G, Young AA, Boileau P, et al. Patterns of loosening of polyethylene keeled glenoid components after shoulder arthroplasty for primary osteoarthritis: results of a multicenter study with more than five years of follow-up. J Bone Joint Surg Am. 2012;94:145–50. https://doi.org/10.2106/JBJS.J.00699.
38. Clavert P, Millett PJ, Warner JJP. Glenoid resurfacing: what are the limits to asymmetric reaming for posterior erosion? J Shoulder Elb Surg. 2007;16:843–8. https://doi.org/10.1016/j.jse.2007.03.015.
39. Gillespie R, Lyons R, Lazarus M. Eccentric reaming in total shoulder arthroplasty: a cadaveric study. Orthopedics. 2009;32:21.
40. Formaini NT, Everding NG, Levy JC, et al. The effect of glenoid bone loss on reverse shoulder arthroplasty baseplate fixation. J Shoulder Elb Surg. 2015;24:e312–9. https://doi.org/10.1016/j.jse.2015.05.045.
41. Martin EJ, Duquin TR, Ehrensberger MT. Reverse total shoulder glenoid baseplate stability with superior glenoid bone loss. J Shoulder Elb Surg. 2017;26:1748–55. https://doi.org/10.1016/j.jse.2017.04.020.
42. Stephens SP, Paisley KC, Jeng J, et al. Shoulder arthroplasty in the presence of posterior glenoid bone loss. J Bone Joint Surg Am. 2015;97:251–9. https://doi.org/10.2106/JBJS.N.00566.
43. Norris TR. Glenoid bone loss in reverse shoulder arthroplasty treated with bone graft techniques. Am J Orthop (Belle Mead NJ). 2018;47. https://doi.org/10.12788/ajo.2018.0016.
44. Sears BW, Johnston PS, Ramsey ML, Williams GR. Glenoid bone loss in primary total shoulder arthroplasty: evaluation and management. J Am Acad Orthop Surg. 2012;20:604–13. https://doi.org/10.5435/JAAOS-20-09-604.
45. Boileau P, Moineau G, Roussanne Y, O'Shea K. Bony increased-offset reversed shoulder arthroplasty: minimizing scapular impingement while maximizing glenoid fixation. Clin Orthop. 2011;469:2558–67. https://doi.org/10.1007/s11999-011-1775-4.
46. Neyton L, Boileau P, Nové-Josserand L, et al. Glenoid bone grafting with a reverse design prosthesis. J Shoulder Elb Surg. 2007;16:S71–8. https://doi.org/10.1016/j.jse.2006.02.002.
47. Hill JM, Norris TR. Long-term results of total shoulder arthroplasty following bone-grafting of the glenoid. J Bone Joint Surg Am. 2001;83-A:877–83.
48. Bateman E, Donald SM. Reconstruction of massive uncontained glenoid defects using a combined autograft-allograft construct with reverse shoulder arthroplasty: preliminary results. J Shoulder Elb Surg. 2012;21:925–34. https://doi.org/10.1016/j.jse.2011.07.009.
49. Melis B, Bonnevialle N, Neyton L, et al. Glenoid loosening and failure in anatomical total shoulder arthroplasty: is revision with a reverse shoulder arthroplasty a reliable option? J Shoulder Elb Surg. 2012;21:342–9. https://doi.org/10.1016/j.jse.2011.05.021.
50. Elhassan B, Christensen TJ, Wagner ER. Feasibility of latissimus and teres major transfer to reconstruct irreparable subscapularis tendon tear: an anatomic study. J Shoulder Elb Surg. 2014;23:492–9. https://doi.org/10.1016/j.jse.2013.07.046.
51. De Biase CF, Ziveri G, De Caro F, et al. Reverse shoulder arthroplasty using a "L" shaped allograft for glenoid reconstruction in a patient with massive glenoid bone loss: case report. Eur Rev Med Pharmacol Sci. 2014;18:44–9.
52. Klein SM, Dunning P, Mulieri P, et al. Effects of acquired glenoid bone defects on surgical technique and clinical outcomes in reverse shoulder arthroplasty. J Bone Joint Surg Am. 2010;92:1144–54. https://doi.org/10.2106/JBJS.I.00778.

53. Boileau P, Morin-Salvo N, Gauci M-O, et al. Angled BIO-RSA (bony-increased offset-reverse shoulder arthroplasty): a solution for the management of glenoid bone loss and erosion. J Shoulder Elb Surg. 2017;26:2133–42. https://doi.org/10.1016/j.jse.2017.05.024.
54. Laver L, Garrigues GE. Avoiding superior tilt in reverse shoulder arthroplasty: a review of the literature and technical recommendations. J Shoulder Elb Surg. 2014;23:1582–90. https://doi.org/10.1016/j.jse.2014.06.029.
55. Hettrich CM, Permeswaran VN, Goetz JE, Anderson DD. Mechanical tradeoffs associated with glenosphere lateralization in reverse shoulder arthroplasty. J Shoulder Elb Surg. 2015;24:1774–81. https://doi.org/10.1016/j.jse.2015.06.011.
56. Frank RM, Romeo AA, Richardson C, et al. Outcomes of Latarjet versus distal tibia allograft for anterior shoulder instability repair: a matched cohort analysis. Am J Sports Med. 2018;46:1030–8. https://doi.org/10.1177/0363546517744203.
57. Frank RM, Shin J, Saccomanno MF, et al. Comparison of glenohumeral contact pressures and contact areas after posterior glenoid reconstruction with an iliac crest bone graft or distal tibial osteochondral allograft. Am J Sports Med. 2014;42:2574–82. https://doi.org/10.1177/0363546514545860.
58. Rabinowitz J, Friedman R, Eichinger JK. Management of glenoid bone loss with anterior shoulder instability: indications and outcomes. Curr Rev Musculoskelet Med. 2017;10:452–62. https://doi.org/10.1007/s12178-017-9439-y.
59. Gupta A, Thussbas C, Koch M, Seebauer L. Management of glenoid bone defects with reverse shoulder arthroplasty-surgical technique and clinical outcomes. J Shoulder Elb Surg. 2018;27:853–62. https://doi.org/10.1016/j.jse.2017.10.004.
60. Antuna SA, Sperling JW, Cofield RH, Rowland CM. Glenoid revision surgery after total shoulder arthroplasty. J Shoulder Elb Surg. 2001;10:217–24. https://doi.org/10.1067/mse.2001.113961.
61. Paul RA, Maldonado-Rodriguez N, Docter S, et al. Glenoid bone grafting in primary reverse total shoulder arthroplasty: a systematic review. J Shoulder Elb Surg. 2019;28:2447–56. https://doi.org/10.1016/j.jse.2019.05.011.
62. Ivaldo N, Mangano T, Caione G, et al. Customized tantalum-augmented reverse shoulder arthroplasty for glenoid bone defect and excessive medialization: description of the technique. Musculoskelet Surg. 2016;100:13–8. https://doi.org/10.1007/s12306-016-0404-5.
63. Jones RB, Wright TW, Roche CP. Bone grafting the glenoid versus use of augmented glenoid baseplates with reverse shoulder arthroplasty. Bull Hosp Jt Dis. 2015;2013(73 Suppl 1):S129–35.
64. Wright TW, Roche CP, Wright L, et al. Reverse shoulder arthroplasty augments for glenoid wear. Comparison of posterior augments to superior augments. Bull Hosp Jt Dis (2013). 2015;73(Suppl 1):S124–8.
65. Jones RB, Wright TW, Zuckerman JD. Reverse total shoulder arthroplasty with structural bone grafting of large glenoid defects. J Shoulder Elb Surg. 2016;25:1425–32. https://doi.org/10.1016/j.jse.2016.01.016.
66. Denard PJ, Lederman E, Parsons BO, Romeo AA. Finite element analysis of glenoid-sided lateralization in reverse shoulder arthroplasty. J Orthop Res. 2017;35:1548–55. https://doi.org/10.1002/jor.23394.
67. Yang C-C, Lu C-L, Wu C-H, et al. Stress analysis of glenoid component in design of reverse shoulder prosthesis using finite element method. J Shoulder Elb Surg. 2013;22:932–9. https://doi.org/10.1016/j.jse.2012.09.001.
68. Jeske H-C, Wambacher M, Dallapozza C, et al. Functional and clinical outcome of total shoulder arthroplasty with oversized glenoid. Arch Orthop Trauma Surg. 2012;132:927–36. https://doi.org/10.1007/s00402-012-1496-5.
69. Uri O, Bayley I, Lambert S. Hip-inspired implant for revision of failed reverse shoulder arthroplasty with severe glenoid bone loss. Improved clinical outcome in 11 patients at 3-year follow-up. Acta Orthop. 2014;85:171–6. https://doi.org/10.3109/17453674.2014.899850.
70. Chammaa R, Uri O, Lambert S. Primary shoulder arthroplasty using a custom-made hip-inspired implant for the treatment of advanced glenohumeral arthritis in the presence of severe glenoid bone loss. J Shoulder Elb Surg. 2017;26:101–7. https://doi.org/10.1016/j.jse.2016.05.027.
71. Dines DM, Gulotta L, Craig EV, Dines JS. Novel solution for massive glenoid defects in shoulder arthroplasty: a patient-specific glenoid vault reconstruction system. Am J Orthop (Belle Mead NJ). 2017;46:104–8.

35 Workup and Management of Infection in Shoulder Arthroplasty

Rui Claro, Luis Barros, Nuno Sampaio Gomes, and Ricardo Sousa

35.1 Introduction

Periprosthetic joint infection (PJI) of the shoulder joint is a rare but a major complication of shoulder arthroplasties. The mean incidence has been reported to be 1.1%; after reverse arthroplasty it can be 3.8% and can reach to 10% in the subgroup of male, young patients operated on with a reverse prosthesis [1–4]. PJI is also the most common reason for revisions of shoulder prosthesis made necessary by pain, stiffness, or loosening [5].

Risk factors associated with periprosthetic shoulder infections are posttraumatic osteoarthritis, previous surgery, repeated cortisone injections, systemic corticosteroid treatment and other immunosuppressive medicaments, rheumatoid arthritis, and diabetes mellitus [1, 6, 7]. Richards et al. [5] studied 4258 patients with shoulder prostheses and found that males were 2.59 times more at risk for infection than females and that reverse total shoulder arthroplasty was associated with a 6.11 higher risk of infection than anatomical shoulder arthroplasty. Trauma-associated prostheses were associated with a 2.98 greater risk of infection.

The microorganisms most commonly associated with shoulder PJI belong to the normal skin flora such as *Staphylococci* and *Cutibacterium acnes* (formerly known as *Propionibacterium acnes*). Recent studies have shown that the *Cutibacterium acnes* is associated with between 31 and 70% of all periprosthetic shoulder infections and causes many more periprosthetic infections in the shoulder than in other joints, probably because of the proximity of the surgical site to the axillary region [5, 6, 8, 9].

Most patients with infected shoulder prosthesis refer pain and/or limited range of motion. However, clear signs and symptoms of infection are not always present especially in chronic cases. Therefore each painful shoulder joint should be considered as potentially infected, especially in early failures or humeral stem loosening [10].

However there is still open debate on what the exact diagnostic strategy or specific cutoff(s)

R. Claro (✉)
Department of Orthopaedics, Centro Hospitalar Universitário do Porto, Porto, Portugal

Shoulder Unit, Orthopedic Department, Centro Hospitalar Universitário do Porto, Porto, Portugal

L. Barros
Department of Orthopaedics, Centro Hospitalar Universitário do Porto, Porto, Portugal

N. Sampaio Gomes
Department of Orthopaedics, Hospital Forças Armadas, Porto, Portugal

Hospital da Luz Arrábida, Porto, Portugal

R. Sousa
Department of Orthopaedics, Centro Hospitalar Universitário do Porto, Porto, Portugal

GRIP (Porto Bone and Joint Infection Unit), Centro Hospitalar Universitário do Porto, Porto, Portugal

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_35

for diagnostic test interpretation in the shoulder should be. Once a diagnosis has been reached, the best chance for a favorable outcome is to follow a rationale and evidence-based treatment approach. Although many of the surgical and medical treatment principles can be extrapolated from what has been learned on the more common hip and knee PJI, specific aspects for the treatment of shoulder PJI are still lacking robust evidence.

35.2 Diagnosis

Although a number of different definitions of periprosthetic joint infection have been proposed over the past few years [11–14], none of them have been specifically developed for shoulder PJI. What they all have in common is the acknowledgment that diagnosis is not always easy and there is no real diagnostic gold standard. Therefore, final diagnosis must rely on a combination of clinical features, laboratory tests (with a special focus on synovial fluid interpretation), microbiological and histological results, and to a lesser degree radiological investigation, such as conventional radiography or scintigraphy.

The principles involved in the diagnosis of a shoulder PJI do not differ from those used to investigate hip or knee arthroplasties. As such, much of the experience gained from them can be used directly for developing shoulder-specific diagnostic tools.

In the recent 2018 International Consensus Meeting (ICM) on Musculoskeletal Infection a study grouped focused specifically on shoulder PJI diagnosis and proposed a new set of criteria [15]. Meeting one of the following criteria is diagnostic of **definite** periprosthetic shoulder infection:

- A sinus tract communicating with the prosthesis is present
- Gross intra-articular pus
- Two positive cultures with phenotypically identical virulent organisms

Table 35.1 Weighted values for all positive tests performed as part of the diagnostic evaluation of a failed shoulder arthroplasty

Minor criteria	Weight
Unexpected wound drainage	4
Single positive tissue culture (virulent organism)	3
Single positive tissue culture (low-virulence organism)	1
Second positive tissue culture (identical low-virulence organism)	3
Humeral loosening	3
Positive frozen section (five PMN in at least five high-power fields)	3
Positive preoperative aspirate culture (low or high virulence)	3
Elevated synovial neutrophil percentage (>80%)[a]	2
Elevated Synovial WBC (>3000 cells/μL)[a]	2
Elevated ESR (>30 mm/h)[a]	2
Elevated CRP (>10 mg/L)[a]	2
Elevated synovial alpha-defensin	2
Cloudy fluid	2

PMN polymorphonuclear leukocyte, *WBC* white blood cell, *ESR* erythrocyte sedimentation rate, *CRP* C-reactive protein
[a]Beyond 6 weeks from recent surgery

Weighted values for all positive tests performed as part of the diagnostic evaluation of a failed shoulder arthroplasty are summed (Table 35.1):

- Six or greater with identified organism = **probable PJI**
- Six or greater *without* identified organism = **possible PJI**
- Six or less:
  - Single positive culture for virulent organism = **possible PJI**
  - Two positive cultures for low-virulence organism = **possible PJI**
  - Negative cultures or only single positive culture for low virulent organism = **PJI unlikely**

To further test the definition, clinical scenarios were proposed and evaluated with the definition (Table 35.2).

Table 35.2 Clinical scenarios of the ICM diagnostic criteria in practice

#	Scenario	Definition
1	Painful shoulder arthroplasty: • Positive aspirate culture (*C. acnes*): 3 points • 1/5 intraoperative cultures positive (*C. acnes*): 1 point • Humeral loosening: 3 points	Probable PJI
2	Painful shoulder arthroplasty: • No aspirate completed • Persistent unexpected wound drainage: 4 points • 2/5 intraoperative cultures positive (*C. acnes*): 1 + 3 = 4 points	Probable PJI
3	Painful shoulder arthroplasty: • Dry aspirate • 2/5 intraoperative cultures positive (MSSA) • Elevated ESR • Elevated CRP	Definite PJI
4	Painful shoulder arthroplasty: • Well-fixed components • 2/5 intraoperative cultures positive (*C. acnes*): 1 + 3 = 4 points • All other tests negative	Possible PJI
5	Painful shoulder arthroplasty: • Persistent unexpected wound drainage: 4 points • 1/5 intraoperative cultures positive (*C. acnes*): 1 point • All other tests negative	Unlikely PJI
6	Painful shoulder arthroplasty: • Persistent unexpected wound drainage: 4 points • 1/5 intraoperative cultures positive (MSSA): 3 points • All other tests negative	Probable PJI

CRP C-reactive protein, *ESR* erythrocyte sedimentation rate, *MSSA* methicillin-sensitive *S. aureus*

35.2.1 History, Physical Examination, and X-Ray

PJI can manifest itself as a myriad of different clinical scenarios. The mode of presentation relates to the pathogenesis and microbial etiology of the infection. Only by taking into consideration the bacterial biofilm paradigm is one able to understand the full spectrum of PJI presentations.

Patients' symptoms may vary from acute, rapid-onset joint pain along with frank inflammatory signs and wound purulence with or without systemic features of infection in the case of acute infections to chronic pain or discomfort, decreased range of motion with or without sinus formation in chronic infections. Even acute postoperative infection can present as subtle prolonged wound discharge. The presence of a sinus tract is the only clinical sign that can be considered highly specific for shoulder PJI.

Patients should be evaluated with high-quality radiographs, in anteroposterior and axillary lateral projections to rule out different causes of shoulder pain and dysfunctions that can mimic or coexist with periprosthetic shoulder infection. Radiographic findings concerning for shoulder PJI include component loosening or migration, radiolucent lines, and osteolysis. Specifically, humeral loosening or early implant loosening or osteolysis (2–3 years after the operation) should significantly raise the suspicion for shoulder PJI [16].

CT scans are often used in revision shoulder arthroplasty for evaluation of the remaining bone stock, implant position and loosening, glenoid component wear, soft tissue swelling, fluid collection, and rotator cuff tendon and muscle pathology. However, the value of CT scan as a direct diagnostic modality for infection is limited. MRI is of little value in the diagnosis of infection because of metal artifact from implants and is rarely used. Ultrasonography can be used to detect surrounding abscesses or (more commonly) to guide arthrocentesis and synovial fluid gathering for subsequent testing.

Nuclear medicine testing is not a first-line imaging technique. Scintigraphy is not useful in the first postoperative year because of false-positive results due to physiological adaptation processes of the bone to the implant. Although the technique using combined leucocyte and bone marrow scintigraphy has been validated for hip and knee arthroplasty [17], little is known about its diagnostic accuracy for chronic periprosthetic shoulder joint infection.

35.2.2 Laboratory Tests

Serological inflammatory markers are often used as an initial screening test in a variety of suspected implant related or otherwise musculoskeletal infections [18]. Although they are nonspecific they are also inexpensive and can be performed with minimal inconvenience. Peripheral leucocyte count is usually within normal range, as is the neutrophil cell distribution. ESR and CRP should be critically evaluated, since they are often not elevated in case of low-grade microorganism such as *Cutibacterium acnes* infection. The CRP value in the blood as a nonspecific test is below 10 mg/L in many cases of periprosthetic infections. Dodson et al. [19] found CRP values higher than 10 mg/L in only 72% of periprosthetic shoulder infections. IL-6 has been shown to be specific but not sensitive for PJI [20]. Thus, it is necessary to use other diagnostic methods in order to prove or exclude the existence of a periprosthetic infection before a revision arthroplasty is carried out.

As a rule, in case of a high clinical suspect for deep infection, aspiration of glenohumeral joint should be performed. Synovial fluid analysis should include not only gram stain and cultures (including aerobes, anaerobes, fungi, and mycobacteria) but also cell count with differential and a number of different biomarkers currently available [21].

The determination of the cell count in the aspirate is the workhorse of preoperative diagnosis. A number of different cutoffs have been proposed and the 3000 cells/μL have been widely adopted in the past few years [14] but most studies include very limited number of shoulder arthroplasties. Moroder et al. [16] established that a cell count of more than 2000/μL and/or more than 70% of polymorph nuclear leucocytes is indicating a late PJI of the shoulder.

Another simple and inexpensive test is the leucocyte esterase strip test. For diagnosis of PJI of total knee and hip arthroplasties, the sensitivity was between 69 and 81% and the specificity between 93 and 100% [22–25]. However, 17–30% of the test are nonreadable because of blood contamination of the aspirate [22–25] and regarding the shoulder specifically there is not enough available experience to really consider it as a useful tool.

A new very promising biomarker is alpha-defensin which is released by leukocytes following contact with bacteria and acts as autogenic antimicrobial agent. Alpha-defensin has the potential advantage of not being affected by systemic inflammatory diseases or previous antibiotic administration [26]. Sensitivity and specificity of the laboratory immunoassay have been reported to be extremely high. However one should not extrapolate these results directly to the commercially available lateral flow test that has been suggested to have significantly lower sensitivity [27, 28]. Regarding total shoulder arthroplasty specifically, Frangiamore et al. [29] also found results to be less favorable than for THA or TKA (63% sensitivity; 95% specificity). These results advise caution in its use.

35.2.3 Microbiology

Traditional cultures of aspirated joint fluid remain an important feature of preoperative diagnosis and should not be disregarded. Ince et al. [30] reported a sensitivity of 81.2% in the diagnosis of PJI of the shoulder. Nevertheless, it is far from being the ideal diagnostic tool to exclude infection. Many studies have demonstrated the failure of culture of the aspirated fluid to provide accurate diagnosis of PJI, especially low sensitivity [31–34].

Obtaining a causative pathogen is not only indicative of infection but it also may help guide treatment choices. It is useful to obtain an exact differentiation of the pathogen and its resistance pattern so that a systemic antibiotic therapy can be planned preoperatively. This information will also enable the addition of specific antibiotics to the cement used in a one-stage or two-stage revision arthroplasty that are tailored to the pathogen concerned. In this way, local and systemic antibiotic treatments can be devised according to the identity and resistance pattern of the infecting pathogen and so avoid the unnecessary, nonspecific use of broad-spectrum antibiotics with all its

disadvantages. In addition, this will also reduce the development of resistance to the antibiotics.

Given its importance, if no synovial fluid is available, an alternative strategy may involve taking biopsies of periprosthetic tissue. Here, the biopsied material is obtained via arthroscopic access. At least five samples should be taken for bacteriological cultivation and should be added by additional samples for histological examination or frozen sections [9].

It is essential to incubate the synovial fluid and biopsy tissue samples for a sufficiently long period, at least 14 days. This extended incubation time is necessary because the bacteria causing the periprosthetic infection occur at a very low concentration in the biofilm and are often sessile; these properties lead to a very low growth rate. Especially, *Cutibacterium acnes* (in 31–70% of the cases the responsible microorganism for PJI of shoulder arthroplasties) is a very slow-growing bacterium and needs a long incubation period for its detection [35–37]. The synovial tissue can also be analyzed using PCR methods to detect the microorganism. The advantage of PCR is that the result is available after few hours and PCR technique can now detect most antibiotic resistances. A disadvantage is the quite high percentage of false-positive results due to the detection of not only living bacteria [35, 38].

The advantage of biopsy is the possibility of combining the different diagnostic methods of cultivation and histological examination on several tissue samples. Dilisio et al. [39] studied 41 shoulder arthroplasties and found that biopsy is more reliable than aspiration of the synovial fluid and could accurately confirm or rule out the presence of an infection. The biopsy method was associated with a sensitivity of 100%, a specificity of 100%, a positive predictive value of 100%, and a negative predictive value of 100%, whereas the aspiration method was found to have a sensitivity of only 16.7%, a specificity of 100%, a positive predictive value of 100%, and a negative predictive value of 58.3%. Therefore, we suggest synovial biopsy in cases where the other indirect and direct diagnostic methods did not lead to a clear decision on periprosthetic infection and could not identify the microorganism.

35.3 Treatment

Time of onset of clinical signs of infection is extremely relevant as it will greatly influence treatment choice. We believe a simple infection classification scheme as proposed by Segawa et al. is a good crude guide in selecting the most appropriate treatment choice [40] (Table 35.3).

35.3.1 Debridement, Antibiotics, and Implant Retention

In most cases, debridement, antibiotics, and implant retention (DAIR) is the first-line treatment alternative for acute postoperative and hematogenous periprosthetic infections. In addition to a short duration of symptoms, it is imperative that the prosthesis is stable. Correct antibiotic therapy with special emphasis on "antibiofilm" drug(s) is also critical [41].

Surgery should include aggressive debridement of the periprosthetic tissue and radical synovectomy. Mobile part exchange has also

Table 35.3 Treatment strategy concerning PJI presentation

Type	Presentation	Definition	First-line treatment strategy
I	Acute postoperative infection	Acute infection in the first 4 weeks after surgery	Debridement, antibiotics, and **implant retention**
II	Chronic infection	Chronic indolent infection after the fourth week	**Exchange revision surgery** (one- or two-stage)
III	Acute hematogenous infection	Acute onset on a previously well prosthesis	Debridement, antibiotics, and **implant retention**
IV	Positive intraoperative cultures	At least two positive intraoperative cultures	Subsequent **antibiotics**

been shown to greatly improve success rates and should be performed whenever possible. This is then followed by a thorough irrigation (also with antiseptic fluids). Some papers find no significant difference in outcome between acute postoperative and hematogenous infections [42, 43]. The problem in clinical practice is how to be sure that a hematogenous infection is really an acute infection and not an exacerbation of a chronic infection and this may explain why some have found late acute hematogenous infections to be associated with a worse outcome [44].

Bacteria causing these infections are mostly unknown at the time of surgery. Therefore broad-spectrum empirical antibiotic treatment is started after surgical debridement and microbiological sample gathering. Once the microorganism is identified, specific antibiotic therapy can be adapted to the susceptibility of the microorganism. The use of "antibiofilm" drug(s) such as rifampicin for staphylococci [43, 45–52] and ciprofloxacin for Gram-negative bacteria [50, 53–55] should be adopted whenever possible.

There is little or no published information on what the duration of antibiotic therapy should actually be. Despite some conflicting evidence, extending therapy for 3 months seems to be sufficient for the majority of cases [48–51, 55]. Firstly, there is no evidence that a prolonged antibiotic treatment has a positive effect on retention of the prosthesis. Secondly, a prolonged antibiotic therapy is more likely to lead to a masking of the infection and a delay in identifying a treatment failure than to prevent it. Thirdly, the level of resistance to the antibiotic is increased when treatment failure occurs after a prolonged antibiotic administration. There is no literature that supports the maintenance of antibiotic therapy until the inflammation parameters have normalized [56–58].

35.3.2 Exchange Revision Surgery

Chronic PJI cases and cases where DAIR is not indicated are often treated with exchange revision surgery. This can be performed as a one- or two-stage procedure.

Two-stage exchange consists of debridement, resection of infected implants, and usually temporary placement of an antibiotic-impregnated cement spacer before reimplantation of a new prosthesis. It is still the most common method for treating an infected shoulder prosthesis as it offers a good compromise between a reliable infection eradication and a satisfying functional outcome. A general advantage of the two-stage concept is that surgical debridement is carried out twice, whereby the second operation enables the eradication of residual organisms remaining after the initial debridement.

