Giuseppe Milano
Andrea Grasso
Angel Calvo
Roman Brzóska
Editors

Management of Failed Shoulder Surgery

European Society of Sports Traumatology, Knee Surgery & Arthroscopy
ESSKA

Springer

Management of Failed Shoulder Surgery

Giuseppe Milano • Andrea Grasso
Angel Calvo • Roman Brzóska
Editors

Management of Failed Shoulder Surgery

Springer

Editors
Giuseppe Milano
Department of Orthopaedics
Catholic University
Rome
Italy

Andrea Grasso
Department of Orthopaedics
Casa di Cura Villa Valeria
Rome
Italy

Angel Calvo
Clínica del Pilar
Zaragoza
Spain

Roman Brzóska
Orthopaedic/Upper Limb
St. Luke's Hospital
Bielsko-Biała
Poland

ISBN 978-3-662-56503-2 ISBN 978-3-662-56504-9 (eBook)
https://doi.org/10.1007/978-3-662-56504-9

Library of Congress Control Number: 2018940098

© ESSKA 2018
This work is subject to copyright. All rights are reserved by the Publisher, whether the whole or part of the material is concerned, specifically the rights of translation, reprinting, reuse of illustrations, recitation, broadcasting, reproduction on microfilms or in any other physical way, and transmission or information storage and retrieval, electronic adaptation, computer software, or by similar or dissimilar methodology now known or hereafter developed.
The use of general descriptive names, registered names, trademarks, service marks, etc. in this publication does not imply, even in the absence of a specific statement, that such names are exempt from the relevant protective laws and regulations and therefore free for general use.
The publisher, the authors and the editors are safe to assume that the advice and information in this book are believed to be true and accurate at the date of publication. Neither the publisher nor the authors or the editors give a warranty, express or implied, with respect to the material contained herein or for any errors or omissions that may have been made. The publisher remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Printed on acid-free paper

This Springer imprint is published by the registered company Springer-Verlag GmbH, DE part of Springer Nature
The registered company address is: Heidelberger Platz 3, 14197 Berlin, Germany

To my father and my mother, fragile and brave persons, for their example of living (GM)

To my family, my Dad, my Mum and my wife. Thanks for always helping and supporting me (AG)

Dedicated to my four sons: Mar, Rocío, Angel and Alex (AC)

To my family, my co-workers and to the whole ESA team (RB)

Preface

In recent years, as an effect of technological innovation of surgical instruments and devices, and also the increasing number of surgical techniques being proposed and validated in the literature, we have seen a considerable expansion of the surgical options for the treatment of shoulder disorders. Furthermore, advancing globalisation, the growth of web-based scientific dissemination and education, and the constant and systematic training and information activities carried out by scientific societies and the research world have all contributed to an overall improvement in the level of theoretical and practical knowledge in the field of shoulder surgery, with the result that there is now very little difference, in terms of quality and surgical efficiency, between the health systems of different countries. All this has contributed to an exponential increase in the number of shoulder repair, reconstruction and replacement surgeries performed every year in the world. Inevitably, this has brought an increase in the number of failures and complications, which have also become more complex to manage, especially in patients with repeated failures. For this reason, the various scientific societies with an interest in shoulder disorders have recently become inclined to examine more closely the problem of shoulder surgery complications and failures, from different perspectives: prevention, diagnosis and management. In particular, European Shoulder Associates (ESA), the ESSKA section devoted to shoulder disorders and surgery, decided that its first biennial meeting should focus on this important and highly topical issue. This meeting, entitled "Management of Failed Shoulder Surgery", was held in Rome on 2–3 October 2015, and this book springs from that event.

We are particularly pleased and proud to have the task of presenting this monograph, which has the same title as the Rome congress, as it offers readers a valuable opportunity to explore aspects of a subject that is both complex and controversial. This is the first time in over a decade that a book has been published that deals exclusively and exhaustively with the management of failed shoulder surgeries, aiming to help us recognise these events, understand why they occur and find successful solutions.

The book is structured in the same way as the Rome meeting. There are five parts, each focusing on a specific area of shoulder surgery: glenohumeral instability surgery, sports injury surgery, standard anatomical shoulder replacement, reverse shoulder replacement and rotator cuff surgery. Each part is made up of chapters that analyse problems and solutions related to complications and failures specific to each surgical procedure. The parts also contain

case studies illustrating the diagnostic and therapeutic approach used by the authors to manage particularly complex cases.

All the speakers at the Rome congress agreed to take part in this book project, and all have provided a contribution, reviewed and updated, on the subject of their particular presentation. Our sincere thanks go to all of them. The enthusiastic support of all the authors has been crucial, helping us to produce volume of great scientific quality. We are confident that readers will appreciate the format the authors have chosen for their chapters, based mainly on a decision-making and problem-solving approach.

Finally, we thank ESSKA's Board for approving and supporting this initiative, and all those at Springer for their great professionalism, and also for the book's excellent quality in both graphic and editorial terms.

Rome, Italy — Giuseppe Milano
Rome, Italy — Andrea Grasso
Zaragoza, Spain — Angel Calvo
Bielsko-Biala, Poland — Roman Brzóska

Contents

Part I

Failed Shoulder Instability Repair

1 Shoulder Instability Repair: Why It Fails

Emilio Calvo, Gia Rodriguez-Vaquero, and David Haeni

1.1 Introduction

The glenohumeral (GH) joint is the least constrained joint in the body and allows a wide range of motion (ROM). On the other hand, it is more susceptible to high rates of instability. In the United States, the incidence of shoulder dislocations is 23 per 100,000 person-years, with the highest rates in adults in their 20s [1]. Anterior shoulder instability is the most frequent, and it is estimated that it affects 1.7% of the population. Current surgical techniques treating anterior shoulder instability are classified in soft tissue and bone augmentation procedures [2]. In the past, the open Bankart repair was considered the "gold standard," obtaining satisfactory surgical results since its first description [3]. Concerns regarding this technique were related to the extensive non-sparing subscapularis approach, immediate postoperative pain, loss of external rotation, and secondary osteoarthritis [4]. With the advent of new techniques and the development of new implants, the arthroscopic Bankart repair showed similar recurrence rates and functional outcomes than the open technique [5, 6]. Despite these results, reported recurrence rates after open or arthroscopic Bankart repair ranges between 5% and 15% [7, 8]. Bone augmentation procedures are usually preferred in young and active patients with recurrent shoulder dislocation in the presence of bone loss (Hill-Sachs lesions and/or bony Bankart) [9]. Recently, a prospective multicenter study found that the Latarjet procedure (open or arthroscopic) improves significantly shoulder function [10].

The main complication after surgical shoulder stabilization (whether open or arthroscopic) is recurrent instability. Revision instability surgery is usually a challenge, and patients with postoperative shoulder instability should be carefully evaluated not only to diagnose the failure but also to clearly identify the underlying causes that determined the outcome and to establish a successful therapeutic strategy [7, 8]. Careful preoperative evaluation is critical for the selection of the best treatment. The clinician must collect detailed information about the cause of the instability, the number and frequency of

E. Calvo (✉)
Shoulder and Elbow Reconstructive Surgery Unit, Department of Orthopedic Surgery and Traumatology, Fundacion Jimenez Diaz, Universidad Autónoma, Madrid, Spain
e-mail: ecalvo@fjd.es

G. Rodriguez-Vaquero
Upper Extremity Surgery Unit, Department of Orthopedic Surgery and Traumatology, Hospital General de Villalba, Madrid, Spain
e-mail: gia.rodriguez@fjd.es

D. Haeni
Shoulder and Elbow Surgery Unit, LEONARDO, Hirslanden Klinik Birshof, Basel, Switzerland

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_1

episodes, the degree of trauma necessary for recurrence, the arm position at the time of the initial injury, and the arm position that provokes symptoms [11].

Any patient with surgical treatment failure after shoulder stabilization can be classified in at least one of the following groups (Table 1.1). The first group is composed of patients in whom the problem was misdiagnosed, either because surgery was not indicated (i.e., voluntary instability), because the specific joint abnormalities to be corrected at surgery were not precisely identified, or because the direction of instability was not adequately understood (i.e., patients with multidirectional instability treated only for anterior instability). Patient-related risk factors may also increase the risk of postoperative recurrence and should be taken into account in the decision-making process in order to offer the best surgical treatment for every patient. Another group of subjects includes properly diagnosed patients in whom the treatment was inadequate, in terms of procedure selection or technical execution. Obviously, there could also be patients with combined misdiagnosis and inadequate treatment leading to surgical treatment failure. The last group includes those patients that were properly diagnosed, and in whom joint abnormalities were recognized and corrected with the optimal procedure, but who suffered a new trauma causing postoperative dislocation or subluxation [12, 13].

Table 1.1 Causes of failure of anterior shoulder stabilization

Misdiagnosis
– Surgical treatment not indicated
– Anatomical abnormalities not identified
– Direction of instability
Patient-related risk factors
– Age, sex
– Number of dislocation
– Type of sport
– Concomitant/trigger disease: Epilepsy, Ehlers-Danlos disease
Surgery-related risk factors
– Technical errors
– Inadequate treatment
– Implant failure: Anchor or graft related
Trauma after surgery
Unknown causes

1.1.1 Misdiagnosis

In order to properly address failed surgical treatment, it is essential first to clearly identify if surgery was indicated. Voluntary GH dislocation tends to occur in the young adult, and it is sometimes related to emotional and psychological problems. Huber et al. showed that voluntary subluxation in the childhood shows usually a favorable long-term outcome with conservative treatment and that is not associated with osteoarthritis [14]. Therefore, recurrent postoperative instability in this setting should be managed conservatively with physical therapy.

Once voluntary instability is ruled out, and considering that instability interferes with patient's activities, the most challenging issue is identifying which is the suitable surgical technique for each patient. For this purpose, it is crucial to recognize the direction of the instability, as well as the abnormalities responsible for recurrence to be addressed. Zabinski et al. [15] reported the comparative results of revision instability surgery in two groups of patients diagnosed of anterior and multidirectional instability, respectively. They found that persistent Bankart lesions were less common and the presence of hyperlaxity was almost constant in those diagnosed of multidirectional instability and concluded that while revision shoulder stabilization is a reliable procedure for patients who have recurrent anterior instability, it is unpredictable in patients who have multidirectional instability with surgical failure and reoperation occurring frequently.

Clinical history and meticulous physical examination allow identifying the direction of the instability, providing evidence about the possible causes of failure and potential associated lesions [16]. Physical examination should be performed always comparing the index shoulder to the contralateral side. The degree of instability (dislocation, subluxations, or apprehension) is also important information. The apprehension test is

performed with the arm hold at 0°–90°–140° abduction and is considered positive for anterior instability if the patient fears subluxation/dislocation or feels high discomfort during the maneuver. The sulcus sign is considered positive if during inferior traction of the shoulder held in neutral position a "sulcus" between acromion and humeral head is appreciated. A positive painful jerk test suggests postero-inferior labrum tear and a surgical repair should be discussed with the patient [17].

Examination under anesthesia before any revision surgery can be useful since it may overcome the clinical examination limitation due to patient's apprehension. Mechanical symptoms, such as catching or locking, may suggest a displaced labral tear, a loose body, or a large osseous defect that is engaging. Instability that occurs in the midrange of motion or during the sleep may indicate an osseous defect. Decreased ROM may be secondary to postoperative stiffness, chondrolysis, GH osteoarthritis, or excessive tension of the capsulolabral ligamentous complex. Loss of strength could be related to rotator cuff tear or neurological injury. Accurate rotator cuff testing should be performed, especially with regard to subscapularis muscle function in patients with previous open surgery. Sachs et al. [18] found that 23% of the patients undergoing open Bankart repair had a deficient subscapularis function and only 57% of them obtained good or excellent results after revision surgery.

Conventional radiography (CR) represents the first level of investigation in postoperative shoulder instability and should include outlet view, "true" anteroposterior view, and the axillary view. With the axillary view, we can evaluate anterior or posterior humeral head subluxation and the state bone graft healing.

Magnetic resonance imaging (MRI) with intra-articular contrast medium (MR arthrography, MRA) can be used both in presurgical and postsurgical care for shoulder instability giving a good assessment of capsulolabral-ligamentous complex and to evaluate postoperative recurrence or complication. MRA identifies soft tissue injuries, rotator cuff tears, humeral avulsion of the glenohumeral ligament (HAGL) lesions, capsulolabral lesions, chondral lesions, and laxity or rupture of the joint capsule better than standard MRI [19]. MRA in abduction and external rotation (ABER) position is useful to identify patients with atraumatic multidirectional instability. The presence of a layer of contrast medium between the humeral head and the anteroinferior glenohumeral ligament (AIGHL) (crescent sign) combined with a triangular-shaped space between the humeral head, AIGHL, and glenoid (triangle sign) has a sensitivity of 86% and specificity of 94% in diagnosing MDI [20].

Computed tomography (CT) can be used for bone evaluation and in cases in which CR does not give enough information about devices positioning. CT arthrography (CTA) is a valid alternative to MRA when susceptibility artifacts are present.

1.1.2 Patient-Related Failure

Several studies have attempted to establish the prognostic factors that may increase the risk of postoperative recurrence following surgical stabilization. Young age and participation in risk activities were identified as major prognostic factors in all of them in addition to the presence of bone defects [21–25]. Age at the first dislocation and male gender have been strongly correlated with a significantly higher risk of recurrent instability after a first dislocation, approaching 80% [21, 26]. Coherently to that, young male patients are more prone to recurrence after primary stabilization [11]. In a study of over 5900 patients, those younger than 20 years had a 12.6% risk of postoperative dislocation and a 7.7% revision rate after primary stabilization, compared to 5.5% and 2.8%, respectively, in patients older than 29 years of age [14]. When compared to adults, young patients usually have higher activity level, more compliant tissue, and decreased muscle bulk. Ninety percent of patients with recurrent dislocations after arthroscopic repair are male [16, 17].

The number of dislocations before stabilization, in addition to the number of previous surgeries, negatively correlates with postsurgical

success [27]. Wasserstein et al. [26] found that patients with three or more dislocations had double the risk for revision surgery and ten times the risk of re-dislocating. Patients with more than one stabilization procedure trended toward lower functional outcomes and less overall satisfaction [28]. These results are likely related to progressive damage tissue.

Collision athletes and contact overhead athletes are more frequently subject to higher energy trauma that can lead to shoulder dislocation and other injuries. In addition, postoperative return to collision sports is associated to a higher risk of new trauma and re-dislocation. Cho et al. [29] and Rhee et al. [30] reported higher instability recurrence rate in active athletes (17.2%) after arthroscopic Bankart repair. Even higher rates are reported in patients who practice collision sports (25–28%). Uhorchak et al. [31] reported outcomes of open Bankart repair, and they found a recurrence of 12% in collision and contact sports athletes. Castagna et al. [32] analyzed the effectiveness of arthroscopic Bankart repair in adolescent athletes who practiced overhead or contact sports at competitive level and reported higher recurrence rate in very high-energy contact sports (rugby) and in high-energy contact sports associated with overhead position of the arm (water polo). Other authors associated contact sports with higher risk of recurrence, but it does not seem to be a contraindication for arthroscopic Bankart repair [33, 34].

Calvo et al. [21] evaluated prospectively 61 patients treated arthroscopically with Bankart repair for recurrent anterior shoulder instability. They developed a risk score for failure of arthroscopic Bankart repair based upon an analysis of the factors that may determine the outcomes (level of satisfaction and degree of stability). Age younger than 28 years, ligamentous laxity, the presence of a fracture of the glenoid rim involving more than 15% of the articular surface, and postoperative participation in contact or overhead sports were associated with a higher risk of recurrence and scored 1, 1.5, and 1 point, respectively. Those patients with a total score of two or more points had a relative risk of recurrence of 43% and should be treated by open surgery. Later, Balg et al. [22] developed the instability severity index score (ISIS) to predict the success of arthroscopic Bankart repair. The ISIS score ranges from 0 to 10, with higher scores predicting a higher risk of recurrence after stabilization. Six risk factors are considered that can predict a higher recurrence rate: age at the surgery (over or below 20), degree and type of preoperative sport, hyperlaxity, and bone loss studied on CR.

Epileptic seizures can cause shoulder dislocation and instability, but these patients follow a characteristic pattern of instability with peculiar structural lesions. Bühler and Gerber [35] studied 34 shoulders in which initial dislocation had been caused by an epileptic seizure. Fifty percent of them had anterior instability and 50% posterior instability. They also found a higher recurrence rate for anterior instability comparing with posterior instability (47 versus 12%) after primary repair. Most of them were associated to poor control of epilepsy disease. Thangarajah et al. [36] followed up 49 patients with recurrent instability with epilepsy for 15 years: 73% of them showed anterior instability, 15% posterior, and 10% multidirectional instability. Eighty percent of all patients showed bone loss. They identified bone loss and persistent postoperative epileptic seizures as the principal factors for recurrent instability. Epileptic medical control and bone block procedure are associated with lower rate of recurrence.

1.1.3 Inadequate Treatment: Anatomic Abnormalities and Technique of Stabilization

Shoulder stabilization surgery should be tailored to the patient and to the specific abnormalities existing in the shoulder. In a cohort of 32 patients surgically revised for recurrent anterior dislocation of the shoulder after surgical repair, Rowe et al. [37] found that an abnormality that had not been adequately addressed and explaining the recurrence could be identified in more than 85% of the patients with postoperative shoulder instability. Moreover, Meeham and Petersen [12] proved in a similar investigation that in almost

half of the cases there is more than one lesion. Therefore, in revision instability surgery, it is crucial to study and identify the specific anatomic abnormalities responsible for the poor outcome. The most frequent abnormalities that can lead to shoulder instability surgery failure are the presence of non-repaired or medially repaired Bankart lesion (Fig. 1.1), poor capsulolabral tissue (Fig. 1.2) or hyperlaxity, and unaddressed bone defects (either on the glenoid or the humeral side) [12, 15, 37, 38].

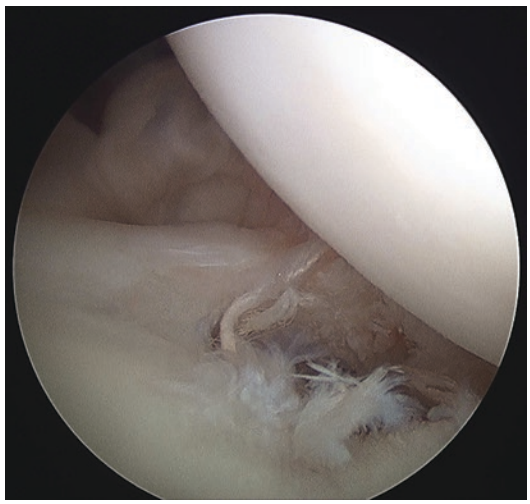

Fig. 1.1 Left shoulder. Arthroscopic view from the posterior portal: medially repaired Bankart lesion

Insufficient labral detachment followed by anatomic re-fixation of a medially healed labrum after multiple episodes of recurrence is probably the most common error during Bankart repair. Anterior labro-ligamentous periosteal sleeve avulsion (ALPSA) lesions have been identified as a risk factor for recurrence comparing with isolated Bankart lesion [27, 39] (Fig. 1.3). This lesion is present more frequently in patients with high number of dislocations. The reason of recurrence after repair may be related to the poor quality of capsulolabral tissue, due to progressive damage. Underestimation of HAGL lesions is also responsible for persistent postoperative instability (Fig. 1.4). A high index of suspicion is necessary to identify and repair this lesion, which can appear in 9% of anterior instability cases [40, 41]. Cases of first-time shoulder dislocation without Bankart lesion and no multidirectional laxity can show a high incidence of HAGL lesions [42].

Poorly positioned anchors have also been associated with recurrence of instability [43]. The number of suture anchors used for primary arthroscopic Bankart repair plays also an important role in the recurrence rate, and three or more anchors are usually recommended in most common cases of anterior shoulder instability [27, 44, 45]. With regard to the type of anchors, data showed no difference in recurrence rate between

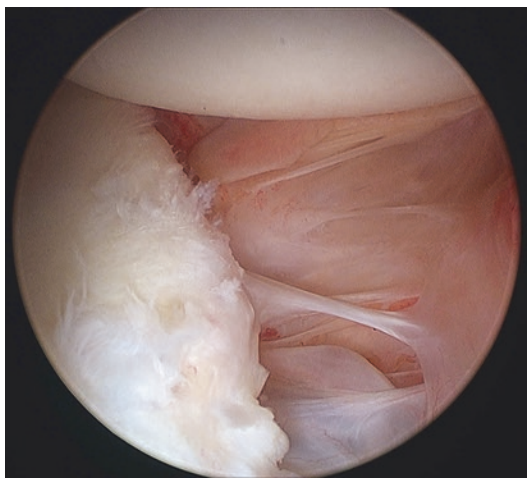

Fig. 1.2 Left shoulder. Arthroscopic view from the anterosuperior portal: poor capsulolabral tissue

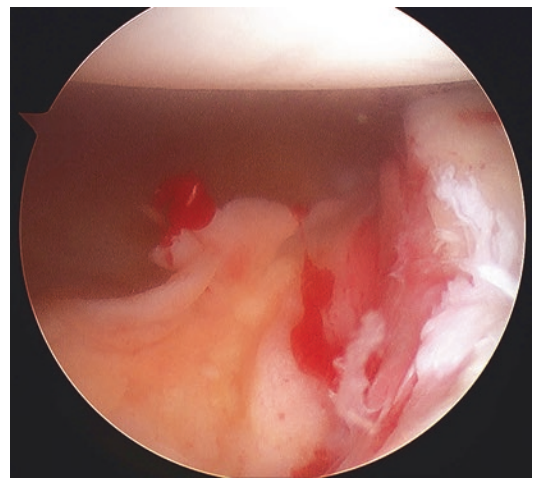

Fig. 1.3 Right shoulder. Arthroscopic view from the anterosuperior portal: ALPSA lesion

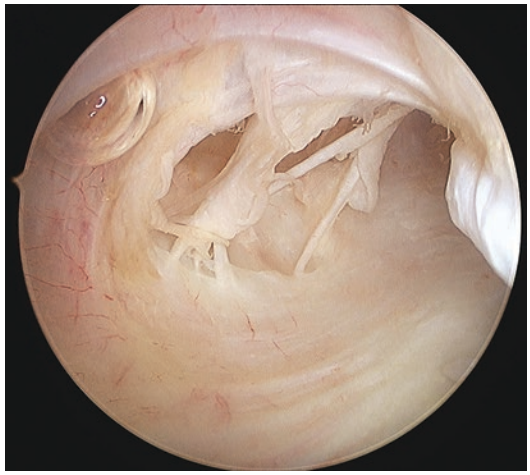

Fig. 1.4 Right shoulder. Arthroscopic view from the posterior portal: HAGL lesion

metal or biodegradable devices [46]. However, a significant difference was found between patients in whom knot-tying and knotless suture anchors were used, with higher rate of recurrence using knotless anchors [47].

One of the most commonly known mistakes includes failure to recognize and address capsular laxity during arthroscopic repair [12, 15, 37, 38]. Hyperlaxity and instability may be coexisting conditions. The difference between instability and hyperlaxity needs to be assessed preoperatively and influences the therapeutic decision. After multiple shoulder dislocations, anterior capsular tissue may be stretched and becomes redundant [12, 28]. Bigliani et al. [48] demonstrated that anterior capsular stretching can occur with or without labral detachment. Rowe et al. [37] showed that 83% of patients with recurrent dislocations after surgical repair had significant capsular laxity.

It is known that the recurrence of instability is significantly higher in patients with anterior glenoid bone defects [21, 23]. Imaging studies are essential for the evaluation of patients with recurrent instability, since it allows the identification and quantification of glenoid bone loss and other possible articular abnormalities. While CR is considered important for bone loss assessment and many different radiographic views have been proposed, CT scan is considered the ideal method to quantify both glenoid and humeral head bone defects. Several authors [49–51] described the glenoid osseous defect as being located anteriorly at approximately the 3 o'clock position (in the right shoulder) and extending toward inferiorly. 3D CT scan with humeral head subtraction facilitates quantification of glenoid bone defect related to the total area and depth of the defect. Glenoid bone defect over 20% have been strongly associated with high risk of recurrence of instability after Bankart repair [45, 52], but Calvo et al. [21] demonstrated that a glenoid bone defect involving 15% of the articular surface represented a higher risk of postoperative failure. Yamamoto et al. [53] introduced the concept of the "glenoid track" determining whether a bipolar lesion was significant. The "glenoid track" concept offers the surgeon the possibility to predict engagement, based on size and morphology lesions [49]. The critical size of a Hill-Sachs lesion is thought to be a volume over 250 mm^3, defined as "large Hill-Sachs lesion" [54]. Recent clinical evidence supports the "on-track" versus "off-track" model in predicting failure of isolated Bankart repair in shoulders with bipolar bone loss [55, 56].

Bone augmentation procedures are preferred to address bone defects, and Latarjet is regarded as the gold standard technique for this condition [57, 58]. Walch et al. [9] conducted a study of 68 shoulders after open Latarjet and reported a recurrence rate of 5.9% after a mean 20-year follow-up. Young and Rockwood [59] studied a population of 39 patients with painful instability after shoulder stabilization performed with an open Bristow procedure and attributed the recurrences to the presence of capsular redundancy in 23 (59%) cases. Other investigations have also found labral defects and capsular elongation at arthroscopic revision of recurrent instability in patients previously operated with bone block procedures [60, 61]. Arthroscopic examination was considered extremely useful in identifying these abnormalities, and labral re-fixation with capsular plication was recommended to stabilize the shoulder [61]. However, other authors have attributed postoperative instability after Latarjet to complications related to the coracoid

graft, either due to malposition, malunion, or nonunion [62]. Gasbarro et al. [63] analyzed the reasons for failure after coracoid transfer procedures in a cohort of 83 patients and considered too inferior or too medial graft placement to be a risk factor for recurrence, as well as single screw fixation of the coracoid graft. Nonunion is a well-known complication after Latarjet procedure that can involve over 9% of the patients, but it has not been clearly associated with a higher risk of recurrence [64]. The coracoid graft can also show osteolysis at its upper half, but this complication does not seem to be correlated with postoperative recurrence either [65–67].

Eden-Hybinette, either open or arthroscopic, has been regarded as the elective technique for failed Latarjet, especially in patients with bone defects [68, 69]. Lunn et al. [70] reported the first series of the Eden-Hybinette revision procedure in a cohort of 46 patients with failed Latarjet and found different risk factors for recurrence such as malposition, lysis, or avulsion of the coracoid graft. Interestingly, the authors identified that ligamentous laxity was present in 14 patients and for the first time incriminated subscapularis weakness as a reason for failure 10 patients (5 patients had a complete rupture of the subscapularis tendon). Calvo et al. [71] reported a series of 11 patients who underwent revision surgery for recurrent instability after Latarjet stabilization. The technique used was based on the specific anatomic abnormalities found at arthroscopy: the coracoid graft inadequately positioned was repositioned with open surgery in three cases; extraarticular capsular reinforcement was performed in four shoulders that showed hyperlaxity or poor capsulolabral tissue and no severe bone defect, while arthroscopic Eden-Hybinette was used in four shoulders with humeral or glenoid bone defects and a nonviable coracoid graft.

Boileau et al. [44] pointed out the role of certain Hill-Sachs defects in the recurrence following surgical stabilization. Recently, Locher et al. [72] assessed the impact of "off-track" Hill-Sachs lesions in a study of 254 patients with anterior instability managed with a Bankart repair. The authors demonstrated that Hill-Sachs "off-track" lesions constitute an important risk factor for recurrence of instability after arthroscopic Bankart repair and need of revision surgery compared to "on-track" defects. "Remplissage" is regarded as the procedure of choice for those patients with "off-track" defects, albeit Latarjet procedure could be a valid alterative in shoulders with "off-track" Hill-Sachs lesions by increasing the articular surface area. However, Millet et al. [11] demonstrated that the presence of "off-track" Hill-Sachs lesions increases the risk for persistent engagement after surgery also after stabilization with the Latarjet technique.

Based on the few comparative studies reported, there is no evidence on the superiority of open or arthroscopic stabilization in terms of recurrence, and the fact that arthroscopic stabilization represents an independent risk for recurrence cannot be sustained. Mohtadi et al. [73] carried out a prospective study of 196 patients randomized to undergo open or arthroscopic soft tissue stabilization and concluded that although there were no differences concerning postoperative quality of life, the recurrence rate was superior after arthroscopic surgery. However, Fabbriciani et al. [74], in a study with a similar design, failed to find differences between the two therapeutic approaches and noticed that the group treated arthroscopically showed superior postoperative mobility over the open group. Moreover, Archetti Netto et al. [75] reported lower failure rates, higher mobility, and fewer complications after arthroscopic Bankart stabilization. With regard to coracoid transfer procedures, there are not published studies on the superiority of the arthroscopic versus the traditional open approach. Arthroscopic surgery is very helpful in identifying articular abnormalities to be amended, and arthroscopic revision stabilization provides satisfactory results [62]. The technique allows direct visualization of the pathology that may be responsible for recurrence, including unexpected causes that can be corrected during the same procedure, such as loose bodies, rotator cuff tears, or chondral lesions [76, 77].

1.1.4 New Trauma

Traumatic injuries to the surgically repaired shoulder are one of the biggest contributors to recurrence. As the majority of those affected are young with initial injuries often due to athletic activities, return to collision sport or overhead throwing sports predisposes this population to reinjury. Tauber et al. [38] reviewed 41 patients and found that 85% of initial shoulder dislocations and 59% of re-dislocations after surgical stabilization were traumatic.

Conclusion

Key factors for successful surgical shoulder stabilization are adequate patient selection, precise surgical technique selection and fulfillment, identification and correction of all joint abnormalities, and integration of patient and surgeon expectations. For this purpose, we must be able to correctly answer the following questions: what are the characteristics of the patient? Which shoulder injuries should be treated? Did the patient have a new trauma responsible for recurrence? Despite all known risk factors for recurrence of instability, there are cases in which it is not possible to establish the cause of primary repair failure.

References

1. Zacchilli MA, Owens BD. Epidemiology of shoulder dislocations presenting to emergency Departments in the United States. J Bone Joint Surg Am. 2010;92:542–9. https://doi.org/10.2106/JBJS.I.00450.
2. Dumont GD, Russell RD, Robertson WJ. Anterior shoulder instability: a review of pathoanatomy, diagnosis and treatment. Curr Rev Musculoskelet Med. 2011;4:200–7. https://doi.org/10.1007/s12178-011-9092-9.
3. Bankart ASB. Recurrent or habitual dislocation of the shoulder-joint. Br Med J. 1923;2:1132–3.
4. Rosenberg BN, Richmond JC, Levine WN. Long-term followup of Bankart reconstruction. Incidence of late degenerative glenohumeral arthrosis. Am J Sports Med. 1995;23:538–44. https://doi.org/10.1177/036354659502300504.
5. Hobby J, Griffin D, Dunbar M, Boileau P. Is arthroscopic surgery for stabilisation of chronic shoulder instability as effective as open surgery? A systematic review and meta-analysis of 62 studies including 3044 arthroscopic operations. J Bone Joint Surg Br. 2007;89:1188–96. https://doi.org/10.1302/0301-620X.89B9.18467.
6. Freedman KB, Smith AP, Romeo AA, et al. Open Bankart repair versus arthroscopic repair with transglenoid sutures or bioabsorbable tacks for recurrent anterior instability of the shoulder: a meta-analysis. Am J Sports Med. 2004;32:1520–7. https://doi.org/10.1177/0363546504265188.
7. Franceschi F, Longo UG, Ruzzini L, et al. Arthroscopic salvage of failed arthroscopic Bankart repair: a prospective study with a minimum follow-up of 4 years. Am J Sports Med. 2008;36:1330–6. https://doi.org/10.1177/0363546508314403.
8. Millar NL, Murrell GAC. The effectiveness of arthroscopic stabilisation for failed open shoulder instability surgery. J Bone Joint Surg Br. 2008;90:745–50. https://doi.org/10.1302/0301-620X.90B6.20018.
9. Walch G. Chronic anterior instability of the shoulder in adults. Summary. Rev Chir Orthop Reparatrice Appar Mot. 2000;86(Suppl 1):147–9.
10. Nourissat G, Neyton L, Metais P, et al. Functional outcomes after open versus arthroscopic Latarjet procedure: a prospective comparative study. Orthop Traumatol Surg Res. 2016;102:S277–9. https://doi.org/10.1016/j.otsr.2016.08.004.
11. Millett PJ, Clavert P, Warner JJP. Open operative treatment for anterior shoulder instability: when and why? J Bone Joint Surg Am. 2005;87:419–32. https://doi.org/10.2106/JBJS.D.01921.
12. Meehan RE, Petersen SA. Results and factors affecting outcome of revision surgery for shoulder instability. J Shoulder Elbow Surg. 2005;14:31–7. https://doi.org/10.1016/j.jse.2004.05.005.
13. Sisto DJ. Revision of failed arthroscopic bankart repairs. Am J Sports Med. 2007;35:537–41. https://doi.org/10.1177/0363546506296520.
14. Huber H, Gerber C. Voluntary subluxation of the shoulder in children. A long-term follow-up study of 36 shoulders. J Bone Joint Surg Br. 1994;76:118–22.
15. Zabinski SJ, Callaway GH, Cohen S, Warren RF. Revision shoulder stabilization: 2- to 10-year results. J Shoulder Elbow Surg. 1999;8:58–65.
16. Johansson K. Multidirectional instability of the glenohumeral joint: an unstable classification resulting in uncertain evidence-based practice. Br J Sports Med. 2016;50:1105–6. https://doi.org/10.1136/bjsports-2016-096094.
17. Kim S-H, Park J-C, Park J-S, Oh I. Painful jerk test: a predictor of success in nonoperative treatment of posteroinferior instability of the shoulder. Am J Sports Med. 2004;32:1849–55.
18. Sachs RA, Williams B, Stone ML, et al. Open Bankart repair: correlation of results with postoperative subscapularis function. Am J Sports Med. 2005;33:1458–62. https://doi.org/10.1177/0363546505275350.
19. Probyn LJ, White LM, Salonen DC, et al. Recurrent symptoms after shoulder instability repair: direct MR arthrographic assessment—correlation with second-look surgical evaluation. Radiology. 2007;245:814–23. https://doi.org/10.1148/radiol.2453061329.