The antibiotic-loaded cement spacer serves a dual purpose of carrying high local doses of antibiotics and reducing soft tissue contractures, arm shortening, and instability. As such, mobilization becomes easier and reimplantation less demanding. However, it is important to consider the risk of recurrent infections and postoperative complications in this challenging patient population. Most studies use gentamicin mixed into the cement provided in the industrially preformed spacer [59]. Some authors use vancomycin and tobramycin as local antibiotics on a regular basis because they have a broader spectrum of activity [60]. Dual antibiotic spacers seem to offer better infection eradication [61]. However, not all bacteria can be successfully treated with these agents (e.g., some Gram-negative organisms) and in some cases custom-made cement spacer mixed with proper antibiotics directed to a specific agent is needed.

After the first surgery antibiotic therapy is customized to the microorganism(s) isolated in microbiology samples and is usually extended for 6 weeks. After infection is deemed to be cured the second stage may be performed. Since the rotator cuff is often insufficient following debridement, reverse shoulder prosthesis is often chosen in the second stage.

The advantage of the one-stage exchange is obvious: only one operation is required eliminating the morbidity associated with the time interval between stages; and the problems associated with the spacer such as fracture, abraded cement particles, or bone resorption can be avoided.

Some conditions must be met for a one-stage exchange to be considered: (1) it is manda-

tory to know the pathogens and their antibiotic susceptibility profile must be favorable; (2) it is mandatory that soft tissues are favorable and allow for correct closure; (3) infection must be fully debridable, without dissecting functional muscles, tendons, or neurovascular structures. Only then can one expect good local infection control with specific antibiotic mixture being added to the bone cement to enable good local antibiotic therapy and good postoperative "antibiofilm" antibiotic therapy. Recent studies using this concept have achieved infection-free survival of between 90 and 100% [1, 30, 62]. Nelson et al. [63] and Cuff et al. [64] did not observe any difference in the level of eradication observed after one-stage and two-stage revisions. Nevertheless, retrospective comparisons should be interpreted with caution as a selection bias to treat more complex cases with a two-stage approach and more straightforward cases with a one-stage protocol is always present.

The functional outcomes of one-stage revisions depend on the integrity of the rotator cuff following debridement and the type of prosthesis used. Klatte et al. [62] showed that the reverse shoulder prosthesis, with a Constant score of 61, was very much better than the bipolar head prosthesis with a Constant score of 56 or a hemiarthroplasty with a Constant score of 43. A study of one-stage revision by Beekman et al. [1] also support for these data with a Constant score of 55.6%. George et al. [65] presented a systematic review of relevant publications and found significantly better clinical outcomes after one-stage revisions (mean Constant score of 51) than after two-stage revisions (mean Constant score of 44). The rates of eradication of infection were similar for all four procedures (86.7% for the resection arthroplasty, 94.7% for the one-stage revision, 90.8% for the two-stage revision, and 95.6% for the permanent spacer). These results support the concept of the one-stage revision if the pathogen has been characterized.

35.3.3 Salvage Procedures

Interestingly, implantation of a spacer after removal of the infected prosthesis often results in a significant clinical improvement and that is why some authors advocate to leave in the implanted spacer. Permanent antibiotic spacer may also be a viable alternative in shoulder PJI, more so than hip or knee arthroplasty, as it has been shown to result in high success rate in infection treatment and acceptable functional outcomes particularly in low-demanding elderly or high-risk patients not eligible for complex revision surgeries [66] (Fig. 35.1).

Simple removal of the infected prosthesis and conversion to a resection arthroplasty resulted in an improved reinfection rate of 30% according to Coste et al. [67] and even of 0% as reported by Romano et al. [68]. However, joint function following resection arthroplasty is considered to be poor.

Suppressive antibiotic therapy is medical treatment alternative that may be considered in very high-risk surgical patients, especially if infection is localized and with no significant

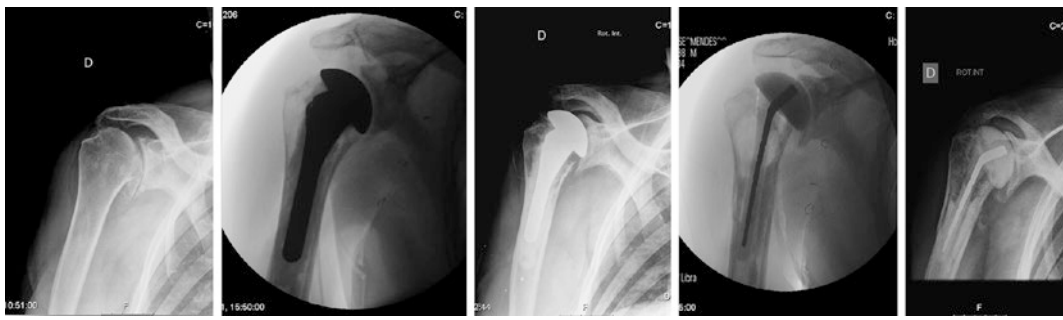


Fig. 35.1 Case of a 56-year-old man. PJI of hemiarthroplasty of right shoulder. Permanent spacer

systemic implications. Although it does not offer a real chance for infection eradication it can improve the patient's quality of life. This option depends on having a susceptible microorganism a patient able to endure chronic antibiotic therapy and should always be appropriately managed by a dedicated infectious diseases expert.

35.4 Conclusion

PJI is an important complication after shoulder arthroplasty and is often associated to high morbidity. It offers a great burden to the patient and a significant technical challenge to the surgeon. Patients with a painful shoulder or limited range of motion should be carefully investigated to rule out a possible infection. While some investigators reported good results with one-stage revisions, more reproducible results have been shown with the two-stage revision. Diagnostic criteria and identification of organisms prior to explant are mandatory to achieve better results.

References

1. Beekman PD, Katusic D, Berghs BM, Karelse A, Wilde L, De Wilde L. One-stage revision for patients with a chronically infected reverse total shoulder replacement. J Bone Joint Surg Br. 2010;92(6):817–22.
2. Singh JA, Sperling J, Schleck C, Harmsen WS, Cofield RH. Periprosthetic infections after total shoulder arthroplasty: a 33-year perspective. J Shoulder Elb Surg. 2012;21(11):1534–41.
3. Hudek R, Gohlke F. [Endoprosthesis infections of the shoulder: diagnosis and therapy algorithm]. Orthopade. 2013;42(7):552–9.
4. Padegimas EM, Maltenfort M, Ramsey ML, Williams GR, Parvizi J, Namdari S. Periprosthetic shoulder infection in the United States: incidence and economic burden. J Shoulder Elb Surg. 2015;24(5):741–6.
5. Richards J, Inacio M, Beckett M, Navarro RA, Singh A, Dillon MT, et al. Patient and procedure-specific risk factors for deep infection after primary shoulder arthroplasty. Clin Orthop Relat Res. 2014;472(9):2809–15.
6. Pottinger P, Butler-Wu S, Neradilek MB, Merritt A, Bertelsen A, et al. Prognostic factors for bacterial cultures positive for Propionibacterium acnes and other organisms in a large series of revision shoulder arthroplasties performed for stiffness, pain, or loosening. J Bone Joint Surg Am. 2012;94(22):2075–83.
7. Bohsali KI, Wirth MA. Complications of shoulder arthroplasty. J Bone Joint Surg Am. 2017;99(3):256–69.
8. Piper KE, Jacobson MJ, Cofield RH, Sperling JW, Sanchez-Sotelo J, Osmon DR, et al. Microbiologic diagnosis of prosthetic shoulder infection by use of implant sonication. J Clin Microbiol. 2009;47(6):1878–84.
9. Fink B, Sevelda F. Periprosthetic joint infection of shoulder arthroplasties: diagnostic and treatment options. Biomed Res Int. 2017;2017:4582756.
10. Weber P, Utzschneider S, Sadoghi P, Andress H-J, Jansson V, Muller PE. Management of the infected shoulder prosthesis: a retrospective analysis and review of the literature. Int Orthop. 2011;35(3):365–73.
11. Parvizi J, Zmistowski B, Berbari EF, Bauer TW, Springer BD, Della Valle CJ, Garvin KL, et al. New definition for periprosthetic joint infection: from the Workgroup of the Musculoskeletal Infection Society. Clin Orthop Relat Res. 2011;469(11)(1528-1132 (Electronic)):2992–4.
12. Osmon DR, Berbari EF, Berendt AR, Lew D, Zimmerli W, Steckelberg JM, et al. Diagnosis and management of prosthetic joint infection: clinical practice guidelines by the Infectious Diseases Society of America. Clin Infect Dis. 2013;56(1):e1–e25.
13. Renz N, Yermak K, Perka C, Trampuz A. Alpha defensin lateral flow test for diagnosis of periprosthetic joint infection: not a screening but a confirmatory test. J Bone Joint Surg Am. 2018;100(9):742–50.
14. Gehrke T, Parvizi J. Proceedings of the International Consensus Meeting on Periprosthetic Joint Infection. 2013. https://www.efort.org/wp-content/uploads/2013/10/Philadelphia_Consensus.pdf.
15. Garrigues GE, Zmistowski B, Cooper AM, Green A. Proceedings from the 2018 International Consensus Meeting on Orthopedic Infections: the definition of periprosthetic shoulder infection. J Shoulder Elb Surg. 2019;28(6S):S8–S12.
16. Moroder P, Gerhardt C, Renz N, Trampuz A, Scheibel M. Diagnostik und Management des Endoprotheseninfekts am Schultergelenk. Obere Extrem. 2016;11(2):78–87.
17. Sousa R, Massada M, Pereira A, Fontes F, Amorim I, Oliveira A. Diagnostic accuracy of combined 99mTc-sulesomab and 99mTc-nanocolloid bone marrow imaging in detecting prosthetic joint infection. Nucl Med Commun. 2011;32(9):834–9.
18. Piper KE, Fernandez-Sampedro M, Steckelberg KE, Mandrekar JN, Karau MJ, Steckelberg JM, et al. C-reactive protein, erythrocyte sedimentation rate and orthopedic implant infection. PLoS One. 2010;5(2):e9358.
19. Dodson CC, Craig E, Cordasco FA, Dines DM, Dines JS, Dicarlo E, et al. Propionibacterium acnes infection after shoulder arthroplasty: a diagnostic challenge. J Shoulder Elb Surg. 2010;19(2):303–7.
20. Grosso MJ, Frangiamore SJ, Saleh A, Kovac MF, Hayashi R, Ricchetti ET, et al. Poor utility of serum interleukin-6 levels to predict indolent peripros-

thetic shoulder infections. J Shoulder Elb Surg. 2014;23(9):1277–81.
21. Marcheggiani Muccioli GM, Guerra E, Roberti di Sarsina T, Alesi D, Fratini S, Cammisa E, et al. Diagnosis and treatment of infected shoulder arthroplasty: current concepts review. Joints. 2018;6(3): 173–6.
22. Colvin OC, Kransdorf MJ, Roberts CC, Chivers FS, Lorans R, Beauchamp CP, et al. Leukocyte esterase analysis in the diagnosis of joint infection: can we make a diagnosis using a simple urine dipstick? Skeletal Radiol. 2015;44(5):673–7.
23. Shafafy R, McClatchie W, Chettiar K, Gill K, Hargrove R, Sturridge S, et al. Use of leucocyte esterase reagent strips in the diagnosis or exclusion of prosthetic joint infection. Bone Joint J. 2015;97-B(9):1232–6.
24. Tischler EH, Cavanaugh PK, Parvizi J. Leukocyte esterase strip test: matched for musculoskeletal infection society criteria. J Bone Joint Surg Am. 2014;96(22)(1535-1386 (Electronic)):1917–20.
25. Wetters NG, Berend KR, Lombardi AV, Morris MJ, Morris MJ, Tucker TL, Della Valle CJ. Leukocyte esterase reagent strips for the rapid diagnosis of periprosthetic joint infection. J Arthroplasty. 2012;27(8 Suppl)(1532–8406 (Electronic)):8–11.
26. Shahi A, Parvizi J, Kazarian GS, Higuera C, Frangiamore S, Bingham J, et al. The alpha-defensin test for periprosthetic joint infections is not affected by prior antibiotic administration. Clin Orthop Relat Res. 2016;474(7):1610–5.
27. Kasparek MF, Kasparek M, Boettner F, Faschingbauer M, Hahne J, Dominkus M. Intraoperative diagnosis of periprosthetic joint infection using a novel alpha-defensin lateral flow assay. J Arthroplasty. 2016;31(12):2871–4.
28. Sigmund IK, Holinka J, Gamper J, Staats K, Bohler C, Kubista B, et al. Qualitative alpha-defensin test (Synovasure) for the diagnosis of periprosthetic infection in revision total joint arthroplasty. Bone Joint J. 2017;99-B(1):66–72.
29. Frangiamore SJ, Saleh A, Grosso MJ, Kovac MF, Higuera CA, Iannotti JP, et al. alpha-Defensin as a predictor of periprosthetic shoulder infection. J Shoulder Elb Surg. 2015;24(7):1021–7.
30. Ince A, Seemann K, Frommelt L, Katzer A, Loehr JF. One-stage exchange shoulder arthroplasty for peri-prosthetic infection. J Bone Joint Surg Br. 2005;87(6):814–8.
31. Ali F, Wilkinson JM, Cooper JR, Kerry RM, Hamer AJ, Norman P, et al. Accuracy of joint aspiration for the preoperative diagnosis of infection in total hip arthroplasty. J Arthroplasty. 2006;21(2): 221–6.
32. Parvizi J, Ghanem E, Menashe S, Barrack RL, Bauer TW. Periprosthetic infection: what are the diagnostic challenges? J Bone Joint Surg Am. 2006;88(Suppl 4) (0021-9355 (Print)):138–47.
33. Qu X, Zanjing Z, Wu C, Jin F, Li H, Wang L, et al. Preoperative aspiration culture for preoperative diagnosis of infection in total hip or knee arthroplasty. J Clin Microbiol. 2013;51(11)(1098-660X (Electronic)):3830–4.
34. Spangehl MJ, Masri BA, O'Connell JX, Duncan CP. Prospective analysis of preoperative and intraoperative investigations for the diagnosis of infection at the sites of two hundred and two revision total hip arthroplasties. J Bone Joint Surg Am. 1999;81(5) (0021-9355 (Print)):672–83.
35. Gollwitzer H, Diehl P, Gerdesmeyer L, Mittelmeier W. [Diagnostic strategies in cases of suspected periprosthetic infection of the knee. A review of the literature and current recommendations]. Orthopade. 2006;35(9):904, 6–8, 10–6.
36. Costerton JW. Biofilm theory can guide the treatment of device-related orthopaedic infections. Clin Orthop Relat Res. 2005;(437):7–11.
37. Neut D, van Horn JR, van Kooten TG, van der Mei HC, Busscher HJ. Detection of biomaterial-associated infections in orthopaedic joint implants. Clin Orthop Relat Res. 2003;(413)(0009-921X (Print)):261–8.
38. Holmes S, Pena Diaz AM, Athwal GS, Faber KJ, O'Gorman DB. Neer Award 2017: a rapid method for detecting Propionibacterium acnes in surgical biopsy specimens from the shoulder. J Shoulder Elb Surg. 2017;26(2):179–85.
39. Dilisio MF, Miller LR, Warner JJ, Higgins LD. Arthroscopic tissue culture for the evaluation of periprosthetic shoulder infection. J Bone Joint Surg Am. 2014;96(23):1952–8.
40. Segawa H, Tsukayama DT, Kyle RF, Becker DA, Gustilo RB. Infection after total knee arthroplasty. A retrospective study of the treatment of eighty-one infections. J Bone Joint Surg Am. 1999;81(10): 1434–45.
41. Sousa R, Abreu MA. Treatment of prosthetic joint infection with debridement, antibiotics and irrigation with implant retention—a narrative review. J Bone Jt Infect. 2018;3(3):108–17.
42. Azzam KA, Seeley M, Ghanem E, Austin MS, Purtill JJ, Parvizi J. Irrigation and debridement in the management of prosthetic joint infection: traditional indications revisited. J Arthroplasty. 2010;25(7):1022–7.
43. Holmberg A, Thorhallsdottir VG, Robertsson O, W-Dahl A, Stefansdottir A. 75% success rate after open debridement, exchange of tibial insert, and antibiotics in knee prosthetic joint infections. Acta Orthop. 2015;86(4):457–62.
44. Wouthuyzen-Bakker M, Sebillotte M, Lomas J, Taylor A, Palomares EB, Murillo O, et al. Clinical outcome and risk factors for failure in late acute prosthetic joint infections treated with debridement and implant retention. J Infect. 2019;78(1):40–7.
45. Choong PF, Dowsey MM, Carr D, Daffy J, Stanley P. Risk factors associated with acute hip prosthetic joint infections and outcome of treatment with a rifampin-based regimen. Acta Orthop. 2007;78(6):755–65.
46. Lora-Tamayo J, Murillo O, Iribarren JA, Soriano A, Sanchez-Somolinos M, Baraia-Etxaburu JM, et al. A large multicenter study of methicillin-susceptible and methicillin-resistant Staphylococcus aureus prosthetic

joint infections managed with implant retention. Clin Infect Dis. 2013;56(2):182–94.
47. Morata L, Senneville E, Bernard L, Nguyen S, Buzele R, Druon J, et al. A retrospective review of the clinical experience of linezolid with or without rifampicin in prosthetic joint infections treated with debridement and implant retention. Infect Dis Ther. 2014;3(2):235–43.
48. Peel TN, Buising KL, Dowsey MM, Aboltins CA, Daffy JR, Stanley PA, et al. Outcome of debridement and retention in prosthetic joint infections by methicillin-resistant staphylococci, with special reference to rifampin and fusidic acid combination therapy. Antimicrob Agents Chemother. 2013;57(1):350–5.
49. Puhto AP, Syrjala H. Short-course antibiotics for prosthetic joint infections treated with prosthesis retention. Clin Microbiol Infect. 2012;18(11):1143–8.
50. Tornero E, Morata L, Martinez-Pastor JC, Angulo S, Combalia A, Bori G, et al. Importance of selection and duration of antibiotic regimen in prosthetic joint infections treated with debridement and implant retention. J Antimicrob Chemother. 2016;71(5):1395–401.
51. Vilchez F, Martinez-Pastor JC, Garcia-Ramiro S, Bori G, Macule F, Sierra J, et al. Outcome and predictors of treatment failure in early post-surgical prosthetic joint infections due to Staphylococcus aureus treated with debridement. Clin Microbiol Infect. 2011;17(3):439–44.
52. Zimmerli W, Trampuz A, Ochsner PE. Prosthetic-joint infections. N Engl J Med. 2004;351(16)(1533–4406 (Electronic)):1645–54.
53. Aboltins CA, Dowsey MM, Buising KL, Peel TN, Daffy JR, Choong PFM, et al. Gram-negative prosthetic joint infection treated with debridement, prosthesis retention and antibiotic regimens including a fluoroquinolone. Clin Microbiol Infect. 2011;17(6):862–7.
54. Rodriguez-Pardo D, Pigrau C, Lora-Tamayo J, Soriano A, del Toro MD, Cobo J, et al. Gram-negative prosthetic joint infection: outcome of a debridement, antibiotics and implant retention approach. A large multicentre study. Clin Microbiol Infect. 2014;20(11):911–9.
55. Grossi O, Asseray N, Bourigault C, Corvec S, Valette M, Navas D, et al. Gram-negative prosthetic joint infections managed according to a multidisciplinary standardized approach: risk factors for failure and outcome with and without fluoroquinolones. J Antimicrob Chemother. 2016;71(9):2593–7.
56. Piso RJ, Elke R. Antibiotic treatment can be safely stopped in asymptomatic patients with prosthetic joint infections despite persistent elevated C-reactive protein values. Infection. 2010;38(4):293–6.
57. Bejon P, Byren I, Atkins BL, Scarborough M, Woodhouse A, McLardy-Smith P, et al. Serial measurement of the C-reactive protein is a poor predictor of treatment outcome in prosthetic joint infection. J Antimicrob Chemother. 2011;66(7):1590–3.
58. Lora-Tamayo J, Euba G, Cobo J, Horcajada JP, Soriano A, Sandoval E, et al. Short- versus long-duration levofloxacin plus rifampicin for acute staphylococcal prosthetic joint infection managed with implant retention: a randomised clinical trial. Int J Antimicrob Agents. 2016;48(3):310–6.
59. Magnan B, Bondi M, Vecchini E, Samaila E, Maluta T, Dall'Oca C. A preformed antibiotic-loaded spacer for treatment for septic arthritis of the shoulder. Musculoskelet Surg. 2014;98(1):15–20.
60. Haddad S, Corona PS, Reverte MM, Amat C, Flores X. Antibiotic-impregnated cement spacer as a definitive treatment for post-arthroscopy shoulder destructive osteomyelitis: case report and review of literature. Strategies Trauma Limb Reconstr. 2013;8(3):199–205.
61. Wouthuyzen-Bakker M, Kheir MM, Moya I, Rondon AJ, Kheir M, Lozano L, et al. Failure after 2-stage exchange arthroplasty for treatment of periprosthetic joint infection: the role of antibiotics in the cement spacer. Clin Infect Dis. 2019;68(12):2087–93.
62. Klatte TO, Junghans K, Al-Khateeb H, Rueger JM, Gehrke T, Kendoff D, et al. Single-stage revision for peri-prosthetic shoulder infection: outcomes and results. Bone Joint J. 2013;95-B(3):391–5.
63. Nelson GN, Davis DE, Namdari S. Outcomes in the treatment of periprosthetic joint infection after shoulder arthroplasty: a systematic review. J Shoulder Elb Surg. 2016;25(8):1337–45.
64. Cuff DJ, Virani NA, Levy J, Frankle MA, Derasari A, Hines B, et al. The treatment of deep shoulder infection and glenohumeral instability with debridement, reverse shoulder arthroplasty and postoperative antibiotics. J Bone Joint Surg Br. 2008;90(3):336–42.
65. George DA, Volpin A, Scarponi S, Haddad FS, Romano CL. Does exchange arthroplasty of an infected shoulder prosthesis provide better eradication rate and better functional outcome, compared to a permanent spacer or resection arthroplasty? A systematic review. BMC Musculoskelet Disord. 2016;17:52.
66. Pellegrini A, Legnani C, Macchi V, Meani E. Two-stage revision shoulder prosthesis vs. permanent articulating antibiotic spacer in the treatment of periprosthetic shoulder infections. Orthop Traumatol Surg Res. 2019;105(2):237–40.
67. Coste JS, Reig S, Trojani C, Berg M, Walch G, Boileau P. The management of infection in arthroplasty of the shoulder. J Bone Joint Surg Br. 2004;86(1):65–9.
68. Romano CL, Borens O, Monti L, Meani E, Stuyck J. What treatment for periprosthetic shoulder infection? Results from a multicentre retrospective series. Int Orthop. 2012;36(5):1011–7.

36 Biomechanics of Failure of Reverse Shoulder Arthroplasty in Rotator Cuff Tear Arthropathy

Selim Ergün, Umut Akgün, and Mustafa Karahan

36.1 Introduction

The cuff tear arthropathy is the most common indication for reverse shoulder arthroplasty (RSA) [1]. The first RSA prostheses were developed in the 1970s. Because the reverse biomechanics were poorly understood, component loosening, insufficiency fractures, and instability were the common complications. Position, dimension, and fixation of the glenoid component were the major concern of early designs.

Second-generation prostheses were developed by Grammont et al. in 1985 [2]. Biomechanical features aimed to medialize the center of rotation by changing the dimensions of the glenosphere from 2/3 sphere to 1/2 sphere (Fig. 36.1) [2]. Because glenoid component loosening was a common cause of failure in the first-generation prostheses, more medialized glenosphere was designed to reduce the lever arm; concurrently baseplate fixation was done with a central peg and two divergent screws to improve stability. However, scapular notching of the inferior border of the glenoid and limited external rotation became the new complications.

The current modern RSAs employ the following biomechanical properties:

1. To adapt individual patient needs and anatomy, altering the components on either side of the joint (modular design).
2. A large glenoid component with no neck to facilitate medialization of the center of rotation and reduced torque on glenoid component.
3. A humeral implant with a valgus angle moves the center of rotation to distal, thereby maximizing the tension of the deltoid muscle to make it a more efficient abductor, as well as increasing stability.
4. A greater impingement-free shoulder range of motion (ROM).

36.2 Biomechanical Etiology of Failure

The reported overall complication rate of RSA (for all indications, not only cuff tear arthropathy) is approximately 15%. Complications related to biomechanical factors are instability, scapular notching, fractures in association with deltoid overtensioning, implant loosening, and component dissociation.

36.2.1 Instability

Despite the semiconstrained structure of RSA, dislocation of humeral component from the glenosphere is an important problem. Instability

S. Ergün (✉) · U. Akgün · M. Karahan
Department of Orthopedics and Traumatology, Acıbadem Mehmet Ali Aydınlar University, Istanbul, Turkey

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_36

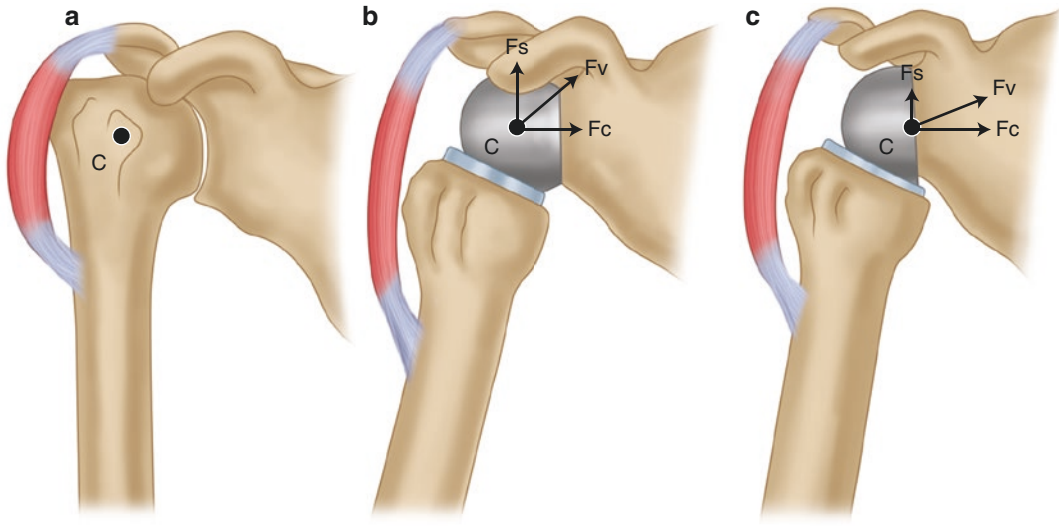


Fig. 36.1 Native shoulder (**a**) has a center of rotation which is in the middle of the humeral head and also a normal tension on deltoid. In RSA, center of rotation is medialized; deltoid muscle is tight, causing a compressive load on glenoid. There are two forces on bone-implant interface of glenoid: compressive (Fc) and shear (Fs) forces. While resultant force (Fv) has a more shear component in the second-generation designs (**b**), modern designs (**c**) with medialized center of rotation have a more compressive force on glenoid

after RSA in cuff tear arthropathy patients has a low rate of 2–3.4% [3, 4]. Insufficient deltoid tension and incorrect component position or version are mostly the reasons for instability. Mechanical impingement between polyethylene onlay and inferior glenoid might be the reason as well. Anterior instability is the most common, with a mechanism of extension, adduction, and internal rotation.

RSA with a medialized center of rotation changes the pulling direction of the deltoid, causing a dislocating effect on the joint [5]. In the native shoulder, the anterior fibers of deltoid are primarily flexor, the middle fibers are abductor, and the posterior fibers are extensor. However, after RSA, all three fibers become primary abductors [6].

Smaller glenosphere size is another risk factor for instability [7]. Langohr et al. showed that in RSAs, larger glenospheres require much more forces to dislocate in both medialized and relatively lateralized center of rotation in a cadaver model [8]. About the effect of medialized or lateralized center of rotation, Henninger et al. found that lateralizing the glenoid resulted in increase in forces required for anterior dislocation, but with a potential risk of acromial stress fracture because of deltoid overtensioning [9]. Opposing the findings of Langohr et al. about the glenosphere size, Gutierrez et al. demonstrated that increasing the ratio of the depth of the humeral implant socket to the radius of the glenosphere has a more notable effect rather than glenosphere size alone [10].

Inferior eccentric positioning of the glenoid component, creating an overhang, is shown to be an important factor to prevent adduction impingement and scapular notching, and it has been shown to increase stability by 17% [11].

Ladermann et al. demonstrated a correlation between shortened humeral length and instability [12]. It is known that both glenosphere lateralization and humeral lengthening improve stability, but with a cost of deltoid overtension and its possible complications. Humeral lateralization, which aims to increase the soft tissue tension without tightening the deltoid, seems to be a good option to prevent instability (Fig. 36.2) [13].

The version of the glenoid and humeral components have an effect on stability as well. Favre et al. demonstrated that 10° change in glenoid and humeral version affected the stability ratio on average by 15% and 27%, respectively [14].

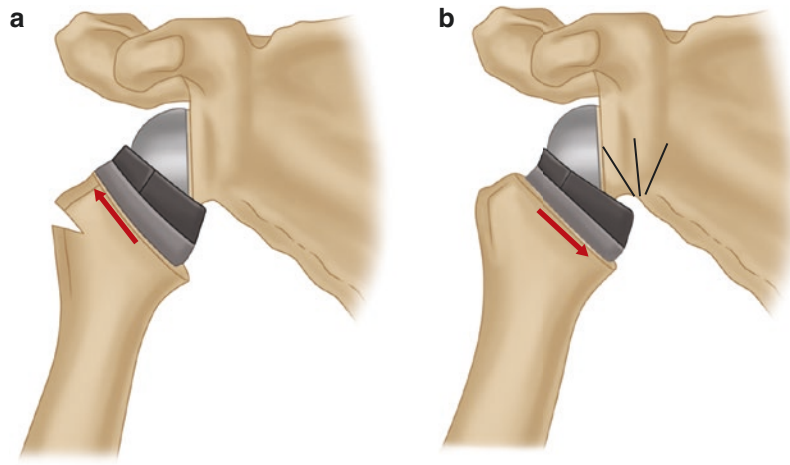


Fig. 36.2 Humeral component lateralization (**a**) increases the soft tissue tension without tightening the deltoid and decreases the risk of notching when compared to medialized humeral component (**b**)

The effect of subscapularis repair on stability is a matter of debate. Although subscapularis muscle is an important dynamic barrier against anterior instability in RSA, several studies contradict its importance. Oh et al. showed that repair of the subscapularis requires an increased load for dislocation [15]. Edwards et al. found an increased risk of instability with an unrepaired subscapularis tendon [16]. However, other studies have shown that whether the subscapularis is repaired or not, the instability rates do not differ [17, 18].

36.2.2 Deltoid Overtension (Acromion and Scapular Fracture)

Acromion and spine of scapula are the regions where deltoid origins. Overtension of the muscle might have multiple reasons: humeral lengthening, glenoid component lateralization, etc. Tight deltoid fibers may cause fatigue fractures in different parts of the scapular bone. These fractures are classified into three patterns:

- Type I: If the fracture occurs in anterior acromion
- Type II: If the fracture occurs posterior to the acromioclavicular joint line
- Type III: If the fracture occurs in the scapular spine

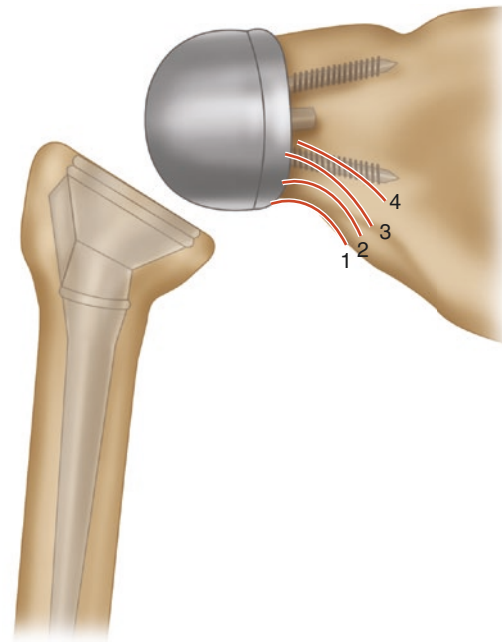


Fig. 36.3 Diagram of the Nerot-Sirveaux classification of scapular notching

36.2.3 Scapular Notching

Notching is the most common reported complication in RSA [19]. The mechanism is erosion of the inferior border of the scapular neck related to repetitive mechanical irritation of the medial rim of the humeral component during adduction of the shoulder. Scapular notching is classified according to the Nerot-Sirveaux grading system (Fig. 36.3).

Factors that may affect the degree of notching are as follows:

1. **Center of rotation:** Lateralizing the center of rotation reduces the incidence of notching. A lateralized center of rotation by lateral offset either with a bone graft or metal augment is closer to physiologic center of rotation. A systematic review by Lawrence et al. showed that scapular notching occurred in only 5.4% of patients with lateral offset, whereas in 45% of patients with second-generation Grammont-style prostheses [20].
2. **Humeral neck-shaft angle:** Anatomic neck-shaft angle of proximal humerus is 135°; early designs had a nonanatomic angle of 155°. Greater nonanatomic angles contribute to scapular notching and deficits in adduction, but they are more stable. To decrease the risk of instability, an angle close to anatomic values (~132.5°) with a larger or lateralized glenosphere is advised.
3. **Glenoid component position, tilt, and size:** Hsu et al. advocated an inferior placement of the glenoid baseplate with inferior inclination to reduce the incidence of scapular notching [21]. However, excessive inferior placement of the glenoid component may compromise glenoid fixation, increase tension in deltoid, and force the surgeon to increase humeral resection. Gutiérrez demonstrated that inferior tilt resulted in least amount of notching, followed by inferior position of the component and lateral offset [22]. Superior tilt has been shown to increase failure rates for nearly all designs and clinical scenarios. Glenosphere size is another factor; it is shown that larger glenospheres are more stable, improve impingement-free ROM, and decrease notching [23–26].

36.2.4 Glenoid Baseplate Loosening

Implant loosening is the most reported complication requiring revision surgery [27]. Glenoid baseplate loosening is more common than humeral implant loosening and has been reported with both medialized and lateralized RSA to be 2.6% and 12%, respectively [28, 29]. This complication can be secondary to prosthetic design, fixation and implantation technique (superior position, superior tilt, anterosuperior approach), and scapular notching.

Although lateralizing the center of rotation results in better function (increased deltoid function and force) [30], early designs with a more lateral center of rotation had high failure rates because of the increase in shear forces on the glenoid baseplate (Fig. 36.1). Current implant designs have a medialized center of rotation. Superior fixation properties (central post screw and peripheral locking screws) and porous coated baseplate to promote osteointegration that increases the baseplate stability [31]. In medialized center of rotation systems, the main biomechanical advantage is reduced torque secondary to no neck on glenosphere. The addition of multiple 5 mm peripheral screws significantly reduced the rate of baseplate failure [31]. James et al. showed that reduced amount of screws (two instead of four) are sufficient for baseplate fixation with no significant negative effect on overall implant baseplate motion [32]. In a recent study by Bitzer et al., it is shown that use of smaller size (3.5 mm) non-locking peripheral screws is a major risk factor for baseplate failure [33]. They also showed that bone grafting to address deficiencies in bony support beneath the baseplate is the other major risk factor [33].

Roche et al. compared the biomechanical response of four generic RSA glenoid baseplates: oval/curved back, oval/flat back, circular/curved back, and circular/flat back, to shear forces. They showed that baseplate shape and size affects fixation strength more than backside geometry [34]. Oval baseplates showed better fixation characteristics than circular counterparts [34].

Because lateralization of the center of rotation has superior biomechanical results, implant designs encourage surgeons to increase the lateral offset of the glenosphere which also increase the rate of baseplate loosening. Boileau et al. described using 10 mm ring-shaped autograft harvested from humeral head as a bony solution to increase the lateralization of the center of

rotation [35]. They named this procedure BIO-RSA (bony increased offset-reversed shoulder arthroplasty), which increased lateral offset, with only 19% scapular notching on CT scan, 98% healing rate of graft to native glenoid bone, and without any reported instability [35].

Recently, Lädermann et al. investigated how the glenoid configurations can lead to changes in parameters like: humeral offset, acromiohumeral distance, range of motion, and rotator cuff muscle length [36]. With a 145° onlay humeral stem and a 36 mm sized and 2 mm inferior eccentric glenosphere, optimum ROM and minimal scapular notching results are found [36].

36.2.5 Component Dissociation

Because the RSA components are often modular and loaded in different directions that can challenge the baseplate and glenosphere, there is a risk of dissociation with impact loading. Cusick et al. reported this complication on 13 patients, and they found that increasing the size of glenoid was the reason for component dissociation [37]. RSAs with 40 mm and 44 mm size glenospheres showed a higher rate of glenosphere dissociation. They explained this relationship to the larger exposed surface area and potential for soft tissue or bony impingement [37].

36.3 Summary

Despite experiences and better understanding of basic biomechanical properties of RSA, complications still occur. With ongoing studies, design of the components is evolving. Lateral offset with inferior overhang of the glenosphere provides the greatest reduction in notching, the most common complication. The use of bone augmentation for a more lateral center of rotation, lateralized humeral implant, and optimal glenoid tilt are emerging areas of interest to minimize the risk. Surgeons should consider all biomechanical principles in each RSA patient to prevent and manage these challenging problems.

References

1. Wall B, Nové-Josserand L, O'Connor DP, Edwards TB, Walch G. Reverse total shoulder arthroplasty: a review of results according to etiology. J Bone Joint Surg Am. 2007;89:1476–85.
2. Grammont P, Trouilloud P, Laffay J, et al. Concept study and realization of a new total shoulder prosthesis. Rhumatologie. 1987;39:407–18.
3. Nolan BM, Ankerson E, Wiater JM. Reverse total shoulder arthroplasty improves function in cuff tear arthropathy. Clin Orthop Relat Res. 2011;469:2476–82.
4. Naveed MA, Kitson J, Bunker TD. The Delta III reverse shoulder replacement for cuff tear arthropathy: a single-centre study of 50 consecutive procedures. J Bone Joint Surg Br. 2011;93:57–61.
5. Boileau P, Watkinson DJ, Hatzidakis AM, Balg F. Grammont reverse prosthesis: design, rationale, and biomechanics. J Shoulder Elb Surg. 2005;14(Suppl S):147–61.
6. Berliner JL, Regalado-Magdos A, Ma CB, Feeley BT. Biomechanics of reverse total shoulder arthroplasty. J Shoulder Elb Surg. 2015;24:150–60.
7. Trappey GJ, O'Connor DP, Edwards TB. What are the instability and infection rates after reverse shoulder arthroplasty? Clin Orthop Relat Res. 2011;469:2505–11.
8. Langohr GDG, Giles JW, Athwal GS, Johnson JA. The effect of glenosphere diameter in reverse shoulder arthroplasty on muscle force, joint load, and range of motion. J Shoulder Elb Surg. 2015;24:972–9.
9. Henninger HB, Barg A, Anderson AE, Bachus KN, Burks RT, Tashjian RZ. Effect of lateral offset center of rotation in reverse total shoulder arthroplasty: a biomechanical study. J Shoulder Elb Surg. 2012;21:1128–35.
10. Gutiérrez S, Keller TS, Levy JC, Lee WE, Luo ZP. Hierarchy of stability factors in reverse shoulder arthroplasty. Clin Orthop Relat Res. 2008;466:670–6.
11. Clouthier AL, Hetzler MA, Fedorak G, Bryant JT, Deluzio KJ, Bicknell RT. Factors affecting the stability of reverse shoulder arthroplasty: a biomechanical study. J Shoulder Elb Surg. 2013;22:439–44.
12. Lädermann A, Williams MD, Melis B, Hoffmeyer P, Walch G. Objective evaluation of lengthening in reverse shoulder arthroplasty. J Shoulder Elb Surg. 2009;18:588–95.
13. Giles JW, Langohr GDG, Johnson JA, Athwal GS. Implant design variations in reverse total shoulder arthroplasty influence the required deltoid force and resultant joint load. Clin Orthop Relat Res. 2015;473:3615–26.
14. Favre P, Sussmann PS, Gerber C. The effect of component positioning on intrinsic stability of the reverse shoulder arthroplasty. J Shoulder Elb Surg. 2010;19:550–6.

15. Oh JH, Shin SJ, McGarry MH, Scott JH, Heckmann N, Lee TQ. Biomechanical effects of humeral neck-shaft angle and subscapularis integrity in reverse total shoulder arthroplasty. J Shoulder Elb Surg. 2014;23:1091–8.
16. Edwards TB, Williams MD, Labriola JE, Elkousy HA, Gartsman GM, O'Connor DP. Subscapularis insufficiency and the risk of shoulder dislocation after reverse shoulder arthroplasty. J Shoulder Elb Surg. 2009;18:892–6.
17. Clark JC, Ritchie J, Song FS, Kissenberth MJ, Tolan SJ, Hart ND, Hawkins RJ. Complication rates, dislocation, pain, and postoperative range of motion after reverse shoulder arthroplasty in patients with and without repair of the subscapularis. J Shoulder Elb Surg. 2012;21:36–41.
18. Friedman RJ, Flurin PH, Wright TW, Zuckerman JD, Roche CP. Comparison of reverse total shoulder arthroplasty outcomes with and without subscapularis repair. J Shoulder Elb Surg. 2017;26:662–8.
19. Farshad M, Gerber C. Reverse total shoulder arthroplasty from the most to the least common complication. Int Orthop. 2010;34:1075–82.
20. Lawrence C, Williams GR, Namdari S. Influence of glenosphere design on outcomes and complications of reverse arthroplasty: a systematic review. Clin Orthop Surg. 2016;8:288–97.
21. Hsu SH, Greiwe RM, Saifi C, Ahmad CS. Reverse total shoulder arthroplasty-biomechanics and rationale. Oper Tech Orthop. 2011;21:52–9.
22. Gutiérrez S, Levy JC, Frankle MA, Cuff D, Keller TS, Pupello DR, Lee WE 3rd. Evaluation of abduction range of motion and avoidance of inferior scapular impingement in a reverse shoulder model. J Shoulder Elb Surg. 2008;17:608–15.
23. Mollon B, Mahure SA, Roche CP, Zuckerman JD. Impact of glenosphere size on clinical outcomes after reverse total shoulder arthroplasty: an analysis of 297 shoulders. J Shoulder Elb Surg. 2016;25:763–71.
24. Langohr GD, Willing R, Medley JB, Athwal GS, Johnson JA. Contact mechanics of reverse total shoulder arthroplasty during abduction: the effect of neck-shaft angle, humeral cup depth, and glenosphere diameter. J Shoulder Elb Surg. 2016;25:589–97.
25. Berhouet J, Garaud P, Favard L. Evaluation of the role of glenosphere design and humeral component retroversion in avoiding scapular notching during reverse shoulder arthroplasty. J Shoulder Elb Surg. 2014;23:151–8.
26. Torrens C, Guirro P, Miquel J, Santana F. Influence of glenosphere size on the development of scapular notching: a prospective randomized study. J Shoulder Elb Surg. 2016;25:1735–41.
27. Fevang BT, Lie SA, Havelin LI, Skredderstuen A, Furnes O. Risk factors for revision after shoulder arthroplasty: 1,825 shoulder arthroplasties from the Norwegian Arthroplasty Register. Acta Orthop. 2009;80:83–91.
28. Frankle M, Siegal S, Pupello D, Saleem A, Mighell M, Vasey M. The Reverse Shoulder Prosthesis for glenohumeral arthritis associated with severe rotator cuff deficiency. A minimum two-year follow-up study of sixty patients. J Bone Joint Surg Am. 2005;87:1697–705.
29. Alentorn-Geli E, Guirro P, Santana F, Torrens C. Treatment of fracture sequelae of the proximal humerus: comparison of hemiarthroplasty and reverse total shoulder arthroplasty. Arch Orthop Trauma Surg. 2014;134:1545–50.
30. Gutierrez S, Levy JC, Lee WE 3rd, Keller TS, Maitland ME. Center of rotation affects abduction range of motion of reverse shoulder arthroplasty. Clin Orthop Relat Res. 2007;458:78–82.
31. Mulieri P, Dunning P, Klein S, Pupello D, Frankle M. Reverse shoulder arthroplasty for the treatment of irreparable rotator cuff tear without glenohumeral arthritis. J Bone Joint Surg Am. 2010;92:2544–56.
32. James J, Allison MA, Werner FW, McBride DE, Basu NN, Sutton LG, Nanavati VN. Reverse shoulder arthroplasty glenoid fixation: is there a benefit in using four instead of two screws? J Shoulder Elb Surg. 2013;22:1030–6.
33. Bitzer A, Rojas J, Patten IS, Joseph J, McFarland EG. Incidence and risk factors for aseptic baseplate loosening of reverse total shoulder arthroplasty. J Shoulder Elb Surg. 2018;27:2145–52.
34. Roche CP, Stroud NJ, Flurin PH, Wright TW, Zuckerman JD, DiPaola MJ. Reverse shoulder glenoid baseplate fixation: a comparison of flat-back versus curved-back designs and oval versus circular designs with 2 different offset glenospheres. J Shoulder Elb Surg. 2014;23:1388–94.
35. Boileau P, Moineau G, Rousanne Y, O'Shea K. Bony increased offset reversed shoulder arthroplasty: minimizing scapular impingement while mazimizing glenoid fixation. Clin Orthop Relat Res. 2011;469:2558–67.
36. Lädermann A, Denard PJ, Boileau P, Farron A, Deransart P, Walch G. What is the best glenoid configuration in onlay reverse shoulder arthroplasty? Int Orthop. 2018;42:1339–46.
37. Cusick MC, Hussey MM, Steen BM, Hartzler RU, Clark RE, Cuff DJ, Cabezas AF, Santoni BG, Frankle MA. Glenosphere dissociation after reverse shoulder arthroplasty. J Shoulder Elb Surg. 2015;24:1061–8.

Revision of Reverse Total Shoulder Arthroplasty: Humeral Component

37

Jean Kany

37.1 Introduction

Reverse total shoulder arthroplasty (RTSA) has dramatically altered the field of shoulder reconstruction. The success of RTSA in improving function in the absence of a functioning rotator cuff has led to a broad range of applications, including massive rotator cuff tear (pseudoparalysis), failed hemi- and anatomic total shoulder arthroplasty (TSA), fractures and their sequelae, rheumatoid arthritis, and tumor reconstruction [1–3]. Enthusiasm for this technique has been tempered by the highly reported complication rate with subsequent drastic increase in the number of revisions [4]. Early reported RTSA complication rates ranged widely from 0 to 75% with revision rates varying from 2 to 10% [4]. The complication and reoperation rates tend to be higher in the revision setting with up to 22% of RTSA performed in the revision setting requiring further revision [4–6]. Revision of a failed RTSA can be technically demanding because of bone loss, soft tissue deficiency, or combined causes such as infection. Overall, the clinical results in a revision RTSA are inferior to those for a primary RTSA [7, 8]. In some cases, a new RTSA cannot be placed because of insufficient or low-quality bone stock, infection, or refractory instability. In these cases salvage surgery is required as conversion to hemiarthroplasty, spacer, or resection arthroplasty. Although poorer than those following primary RTSA, the outcomes following revision RTSA are superior to alternative salvage strategies [9–11].

Even if glenoid loosening should be the main cause for TSA revisions [12], on the contrary in RTSA complications seem to be more frequent on the humeral side such as instability due to malpositioned humeral implant.

The purpose of this study is to focus on the humeral side revisions after failed RTSA with special interest in instability, as it is the main cause for complications and revisions.

37.2 Reverse Shoulder Arthroplasty: Revision on the Humeral Side

We will consider the main postoperative complications after RTSA requiring revision and the surgical strategies for revision reported in the literature.

37.2.1 Preoperative Evaluation and Planning

A thorough preoperative evaluation is crucial when considering revision RTSA since associated

J. Kany (✉)
Clinique de l'Union, Ramsay, Saint Jean, France
e-mail: jean.kany@epaule-toulouse.com

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_37

complications tend to be underestimated such as implant loosening and infection, implant instability, and humeral shortening due to bone loss and implant instability. Preoperative evaluation should include diagnostic imaging as the following: comparative AP shoulder radiographs which include the entire prosthesis in neutral rotation, looking for implant loosening and/or medialization, comparative humeral radiographs with millimeter scale to quantify bone defect and humeral shortening by measuring the two humeri following Läderman et al. [13], and CT scan to assess the orientation of implants in the axial plane, glenoid, and humeral bone stock and fatty infiltration of the rotator cuff muscles. Furthermore, preoperative evaluation should include also biological assessment, with blood count, erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) to screen for associated latent infection or an unexpected positive culture. Finally, bone scintigraphy and/or joint aspiration may be obtained to rule out any concomitant infection even if there are lots of false-negative results. Subscapularis deficiency can be very difficult to diagnose either with arthro-CT scan (as there is a neo-capsule around the prosthesis preventing leak) or even with MRI (as there are some artifacts). Electromyography and nerve conduction studies are relevant when there is a concern for axillary nerve injury. It is important to determine the exact prosthesis that is being revised, which often requires obtaining the operative note from the primary surgery. All the instruments and implants from the manufacturer for both the prosthesis to be revised and the planned new prosthesis should be obtained. Revision instruments should be available at surgery especially if either a humeral "window" or a "sarcophagi" technique is needed to remove the humeral stem. Often, bone graft is required and the patient should be consented for both autograft and allograft.

37.2.2 Causes of RSA Instability

Prosthetic instability after RTSA is the main cause for re-intervention [4, 14–16]. It is the most difficult complication to manage, as seen from the high rate of recurrence. Implant malpositioning in the horizontal plane (humeral version on CT) and/or in the vertical plane (humeral component too high) is/are the most frequent error. Overlooking or ignoring any of the abovementioned details can lead to malpositioning of the new implants and to failure in restoring humeral length and lateralization, with risk of recurrent instability and multiple re-interventions. Special concerns must be taken in the case of shortened humerus due to a proximal bone loss secondary to implant migration, greater tuberosity lysis, bone resection secondary to acute fracture or fracture sequelae, and humeral resection for tumor. A "cam effect" mediated by posterior scared soft tissue or distal glenoid ossification spikes inducing a leverage effect. Soft tissue deficiency such as subscapularis tear and/or deltoid atrophy due to axillary palsy may also induce instability.

37.2.2.1 Why Does the Humerus Length Influence the RSA Stability?

The tensioned deltoid provides the stable fulcrum essential for prosthesis stability. The failure to restore sufficient deltoid tension may result in prosthetic instability [13]. A shortening of the arm and thus a lack of retensioning of the deltoid constantly lead to a potential cause of instability. This situation may be accounted for by relative humeral shortening compared to the contralateral side. Läderman et al. found out a strong correlation between humeral length and incidence of dislocation [17]. Shortening of the humerus postoperatively compared to preoperatively or shortening compared to the contralateral humeral length was observed in all cases of dislocation. When sufficient deltoid tension was not restored, a significantly higher risk of instability ($p < 0.0001$) was reported. Deltoid tension produced by the lowered humerus increases muscle fiber recruitment of the anterior and posterior deltoid that makes up for a deficient rotator cuff. However excessive lengthening is not advisable because it could increase the risk of complications such as neurologic damage [17], permanent arm abduction [1], or acromial fracture [17].

37.2.2.2 Preoperative Humerus Length Assessment Is Mandatory

Recommendations are numerous to assess the intraoperative prosthetic stability: difficulty to reduce the implanted prosthesis, pistoning with axial traction on the arm, looking for a "cam effect" throughout a full range of motions [18], tensioning the conjoint tendon after reduction with the arm at the side and the elbow extended [1], and free glenohumeral motion without scapula-thoracic motion between 0 and 60° of abduction [19]. Nevertheless intraoperative assessment of deltoid tension can be difficult because those criteria are evaluated in the patient under general and/or locoregional anesthesia. Läderman et al. showed that bilateral scaled humeri X-rays and preoperative templating help the surgeon to assess the correct length and deltoid tension especially in cases of revision surgery or fracture sequelae [13]. Boileau has published a discussion using preoperative and postoperative external measurements to describe retensioning of the deltoid [1].

One of the limits of the bilateral scaled humeri X-rays is presence of fibrosis, scar, and retraction of soft tissue. Indeed, after extensive release of the retracted soft tissue and adequate correction of the humerus length, care must be taken of the muscle (deltoid), tendon (subscapularis), and skin closure. This must be anticipated at the time of the incision to plan any flap for tissue reconstruction if needed.

37.2.2.3 How to Correct an Inadequate Humerus Length

Influence of the Prosthesis Design

It is critically important to know the different combinations of design parameters that influence biomechanical changes. Each prosthesis design has got its own principles and must be understood. Here are some general considerations.

The humeral offset is defined as the horizontal distance between the intramedullary canal and the humeral stem axis to the center of the humeral liner. This humeral offset depends on the prosthesis design but can be modified by an additional humeral spacer component to prevent any recurrent instability (Figs. 37.1 and 37.2).