20. Schaeffeler C, Waldt S, Bauer JS, et al. MR arthrography including abduction and external rotation images in the assessment of atraumatic multidirectional instability of the shoulder. Eur Radiol. 2014;24:1376–85. https://doi.org/10.1007/s00330-014-3133-x.
21. Calvo E, Granizo JJ, Fernández-Yruegas D. Criteria for arthroscopic treatment of anterior instability of the shoulder: a prospective study. J Bone Joint Surg Br. 2005;87:677–83. https://doi.org/10.1302/0301-620X.87B5.15794.
22. Balg F, Boileau P. The instability severity index score. A simple pre-operative score to select patients for arthroscopic or open shoulder stabilisation. J Bone Joint Surg Br. 2007;89:1470–7. https://doi.org/10.1302/0301-620X.89B11.18962.
23. Ahmed I, Ashton F, Robinson CM. Arthroscopic Bankart repair and capsular shift for recurrent anterior shoulder instability: functional outcomes and identification of risk factors for recurrence. J Bone Joint Surg Am. 2012;94:1308–15. https://doi.org/10.2106/JBJS.J.01983.
24. Voos JE, Livermore RW, Feeley BT, et al. Prospective evaluation of arthroscopic bankart repairs for anterior instability. Am J Sports Med. 2010;38:302–7. https://doi.org/10.1177/0363546509348049.
25. Flinkkilä T, Hyvönen P, Ohtonen P, Leppilahti J. Arthroscopic Bankart repair: results and risk factors of recurrence of instability. Knee Surg Sports Traumatol Arthrosc. 2010;18:1752–8. https://doi.org/10.1007/s00167-010-1105-5.
26. Wasserstein D, Dwyer T, Veillette C, et al. Predictors of dislocation and revision after shoulder stabilization in Ontario, Canada, from 2003 to 2008. Am J Sports Med. 2013;41:2034–40. https://doi.org/10.1177/0363546513492952.
27. Randelli P, Ragone V, Carminati S, Cabitza P. Risk factors for recurrence after Bankart repair a systematic review. Knee Surg Sports Traumatol Arthrosc. 2012;20:2129–38. https://doi.org/10.1007/s00167-012-2140-1.
28. Marquardt B, Garmann S, Schulte T, et al. Outcome after failed traumatic anterior shoulder instability repair with and without surgical revision. J Shoulder Elbow Surg. 2007;16:742–7. https://doi.org/10.1016/j.jse.2007.02.132.
29. Cho NS, Hwang JC, Rhee YG. Arthroscopic stabilization in anterior shoulder instability: collision athletes versus noncollision athletes. Arthroscopy. 2006;22:947–53. https://doi.org/10.1016/j.arthro.2006.05.015.
30. Rhee YG, Ha JH, Cho NS. Anterior shoulder stabilization in collision athletes: arthroscopic versus open Bankart repair. Am J Sports Med. 2006;34:979–85. https://doi.org/10.1177/0363546505283267.
31. Uhorchak JM, Arciero RA, Huggard D, Taylor DC. Recurrent shoulder instability after open reconstruction in athletes involved in collision and contact sports. Am J Sports Med. 2000;28:794–9. https://doi.org/10.1177/03635465000280060501.
32. Castagna A, Delle Rose G, Borroni M, et al. Arthroscopic stabilization of the shoulder in adolescent athletes participating in overhead or contact sports. Arthroscopy. 2012;28:309–15. https://doi.org/10.1016/j.arthro.2011.08.302.
33. Mazzocca AD, Brown FM, Carreira DS, et al. Arthroscopic anterior shoulder stabilization of collision and contact athletes. Am J Sports Med. 2005;33:52–60. https://doi.org/10.1177/0363546504268037.
34. Grana WA, Buckley PD, Yates CK. Arthroscopic Bankart suture repair. Am J Sports Med. 1993;21:348–53. https://doi.org/10.1177/036354659302100304.
35. Bühler M, Gerber C. Shoulder instability related to epileptic seizures. J Shoulder Elbow Surg. 2002;11:339–44. https://doi.org/10.1067/mse.2002.124524.
36. Thangarajah T, Lambert S. The management of recurrent shoulder instability in patients with epilepsy: a 15-year experience. J Shoulder Elbow Surg. 2015;24:1723–7. https://doi.org/10.1016/j.jse.2015.04.008.
37. Rowe CR, Zarins B, Ciullo JV. Recurrent anterior dislocation of the shoulder after surgical repair. Apparent causes of failure and treatment. J Bone Joint Surg Am. 1984;66:159–68.
38. Tauber M, Resch H, Forstner R, et al. Reasons for failure after surgical repair of anterior shoulder instability. J Shoulder Elbow Surg. 2004;13:279–85. https://doi.org/10.1016/S1058274604000254.
39. Porcellini G, Campi F, Pegreffi F, et al. Predisposing factors for recurrent shoulder dislocation after arthroscopic treatment. J Bone Joint Surg Am. 2009;91:2537–42. https://doi.org/10.2106/JBJS.H.01126.
40. Wolf EM, Siparsky PN. Glenoid avulsion of the glenohumeral ligaments as a cause of recurrent anterior shoulder instability. Arthroscopy. 2010;26:1263–7. https://doi.org/10.1016/j.arthro.2010.06.005.
41. Flury M, Rickenbacher D, Audigé L. Arthroscopic treatment of anterior shoulder instability associated with a HAGL lesion-a case series. J Shoulder Elbow Surg. 2016;25:1989–96. https://doi.org/10.1016/j.jse.2016.02.030.
42. Savoie FH, Holt MS, Field LD, Ramsey JR. Arthroscopic management of posterior instability: evolution of technique and results. Arthroscopy. 2008;24:389–96. https://doi.org/10.1016/j.arthro.2007.11.004.
43. Kim S-H, Ha K-I, Kim Y-M. Arthroscopic revision Bankart repair: a prospective outcome study. Arthroscopy. 2002;18:469–82. https://doi.org/10.1053/jars.2002.32230.
44. Boileau P, Villalba M, Héry J-Y, et al. Risk factors for recurrence of shoulder instability after arthroscopic Bankart repair. J Bone Joint Surg Am. 2006;88:1755–63. https://doi.org/10.2106/JBJS.E.00817.
45. An VVG, Sivakumar BS, Phan K, Trantalis J. A systematic review and meta-analysis of clinical and patient-reported outcomes following two procedures for recurrent traumatic anterior instability of the shoulder: Latarjet procedure vs. Bankart repair. J Shoulder Elbow Surg. 2016;25:853–63. https://doi.org/10.1016/j.jse.2015.11.001.

46. Thal R. Knotless suture anchor: arthroscopic bankart repair without tying knots. Clin Orthop. 2001;390:42–51.
47. Cho NS, Lubis AMT, Ha JH, Rhee YG. Clinical results of arthroscopic bankart repair with knot-tying and knotless suture anchors. Arthroscopy. 2006;22:1276–82. https://doi.org/10.1016/j.arthro.2006.07.005.
48. Bigliani LU, Kelkar R, Flatow EL, et al. Glenohumeral stability. Biomechanical properties of passive and active stabilizers. Clin Orthop. 1996;330:13–30.
49. Griffith JF, Antonio GE, Tong CWC, Ming CK. Anterior shoulder dislocation: quantification of glenoid bone loss with CT. AJR Am J Roentgenol. 2003;180:1423–30. https://doi.org/10.2214/ajr.180.5.1801423.
50. Saito H, Itoi E, Sugaya H, et al. Location of the glenoid defect in shoulders with recurrent anterior dislocation. Am J Sports Med. 2005;33:889–93. https://doi.org/10.1177/0363546504271521.
51. Ji J-H, Kwak D-S, Yang P-S, et al. Comparisons of glenoid bony defects between normal cadaveric specimens and patients with recurrent shoulder dislocation: an anatomic study. J Shoulder Elbow Surg. 2012;21:822–7. https://doi.org/10.1016/j.jse.2011.10.016.
52. Shibata H, Gotoh M, Mitsui Y, et al. Risk factors for shoulder re-dislocation after arthroscopic Bankart repair. J Orthop Surg. 2014;9:53. https://doi.org/10.1186/s13018-014-0053-z.
53. Yamamoto N, Itoi E, Abe H, et al. Contact between the glenoid and the humeral head in abduction, external rotation, and horizontal extension: a new concept of glenoid track. J Shoulder Elbow Surg. 2007;16:649–56. https://doi.org/10.1016/j.jse.2006.12.012.
54. Hartzler RU, Bui CNH, Jeong WK, et al. Remplissage of an off-track Hill-Sachs lesion is necessary to restore biomechanical glenohumeral joint stability in a bipolar bone loss model. Arthroscopy. 2016;32:2466–76. https://doi.org/10.1016/j.arthro.2016.04.030.
55. Di Giacomo G, Itoi E, Burkhart SS. Evolving concept of bipolar bone loss and the Hill-Sachs lesion: from "engaging/non-engaging" lesion to "on-track/off-track" lesion. Arthroscopy. 2014;30:90–8. https://doi.org/10.1016/j.arthro.2013.10.004.
56. Tokish JM, Shaha JS, Cook JB, et al. Predictive value and clinical validation of the "on-track" vs. "off-track" concept in bipolar bone loss in anterior glenohumeral instability. Orthop J Sports Med. 2015;3:2325967115S00089. https://doi.org/10.1177/2325967115S00089.
57. Anderl W, Kriegleder B, Heuberer PR. All-arthroscopic implant-free iliac crest bone grafting: new technique and case report. Arthroscopy. 2012;28:131–7. https://doi.org/10.1016/j.arthro.2011.10.027.
58. Walch G. Luxation récidivante antérieure de l'épaule. Rev Chir Orthop. 1991;77:177–91.
59. Young DC, Rockwood CA. Complications of a failed Bristow procedure and their management. J Bone Joint Surg Am. 1991;73:969–81.
60. Cuéllar A, Cuéllar R, de Heredia PB. Arthroscopic revision surgery for failure of open Latarjet technique. Arthroscopy. 2017;33:910–7. https://doi.org/10.1016/j.arthro.2016.09.017.
61. Castagna A, Garofalo R, Melito G, et al. The role of arthroscopy in the revision of failed Latarjet procedures. Musculoskelet Surg. 2010;94(Suppl 1):S47–55. https://doi.org/10.1007/s12306-010-0060-0.
62. Boileau P, Richou J, Lisai A, et al. The role of arthroscopy in revision of failed open anterior stabilization of the shoulder. Arthroscopy. 2009;25:1075–84. https://doi.org/10.1016/j.arthro.2009.04.073.
63. Gasbarro G, Giugale JM, Walch G, Lin A. Predictive surgical reasons for failure after coracoid process transfers. Orthop J Sports Med. 2016;4:2325967116676795. https://doi.org/10.1177/2325967116676795.
64. Gupta A, Delaney R, Petkin K, Lafosse L. Complications of the Latarjet procedure. Curr Rev Musculoskelet Med. 2015;8:59–66. https://doi.org/10.1007/s12178-015-9258-y.
65. Di Giacomo G, de Gasperis N, Costantini A, et al. Does the presence of glenoid bone loss influence coracoid bone graft osteolysis after the Latarjet procedure? A computed tomography scan study in 2 groups of patients with and without glenoid bone loss. J Shoulder Elbow Surg. 2014;23:514–8. https://doi.org/10.1016/j.jse.2013.10.005.
66. Di Giacomo G, Costantini A, de Gasperis N, et al. Coracoid graft osteolysis after the Latarjet procedure for anteroinferior shoulder instability: a computed tomography scan study of twenty-six patients. J Shoulder Elbow Surg. 2011;20:989–95. https://doi.org/10.1016/j.jse.2010.11.016.
67. Haeni DL, Opsomer G, Sood A, et al. Three-dimensional volume measurement of coracoid graft osteolysis after arthroscopic Latarjet procedure. J Shoulder Elbow Surg. 2017;26:484–9. https://doi.org/10.1016/j.jse.2016.08.007.
68. Giannakos A, Vezeridis PS, Schwartz DG, et al. All-arthroscopic revision eden-hybinette procedure for failed instability surgery: technique and preliminary results. Arthroscopy. 2017;33:39–48. https://doi.org/10.1016/j.arthro.2016.05.021.
69. Mizuno N, Denard PJ, Raiss P, et al. Long-term results of the Latarjet procedure for anterior instability of the shoulder. J Shoulder Elbow Surg. 2014;23:1691–9. https://doi.org/10.1016/j.jse.2014.02.015.
70. Lunn JV, Castellano-Rosa J, Walch G. Recurrent anterior dislocation after the Latarjet procedure: outcome after revision using a modified Eden-Hybinette operation. J Shoulder Elbow Surg. 2008;17:744–50. https://doi.org/10.1016/j.jse.2008.03.002.
71. Calvo E, Rojas D, Morcillo D, et al. Management of the failed Latarjet procedure. In: Boileau P, editor. Shoulder concepts: arthroscopy, arthroplasty & fractures. Montpellier: Lippincott Williams & Wilkins; 2012.
72. Locher J, Wilken F, Beitzel K, et al. Hill-Sachs off-track lesions as risk factor for recurrence of instability after arthroscopic bankart repair. Arthroscopy. 2016;32:1993–9. https://doi.org/10.1016/j.arthro.2016.03.005.
73. Mohtadi NGH, Chan DS, Hollinshead RM, et al. A randomized clinical trial comparing open and

arthroscopic stabilization for recurrent traumatic anterior shoulder instability: two-year follow-up with disease-specific quality-of-life outcomes. J Bone Joint Surg Am. 2014;96:353–60. https://doi.org/10.2106/JBJS.L.01656.

74. Fabbriciani C, Milano G, Demontis A, et al. Arthroscopic versus open treatment of Bankart lesion of the shoulder: a prospective randomized study. Arthroscopy. 2004;20:456–62. https://doi.org/10.1016/j.arthro.2004.03.001.
75. Archetti Netto N, Tamaoki MJS, Lenza M, et al. Treatment of Bankart lesions in traumatic anterior instability of the shoulder: a randomized controlled trial comparing arthroscopy and open techniques. Arthroscopy. 2012;28:900–8. https://doi.org/10.1016/j.arthro.2011.11.032.
76. Araghi A, Prasarn M, St Clair S, Zuckerman JD. Revision anterior shoulder repair for recurrent anterior glenohumeral instability. Bull Hosp Jt Dis. 2005;62:102–4.
77. Tamai K, Higashi A, Tanabe T, Hamada J. Recurrences after the open Bankart repair: a potential risk with use of suture anchors. J Shoulder Elbow Surg. 1999;8:37–41.

How to Manage Failed Anterior Arthroscopic Repair

2

Vito Bongiorno

2.1 Introduction

Arthroscopic Bankart repair is the most performed procedure for the treatment of post-traumatic capsulo-labral detachment [1, 2]. Although some authors still perform open Bankart repair claiming better results, the arthroscopic technique is considered the gold standard for anterior stabilization [3, 4].

Recurrence of instability after arthroscopic Bankart repair is increasing over years probably because of the large number of procedures done by surgeons performing few procedures per year (i.e., lack of adequate training) [5]. Another cause might be the excessive use of arthroscopic procedures in the attempt to treat lesions that needed to be treated differently [6, 7].

Management of failed arthroscopic repair for anterior shoulder instability needs to focus mainly on the reasons for failure. Except for new trauma occurring on the operated shoulder at least 9–12 months after primary surgery in a patient that has fully recovered, other causes may be wrong surgical technique or, more often, wrong indication. Accurate analysis of the preoperative imaging and clinical history of the patient can be very helpful in understanding the cause of failure.

V. Bongiorno
Polyclinique Notre Dame, Draguignan, France

2.2 Anterior Shoulder Instability: Spectrum of Disease

Traumatic anteroinferior dislocation of the shoulder causes a spectrum of lesions that determine the basis for further episodes of instability.

Instability arises from either soft tissue and/or bone damage. Capsulolabral complex is usually detached and stretched when a trauma occurs, and this lesion can be extended to the inferior labrum and in worst cases involve the whole labrum in a 360° detachment from the glenoid. Anterior glenoid rim may be involved. On the other side, posterior aspect of the humeral head is usually damaged because of the impact with the anteroinferior glenoid (Hill-Sachs lesion). This lesion can be very medial and deep on the cartilage surface, thus engaging with the anterior glenoid rim and causing dislocation. Di Giacomo et al. [8] introduced the new concept of "on-track/off-track lesion" to establish an algorithm for the treatment of the anterior instability.

The size of anteroinferior glenoid bone loss has been widely investigated to establish a treatment algorithm and any further recurrence [9]. Addressing glenoid bone loss must take into account the type of lesion. In case of glenoid fracture with a small fragment, especially in recurrent instability, fragment can undergo resorption, and bone loss is typically larger than the bone fragment as it appears on computed tomography (CT) scan [10].

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_2

2.3 Causes of Failure

A new high-energy trauma is a possible cause of failure if it occurs at least after 9–12 months from surgery and after total recovery and return to sport/work. Unfortunately, many times the reason of failure is related to errors in surgical technique (Fig. 2.1) or indication.

Indeed, the number of anchors, number of sutures, and position of the lower anchor are all crucial factors determining the outcome of the repair [11]. However, wrong indication is probably the most common cause of failure. According to Boileau et al. [11], factors like age at surgery (less than 20 years), type (overhead) and level (competitive) of sport participation, hyperlaxity, and bone loss either at the level of anteroinferior glenoid rim and the posteromedial humeral head are all concurrent to increased failure rate. Porcellini et al. [12] also described a higher risk of recurrence following arthroscopic Bankart repair on the basis of sex, age, and time from the first dislocation to surgery.

Randelli and Taverna [13] published a paper analyzing a young athlete after first dislocation and observed that patients with high-demanding shoulder activities had higher risk of failure, thus suggesting early surgical stabilization. Aboalata et al. [14] also described better clinical and radiological results in patients stabilized after first dislocation, and Crall et al. [15] evaluated the cost-effectiveness of this strategy showing better overall results with early surgery.

2.4 Evaluation

The first thing to analyze in case of failure of anterior stabilization is preoperative clinical history and imaging. In addition, surgical report and postoperative rehabilitation protocol should be analyzed. In case of failure of arthroscopic Bankart repair, accurate assessment of soft tissues and bone loss, either on the glenoid and on the humeral head, is highly recommended. Magnetic resonance arthrography (MRA) and CT scan are the most accurate imaging studies (Fig. 2.2).

2.5 Treatment Algorithm

After a Bankart repair, failure can be diagnosed when recurrence of dislocation does occur. When regained stability is enough for a sedentary lifestyle, the patient cannot return to full sport activities but does not always complain of persistent

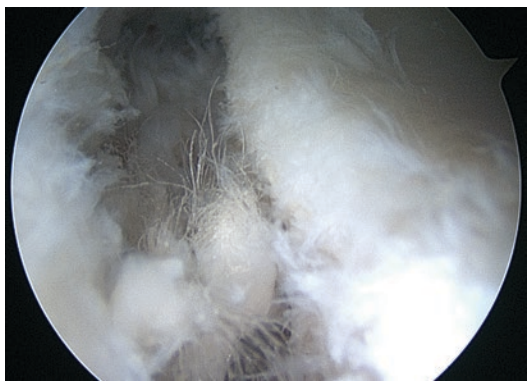

Fig. 2.1 Right shoulder. Arthroscopic view from anterosuperior portal. Surgical error: previous anchors were not positioned on the glenoid rim

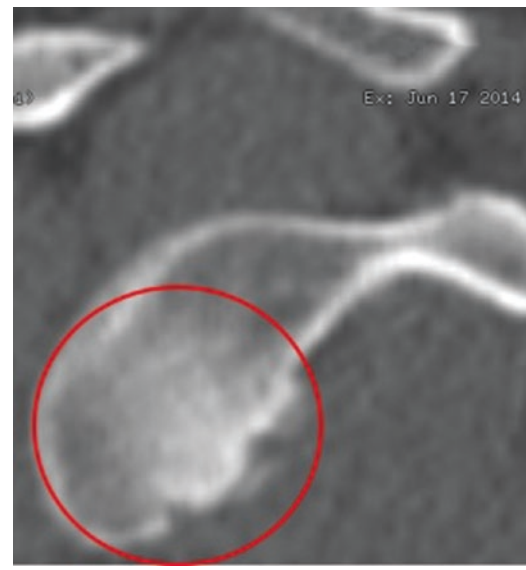

Fig. 2.2 Right shoulder. 2D CT scan. Anchor insertion site is usually the weakest point. Glenoid bone loss is calculated

instability. This explains some good results reported in short-term follow-up studies of untreated failed arthroscopic Bankart repair. Most of the times, patients are limited by a permanent minor instability, which can be painful and evolve toward more symptomatic instability and/or osteoarthritis (OA) over time.

Revision by new soft tissue repair to the glenoid has limited indications as a first solution for Bankart failure since soft tissue quality decreases over time so the hammock effect would not be reproduced properly by the weak ligament (Fig. 2.3). Nevertheless, in case of failure, revision by arthroscopic soft tissue repair should be always considered. If no glenoid erosion nor fractures are evident and, during arthroscopic assessment, the capsule-ligamentous structures show good appearance, thickness, and resistance under tension, a new soft tissue repair can be considered. The repair should be done with at least three double-loaded anchors by shifting the capsule and ligaments from posterior to anterior and from inferior to superior.

Humeral head Hill-Sachs "remplissage" has to be taken into account when the bone loss is deep and medial because it can be engaging and determine failure of treatment and early OA in a stabilized shoulder [16].

Coracoid transfer as described by Latarjet-Patte or Bristow, also performed arthroscopically as described by Lafosse et al. [17] and Boileau et al. [18], is a reliable solution in case of failed anterior arthroscopic repair with severe glenoid bone loss, insufficient ligaments, and/or large Hill-Sachs lesion. Particularly, in case of failure of previous surgery, the sling effect of the conjoint tendon is necessary to achieve the needed stability for patients demanding high performances (sport or work).

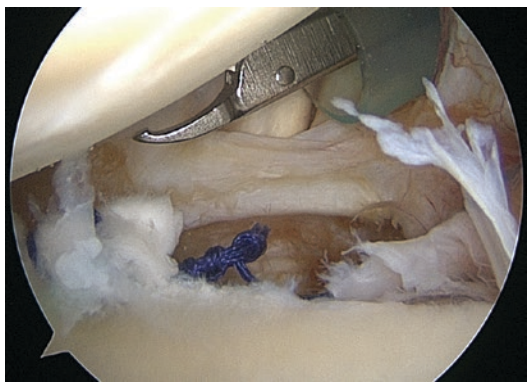

Fig. 2.3 Right shoulder. Arthroscopic view from posterior portal: soft tissue insufficiency

A simple bone graft would not be enough without proper ligament complex. This is a necessary condition when considering only isolated bone graft glenoid augmentation. Conversely, an isolated bone graft with iliac crest (open or arthroscopic) can be the only solution in case of failure of anterior stabilization with Latarjet or Bristow procedure [19, 20].

References

1. Castagna A, Garofalo R, Conti M, Flanagin B. Arthroscopic Bankart repair: have we finally reached a gold standard? Knee Surg Sports Traumatol Arthrosc. 2016;24(2):398–405.
2. Browe DP, Rainis CA, McMahon PJ, Debski RE. Injury to the anteroinferior glenohumeral capsule during anterior dislocation. Clin Biomech (Bristol, Avon). 2013;28(2):140–5.
3. Virk MS, Manzo RL, Cote M, Ware JK, Mazzocca AD, Nissen CW, Shea KP, Arciero RA. Comparison of time to recurrence of instability after open and arthroscopic Bankart repair techniques. Orthop J Sports Med. 2016;4(6):2325967116654114.
4. Wang L, Liu Y, Su X, Liu S. A meta-analysis of arthroscopic versus open repair for treatment of Bankart lesions in the shoulder. Med Sci Monit. 2015;21:3028–35.
5. Middleton RM, Vo A, Ferguson J, Judge A, Alvand A, Price AJ, Rees JL. Can surgical trainees achieve arthroscopic competence at the end of training programs? A cross-sectional study highlighting the impact of working time directives. Arthroscopy. 2017;33(6):1151–8.
6. Ho AG, Gowda AL, Michael Wiater J. Evaluation and treatment of failed shoulder instability procedures. J Orthop Traumatol. 2016;17(3):187–97.
7. Walch G, Boileau P, Levigne C, Mandrino A, Neyret P, Donell S. Arthroscopic stabilization for recurrent anterior shoulder dislocation: results of 59 cases. Arthroscopy. 1995;11(2):173–9.
8. Di Giacomo G, Itoi E, Burkhart SS. Evolving concept of bipolar bone loss and the hill-Sachs lesion: from "engaging/non-engaging" lesion to "on-track/off-track" lesion. Arthroscopy. 2014;30(1):90–8.
9. Baudi P, Righi P, Bolognesi D, Rivetta S, Rossi Urtoler E, Guicciardi N, Carrara M. How to identify and calculate glenoid bone deficit. Chir Organi Mov. 2005;90(2):145–52.

10. Nakagawa S, Mizuno N, Hiramatsu K, Tachibana Y, Mae T. Absorption of the bone fragment in shoulders with bony Bankart lesions caused by recurrent anterior dislocations or subluxations: when does it occur? Am J Sports Med. 2013;41(6):1380–6.
11. Boileau P, Villalba M, Héry JY, Balg F, Ahrens P, Neyton L. Risk factors for recurrence of shoulder instability after arthroscopic Bankart repair. J Bone Joint Surg Am. 2006;88(8):1755–63.
12. Porcellini G, Campi F, Pegreffi F, Castagna A, Paladini P. Predisposing factors for recurrent shoulder dislocation after arthroscopic treatment. J Bone Joint Surg Am. 2009;91(11):2537–42.
13. Randelli P, Taverna E. Primary anterior shoulder dislocation in young athletes: fix them! Knee Surg Sports Traumatol Arthrosc. 2009;17(12):1404–5.
14. Aboalata M, Plath JE, Seppel G, Juretzko J, Vogt S, Imhoff AB. Results of arthroscopic Bankart repair for anterior-inferior shoulder instability at 13-year follow-up. Am J Sports Med. 2017;45(4):782–7.
15. Crall TS, Bishop JA, Guttman D, Kocher M, Bozic K, Lubowitz JH. Cost-effectiveness analysis of primary arthroscopic stabilization versus nonoperative treatment for first-time anterior glenohumeral dislocations. Arthroscopy. 2012;28(12):1755–65.
16. Purchase RJ, Wolf EM, Hobgood ER, Pollock ME, Smalley CC. Hill-sachs "remplissage": an arthroscopic solution for the engaging hill-sachs lesion. Arthroscopy. 2008;24(6):723–6.
17. Lafosse L, Lejeune E, Bouchard A, Kakuda C, Gobezie R, Kochhar T. The arthroscopic Latarjet procedure for the treatment of anterior shoulder instability. Arthroscopy. 2007;23(11):1242.e1–5.
18. Boileau P, Mercier N, Old J. Arthroscopic Bankart-Bristow-Latarjet (2B3) procedure: how to do it and tricks to make it easier and safe. Orthop Clin North Am. 2010;41(3):381–92.
19. Giannakos A, Vezeridis PS, Schwartz DG, Jany R, Lafosse L. All-arthroscopic revision Eden-Hybinette procedure for failed instability surgery: technique and preliminary results. Arthroscopy. 2017;33(1):39–48.
20. Taverna E, Golanò P, Pascale V, Battistella F. An arthroscopic bone graft procedure for treating anterior-inferior glenohumeral instability. Knee Surg Sports Traumatol Arthrosc. 2008;16(9):872–5.

How to Manage Failed Anterior Open Repair

3

Boris Poberaj

3.1 Introduction

The recurrence of instability has been noted in all stabilization procedures for anterior shoulder instability. Open Bankart repair has been for many years the golden standard approach, with a better success rate than the arthroscopic procedure. There are many modifications of classical Bankart procedure with or without coracoid osteotomy and other open techniques for anterior stabilization, such as Putti-Platt, Bojtchev, Bristow, Latarjet, etc. The reasons for surgical failure are related to technical errors, unrecognized pathology like major glenoid bone loss or Hill-Sachs lesion, missed HAGL lesion, and neglecting increased ligament laxity. On the other side, patient's compliance and risk factors like participation in contact sports at a competitive level or prolonged time from first dislocation to surgery can play an important role in recurrence. The most devastating consequence due to failed stabilization surgery is early osteoarthritis (OA) due to hardware or bone block malposition.

3.2 Clinical Approach and Diagnostics

Patients may very well recognize the episode of shoulder re-dislocation or subluxation, as they have previous experiences with it. It is important to take into consideration the history of the injury mechanism.

Clinical examination consists of stability tests and range of motion measurement, especially external rotation at the side and at 90° of abduction. SLAP tests and rotator cuff tests should be included, particularly for subscapularis tendon, which is always taken down during the open surgical approach. Further investigations are planned according to previous surgical intervention. Detailed surgical report, together with postoperative scars, would give us an important information about the type of soft tissue or bone block procedures that were done and also about the implants. X-ray assessment is always the first choice followed by magnetic resonance imaging (MRI) or computed tomography (CT) with or without arthrography. The interpretation of results is in general quite challenging due to implant artifacts, postoperative anatomical changes, scar tissue, etc. This is specifically true for soft tissue assessment, whereas bone defects are more easily assessed. Beside bone integrity of the glenoid and humeral head, coracoid process integrity should be assessed in order to exclude coracoid fracture, which can compromise

B. Poberaj
Aspetar Hospital, Doha, Qatar
e-mail: Boris.Poberaj@aspetar.com

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_3

coracoid transfer in revision procedure. Finally, arthroscopy is additional and a very important tool to get the most comprehensive information about intra-articular pathological changes. Thus, arthroscopy is a mandatory part of revision surgical planning.

3.3 Surgical Planning

The final goal of revision surgery is a "stable shoulder." As for the primary surgical stabilization planning, individual risk factors for each patient must be taken into account, such as age, type of sports activity, patient compliance, etc. Other important considerations should be questioned as follows: What initiated failure? What is the quality of previously addressed capsulolabral complex and extent of bony defects of the glenoid and humeral head? What are the anatomical rearrangements due to initial bone block procedure?

The importance of customized procedure for each case is the mainstay of surgical planning.

3.4 Surgical Revision Options

There is no universal technique for all failed anterior instability repairs. Revision surgery should always start with arthroscopic evaluation. Quality of previously addressed tissues, like capsule and ligaments, should be carefully assessed, as this plays a conclusive role in decision-making to proceed with soft tissue reconstruction or bone block procedure. In addition, arthroscopy provides valuable information about rotator cuff, cartilage, LHB, and possible loose bodies.

Advances in arthroscopic techniques with new, small-sized implants have contributed with improving results of revision arthroscopic stabilization procedures. Small-sized implants can be easily positioned between previous placed anchors. The number and the type of suture anchors (single- or double-loaded anchors) to be used in revision setting have not been clearly established, albeit at least three to four anchors are recommended [1, 2]. The potential advantage of arthroscopy in soft tissue reconstruction consists in addressing the instability problem more comprehensively, with the option of additional re-tensioning of posterior capsule or remplissage procedure. In addition, arthroscopic approach preserves subscapularis function. Despite the fact, there have been no randomized controlled studies comparing outcomes between arthroscopic and open revision Bankart repair [1].

Revision cases with glenoid bone deficiency and/or poor soft tissue quality need some kind of bone block reconstruction [3], such as modifications of the Latarjet procedure or free bone blocks procedures by use of autograft from the iliac crest or allografts. Most of these procedures can be performed arthroscopically by experienced surgeons.

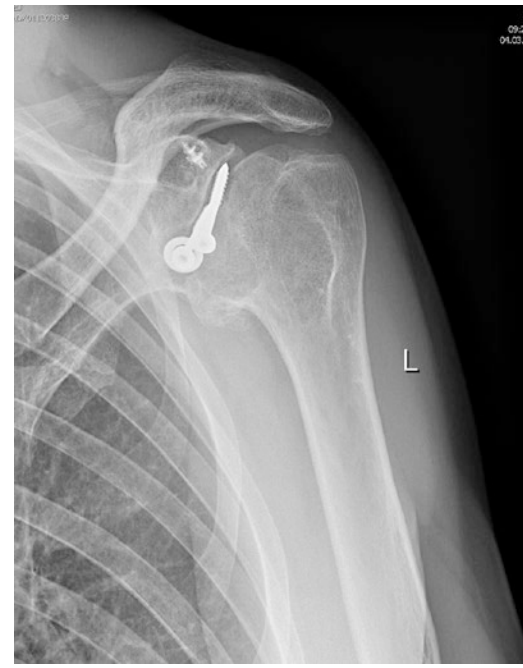

Fig. 3.1 Latarjet procedure in a kickboxer with recurrent instability after high-energy trauma. X-ray shows bended screws

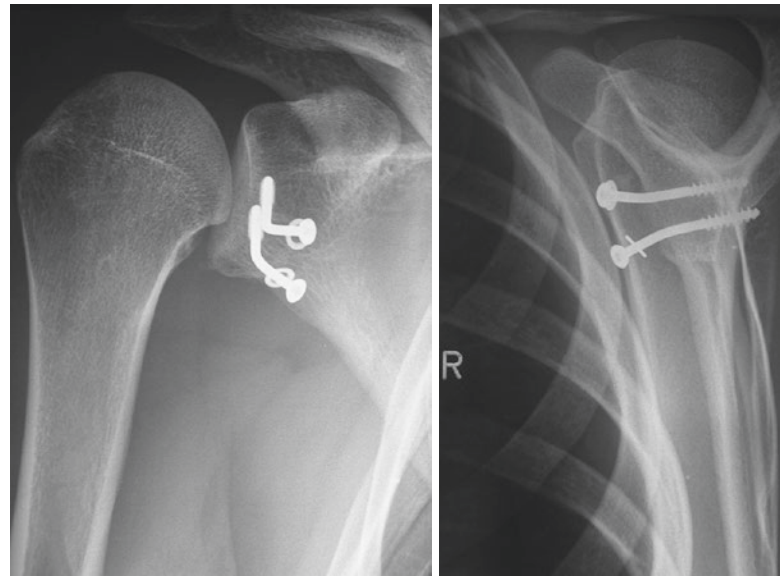

Fig. 3.2 Failed Latarjet with displaced hardware in epileptic patient. Advanced OA is present

In case of failure of previous bone block procedure, the surgical solution becomes more complex (Fig. 3.1). Again, arthroscopy helps to assess intra-articular pathology and in some cases can even be the technique of choice. Failed bone block procedure does not necessarily mean a fractured and displaced bone graft. In such cases, preserved capsule-ligamentous complex can be arthroscopically re-tensioned and remplissage can be added.