A glenosphere with a center of rotation (CoR) of 5 mm or less lateral to the glenoid face is regarded as a medialized glenoid (MG), and a glenosphere with a CoR greater than 5 mm lateral to the glenoid face is regarded as a lateralized glenoid (LG). MG designs are associated with less deltoid wrapping, which reduces the horizontal stabilizing compressive force vector of the deltoid and may increase the risk of dislocation if not addressed on the humeral side. As a result of preoperative glenoid bone erosion, all the downsides of MG devices can be further exaggerated with instability. The glenosphere thickness is directly related to the lateralization of the CoR relative to the glenoid face.

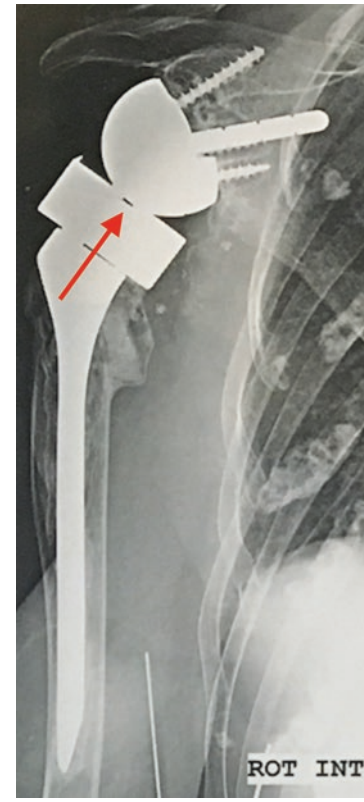


Fig. 37.1 This additional implant lengthens and lateralizes the humerus. Therefore it modifies both the humeral offset and the deltoid wrap angle

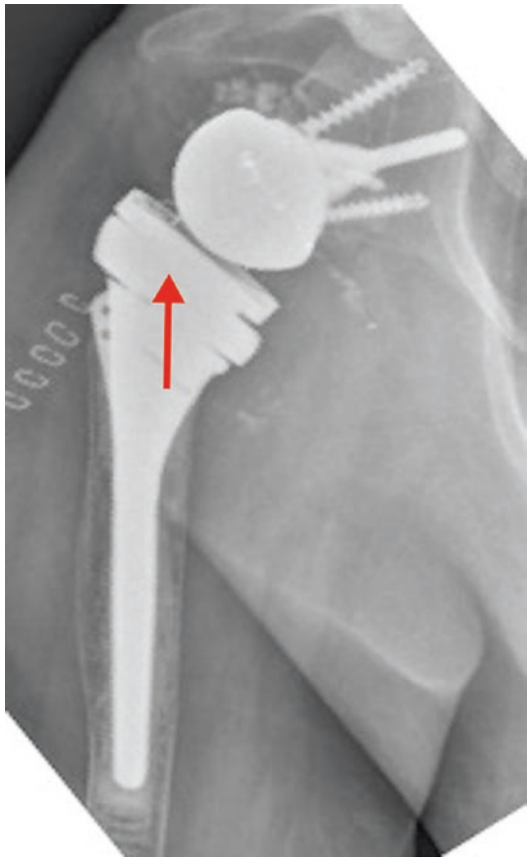


Fig. 37.2 This additional implant lengthens but does not lateralize the humerus. Therefore it modifies neither the humeral offset nor the deltoid wrap angle

Indeed the acromiohumeral distance depends on the thickness of the polyethylene insert, the size of the implant, the use of an eccentric glenosphere, and the position of the glenosphere in the vertical plane. The more lateral the humeral component, the greater the deltoid wrapping and also the more anatomic rotator cuff muscle tensioning (Fig. 37.3).

Therefore the effective treatment of dislocations may consist in adding a metallic extension to the humeral neck (with or without any humeral offset modification) (Figs. 37.1 and 37.2), an enlarged polyethylene liner component, a larger diameter or eccentric glenosphere, or combined procedures to improve the tension of the deltoid [17].

Original Grammont humeral stems have an intraosseous metal inlay that medializes the humerus [18]. In changing prosthesis, humeral length and lateralization can be increased by changing from inlay to onlay.

Influence of the Technique

The use of a structural humerus allograft can be put forward to lower the humerus and to retension the deltoid in case of significant bone loss to maximize the long stem fixation and to prevent any loosening or rotation (Fig. 37.4a, b). This option retensions the deltoid (by restoring its wrapping angle) as well as the remaining cuff and increases the stability and the compressive forces.

37.2.3 Revision Technique

37.2.3.1 Exposure

The typical approach for revision RTSA is a deltopectoral approach, which may be extended distally to expose the humeral shaft in case of window, osteotomy, or even sarcophagi to remove the prosthesis followed by cerclage reconstruction. Antibiotics, after multidisciplinary discussion, are held in all revision cases until intraoperative specimens are obtained to prevent unexpected positive cultures. Cultures should be held for 2–3 weeks to rule out *Cutibacterium acnes* infection [20, 21]. It is often difficult to establish tissue planes in revision surgery. The subscapularis tendon, if present, is either peeled or divided, or the lesser tuberosity insertion is osteotomized to gain access to the glenohumeral joint. The axillary nerve should be palpated and preserved throughout the approach. This step is mandatory as neurological lesions are one of the main complications after RTSA revision. Once the glenohumeral joint is entered, the prosthesis is exposed and assessed for position and stability. When unstable, inappropriately positioned, or infected the stem does require removal. Bone resection should be minimized. Thin osteotomes can be used to break up the implant-bone or bone-cement interface within the metaphysis. If the stem cannot be dislodged, a humerotomy is performed stopping just short of the stem tip behind the bicipital groove using a motorized

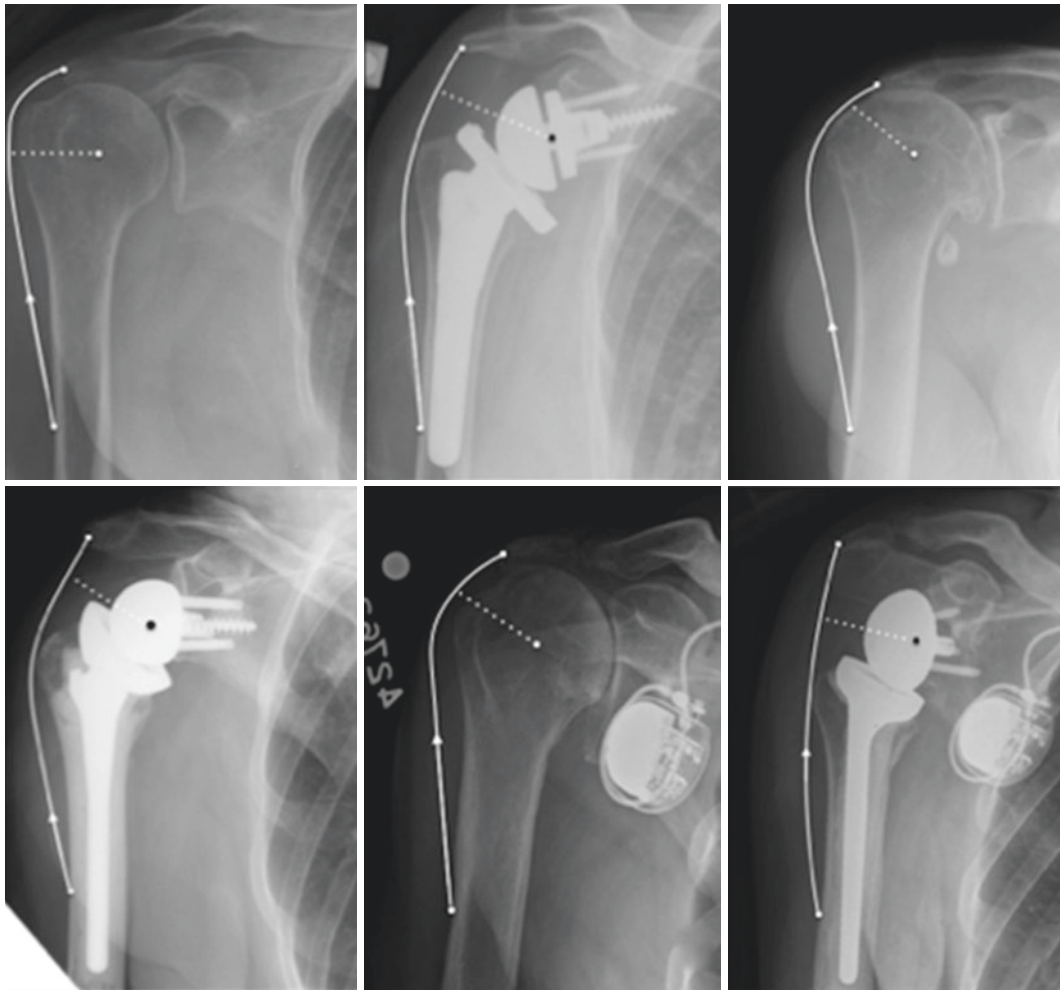


Fig. 37.3 The deltoid wrap angle depends on the prosthesis design: the more lateral the humeral component, the greater the deltoid wrapping

saw. Multiple techniques have been described in the literature for stem removal. We are used to performing a full, retractable cortical sarcophagi along the length of the stem by an extended transversally osteotomy pediculated with the pectoralis major/latissimus dorsi and teres major: the idea is to split the bone from cement and open the cortical bone to free the implant [22]. With revisions of infected cemented stems, the cement mantle must be removed in its entirety. If noninfected, the cement mantle can sometimes be preserved and the revision (smaller) implant may be cemented into the existing cement mantle stem with the correct level and version. Cerclage wires or cables are carefully placed around the osteotomy or sarcophagi site to recreate an intact cylinder prior to reaming and/or placement of the revision implant. The radial nerve should be protected during the passage of cerclage wires/cables. Allograft struts may be added for thin or deficient diaphyseal humeral bone. The revision stem should be extended 2.5 cortical diameters distal to the osteotomy site and may be longer than the revised one.

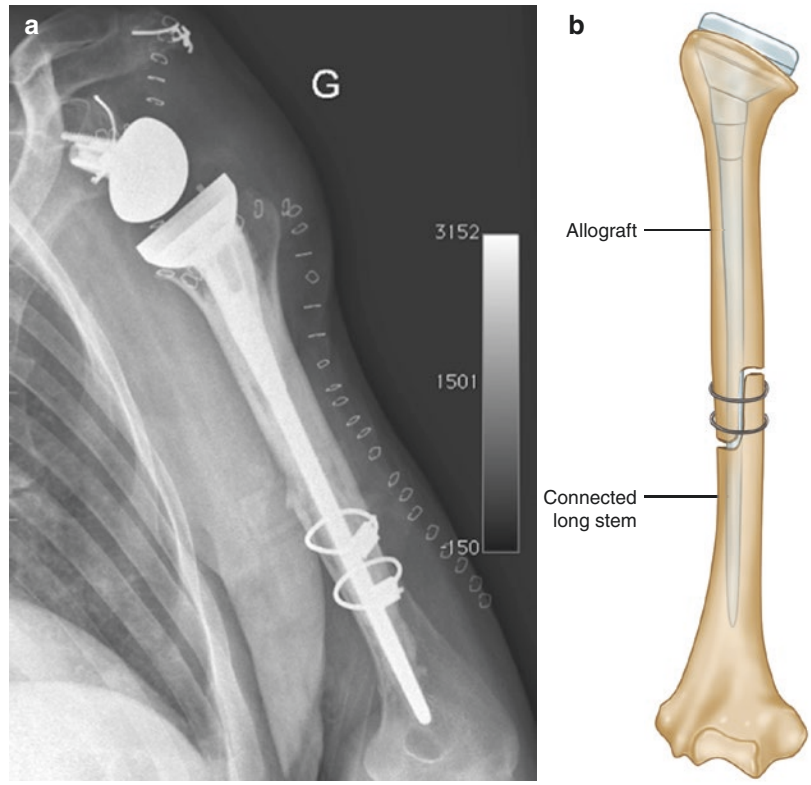


Fig. 37.4 (**a**, **b**) The use of a structural humerus allograft can be useful in case of significant bone loss to maximize the long stem fixation and to prevent any loosening or rotation

37.2.3.2 Management of Humeral Shortening

When shortening is less than ±15 mm (this depends on the company implant designs) and if there is no humeral malpositioning or loosening, humeral height can be restored by adding a thicker humeral baseplate without changing implants. When shortening exceeds ±15 mm, the humeral implant may need to be replaced and positioned at "the correct height," based on measurement of the length of the contralateral humerus. When shortening exceeds 5 cm, structural humeral bone graft (or massive reconstruction prosthesis) can improve shoulder stability and prevent loosening [20, 23–27]. The long-stemmed humeral implant is often cemented into the proximal humeral graft and then fixed distally by press fit or cement. Rotational stress should be neutralized by a "step cut" between graft and shaft (Fig. 37.4a, b). When proximal humeral resection is substantial, we opt for an uncemented humeral implant locked by screws to neutralize rotational force.

Reinsertion of the subscapularis tendon can improve internal rotation and RTSA stability. Unfortunately most of the time this reinsertion is not possible, this being either due to the absence of soft tissue or to the implant lateralization that increases the tension with high risk of rupture. We do not recommend any latissimus dorsi muscle-tendon transfer on the humeral allograft in case of external rotator deficit, as there is a high risk of instability recurrence due to absence of efficient subscapularis.

37.2.3.3 Management of a Chronic Infected RTSA

RTSA instability can be combined with infection with or without preoperative evidence. Implant replacement is the attitude of choice but involves advanced surgery due to possible intra- and postoperative complications. The technical difficulty lies in removing the implant because of frequent absence of loosening with risk of humeral fracture and bone defect. One-step replacement (humeral component fixed with antibiotic-

bearing cement) gives better functional results, with lower rates of morbidity and complications without any greater risks of recurrent infection especially if the implicated bacteria are known in advance, which is a very rare situation [28–30]. Most of the time infection is either a preoperative finding (fluid inside) or a 3-week postoperative period diagnosis, thanks to unexpected positive cultures. This is the reason why we recommend a systematic double antibiotherapy for 3 weeks until results of culture samples.

In very uncommon situations a two-step procedure could be preferable, despite greater morbidity, with insertion of an antibiotic-bearing cement spacer to fill the resulting space and provide local antibiotic therapy for 6–8 weeks. We routinely use a custom made spherical spacer ("tennis ball" shape) in two-stage revisions as a temporary interposition, but in certain cases they can be a permanent solution if the patient is unable or unwilling to be subjected to another surgical operation [9]. This special shape allows a double mobility of the shoulder but may be unstable. It is our impression that shoulder comfort and mobility can be enhanced by tensioning of the deltoid and lateralization of the humeral shaft, thanks to a spacer.

Prosthesis removal alone (resection arthroplasty without permanent spacer) could be a salvage option too and should be considered in low-demanding and fragile patients, resistant infection, or after failure of other surgical techniques [10]. Most authors agree on the fact that resection arthroplasty is a valid therapeutic option: those patients get reliable pain relief but outcomes concerning function cannot be always predicted if compared to those when the implant is conserved or replaced [11, 31, 32].

37.3 Proximal Humeral Bone Loss: Algorithm

Gohlke and Werner in 2006 defined five different humerus zones in case of inadequate length: the metaphyseal bone loss (zone 1), the rotator cuff (zone 2), above the deltoid insertion (pectoralis major, latissimus dorsi, and the teres major) (zone 3), beyond the deltoid insertion, and close to the elbow joint (Fig. 37.5) [32].

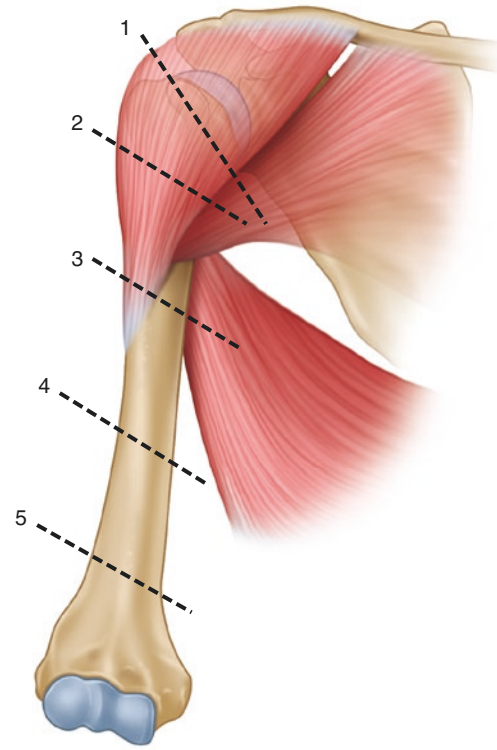


Fig. 37.5 The five different humerus zones described by Gohlke and Werner [32]. Beyond the zone 2, a structural humeral allograft is recommended to prevent a possible stem loosening

1. **Zones 1 and 2 (Proximal Metaphysis Bone Loss)**

   On the glenoid side, a greater glenosphere with an inferior tilt and a thicker baseplate with a metallic spacer on the humeral side can lower the humerus about 15 mm. A glenoid bone graft (bio-RSA) will lateralize the humerus and will restore the deltoid wrapping angle.
2. **Zones 3 and 4 (Proximal Diaphysis Bone Loss)**

   A longer and more proud humeral stem will be mandatory with a structural humeral allograft to prevent any possible loosening.

37.4 Conclusion

The indications and usage of reverse shoulder arthroplasty keep on expanding, with revision of RTSA occurring more often. The most common indications for humeral component revision of RTSA are instability, infection, and loosening. All those causes can be combined. Surgical challenges include proximal humeral bone loss and unexpected positive cultures.

Failing to tension of the deltoid may result in prosthetic instability recurrence and iterative revision. A preoperative deficit humeral length (vertical imbalance) must be evaluated with bilateral scaled humeri X-rays. Different combinations of design parameters influence biomechanical changes (vertical and horizontal imbalance) and are specific to prosthesis for proximal metaphysis bone loss. Structural humeral allograft seems mandatory for proximal diaphysis bone loss. Intraoperative stability assessment searching for an inferior or posterior impingement is important (came effect). Care must be taken of regarding soft tissue since lengthening and lateralizing the humerus may make the closure difficult. Finally, failing to adequately diagnose associated low-grade infection can be prevented, thanks to a 3-week postoperative period antibiotherapy. In some cases where revision components cannot be implanted, salvage options, including resection arthroplasty, can provide good pain relief but limited function.

References

1. Boileau P, Watkinson DJ, Hatzidakis AM, Balg F. Grammont reverse prosthesis: design, rationale, and biomechanics. J Shoulder Elb Surg. 2005;14:47–61.
2. Boileau P, Watkinson D, Hatzidakis AM, Hovorka I. Neer Award 2005: the Grammont reverse shoulder prosthesis: results in cuff tear arthritis, fracture sequelae, and revision arthroplasty. J Shoulder Elb Surg. 2006;15:527–40.
3. Wall B, Nove-Josserand L, O'Connor DP, Edwards TB, Walch G. Reverse total shoulder arthroplasty: a review of results according to etiology. J Bone Joint Surg Am. 2007;89:1476–85.
4. Zumstein MA, Pinedo M, Old J, Boileau P. Problems, complications, reoperations, and revisions in reverse total shoulder arthroplasty: a systematic review. J Shoulder Elb Surg. 2011;20:146–57.
5. Groh GI, Groh GM. Complications rates, reoperation rates, and the learning curve in reverse shoulder arthroplasty. J Shoulder Elb Surg. 2014;23:388–94.
6. Kempton LB, Ankerson E, Wiater JM. A complication based learning curve from 200 reverse shoulder arthroplasties. Clin Orthop Relat Res. 2011;469:2496–504.
7. Austin L, Zmistowski B, Chang ES, Williams GR Jr. Is reverse shoulder arthroplasty a reasonable alternative for revision arthroplasty? Clin Orthop Relat Res. 2011;469:2531–7.
8. Walker M, Willis MP, Brooks JP, Pupello D, Mulieri PJ, Frankle MA. The use of the reverse shoulder arthroplasty for treatment of failed total shoulder arthroplasty. J Shoulder Elb Surg. 2012;21:514–22.
9. Brodt S, Hofmann GO, Tiemann AH. Preservation of the shoulder joint by the use of a hybrid-spacer after septic loosening of a reversed total shoulder joint arthroplasty: a case report. Strategies Trauma Limb Reconstr. 2010;5:111–4.
10. Verhelst L, Stuyck J, Bellemans J, Debeer P. Resection arthroplasty of the shoulder as a salvage procedure for deep shoulder infection: does the use of a cement spacer improve outcome? J Shoulder Elb Surg. 2011;20:1224–33.
11. Muh SJ, Streit JJ, Lenarz CJ, McCrum C, Wanner JP, Shishani Y, Moraga C, Nowinski RJ, Edwards TB, Warner JJ, Walch G, Gobezie R. Resection arthroplasty for failed shoulder arthroplasty. J Shoulder Elb Surg. 2013;22:247–52.
12. Bohsali KI, Wirth MA, Rockwood CA. Complications of total shoulder arthroplasty. J Bone Joint Surg Am. 2006;88:2279–92.
13. Läderman A, Williams MD, Melis B, Hoffmeyer P, Walch G. Objective evaluation of lengthening in reverse shoulder arthroplasty. J Shoulder Elb Surg. 2009;8:588–95.
14. Trappey GJT, O'Connor DP, Edwards TB. What are the instability and infection rates after reverse shoulder arthroplasty? Clin Orthop Relat Res. 2011;469:2505–11.
15. Gallo RA, Gamradt SC, Mattern CJ, et al. Instability after reverse total shoulder replacement. J Shoulder Elb Surg. 2011;20:584–90.
16. Farshad M, Gerber C. Reverse total shoulder arthroplasty from the most to the least common complication. Int Orthop. 2010;34:1075–82.
17. Läderman A, Walch G, Lubbeke A, Drake GN, Melis B, Bacle G, Collin P, Edwards BT, Sirveaux F. Influence of arm lengthening in reverse shoulder arthroplasty. J Shoulder Elb Surg. 2012;21:336–41.
18. Grammont P, Latfay J, Deries X. Etude et réalisation d'une nouvelle prothèse d'epaule. Rhumatologie. 1987;39:407–18.
19. Valenti P, Katz D. Comment implanter une prothèse inversée? In: Maîtrise Orthopédique. 2005;148.

20. Boileau P. Complications and revision of reverse total shoulder arthroplasty. Orthop Traumatol Surg Res. 2016;102:S33–44.
21. Garver J, Mata-Fink A, Zhu D, Javier F, Gallacher S, Blaine TA. Revision strategies in reverse total shoulder arthroplasty. J Shoulder Elb Arthroplasty. 2017;1:1–10.
22. Gohlke F, Rolf O. Revision of failed fracture hemiarthroplasties to reverse total shoulder prosthesis through the trans-humeral approach: method incorporating a pectoralis-major-pedicled bone window. Oper Orthop Traumatol. 2007;19:185–208.
23. Boileau P, Melis B, Duperron D, Moineau G, Rumian AP, Han Y. Revision surgery of reverse shoulder arthroplasty. J Shoulder Elb Surg. 2013;22:1359–70.
24. Budge MD, Moravek JE, Zimel MN, Nolan EM, Wiater JM. Reverse total shoulder arthroplasty for the management of failed shoulder arthroplasty with proximal humeral bone loss: is allograft augmentation necessary? J Shoulder Elb Surg. 2013;22:739–44.
25. Chacon A, Virani N, Shannon R, Levy JC, Pupello D, Frankle M. Revision arthroplasty with use of a reverse shoulder prosthesis-allograft composite. J Bone Joint Surg Am. 2009;91:119–27.
26. Abdeen A, Hoang BH, Athanasian EA, Morris CD, Boland PJ, Healey JH. Allograft-prosthesis composite reconstruction of the proximal part of the humerus: functional outcome and survivorship. J Bone Joint Surg Am. 2009;91:2406–15.
27. Dudkiewicz I, Velkes S, Oran A, Pritsch M, Salai M. Composite grafts in the treatment of osteosarcoma of the proximal humerus. Cell Tissue Bank. 2003;4:37–41.
28. Amaravathi RS, Kany J, Melet M, Katz D, Sauzieres P, Valenti P, Gandois JM. Analysis of infection in shoulder arthroplasty: a multicentre study. Eur J Orthop Surg Traumatol. 2012;22:145–50.
29. Jacquot A, Sirveaux F, Roche O, Favard L, Clavert P, Molé D. Surgical management of the infected reversed shoulder arthroplasty: a French multicentric of reoperation in 32 patients. J Shoulder Elb Surg. 2015;24:1713–22.
30. Beeckman PDA, Katusic D, Berghs BM, Karelse A, de Wilde L. One stage revision for patients with chronically infected reverse total shoulder replacement. J Bone Joint Surg Br. 2010;92:817–22.
31. Casier S, Middernacht B, Van Tonge A, DeWilde L. Revision of reversed shoulder arthroplasty: is a reoperation possible? Obere Extrem. 2017;12:16–24.
32. Gohlke F, Rolf O, Werner CH. Results of reverse arthroplasty in revision of failed hemiarthroplasties. In: Reverse shoulder arthroplasty: clinical results, complications, revision. Nice Shoulder Course. Sauramps Medical; 2006. p. 209–15.

Case Example 1: Failure of Rotator Cuff Repair

38

Roger Hackney

38.1 Introduction

Failure of rotator cuff repairs is more common than we like to think. There are studies which have shown up to a 94% failure rate in repairing very large and massive tears [1]. Even moderate tears of the rotator cuff were found to have a 40% recurrence rate in the UKUFF trial, a figure not different from much of the literature [2]. Of course, the recurrence rate does depend upon how hard you look for tears. Some studies have used arthrographic techniques, where any leakage into the subacromial space counts as a recurrence; some studies use ultrasound scan [3, 4]. However, my radiology colleagues tell me it can actually be very difficult to determine whether the changes seen are post-surgical artefact or truly recurrence of the tendon tear. The same applies to MRI scan. The literature does not supply data on whether there has been a complete recurrence of the original tear or just a small failure of complete closure. A non-watertight repair of a large to massive tear is not necessarily a poor procedure. This may be why the literature also suggests that, certainly for the first year or two, there is very little difference in outcome between the two groups [5].

In clinical practice we see patients in whom the repair simply pulls apart. The rotator cuff tendon has lost its capacity for healing, the muscle does not recover and function is never regained or is lost. The risk factors for recurrence of tear include quality of tendon, tension of the repair, number of tendons involved (size of tear) and the fatty degeneration index (FDI) [6, 7].

The scenario of complete recurrence of tear is undoubtedly more likely when dealing with large to massive tears. What are the options for this category of patient?

I describe two patients with different causes of recurrence where a synthetic patch was used to effect the revision repair of otherwise non-repairable rotator cuff tendons.

Case Example 1

Mr. DS is a 69-year right arm dominant retired man who initially presented with a typical history of a painful shoulder due to rotator cuff disease. He complained of pain with overhead activity, an inability to lie on his shoulder at night with sleep disturbance, difficulty dressing and undressing but, most importantly for him, pain on playing golf. Mr. DS is an avid golfer who plays several times a week when fit, but was struggling to play a full round without severe pain.

Examination revealed pain and mild weakness on stressing supraspinatus. Investigation with dynamic ultrasound scanning revealed a moderate-sized tear of the rotator cuff. He had failed to ben-

R. Hackney (✉)
Spire Hospital Leeds, Roundhay Hall, Leeds, UK
e-mail: roger.hackney@nhs.net

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_38

efit from physiotherapy and elected to undergo an arthroscopic repair of his rotator cuff.

At surgery a moderate tear of supraspinatus measuring just over 1 cm longitudinally and 1 cm transversely was found. A simple single-row repair using a single anchor was performed. The repair was watertight at the end of the procedure. Standard protocol following this is to spend 4 weeks in a sling with passive range of motion rehabilitation only.

At the end of 4 weeks he represented with a painful swelling of the shoulder, with some pain. He had discomfort on moving the shoulder. The appearances were of a low-grade infection. This appeared to be superficial on initial examination and a course of antibiotics (flucloxacillin) was prescribed. There were no signs of joint infection, with a full passive range of motion achievable with minimal pain. However, after 2 weeks, the swelling had not settled and the wound looked worse.

Surgical exploration of the wound was undertaken. The findings were of significant fatty necrosis and fluid extending through to the joint. The rotator cuff repair had come apart and had significantly increased in size. A surgical debridement was performed and all foreign material removed. The wound was not closed and required two further open debridements to remove recurrent infection. The wound was subsequently closed when all tissues appeared free of infection.

Prolonged cultures grew *Propionibacterium acnes*. He was treated according to advice from the microbiology team with 6 weeks teicoplanin, intravenously, via a long line.

Mr. DS continued to receive physiotherapy and 4 months later had regained some overhead activity. However, his arm felt weak and he was unable to return to golf without pain. Oxford Shoulder Score was 17. He expressed a wish to undergo revision repair.

An MRI scan showed a tear of 3 × 1.5 cm with significant loss of bulk of supraspinatus.

Advice from the microbiologists was that a 6-week course of teicoplanin should have removed any intra-articular *P. acnes* and that normal prophylaxis was all that would be required. Nonetheless Mr. DS was counselled on the risks of recurrent infection.

He elected to undergo revision. With large recurrent tears it is my practice to consent patients to an arthroscopic repair if the tendon quality is good and the tendon can be mobilised to allow a repair without undue tension.

At shoulder arthroscopy the tear was found to be large to massive with a large area of uncovered humeral head. The quality of the tendon was poor and arthroscopic mobilisation of the tendon failed to bring the edges of the tendon together without significant tension for even a partial repair.

Mr. DS had several risk factors for recurrence of tear. The Goutallier score for supraspinatus was 3, the size of the tear was large (two tendons were involved), the quality of tendon was poor and high tension would have been present with a conventional repair.

The risk of recurrent tear for this patient from a standard arthroscopic repair was very high. The author's experience with patch augmentation using the Leeds-Kuff Patch in revision surgery is that the risk of re-recurrence is dramatically reduced.

Mr. DS underwent an open repair augmented with the Leeds-Kuff Patch. A lateral deltoid splitting approach is used peeling the deltoid from the anterolateral acromion. The arm is held in a shoulder holder, allowing stable access to the rotator cuff. The tendon was not completely repairable despite extensive mobilisation using both arthroscopic and open techniques. At open surgery the tendon is mobilised using stay or traction sutures with number two Ethibond sutures. A partial repair was performed using the traction sutures and Orthocord to the apex of the repair. A Leeds-Kuff Patch was applied, the gap bridged by the patch material which was sutured over the repair using the Ethibond sutures with additional interrupted Orthocord sutures. A meticulous closure of deltoid was performed using one Ethibond sutures to reattach the deltoid muscle into its anatomical position. A subcuticular closure is used for excellent cosmesis.

As is often the case, he reported that the repair felt stronger and more secure than following his initial arthroscopic repair. At 2 months following his revision repair with patch he had lost all the aching he had prior to surgery and was progressing well with supervised rehabilitation. He had

regained full overhead activity. He was able to start to play golf again after 3 months, much to his delight. At 6-month review Mr. DS' Oxford Shoulder Score had improved from 17 to 48, a normal pain-free shoulder, and he was able to return to playing golf.

Although the patch repair is undoubtedly stronger than a conventional repair my practice is to follow my standard protocol until evidence can be gained that it is safe to undergo accelerated rehabilitation.

Case Example 2

Mr. IG is a right-handed electrician who worked for a local museum. He is a very large man with a moderate-sized tear of the left rotator cuff that was causing pain and weakness with overhead activity. He was struggling at work. Examination revealed a reluctance to move beyond 60° forward flexion of abduction because of pain. Supraspinatus was weak but infraspinatus was nearly flail or pseudo-paralytic. An ultrasound scan revealed only a moderate-sized tear of the rotator cuff. He had undergone a course of physiotherapy prior to referral and chose the option of repair of his rotator cuff.

He underwent an arthroscopic repair of his rotator cuff. An small os acromiale was noted, and an arthritic, painful acromio-clavicular joint was excised. He progressed remarkably quickly but unfortunately the physiotherapist supervising him commenced lifting with weights 2 months after his repair, against the standard protocol. Mr. IG felt a sudden pull in his shoulder with loss of function. A further ultrasound scan revealed a recurrence of tear measuring at 3 × 1.7 cm. He failed to improve with further physiotherapy. It was elected to redo the surgery with a low threshold for patch augmentation. At arthroscopy the tendon was found to be significantly more tendinopathic than had been recognised on the ultrasound scans or at the time of the previous repair. Poor quality of tendon probably explained the failure of the original repair, and patch augmentation was determined to be the best option.

An open repair was performed with partial repair of the tendon achievable without undue tension on the repair. A patch was applied over the repair, bridging a moderate sized defect.

Mr. IG underwent a standard rehabilitation protocol. At 6 months his Oxford Shoulder Score had improved from 20 to 48 with normal shoulder function. He returned to full duties at work without any pain. He had full power with overhead activity and was able to perform all his duties as an electrician.

38.2 Summary

The UKUFF trial [2] produced a recurrence rate of 40% for moderate tears of the rotator cuff repaired either arthroscopically, mini-open or open. The literature indicates that not all recurrences of tear become symptomatic during early follow up, but clinical practice suggests that over a longer period a significant number of patients return with symptoms attributable to a recurrent tear.

When patients represent with symptoms, the tears are often increased in size; there is a greater degree of muscle atrophy. My anecdotal experience suggests that it is patients with more tendinopathic rotator cuff tendons and particularly those where a suture has pulled out of the tendon during the initial repair that are more likely to fail.

It seems evident that it is not the method of repair, whether it single or double row or open that is the concern, but the biology of the tendon itself. The healing potential for such tendons is poor. When recurrence of tear occurs, it is therefore illogical to try and repeat the same procedure in the expectation of a different outcome—a definition of madness often attributed to Einstein.

Patch augmentation alters the biology. An ideal patch will allow native tissue to grow into the patch; be strong enough to withstand traction from sutures during repair, even allowing early mobilisation; and produce a strong, complete rotator cuff. The material must not produce any adverse tissue reactions/histological response. This has been the case with xenografts, where outcomes have not been very successful. Human skin shows very little native tissue ingrowth. Where the patch is used to bridge large defects, the patch must retain its strength during the period of tissue ingrowth, or the

repair will fail. This was my experience with Artelon, which did not produce sufficient tissue ingrowth to bridge large gaps. The strength of the graft was not sufficient to hold the rotator cuff repair intact once the patch material biodegrades.

The Leeds-Kuff Patch is not biodegradable. The pull-out strength is equivalent to triple thickness of dermal graft. The tendon would fail before this is reached. The patch allows excellent tissue ingrowth and can bridge reasonably large defects with a successful outcome. What is not yet known is whether muscle atrophy with a Goutallier score of 3 or more is recoverable. A patient of mine who had an arthroscopic repair of a tear which recurred worked as a joiner. He underwent a patch repair with the Leeds-Kuff Patch but took 18 months to regain full power in overhead activity.

Where recurrences of tears occur, it is the author's opinion and experience that augmentation with a synthetic patch, the Leeds-Kuff Patch, dramatically reduces the risk of recurrence and can lead to full recovery (Figs. 38.1, 38.2, 38.3, 38.4, 38.5, and 38.6).

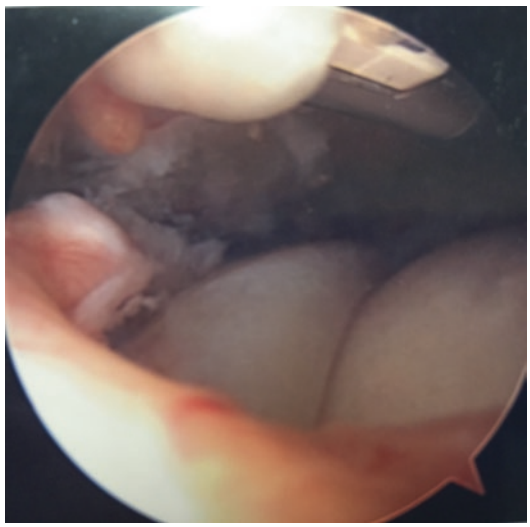


Fig. 38.1 Initial view of retraction of the rotator cuff. A 49-year-old painter and decorator with a massive tear of the rotator cuff. The tendon can be seen lying behind the glenoid, but is of good thickness and quality

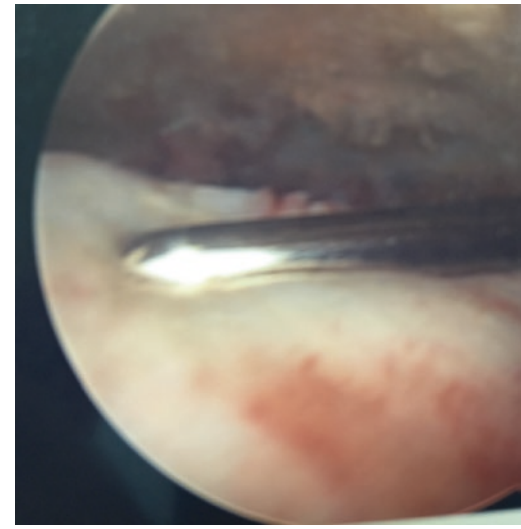


Fig. 38.2 Post-mobilisation of rotator cuff tendon. After aggressive arthroscopic mobilisation the rotator cuff could be mobilised to partly cover the humeral head, but the tendon was not fully repairable

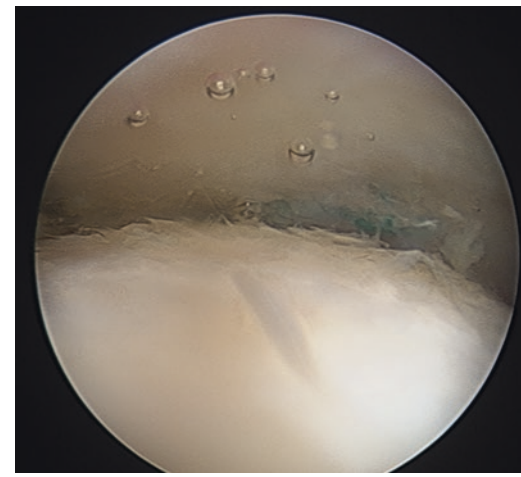


Fig. 38.3 Intra-articular view following release. Postoperatively he developed stiffness and this failed to resolve. At 6 months post-repair he underwent an elective capsular release of the rotator interval and clearance of adhesions in the subacromial space. The patch material has been completely encased in native tissue. The damage to the superior surface of the humeral head from abrasion against the acromion prior to repair can still be seen

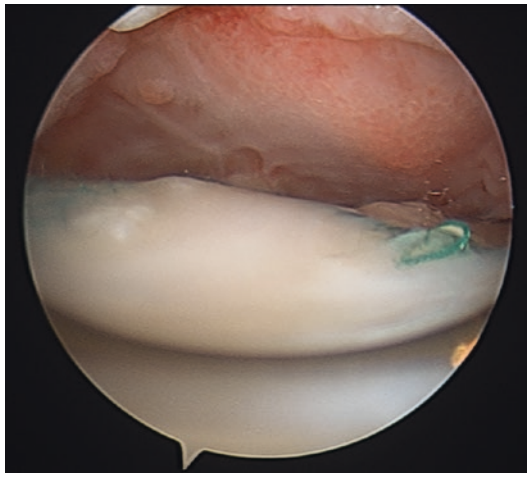


Fig. 38.4 Subacromial view. The patch material is completely enclosed in native tissue. The green material is the reinforcement around the circumference of the patch. Six weeks following the release he had a full pain-free range of motion and was back to work and not and but as a painter and decorator

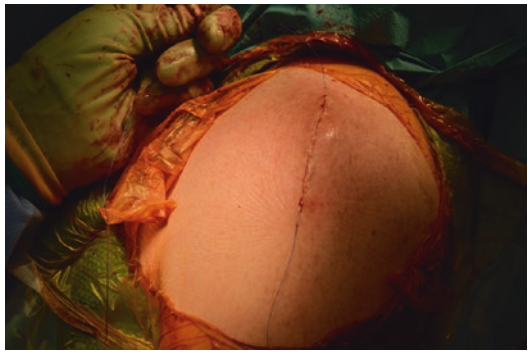


Fig. 38.5 Meticulous closure of deltoid is vital to preserve shoulder function. Subcuticular closure of the skin produces an excellent cosmetic result

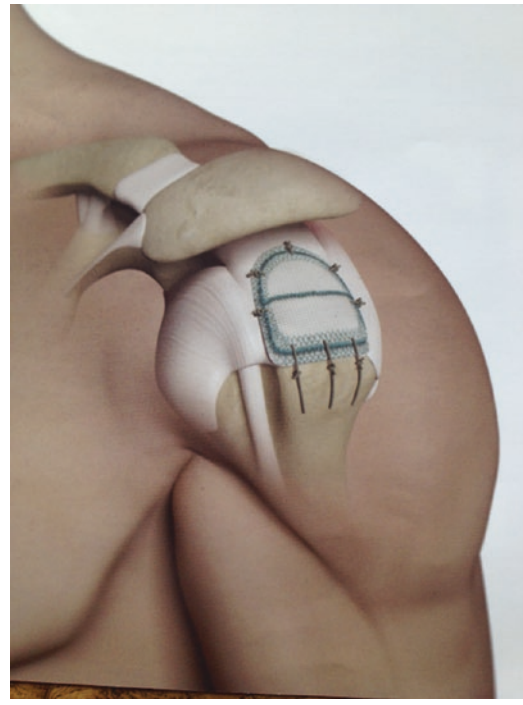


Fig. 38.6 Schematic of use of the Leeds-Kuff Patch (Tm Xiros Leeds UK)

Disclosure The author receives royalties for the Leeds-Kuff Patch not implanted by himself.

References

1. Harryman DT, Mack LA, Wang KY, Jackins SE, Richardson ML, Matsen FA. Repairs of the rotator cuff. Correlation of functional results with integrity of the cuff. J Bone Joint Surg Am. 1991;73:982–9.
2. Carr A, Cooper C, Campbell MK, Rees J, Moser J, Beard DJ, Fitzpatrick R, Gray A, Dawson J, Murphy J, Bruhn H, Cooper D, Ramsay C. Effectiveness of

open and arthroscopic rotator cuff repair (UKUFF): a randomised controlled trial. Health Technol Assess. 2015;19:1–218.
3. Collin P, Yoshida M, Delarue A, Lucas C, Jossaume T, Lädermann A. French Society for Shoulder and Elbow (SOFEC). Evaluating postoperative rotator cuff healing: prospective comparison of MRI and ultrasound. Orthop Traumatol Surg Res. 2015;101:S265–8.
4. De Jesus JO, Parker L, Frangos A, Nazarian LN. Accuracy of MRI, MR arthrography and ultrasound in the diagnosis of rotator cuff tears: a meta-analysis. AJR Am J Roentgenol. 2009;192:1701–7.
5. Vastamäki M, Lohman M, Borgmästars N. Rotator cuff integrity correlates with clinical and functional results at a minimum 16 years after open repair. Clin Orthop Relat Res. 2013;471:554–61.
6. Le BT, Wu XL, Lam PH, Murrell GA. Factors predicting rotator cuff retears: an analysis of 1000 consecutive rotator cuff repairs. Am J Sports Med. 2014;42:1134–42.
7. Goutallier D, Postel J, Bernageau J, Lavau L, Voisin M. Fatty muscle degeneration in cuff ruptures. Pre- and postoperative evaluation by CT scan. Clin Orthop Relat Res. 1994;304:78–83.

39 Case Example 2: Combined Massive Rotator Cuff Tear and Recurrent Shoulder Instability

Benjamin Marjanovič, Marko Nabergoj, and Boris Poberaj

39.1 Introduction

Recurrent shoulder instability which results from a massive rotator cuff tear is uncommon [1]. Massive rotator cuff tear is defined as a complete detachment of at least two tendons [2]. The term shoulder instability is used to refer to the inability to maintain the humeral head in the glenoid fossa. Glenohumeral joint is the most mobile joint in our body. Its significant range of motion is achieved due to the lack of bony constraints which sets the stage for pathologic instability. This balance between shoulder mobility and stability is related to a complex combination of dynamic and static stabilizers around the shoulder joint.

Static stabilizers are as follows:

1. Vacuum effect provided by intracapsular negative pressure, suction effect of the glenoid labrum against humeral head, and adhesion-cohesion effect between two wet surfaces.
2. Bonny geometry: Normally the glenoid is anteverted in relation to the humerus which prevents posterior instability. A loss of that physiological version may affect the stability as we see in glenoid dysplasia.
3. Glenoid labrum increases the glenoid surface, serves as attachment for the glenohumeral ligaments and the long head of biceps, and prevents translation of the humeral head.
4. Glenohumeral capsule works as a socket. Together with glenohumeral ligaments it stabilizes the humeral head in all directions.

Dynamic stabilizers are as follows:

1. Proprioception: Glenohumeral capsule with its receptors sends information to periscapular muscles. In case of stretching of the capsule, the periscapular muscles contract, which prevents dislocation.
2. Rotator cuff (RC) creates concavity compression mechanism which maintains the center of rotation and stabilizes the shoulder at middle range of motion (ROM) when the ligamentous structures are lax and at terminal ROM through muscle activity that limits motion and decreases strain on the glenohumeral ligaments [3].

Disruption of the balance between dynamic and static stabilizers due to their loss of integrity may lead to instability.

Case Example

A 63-year-old female patient presented to our outpatient department was complaining of a right

B. Marjanovič · M. Nabergoj (✉)
Orthopaedic Hospital of Valdoltra, Ankaran, Slovenia
e-mail: Benjamin.marjanovic@ob-valdoltra.si; marko.nabergoj@ob-valdoltra.si

B. Poberaj
Aspetar Orthopaedic and Sports Medicine Hospital, Doha, Qatar

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_39

shoulder pain lasting for a year, which has been worsened after a fall directly on the shoulder 6 days prior. The injury was managed initially by another institution. On the day of the injury she was examined in the emergency department where fracture of the injured shoulder was excluded on plain radiographs. On examination in our hospital she complained of inability to use her right upper limb in everyday activities. She complained of shoulder instability. She had up to five shoulder dislocations per day after the injury. The dislocated shoulder was relocated by herself. Before the injury she had a feeling of instability but no dislocations. Pain was present in every movement of the affected arm. She complained of a shoulder pain at night, which frequently woke her up. Before the recent injury she performed one cycle of physiotherapy (hydrogymnastics, laser therapy, exercises for the shoulder, diadynamic therapy) prescribed by her physiatrist, which was unsuccessful. Four months prior she had an ultrasound examination of her right shoulder, which showed a complete rupture of the supraspinatus tendon with subacromial bursitis. During the examination of her shoulder an atrophy of the supraspinatus muscle region was present. Active anteflexion and abduction was up to 90° but very painful, active retroflexion up to 20°. Jobe test, belly-press test, infraspinatus test, anterior apprehension test, and sulcus sign test were positive. Function of the axillary nerve was intact; no motor or sensory deficits were identified during neurological examination. The neck was well movable and painless. X-ray of the right shoulder was performed (Fig. 39.1).

Computed tomography arthrography (CTA) of her right shoulder showed medium-sized Hill-Sachs lesion, Bankart lesion with continuation along the anterior labrum into a minor superior labrum anterior to posterior (SLAP) lesion, complete rupture of supraspinatus (SSP) tendon with retraction about 1.5 cm laterally from the acromioclavicular joint, partial rupture of infraspinatus (ISP) tendon, moderate atrophy of the SSP

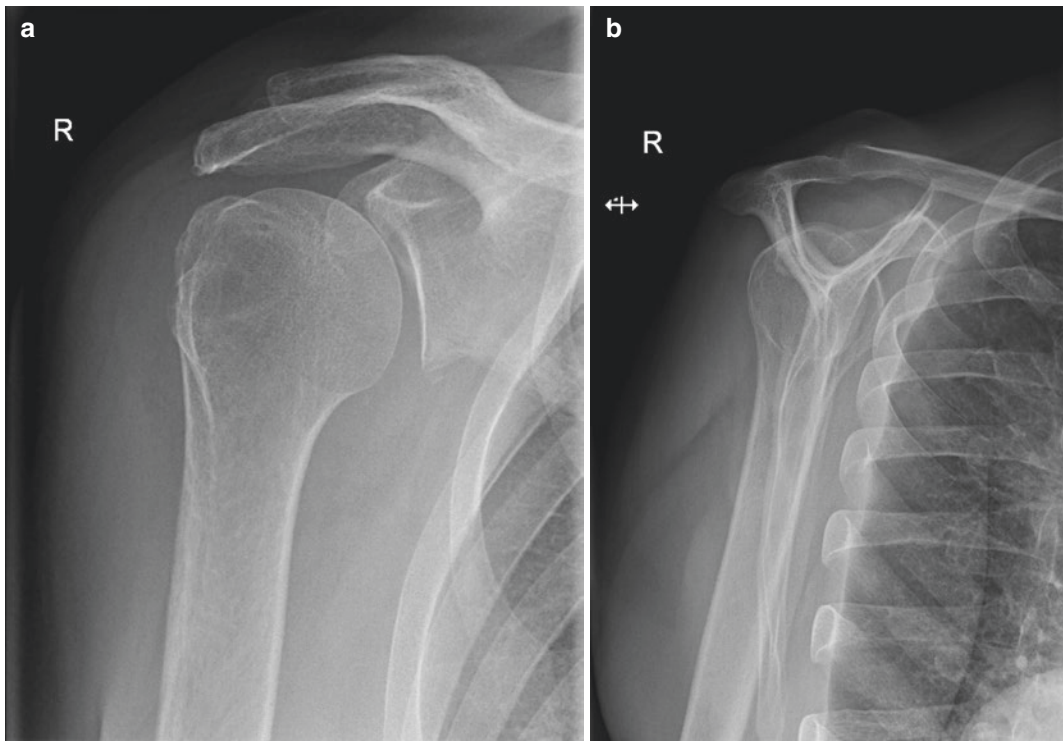


Fig. 39.1 X-ray of the right shoulder with AP view (**a**) and Y view (**b**): A slight cranial translation of the humeral head without glenohumeral arthrosis is seen

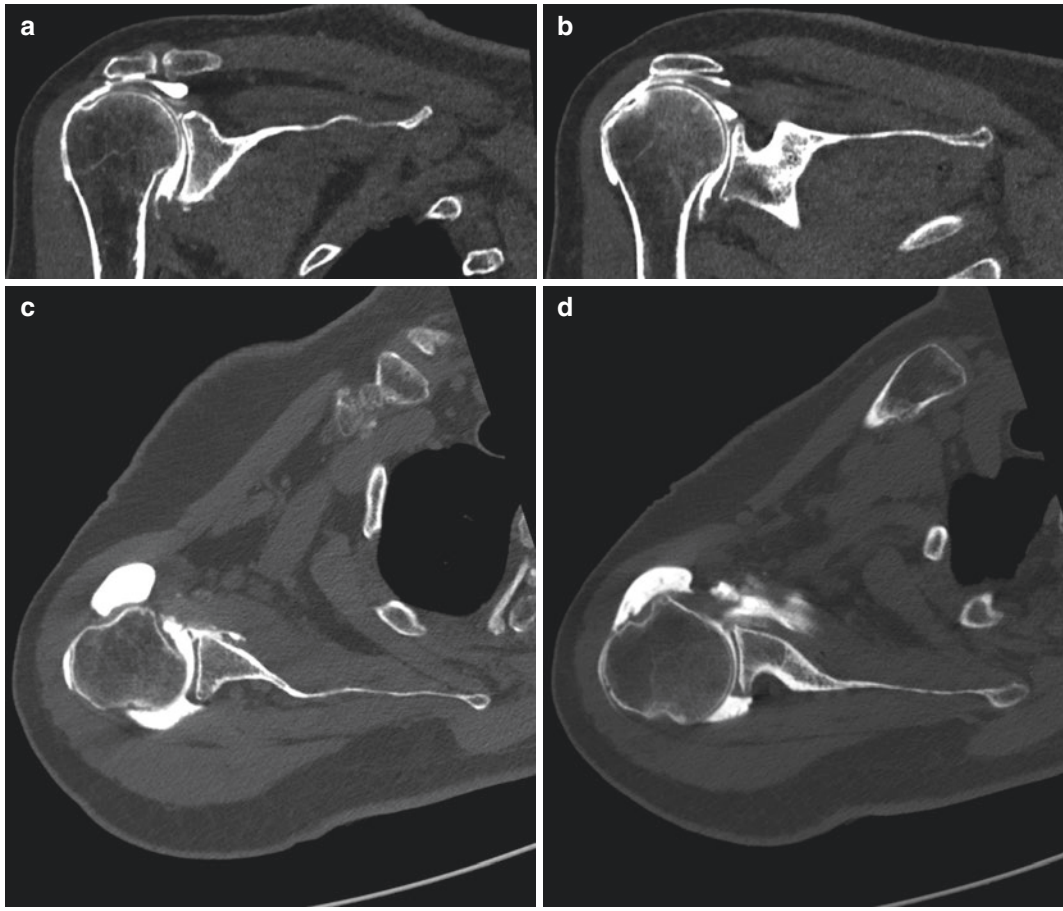


Fig. 39.2 CTA of the right shoulder: Coronal reconstruction shows a minor SLAP lesion (**a**) and a SSP lesion with cranial translation (**b**). On transverse reconstruction a Bankart lesion (**c**) and a Hill-Sachs lesion (**d**) were seen

and ISP muscle, and absence of glenohumeral joint arthrosis (Fig. 39.2).