In more devastating cases with bone block fracture displacement and hardware damage (Fig. 3.2), the surgical solution is an open approach with hardware removal, bone block replacement, and viable soft tissue reconstruction.

Finally, glenohumeral OA represents the late sequela of instability history. In general, it is not possible to determine whether dislocations, first surgery, or revision surgery are potential triggers, unless the hardware conflict or bone block malposition is clearly find to be the cause (Fig. 3.3). These cases usually represent with stiffness and pain and they are "super stable." Arthroscopy might help in the early stages of OA with joint debridement, capsulotomy, and even hardware or bone block conflict removal (Fig. 3.4).

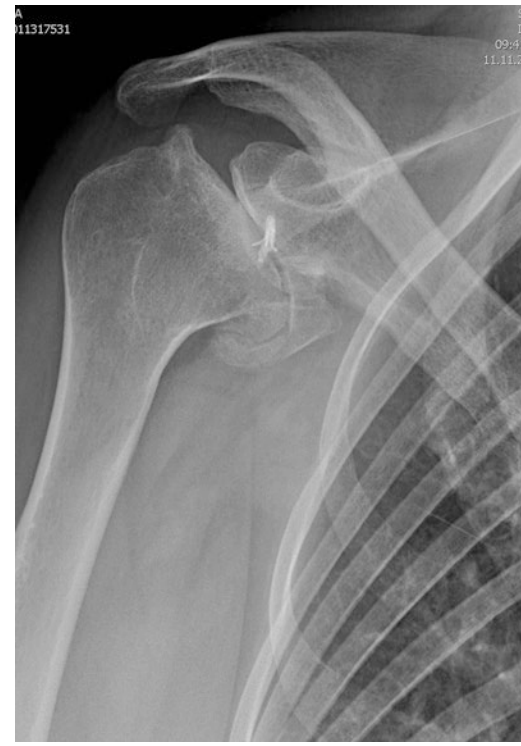

Fig. 3.3 Advanced OA secondary to open Bankart repair with metallic anchor in direct conflict with humeral head

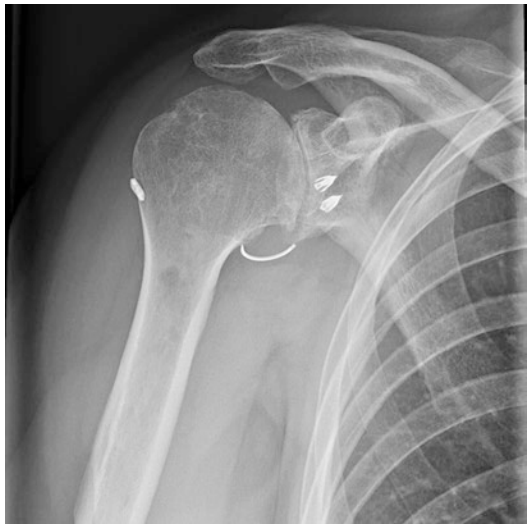

Fig. 3.4 Early OA secondary to open Bankart repair. Metallic anchors present with "forgotten" needle inside the soft tissue

References

1. Abouali JAK, Hatzantoni K, Holtby R, Veillette C, Theodoropoulos J. Revision arthroscopic Bankart repair. Arthroscopy. 2013;29:1572–8.
2. Neviaser AS, Benke MT, Neviaser RJ. Open Bankart repair for revision of failed prior stabilization: outcome analysis at a mean of more than 10 years. J Shoulder Elbow Surg. 2015;24:897–901.
3. Friedman LGM, Griesser MJ, Miniaci AA, Jones MH. Recurrent instability after revision anterior shoulder stabilization surgery. Arthroscopy. 2014;30:372–81.

4 Management of Failed Posterior and Multidirectional Instability Repair

Roman Brzóska, Hubert Laprus, Paweł Ranosz, Patryk Kłaprocz, and Tomasz Rynkiewicz

4.1 Introduction

It is well known that posterior and multidirectional instability (MDI) of the glenohumeral (GH) joint are less common than anterior instability. Posterior dislocation is estimated to occur in around 5% of all GH dislocations [1–5]. The prevalence of posterior instability (PI) and MDI is highest in young men (twice more often than in women) and in elderly patients. In young patients instability is mainly produced by traumatic high-energy accidents and sport injuries, especially in contact and throwing sports. In the elderly, it is mainly caused by traumas like high altitude falls [3, 6, 7].

Comprehensive nonoperative treatment should be the first step of management of PI and MDI. Patients who have failed several months of conservative therapy might be proper candidates for operative treatment.

The results of surgical treatment of PI and MDI are significantly worse than those reported for anterior instability. Failure rate of surgical treatment is 30–70% for open procedures [8], while arthroscopic procedures have success rate higher than 90% [8–10]; therefore, the latter should be the treatment of choice.

When recurrent shoulder instability occurs after surgery, a surgeon must consider why the previous surgery has failed. Main reasons for failure of primary surgery in PI and MDI are inadequate preoperative assessment and wrong initial diagnosis.

The purpose of this chapter is to underline the common mistakes in PI and MDI repair and to present how to perform a successful revision surgery.

4.2 Wrong Diagnosis at First Repair

In order to avoid future complications, a surgeon must be certain to discriminate PI from MDI. It must be determined if there is a traumatic accident in patient's history or the symptoms appeared because of recurrent microtraumas. A thorough physical exam should be performed to test all types of instability. Specific tests include the jerk test and the Kim test [11]. To assess osseous and

R. Brzóska (✉)
Orthopaedic/Upper Limb, St. Luke's Hospital, Bielsko-Biała, Poland
e-mail: brzoska@lukasza.pl

H. Laprus · P. Kłaprocz
Department of Orthopedics, St Luke's Hospital, Bielsko-Biała, Poland
e-mail: laprus@lukasza.pl

P. Ranosz
Boni Fratres Catoviensis, Katowice, Poland

T. Rynkiewicz
Ludwik Rydygier Memorial Specialized Hospital, Krakow, Poland

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_4

soft tissues, imaging studies like computed tomography (CT) and magnetic resonance imaging (MRI) should be performed. A 3-Tesla MRI and MR arthrography (MRA) with intra-articular contrast medium can be helpful to enhance the visualization of concomitant lesions associated with recurrent instability, such as reverse humeral avulsion of the glenohumeral ligaments (rHAGL lesion) [12]. If there are no posterior labral lesions or posterior glenoid defects, the rHAGL lesion should be considered as cause of instability.

Posterior instability may result from subtle posterior subluxations as well as from a traumatic event resulting in dislocation. Differential diagnosis between MDI and subluxation-type posterior instability requires a thorough physical exam consisting of anterior, posterior, and inferior relocation tests with the patient in supine position with the arm in 90° of abduction.

Generalized ligament laxity (GLL) might be a substantial ground for operative failure as well. GLL has to be assessed in patients with MDI and PI symptoms. The Beighton score is a simple and reliable tool to assess laxity, and the score of 4 or more (out of 9 points) entitles to the clinical diagnosis of GLL [13]. These patients are potential candidates for recurrent instability and require individualized treatment. According to Tillander et al., it is known that successful treatment in cases with multidirectional hyperlaxity essentially depends on presenting symptoms [14]. For patients in whom instability is the main symptom, solely conservative treatment might be insufficient, and a combination of surgery and rehabilitation is recommended. Patients presenting only pain without symptoms of previous instability are a problematic group, because long-term outcome is less satisfactory.

Damaged posterior labrum is a significant pain producer, and its mechanical stimulus during posterior instability is painful. Positive Kim test that relies on irritating damaged posterior labrum unambiguously proves it in contrast to anterior labrum lesions, which do not give symptoms until anterior instability occurs [11]. The pain is a helpful indicator in differentiation of instability causes. Non-traumatic MDI caused by hyperlaxity without labral injury typically proceeds painlessly as opposed to post-traumatic labral damage, which causes severe pain. Nevertheless, dysplastic painful labrum might be a trap in the diagnostic process and should be investigated.

4.3 Postoperative Complications

The patients with unsatisfactory postoperative outcome typically report persistent pain, subjective instability, decreased range of motion (ROM), or painful cracking in the joint area. For arthroscopic methods, persistent pain concerns around 12% of patients, and insufficient stabilization involve 8–9%, whereas other complications are observed rather uncommonly [8].

4.3.1 Bone Graft Healing

Complications of bone block in posterior bony augmentation procedures as treatment of PI and MDI are well demonstrated in the literature [6, 15–17] . Due to a difficult access to posterior glenoid, the risk of bone block malposition is higher than for coracoid process malposition in the Latarjet procedure. Additionally, blood supply of posterior bone block area is typically less effective than anterior glenoid, and osteolysis of the graft might proceed faster.

Graft osteolysis is the most frequent long-term complication, but it is rather a radiological finding without clinical implications. On the other hand, complete graft resorption is reported by many authors and should always be considered as the possible reason for recurrent instability requiring revision procedures [15–17]. Osteolysis usually starts and proceeds rapidly on the superficial part of the graft. Deeper areas do not suffer from lysis because of appropriate blood supply from previously abraded glenoid. Osteolysis of the unhealed graft might cause its fracture, and free bone fragments can produce typical blocking symptoms like sudden pain, reduced ROM, or audible cracking. Decreased glenoid dimension due to graft lysis can lead to recurrence of PI and requires revision surgery.

Proper posterior glenoid abrasion and optimal graft sizing, placement, and fixation lead to proper anatomical joint restoration and appear to be the most important factors that reduce the risk of osteolysis and guarantee satisfactory long-term outcomes.

4.3.2 Painful Hardware

The use of implants for GH soft tissue fixation is changing constantly. Biodegradable suture anchors have largely replaced other kind of fixation devices like metallic anchors and point tack fixation devices, but issues with them have not been resolved so far. The clinical symptoms of these complications typically reveal as audible crepitus, growing pain, and restricted ROM that might be caused by intra-articular prominent parts of the implants. To confirm implant malposition, radiographs and MRI might be necessary. Removal of arthroscopic failed implants should be considered to prevent chondral attrition and injury [18].

Drilling malposition, imprecise measuring, and lack of adequate screw length are the most common causes of implanting too long screws. Usually small protrusion is asymptomatic, but patients who present prolonged pain or painful cracking need revision to remove the devices. K-wire breakage during advancement through the glenoid has also been reported [17]. Generally, a broken K-wire stays stable, but sometimes it might migrate anteriorly and contribute to pain symptoms.

4.3.3 Rehabilitation

Many reasons have been reported for rehabilitation failure, such as wrong initial diagnosis, incomplete or inappropriate rehabilitation, low rehabilitation frequency and motivation, weak sensorimotor abilities, and psychological problems.

Regardless of whether a patient with PI or MDI of the shoulder has gone through surgery or not, rehabilitation program usually consists of four phases [19–21]:

1. Immobilization and pain management
2. Initial muscle activation and strengthening; gradual ROM restoration
3. Regaining shoulder girdle muscle strength and endurance
4. Functional and sport-specific exercises

Each of the abovementioned phases has to be conducted carefully and precisely. Patient can move to the next phase of rehabilitation treatment after an objective assessment of achieved goals. During every phase, many therapeutic failures may happen as result of patient and/or physiotherapist fault. These mistakes include early joint strengthening (when the shoulder is still painful or symptoms come back), progressing to strengthening and specific task training when full ROM is not achieved, and too early return to sports activities [21]. Reasons for these mistakes might vary, depending on proper cooperation between patient and clinician. Indeed, rehabilitation programs should be individualized and focused on neuromuscular performance, and no standardized rehabilitation programs can be applied to every patient [22, 23].

Proprioception is a crucial function for appropriate motor control of the shoulder joint [24, 25]. Some of the patients present better joint position sense than others [26]. These patients seem to have greater benefit from rehabilitation. Conversely, patients with poor joint position sense are not able to undergo optimal rehabilitation.

Proper shoulder girdle function depends on correct scapulothoracic movement pattern, and coordination disturbance is very common in shoulder instability [27], albeit its importance is poorly understood. Many patients with positional instability who present transient scapula winging develop subluxation, but there is no evidence of long thoracic nerve dysfunction [5]. Nevertheless, patients with PI or MDI and weak serratus anterior muscle function have an increased disability by protraction and upward rotation of the scapula [24, 28, 29]. It is still unclear whether the asynchrony of scapulothoracic motion is contributory to instability or whether it is acquired compensatory mechanism to prevent posterior escape of the humeral head.

Shoulder instability might be a compensatory effect of restrictions in different body parts, so that whole body functional examination should be always perform initially. Every muscle balance restriction especially in professional atheltes has to be removed if present. After that proper instability rehabilitation protocol could be input [23].

Psychological problems in patients with shoulder instability are also reported [30]. They are able to subluxate shoulder voluntarily using unbalanced muscle force and most often develop instability in adolescence [31]. Rowe et al. reported that if psychological disorders have not been cured before instability treatment, the final outcome is very poor [30].

4.3.4 Adhesive Capsulitis

Adhesive capsulitis is a complication caused by scarring between shoulder tissue layers and requires an individualized treatment protocol. Restriction of passive internal and external rotation caused by contracted coracohumeral ligament and rotator interval is typically observed. Open procedures can cause excessive bleeding and lead to capsulitis more often than arthroscopic treatment. Moreover, prolonged postoperative immobilization is a risk factor for capsulitis development [18], so that postoperative rehabilitation based on early mobilization and passive ROM restoration seems advisable.

Forsythe suggests that capsulitis might be caused by too aggressive rotator interval closure [18]. Harryman reveals that during rotator interval closure by shifting middle glenohumeral ligament (MGHL) to the superior glenohumeral ligament (SGHL), loss of external rotation is often observed, especially in PI and MDI surgery, when excessive rotator interval closure might be the result of imbrication in the adduct arm. In these cases, successful adhesion treatment requires arthroscopic rotator interval release [32].

Biceps inflammation and adhesions might produce capsulitis too. Tonino suggests that intraarticular biceps adhesions should be treated by arthroscopic excision of the biceps tendon [33]. Indeed, we observed very good results with nonoperative treatment of postoperative capsulitis, and therefore we suggest the conservative approach as treatment of choice.

4.3.5 Postoperative Neurological Deficits

Postoperative neurological deficits are reported in 0.2–3% of all shoulder arthroscopic procedures and in 8% of open procedures. The majority of neurological injuries are minor cutaneous nerve lesions. Reliable survey performed in a specialized shoulder surgery center revealed that during 10 years of observation, two cases of neurological deficits were caused by open surgery for PI/MDI and none by arthroscopic procedures [8, 34]. During arthroscopic treatment of PI, posterolateral portal is most often used to visualize posterior and posteroinferior aspect of the shoulder. This portal is typically safe for neurovascular structures like axillary nerve, especially if the surgeon avoids to insert the needle too deeply during portal creating [35].

4.3.6 Osteoarthritis and Chondral Lesions

Osteoarthritis (OA) and chondral lesions might be caused by PI and MDI. Although successful treatment of recurrent PI/MDI is required to avoid early OA, surgical procedures might be a cause of OA. Posterior bone block techniques have proven, satisfactory long-term results; however, late postoperative osteoarthritis caused by the graft has been reported [15]. A recent systematic review revealed that about one-third of patients after bone block procedures for the treatment of PI developed late OA [36, 37]. Unfortunately, OA can also develop as consequence of soft tissue procedures and conservative treatment as well [36]. Reverse Hill-Sachs lesion and posterior glenoid bone loss are significant predictors of late OA [38]. Patients with OA may be asymptomatic or may present symptoms like increasing persistent pain and decreasing

ROM. Conventional radiographs show narrowing of the GH joint space. High-field MRI can show chondral thinning in the early phase of the disease. In those cases, chondral restoring procedures or arthroplasty should be considered, if necessary.

4.3.7 Traumatic Recurrent Posterior Instability

Recurrent instability due to traumatic accident after previous surgery is challenging to treat. Revision surgical procedure has to be chosen according to that performed during the previous surgery. Combinations of bony and soft tissue procedures are usually recommended. For patients who underwent soft tissue treatment like posterior capsulorrhaphy and labral repair/augmentation, capsular shift/plication, modified McLaughlin technique, or infraspinatus advancement, a posterior bony procedure is suggested. On the contrary, for patients treated by bone augmentation, bone defect filling or glenoid axis correction should be considered [6–8, 36, 39].

4.4 Conservative Treatment of PI and MDI

4.4.1 Conservative Treatment of PI

Although many authors agree that treatment of PI of the shoulder should begin with rehabilitation [6, 8, 40, 41], there are a few reports assessing rehabilitation in PI. Results of conservative treatment reveal that recurrence rate is significantly higher in patients with previous failed surgery [39, 42]. Proper strengthening and proprioceptive training diminish pain and improve joint stability in almost two-third of patients with PI and MDI of the shoulder [6, 40]. Rehabilitation programs are more effective for patients with GLL and for those who present instability based on microtraumas. Nonoperative treatment is less satisfactory in traumatic cases [6, 39, 40].

Burkhead and Rockwood, using their own rehabilitation protocol, reported satisfactory results in 70–89% of atraumatic cases, but only in 16% of traumatic ones [40]. Kiss et al. noted 23% of recurrence at their study, where a rehabilitation program begun from patient's education and explanation of baseline condition. Proprioceptive exercises were recommended to improve joint position sense, to reeducate movement pattern and boost strength of the scapula and GH joint. Instruments as mirrors, closed circuit television, PNF patterns, and biofeedback were used to correct and train scapula and GH joint. Stabilization was obtained by muscle balance improvement with proprioceptive and strengthening exercises in closed kinetic chain. Patients performed stamina training as well. Occupational therapy and home exercise program were recommended to maintain shoulder function [42].

Takwale et al. also presented a rehabilitation protocol for involuntary shoulder instability. They identified abnormal movement pattern and muscle activity. Most of the patients with PI demonstrate less active external rotators, posterior deltoid and hyperactive of internal rotators, anterior deltoid, and latissimus dorsi. Additionally, patients medially rotate the GH joint and then initiate movement of the shoulder with a reversed scapular action so that the inferior angle "wings out" and the scapula is prevented from protracting forward and upward in the usual smooth manner. After appropriate concentric and eccentric muscle work of scapula is obtained, the next step consists of initiating right activation of movement pattern of GH joint [41]. In order to centralize humeral head in the glenoid, activation of external rotators is recommended most often [41, 43].

4.4.2 Conservative Treatment of MDI

Exercises seem to be beneficial for atraumatic MDI [19, 40, 42, 44–46]. Current literature indicates that conservative treatment focused on strengthening of the rotator cuff, pectoralis major, biceps brachii, triceps brachii, and deltoid muscles increases muscle activity, but not as much as rehabilitation following arthroscopic

capsular shift. Only patients surgically treated and supported by prolonged proper postoperative rehabilitation achieve complete normal muscle activation. Similar results were observed during evaluation of scapulothoracic rhythm and relative displacement between the rotation centers of the humerus and scapula. Improvement in scapula kinematics and humeral head centralization can be achieved after rehabilitation only, but complete physiological function was regained after surgery and postoperative rehabilitation [44, 47].

Ide et al. tested rehabilitation training for MDI by using orthosis that holds scapula in upward rotation. The program lasted 8 weeks and consisted of isometric exercises for rotator cuff and scapula stabilizers. Later on, isotonic exercises with elastic bands and wall push-up exercises were added. Outcomes demonstrated significant change in Rowe score, increased external and internal rotation strength, and decrease in IR/ER ratio as well [48].

MDI patients have altered movement and muscle activation patterns [28, 49, 50]. However, we do not know whether it is the cause or the effect or perhaps just an adaptative process allowing function in a certain spectrum of disorder. Nevertheless, elimination of existing deficits seems a reasonable objective. Main goal of appropriate rehabilitation course are regaining of correct scapula position and achieving good motion and stability of scapulothoracic articulation. Afterward, we can build up a proper stabilization of the GH joint.

4.5 Surgical Management of Failed PI and MDI Repair

Revision surgery after failed surgical treatment should aim to restore anatomical conditions within the posterior joint structures as closely as possible. Procedures performed on soft tissues are possible in the absence of any significant bone loss. These procedures may turn out to be effective only in the case of a recurring mechanical damage to the posterior glenoid labrum and joint capsule, as well as in the case of traumatic injury to the humeral attachment site of the posterior capsule. In other cases, procedures aiming to fill bone defects or correcting pathological glenoid retroversion are recommended [51].

4.5.1 Posterior Capsule-Labral Complex Repair

This procedure is successful only in the cases without degenerative damage after recurrent separation from glenoid [3, 9, 11, 51]. If the labrum has been cut by previously placed stitches, its restitching becomes impossible, and alternative reconstruction technique should be considered.

Arthroscopic procedure initiates by establishing the posterior viewing portal (Fig. 4.1). The next step is to make the anterolateral portal above the long head of the biceps tendon (in the rotator cuff). Transferring the optics to this port makes it possible to visualize the posterior glenoid area and to assess the damage of the posterior structures (Fig. 4.2). If a decision to repair the posterior labrum is made, a posterolateral working portal located below posterior acromion is created, slightly lateral (about 2 cm) to the posterior access. Both the arthroscopic tools and fixing implants are

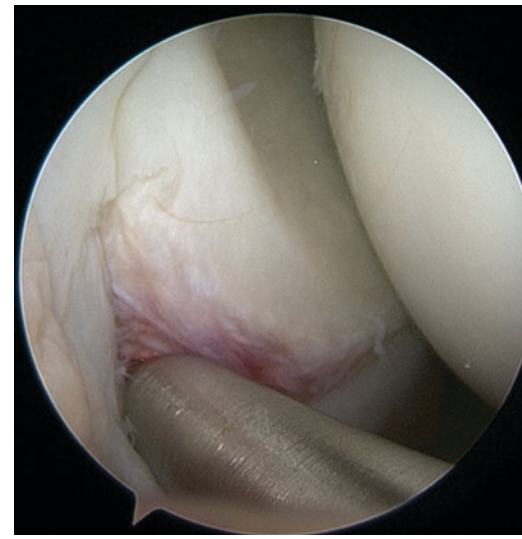

Fig. 4.1 Posterolateral portal view. Posterior labrum reinjury

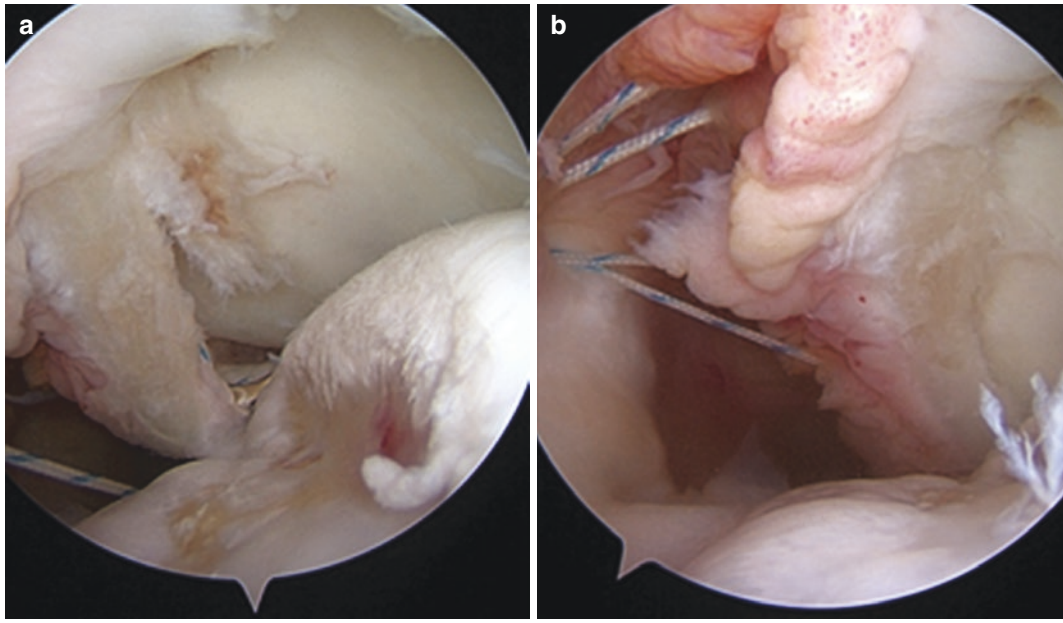

Fig. 4.2 (**a**, **b**) Anterolateral portal view. Posterior labrum reinjury and repair

inserted into the joint through this portal. Before fixing the labrum, posterior edge of the glenoid should be exposed and prepared, for the labrum to be attached to the bleeding bony surface. Labrum mobilization should allow lifting it upward the glenoid. Three fixing implants placed with mattress stitches are usually used for repair. Stitches surrounding the labrum should be avoided, as they can lead to ischemia, labral reinjury, and damages to the cartilage. Mattress stitches avoid contact between the hard stitch material and cartilage of the humeral head, which may contribute to early OA, especially when glenoid retroversion and fixed posterior humeral head subluxation exist [3, 9, 11, 51, 52]. The stitches are usually tied from the posterior access. After stitching the labrum, its stability is checked, and then shoulder stability is assessed by performing internal rotation and posterior shift of the humerus. If the humeral head fracture (Perthes fracture) overlaps with the reconstructed posterior joint labrum, a posterior engagement occurs during this maneuver, and an arthroscopic modified McLaughlin procedure should be considered [38].

4.5.2 Modified McLaughlin Procedure

This procedure can be performed when the fracture in the anterior quadrant of the humeral head (Perthes fracture) is in continuity with the attachment of the subscapularis tendon on the lesser tuberosity. The scope is located in the anterolateral port, and the anterior portal is used as working portal. After fracture debridement, an anchor is placed in its central part [38]. In order to adequately mobilize the subscapularis tendon, it is necessary to cut the connection between the middle glenohumeral ligament (MGHL) and the subscapularis tendon (Fig. 4.3). One double-loaded anchor and two mattress sutures are used to fix the tendon, thus creating tenodesis of the subscapularis into the Perthes fracture (Fig. 4.4). When the fracture involves a large area on the vertical plane, two anchors may be used. In very large bone defect bone, graft is necessary (Fig. 4.5). In case of recurrent MDI with anterior instability combined with PI, inferior glenohumeral ligament (IGHL) repair or anterior capsular plication is necessary before performing McLaughlin procedure.

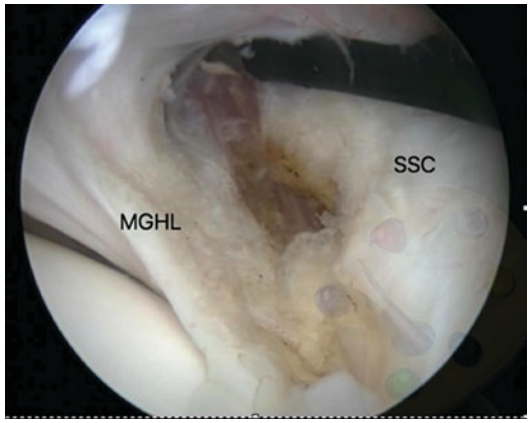

Fig. 4.3 Cutting the connection between middle glenohumeral ligament (MGHL) and subscapularis tendon (SSC)

4.5.3 Open Posterior Bone Block Procedure

This procedure is performed from a posterior approach. The access to the posterior glenoid is achieved by splitting the infraspinatus muscle fibers. Bone graft is fixed with cortical screws. The significant difficulty of this technique is the angle of screws introduction, and in the course of drilling holes in the glenoid, one must be especially careful to avoid neurovascular complications [18]. In these cases, using an arthroscope to verify the length of the screws and secure the front of the glenoid during drilling is an invaluable assistance.

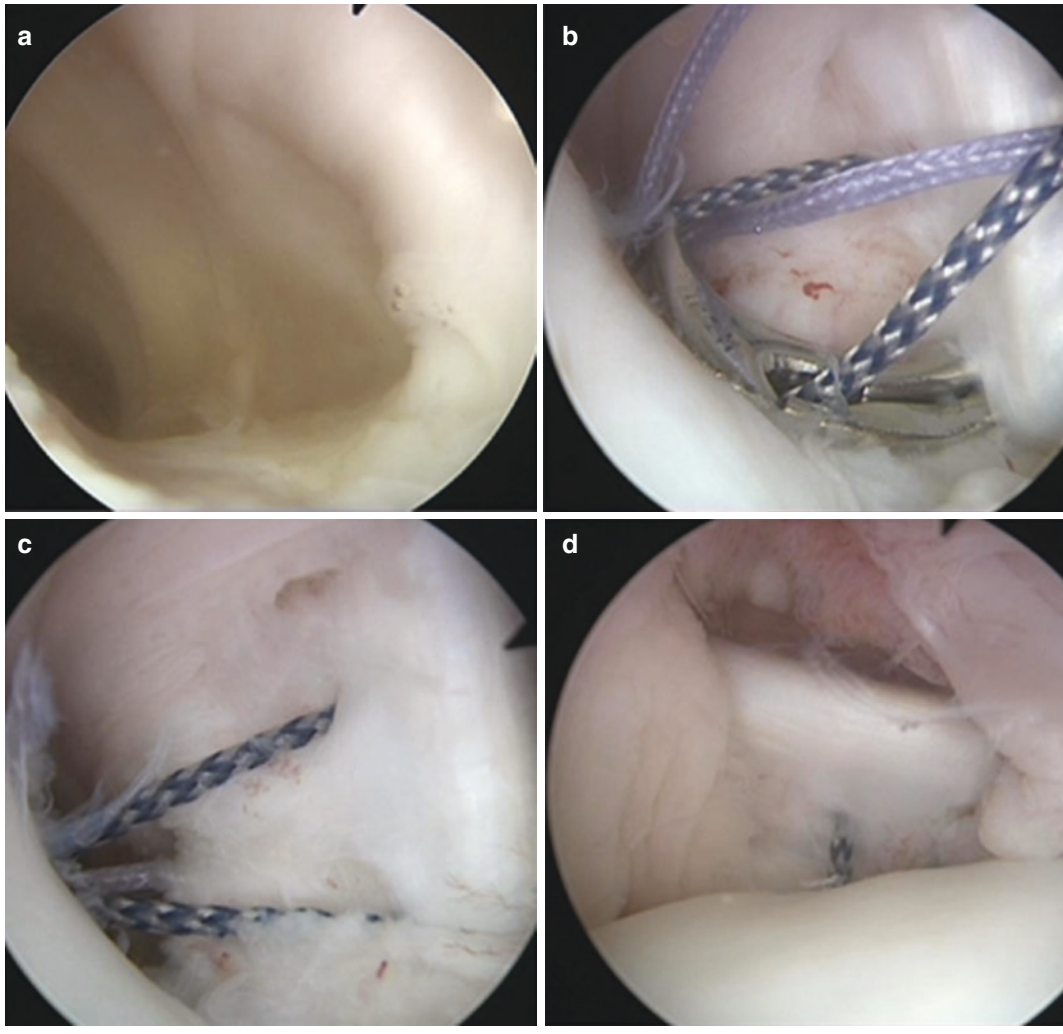

Fig. 4.4 (**a**) Perthes lesion. (**b**) Subscapularis fixation. (**c**) Threads into the tendon. (**d**) Final effect

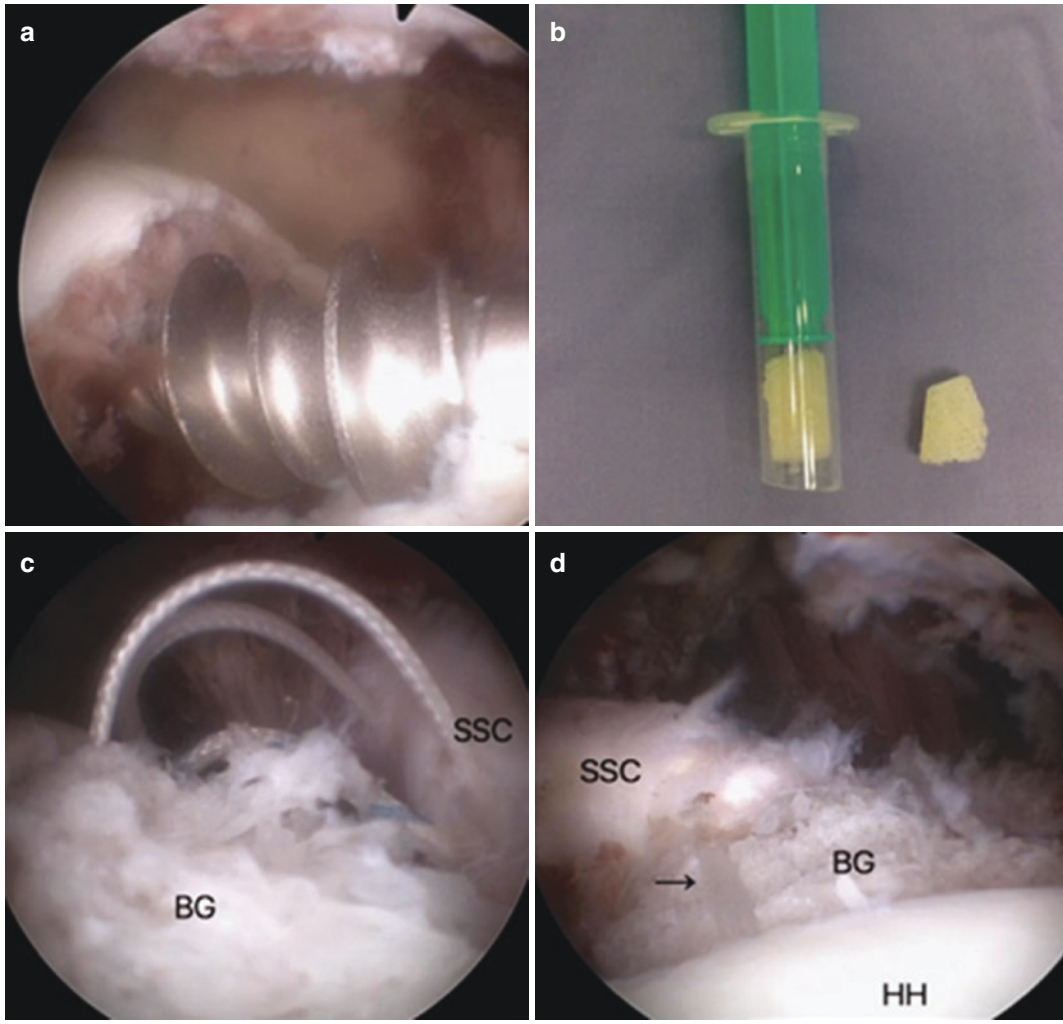

Fig. 4.5 (**a**) Anchor screwing into the humeral head (HH). (**b**) Syringe with graft prepared to insert into the anterolateral portal. (**c**) Graft fixation (*BG* bone graft, *SSC* subscapularis tendon). (**d**) Final effect (arrow: thread)

4.5.4 Arthroscopic Posterior Bone Block Procedure

The use of this technique, in conjunction with the posterior labrum-ligament repair seems to be the most suitable to restore shoulder anatomy. Furthermore, arthroscopic technique allows to spare the posterior deltoid and external rotator muscles and reduces the risk of nerve injuries.

There are two techniques described in literature, which differ in the technique of graft fixation. The first uses cannulated cortical screws, the same used in the Latarjet technique [17, 53, 54] (Fig. 4.6); the second one uses suture anchors. Bone loss can be replenished with allograft or autograft. The latter is the reference standard, as biological safety and good healing were confirmed by literature. Suggested graft size is about 2 cm × 1 cm × 1 cm. In the screw fixation technique, the graft should be a little bit thicker taking into account the expected partial resorption.

Arthroscopic standard posterior portal is performed. In the technique preferred by the authors, the anterolateral portal is created in the rotator interval, above biceps retinaculum. Shifting the

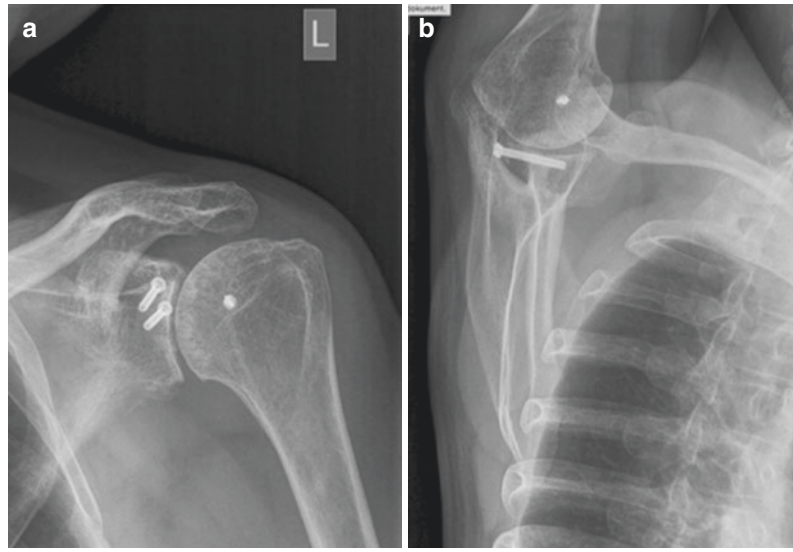

Fig. 4.6 (**a**, **b**) Posterior bone block with modified McLaughlin procedure

scope in front allows a precise assessment of the posterior glenoid loss and posterior soft tissues. At the same time, a third posterolateral portal is created, lateral (about 1.5 cm) to the posterior portal. Its vertical location is identified along a perpendicular line to the bone loss, as assessed with a spinal needle. After separating soft tissues and preparing the bone bed for graft placement on the posterior aspect of the scapular neck, the posterior portal is enlarged to allow the introduction of instruments for fixing the cannulated screws. The graft can be introduced into the joint directly through the posterior portal, after securing it to a guide system (i.e., instrument set for arthroscopic Latarjet procedure). Alternatively, the graft is introduced from the conventional anterior portal through a cannula or a plastic tube (syringe) prepared for this purpose. In this case, the fixing device is connected to the graft inside the joint. Regardless of the introduction technique, the graft must be prepared by drilling the holes for the screws and smoothing the edges [17]. Correct graft placement is rather challenging. It should be at the level of the posterior glenoid loss and around its equator. Graft placed too proximal (above the equator) does not negatively affect joint stability, but is more prone to resorption, and the exposed screw heads may damage the infraspinatus muscle.

4.5.5 Posterior Osteotomy of the Scapula Neck

Glenoid osteotomy is performed either as a revision procedure after failed posterior bone block or as treatment of choice in case of excessive glenoid retroversion (>15°). Osteotomy is traditionally an open procedure, but there is a possibility to perform it arthroscopically.

Before surgery, the localization and direction of osteotomy and opening extent have to be measured based on a CT scan.

It is possible to perform the procedure both in lateral decubitus and in beach-chair position. The incision starts 2.5 cm medially to the posterolateral acromial margin and goes down toward to the axillary crease. After deltoid muscle splitting, division of the infraspinatus is made paying attention to the axillary nerve. The next step is opening the joint capsule above the neck of the scapula. Typically osteotomy is performed parallel to glenoid cavity, being careful not to injure the suprascapular nerve which is supposed to be previously visualized and palpated before the retractor positioning.

Osteotomy starts on the spinoglenoid notch and runs down to the inferior margin of the scapula neck. Before cutting off, two K-wires have to be drilled laterally to the osteotomy. Cutting is per-

formed by chisel or oscillating saw aiming toward the base of the coracoid process and parallel to the K-wires. Osteotomy opening, as measured before surgery, has to be confirmed intraoperatively. A wedge-shaped bone graft is harvested from the iliac crest and inserted into the osteotomy line. Some authors recommend graft dimensions of minimum 3 cm in length and 1.5 cm in height.

Recent modification of the technique suggests stabilizing the graft with a buttress radius plate. In patients with loose posterior capsule, additional capsulorrhaphy can be performed [55, 56].

References

1. Ahrens P. Arthroscopic treatment of posterior shoulder instability. Arthroscopy. 2015;31(3):555–60.
2. Blomquist J, Solheim E, Liavaag S, Schroder CP, Espehaug B, Havelin LI. Shoulder instability surgery in Norway: the first report from a multicenter register, with 1-year follow-up. Acta Orthop. 2012;83(2):165–70.
3. Bradley JP, McClincy MP, Arner JW, Tejwani SG. Arthroscopic capsulolabral reconstruction for posterior instability of the shoulder: a prospective study of 200 shoulders. Am J Sports Med. 2013;41(9):2005–14.
4. Chalmers PN, Hammond LJ, Juhan T, Romeo AA. Revision posterior shoulder stabilization. J Shoulder Elb Surg. 2013;22(9):1209–20.
5. Robinson CM, Aderinto J. Recurrent posterior shoulder instability. J Bone Joint Surg Am. 2005;87(4):883–92.
6. Fronek J, Warren RF, Bowen M. Posterior subluxation of the glenohumeral joint. J Bone Joint Surg Am. 1989;71:205–16.
7. Robinson CM, Seah M, Akhtar MA. The epidemiology, risk of recurrence, and functional outcome after an acute traumatic posterior dislocation of the shoulder. J Bone Joint Surg Am. 2011;93(17):1605–13.
8. Provencher MT, LeClere LE, King S, McDonald LS, Frank RM, Mologne TS, Ghodadra NS, Romeo AA. Posterior instability of the shoulder: diagnosis and management. Am J Sports Med. 2011;39(4):874–86.
9. Bradley JP, Baker CL, Kline AJ, Armfield DR, Chhabra A. Arthroscopic capsulolabral reconstruction for posterior instability of the shoulder: a prospective study of 100 shoulders. Am J Sports Med. 2006;34(7):1061–71.
10. Provencher MT, Bell SJ, Menzel KA, Mologne TS. Arthroscopic treatment of posterior shoulder instability: results in 33 patients. Am J Sports Med. 2005;33(10):1463–71.
11. Kim SH, Park JS, Jeong WK, Shin SK. The Kim test: a novel test for posteroinferior labral lesion of the shoulder-- a comparison to the jerk test. Am J Sports Med. 2005;33(8):1188–92.
12. Antosh IJ, Tokish JM, Owens BD. Posterior shoulder instability: current surgical management. Sports Health. 2016;8(6):520–6.
13. Johnson SM, Robinson CM. Shoulder instability in patients with joint hyperlaxity. J Bone Joint Surg Am. 2010;92(6):1545–57.
14. Tillander B, Lysholm M, Norlin R. Multidirectional hyperlaxity of the shoulder: results of treatment. Scand J Med Sci Sports. 1998;8:421–5.
15. Barbier O, Ollat D, Marchaland JP, Versier G. Iliac bone-block autograft for posterior shoulder instability. Orthop Traumatol Surg Res. 2009;95(2):100–7.
16. Smith T, Goede F, Struck M, Wellmann M. Arthroscopic posterior shoulder stabilization with an iliac bone graft and capsular repair: a novel technique. Arthrosc Tech. 2012;1(2):e181–5.
17. Schwartz DG, Goebel S, Piper K, Kordasiewicz B, Boyle S, Lafosse L. Arthroscopic posterior bone block augmentation in posterior shoulder instability. J Shoulder Elb Surg. 2013;22(8):1092–101.
18. Forsythe B, Ghodadra N, Romeo AA, Provencher MT. Management of the failed posterior/multidirectional instability patient. Sports Med Arthrosc. 2010;18(3):149–61.
19. Bahu MJ, Trentacosta N, Vorys GC, Covey AS, Ahmad CS. Multidirectional instability: evaluation and treatment options. Clin Sports Med. 2008;27:671–89.
20. Misamore GW, Sallay PI, Didelot W. A longitudinal study of patients with multidirectional instability of the shoulder with seven- to ten-year follow-up. J Shoulder Elb Surg. 2005;14:466–70.
21. Wilk KE, Macrina LC, Reinold MM. Non-operative rehabilitation for traumatic and atraumatic glenohumeral instability. N Am J Sports Phys Ther. 2006;1(1):16–31.
22. Watson L, Warby S, Balster S, Lenssen R, Pizzari T. The treatment of multidirectional instability of the shoulder with a rehabilitation program: part 1. Shoulder Elbow. 2016;8(4):271–8.
23. Cools AM, Borms D, Castelein B, Vanderstukken F, Johansson FR. Evidence-based rehabilitation of athletes with glenohumeral instability. Knee Surg Sports Traumatol Arthrosc. 2016;24(2):382–9.
24. Myers JB, Lephart SM. The role of the sensorimotor system in the athletic shoulder. J Athl Train. 2000;35(3):351–63.
25. Riemann B, Lephart SM. The sensorimotor system, part I: the physiologic basis of functional joint stability. J Athl Train. 2002;37(1):71–9.
26. Herrington L, Horsley I, Rofl C. Evaluation of shoulder joint position sense in both asymptomatic and rehabilitated professional rugby players and matched controls. Phys Ther Sport. 2010;11(1):18–22.
27. Warner JJ, Micheli LJ, Arslanian LE, Kennedy J, Kennedy R. Scapulothoracic motion in normal shoulders and shoulders with glenohumeral instability and impingement syndrome. A study using Moire topographic analysis. Clin Orthop. 1992;285:191–9.

28. Morris AD, Kemp GJ, Frostick SP. Shoulder electromyography in multidirectional instability. J Shoulder Elb Surg. 2004;13:24–9.
29. Young MS. Electromyographic biofeedback use in the treatment of voluntary posterior dislocation of the shoulder: a case study. J Orthop Sports Phys Ther. 1994;20(3):171–5.
30. Rowe CR, Pierce DS, Clarks JG. Voluntary dislocation of the shoulder: a preliminary report on a clinical, electromyographic and psychiatric study of twenty-six patients. J Bone Joint Surg Am. 1973;55-A:445–59.
31. Kessel L. Posterior shoulder dislocation. In: Kessel L, Bayley I, editors. Clinical disorders of the shoulder. 2nd ed. New York: Churchill Livingstone; 1986. p. 125–37.
32. Harryman DT, Sidles JA, Harris SL, Matsen FA. The role of the rotator interval capsule in passive motion and stability of the shoulder. J Bone Joint Surg Am. 1992;74(1):53–66.
33. Tonino PM, Gerber C, Itoi E. Complex shoulder disorders: evaluation and treatment. J Am Acad Orthop Surg. 2009;17:125–36.
34. Carofino BC, Brogan DM, Kircher MF, Elhassan BT, Spinner RJ, Bishop AT, Shin AY. Iatrogenic nerve injuries during shoulder surgery. J Bone Joint Surg Am. 2013;95(18):1667–74.
35. Meyer M, Graveleau N, Hardy P, Landreau P. Anatomic risks of shoulder arthroscopy portals: anatomic cadaveric study of 12 portals. Arthroscopy. 2007;23(5):529–36.
36. Castagna A, Snyder SJ, Conti M, Borroni M, Massazza G, Garofalo R. Posterior humeral avulsion of the glenohumeral ligament: a clinical review of 9 cases. Arthroscopy. 2007;23(8):809–15.
37. Hurley JA, Anderson TE, Dear W, Andrish JT, Bergfeld JA, Weiker GG. Posterior shoulder instability. Surgical versus conservative results with evaluation of glenoid version. Am J Sports Med. 1992;20:396–400.
38. Martetschläger F, Padalecki JR, Millett PJ. Modified arthroscopic McLaughlin procedure for treatment of posterior instability of the shoulder with an associated reverse Hill-Sachs lesion. Knee Surg Sports Traumatol Arthrosc. 2013;21(7):1642–6.
39. Schwartz E, Warren RF, O'Brien SJ, Fronek J. Posterior shoulder instability. Orthop Clin North Am. 1987;18(3):409–19.
40. Burkhead WZ Jr, Rockwood CA Jr. Treatment of instability of the shoulder with an exercise program. J Bone Joint Surg Am. 1992;74:890–6.
41. Takwale VJ, Calvert P, Rattue H. Involuntary positional instability of the shoulder in adolescents and young adults. Is there any benefit from treatment? J Bone Joint Surg Br. 2000;82(5):719–23.
42. Kiss J, Damrel D, Mackie A, Neumann L, Wallace WA. Non-operative treatment of multidirectional shoulder instability. Int Orthop. 2001;24(6):354–7.
43. McIntyre K, Bélanger A, Dhir J, Somerville L, Watson L, Willis M, Sadi J. Evidence-based conservative rehabilitation for posterior glenohumeral instability: a systematic review. Phys Ther Sport. 2016;22:94–100.
44. Illyes A, Kiss J, Kiss RM. Electromyographic analysis during pull, forward punch, elevation and overhead throw after conservative treatment or capsular shift at patient with multidirectional shoulder joint instability. J Electromyogr Kinesiol. 2009;19:e438–47.
45. Saccomanno MF, Fodale M, Capasso L, Cazzato G, Milano G. Generalized joint laxity and multidirectional instability of the shoulder. Joints. 2014;1(4):171–9.
46. Warby SA, Pizzari T, Ford JJ, Hahne AJ, Watson L. The effect of exercise-based management for multidirectional instability of the glenohumeral joint: a systematic review. J Shoulder Elb Surg. 2014;23:128–42.
47. Kiss RM, Illyes A, Kiss J. Physiotherapy vs. capsular shift and physiotherapy in multidirectional shoulder joint instability. J Electromyogr Kinesiol. 2010;20:489–501.
48. Ide J, Maeda S, Yamaga M, Morisawa K, Takagi K. Shoulder strengthening exercise with an orthosis for multidirectional shoulder instability: quantitative evaluation of rotational shoulder strength before and after the exercise program. J Shoulder Elb Surg. 2003;12:342–5.
49. Barden JM, Balyk R, Raso VJ, Moreau M, Bagnall K. Atypical shoulder muscle activation in multidirectional instability. Clin Neurophysiol. 2005;116:1846–57.
50. Illyes A, Kiss RM. Kinematic and muscle activity characteristics of multidirectional shoulder joint instability during elevation. Knee Surg Sports Traumatol Arthrosc. 2006;14:673–85.
51. Chen D, Goldberg J, Herald J, Critchley I, Barmare A. Effects of surgical management on multidirectional instability of the shoulder: a meta-analysis. Knee Surg Sport Traumatol Arthrosc. 2016;24(2):630–9.
52. Brzoska R, Blasiak A, Hldaki W, Streit JJ, Toussaint B, Solecki W. Arthroscopic Bankart repair using Extracapsular suture between the glenohumeral ligaments and subscapularis tendon (BLS technique): a technical note. Tech Shoulder Surg. 2012;13:86–9.
53. Lafosse L, Boyle S. Arthroscopic Latarjet procedure. J Shoulder Elb Surg. 2010;19:2–12.
54. Boileau P, Hardy MB, McClelland WB Jr, Thélu CE, Schwartz DG. Arthroscopic posterior bone block procedure: a new technique using suture anchor fixation. Arthrosc Tech. 2013;2(4):e473–7.
55. Ortmaier R, Moroder P, Hirzinger C, Resch H. Posterior open wedge osteotomy of the scapula neck for the treatment of advanced shoulder osteoarthritis with posterior head migration in young patients. J Shoulder Elb Surg. 2017;26(7):1278–86. pii: S1058-2746.
56. Hawkins RH. Glenoid osteotomy for recurrent posterior subluxation of the shoulder: assessment by computed axial tomography. J Shoulder Elb Surg. 1996;5:393–400.

5 Failed Arthroscopic Anterior Instability Repair: Case Example

Emmanouil Antonogiannakis, Emmanouil Brilakis, and Anastasios Deligeorgis

5.1 Case Presentation

This case concerns a 24-year-old man who has been reoperated arthroscopically for recurrent anterior instability with a bone block procedure as described by Taverna et al. [1].

This right-handed young man had been operated for the first time 3.5 years earlier due to recurrent anterior instability of his right shoulder. He had suffered five episodes of subluxation/dislocation before the first operation and underwent an arthroscopic Bankart repair. He followed a rehabilitation program and returned to his daily life activities. Twelve months later, after a low-energy trauma, he re-dislocated his right shoulder, and after that he suffered two more episodes of dislocation, the last while sleeping.

The patient was a recreational basketball player. During the clinical examination made before the second operation, he complained for mild pain, mostly during forceful movements, probably because the last episode happened a week before. He had full range of motion (ROM), 180° of forward flexion, 90° of external rotation at 0° of abduction, and at 90° of abduction and internal rotation to T10 vertebra (Fig. 5.1). He had no restrictions in daily activities and little difficulty sleeping on his affected shoulder, but he was not able to perform his usual sport/leisure activities. The clinical examination revealed positive in both the apprehension and the relocation tests, and the anterior drawer test was also positive (+++). There was minimum posterior translation (+) and a positive sulcus sign (++). Mild general ligamentous laxity was considered to be present since knee recurvatum, and elbow hyperextension was observed. His medical record was negative for any other pathology, drug consumption, or allergy, except for G6PD deficiency.

The radiological assessment, with magnetic resonance imaging (MRI) and three-dimensional computer tomography CT (3D CT), revealed a re-tear of the anterior labrum, as well as a humeral bone defect (Hill–Sachs lesion) of 25 mm. A 23% bone defect of the glenoid defect was calculated on the en face reconstructed view of a CT scan, with the humeral head eliminated, according to the circle method described by Sugaya et al. [2] (Fig. 5.2). Glenoid bone loss was calculated without comparison with the contralateral shoulder as measurement by comparison was not reliable due to a 15% bone loss on the left shoulder (he had dislocated his left shoulder too).

We decided to treat the patient with a bone block procedure using the technique described by Taverna et al. [1]. A bone block of 2 cm × 1 cm × 1 cm was obtained from the anterior iliac crest and was prepared with special instruments (Smith & Nephew, London, UK) (Fig. 5.3). A standard arthroscopy was performed

E. Antonogiannakis · E. Brilakis (✉) · A. Deligeorgis
3rd Orthopaedic Department, Hygeia Hospital, Athens, Greece

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_5

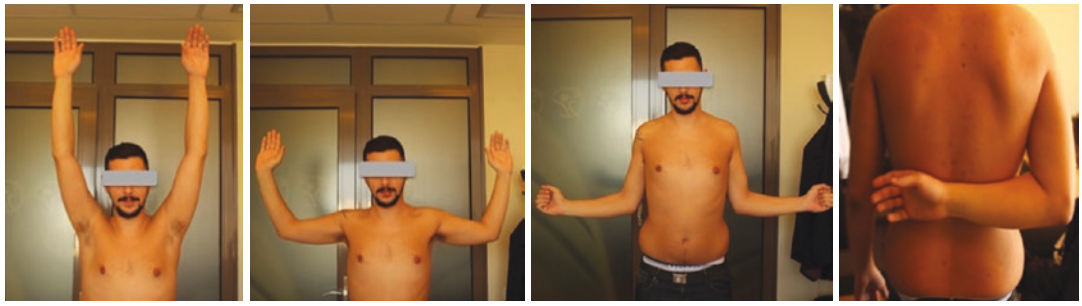

Fig. 5.1 Preoperative ROM of the patient

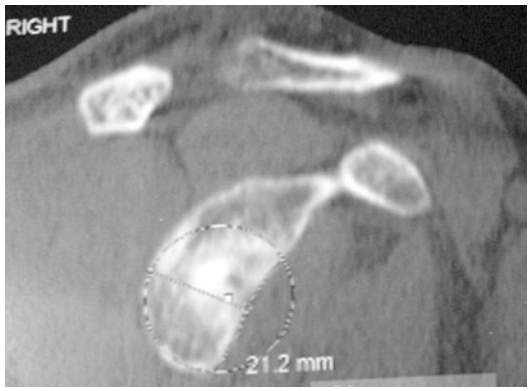

Fig. 5.2 Preoperative 3D CT scan. A 23% glenoid bone loss was calculated on the en face reconstructed view of a CT scan with the humeral head eliminated according to the circle method [2]

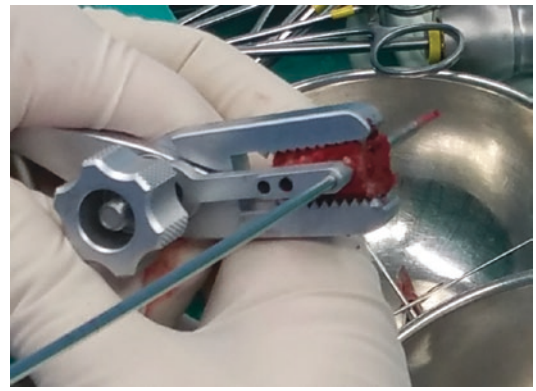

Fig. 5.3 Bone block of 2 cm × 1 cm × 1 cm was obtained from the anterior iliac crest and was prepared with special instruments (Smith & Nephew, London, UK)

in the lateral decubitus position under general anesthesia. The affected shoulder was placed in 70° of abduction and 15° of forward flexion with 3 kg of traction. The glenohumeral joint was inspected from the posterior and the anterior portal, and a diagnostic examination of the joint was performed in order to assess the severity of the glenoid bone defect and the tissue quality of the capsule. After that, the anterior labrum-capsule complex was freed completely from the glenoid neck and was mobilized. With the use of a special guide (Smith & Nephew), two parallel 2.8 mm tunnels were drilled 5 mm deeper from the articular surface of the glenoid. Three double-loaded soft anchors were inserted (Juggerknot 1.5; Zimmer Biomet, Warsaw, IN, USA) at 5, 3, and 1 o'clock, and a 13 mm metal cannula was introduced through the rotator interval. From this cannula the bone block was inserted into the joint and set in position. Two sets of round buttons (pairs of two, four in total) connected with No. 5 high-strength sutures stabilized the bone graft to the anterior glenoid rim. Then, remplissage of the posterior capsule and the infraspinatus was performed. The Hill–Sachs lesion was abraded with a burr, and one double-loaded absorbable anchor (Lupine; DePuy Mitek, Raynham, MA, USA) was inserted in the lesion through a posterolateral accessory portal. With the use of a suitable suture passing instrument (Bird Beak; Arthrex, Naples, FL, USA), the sutures were passed through the capsule and the infraspinatus tendon in a mattress fashion and tied over the lesion bringing the capsule-tendon complex in firm contact with the abraded surface of the humeral head. When we were satisfied with the filling of the humeral head defect, we continued with the repair of the anterior complex. In this way, the tension of the

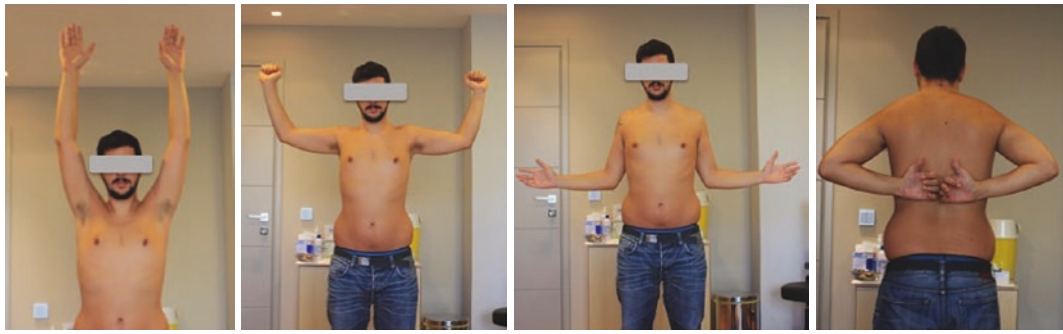

Fig. 5.4 Postoperative ROM of the patient

anterior and posterior capsule was restored, and the bone block became extra-articular.

The operated shoulder was protected in a sling for 6 weeks. The patient was allowed to remove the sling for exercising. Activities of daily living were allowed after the first week as long as the motion of the shoulder was pain-free and was restricted to the front part of the body (90° of forward flexion, internal rotation to the belly, and no more than 10° of external rotation). Active-assisted exercises were started during the fourth postoperative week increasing the ROM gradually. Overhead activities were allowed 3 months postoperatively and sporting activities after 6 months, increasing gradually.

At the latest follow-up (1 year), the active forward flexion was 180°, the active external rotation at 0° of abduction was 90°, the external rotation at 90° of abduction was 90°, and the internal rotation was at T10 level (Fig. 5.4). The scoring systems we used evaluated the shoulder function as fully recovered (Table 5.1). The patient returned to his daily activities with no restrictions, and he has started playing basketball again (recreational athlete). The radiological evaluation with standard anteroposterior (AP) view in neutral position revealed the position of the graft and the buttons (Fig. 5.5). The graft was considered consolidated to the glenoid in accepted position by CT scan (Fig. 5.6) and 3D CT reconstruction (Fig. 5.7). The same method used preoperatively for calculating the glenoid bone defect was used to define that the diameter of the inferior glenoid was restored (Fig. 5.8). No adverse events were noted since the last follow-up.

Table 5.1 Functional evaluation of the patient's shoulder pre- and postoperatively

Scoring system	Preoperative score (max)	Postoperative (max)
Rowe score	50 (100)	100 (100)
Walsh-Duplay score	55 (100)	100 (100)
ASES score	88.33 (100)	100 (100)
Oxford instability score	20 (48)	46 (48)

5.1.1 Patient Perspective

At the last follow-up (1 year postoperatively), the patient was asked to describe his experience:

> I had a failed arthroscopic anterior instability repair 4½ years ago. One year ago I was re-operated because I was unable to perform sport activities without restriction. This time I was told that a bone block procedure would be performed. My experience is that I had no serious pain after the operation in my shoulder. Only the donor site of the graft was painful and I was slightly limping for the first 2 weeks, until the removal of stiches, as I can remember. I started the rehabilitation protocol a month after the operation and I am feeling very satisfied with my decision to have a second surgery. One year later, I have returned to all my daily activities, I am going to the gym and I play basketball that is my favorite sport. I am not feeling any instability from my shoulder, I have no restrictions even in sports and I am confident that in the near future I will be able to perform even better. This is very important for me, as having my shoulder suddenly dislocated was very distressing and interfered a lot with my social life, and my sporting activities. It was always in my mind.

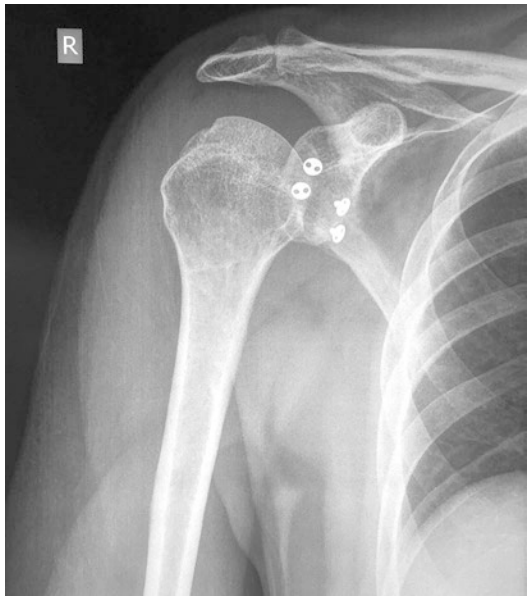

Fig. 5.5 Postoperative radiological evaluation with standard anteroposterior (AP) view in neutral position revealed the position of the graft and the buttons at 1-year follow-up

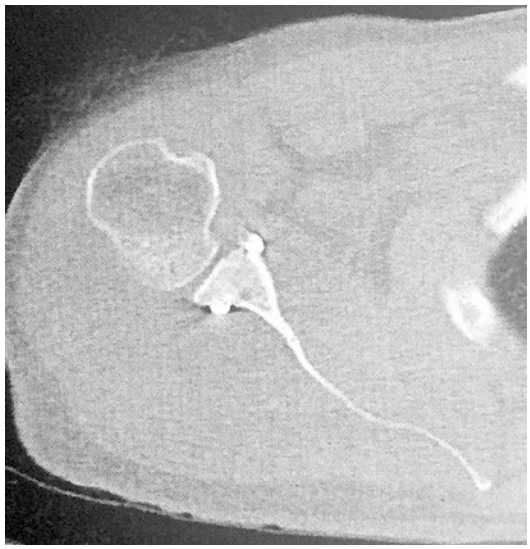

Fig. 5.6 Postoperative CT scan at 1-year follow-up

5.2 Discussion

The operative techniques and the instrumentation that a shoulder surgeon can use for successfully repairing anterior glenohumeral instability arthroscopically have sufficiently advanced nowadays [3]. Since 1923, when Bankart described the detachment of the anterior labrum from the glenoid as the essential lesion for anterior shoulder instability, open or arthroscopic repair of the detached labrum is widely used for the management of patients with traumatic anterior shoulder instability. However, increased failure rates have been reported when bony defects exist either at the glenoid or at the humeral head side and are not addressed during the operation [4]. Recurrent instability in patients with large humeral head bone defects has been attributed at least partially to the engagement of the Hill–Sachs lesion with the anterior rim of the glenoid. The concept of engaging Hill–Sachs lesion was first described by Burkhart and De Beer [3] and was evolved to the concept of on-track/off-track lesions by Di Giacomo et al. [5]. All the above support the concept that the combination of the bony lesions (glenoid and humeral side) is important and not the severity of each lesion separately.

Bone block procedures (coracoid, iliac bone graft, allografts) have been described as the golden standard for the management of anteroinferior glenohumeral instability with significant bone loss [6–10]. The described technique has been used for acute cases with very good results, but we have used the same technique for the management of chronic and revision cases providing that we were able to restore the tension of the anterior and posterior capsule. Otherwise, a procedure for reinforcing the anterior capsule as well is necessary.