Due to the evidence of gross instability both an arthroscopic stabilization and RC reconstruction of the injured shoulder was advised. We decided not to implant a reverse total shoulder prosthesis due to the absence of glenohumeral joint arthrosis. Arthroscopy of the shoulder confirmed the radiologically identified pathological lesions and additional finding was a rupture of the upper part of the subscapularis tendon, medially subluxated long head of the biceps tendon, and not a partial but a complete rupture of ISP tendon. During the surgery we performed an arthroscopic Bankart repair, remplissage and reconstruction of the ISP to the footprint, reconstruction of subscapularis, SSP, and long head of the biceps tenotomy. After the surgery the patient started with rehabilitation. Six months postoperatively the patient was satisfied with the result. She had no feeling of instability, no pain in her shoulder with a very good range of motion (Fig. 39.3).

39.2 Discussion

The prevalence of traumatic anterior shoulder dislocations in the elderly population has been increasing due to prolonged life expectancy [1, 4]. Shoulder dislocations in the elderly have a different pathological spectrum with specific surgical implications compared to the younger population. Especially the RC tears have been the

Fig. 39.3 Patient and her ROM 6 months postoperatively

predominant pathological lesion that induce shoulder instability in the elderly population [1]. Robinson et al. have shown that a massive RC tear is one of the most important risk factors for early re-dislocation within a week after a first-time anterior traumatic dislocation [5].

As described earlier one of the main dynamic stabilizers is represented by the RC through concavity compression mechanism. Pouliart et al. have shown in a cadaveric study that smaller capsuloligamentous lesions were needed to lead to dislocation in the presence of RC deficient model [6]. Rowe found similar rates of shoulder dislocations among patients younger or older than 45 years of age [7]. Gumina and Postacchini have reported an incidence of shoulder instability in patients above 60 years of age to be up to 20% of acute anterior dislocations [8]. One of the major differences between shoulder instability in younger and elderly patients is the low recurrence rate in the elderly compared to the high recurrence rate in young population. The percentage of recurrent shoulder dislocations in patients bellow the age of 20 is reported to be between 68 and 95%, whereas in patients older than 60 years between 11 and 31% [9–11].

Shoulder dislocation may result in a RC tear, both in younger sportsmen or older patients with age-related degenerative tendinopathy. Shoulder dislocations in elderly are more commonly associated with neurovascular injuries, fractures, and RC tears. In younger patients, it is thought that anterior dislocation often causes more damage to the anterior capsulolabral support structures.

Whereas in elderly, it has been considered that dislocation results more in a disruption of the posterior support structures such as the posterosuperior RC, while it rarely affects the anterior glenoid labrum and/or capsular structures [12]. Craig popularized the posterior mechanism of instability in a reported series of three patients older than 60 years who had suffered a RC tear combined with recurrent instability after an anterior dislocation. He proposed that recurrent anterior instability may be a result of posterior RC failure [13].

The prevalence of RC tears after an anterior shoulder dislocation is estimated to be between 7 and 32% and is more common with advancing age. The percentage of elderly patients with this comorbidity is 34–100% [1]. This age-related pathophysiology of anterior shoulder dislocation is attributed to deterioration of the structure and mechanical properties of RC tendons [14]. A cadaveric study by Lehman et al. has shown an age-related increase in full-thickness RC tears. In cadavers aged under 60 years the incidence of RC tears was 6% whereas in those above 60 years of age the incidence rose to 30% [15]. Gombera et al. postulated in their systematic review that pain and weakness persisting for up to 3 weeks after an anterior dislocation should set a high suspicion for a RC tear and further investigations should be taken [16].

Once the patient is diagnosed with a combination of shoulder instability and RC lesion, treatment options must be considered. If a patient has a balanced RC lesion, minimal pain and absence of recurrent instability conservative treatment may be considered [17]. However, in patients who continue to suffer from persistent pain and shoulder dysfunction due to recurrent dislocations, surgery may result in less pain and improved function [16].

There is no clear consensus as to whether either the RC tear or the anterior capsulolabral complex injury or both should be treated surgically if they occur simultaneously in a patient with recurrent anterior dislocation of the shoulder. Itoi and Tabata reported a satisfactory outcome in 8 out of 11 (73%) patients when only the RC tear was repaired while the Bankart lesion was left unrepaired [18]. On the other hand, Gumina and Postacchini argued that both RC tear and Bankart lesion must be repaired to achieve satisfactory result [8]. Shin et al. suggested that since massive RC tears alone can induce shoulder joint instability, their repair is sufficient to stabilize the joint, while the Bankart lesion is left unrepaired [19]. In this cases anterior capsulolabral repair does not affect the stability of the joint [1]. However, when the size of RC tear was small to medium, Bankart repair should be performed as well, since small RC tear alone is insufficient to induce instability of the shoulder, which in this case appears to be in a large part caused by the anterior capsulolabral lesion [19].

39.3 Summary

Rotator cuff plays an important role in preventing glenohumeral instability as it stabilizes and centers the humeral head in the glenoid fossa through concavity compression mechanism. Capsulolabral lesion is a very common finding in a dislocated shoulder but a single dislocation event may also disrupt a rotator cuff, which is already weakened from overuse injury in younger athletes and from age-related degeneration in elderly. We must be careful in assessing a patient with persisting pain and weakness up to 3 weeks after an anterior shoulder dislocation and consider further investigations to exclude a rotator cuff injury. Surgical repair of either the rotator cuff, the capsulolabral structure, or both has been proposed in patients with recurrent anterior shoulder dislocation. Currently, there are no definitive answers on which surgical treatments bring the best clinical results.

References

1. Shin S, Ko Y, Lee J. Traumatic anterior shoulder dislocation in elderly patients. Arthrosc Orthop Sports Med. 2016;3(1):24–31.
2. Gerber C, Fuchs B, Hodler J. The results of repair of massive tears of the rotator cuff. J Bone Joint Surg Am. 2000;82(4):505–15.
3. Gasbarro G, Bondow B, Debski R. Clinical anatomy and stabilizers of the glenohumeral joint. Ann Jt. 2017;2:58.

4. Mahishale V. Ageing world: health care challenges. J Sci Soc. 2015;42(3):138–43.
5. Robinson CM, Kelly M, Wakefield AE. Redislocation of the shoulder during the first six weeks after a primary anterior dislocation: risk factors and results of treatment. J Bone Joint Surg Am. 2002;84–A(9):1552–9.
6. Pouliart N, Gagey O. Concomitant rotator cuff and capsuloligamentous lesions of the shoulder: a Cadaver Study. Arthroscopy. 2006;22(7):728–35.
7. Rowe CR. Prognosis in dislocations of the shoulder. J Bone Joint Surg Am. 1956;38–A(5):957–77.
8. Gumina S, Postacchini F. Anterior dislocation of the shoulder in elderly patients. J Bone Joint Surg Br. 1997;79(4):540–3.
9. McLaughlin HL, MacLellan DI. Recurrent anterior dislocation of the shoulder. II. A comparative study. J Trauma. 1967;7(2):191–201.
10. Levy O, Pritsch M, Rath E. An operative technique for recurrent shoulder dislocations in older patients. J Shoulder Elb Surg. 1999;8(5):452–7.
11. Rapariz JM, Martin-Martin S, Pareja-Bezares A, Ortega-Klein J. Shoulder dislocation in patients older than 60 years of age. Int J Shoulder Surg. 2010;4(4):88–92.
12. Paxton ES, Dodson CC, Lazarus MD. Shoulder instability in older patients. Orthop Clin North Am. 2014;45(3):377–85.
13. Craig EV. The posterior mechanism of acute anterior shoulder dislocations. Clin Orthop Relat Res. 1984;(190):212–6.
14. Maier M, Geiger EV, Ilius C, Frank J, Marzi I. Midterm results after operatively stabilised shoulder dislocations in elderly patients. Int Orthop. 2009;33(3):719–23.
15. Lehman C, Cuomo F, Kummer FJ, Zuckerman JD. The incidence of full thickness rotator cuff tears in a large cadaveric population. Bull Hosp Jt Dis. 1995;54(1):30–1.
16. Gombera MM, Sekiya JK. Rotator cuff tear and glenohumeral instability: a systematic review. Clin Orthop Relat Res. 2014;472(8):2448–56.
17. Porcellini G, Caranzano F, Campi F, Pellegrini A, Paladini P. Glenohumeral instability and rotator cuff tear. Sports Med Arthrosc Rev. 2011;19(4):395–400.
18. Itoi E, Tabata S. Rotator cuff tears in anterior dislocation of the shoulder. Int Orthop. 1992;16(3):240–4.
19. Shin S-J, Yoo JC, McGarry MH, Jun BJ, Lee TQ. Anterior capsulolabral lesions combined with supraspinatus tendon tears: biomechanical effects of the pathologic condition and repair in human cadaveric shoulders. Arthroscopy. 2013;29(9):1492–7.

40 Case Example 3: Reverse Arthroplasty Versus Other Treatment Options

Pascal Gleyze, Nikos Tzanakakis, and Konstantina Moraiti

40.1 Introduction

In certain cases, the indications for shoulder surgery have never been clear [1–12]. They have been described as "cuff arthropathy and other clinical cases with a variable combination of arthropathy and cuff lesions." These terms refer to complex and intercorrelated lesions between cuff, cartilage, and impaired shoulder function which lead to a huge variety of consequences.

The analysis of these factors is an important point that we must focus on, in order to set the appropriate surgical treatment. This necessitates the profound understanding of how multiple factors are cumulated to create and contribute to the evolution of (1) the anatomical lesion, (2) the clinical status, and (3) the natural history.

Before decision making and proposing a reverse shoulder arthroplasty (RSA) versus other treatment options, we have to evaluate, for each patient, the actual staging of the lesions and the functional demands of the patient and estimate what could be the evolution for each of the components of this complex pathology: (1) the cuff, (2) the cartilage, and (3) the shoulder function.

P. Gleyze (✉) · N. Tzanakakis
Department of Orthopedic and Trauma Surgery, Centre Hospitalier des 3 Frontières, Saint Louis, France
e-mail: Nikolaos.TZANAKAKIS@CLIN3F.fr

K. Moraiti
Hopital Nord Franche Comté, Trevenans, France

This process takes time, it demands a lot of thinking and experience, and at the end it may be difficult to make the right decision, especially if we choose a treatment without immediate and spectacular results like the reverse shoulder arthroplasty. We are living in a time of "limited efforts" from the patients who are asking for immediate and perfect results [13–15]. Surgeons are also seeking for a great reputation of efficiency and they need to preserve their "image" by all means, while, at the same time, they have to confront the influence of industrial and commercial pressure.

Under all these circumstances, we note nowadays an "overindication" of reverse shoulder arthroplasty. The significant augmentation of the rate of reverse shoulder arthroplasty is not normal according to the population health evolution (from 15 to 64% in France in less than 10 years) [16].

We do think that initially all patients should go through a period of rehabilitation and a cuff repair when possible [4, 17–20]. In addition, all traumatic cases or cases with degenerative arthropathies with preserved cuff should be treated with an anatomic shoulder arthroplasty even if a limited cuff repair should be performed at the same time.

This chapter tries to demonstrate, through several clinical cases, the importance of a thorough clinical examination, with special attention to muscle equilibrium among the shoulder,

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_40

the scapula, and the "cervical-to-shoulder zone." This encounter with the patient is of paramount importance for decision making and it also serves the opportunity to give clear explanation about all the different options that we can propose to him. This sharing time with the patient is precious, especially when the clinical examination and history are not clear, with potential hidden problems that could influence our decision [20].

40.2 Practical Guideline for Decision Making and Treatment Options

1. **What has to be checked as fundamental criteria:**
   (a) *Age, activity, and activity level* of the patient (active or not): Actual and in the future.
   (b) *Muscles:*
      - "Shoulder": Cuff and deltoid muscles, history (sport, hard workers, etc.), state at examination, actual and in the future
      - "Regional compensation": Trapezius muscle, paracervical muscles, scapula stabilizers, etc.
   (c) *Cartilage* of glenohumeral joint but also acromioclavicular joint.
   (d) *Clinical examination:*
      - Mechanic/inflammatory pain
      - Active/passive range of motion
      - Handicap
      - Clear clinical conflict (precise painful point in adduction internal rotation test)
      - Analytic muscular testing
2. **What can we propose to the patient according to the combination of anatomical state, age, and activity level?**
   (a) Nothing, if no pain, no functional limitation, and no danger for future degeneration
   (b) Rehabilitation and self-rehabilitation exercises with precise, intensive, and realistic protocol
   (c) Acromioplasty with or without partial or complete cuff repair
   (d) Patch, graft, or spacer techniques
   (e) Surface, anatomic, or reverse arthroplasty

A precise analysis of mentioned factors, giving the time needed to propose and explain to the patient the benefits and risks for each treatment option, and of course mastering all of the possible surgical techniques are fundamental for a good medical practice and decision making. Proposing in every case a RSA will only mask our deficiency.

RSA for everybody will give satisfaction to all but the price will be paid later on. Specifically, the biomechanical constrain of the prosthesis design, the definitive sacrifice of residual cuff, and the bone capital lost will make easy to jump from heaven to hell if anything during and/or after the procedure fails and especially if the functional demand of the patient is over the limited biomechanical service given by the implant.

Irreparable cuff tear with severe arthropathy for patient with limited functional demand is the actual absolute indication for RSA as defined by scientific societies and should be followed.

40.3 Clinical Case 1: When We Do Think We Have No Choice

1. **Presentation of the case:**
   (a) *Profile:* Male, 66 years old
   (b) *Medical history:* 2-year evolution with suspected limited cuff rupture (supraspinatus) and 4 months limited active ROM/pain medium/important handicap/constant score (36/100). Pseudoparalytic shoulder
   (c) *Prior treatment:* Rehabilitation 4 months
   (d) *Clinical examination:*
      - Pain: Limited during the day/no pain at night
      - Handicap: Major handicap
      - Active range of forward flexion: 70°
      - Passive range of forward flexion: 170°
      - Clinical anterior superior conflict: No
      - Muscular testing:
        – Supraspinatus: OK (deltoid compensation)

  - Infraspinatus: OK
  - Subscapularis: OK

(e) *Muscles:*
- *Cuff and deltoid:* Massive rotator cuff tear of supraspinatus (fatty infiltration stage 3) + infraspinatus (fatty degeneration stage 2) + subscapularis (no fatty degeneration)
- *Regional compensation:* Major compensation, trapezius, and paracervical (permanent contraction/trumpet sign and pain on the muscular bodies)

(f) *Cartilage:* No arthropathy, centered, not medialized/limited superior translation (Visotsky 1A/Hamada 1/Samilson/Sirveaux E0)

(g) *Synthesis of the case:* Pain, limited function, major compensation, no arthrosis. Failure of rehabilitation

(h) *Arthro-CT scanner* (Fig. 40.1)

2. **Therapeutic options:**
   (a) Rehabilitation and intensive self-rehabilitation (2–3 h a day)
   (b) Acromioplasty ± partial repair
   (c) Reverse shoulder arthroplasty
3. **Therapeutic decision:**
   (a) My first decision: Continuing rehabilitation with addition of intensive self-rehabilitation/3 months (more than 2 h a day reported on special following document). However, failure of this first option: absolutely no improvement on pain, handicap, ROM.
   (b) My second decision after those 3 months of intensive self-rehabilitation and physiotherapy: Acromioplasty and partial repair. Unfortunately, no partial repair was possible during the procedure. Only acromioplasty was performed. Results: Stiffness and no pain at 3 months, com-

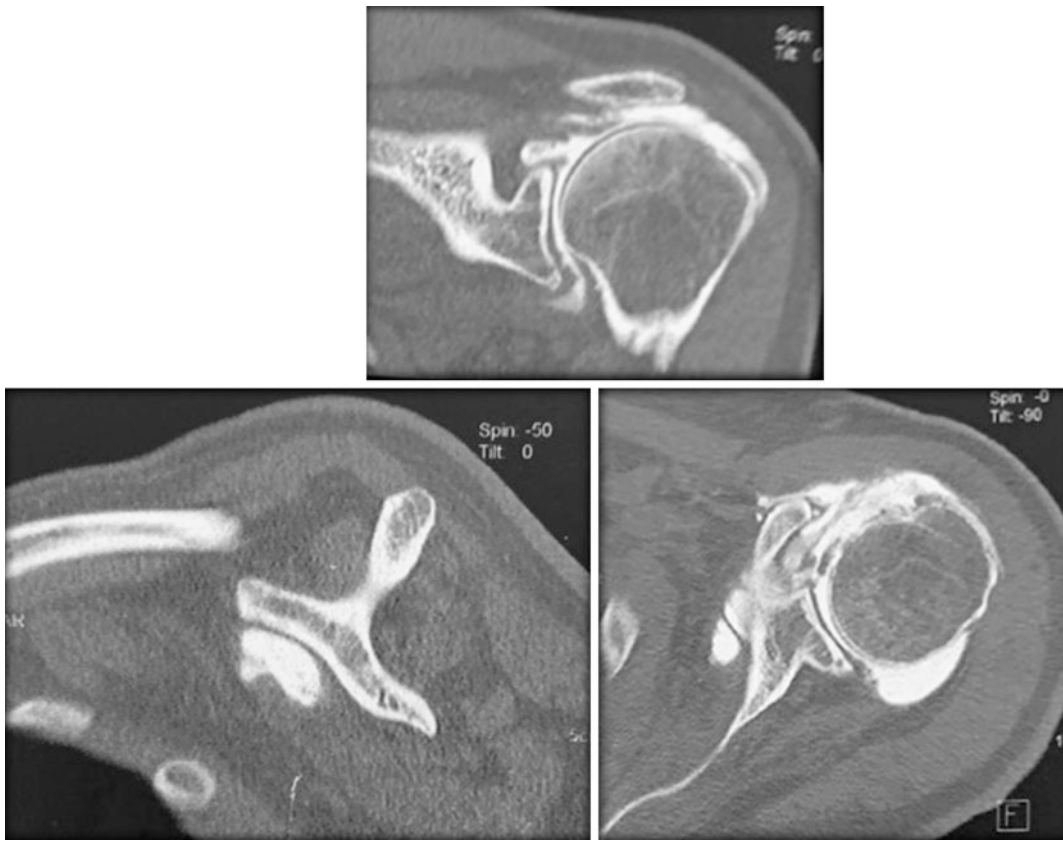


Fig. 40.1 Case 1; CT arthrogram of the patient showing massive rotator cuff tear with reduced acromiohumeral distance

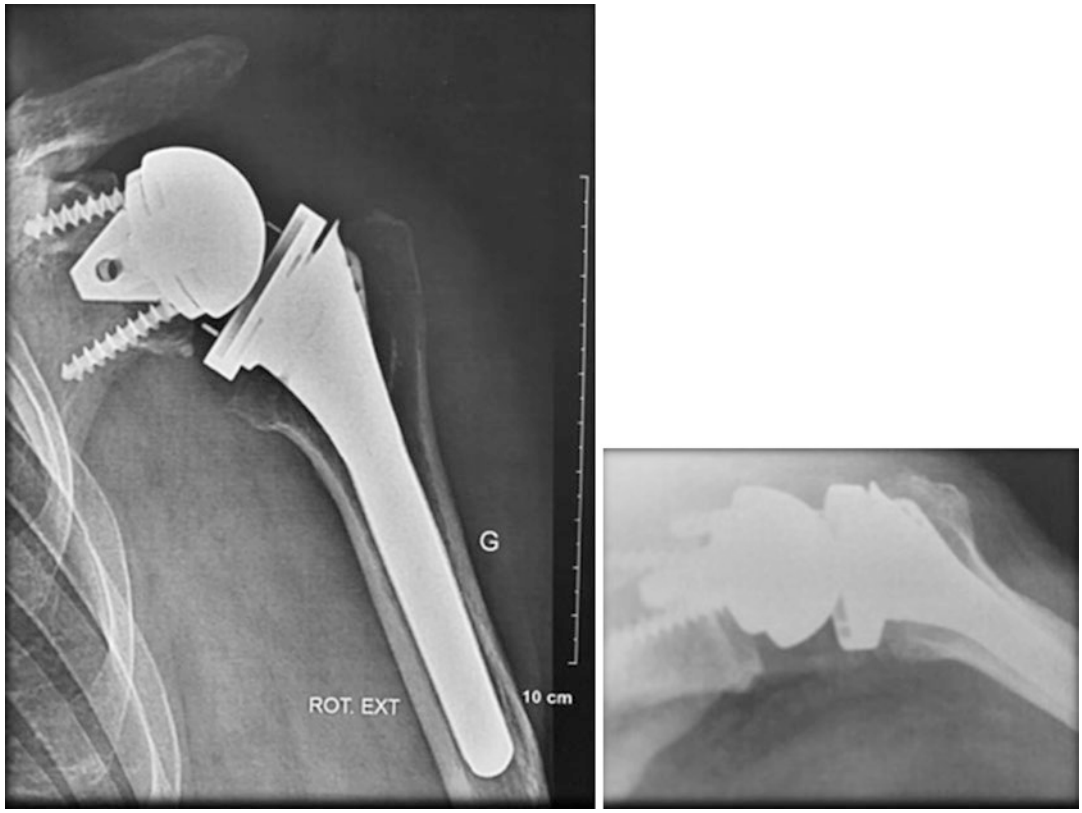


Fig. 40.2 Case 1; x-ray of the shoulder after surgical treatment with reverse shoulder arthroplasty

plete passive range of motion at 6 months, no pain but still pseudoparalytic shoulder with major trapezius compensation.

(c) My third decision 9 months after acromioplasty: Reversed shoulder prosthesis, no operative difficulty (Fig. 40.2).

4. **Final results:** *the clinical outcome in* 3 months = 6 months = revisions ⇒ bad result. Moderate/no pain night and day with an active ROM below 70° with still a major regional compensation and important handicap. Ten points of Constant score improvement. The clinical examination was equivalent as it was the first time the patient came (Fig. 40.3).
5. **Analysis:**
   (a) The initial clinical image was due to a massive cuff tear with a permanent and major compensation. Antalgic reflex position create "regional muscle compensation" and is always present when shoulder pain after trauma, recent cuff rupture, or other etiology but should disappear with the use of the shoulder, rehabilitation protocols, self-control of the trapezius, and over time. Sometimes it persists and becomes part of the spontaneous using of the shoulder as new "corporeal image."

(b) This "compensation" reflex stabilizes the shoulder and blocks the function of the residual cuff muscles and deltoid. Forward flexion and abduction are not possible when trapezius and paracervical muscles are contracted because the shoulder moves into the scapulothoracic joint and oblige to spine adaptation. The "trumpet sign" shows this regional compensation. This clinical sign is a consequence of the massive cuff tear but can also be a causality of the impossible active range of motion when this reflex is maintained by the patient as a systematic antalgic reflex day after day.

(c) This explains why the patient's shoulder was stiff, why it was not moving before

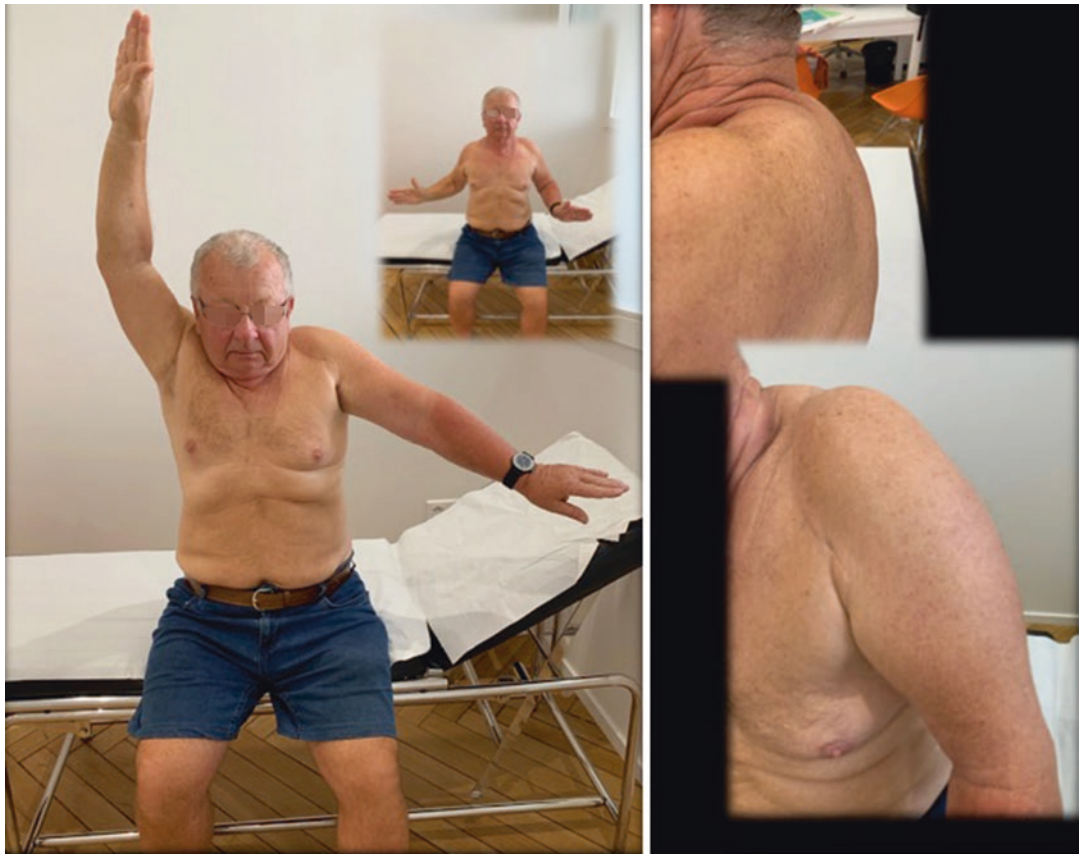


Fig. 40.3 Case 1; final clinical examination 18 months after surgical treatment with reverse shoulder arthroplasty showing the same range of motion as preoperatively

the prosthesis, and why its function was not improved after treatment: it was just due to the persistence of this antalgic reflex.

(d) We can state that RSA for this patient was not a good option because he had no arthropathy and because the handicap and limited function was only due to the "compensation reflex" and not to absence of the cuff. Acromioplasty was not a good option either because he had no clinical conflict either because the endoscopic reparation during the procedure was not possible.

(e) What could help us make the right decision? The key point is to know the exact potential of the residual cuff and deltoid. This can be done by evaluating the physiologic age of the patient and the status of the muscles (trophicity and fatty degeneration) [12, 17, 21]. A combination of MRI or CT scan imaging with a precise description of the fatty degeneration of each muscle is vital specially when it is possible to individualize the state of the superior and inferior part of the infraspinatus. The fatty degeneration of the inferior part of the infraspinatus will determine the "red line frontier." If it is preserved, the patient should be able to move full ROM specially if he has no significant arthropathy. From a biomechanical point of view, the preserved inferior part of the infraspinatus in combination with an efficient subscapularis and a good deltoid is enough to have full range of

motion in most cases. Further posterior extension of the rupture can be accepted as the limit that will give us the decision for RSA.