The technique used in the present case has two main advantages. First, it is an all-arthroscopic technique in which the guide is used from the posterior portals and the graft is introduced from the standard anterior portal, thus avoiding the anterior neurovascular structures. Second, avoiding the use of screws near the joint lessens the risk of possible complications in case of absorption of the graft and recurrence of the instability or in case of future glenoid erosion due to osteoarthritis. Indeed, the advantages of arthroscopic technique for shoulder stabilization are well-established and include less soft-tissue dissection, thorough inspection of the glenohumeral joint and access to all structures of the joint. The

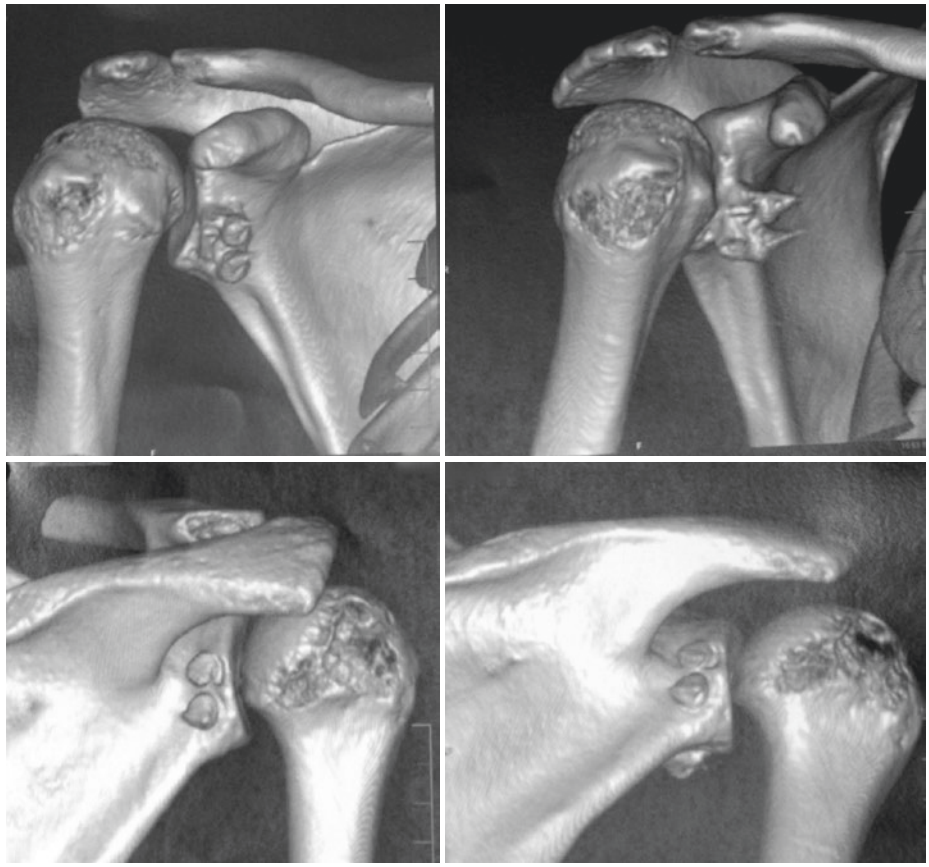

Fig. 5.7 Postoperative 3D CT scan reconstruction at 1-year follow-up showing consolidated graft to the glenoid in acceptable position

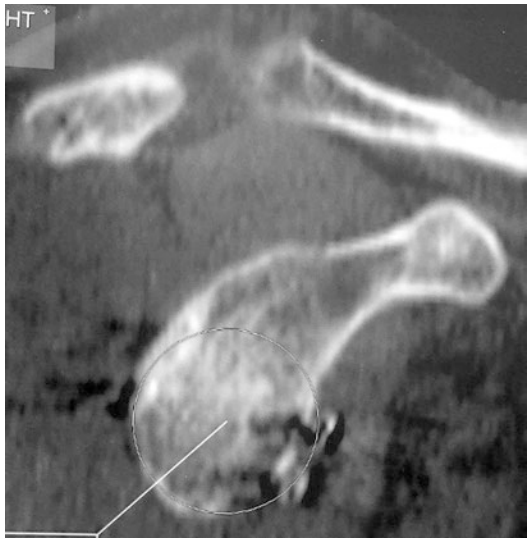

Fig. 5.8 Postoperative 3D CT scan. The same method used preoperatively for calculating the glenoid bone defect was used to define that the diameter of the inferior glenoid was restored

combination of bone block procedure with remplissage increases the tension of the posterior capsule and introduces one more stabilizing factor without influencing the shoulder ROM after the early postoperative period [11].

The most important disadvantage of this procedure is the use of the iliac crest as a source of the bone graft, which is responsible for donor site-related complications [12]. The usual problem for the patient is that the harvest site may be painful. However, pain lasts only during the early postoperative period (2–3 weeks), and it is well tolerated by the properly informed patient.

Conclusion

The arthroscopic use of autologous bone graft in combination with anterior capsule retensioning and posterior remplissage allowed an anatomic repair in the case described, offering a very good outcome until the most recent

follow-up (1 year). The described procedure provided a suitable solution for the management of failed arthroscopic anterior instability repair.

References

1. Taverna E, D'Ambrosi R, Perfetti C, Garavaglia G. Arthroscopic bone graft procedure for anterior inferior glenohumeral instability. Arthrosc Tech. 2014;3(6):e653–60.
2. Sugaya H, Moriishi J, Kanisawa I, Tsuchiya A. Arthroscopic osseous Bankart repair for chronic recurrent traumatic anterior glenohumeral instability. J Bone Joint Surg Am. 2005;87(8):1752–60.
3. Kim SJ, Kim SH, Park BK, Chun YM. Arthroscopic stabilization for recurrent shoulder instability with moderate glenoid bone defect in patients with moderate to low functional demand. Arthroscopy. 2014;30:921–7.
4. Burkhart S, De Beer J. Traumatic glenohumeral bone defects and their relationship to failure of arthroscopic Bankart repairs: significance of the inverted-pear glenoid and the humeral engaging Hill–Sachs lesion. Arthroscopy. 2000;16(7):677–94.
5. Di Giacomo G, Itoi E, Burkhart S. Evolving concept of bipolar bone loss and the Hill–Sachs lesion: from "engaging/non-engaging" lesion to "on-track/off-track" lesion. Arthroscopy. 2014;30(1):90–8.
6. Taverna E, Golanò P, Pascale V, Battistella F. An arthroscopic bone graft procedure for treating anterior-inferior glenohumeral instability. Knee Surg Sports Traumatol Arthrosc. 2008;16:872–5.
7. Warner JJ, Gill TJ, O'Hollerhan JD, Pathare N, Millett PJ. Anatomical glenoid reconstruction for recurrent anterior glenohumeral instability with glenoid deficiency using an autogenous tricortical iliac crest bone graft. Am J Sports Med. 2006;34:205–12.
8. Scheibel M, Kraus N, Diederichs G, Haas NP. Arthroscopic reconstruction of chronic antero-inferior glenoid defect using an autologous tricortical iliac crest bone grafting technique. Arch Orthop Trauma Surg. 2008;128:1295–300.
9. Kraus N, Amphansap T, Gerhardt C, Scheibel M. Arthroscopic anatomic glenoid reconstruction using an autologous iliac crest bone grafting technique. J Shoulder Elbow Surg. 2014;23(11):1700–8.
10. Anderl W, Kriegleder B, Heuberer PR. All-arthroscopic implant-free iliac crest bone grafting: new technique and case report. Arthroscopy. 2012;28:131–7.
11. Brilakis E, Mataragas E, Deligeorgis A, Maniatis V, Antonogiannakis E. Midterm outcomes of arthroscopic remplissage for the management of recurrent anterior shoulder instability. Knee Surg Sports Traumatol Arthrosc. 2016;24(2):593–600.
12. Arrington ED, Smith WJ, Chambers HG, Bucknell AL, Davino NA. Complications of iliac crest bone graft harvesting. Clin Orthop Relat Res. 1996;329:300–9.

Failure Posterior Instability Repair: Case Example

6

Angel Calvo, Nestor Zurita, Alfredo Rodríguez, and Pablo Carnero

6.1 Introduction

Different classifications of shoulder instability have been proposed in order to facilitate treatment decision-making [1].

The classic classifications of shoulder instabilities include two generic types (TUBS and AMBRII), and a new concept of microinstability (AIOS) was more recently introduced [2]. However, some situations cannot be classified in these patterns of instability. This is one of the cases, consisting of a pseudo-neurologic pattern of instability with normal electromyography (EMG) findings.

6.2 Case Presentation

A 16-year-old girl suffered a dislocation to her left shoulder during swimming 2 years before. She was treated by closed reduction under anesthesia in an emergency service. Since then, the patient had daily recurrent episodes of shoulder dislocation. The right shoulder was asymptomatic.

A. Calvo (✉) · A. Rodríguez · P. Carnero
Arthrosport Zaragoza, Zaragoza, Spain
e-mail: angelcalvo@me.com

N. Zurita
IMED Elche Hospital, Elche, Spain

The patient underwent a rehabilitation program for improving muscle control and strength, but dislocation episodes were more and more frequent. Several orthopedic consultations confirmed the diagnosis of multidirectional instability (MDI), and when she came to our attention, she already had two previous arthroscopic capsular plication procedures that failed. Examination under anesthesia had confirmed a predominantly posterior instability.

The patient came to our office with a permanent 45° abduction sling, because the shoulder dislocated in with the arm at the side of the body. Clinical exam was impossible, but Gagey's test was very positive in the contralateral shoulder.

The X-rays showed an abnormal humeral head position displaced inferiorly with the arm at 0° abduction that centered at 45° (Fig. 6.1).

Magnetic resonance arthrography (MRA) showed a huge capsular volume suggesting a capsular lesion, but the humeral head remained centered in both planes during the MR in supine decubitus. No significant bone defects were detected (Fig. 6.2). All EMGs previously performed were normal.

The patient underwent an open Latarjet procedure, but during the postoperative rehabilitation, the patient suffered multiple instability and "dead arm syndrome" episodes. She did not have spontaneous dislocations with the arm at the side of the body, but she suffered subluxations and dislocation sensations during the daily activities.

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_6

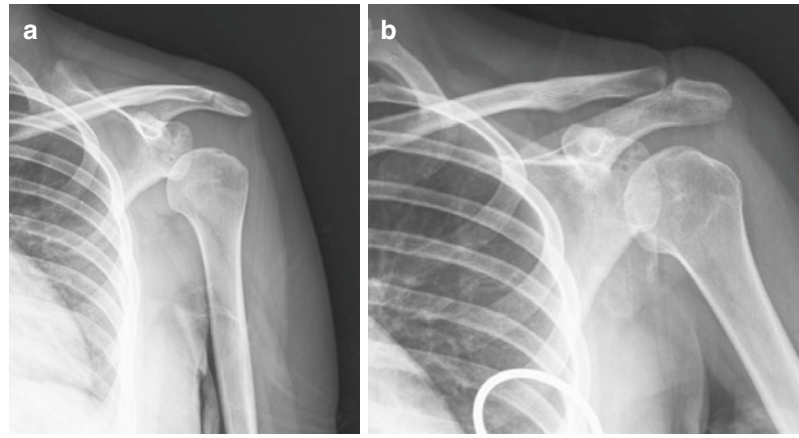

Fig. 6.1 (**a**, **b**) Left shoulder at 0° abduction and with a 45° abduction sling

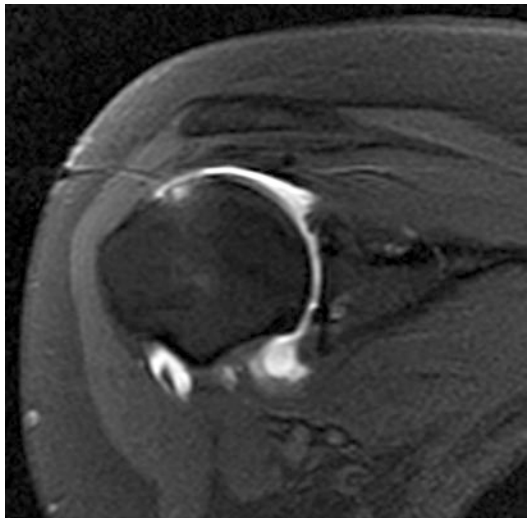

Fig. 6.2 MRA showing a normally centered humeral head

X-rays and computed tomography (CT) showed a correct placement and healing of the coracoid process autograft (Fig. 6.3).

The clinical exam revealed a scapula dyskinesia with an increased pattern of external rotation movement in the left scapula (Fig. 6.4). New EMG studying axillary, suprascapular, accessory spinal, and long thoracic nerves was normal and symmetrical.

We suggested an intense program of muscular reeducation of the scapula during 6 months with an expert physical therapist. After 1-year follow-up, the patient was satisfied and she was able to do normally her daily activities but unable to practice any sport activity involving the affected arm.

6.3 Discussion

This is an unusual case of predominantly posterior-inferior MDI of the shoulder with multiple failed surgical treatments. Initial X-rays suggested a neurological disorder with a typical pattern of axillary nerve palsy and Parsonage-Turner syndrome. However, EMG was normal. In our opinion, it was a false radiographic paralytic pattern due to a malposition of the scapula, which was evident in a preoperative X-ray of the thorax (Fig. 6.5). This condition associated to hyperlaxity can reproduce instability symptoms.

Evaluation of range of motion (ROM) should take into account of motion outside of the glenohumeral (GH) joint. Indeed, GH joint is responsible of the first 120° of abduction and elevation. For the other third of shoulder ROM, we use the movement of the scapula (elevation and external rotation), acromioclavicular joint, and sternoclavicular joint.

In our opinion, it is impossible to classify this pattern of instability into one of the standard patterns TUBS, AMBRII, and AIOS [1, 2]. The Stanmore classification introduced new concepts

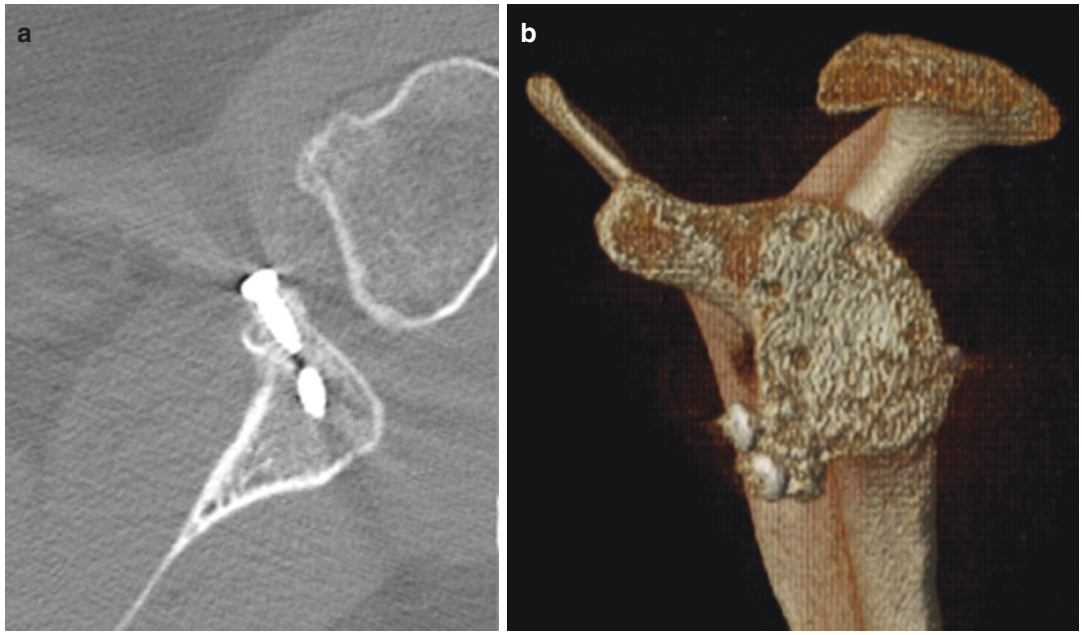

Fig. 6.3 (**a**, **b**) Latarjet procedure. CT scans showing good placement and healing of the coracoid graft

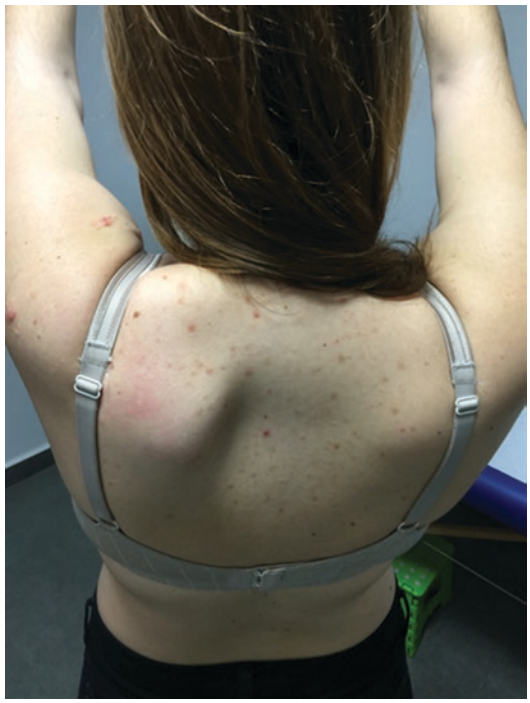

Fig. 6.4 Clinical exam after the failed Latarjet procedure shows scapula dyskinesia

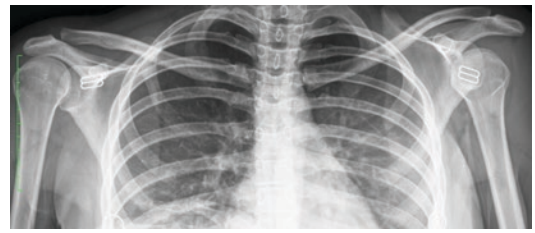

Fig. 6.5 Preoperative X-ray of the thorax shows static malposition of the left scapula

related to atraumatic instability associated to muscular patterning [3]. For the present case, we suggested using the term "instability related to scapular dyskinesia associated with hyperlaxity" (ISDAH).

All the surgical procedures in this type of patients are going to fail, and the treatment should be based on physical therapy for scapular muscle reeducation. An accurate preoperative diagnosis and patient selection are the keys for successful treatment of such unstable shoulders [4].

References

1. Habermeyer P, Magosch P, Lichtenberg S. Shoulder instability. Classification and treatment. Orthopade. 2004;33(7):847–72.
2. Castagna A, Nordenson U, Garofalo R, Karlsson J. Minor shoulder instability. Arthroscopy. 2007;23(2): 211–5.
3. Lewis A, Kitamura T, Bayley JIL. The classification of shoulder instability: new light through old windows! Curr Orthop. 2004;18:97–108.
4. Ho AG, Gowda AL, Michael Wiater J. Evaluation and treatment of failed shoulder instability procedures. J Orthop Traumatol. 2016;17(3):187–97.

Failed Open Anterior Instability Repair: Case Example

7

Ettore Taverna and Vincenzo Guarrella

7.1 Introduction

Open anterior instability repair is usually indicated in cases of instability with bone loss (on the glenoid side or on the humeral side) that must be restored [1]. In this case, the most used surgical procedure is the Latarjet procedure, in which a coracoid bone graft is transferred on the anteroinferior margin of the glenoid. The success of this challenging technique relies on the effect of bone stock replacement, on the sling effect of the conjoint tendon, and on capsular reconstruction obtained by suturing a stump of the coracoacromial ligament attached to the coracoid graft to the remaining capsule [2]. This procedure is more commonly performed in an open fashion, albeit arthroscopically assisted or arthroscopic Latarjet is currently used [3–5]. Outcomes of this procedure are good and reliable, even better than arthroscopic Bankart repair, especially in the context of bone loss [6], albeit clinical and radiological results are related to the surgical technique and hardware placement. Nevertheless, many surgeons are concerned about possible complications of the Latarjet procedure and consider it as the last treatment option [7–10].

In our experience, a failed open Latarjet procedure can be successfully treated with an arthroscopic bone block procedure [11]. After hardware removal and the preparation of the glenoid rim, a bone allograft or autograft from the iliac crest is placed on the anterior margin of the glenoid with the help of an arthroscopic guide, and its fixation is obtained with a double-button system, tensioned with a dedicated device. At the end of the procedure, a standard soft tissue repair is performed (if possible) with arthroscopic suture anchors.

7.2 Case Presentation

A 23-year-old male, otherwise healthy, underwent an open Latarjet procedure for recurrent dislocations (seven episodes) of his right shoulder. The procedure was performed elsewhere. After 6 months of rehabilitation, he experienced a new dislocation from playing volleyball during a forced abduction and external rotation movement. A computed tomography (CT) scan was performed to assess hardware and graft positioning of the previous Latarjet procedure (Fig. 7.1).

The images showed a nonconsolidated bone graft positioned too high on the glenoid rim. Screws appeared too angulated on the glenoid neck, and we considered the failure as a consequence of a malpositioning of the coracoid graft and hardware.

An arthroscopic hardware removal and bone block procedure was planned. The procedure was performed in beach chair position, under general

E. Taverna · V. Guarrella (✉)
IRCCS Istituto Ortopedico Galeazzi, Chirurgia della Spalla, Milano, Italy
e-mail: Ettore.Taverna@eoc.ch

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_7

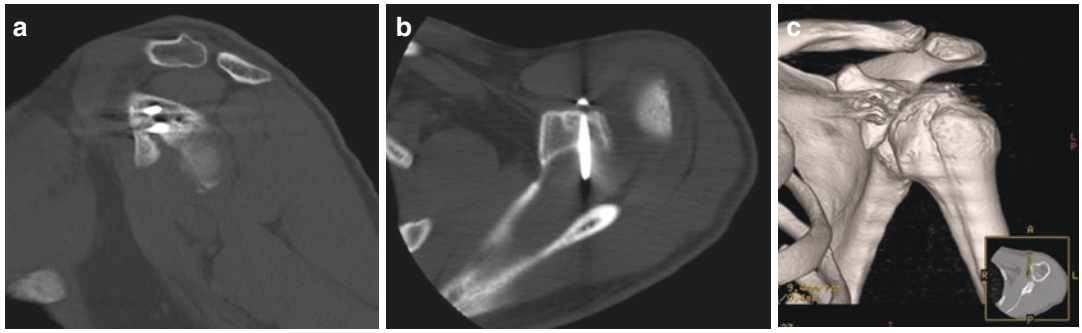

Fig. 7.1 (**a**–**c**) Preoperative CT scan: bone graft and hardware malpositioning

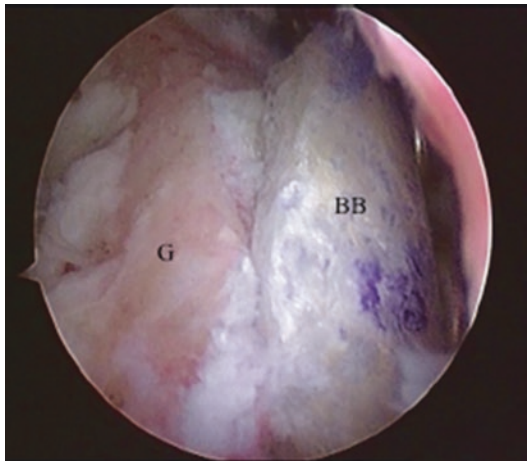

Fig. 7.2 Arthroscopic view from anterior portal. The bone block is perfectly flush with the anterior glenoid rim (*BB* bone block, *G* glenoid)

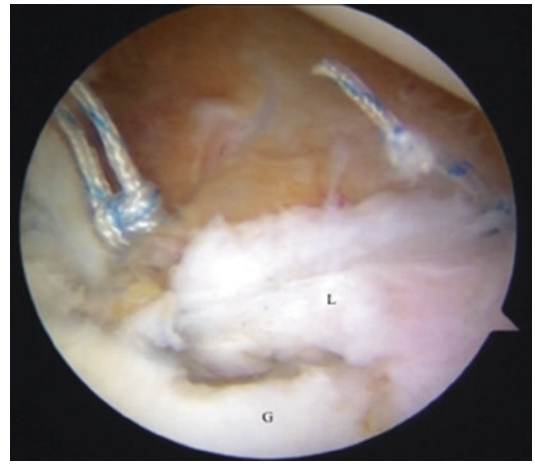

Fig. 7.3 Arthroscopic view from posterior portal. The anterior labrum and capsule are repaired to the glenoid rim with suture anchors, and a standard Bankart repair is performed (*G* glenoid, *L* labrum)

anesthesia and interscalene block. Diagnostic arthroscopy confirmed a high graft placement and an anteroinferior bone defect of the glenoid. The hardware removal was easily achieved through the rotator cuff interval with a screwdriver. A demineralized and decellularized bone allograft from the iliac crest (LifeNet Health, Virginia Beach, VA, USA) was used for the bone grafting procedure. It was positioned with the help of an arthroscopic guide on the anterior-inferior glenoid neck after labral detachment (labrum was not treated during the previous surgery) and glenoid rim preparation. Fixation was achieved with two pairs of arthroscopic round Endobuttons (Smith & Nephew Inc., Andover, MA, USA), and compression was optimized with a tensioner device (Fig. 7.2).

At the end of the procedure, a standard Bankart repair was performed with suture anchors in order to repair the capsule and the labrum and to leave the graft extraarticular (Fig. 7.3).

After surgery, the shoulder was protected in a sling with 15° of abduction for 3 weeks; then passive- and active-assisted range of motion (ROM) exercises were started and prosecuted for 4 weeks. At the 7th week after surgery, active ROM exercises were started, avoiding weights and rubber bands until the 14th week. From the 15th week, strengthening exercises were introduced, and sports activity (volleyball) was resumed between 5 and 6 months after surgery.

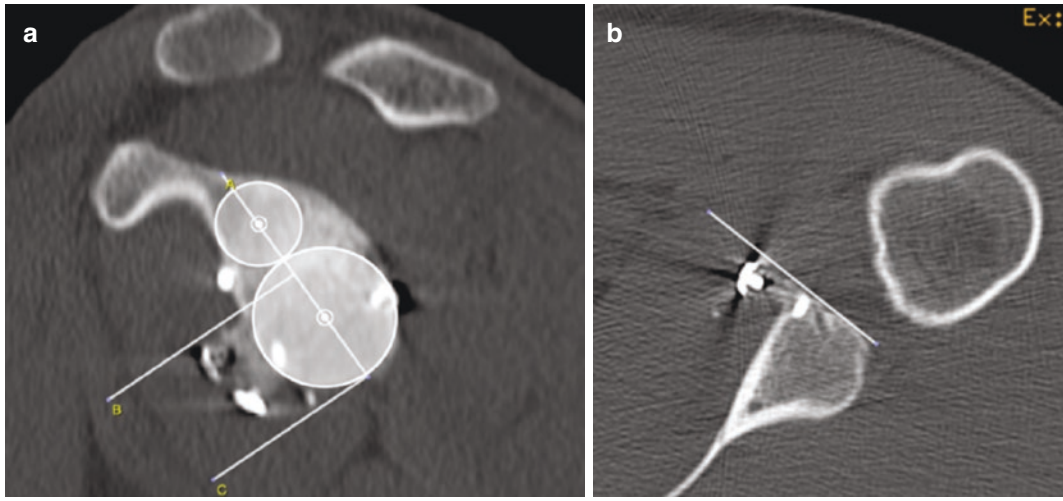

Fig. 7.4 (**a**, **b**) CT scans at 1-year follow-up show bone graft healing and remodeling

CT scan at 1-year follow-up showed bone integration and optimal positioning of the bone graft in the lower half of the glenoid and flush to the glenoid rim (Fig. 7.4). The patient did not experience further episodes of instability and did not complain about pain or loss of external rotation. He went back to all his living and sports activities.

References

1. Provencher MT, Bhatia S, Ghodadra NS, Grumet RC, Bach BR Jr, Dewing CB, LeClere L, Romeo AA. Recurrent shoulder instability: current concepts for evaluation and management of glenoid bone loss. J Bone Joint Surg Am. 2010;92(suppl 2):133–51.
2. Walch G, Boileau P. Latarjet-Bristow procedure for recurrent anterior instability. Tech Shoulder Elbow Surg. 2000;1:256–61.
3. Taverna E, Ufenast H, Broffoni L, Garavaglia G. Arthroscopically assisted Latarjet procedure: a new surgical approach for accurate coracoid graft placement and compression. Int J Shoulder Surg. 2013;7(3):120.
4. Lafosse L, Boyle S. Arthroscopic Latarjet procedure. J Shoulder Elbow Surg. 2010;19:2–12.
5. Boileau P, Gendre P, Baba M, Thélu CÉ, Baring T, Gonzalez JF, Trojani C. A guided surgical approach and novel fixation method for arthroscopic Latarjet. J Shoulder Elbow Surg. 2016;25(1):78–89.
6. Zimmermann SM, Scheyerer MJ, Farshad M, Catanzaro S, Rahm S, Gerber C. Long-term restoration of anterior shoulder stability: a retrospective analysis of arthroscopic Bankart repair versus open Latarjet procedure. J Bone Joint Surg Am. 2016;98(23):1954–61.
7. Zuckerman JD, Matsen FA. Complications about the glenohumeral joint related to the use of screws and staples. J Bone Joint Surg Am. 1984;66:175–80.
8. Young DC, Rockwood CA. Complications of a failed Bristow procedure and their management. J Bone Joint Surg Am. 1991;73:969–81.
9. Rowe C, Zarins B, Ciullo J. Recurrent anterior dislocation of the shoulder after surgical repair. Apparent causes of failure and treatment. J Bone Joint Surg Am. 1984;66(2):159.
10. Athwal GS, Meislin R, Getz C, Weinstein D, Favorito P. Short-term complications of the arthroscopic Latarjet procedure: a north American experience. Arthroscopy. 2016;32(10):1965–70.
11. Taverna E, D'Ambrosi R, Perfetti C, Garavaglia G. Arthroscopic bone graft procedure for anterior inferior glenohumeral inastability. Arthrosc Tech. 2014;3(6):e653–60.

Part II

Failed Biceps and Acromioclavicular Joint Treatment

The Disabled Throwing Shoulder: When and How to Operate

8

Nuno Gomes, Ricardo Aido, and Joana Gomes

8.1 Introduction

The throwing athletes provide a demanding challenge to the orthopedic surgeon. The repetitive overhead motion they are subjected places high forces on the shoulder, predisposing them to injuries. The American baseball pitcher is often remembered as the paradigmatic throwing athlete, not only because he is a perfect example of the athlete that may suffer from pathologic conditions of a throwing shoulder but also because many of the scientific literature on this clinical entity originates from the Americas. Nevertheless, those conditions can be seen in many other athletes who are involved in repetitive overhead throwing such as handball, volleyball, or basketball, as well as in athletes who participate in non-throwing sports that include repetitive overhead motion, such as tennis and swimming. As a result, pathology of the throwing shoulder is common in orthopedic practice all over the world, both in recreational and professional athletes.

N. Gomes (✉)
Hospital Forças Armadas - Polo do Porto, Porto, Portugal

Hospital da Luz Arrábida, Porto, Portugal

R. Aido
Centro Hospitalar Póvoa de Varzim – Vila do Conde, Póvoa de Varzim, Portugal

J. Gomes
Hospital Srª da Oliveira, Guimarães, Portugal

The overhead movement demands a complex balance between the static and dynamic stabilizers of the glenohumeral (GH) joint, which has been referred to as the *throwers paradox* [1, 2] due to the need for both hypermobility and stability, allowing overhead activity without subluxation. To obtain the supraphysiologic range of motion (ROM) that is critical for successful overhead athletes, adaptations of the GH joint are needed, in the form of both osseous and soft tissue changes [3, 4].

The throwing motion can be divided into six phases: windup, early cocking, late cocking, acceleration, deceleration, and follow-through [5, 6]. During the late cocking stage of throwing, the arm reaches maximal external rotation, abduction, and extension, which is a position where the anterior band of the inferior glenohumeral ligament (IGHL), the primary static stabilizer to anterior translation, is under maximal strain [7]. The recurrent stretching of these capsule-ligamentous restraints results in microtears, leading to an increased anterior capsule laxity [3] and posterosuperior labral tearing, the so-called peel-back mechanism, a combination that produces a supplementary gain in external rotation in overhead athletes [2, 8–10]. These alterations become established over time, with lengthening of the anterior capsule along with posterior contracture, resulting in translation of the head and an increased contact between the greater tuberosity and posterosuperior glenoid

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_8

when throwing. It is the internal impingement between the articular side of the posterior cuff and the labrum in abduction and external rotation of the arm. However, these alterations do not necessarily have a clinical expression, and several factors may contribute to turn these asymptomatic shoulders into symptomatic.

The disabled throwing shoulder (DTS) is a clinical entity that has deserved much study in the past years, and an update on current knowledge [11] reflects the progress in the understanding of this problem and highlights what is and what is still not known, thus suggesting future directions.

8.2 Spectrum of Pathology in the Disabled Throwing Shoulder

Different theories try to explain the abnormalities found in a throwing shoulder. The explanation would most likely be a combination of all the theories and findings.

8.2.1 Kinetic Chain Alterations

The kinetic chain is a sequential coordinated chain of force development and a kinematic chain of sequential body positions and motions that develop and regulate the overhead throwing motion [11, 12]. It allows throwing an object or doing that throwing movement with less effort and with a velocity that is directly correlated to the speed of the trunk rotation and the body segments used, with the slowest being arm use alone and the fastest being use of the legs, hip, and trunk together [13].

The clinical implications for injury risk are obvious but recurrently underestimated, or even forgotten, by physicians. Flexing the knees in the cocking phase and rotating the contralateral hip are steps of the throwing motion that everyone would recognize. The body works as a unit in performance, and a limitation in any of those due to a lesion, like a decreased hip ROM, has been associated with shoulder injury and a poor throwing mechanics [14]. However, long-term studies are still lacking, and it is not clear whether the kinetic chain alterations are a cause or an effect of other lesions or whether their early identification and correction have significant impact on improved performance or decreased injury incidence [11].

8.2.2 Scapular Dynamic Dysfunction

The three-dimensional motion of the scapula when throwing is well documented [15], and any deviations from those patterns have been shown to have implications for injury [11, 16]. Any abnormal movement of the scapula is known as scapula dyskinesis and is an important sign of an underlying shoulder disorder and a guide to rehabilitation. The clinical presentation may vary, with some overlap between the different patterns, but scapula malposition is a common finding, consisting of increased abduction, protraction, and inferior translation (Fig. 8.1). There is an altered rest position, with medial border prominence, and coracoid pain due to pectoralis minor retraction and weakness of serratus anterior and lower trapezius. Burkhart et al. [17] referred to an overuse muscular fatigue syndrome in the throwing athlete as the SICK scapula (scapular malposition, inferior medial border prominence, coracoid pain and malposition, and dyskinesis of scapular movement), where the most common presenting complaint is anterior shoulder pain in the region of the coracoid. This clinical condition can easily be confused with pain due to anterior instability and an apparent "dropped" scapula in his dominant symptomatic shoulder.

Scapular dyskinesis is found in association with DTS in a large percentage of cases with symptoms due to internal impingement, anterior capsular laxity, labral injury, or rotator cuff weakness [11, 18, 19], and addressing that dyskinesis has been shown to decrease impingement symptoms, improve rotator cuff strength, and decrease symptoms in labral injury [11, 20]. Scapular dyskinesis is present if manual stabilization of a protracted scapula increases strength in patients with apparent rotator cuff weakness [21].

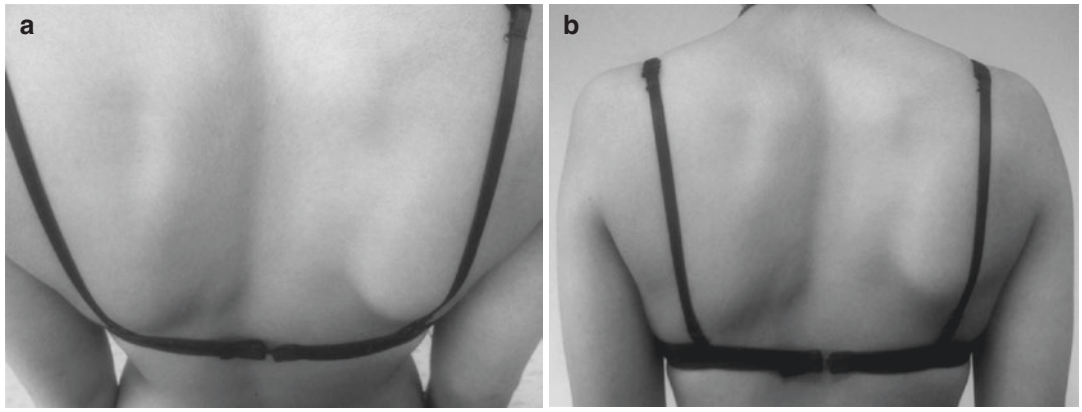

Fig. 8.1 Scapular dyskinesis is characterized by an abnormal movement of the scapula and a malposition at rest. An increased abduction, protraction, and inferior translation, with medial border prominence, is the typical finding (right shoulder)

8.2.3 Glenohumeral Rotation Alterations

Alterations of the GH rotations are classical in these patients and probably the most consistently found changes associated with shoulder injury. Together with a loss of internal rotation (IR), usually referred to as glenohumeral internal rotation deficit (GIRD) [19], external rotation (ER) is usually found to be asymmetrically increased on the dominant side of throwers, evidencing the adaptations these shoulders suffer in order to meet their needs. However, the total rotational ROM (TROM = IR + ER) is usually symmetric in throwers and servers but should not exceed 186° [11]. Wilk et al. [22] reported that the TROM in the throwing shoulders of professional baseball pitchers is within 5° of the non-throwing shoulder and that a bilateral difference outside that range was a contributing factor to shoulder injuries. Likewise, GIRD should be defined as side-to-side asymmetry greater than 18° and may be considered predictive for shoulder injury, together with the deficit of TROM [11]. In a more recent prospective study, the same authors reported that deficit in ER is associated with an increased risk of injury, while deficits in IR and TROM are not [23]. This demonstrates that there is still much to understand and demonstrate about this subject.

Capsule-ligamentous changes in these shoulders have been well documented and result from their exposure to extreme stresses. The recurrent stretching of the anterior capsule in maximal external rotation leads to an increased capsular laxity and an established increased ER. Over time, this process leads to posterior shoulder tightness, with a thickened posterior capsule and superior labrum injuries that have been associated to the IR deficits. Nevertheless, this association does not account for those cases without thickened capsules on the magnetic resonance imaging (MRI) and for the rapid changes in the magnitude of IR deficit after a throwing exposure or stretching programs. Muscle might play a role in this phenomenon by increase in posterior muscles stiffness—thixotropy—as a response to chronic muscular strain [11].

8.2.4 Pathoanatomy of the Disabled Throwing Shoulder

Several types of lesions may be found in a throwing shoulder, besides the dynamic alterations mentioned above. Imaging is invaluable in order to diagnose those lesions, which can be confirmed by MRI, MR arthrography (MRA), or computed tomography (CT) arthrography (CTA), besides ultrasounds (US) that may also play a role in evaluating the cuff and the long head of biceps tendon (LHBT). However, some of these lesions, namely, rotator cuff disease (including partial-thickness cuff tears), may be present in

asymptomatic throwers [24]. Extreme care is therefore needed to distinguish between lesions that are responsible for symptoms and in need of surgery and those that are not.

Capsulolabral injuries are among the most common findings. The excessive laxity that is commonly present in throwers is generally associated to a large anteroinferior pouch due to chronic capsular stretching that may go along with anteroinferior labral lesions (Fig. 8.2). As the anterior capsule fails and the humeral head translates anteriorly during the throwing cycle, the greater tuberosity of the humeral head can contact the posterosuperior glenoid rim, pinching the rotator cuff and the posterosuperior labrum between these two structures (Fig. 8.3).

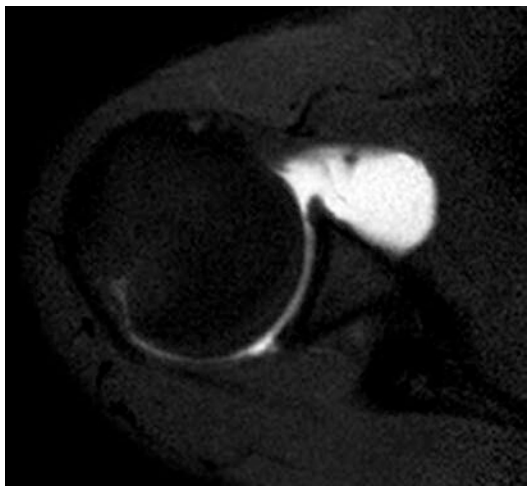

Fig. 8.2 MRA of a right shoulder (axial view) with a distended anteroinferior capsule

Superior labrum from anterior to posterior (SLAP) lesions (Fig. 8.4) can be present in this population because of the peel-back mechanism of the superior labrum with the arm in the cocked position of abduction and external rotation (ABER) (Fig. 8.5). The patient can complain of a "dead arm" or inability to throw at the same level because of pain or discomfort in the shoulder [25].

Rotator cuff pathology in this setting is usually the result of degeneration over years of repeated tendon stress and microtrauma. Most rotator cuff tears in the throwing athlete are partial and articular sided in the supraspinatus (Figs. 8.6 and 8.7), implying internal impingement at maximal cocking as the likely mechanism [26]. However, all the throwing motion can be aggressive to the cuff tendons. Early cocking, acceleration, and deceleration phases result in tensile stresses on the cuff tissue, as well as compression against the coracoacromial arch. These phenomena can be even more blatant when

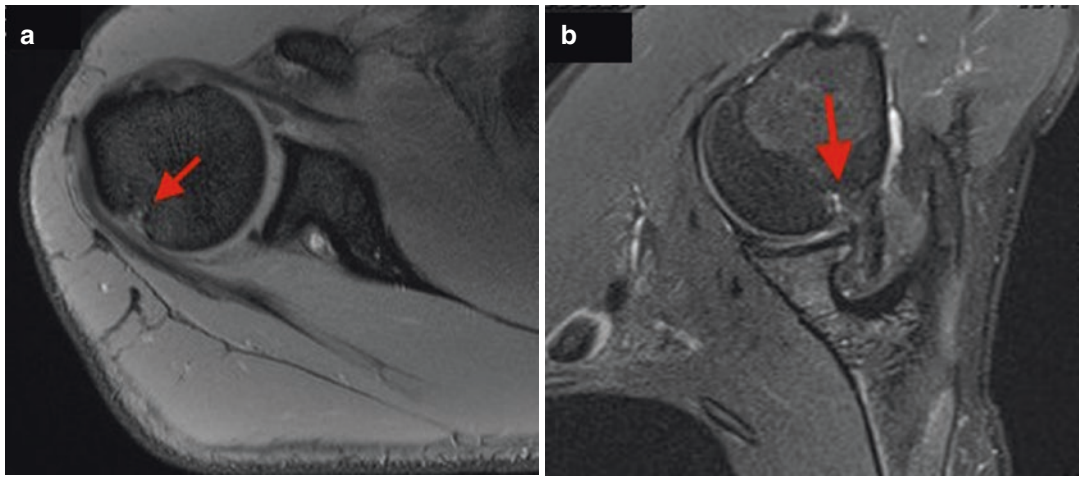

Fig. 8.3 MRI of a right shoulder with internal impingement, pinching of the articular side of the rotator cuff and posterosuperior labrum between the greater tuberosity and glenoid rim. Bony erosions in the same topography on the humeral head are also evident (arrows). (**a**) Axial view. (**b**) Abducted and externally rotated (ABER) view

weakness of the cuff muscles, fatigue, or improper mechanics is present.

Another mechanism of injury to the cuff is related to the motion of the LHBT during abduction and external rotation. The LHBT can override the lesser tuberosity, resulting in inflammation and aggression to the pulley structures that stabilize it in the biceps groove. This will potentially contribute to the progression of a partial-thickness articular-sided tear of the subscapularis tendon that will perpetuate the instability and subluxation of the LHBT with concomitant painful biceps tendonitis (Fig. 8.8).

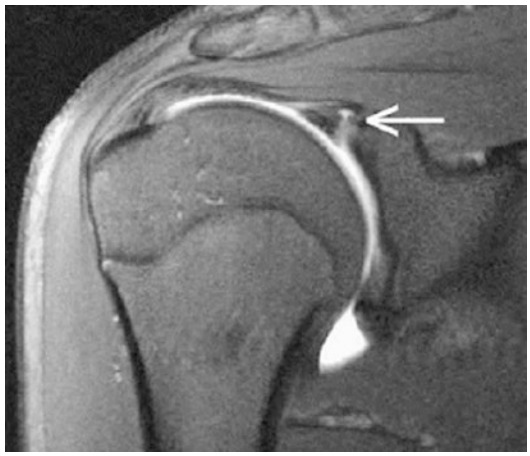

Fig. 8.4 MRA of a right shoulder (coronal view). A SLAP lesion (arrow) is evident

The Bennett lesion is an extra-articular ossification of the posteroinferior capsule at its insertion on the glenoid and typical of throwers (Fig. 8.9). It seems to be related to the traction on the posterior band of the IGHL during the deceleration phase of the throwing cycle [6, 26]. The presence of this bony spur does not imply a clinical manifestation, since in symptomatic shoulders there is generally an associated lesion of the cuff or of the posterior labrum. However, posterior joint laxity, no deficit of internal rotation, and an avulsed fragment on CT scan were determined to be the characteristic clinical features in the shoulders with a painful Bennett lesion [27].

Acromioclavicular (AC) joint osteoarthritis (OA) is neither common nor typical of throwers, but it can also be present in these and be a cause of pain and functional limitation, namely, with repetitive horizontal adduction during follow-through, just like with the crossarm (or cross adduction) test (Fig. 8.10) that is used to search for AC joint pain [28]. In the throwing athlete, isolated AC joint pathology is more likely because of overzealous upper body training, like weight lifting, or a prior trauma than of the

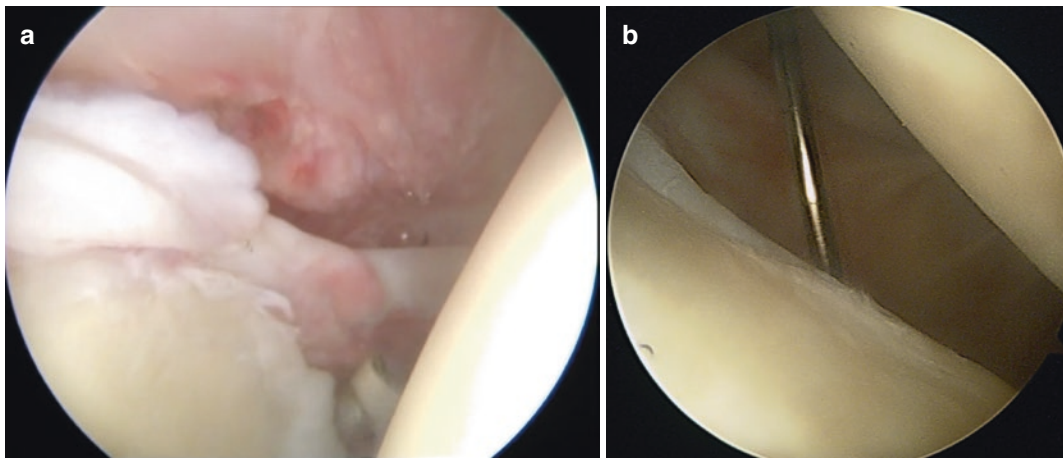

Fig. 8.5 Arthroscopic view of a right shoulder from a standard posterior viewing portal. (**a**) SLAP lesion and irregular posterosuperior labrum with a *mirror* lesion on the undersurface of the posterior supraspinatus from internal impingement. (**b**) Anteroinferior capsular distension and hypoplastic labrum

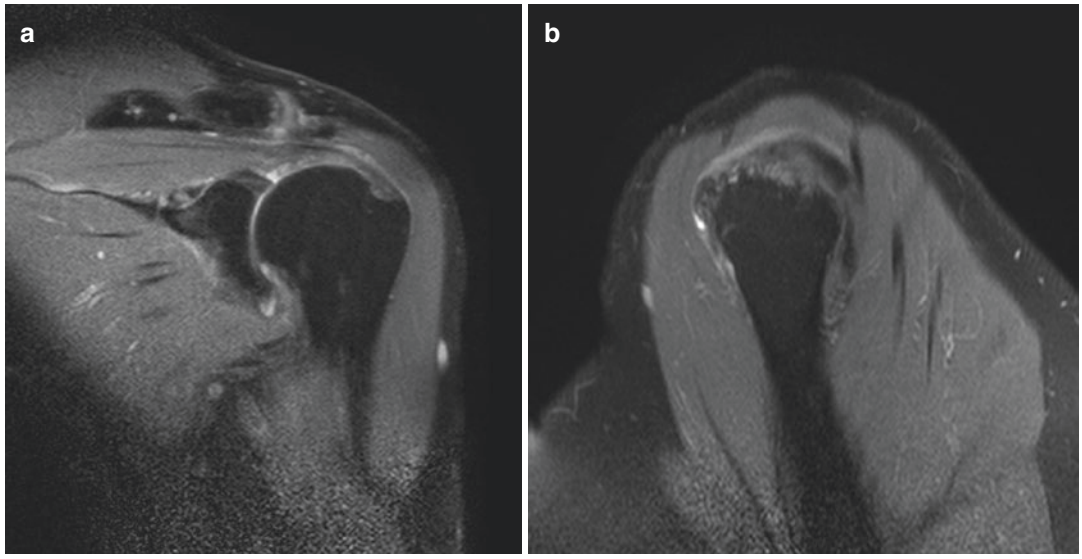

Fig. 8.6 MRI of a left shoulder with an articular-sided fraying of the supraspinatus tendon. Bony erosion on the humeral head at the footprint is present. (**a**) Coronal view. (**b**) Sagittal view

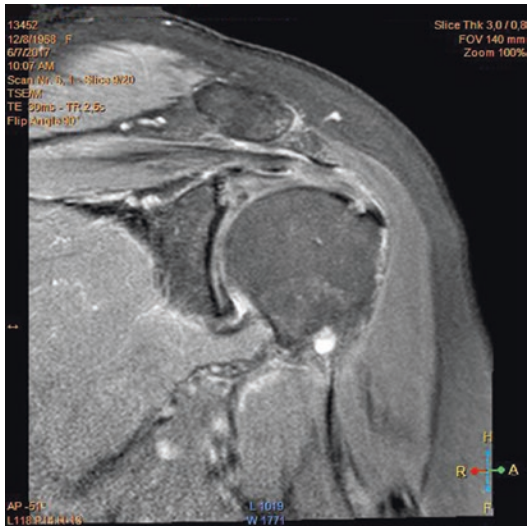

Fig. 8.7 MRI of a right shoulder (coronal view) with a partial-thickness articular-sided tear of the supraspinatus tendon

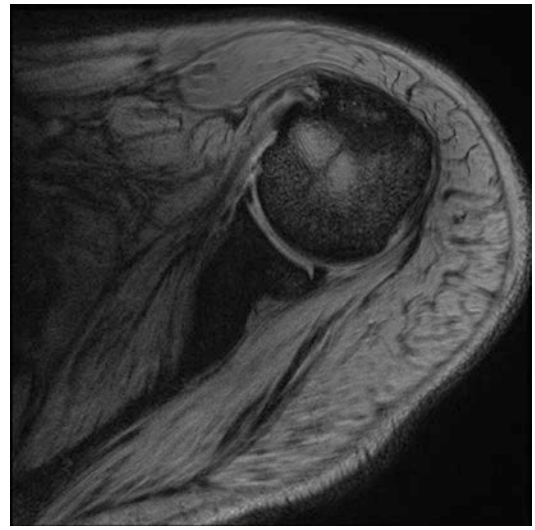

Fig. 8.8 MRI of a left shoulder (axial view) with a partial-thickness articular-sided tear of the subscapularis tendon and an unstable long biceps tendon

repetitive motion itself [26]. The presence of joint effusion, bone edema, and cysts on MRI is important for the diagnosis (Fig. 8.11), especially when the plain X-ray is normal.

8.3 Conservative Treatment

The first approach to a DTS must always be non-operative and address the alterations described

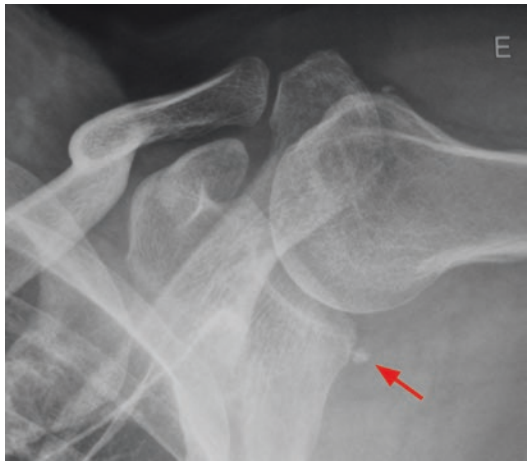

Fig. 8.9 X-ray of a left shoulder (axial view) showing an ossification of the posteroinferior capsule (Bennett lesion) (arrow)

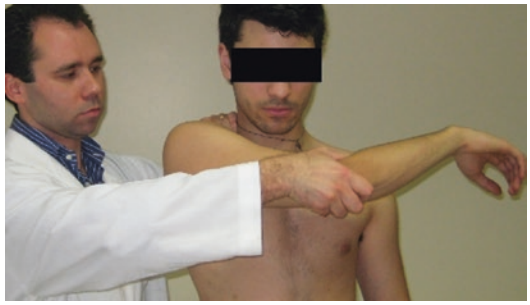

Fig. 8.10 Crossarm or cross adduction test for AC joint pathology

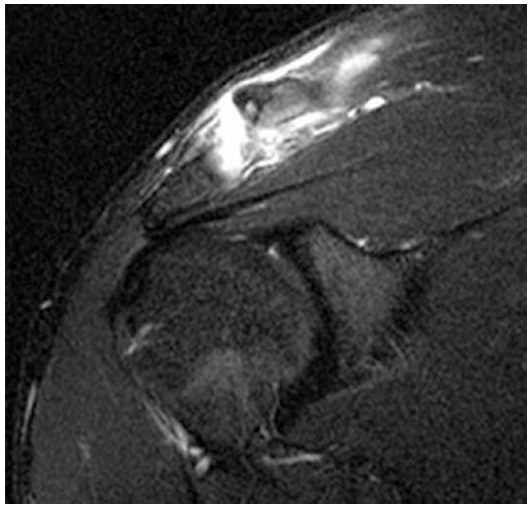

Fig. 8.11 MRI of a right shoulder (coronal view) showing OA of AC joint with effusion, bone edema, and cysts

above. Rest and simple pain relief should be the first-line management. Rehabilitation is then crucial for the correction of any imbalance or dysfunction, but clear guidelines on the best procedure are still lacking. Nevertheless, it is the physician's responsibility to identify all the alterations that come along with a painful shoulder in a thrower, besides what is evident on imaging, and to decide which ones are eligible for and could benefit from physical therapy. Given the specificity of this problem, these patients should be sent to rehabilitation facilities with experience in treating these situations.

In general, exercises focused on rotator cuff and scapular stabilizers strengthening, combined with posterior stretching in patients with GIRD, should be performed. This approach should be dictated by a thorough physical examination looking for alterations in the kinetic chain, scapular dyskinesis, or GH rotation deficits that may be subtle and less obvious to shoulder physical testing and imaging.

Non-operative treatment plays a key role in reducing the need for surgery. It should follow a sequential, progressive, three-phased approach that highlights the entire kinetic chain while restoring GH joint mobility [21, 29].

The first phase (the acute phase) focuses on anti-inflammatory measures to prevent pain and on mobilization to restore the GH motion. In order to address GIRD, passive stretching of the posterior soft tissues should be done to maintain the total arc of motion, utilizing the cross-body stretch and modified sleeper stretch. The second phase (the recovery phase) will work on the kinetic chain through resistance training involving the lower limb and trunk, with core and scapular stabilization. The third phase (the functional phase) is characterized by endurance-based etxercises and repetition of sport-specific, functional movement patterns necessary for regaining arm strength and for a gradual return to throwing. Plyometric exercise, shoulder end-range stabilization drills, and isotonic strengthening are key components of this phase, facilitating inhibited or weak scapular muscles in order to restore scapular motor control and proprioception.

8.4 Surgical Treatment

"When and how to operate" is still a simple question yet with a difficult answer. Outcomes after surgery in throwing athletes are often hard to interpret because of the lack of standardized diagnosis, the duration and type of non-operative treatment, the techniques used at surgery, and the postoperative rehabilitation protocol. Sciascia et al. [30], in a recent systematic review, concluded that the rate of return to participation after shoulder surgery within the literature is inconsistent. In another review by Harris et al. [31], the rate of return to sport 1 year after surgery in 287 elite throwers was around 68%, but below preinjury levels, despite improvement in terms of pain and disability.

The surgeon must bear in mind that a throwing shoulder has normal adaptive changes, and therefore the aim of an operative intervention may not necessarily be the anatomical reconstruction of all the lesions, but the recreation of the anatomy inherent to that throwing shoulder. For this reason, operative intervention should be considered with a "less-is-more" approach, and, in cases of intraoperative decision, a minimalist approach often provides superior outcomes over aggressive surgical intervention [21]. Furthermore, standardized protocols for the postoperative rehabilitation are also lacking [32], which adds another variable of difficult control to the treatment equation.

8.4.1 Labral Injuries and Instability

Laxity can be physiologic or pathologic in the throwing athlete. Frank instability is less common in the throwing athlete, but acquired laxity may be present as a result of the repetitive throwing motion and actually be necessary for performance at high levels. However, excessive laxity can lead to pathologic conditions in the shoulder [3], and the term instability is often used to describe that acquired laxity. Instability in this setting often presents as a vague complaint of shoulder pain, and comparison with the contralateral shoulder is important in assessing GH laxity. The Jobe relocation test (Fig. 8.12) is helpful in this assessment, noting the presence of apprehension in case of instability, but the presence of posterior pain with this test, in abduction and maximal external rotation, may be due to internal impingement even in the absence of an established instability.

When a trial of non-operative management fails, a diagnostic arthroscopy may reveal anteroinferior capsulolabral damage, posterosuperior labral damage due to internal impingement, humeral head subluxation, or undersurface tears of the supraspinatus or infraspinatus tendons (Fig. 8.13). With gross laxity of the capsule, confirmed by a positive drive-through sign, a stabilization procedure may be indicated to reinforce the anterior capsule. An arthroscopic anterior capsulorrhaphy with the use of sutures or suture anchors is the procedure of choice, with labral repair when necessary (Fig. 8.14). The objective is to reduce any redundancy of the anterior capsule, but care must be taken not to overtighten the repair, with risks of postoperative stiffness.

8.4.2 SLAP and Biceps Lesions

SLAP lesions are typical findings in throwers and have deserved a specific overview in a few studies. Boileau et al. [33] were the first to compare

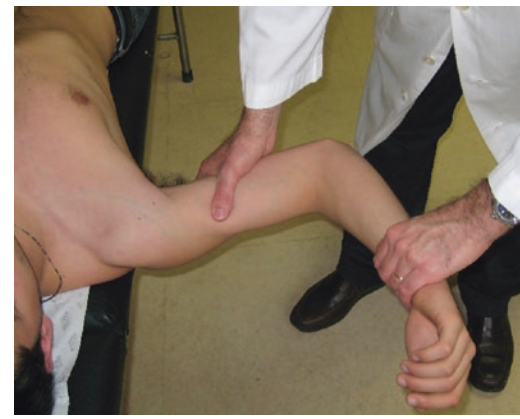

Fig. 8.12 Jobe relocation test (or Fowler test): patient is placed supine with the arm in 90° of abduction and maximal external rotation. The physician then applies a posteriorly directed force on the humeral head, which alleviates the sense of apprehension. Pain that is elicited with this test may be a sign of internal impingement

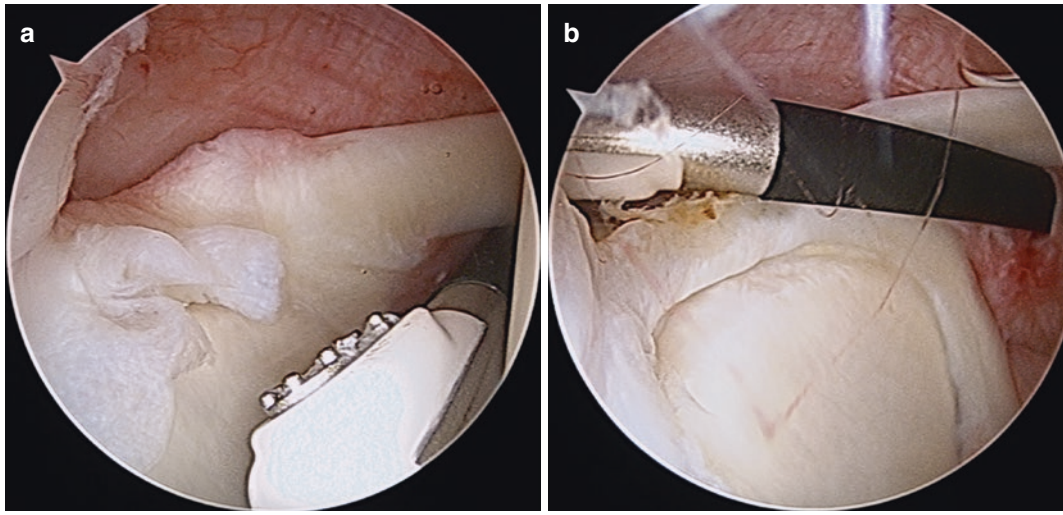

Fig. 8.13 Arthroscopic view of a right shoulder with signs of internal impingement from a standard posterior viewing portal. (**a**) Fraying of the posterosuperior labrum and undersurface of the cuff. (**b**) After debridement with radiofrequency

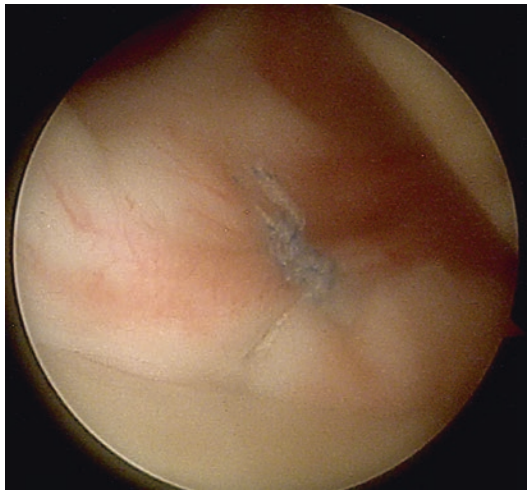

Fig. 8.14 Arthroscopic view of a right shoulder from a standard posterior viewing portal: anteroinferior capsulolabral suture plication

labral repairs with biceps tenodesis in throwers with SLAP II lesions. The background for that study was precisely the existence of recurrent reports on overhead athletes having an inconsistent return to their previous level of sport and satisfaction after arthroscopic repair of SLAP lesion. They found that 60% (6/10) of the patients were disappointed after repair because of persistent pain or inability to return to their previous level of sports participation. In the tenodesis group, the Constant score improved from 59 to 89 points, and 93% (14/15) were satisfied or very satisfied.

Another recent report [34] confirmed the difficulty of returning to sport after SLAP repair in 68 players who had a failure of a primary attempt of guided physical therapy. The conservative approach was similar in all cases and included correction of scapular dyskinesis, posterior capsular stretching to address GIRD, and a gradual return to throwing through stepwise increases as demonstrated by pain-free throwing. Of those who underwent surgery, half went back to play but only 7% did it at the preinjury level of performance. This study stresses the importance of a specifically oriented rehabilitation protocol before indicating surgery to an overhead athlete as well as the poor prognosis after surgery in terms of returning to the same level of performance in throwing.

Moore-Reed et al. [35] carried out a level II prospective study in 58 patients to identify variables predictive of failure of rehabilitative treatment for SLAP lesions. They concluded that a structured rehabilitation program resulted in modification of symptoms and improved function at 6-week follow-up in over half of patients. On initial evaluation, the presence of a painful

arc of overhead motion, indicating loss of normal GH kinematics, and the presence of forward shoulder posture, indicating an altered scapular position, represent negative predictive factors for success of rehabilitation. The authors argue these findings are important when deciding the treatment strategy of a SLAP lesion, as the presence of those factors is associated with failure to achieve a satisfactory improvement and surgery may be recommended.

After failure of non-operative treatment, repair has long been the primary treatment option for symptomatic SLAP lesions of the shoulder. An analysis of data from the American Board of Orthopaedic Surgery Certification Examination Database in 2014 [36] helped to objectivize growing evidence to support both biceps tenotomy and tenodesis as effective alternative treatments for SLAP lesions, as stated above. Practice trends for orthopedic board candidates indicate that the proportion of SLAP repairs has decreased over time, with an increase in biceps tenodesis and tenotomy, an evidence that was even more clear when applicants had shoulder- and elbow-specific fellowship training. These applicants performed less SLAP repairs than those with general orthopedic training, probably due to the fact they are more aware of the clinical results and current trends in the treatment of SLAP tears.

That being so, indication for a SLAP repair must be judicious and probably limited to cases where an isolated trauma, usually with sudden biceps contraction, is identified and to younger athletes [37]. A repair of a chronic SLAP lesion which has developed over time in a thrower may lead to overtightening of the LHBT insertion that limits its physiological peel-back motion and the ability to throw. Therefore, when repair of a SLAP lesion is the choice, there must be concern on how to fix it. Laboratorial studies have shown that there is a tendency to failure of the repair with overtight fixations, including those with anchors anterior to or too close to the root of the LHBT [38, 39], suggesting that one or two posterior anchors, according to the size of the lesion, would be the most appropriate. Furthermore, we and other authors advocate the use of knotless anchors in an effort to provide a low-profile implant that minimizes abrading of the undersurface of the cuff from the knot stack.

Considering these facts, better outcomes may be expected with either biceps tenotomy or tenodesis in many SLAP lesions. In a recent report, Friedman et al. [40] showed that despite increased demands and activity placed on biceps function in a younger population, there are no differences in functional and subjective outcome measurements. The choice between biceps tenotomy and tenodesis for pathology of the proximal biceps tendon can continue to be based on surgeon and patient preference.

Although tenodesis would be a preferred option in a young thrower, there is no consensus regarding the type of fixation. It can be performed by means of sutures to the soft tissues (Fig. 8.15), high or low in the groove, either with anchors, interference screws (Fig. 8.16) or suspensory systems, or subpectoral with a mini-open approach.

The rationale behind the subpectoral approach is the avoidance of the risk of postoperative pain after more proximal fixations. It is argued that the presence of hidden lesions of the LHBT on its course in the groove, as well as inflammatory reaction to the fixation site under the coracoacromial arch, is responsible for residual pain after proximal tenodesis, suggesting a distal fixation would avoid it [41]. That has been the basis for the wide acceptance of subpectoral fixations in throwers, using interference screws for its high strength of fixation. However, reports on humeral fractures due to bony weakening on the fixation site have brought a concern around usage of screws [42, 43]. A recent report on a suspensory button bicortical fixation showed no major complications and a high rate of success (Fig. 8.17) [44].

Pathology of the LHBT, such as tenosynovitis or instability due to pulley or partial tears of the subscapularis tendon, may not be so obvious to diagnose. This is especially true when MRI shows a SLAP lesion and other lesions pass undiagnosed, either because the attention is focused on the most blatant finding or because the altered imaging signs of the intra-articular part of the LHBT are often hard to identify. Clinical examination is often the best clue for the correct

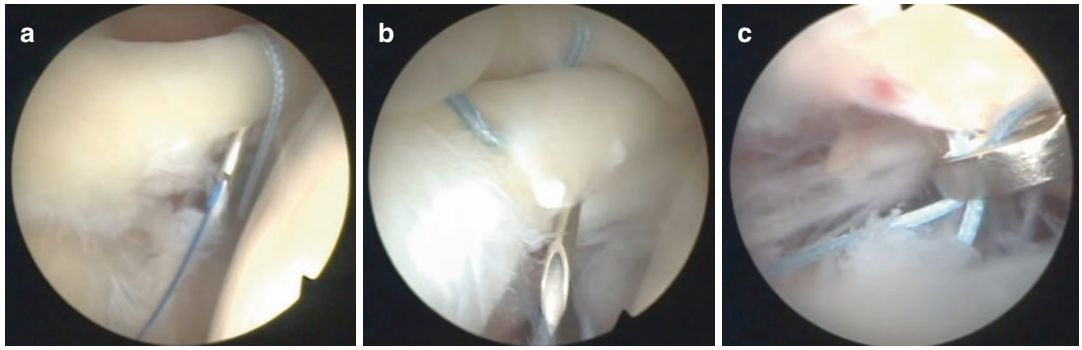

Fig. 8.15 Percutaneous intra-articular transtendon tenodesis (PITT technique) of the LHBT in a right shoulder. (**a**) #2 ultra-high-molecular-weight polyethylene (UHMWPE) suture around the long biceps tendon. A PDS suture is passed through the tendon using a percutaneous needle in order to shuttle one end of the #2 UHMWPE suture. (**b**) Preparation for shuttling the other end of the #2 suture through a perforation a few mm from the first. (**c**) Subacromial view of the two suture limbs that embrace the long biceps tendon and cuff/pulley tissue before being tied

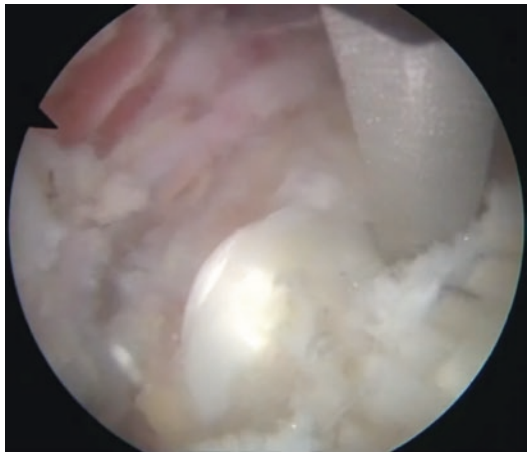

Fig. 8.16 Arthroscopic bursal view of a left shoulder. Proximal tenodesis of the LHBT is performed with an interference screw. The tendon proximal stump and the guide for the screw are inside the humeral hole on the bicipital groove

diagnosis, due to the low specificity of the available tests. In our practice, anterior pain at palpation on the groove and at maximal forward elevation, along with positive palm-up test (or Speed's test) and Yergason test, gives a good evidence of the origin of the pain in the LHBT. Checking the stability and integrity of the LHBT during diagnostic arthroscopy is mandatory in these cases (Fig. 8.18). The role of the US imaging in the office in these cases is invaluable, since it allows a dynamic assessment of a large portion of the tendon. In case of positive findings and failure of conservative measures, including US-guided injections, either tenotomy or tenodesis must be considered.