6. **Conclusion:** No arthropathy with a residual inferior part of the infraspinatus and a good deltoid should be able to allow to the patient full range of motion with no pain or limited pain and no handicap or limited handicap.
   (a) If a clear clinical conflict sign is present and can explain the pain, acromioplasty with ± partial cuff repair could be proposed.
   (b) If there is no pain, the educational control of the compensation should be enough to make this shoulder recover without pain, full range of motion, and limited handicap in correlation with limited muscular forces.
   (c) Natural evolution of this patient's pathology will be a progressive severe cuff arthropathy. But due to the fact that it is difficult to precisely know and explain to the patient when and how this cuff arthropathy will oblige him to a RSA, the only solution is to have regular follow-ups and to propose RSA when the time comes, which can be in 10 years' time. This will then be a justified surgical procedure with an adequate benefit risk ratio. *Prevention arthroplasty is not acceptable* [22].
   (d) In the case presented, this is no hope for favorable evolution for the glenohumeral joint, with or without arthroplasty, if the corporeal image of the patient is to lock the shoulder before starting any movement.

40.4 How to Define the "Red Line Criteria" for RSA Indication

1. **Case 2: Good result with rehabilitation**
   Male, 72 years old, active profile.

   Well-known old cuff tear (supraspinatus) for 4 years. Fall on the shoulder 4 months ago and pseudoparalytic shoulder, "trumpet sign," and limited stiffness in forward flexion (130°) and external rotation (20°). On arthro-CT scan a diagnosis of massive cuff tear was made with fatty degeneration grade 3 for supraspinatus and with a preserved posterior part of infraspinatus. Eccentric arthropathy was present as well (Fig. 40.4).

   The patient already had three proposals of RSA. At the time of the first consultation, he had already lost his faith in regaining his shoulder function.

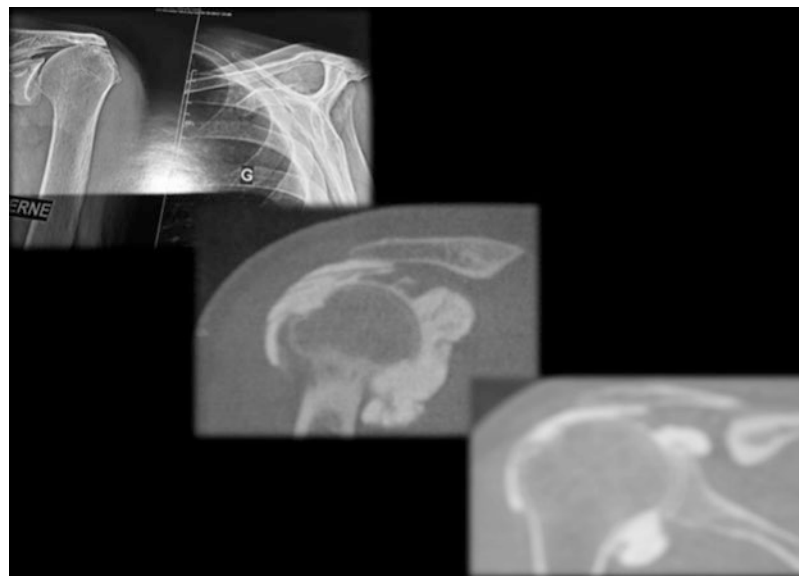


Fig. 40.4 Case 2; x-ray of the shoulder and CT arthrogram preoperatively

(a) We proposed intensive rehabilitation and self-stretching over the pain limits. The patient has followed this protocol with more than 2 h per day over the pain limits: pain-free and complete recover after 3 months with a preserved perfect clinical result and no arthropathy evolution 5 years after (Fig. 40.5).

(b) This clinical case is exactly the same as the prior case except that he had arthropathy, but the results are finally very good because the patient achieved a good shoulder mobility.

2. **Case 3: Temporally limited results due to insufficiency of the rehabilitation and self-rehabilitation exercises**

Female, 66 years old, not an active profile, no stiffness, limited centered arthropathy, pain night and day, and important handicap.

No compensation but no real self-rehabilitation work, bad results after 3 months, and still in depressed psychological condition. What to do?

(a) We asked her to continue the rehabilitation protocol and we explained to her that she was in good progress and that she had only to pass horizontal elevation.

(b) In general, if the patient is able to maintain the arm over horizontal level, he should be able to gain full active range of motion if he works hard on rehabilitation and self-rehabilitation. Our patient, 6 weeks after the consultation, recovered full ROM and had no pain night and day. This good clinical result is still preserved, after 2 years (Fig. 40.6).

3. **Case 4: No need of a doctor, don't touch me**

Male, 75 years old, very active profile.

Fig. 40.5 Case 2; clinical examination of the patient before treatment and after 3 months following intensive rehabilitation

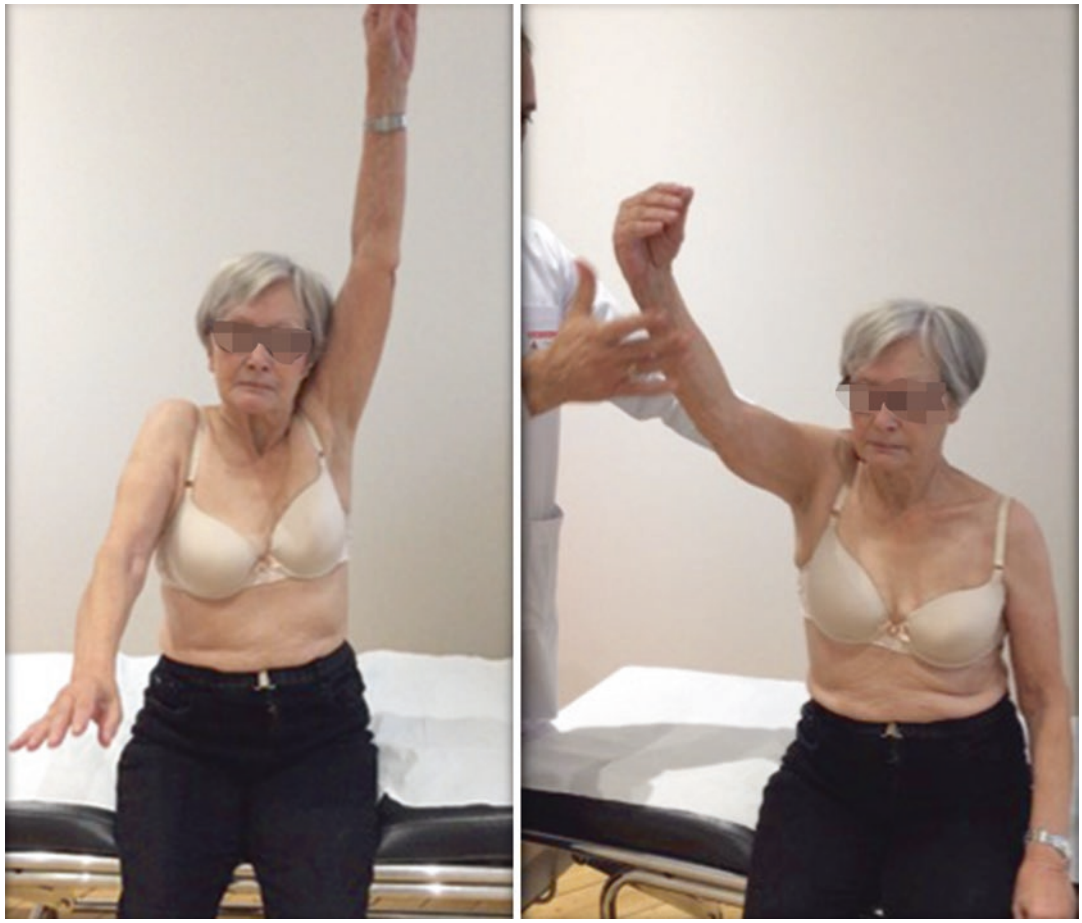


Fig. 40.6 Case 3; clinical presentation of the patient at first follow-up at 6 weeks following self-rehabilitation protocol

Massive cuff tear involving the posterior part of the infraspinatus. Eccentric arthropathy. Limited passive ROM.

(a) No pain, no handicap (as described by the patient). "No doctors needed"; the patient consulted for the knee. So, no treatment and a happy active guy (Fig. 40.7).

4. **Case 5: Acromioplasty and partial repair of post part of infraspinatus**

Male, 68 years old, active, rupture of the supraspinatus more than 5 years ago. Due to an excessive effort 3 months ago, he presented a complete supra- and infraspinatus rupture (no fatty degeneration of the infraspinatus). Eccentric arthropathy. Pseudoparalytic shoulder.

(a) The patient had acromioplasty and repair of the infraspinatus, no repair of the supraspinatus (fatty degeneration).

(b) Clinical result at 3 months, no pain, full ROM, and limited force in external rotation (Fig. 40.8).

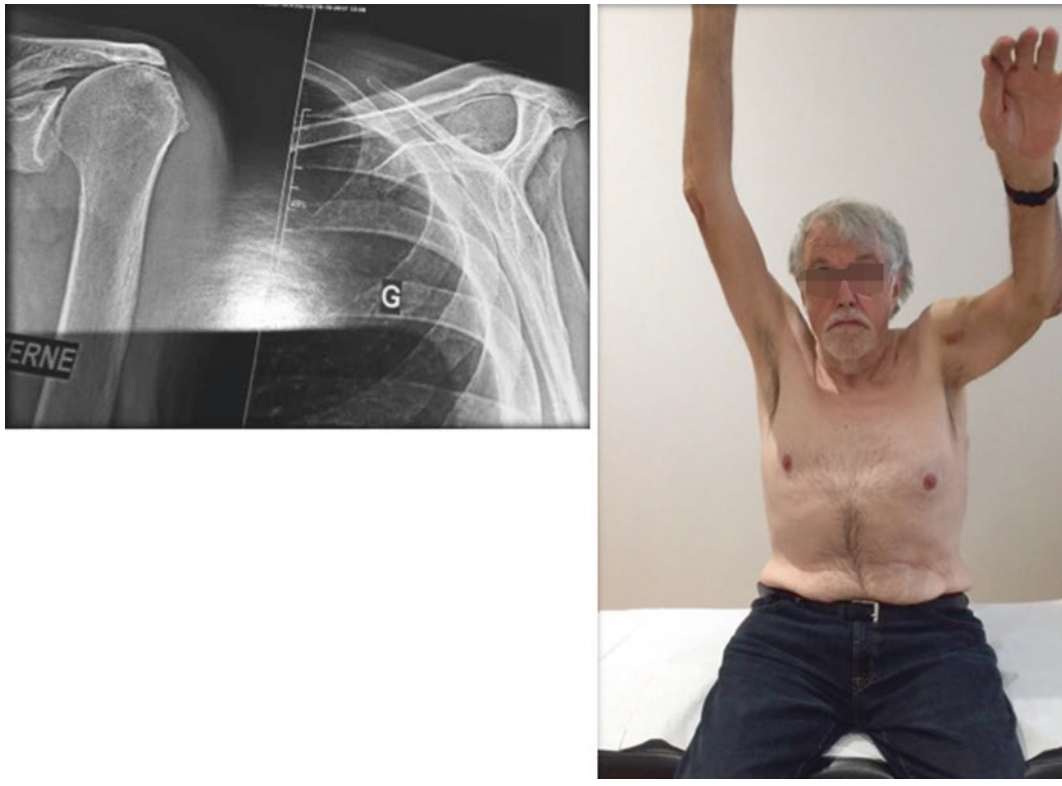


Fig. 40.7 Case 4; patient with massive rotator cuff tear but preserved shoulder function and without pain

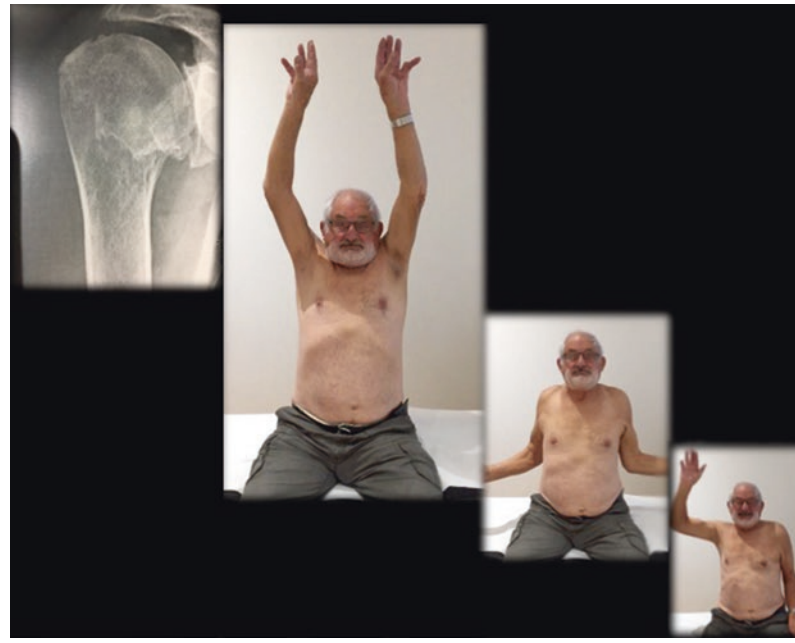


Fig. 40.8 Case 5; clinical result in a patient with massive rotator cuff tear and eccentric arthropathy 3 months after treatment with partial repair, subacromial debridement, and acromioplasty

40.5 Summary

We must never propose any surgical solution before we are sure that we have reached the far end of what nature can give. This means that we have to insist on muscle reinforcement, stretching, self-exercise, and physiotherapy on a very intensive way for a minimum of 4 weeks before making the decision of RSA or not. And we should be aware of the compensation reflex which may mask the real potential of the shoulder.

References

1. Walch G, Bacle G, Lädermann A, Nové-Josserand L, Smithers CJ. Do the indications, results, and complications of reverse shoulder arthroplasty change with surgeon's experience? J Shoulder Elb Surg. 2012;21(11):1470–7.
2. Mulieri P, Dunning P, Klein S, Pupello D, Frankle M. Reverse shoulder arthroplasty for the treatment of irreparable rotator cuff tear without glenohumeral arthritis. J Bone Joint Surg Am. 2010;92(15):2544–56. https://doi.org/10.2106/JBJS.I.00912.
3. Petrillo S, Longo UG, Papalia R, Denaro V. Reverse shoulder arthroplasty for massive irreparable rotator cuff tears and cuff tear arthropathy: a systematic review. Musculoskelet Surg. 2017;101(2):105–12. https://doi.org/10.1007/s12306-017-0474-z.
4. Allert JW, Sellers TR, Simon P, Christmas KN, Patel S, Frankle MA. Massive rotator cuff tears in patients older than sixty-five: indications for cuff repair versus reverse total shoulder arthroplasty. Am J Orthop (Belle Mead NJ). 2018;47(12). https://doi.org/10.12788/ajo.2018.0109.
5. Boileau P, Sinnerton RJ, Chuinard C, Walch G. Arthroplasty of the shoulder. J Bone Joint Surg Br. 2006;88(5):562–75.
6. Erickson BJ, Bohl DD, Cole BJ, Verma NN, Nicholson G, Romeo AA, Harris JD. Reverse total shoulder arthroplasty: indications and techniques across the world. Am J Orthop (Belle Mead NJ). 2018;47(9). https://doi.org/10.12788/ajo.2018.0079.
7. Lindbloom BJ, Christmas KN, Downes K, Simon P, McLendon PB, Hess AV 2nd, Mighell MA, Frankle MA. Is there a relationship between preoperative diagnosis and clinical outcomes in reverse shoulder arthroplasty? An experience in 699 shoulders. J Shoulder Elb Surg. 2019;28(6S):S110–7. https://doi.org/10.1016/j.jse.2019.04.007.
8. Frankle M, Siegal S, Pupello D, Saleem A, Mighell M, Vasey M. The reverse shoulder prosthesis for glenohumeral arthritis associated with severe rotator cuff deficiency. A minimum two-year follow-up study of sixty patients. J Bone Joint Surg Am. 2005;87(8):1697–705.
9. Wall B, Nové-Josserand L, O'Connor DP, Edwards TB, Walch G. Reverse total shoulder arthroplasty: a review of results according to etiology. J Bone Joint Surg Am. 2007;89(7):1476–85.
10. Cuff D, Pupello D, Virani N, Levy J, Frankle M. Reverse shoulder arthroplasty for the treatment of rotator cuff deficiency. J Bone Joint Surg Am. 2008;90(6):1244–51. https://doi.org/10.2106/JBJS.G.00775.
11. Nolan BM, Ankerson E, Wiater JM. Reverse total shoulder arthroplasty improves function in cuff tear arthropathy. Clin Orthop Relat Res. 2011;469(9):2476–82. https://doi.org/10.1007/s11999-010-1683-z.
12. Ek ET, Neukom L, Catanzaro S, Gerber C. Reverse total shoulder arthroplasty for massive irreparable rotator cuff tears in patients younger than 65 years old: results after five to fifteen years. J Shoulder Elb Surg. 2013;22(9):1199–208. https://doi.org/10.1016/j.jse.2012.11.016.
13. Daw RLC, Gibson J, Prescot D, Bonnett L, Smith M. What is the correlation between patient-reported outcome measure (PROM) scores and patient satisfaction following elective reverse total shoulder replacement? Shoulder Elbow. 2019;11(2 Suppl):42–7. https://doi.org/10.1177/1758573217744178. Epub 2017 Dec 14.
14. Rauck RC, Swarup I, Chang B, Ruzbarsky JJ, Dines DM, Warren RF, Henn RF 3rd, Gulotta LV. Preoperative patient expectations of elective reverse shoulder arthroplasty. J Shoulder Elb Surg. 2019;28(7):1217–22. https://doi.org/10.1016/j.jse.2018.12.008. Epub 2019 Mar 20.
15. MacInnes SJ, Mackie KE, Titchener A, Gibbons R, Wang AW. Activity following reverse total shoulder arthroplasty: what should surgeons be advising? Shoulder Elbow. 2019;11(2 Suppl):4–15. https://doi.org/10.1177/1758573218793648. Epub 2018 Aug 8. Review.
16. Rapport DREES—Ministère des Solidarités et de la Santé—La santé des Français en 2017, Direction de la Recherche des Etudes de l'Evaluation et des Statistiques.
17. Gladstone JN, Bishop JY, Lo IK, Flatow EL. Fatty infiltration and atrophy of the rotator cuff do not improve after rotator cuff repair and correlate with poor functional outcome. Am J Sports Med. 2007;35(5):719–28. https://doi.org/10.1177/0363546506297539.
18. Gerber C, Fuchs B, Hodler J. The results of repair of massive tears of the rotator cuff. J Bone Joint Surg Am. 2000;82(4):505–15. https://doi.org/10.2106/00004623-200004000-00006.
19. Denard PJ, Ladermann A, Jiwani AZ, Burkhart SS. Functional outcome after arthroscopic repair of massive rotator cuff tears in individuals with pseudoparalysis. Arthroscopy. 2012;28(9):1214–9. https://doi.org/10.1016/j.arthro.2012.02.026.

20. Werner BC, Wong AC, Mahony GT, Craig EV, Dines DM, Warren RF, Gulotta LV. Causes of poor postoperative improvement after reverse total shoulder arthroplasty. J Shoulder Elb Surg. 2016;25(8):e217–22. https://doi.org/10.1016/j.jse.2016.01.002.
21. Goutallier D, Postel JM, Gleyze P, Leguilloux P, Van Driessche S. Influence of cuff muscle fatty degeneration on anatomic and functional outcomes after simple suture of full-thickness tears. J Shoulder Elb Surg. 2003;12(6):550–4. https://doi.org/10.1016/S1058-2746(03)00211-8.
22. Hartzler RU, Steen BM, Hussey MM, Cusick MC, Cottrell BJ, Clark RE, Frankle MA. Reverse shoulder arthroplasty for massive rotator cuff tear: risk factors for poor functional improvement. J Shoulder Elb Surg. 2015;24(11):1698–706. https://doi.org/10.1016/j.jse.2015.04.015.

41 Case Example 4: Massive Rotator Cuff Tear and Patient-Specific Rehabilitation in Sportsmen

Ettore Taverna, Vincenzo Guarrella, Baldo Arcuri, and Marianna Vitale

41.1 Case Report

An otherwise healthy 40-year-old right-hand-dominant male, agonist climber, came to our attention complaining about weakness and pain in his right shoulder due to fall on an outstretched hand while climbing 1 month before. He noted pain in the anterior and superior aspect of his dominant right shoulder exacerbated with overhead activity. He denied previous trauma, instability, or associated symptoms. Radiographs that were made after the injury were negative for bone fracture.

Clinical examination revealed an active range of motion (ROM) significantly decreased with a painful forward elevation of 100°, painful abduction of 100°, internal rotation of L5, and external rotation with the elbow at the side of 20°. Passive ROM was complete. Rotator cuff testing showed severe weakness on Jobe and Whipple tests and lift-off and belly-press tests were also positives. No signs of glenohumeral instability were detected. Neck evaluation showed a pain-free complete range of motion. Patient's posture evaluation pointed a thoracic kyphotic posture and forward head posture.

MRI discloses a high-grade, full-thickness tear of supraspinatus tendon without gross retraction, diffuse enlargement, and edema of subscapularis tendon consistent with tendinosis (Figs. 41.1 and 41.2). AC joint synovitis narrowing the subacromial space was also detected.

The patient was initially managed conservatively (analgesics, anti-inflammatory drugs, rehabilitation, and subacromial corticosteroid injections); nevertheless, the symptoms failed to resolve and the patient continued to complain weakness and pain in the involved shoulder. Therefore, surgical repair was offered, which was subsequently performed.

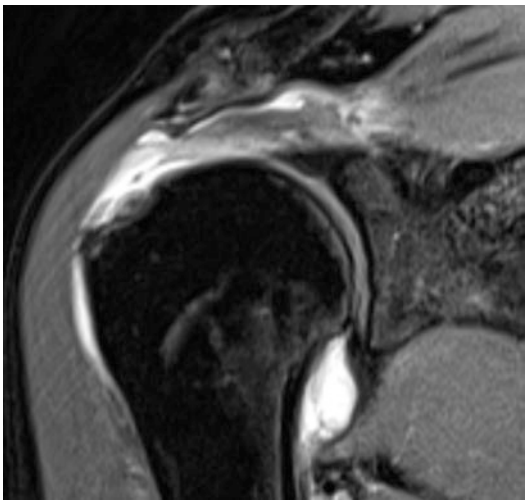


Fig. 41.1 MRI in coronal plane showing full-thickness tear of supraspinatus tendon without marked retraction

E. Taverna · V. Guarrella (✉)
IRCCS Istituto Ortopedico Galeazzi, Milan, Italy
e-mail: ettore.taverna@eoc.ch

B. Arcuri · M. Vitale
Universita' degli Studi di Milano, Milan, Italy

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_41

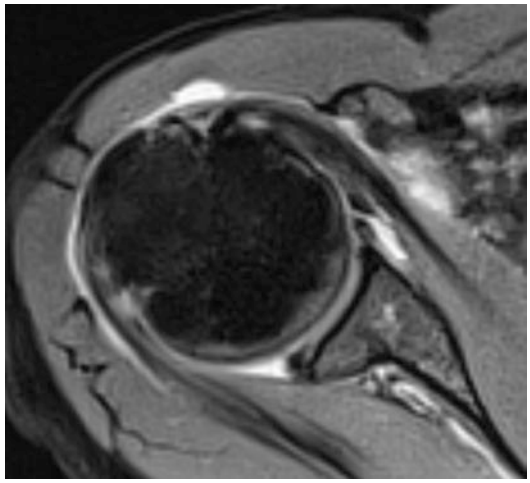


Fig. 41.2 MRI in transverse plane showing enlargement and edema of subscapularis tendon consistent with tendinosis

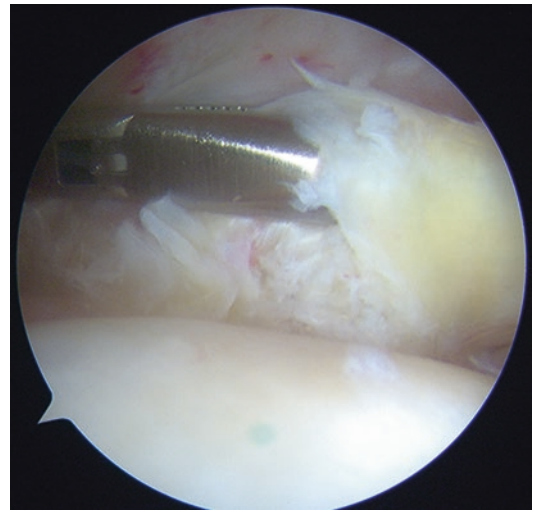


Fig. 41.3 Arthroscopic view through standard arthroscopic portal showing a large rotator cuff lesion involving the supraspinatus and the upper part of the subscapularis tendon

41.2 Surgical Treatment

Glenohumeral examination through standard arthroscopic portals showed a large rotator cuff lesion involving the supraspinatus with the upper part of the subscapularis tendon involvement (Fig. 41.3). A titanium double-loaded anchor was inserted into the lesser tuberosity and the sutures were passed through the subscapularis tendon which was repaired with lasso loop knots (Fig. 41.4). Tenodesis of the long head of the biceps that appears medially subluxated was also performed. No other intra-articular lesions were detected.

Subacromial examination showed signs of subacromial impingement in the presence of reactive bursitis. Partial bursectomy with radio frequency and motorized shaver was performed. A minimal anterolateral acromioplasty with a burr was also performed.

Full-thickness rupture of the supraspinatus was shown (Fig. 41.5). After tendon and foot print preparation the superior cuff was repaired with two triple-loaded titanium anchors inserted on the greater tuberosity and sliding knots (Fig. 41.6).

The dynamic test showed the good coverage of the humeral head. After portal closure and medication an abduction sling was positioned.

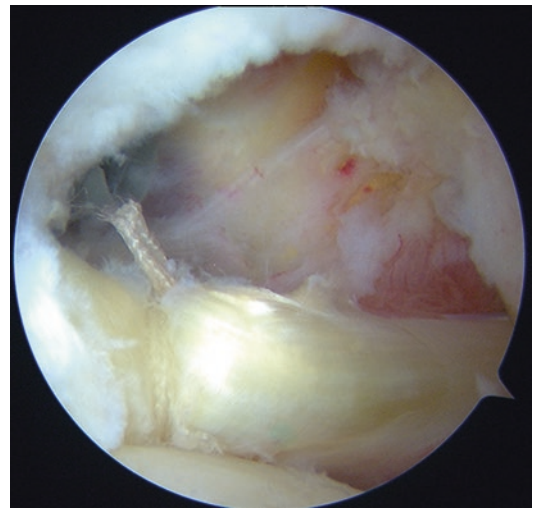


Fig. 41.4 Subscapularis repair on a titanium double-loaded anchor with lasso loop knots

41.3 Rehabilitation

Before surgery, the patient was informed in details about the duration, structure, and content of the rehabilitation measure.