8.4.3 Rotator Cuff Tears

When physical therapy focused on tissue-specific stretching and strengthening of the rotator cuff muscles fails, arthroscopy is used to definitively diagnose and treat rotator cuff tears. The three options for surgical treatment of rotator cuff tears in throwers include debridement, repair of tendon delamination with sutures, and tendon-to-bone repair. Several authors reported low rates of return to preinjury levels in throwers after repair to bone of partial-thickness tears, confirming evidences that repairing the tendon to bone may tether the compensatory anatomy of the rotator cuff and lead to less-than-ideal outcomes [21, 45–48]. There is no consensus on the best option, but, for that reason, some recent studies state that a minimalist approach is the best choice, with either debridement or intratendinous repair of delaminated tears as opposed to tendon-to-bone repair with suture anchors [49, 50]. However, there is limited evidence to support this option, and the surgical repair should prevent progression of the tear, without limiting the substantial range of motion that is required for competitive play.

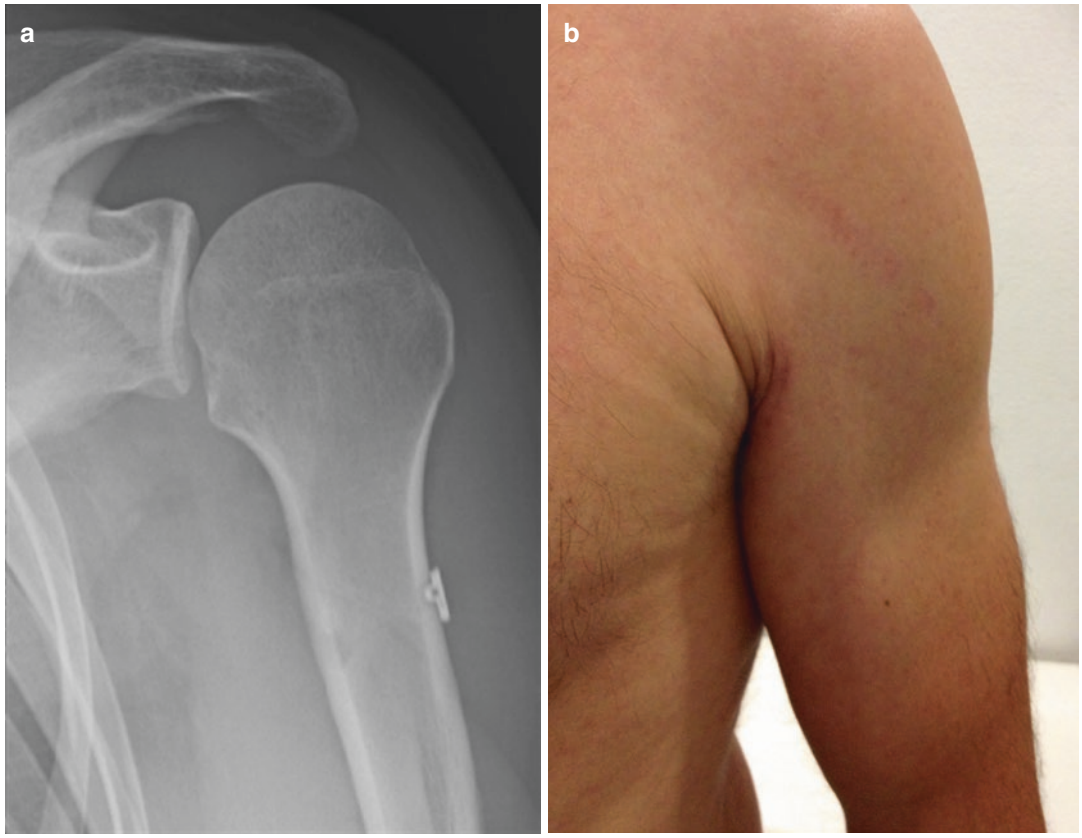

Fig. 8.17 Subpectoral tenodesis and bicortical fixation with a suspensory button. (**a**) Postoperative X-ray. (**b**) Postoperative appearance: good cosmesis and no Popeye sign

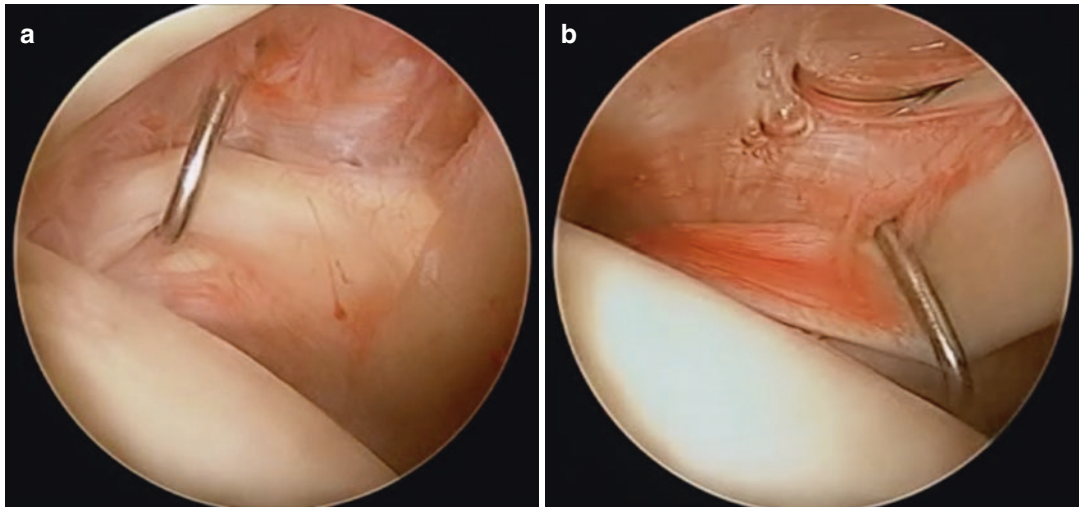

Fig. 8.18 Arthroscopic view of a left shoulder from a standard posterior viewing portal. (**a**) Fraying of the undersurface of the subscapularis tendon is evident at inspection with a probe. (**b**) Tenosynovitis of the LHBT was identified only after traction with a probe

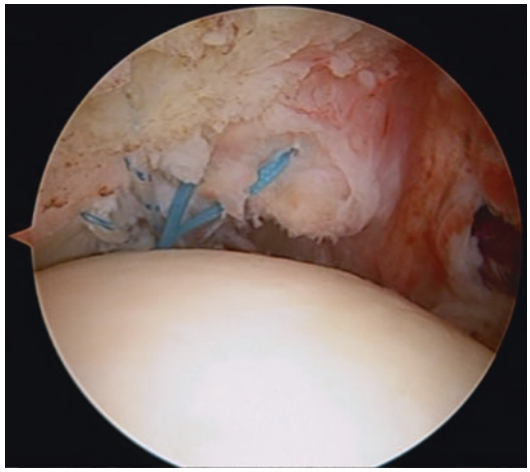

Fig. 8.19 Arthroscopic view of a left shoulder from a standard posterior viewing portal. A partial-thickness articular-sided tear of the supraspinatus tendon is evident. Four suture limbs from an anchor have been passed through the supraspinatus tendon. Subsequent knot tying in the subacromial space will complete the transtendinous repair

Castagna et al. [51] reported on the repairs of deep partial-thickness tears of the supraspinatus and found no clinical or subjective difference between transtendon repair and repair after completion of the partial rupture. However, the mean age of the study group was 51 years old and not focused on throwers, preventing a straightforward application to the case of overhead athletes. Still, in case of a deep partial-thickness articular-sided tear with lack of strength of the supraspinatus, a repair to bone may be considered (Fig. 8.19).

8.4.4 Acromioclavicular Joint Pathology

OA of the AC joint may be present due to overuse, but is not typical of the throwing athlete. A conservative approach is mandatory, and with anti-inflammatory measures and adaptation of the bodybuilding exercises, the athlete may perform in a regular basis. Surgical excision of the lateral end of the clavicle should only be considered after failure of these measures, including joint injections.

Conclusion

Absolute consensus with respect to the treatment of a painful shoulder in throwing athletes does not exist because of the intricacy of the pathoanatomy of the thrower's shoulder, the extreme demand for the joint, and lack of standardized surgical techniques and treatment principles. The athlete must be aware that return to prior abilities is less consistent than in general population, in order to ease management of expectations and the recovery process.

A conservative approach is the mainstay of the treatment of a DTS, and familiarity with the rehabilitation principles of this clinical entity by the physicians, namely, the orthopedic surgeon, is mandatory to be able to identify where the treatment should focus and avoid unnecessary or unsuccessful surgical interventions. Decision to operate on a thrower's shoulder must only take place after exhausting the conservative alternatives.

References

1. Wilk KE, Arrigo C. Current concepts in the rehabilitation of the athletic shoulder. J Orthop Sports Phys Ther. 1993;18(1):365–78.
2. Lauchlan Chambers L, Altchek DW. Microinstability and internal impingement in overhead athletes. Clin Sports Med. 2013;32:697–707.
3. Braun S, Kokmeyer D, Millett PJ. Shoulder injuries in the throwing athlete. J Bone Joint Surg Am. 2009;91(4):966–78.
4. Kurokawa D, Yamamoto N, Ishikawa H, Nagamoto H, Takahashi H, Muraki T, Tanaka M, Sato K, Itoi E. Differences in humeral retroversion in dominant and nondominant sides of young baseball players. J Shoulder Elb Surg. 2017;26(6):1083–7. https://doi.org/10.1016/j.jse.2016.11.051. [Epub ahead of print].
5. Park SS, Loebenberg ML, Rokito AS, et al. The shoulder in baseball pitching: biomechanics and related injuries—part 1. Bull Hosp Jt Dis. 2002;61(1–2):68–79.
6. Park SS, Loebenberg ML, Rokito AS, et al. The shoulder in baseball pitching: biomechanics and related injuries—part 2. Bull Hosp Jt Dis. 2002;61(1–2):80–8.
7. Malicky DM, Kuhn JE, Frisancho JC, et al. Nonrecoverable strain fields of the anteroinferior glenohumeral capsule under subluxation. J Shoulder Elb Surg. 2002;11:529–40.

8. Jobe CM. Posterior superior glenoid impingement: expanded spectrum. Arthroscopy. 1995;11(5):530–6.
9. Wilk KE, Arrigo CA, Andrews JR. Current concepts: the stabilizing structures of the glenohumeral joint. J Orthop Sports Phys Ther. 1997;25(6):364–79.
10. Burkhart SS, Morgan CD. The peel-back mechanism: its role in producing and extending posterior type II SLAP lesions and its effect on SLAP repair rehabilitation. Arthroscopy. 1998;14:637–40.
11. Kibler WB, Kuhn JE, Wilk K, Sciascia A, Moore S, Laudner K, Ellenbecker T, Thigpen C, Tim Uhl T. The disabled throwing shoulder: spectrum of pathology – 10 year update. Arthroscopy. 2013;29(1):141–61.
12. Davids K, Glazier P, Araújo D, Bartlett R. Movement systems as dynamical systems: the functional role of variability and its implications for sports medicine. Sports Med. 2003;33:245–60.
13. Hirashima M, Kadota H, Sakurai S, Kudo K, Ohtsuki T. Sequential muscle activity and its functional role in the upper extremity and trunk during overarm throwing. J Sports Sci. 2002;20:301–10.
14. Robb AJ, Fleisig G, Wilk K, Macrina L, Bolt B, Pajaczkowski J. Passive ranges of motion of the hips and their relationship with pitching biomechanics and ball velocity in professional baseball pitchers. Am J Sports Med. 2010;38:2487–93.
15. Myers JB, Laudner KG, Pasquale MR, Bradley JP, Lephart SM. Scapular position and orientation in throwing athletes. Am J Sports Med. 2005;33:263–71.
16. Ludewig PM, Reynolds JF. The association of scapular kinematics and glenohumeral joint pathologies. J Orthop Sport Phys Ther. 2009;39:90–104.
17. Burkhart SS, Morgan CD, Ben Kibler WB. The disabled throwing shoulder: spectrum of pathology part III: the SICK scapula, scapular dyskinesis, the kinetic chain, and rehabilitation. Arthroscopy. 2003;19(6):641–61.
18. Laudner KG, Myers JB, Pasquale MR, et al. Scapular dysfunction in throwers with pathologic internal impingement. J Orthop Sports Phys Ther. 2006;36(7):485–94.
19. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: Spectrum of pathology. Part I: pathoanatomy and biomechanics. Arthroscopy. 2003;19(4):404–20.
20. Kibler WB. Rehabilitation of rotator cuff tendinopathy. Clin Sports Med. 2003;22:837–48. Arthroscopy (2003);19(6):641-661.
21. Brabston EW, Galdi B, Ciccone J, Yenchak A, Ahmad CS. The thrower's shoulder: an update. JBJS Rev. 2015;3(4):e4.
22. Wilk KE, Macrina LC, Fleisig GS, et al. Correlation of glenohumeral internal rotation deficit and total rotational motion to shoulder injuries in professional baseball pitchers. Am J Sports Med. 2011;39(2):329–35.
23. Wilk KE, Macrina LC, Fleisig GS, Aune KT, Porterfield RA, Harker P, Evans TJ, Andrews JR. Deficits in glenohumeral passive range of motion increase risk of shoulder injury in professional baseball pitchers: a prospective study. Am J Sports Med. 2015;43(10):2379–85.
24. Connor PM, Banks DM, Tyson AB, Coumas JS, D'Alessandro DF. Magnetic resonance imaging of the asymptomatic shoulder of overhead athletes: a 5- year follow-up study. Am J Sports Med. 2003;31:724–7.
25. Burkhart SS, Morgan C. SLAP lesions in the overhead athlete. Orthop Clin North Am. 2001;32(3):431–41, viii.
26. Cohn RM, Jazrawi LM. The throwing shoulder: the orthopedist perspective. Magn Reson Imaging Clin N Am. 2012;20:261–75.
27. Nakagawa S, Yoneda M, Hayashida K, Mizuno N, Yamada S. Posterior shoulder pain in throwing athletes with a Bennett lesion: factors that influence throwing pain. J Shoulder Elb Surg. 2006;15(1):72–7.
28. Gomes N. Exame Clínico. In: Ombro O, editor. Lidel; 2009. pp. 29–50.
29. Wilk KE, Meister K, Andrews JR. Current concepts in the rehabilitation of the overhead throwing athlete. Am J Sports Med. 2002;30(1):136–51.
30. Sciascia A, Myers N, Kibler WB, Uhl TL. Return to preinjury levels of participation after superior labral repair in overhead athletes: a systematic review. J Athl Train. 2015;50(7):767–77.
31. Harris JD, Frank JM, Jordan MA, Bush-Joseph CA, Romeo AA, Gupta AK, Abrams GD, McCormick FM, Bach BR Jr. Return to sport following shoulder surgery in the elite pitcher: a systematic review. Sports Health. 2013;5(4):367–76.
32. Brand H, van der Linde JA, van Deurzen DFP, van den Bekerom MPJ. Lacking evidence for rehabilitation following arthroscopic Bankart repair: a systematic review. J ISAKOS. 2017;2(1):14–20.
33. Boileau P, Parratte S, Chuinard C, Roussanne Y, Shia D, Bicknell R. Arthroscopic treatment of isolated type II SLAP lesions. Am J Sports Med. 2009;37(5):929–36.
34. Fedoriw WW, Ramkumar P, McCulloch PC, Lintner DM. Return to play after treatment of superior labral tears in professional baseball players. Am J Sports Med. 2014;42(5):1155–60. Epub 2014 Mar 27.
35. Moore-Reed SD, Kibler WB, Sciascia AD, Uhl T. Preliminary development of a clinical prediction rule for treatment of patients with suspected SLAP tears. Arthroscopy. 2014;30(12):1540–9.
36. Patterson BM, Creighton RA, Spang JT, Roberson JR, Kamath GV. Surgical trends in the treatment of superior Labrum anterior and posterior lesions of the shoulder. Am J Sports Med. 2014;42(8):1904–10.
37. Ek ET, Shi LL, Tompson JD, Freehill MT, Warner JJ. Surgical treatment of isolated type II superior labrum anterior-posterior (SLAP) lesions: repair versus biceps tenodesis. J Shoulder Elb Surg. 2014;23(7):1059–65.
38. Yoo JC, Ahn JH, Lee SH, Lim HC, Choi KW, Bae TS, Lee CY. A biomechanical comparison of repair techniques in posterior type II superior labral anterior and posterior (SLAP) lesions. J Shoulder Elb Surg. 2008;17(1):144–9.

39. Morgan RJ, Kuremsky MA, Peindl RD, Fleischli JE. A biomechanical comparison of two suture anchor configurations for the repair of type II SLAP lesions subjected to a peel-back mechanism of failure. Arthroscopy. 2008;24(4):383–8.
40. Friedman JL, FitzPatrick JL, Rylander LS, Bennett C, Vidal AF, McCarty EC. Biceps Tenotomy versus Tenodesis in active patients younger than 55 years: is there a difference in strength and outcomes? Orthop J Sports Med. 2015;3(2):2325967115570848.
41. Moon SC, Cho NS, Rhee YG. Analysis of "hidden lesions" of the extra-articular biceps after subpectoral biceps Tenodesis: the subpectoral portion as the optimal Tenodesis site. Am J Sports Med. 2015;43:63–8.
42. Sears BW, Spencer EE, Getz CL. Humeral fracture following subpectoral biceps tenodesis in 2 active, healthy patients. J Shoulder Elb Surg. 2011;20(6):e7–11.
43. Beason DP, Shah JP, Duckett JW, Jost PW, Fleisig GS, Cain EL Jr. Torsional fracture of the humerus after subpectoral biceps Tenodesis with an interference screw: a biomechanical cadaveric study. Clin Biomech (Bristol, Avon). 2015;30(9):915–20.
44. Gomes N, Silva MR, Pereira H, Aido R, Sampaio R. Long biceps subpectoral Tenodesis with suspensory button and bicortical fixation. Arthrosc Tech. 2017;6(4):e1049–55.
45. Van Kleunen JP, Tucker SA, Field LD, Savoie FH. Return to high-level throwing after combination infraspinatus repair, SLAP repair, and release of glenohumeral internal rotation deficit. Am J Sports Med. 2012;40(11):2536–41. Epub 2012 Oct 10.
46. Ide J, Maeda S, Takagi K. Arthroscopic transtendon repair of partial-thickness articular-side tears of the rotator cuff: anatomical and clinical study. Am J Sports Med. 2005;33(11):1672–9. Epub 2005 Aug 10.
47. Liem D, Lichtenberg S, Magosch P, Habermeyer P. Arthroscopic rotator cuff repair in overhead-throwing athletes. Am J Sports Med. 2008;36(7):1317–22. Epub 2008 Apr 28.
48. Mazoue CG, Andrews JR. Repair of full- thickness rotator cuff tears in professional baseball players. Am J Sports Med. 2006;34(2):182–9. Epub 2005 Oct 31.
49. Shaffer B, Huttman D. Rotator cuff tears in the throwing athlete. Sports Med Arthrosc Rev. 2014;22(2):101–9.
50. Kuhn JE. Current concepts: rotator cuff pathology in athletes—a source of pain or adaptive pathology? Curr Sports Med Rep. 2013;12(5):311–5.
51. Castagna A, Borroni M, Garofalo R, Rose GD, Cesari E, Padua R, Conti M, Gumina S. Deep partial rotator cuff tear: transtendon repair or tear completion and repair? A randomized clinical trial. Knee Surg Sports Traumatol Arthrosc. 2015;23(2):460–3.

How to Manage Failed Slap Repair

9

Néstor Zurita and Angel Calvo Díaz

9.1 Introduction

Injuries to the superior labrum from anterior to posterior (SLAP) represent a significant cause of shoulder pain especially in people involved in repetitive overhead activities [1]. Indeed, understanding of etiology and pathology of SLAP lesions is mainly related to the development of arthroscopic techniques [2, 3].

Isolated SLAP lesions are rare (about 10%) and normally are associated with other injuries to the long head of biceps (LHB) tendon, rotator cuff, and/or to glenohumeral instability. In this context, establishing the diagnosis of a SLAP lesion is difficult when injuries are overlapped [4, 5].

Treatment of SLAP lesions is still controversial even after diagnostic shoulder arthroscopy [4, 6]. There are different treatment modalities according to the pattern of SLAP lesion. Moreover, treatment plan depends on age and functional level of the patient [7].

Although several studies analyzed injury mechanism and symptoms and correlation between them [8, 9], there is no clear evidence about factors that can predict success or failure of SLAP repair [10].

N. Zurita (✉)
IMED Elche Hospital, Elche, Spain

A. C. Díaz
Arthrosport Zaragoza, Zaragoza, Spain

In the present chapter, we aimed to analyze and discuss common problems concerning diagnosis, surgical treatment, and management of failed SLAP repair.

9.2 How to Prevent Diagnostic Errors

SLAP tears have been recognized as a common cause of shoulder pain and dysfunction in specialized patient populations, namely, athletes taking part in overhead activities and heavy-duty workers [11, 12].

Clinical diagnosis of SLAP lesion is extremely challenging, because there are no unique clinical findings associated with this pathology.

There are many clinical tests described to detect a SLAP lesion. They are usually sensitive but not specific (1). The most frequently reported are:

- Active compression test/O'Brien's test
- Biceps load test II
- O'Driscoll's dynamic labral shear test
- Speed's test
- Labral tension test

No convincing data confirmed superiority of one of these tests for diagnostic accuracy for SLAP lesions, excepting the biceps load test II (Fig. 9.1) [13–17].

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_9

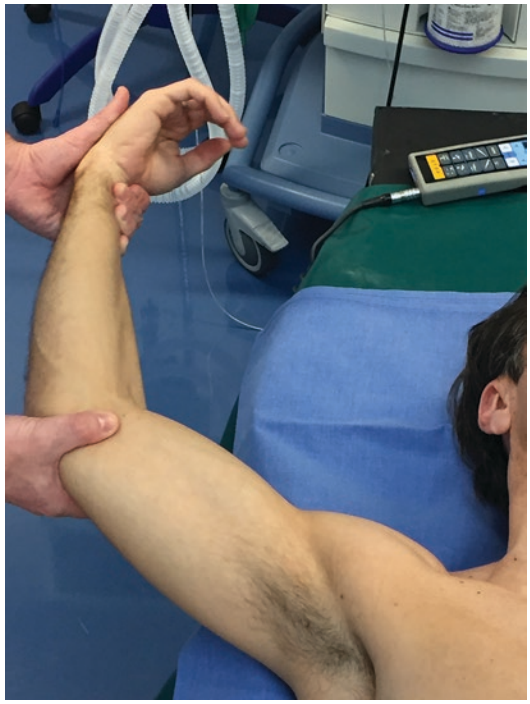

Fig. 9.1 Biceps load test II

Indeed, a correct diagnostic approach should pay great attention to the pathomechanics of SLAP lesions, which is mainly related to one of the following injury mechanisms:

- Hyperabduction and external rotation during throwing or climbing sports produces an increase of shear and compressive forces on the glenohumeral joint and strain on the rotator cuff and capsulolabral structures [17]. Maximal abduction and external rotation produces a twist at the base of the LHB tendon that transmits torsional force to the area, thus creating a SLAP lesion by a "peel-back" mechanism [18].
- Repetitive microtraumas by raising the arm over the shoulder with rotational components in manual workers.
- Scapula dyskinesia. When the scapula does not perform its action properly, its malposition decreases normal shoulder function and causes visible alterations in scapular position and motion patterns [7].

Diagnostic tools are necessary for a correct handling in the detection of a SLAP lesion. Currently, magnetic resonance arthrography (MRA) is the gold standard imaging method to diagnose a SLAP lesion, as the intra-articular injected contrast medium expands the joint capsule, outlines intra-articular structures, and leaks into tears [19]. SLAP lesions are best seen on coronal oblique sequences in the abduction-external rotation (ABER) position, as the contrast medium fills the gap between glenoid and superior labrum [20]. However, the high prevalence of false positive cases at MRA makes necessary a detailed correlation of the exam with clinical history and physical examination in order to achieve a correct diagnosis.

9.3 Surgical Treatment

The first question we should ask, once surgery started, is what to do with anatomical variants of the anterior and superior labrum like the sublabral foramen and the Buford complex.

Indeed, several studies reported on these anatomical variants related to a higher rate of SLAP injuries. In this way, when a patient suffers from symptoms and no other injuries are detected at diagnostic arthroscopy, it may be necessary to repair these variants [21].

Assessment of associated pathology is critical for the success of the procedure. Therefore, SLAP repair has consistent risk of failure when associated shoulder instability is not adequately recognized and addressed. Similarly, procedure is frequently unsuccessful for repair of type IV SLAP lesions or when severe degenerative changes to the LHB tendon are present. Associated supraspinatus or subscapularis tears that involve the biceps groove are signs of biceps instability. In this context, LHB tenotomy or tenodesis should be considered to avoid failure of the procedure.

Waterman et al. [22] revealed that arthroscopic subacromial interventions for associated rotator cuff tears, impingement, and/or symptomatic acromioclavicular osteoarthritis occurred in nearly one-third of patients with type II SLAP repair (30.7%) in a military population. Interestingly, patients with concomitant treatment of rotator cuff tears had a significantly higher return-to-duty rate than those service members with isolated SLAP repair, while

arthroscopic subacromial decompression and/or distal clavicle excision failed to yield significantly improved rates of functional return.

The presence of instability injuries associated with SLAP lesions requires combined surgical repair of SLAP and instability injuries [22].

A special case is the type II SLAP lesion. Frank et al. [10] analyzed prognostic factors significantly associated with failure of surgical treatment. The authors observed that when revision surgery was used as indicator of failure, the most significant predictors were overhead throwing sports and age less than 20 years; when ASES scoring system was considered (score less than 50 as indicator of failure), the most significant prognostic factors were age greater than 40 years, heavy laborers, users of tobacco and/or alcohol, diabetics, and/or patients who present with persistent anterior shoulder pain (symptoms consistent with persistent SLAP lesion or bicipital groove tenderness). In these cases, the best treatment option was LHB tenodesis or tenotomy.

9.4 Tips and Tricks to Improve Surgical Management

We have to consider that SLAP injuries (especially the part of the lesion) are located in a difficult position for suturing, which can cause iatrogenic damage to the labrum or to the LHB tendon.

Even a correct repair of the labrum but too close to the biceps tendon can produce a choke phenomenon that can make the surgery fail. Indeed, biceps insertion is mostly located on posterior aspect of the superior labrum, and this should be taken into consideration when a SLAP repair is attempted; then care should be taken to avoid overtensioning of the anterior part of the labrum, thus leaving enough mobility to the tendon to avoid persistent pain.

Using the Neviaser portal for suture passage is a useful suggestion to avoid damage to the surrounding tissues during SLAP repair (Fig. 9.2). We can use direct or indirect suture repair. In my experience, I like to use an Abbocath cannulated needle with a monofilament to facilitate the access to the labrum and to cause less damage as possible to the surrounding soft tissues (Fig. 9.3).

Several repair devices can be used for SLAP repair, such as nonabsorbable or absorbable, knotted or knotless suture anchors. Metal suture anchors are the most commonly used over time. However, some complications have been reported, like articular surface damage, migration, and artifact production in postoperative MR imaging (MRI) [7].

Absorbable tacks and anchors are viable alternative to metal implants [23]. Although no

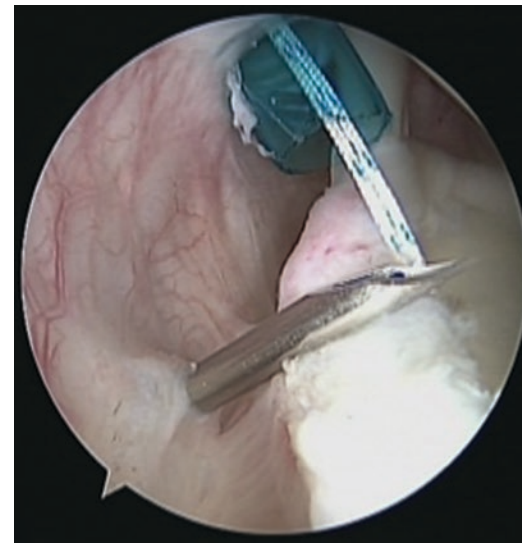

Fig. 9.2 Abbocath cannulated needle through the Neviaser portal

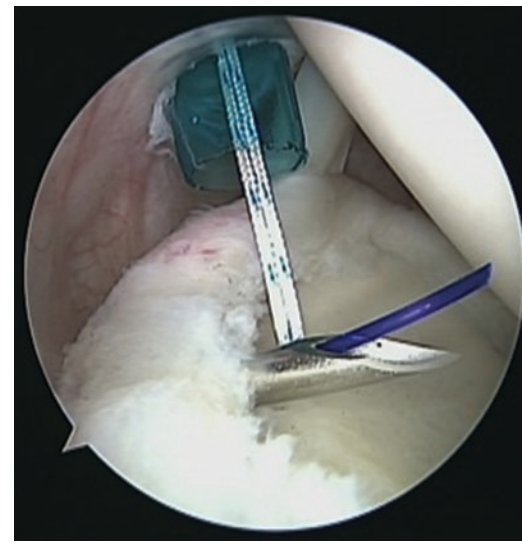

Fig. 9.3 Abbocath cannulated needle is used as an indirect suture method with a monofilament

significant differences have been reported between metal and absorbable devices for intra-articular use [24], foreign body reactions, synovitis, and chondral damage were described with the use of absorbable devices [25, 26]. The newest absorbable anchors are designed to decrease complication rates [27, 28]. However, McCarthy et al. [29] described several cases of papillary synovitis, chondral damage, and giant cell reactions. Probably, the use of all-suture systems could reduce the risk of the abovementioned adverse effects by preserving the bone stock and reducing the contact with foreign material.

Knotless anchors are another viable option. Kocaoglu et al. [30] reported results of repair with knotless anchors that were comparable with those obtained with standard anchors. Biomechanical studies showed that initial fixation strength of knotless anchors was similar to that of standard suture anchors. Furthermore, the absence of knots in the intra-articular environment may reduce the risk of mechanical irritation to the joint surfaces [31].

9.5 What Can We Do When All Previous Treatment Failed

Clinical outcome of SLAP repair has been reported as good to excellent in 63–100% of the patients; therefore, approximately one-third of patients are still dissatisfied after the procedure [32].

The highest prevalence of SLAP repair is reported in the 20–29 and 40–49 year ranges [2]. In this context, there are conflicting results about type II SLAP repair in patients older than 40 years. Boileau et al. [33] found that LHB tenodesis is superior to SLAP repair in older population. Conversely, Alpert et al. [34] showed that repair of type II SLAP lesions using suture anchors can provide good results in patients older than age 40.

Regarding functional outcomes, Morgan et al. [18] published a retrospective review of 102 patients who underwent arthroscopic repair of type II SLAP tears. They reported worst results in athletes respect to the general population. Provencher et al. [3] found that after SLAP repair, 37% of the patients cannot get a reliable return to the previous activity level with a 28% revision rate.

Biceps tenodesis has demonstrated utility in carefully select patients with superior labral pathology, particularly those of older chronological age and/or non-throwing athletes [33]. Waterman et al. [22] found that 31 patients required revision surgery for failed SLAP repair. Of those, 25 patients underwent secondary subpectoral biceps tenodesis with a 76% return-to-duty rate. Conversely, revision SLAP repair was associated with significantly lower rates of return to duty (16.7%), which is consistent with prior reports in the literature indicating poor clinical outcomes with revision repair [35].

Nonoperative treatment is also largely unsuccessful for treatment of persistent symptoms after prior arthroscopic SLAP repair, with only 29% of patients reporting good to excellent results without further surgery [36].