Basically, the rehabilitation process of the shoulder was divided into different phases

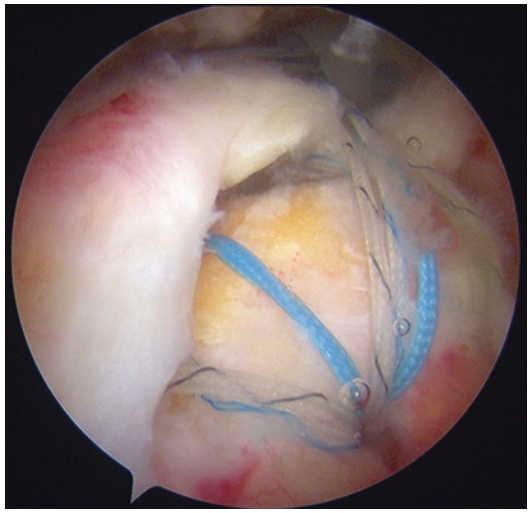


Fig. 41.5 Full-thickness rupture of the supraspinatus. Tendon and foot print are prepared, anchor is inserted in greater tuberosity, and sutures are passed through tendon

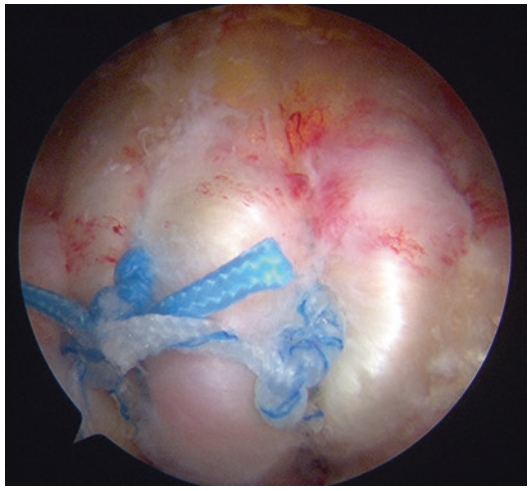


Fig. 41.6 Final view on rotator cuff—supraspinatus repair with two triple-loaded titanium anchors inserted on the greater tuberosity and sliding knots

depending on the functional limitations and the degree of soft tissue irritation. In the course of these phases, a consistent medical examination of the current state of the patient was performed in order to adapt the rehabilitation measures according to the healing process. Rehabilitation should always be pain-free [4].

41.3.1 Phase 1, Acute Phase

In the acute phase, which begins the day after surgery and lasts 3 weeks, the arm was kept in a 20° abduction sling and the patient was instructed not to start physiotherapy to lower the risk of cuff retear. Elbow, hand, and wrist motion was permitted immediately after surgery and performed independently at home. Pain management was based on cryotherapy and oral analgesics.

Two weeks postoperatively, the wound was considered well healed without signs or symptoms of infection. Cutaneous stiches were removed at this stage. In order to avoid contractures and adhesions, the patient was advised to start gentle passive pendulum exercises but was still kept in the abduction sling.

41.3.2 Phase 2, Early Convalescence Phase

In this phase, the target of our protocol was full regain of passive range of motion. The abduction sling was removed. The physiotherapist initiated gentle manual resistance for scapular protraction-retraction and elevation-depression and gentle, submaximal glenohumeral isometrics in all planes. By 8 weeks from surgery, the athlete had achieved a passive range of motion of 140° of forward flexion (Fig. 41.7), 130° of abduction, 80° of external rotation with the elbow at the side, and L5 of internal rotation (Fig. 41.8). The athlete still complained of anterior shoulder pain, typically at end range of motion of internal rotation.

41.3.3 Phase 3, Late Convalescence Phase

In the late convalescence phase, the focus is on active mobilization, improvement of coordination, and strengthening. From the 8th to 12th week, closed kinematic chain exercises started with glenohumeral centering and stabilization through careful concentric and eccentric exercises. It was emphasized the rebuilding and

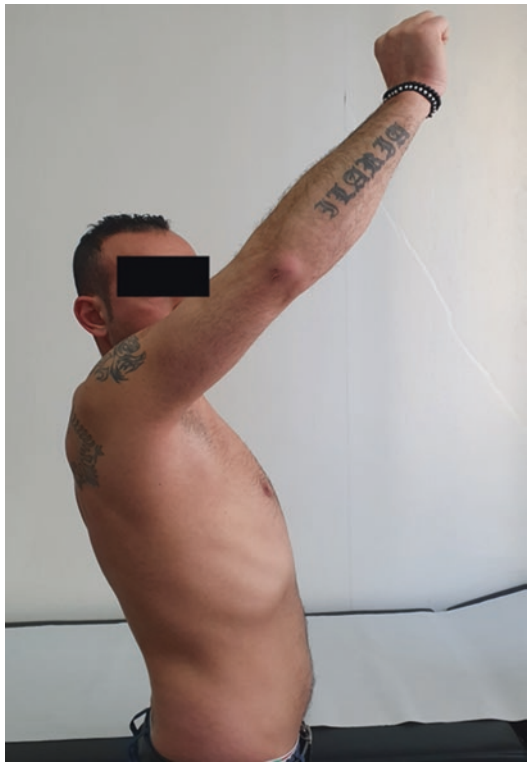


Fig. 41.7 Functional result of the athlete 8 weeks from the surgery with 140° of forward flexion

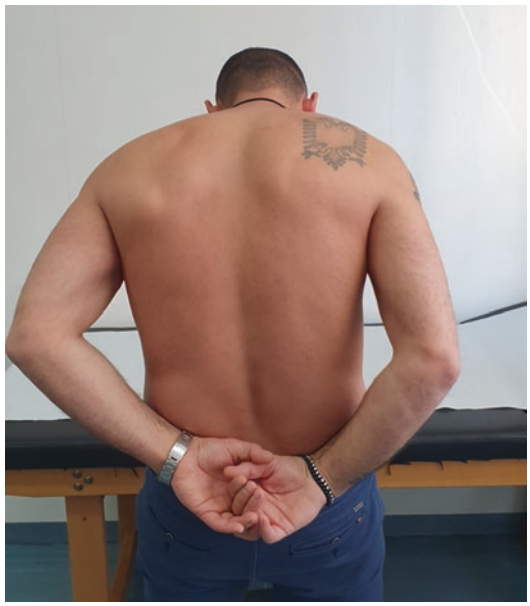


Fig. 41.8 Internal rotation of the patient 8 weeks after surgical treatment

strengthening of the trunk muscles (mm. transversus abdominis, multifidi, and rotatores) as well as the progressive isotonic muscle building within the framework of the kinetic chain. By the end of this phase the patient was pain-free in normal daily activities.

41.3.4 Phase 4, Functional Phase

After 3 months the functional phase of rehabilitation was started. Sports-specific exercises were planned; the endurance was trained by increasing the repetitions against lower resistances. At 6 months the patient had a complete active and passive range of motion, could perform sports-specific activities without symptoms, and recovered about 90% of normal strength. Return to sport was allowed at this stage.

41.4 Conclusion

Tendon healing requires a few months to occur, especially in case of large or massive rotator cuff tears; therefore aggressive rehabilitation protocols are not indicated even in case of young sportsman patients. In this kind of patients sport-specific exercises can be started between the fourth and fifth months postoperatively. If possible, avoid anti-inflammatory drugs in the first 2 months to not interfere with normal inflammatory process.

References

1. Van der Heijden GJ. Shoulder disorders: a state-of-the-art review. Baillieres Best Pract Res Clin Rheumatol. 1999;13:287–309.
2. Taylor W. Musculoskeletal pain in the adult New Zealand population: prevalence and impact. N Z Med J. 2005;118:U1629.
3. Dodson CC, Bedi A, Sahai A, Potter HG, Cordasco FA. Complete rotator cuff tendon avulsion and glenohumeral joint incarceration in a young patient: a case report. J Shoulder Elb Surg. 2010;19:e9–e12.
4. Dreinhöfer KE, Schüler S, Schäfer M, Ohly T. Rehabilitative konzepte und rückkehr zum sport nach eingriffen an der schulter. Orthopade. 2014;43:256–64.

Case Example 5: Revision Arthroscopic Rotator Cuff Repair

42

Ángel Calvo Díaz, Pablo Carnero Martín de Soto, and Néstor Zurita Uroz

42.1 Introduction

Repair of rotator cuff tears has become one of the most common orthopedic procedures performed worldwide. Scientific literature about repair techniques and prognostic factors that influence outcomes is abundant. However, there are no clear guidelines on how to manage its main complication: the recurrence of the tear.

There have been published few reports about revision arthroscopic rotator cuff repairs and the outcomes are mainly unpredictable [1, 2]. While it has been demonstrated that advanced age, tear size, and fatty degeneration are important prognostic factors for re-tear after primary repair, the variables that affect results after revision repairs are in debate [3, 4]. These facts contribute to make revision rotator cuff repairs a big challenge for surgeons, so its indication has to be done with huge caution.

It is crucial to identify the reason why the cuff re-tears. An inadequate previous surgical technique can be corrected during revision surgery. However, a biological failure in the healing process is difficult to solve. A new attempt to repair the cuff should be avoided if the surgeon foresees low potential of healing, though we do not know which factors would predict it.

Á. Calvo Díaz · P. Carnero Martín de Soto (✉)
Arthrosport Zaragoza, Zaragoza, Spain

N. Zurita Uroz
IMED Elche Hospital, Alicante, Spain

42.2 Case Presentation

We present the case of a 56-year-old male patient, builder, who suffered a complete traumatic rotator cuff tear on his right dominant shoulder after falling from 1.5 m height while working. No medical comorbidities were present.

A repair was performed 1 month later in another center. An isolated torn supraspinatus tendon was found with 2 cm medial retraction and sized 1 cm from anterior to posterior. A double-row-configuration repair was performed using double-loaded PEEK anchors, one medial and one lateral. Acromioplasty was done and the long head of biceps tendon was left intact. He was immobilized after surgery with a 30° abduction brace. Passive motion and pendulum exercises were allowed at week 6 after surgery. Active elevation and rotations were delayed until week 10, and different exercises were allowed progressively.

The evolution was correct until week 8, when he started suffering some discomfort during rehabilitation exercises that did not impede its completion. The symptoms evolved to pain with exercises and during nights with no influence on range of motion (ROM).

The patient arrived to our office 13 months after primary surgery with moderate pain at rest and severe during movements. He had limited last degrees of active ROM because of pain, reaching 155° forward flexion, 140° abduction, 70° of external rotation at 0° of abduction, 60° of exter-

N. Sampaio Gomes et al. (eds.), *Massive and Irreparable Rotator Cuff Tears*,
https://doi.org/10.1007/978-3-662-61162-3_42

nal rotation at 90° of abduction, and reaching L5 on internal rotation. Passive ROM was complete. Lag tests for all rotator cuff tendons were negative. Strength reached 4/5 on Daniel's scale for forward flexion, abduction, and external rotation at 0° of abduction. Acromioclavicular joint was not painful, but palpation of bicipital groove was.

Plain radiographs showed no upper migration of the humeral head nor glenohumeral joint osteoarthritis (Fig. 42.1). Magnetic resonance imaging (MRI) demonstrated a type II retear according to Cho et al. [5], involving supra- and infraspinatus tendons (Fig. 42.2). Maximum medial-to-lateral retraction of the edge of the tendon stump to the footprint was 4 cm; the anterior-to-posterior size was 3 cm. Fatty infiltration of the supraspinatus muscle belly was Goutallier stage 2; muscle atrophy was stage 1.

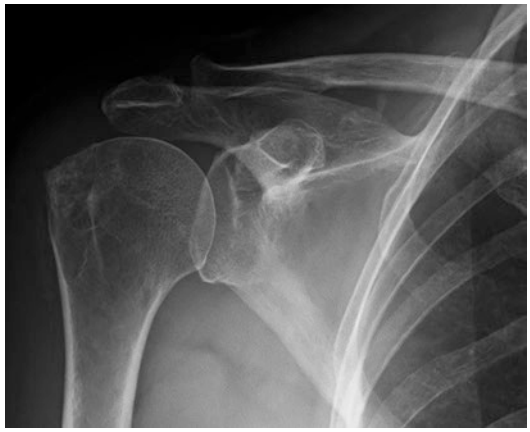


Fig. 42.1 X-ray showing no upper migration of the humeral head

Decision on treating the patient with a new rotator cuff repair was made for several reasons: first, he was a young patient; second, he was a manual worker who required adequate strength to perform his job properly; third, the quality of the tissues and retraction of the edges of the tendons could allow anatomical reduction, stable fixation, and reasonable healing potential although the tear seemed to be on the musculotendinous junction; and forth, the patient had showed in the past excellent adherence to rehabilitation programs.

42.3 Surgical Technique

A shoulder arthroscopy was performed under general anesthesia and interscalene nerve blockade in lateral decubitus position. During intra-

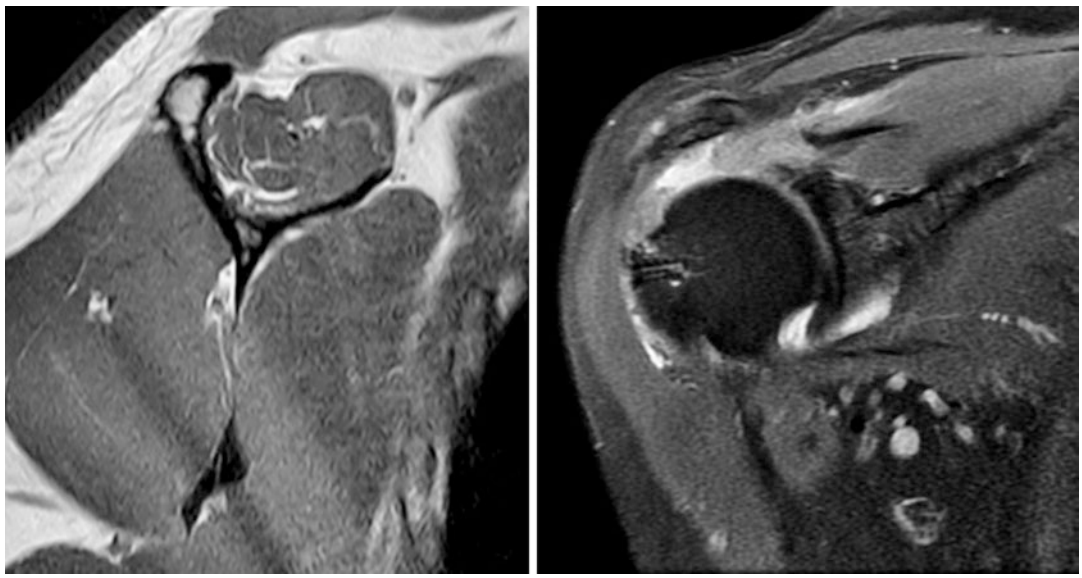


Fig. 42.2 MRI: (left) no intense fatty degeneration of supraspinatus muscle belly; (right) retear at the musculotendinous junction

articular inspection no chondral injuries were visible and biceps tenotomy was done. The scope was then placed at the subacromial space. The bursa was excised and extensive debridement was carried out until obtaining full visualization of the acromioclavicular joint, the trapezoid ligament, and the base of the coracoid process anteriorly and the spine of the scapula posteriorly. These anatomic landmarks would be used posteriorly as references to obtain proper orientation. A large medial-to-lateral and anterior-to-posterior sized tear was observed with no tendinous tissue remaining on the footprint (Fig. 42.3). Previous knots were cut and the lateral anchor from primary repair was extracted. Enough bony surface on the cuff footprint was prepared to place more anchors (Fig. 42.4). Mobility of the tendon edges was assessed with a grasper and complete coverage of the footprint without tension was observed after erasing adherences between the cuff tendons and the bony surfaces of the scapula. The liberation reached posteriorly the base of the spine of the scapula. At this point, the shaver was exclusively used in order to avoid damage of the suprascapular nerve when it passes under the spinoglenoid ligament. No anterior or posterior interval-slide technique was necessary to obtain full mobilization of the tendon. Then the scope was placed at the anterolateral portal for full visualization of the anterior edge of the tear. A Neviaser portal was used for suture passing through the tendon with direct graspers. Once passed, the limbs were retrieved trough the anterior or posterior portals and progressively tied. A double-row configuration repair was used, with two double-loaded anchors of PEEK medially and two laterally, tied from lateral to medial and from posterior to anterior. Full coverage of the footprint was obtained (Fig. 42.5).

The arm was placed on a sling and gentle pendulum and auto-passive flexion exercises were allowed from the beginning. Passive rotations and active flexion were permitted at the eighth week with progression to full active ROM. Resisted exercises were allowed at the 16th week and return to work at 6 months. At the end of the 1-year follow-up, the patient was pain-free and able to complete his daily life activities and job. Active ROM was almost completely restored, reaching 170° of forward flexion, 160° of abduction, 80° of external rotation with arm placed at 0° of abduction, 75° of external rotation with 90° of abduction of the arm, and touching L1 on internal rotation. Strength was 5/5 at all movements and slightly inferior compared to the contralateral side. The patient was satisfied and would recommend the treatment.

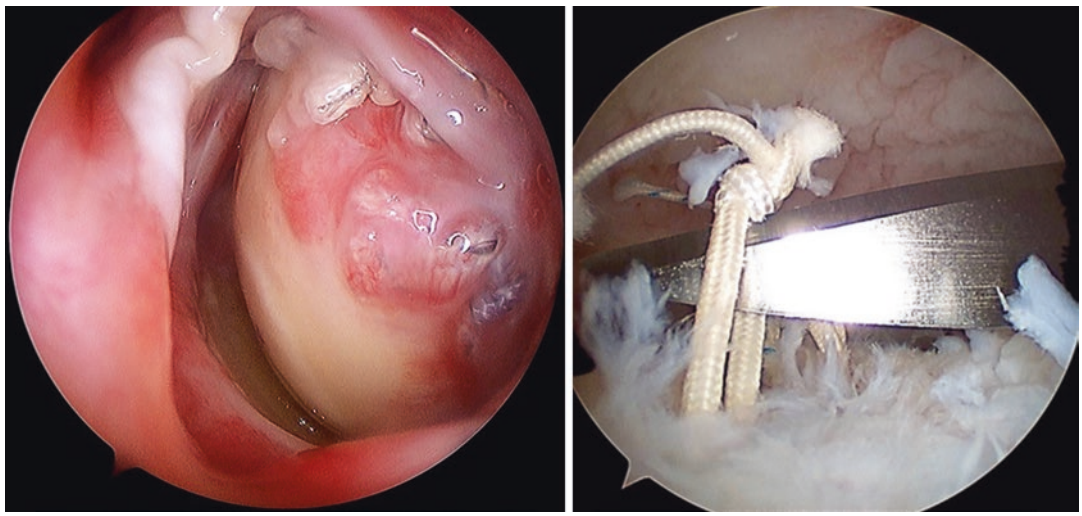


Fig. 42.3 (Left) Retear of the cuff with no tendon stumps at the footprint; (right) previous knots cut

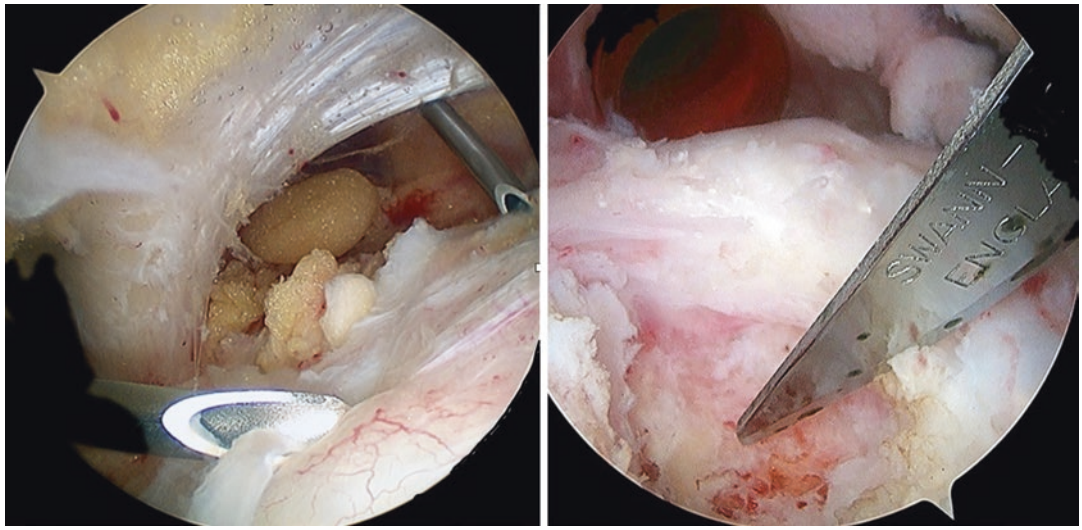


Fig. 42.4 Design of the repair: (left) location of the Neviaser and the posterior portals; the spine of the scapula acts as landmark; (right) cuff footprint once extracted the lateral anchor from previous repair

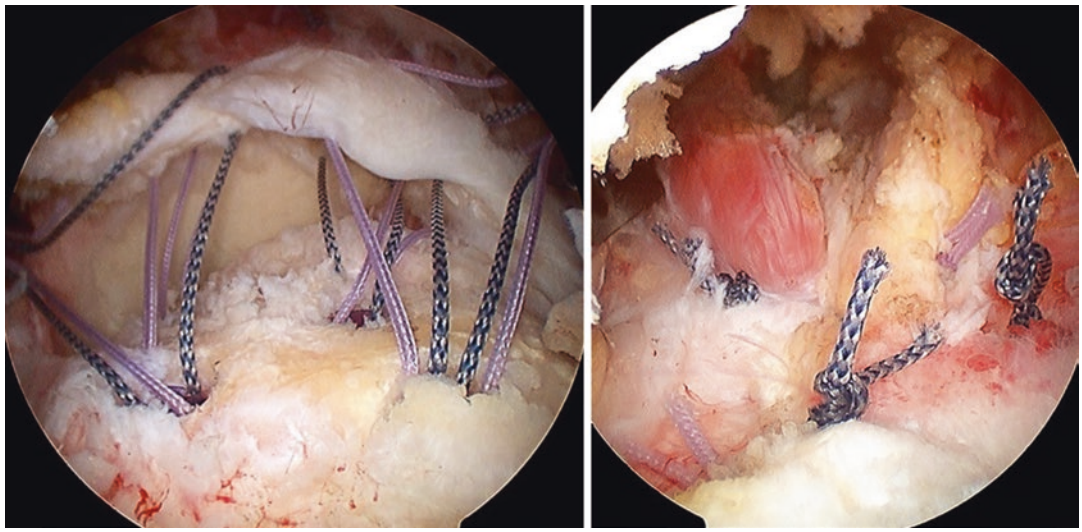


Fig. 42.5 (Left) Suture limbs passing through the tendon in double-row configuration, view from the anterolateral portal; (right) final repair

42.4 Discussion

Healing of the tendon is surgeon's main concern when the decision of repairing a re-tear of the rotator cuff is taken. Failure after primary repair usually happens during the first months without any traumatic event what demonstrates a biological deficit on the healing process [6]. Identifying factors that may lead to non-healing of the tendon is the key to make a correct indication when a retear appears.

In this case, we found several good prognostic factors that made us choosing a new repair of the cuff. The absence of intense fatty degeneration, atrophy, and retraction of the edge of the tendon encouraged us to repair it again. Gentle manage of

soft tissues, extensive debridement of the footprint, and obtaining a tension-free stable repair were the surgeon-dependent factors to obtain proper healing. However, we assumed that a high probability of failure existed as happened at first repair.

Revision rotator cuff repairs are, in general, safe procedures that allow good functional outcomes though inferior than those obtained with primary repairs [2, 7]. Moreover, the rate of failure in these situations remains high enough to consider other treatment options [8]. Other alternatives, as the addition of grafts reinforcing the repair or bridging the gap, have been proposed to improve clinical outcomes and enhance healing of the tendon to the footprint, with promising results [9].

In conclusion, revision repair of rotator cuff retear is a valid option in young patients in the absence of bad prognostic factors for tendon healing.

References

1. Piasecki DP, Verma NN, Nho SJ, Bhatia S, Boniquit N, Cole BJ, Nicholson GP, Romeo AA. Outcomes after arthroscopic revision rotator cuff repair. Am J Sports Med. 2010;38(1):40–6.
2. Valencia Mora M, Morcillo Barrenechea D, Martín Ríos MD, Foruria AM, Calvo E. Clinical outcome and prognostic factors of revision arthroscopic rotator cuff tear repair. Knee Surg Sports Traumatol Arthrosc. 2017;25(7):2157–63.
3. Saccomanno MF, Sircana G, Cazzato G, Donati F, Randelli P, Milano G. Prognostic factors influencing the outcome of rotator cuff repair: a systematic review. Knee Surg Sports Traumatol Arthrosc. 2016;24(12):3809–19.
4. Oh JH, Kim SH, Ji HM, Jo KH, Bin SW, Gong HS. Prognostic factors affecting anatomic outcome of rotator cuff repair and correlation with functional outcome. Arthroscopy. 2009;25(1):30–9.
5. Cho NS, Lee BG, Rhee YG. Arthroscopic rotator cuff repair 27. using a suture bridge technique: is the repair integrity actually maintained. Am J Sports Med. 2011;39(10):2108–16.
6. Mihata T, Watanabe C, Fukunishi K, Ohue M, Tsujimura T, Fujiwara K, Kinoshita M. Functional and structural outcomes of single-row versus double-row versus combined double-row and suture-bridge repair for rotator cuff tears. Am J Sports Med. 2011;39(10):2091–8.
7. Shamsudin A, Lam PH, Peters K, Rubenis I, Hackett L, Murrell GA. Revision versus primary arthroscopic rotator cuff repair: a 2-year analysis of outcomes in 360 patients. Am J Sports Med. 2015;43(3):557–64.
8. Lädermann A, Denard PJ, Burkhart SS. Management of failed rotator cuff repair: a systematic review. J ISAKOS. 2016;1(1):32–7.
9. Ono Y, Dávalos Herrera DA, Woodmass JM, Boorman RS, Thornton GM, Lo IK. Graft augmentation versus bridging for large to massive rotator cuff tears: a systematic review. Arthroscopy. 2017;33(3):673–80.