References

1. Knesek M, Skendzel JG, Dines JS, Altchek DW, Allen AA, Bedi A. Diagnosis and management of superior labral anterior posterior tears in throwing athletes. Am J Sports Med. 2013;41:444–60.
2. Park JY, Chung SW, Jeon SH, Lee JG, Oh KS. Clinical and radiological outcomes of type 2 superior labral anterior posterior repairs in elite overhead athletes. Am J Sports Med. 2013;41:1372–9.
3. Provencher MT, McCormick F, Dewing C, McIntire S, Solomon D. A prospective analysis of 179 type 2 superior labrum anterior and posterior repairs: outcomes and factors associated with success and failure. Am J Sports Med. 2013;41:880–6.
4. Grauer JD, Paulos LE, Smutz WP. Biceps tendon and superior labral injuries. Arthroscopy. 1992;8:488–97.
5. Maffet MW, Gartsman GM, Moseley B. Superior labrum-biceps tendon complex lesions of the shoulder. Am J Sports Med. 1995;23:93–8.
6. Gobezie R, Zurakowski D, Lavery K, Millett PJ, Cole BJ, Warner JJ. Analysis of interobserver and intraobserver variability in the diagnosis and treatment of SLAP tears using the Snyder classification. Am J Sports Med. 2008;36:1373–9.
7. Aydin N, Sirin E, Arya A. Superior labrum anterior to posterior lesions of the shoulder: diagnosis and arthroscopic management. World J Orthop. 2014;5(3):344–50.
8. Katz LM, Hsu S, Miller SL, et al. Poor outcomes after SLAP repair: descriptive analysis and prognosis. Arthroscopy. 2009;25(8):849–55.

9. Park JH, Lee YS, Wang JH, Noh HK, Kim JG. Outcome of the isolated SLAP lesions and analysis of the results according to the injury mechanisms. Knee Surg Sports Traumatol Arthrosc. 2008;16(5):511–5.
10. Frank R, Nho SJ, McGill KC, Grumet RC, Cole BJ, Verma NN, Romeo AA. Retrospective analysis of arthroscopic superior labrum anterior to posterior repair: prognostic factors associated with failure. Adv Orthop. 2013;2013:1–7.
11. Mazzocca AD, Cote MP, Solovyova O, Rizvi SH, Mostofi A, Arciero RA. Traumatic shoulder instability involving anterior, inferior, and posterior labral injury: a prospective clinical evaluation of arthroscopic repair of 270° labral tears. Am J Sports Med. 2011;39:1687–96.
12. Van Kleunen JP, Tucker SA, Field LD, Savoie FH. Return to high-level throwing after combination infraspinatus repair, SLAP repair, and release of glenohumeral internal rotation deficit. Am J Sports Med. 2012;40:2536–41.
13. Cook C, Beaty S, Kissenberth MJ, Siffri P, Pill SG, Hawkins RJ. Diagnostic accuracy of five orthopedic clinical tests for diagnosis of superior labrum anterior posterior (SLAP) lesions. J Shoulder Elb Surg. 2012;21:13–22.
14. Karlsson J. Physical examination tests are not valid for diagnosing SLAP tears: a review. Clin J Sport Med. 2010;20:134–5.
15. Bennet WF. Specificity on the speed's test: arthroscopic technique for evaluating the biceps tendon at the level of bicipital groove. Arthroscopy. 1998;14:789–96.
16. Rockwood AC, Matsen FA, Wirth MA, Lippitt SB, editors. The shoulder. Amsterdam: Elsevier; 2009. p. 145–76.
17. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology part I: pathoanatomy and biomechanics. Arthroscopy. 2003;19:404–20.
18. Morgan CD, Burkhart SS, Palmeri M, Gillespie M. Type II SLAP lesions: three subtypes and their relationships to superior instability and rotator cuff tears. Arthroscopy. 1998;14:553–65.
19. Jee WH, McCauley TR, Katz LD, Matheny JM, Ruwe PA, Daigneault JP. Superior labral anterior posterior (SLAP) lesions of the glenoid labrum: reliability and accuracy of MR arthrography for diagnosis. Radiology. 2001;218:127–32.
20. Bedi A, Allen A. Superior labral lesions anterior to posterior-evaluation and arthroscopic management. Clin Sports Med. 2008;27:607–30.
21. Kanatli U, Ozturk BY, Bolukbasi S. Anatomical variations of te anterosuperior labrum: prevalence and association with type II superior labrum antero-superior (SLAP). J Shoulder Elb Surg. 2010;19(8):1199–203.
22. Waterman BR, Arroyo W, Heida K, Burks R, Pallis M. SLAP repairs with combined procedures have lower failure rate than isolated repairs in a military population. Surgical outcomes with minimum 2-year follow-up. Orthop J Sports Med. 2015;3(8):2325967115599154.
23. Ozbaydar M, Elhassan B, Warner JJ. The use of anchors in shoulder surgery: a shift from metallic to bioabsorbable anchors. Arthroscopy. 2007;23:1124–6.
24. Milano G, Grasso A, Santagada DA, Saccomanno MF, Deriu L, Fabbriciani C. Comparison between metal and biodegradable suture anchors in the arthroscopic treatment of traumatic anterior shoulder instability: a prospective randomized study. Knee Surg Sports Traumatol Arthrosc. 2010;18:1785–91.
25. Freehill MQ, Harms DJ, Huber SM, Atlihan D, Buss DD. Poly-L-lactic acid tack synovitis after arthroscopic stabilization of the shoulder. Am J Sports Med. 2003;31:643–7.
26. Wilkerson JP, Zvijac JE, Uribe JW, Schürhoff MR, Green JB. Failure of polymerized lactic acid tacks in shoulder surgery. J Shoulder Elb Surg. 2003;12:117–21.
27. 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.
28. Stein T, Mehling AP, Ulmer M, Reck C, Efe T, Hoffmann R, Jäger A, Welsch F. MRI graduation of osseous reaction and drill hole consolidation after arthroscopic Bankart repair with PLLA anchors and the clinical relevance. Knee Surg Sports Traumatol Arthrosc. 2012;20:2163–73.
29. McCarty LP, Buss DD, Datta MW, Freehill MQ, Giveans MR. Complications observed following labral or rotator cuff repair with use of poly-L-lactic acid implants. J Bone Joint Surg Am. 2013;95:507–11.
30. Kocaoglu B, Guven O, Nalbantoglu U, Aydin N, Haklar U. No difference between knotless sutures and suture anchors in arthroscopic repair of Bankart lesions in collision athletes. Knee Surg Sports Traumatol Arthrosc. 2009;17:844–9.
31. Uggen C, Wei A, Glousman RE, ElAttrache N, Tibone JE, McGarry MH, Lee TQ. Biomechanical comparison of knotless anchor repair versus simple suture repair for type II SLAP lesions. Arthroscopy. 2009;25:1085–92.
32. Brockmeier SF, Voos JE, Williams RJ, Altchek DW, Cordasco FA, Allen AA. Outcomes after arthroscopic repair of type-II SLAP lesions. J. Bone Joint Surg. A. 2009;91(7):1595–603.
33. Boileau P, Parratte S, Chuinard C, Roussanne Y, Shia D, Bicknell R. Arthroscopic treatment of isolated type II SLAP lesions: biceps tenodesis as an alternative to reinsertion. Am J Sports Med. 2009;37:929–36.
34. Alpert JM, Wuerz TH, O'Donnell TF, Carroll KM, Brucker NN, Gill TJ. The effect of age on the outcomes of arthroscopic repair of type II superior labral anterior and posterior lesions. Am J Sports Med. 2010;38:2299–303.
35. Park S, Glousman RE. Outcomes of revision arthroscopic type II superior labral anterior posterior repairs. Am J Sports Med. 2011;39:1290–4.
36. Katz LM, Hsu S, Miller SL, et al. Poor outcomes after SLAP repair: descriptive analysis and prognosis. Arthroscopy. 2009;25:849–55.

Biceps Disorders: When and How to Operate

10

Sebastian Kwisda and Matthias Flury

10.1 Anatomy and Pathologies of the Long Head of the Biceps

10.1.1 Inside Lesions

The glenoid labrum is a collagenous rim that is circumferentially connected to the glenoid [1]. There are known variations of its anatomy such as the sublabral hole and the Buford complex, which need to be differentiated from labral pathologies during arthroscopy [1–6]. While the inferior labrum is connected firmly to the glenoid, the superior labrum has a meniscal loose attachment of thin elastic tissue [1, 7]. The long head of the biceps tendon (LHBT) originates from the supraglenoid tubercle on the 12 o'clock position and from the superior labrum. The bony attachment is approximately 5 mm medial to the glenoid's edge. Vangsness et al. [8] described four different types of LHBT attachments to the glenoid labrum:

- Type I attaches completely to the posterior labrum.
- Type II attaches mainly to the posterior labrum with some anterior remnants.
- Type III attaches in equal parts to the anterior and posterior labrum.
- Type IV attaches mainly to the anterior biceps labrum.

The majority of the LHBTs, which originate from the superior labrum, are either type I or II [8, 9]. Labral vascularity originates from the suprascapular, anterior humeral circumflex and the posterior humeral circumflex arteries. There is no transosseous vascularity from the glenoid bone [1]. The limited vascularization of the superior labrum is of particular interest because it limits the inherent healing abilities after surgical reattachment, for instance, after superior labrum anterior and posterior (SLAP) repair.

There are many variations in origins and morphology of the LHBT. In around 9–20%, there can be accessory heads of the LHBT. Their origins vary between the greater tuberosity close to the articular capsule, the articular surface of the glenohumeral joint, and the coracoid process. Variations also include confluent LHBT with the rotator cuff or the capsule or at the anterior border of the subscapularis tendon. There is also a known bifurcated form of the tendon [10–17] (Fig. 10.1).

Inside lesions comprise mainly SLAP lesions and dynamic incarcerations of the LHBT. Injuries to the anterior-superior labrum close to the origin of the LHBT were initially described by Andrews in 1985 [18]. In 1990, Snyder et al. coined the

S. Kwisda · M. Flury (✉)
Schulthess Clinic, Zurich, Switzerland
e-mail: matthias.flury@kws.ch

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_10

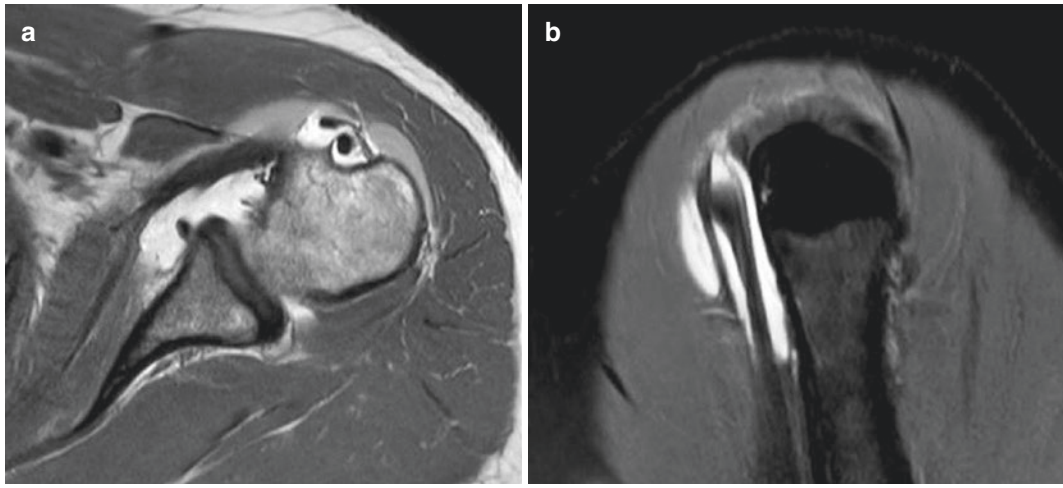

Fig. 10.1 (**a**) Axial T2-weighted MRI scan of a biparted LHBT. (**b**) Sagittal T2-weighted MRI scan of a biparted LHBT

term SLAP and subdivided it into four different types [19]. The classification introduced by Snyder remains widely accepted but has been repeatedly modified [20–22].

Type I lesions are described as a degeneration and fraying of the superior labrum while the labrum still remains firmly attached to the glenoid. These lesions occur mainly in an elderly population.

Type II lesions are described as detachments of the labrum together with the origin of the LHBT from the supraglenoid tubercle. These lesions must be carefully differentiated from normal variations of the biceps anchor during arthroscopic surgery. Non-pathologic variations may show articular cartilage under the loose meniscal attachment and show no signs of inflammation or fraying. Type II lesions are the most common ones, prevalence ranging from 21% to 55% [19, 21, 23, 24] of all SLAP lesions.

Type III lesions are bucket-handle-like lesions with the labral tear mostly beneath the anchor of the LHBT. The origin of the LHBT remains attached to the supraglenoid tubercle; therefore, the LHBT is stable. Mechanical locking of the mobile fragment can cause severe pain.

Type IV lesions are bucket handle tears that expand to the anchor of the LHBT and sometimes the tendon itself. Type IV lesions are less common with a prevalence of 4–15% [19, 21, 23, 24] (Fig. 10.2).

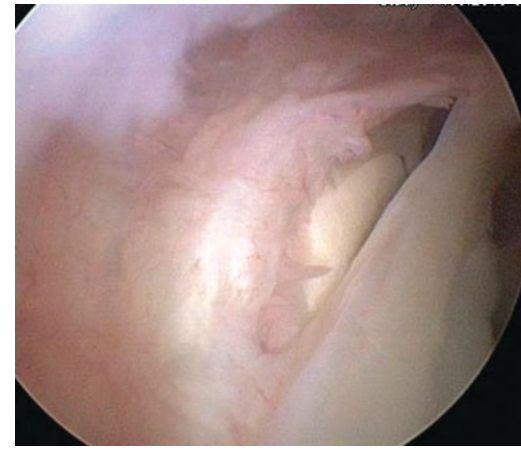

Fig. 10.2 Right shoulder, posterior viewing portal. SLAP IV lesion, the glenoid is visible through the bucket handle tear. The LHBT is still attached to the labrum and pulls the tear toward the joint

Type I and III lesions are considered stable since the anchor of the LHBT is not detached from its origin. In case of persistent pain, surgical options involve arthroscopic debridement for type I lesions and refixation or resection of the bucket handle for type III lesions.

Type II and IV tears are not considered stable, and the optimal surgical treatment is still a matter of debate. There are many known cofactors that

may influence the surgical outcome. Various studies suggest that SLAP lesions are commonly accompanied by other pathologies, including Bankart lesions, rotator cuff tears, injuries to the acromioclavicular joint, and glenohumeral chondral lesions [19, 21, 23–25]. Injury mechanisms vary widely throughout the literature but can be subdivided in acute traumatic injuries and depletion injuries that mainly occur in overhead athletes. Other potential influencing factors include patient age, quality of labral tissue, and patient's activity level. The current surgical options for SLAP II and IV lesions involve labral repair, biceps tenodesis, or tenotomy. A systematic review conducted by Sayde et al. assesses outcomes after SLAP repair with isolated SLAP II lesions. The results showed that 83% of patients had "good-to-excellent" subjective outcome (patient satisfaction), and 73% of them could return to their previous level of activity, whereas only 63% of overhead athletes could return to their previous level of play [26]. In a more recent study, Fedoriw et al. retrospectively reviewed outcomes of professional baseball players with SLAP tears depicted in magnetic resonance imaging (MRI) scans. Patients had either been treated conservatively or with SLAP repair. Both groups had poor return to play rates with 40% vs. 48% and even worse rates for return to their previous level with 22% vs. 7% [27]. While successful outcomes after SLAP repairs in an ordinary population range from 65% to 94% [25, 28–31], rates for return to sport are significantly lower, ranging from 20% to 87% [31, 32]. Boileau et al. compared outcomes of SLAP repair and biceps tenodesis for patients with isolated type II lesions. Their results showed that 80% of the patients in the tenodesis group were subjectively satisfied and 87% could return to their previous level of sport. These results stood in vast contrast to the results of the SLAP repair group, where 40% were subjectively satisfied and only 20% could return to sports [33]. Ek et al. found no significant difference for patients with isolated SLAP II lesions after either tenodesis or SLAP repair in ASES score, patient satisfaction, or return to previous sporting level [34]. However, there was a selection bias in both studies due to a significant age difference between the groups: patients who received SLAP repair were significantly younger than those in tenodesis groups which could imply higher functional requirements [33, 34]. Denard et al. compared outcomes after arthroscopic biceps tenodesis with SLAP repair for isolated type II lesions in patients older than 35 years. They found no significant difference in ASES score, UCLA score, and functional outcome. At the same time, patients had shorter rehabilitation time, more predictable outcomes, higher subjective satisfaction, and return to activity with biceps tenodesis compared to SLAP repair [35]. The good results of biceps tenodesis as a salvage procedure after failed SLAP repairs led authors to turn to biceps tenodesis as primary treatment option for SLAP tears. Gottschalk et al. treated 29 patients with SLAP II and IV tears with primary subpectoral biceps tenodesis. Ninety percent of the patients could return to their previous level of sport [36]. These results were confirmed by Gupta et al., who evaluated 28 patients after primary subpectoral biceps tenodesis and had excellent results for all validated outcome measures [37].

In our opinion, patients' ages and their individual demands are important determinants for the choice of surgical treatment for SLAP lesions. According to the current literature [38], we use the following treatment algorithm for SLAP II and IV lesions: SLAP repair is a viable option for patients younger than 35 years with acute trauma and high demands; older patients are either treated with tenotomy or tenodesis according to the patient's individual needs.

Incarceration of the LHBT between the glenoid and the humeral head occurs while the arm is in forward flexion and internal rotation. Incarceration by itself is not pathological but may be the cause of biceps tendinitis and/or partial rupture through attrition of the chondral surface of the anteromedial aspect of the humeral head's surface. Repeated incarceration may lead to dilation of the tendon and edema with progressive cellular infiltration. Adhesions between the tendon sheath and the tendon itself occur alongside synovial proliferation and fibrosis [39]. Over time, these reorganizing mechanisms can become

symptomatic and the cause of severe pain and limited motion. Alpantaki et al. found an extensive network of sympathetic and sensory nerves in the LHBT that were more dense in the proximal segment [40]. These findings support the theory that tendinitis of the proximal part of the LHBT may be the source of severe pain. Boileau et al. described an "hourglass biceps" deformity, in which the proximal hypertrophy of the LHBT leads to an entrapment of the tendon in the glenohumeral joint [41]. In case of SLAP lesions combined with tendinitis of the LHBT, we do not perform SLAP repairs.

10.1.2 Junction Lesions

The junction is characterized as the intra-articular part of the LHBT from the articular margin to the exit from the glenohumeral joint and from the stabilizing and constraining biceps pulley. The LHBT, measured from its origin at the supraglenoid tubercle to the musculotendinous junction, has an average length of 99–138 mm [42–44]. The intra-articular diameter of the tendon is approximately 6.6 mm, while the extra-articular diameter is slightly smaller with an average of 5.1–6 mm [44, 45]. Swelling of the synovium, which is associated with an inflammatory process, may cause a mismatch between the enlarged tendon and the noncompliant intertubercular groove, leading to stenosis of the tendon in the groove. The anterior humeral circumflex artery, the vincular attachments, and the labrum tributaries provide the blood supply for the LHBT [1, 46]. There are two zones of the LHBT, namely, a traction zone and a sliding zone. In the sliding zone, the tendon is in direct contact with the bony intertubercular groove. In the traction zone, the intratendinous blood supply resembles the vascularization of other tendons, while vascularization in the sliding zone is considerably lower [47]. This hypovascular region runs from the articular margin to the intertubercular groove and makes the tendon more susceptible to degeneration and/or rupture [46]. Many authors suggest that the shape of the intertubercular groove is directly involved in the pathomechanics of LHBT ruptures [48–50]. Shallow grooves are commonly associated with instability of the LHBT, whereas narrow grooves are believed to be accompanied by a sharp medial wall. Bony changes of the intertubercular groove like osteophytes and spurs on the floor of the groove may erode or wear down the tendon and lead to tendinitis or rupture [50]. Even though changes of the groove may be the cause of LHBT problems, some of the bony changes may be a result of underlying soft tissue changes around the groove. The LHBT passes directly under the critical zone of the supraspinatus through the rotator interval. Refior et al. found that the most proximal part of the tendon close to the origin as well as the segment that exits the joint is most susceptible to microscopic degenerative changes [51].

Arthroscopy is the gold standard for visualization of pathologies in the junction area, but numerous studies suggest a limited visibility of tunnel lesions. Average excursion of the LHBT during arthroscopic pull test is 15–19 mm in cadaveric studies and only 14 mm in vivo [42, 52, 53]. Moon et al. reported that 79% of intra-articular biceps tears showed "hidden lesions" beyond the bicipital groove [54]. A recent study of Gilmer et al. [52] compared visualization of the pathologies of the LHBT during arthroscopic and subpectoral tenodesis and found that significantly ($p < 0.001$) more "hidden lesions" could be diagnosed in the tenodesis group. Due to the risk of persisting symptoms caused by "hidden lesions," we mainly use subpectoral tenodesis or tenotomy for patients with tendinitis or partial ruptures in the junction area.

Junctional stabilization of the LHBT bears on a capsule-ligamentous stabilizing complex also known as biceps pulley. It is formed by coalescing fibers of the superior glenohumeral ligament (SGHL), the coracohumeral ligament (CHL), as well as parts of the subscapularis and supraspinatus tendons. The biceps pulley stabilizes the LHBT as it exits the joint [55]. Braun et al. differentiated lesions of the biceps pulley in anteromedial and posterolateral according to the force vector [56] (Fig. 10.3).

In their study containing 207 patients who underwent arthroscopic surgery of the shoulder,

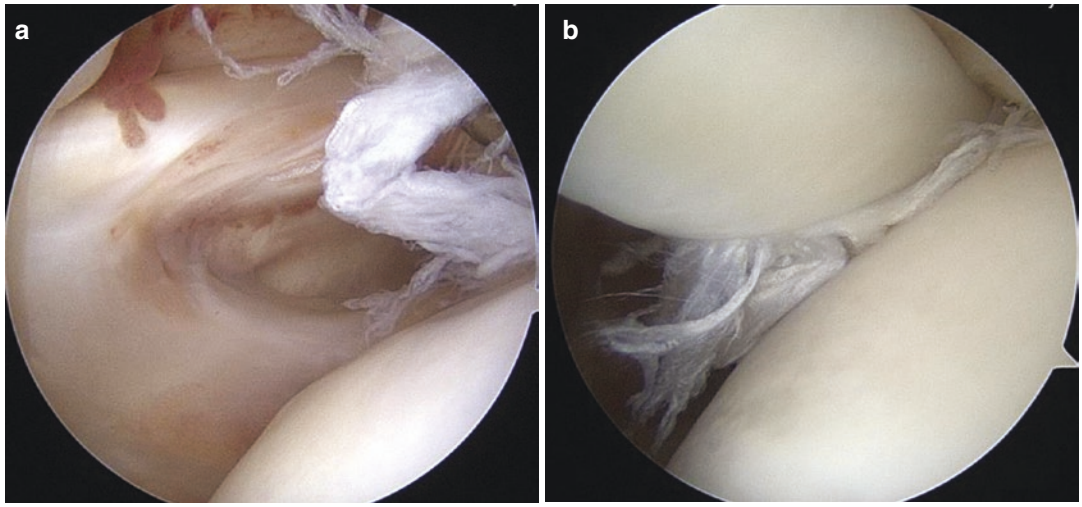

Fig. 10.3 Right shoulder, posterior viewing portal. (**a**) Anteromedial lesion of the biceps pulley with cranial split of the subscapularis tendon and medial instability of the LHBT. (**b**) Fraying of the biceps pulley with no current signs of synovitis due to instability of the LHBT

32% had lesions of the biceps pulley. These lesions were highly associated with instability and/or subluxation of the LHBT [57]. Lesions of the anteromedial pulley occurred slightly more often than those of the posterolateral pulley [56]. Bennett introduced a classification system based on the injured structures [58]. Surgical options for a displaced tendon consist of tenotomy, tenodesis, as well as relocation and reconstruction of the biceps pulley. In 1994, Walch et al. published their results after exploration of the rotator interval during the repair of 116 apparently isolated supraspinatus tendon tears. They found "hidden lesions" of the coracohumeral ligament, the SGHL, and the superior portion of the subscapularis tendon in 19 patients. Fourteen of them had a subluxated LHBT and two had ruptured LHBT. The supraspinatus tendon and the torn structures of the biceps pulley were reinserted after the LHBT was recentered in the groove. Patients were reviewed after a mean follow-up of 20 months. Re-rupture of the LHBT was observed in 25% of the patients after recentering of the tendon [59]. McClelland et al. examined a series of 16 patients with combined lesion of the subscapularis tendon and biceps pulley. Patients were treated with relocation of the LHBT and reconstruction of the pulley system combined with rotator cuff repair. After a mean follow-up of 26 months, mobility and location of the LHBT were evaluated during ultrasound scanning. Only 8 of the 16 patients had a static tendon. Four out of six patients that had received additional groove deepening showed a stable tendon during dynamic ultrasound evaluation [60]. These results stand in contrast to the findings of Bennett et al., who could show significant improvement after a 2-year follow-up in postoperative ASES scores; total, subjective, and objective Constant score; and VAS after arthroscopic sheath repair in 18 patients, who had lesions that had affected both the medial and lateral wall of the bicipital sheath [61]. The literature shows mixed results after pulley repair. Depending on a patient's age, individual demand, and activity level as well as comorbidities, we either use tenotomy or tenodesis in case of instability of the LHBT.

10.1.3 Extra-Articular Lesions

The extra-articular segment of the LHBT or bicipital tunnel extends from the articular margin to the subpectoral region [62]. This segment is subdivided into three separate zones: zone 1 consists of the osseous bicipital groove with its

aperture created by the pulley system to the distal margin of the subscapularis tendon (DMSS); zone 2 extends from the DMSS to the proximal margin of the pectoralis major tendon (PMPM); and zone 3 comprises the subpectoral region. Zone 1 has close osseous borders with an average depth of 4.3 mm and a proximal width of 8.8 mm. These borders are prone to degenerative changes with osteophyte formation [63]. In zone 2, the groove becomes shallower with overlying periosteum. Zone 1 and 2 show similar characteristics, like the presence of synovium [62]. Zone 3 has a mainly flat osseous floor. The medial boundaries of the LHBT consist of loose connective tissue with an increase in space compared to zone 1 and 2 [62]. Due to this increased space, suppressing lesions such as scars, osteophytes, or loose articular bodies have less consequences. Patients with chronical symptoms of the biceps often have lesions of the bicipital tunnel that may go unnoticed during diagnostic arthroscopic evaluation [42]. The bicipital tunnel syndrome as described by Taylor et al. has a prevalence of close to 50% [42] in this collective. About one third of the lesions of the BLC are concealed by the bicipital tunnel and are underestimated during arthroscopic surgery [52]. About 80% of LHBT tears extend over the articular margin in the bicipital tunnel and show signs of synovitis [54] (Fig. 10.4).

In their retrospective evaluation of 277 patients who underwent subdeltoid transfer of the LHBT to the conjoint tendon, Taylor et al. found "hidden lesions" in 47% of their patients.

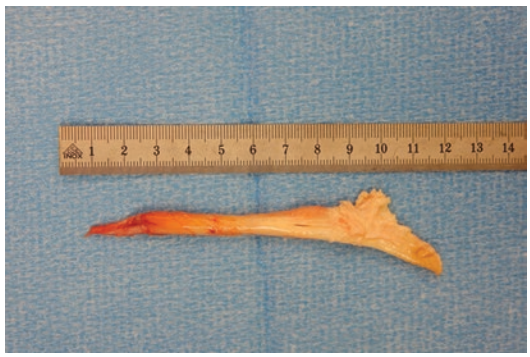

Fig. 10.4 Tendinitis and fraying of the LHBT in zones 1 and 2. The intra-articular region appears healthy without signs of synovitis (proximal right, distal left)

Adhesions and/or scarring were most common and accounted for 48% [42].

In three systematic reviews, both tenotomy and tenodesis were deemed effective treatment options for lesions of the BLC. 74–77% of patients had good to excellent results, but in 19–24% the symptoms of the LHBT persisted even after surgery [64–66]. A systematic review by Taylor et al. assessing the clinical impact of biceps tunnel decompressing versus non-decompressing techniques showed higher postoperative Constant scores after tunnel decompression. The authors note that the literature mainly consists of single-cohort retrospective observational trials that have probably been influenced by publication bias [67]. Even though many contributing factors can influence surgical outcomes, and the literature does not show evidence for our claim as yet, we believe that missed and untreated bicipital tunnel disease can be a decisive factor in persisting pain after surgery. For this reason we prefer decompressing techniques or resection techniques such as the subpectoral fixation for tenodesis over non-decompressing ones.

10.2 Surgical Options

10.2.1 Tenotomy or Tenodesis

Up to date surgicalmanagement involves tenotomy and multiple techniques for tenodesis [68–75]. Authors advocating tenotomy argue that it is simple to perform and well tolerated; it shortens operating time as well as rehabilitation and allows early return to activity [66, 76, 77]. Authors report good to excellent outcomes for the majority of their patients [76–78]. However, tenotomy is associated with a cosmetic deformity known as "popeye" deformity, possible muscle cramping, decreased supination peak torque, and fatigue pain [76–81].

Postoperative results after tenodesis show similar results compared to tenotomy [74, 75, 82–84]. Proponents of tenodesis advocate that a close restauration of normal anatomy produces better length-tension relation, and therefore

supination and elbow flexion strength is maintained. Less biomechanical changes after surgery, fewer cosmetic deformities, and less cramping justify longer rehabilitation and operating time as well as increased costs [74, 75, 85, 86]. Revision for biceps tenodesis-related issues is rare [87]. There are numerous studies comparing outcomes after tenotomy and various tenodesis techniques with no clear consensus on optimal surgical treatment of LHBT lesions [66, 81, 88, 89].

Recently, three systematic reviews [64–66] showed a significant improvement in objective outcome parameters after tenotomy and tenodesis. Seventy seven percent of the patients showed good to excellent results after tenotomy, with cosmetic deformity ranging from 41% to 43%. Persistent postoperative bicipital pain was found in 19%. After tenodesis procedures 74% showed good to excellent results, with postoperative deformity ranging from 8% to 25%. Persisting pain occurred in 24% of patients. The four surveys that directly compared the two methods found no significant difference in outcome, except for the cosmetic deformity. Available biomechanical data showed lower load to failure after tenotomy compared with tenodesis (81.6 vs. 233.5 N). However, the authors note that due to the lack of prospective randomized trials it remains difficult to recommend one method.

Up to date, surgical recommendations are most commonly based on patients' age due to a lack of high-level evidence [90]. Duff and colleagues [91] looked at patients' acceptance after 117 tenotomies regardless of their age and physical activity and found no significant difference in cramping, weakness, or deformity between younger manually active and older sedentary groups; 95% of their patients were satisfied or very satisfied with surgical outcome; 3% were concerned with cosmetic deformity but did not request correction; and 19% had postoperative muscle cramping.

More recently, Galdi et al. [92] tried to determine predictive factors for patients' preferences for biceps tenotomy or tenodesis. They developed a biceps-specific questionnaire to evaluate which factors influence patient's perception of a successful surgical outcome. The authors analyzed preferences of 100 patients (51 male, 49 female) after they had been given a short summary on available literature and found an overall preference for tenodesis. Surprisingly, age was not a statistically significant variable for predicting which method a patient might choose. Significant predictors toward preference for tenodesis were female sex, concern regarding cosmetic deformity, and importance of pain relief. Factors influencing preference for tenotomy were male sex, higher pain scores, concerns regarding the usage of hardware, and recovery time. Factors with no significant predictive value were patients' age, body mass index, concern of postoperative muscle cramping, importance of elbow flexion strength, occupation, and income level. According to this data, choosing either tenotomy or tenodesis should not be mainly based on patient's age, and surgeons should take individual needs into account.

10.2.2 Techniques and Biomechanical Requirements for Tenodesis

Over the years, a number of different techniques for biceps tenodesis have been described and evaluated. They can be classified according to fixation technique (soft tissue to soft tissue vs. soft tissue to bone), surgical exposure (open vs. arthroscopic approaches), and decompression vs. non-decompression of the bicipital sheath [75, 79, 93–102]. In recent years, indications for proximal biceps tenodesis were expanded to include primary and revision of SLAP lesions that have historically been treated with SLAP repair and, in case of failure, revision repair [33, 103, 104]. As the popularity of tenodesis increases fast [105], different fixation options have been publicized. Fixation techniques involve soft tissue tenodesis [106–108], keyhole procedure [94], transfer to the conjoint tendon [109], suture anchor [106, 108, 110], screw fixation [72, 111], and button fixation [112].

The force necessary to lift the arm against gravity is approximately 75 N. Lifting the arm while holding a weight of 2.5 kg requires around

300 N [113]. Minimal mechanical requirements on tenodesis for initial stability after surgery should therefore reach 100–200 N. Several authors assessed the biomechanical stability of before mentioned fixation techniques. Ultimate load to failure (UFL) was less than 100 N for soft tissue tenodesis, 101 N for keyhole technique, 129–164 N for suture anchors, 169–174 N for buttons, and 252 N for button in combination with an interference screw. Isolated interference screw fixation had a UFL ranging from 150 N to 165 N and from 205 N to 252 N when a whip-stitch was performed [108, 111, 114–116].

Several studies investigated the optimal location of tenodesis. All-arthroscopic LHBT tenodesis can be performed in the rotator interval, at the groove entrance, or in a suprapectoral position close to proximal border of the pectoralis major tendon. Subpectoral tenodesis is usually performed through an open or mini-open approach at the inferior border of the pectoralis major tendon.

Tenodesis at the articular margin allows an all-arthroscopic surgery with visualization of intra-articular portion of the LHBT. It is an elegant method, especially in combination with rotator cuff reconstruction since the bicipital sheath is not decompressed (Fig. 10.5). Lee et al. examined 84 patients who had undergone arthroscopic biceps tenodesis and found that 11 patients (12.9%) showed postoperative "popeye" deformity. Brady et al. evaluated the largest retrospective cohort of soft tissue-to-bone tenodesis at the articular margin with a collective of 1083 patients and found a significant improvement in objective outcome scores and low revision rates with 4.1% [87]. Werner et al. compared the outcomes of 249 patients who had undergone either arthroscopic suprapectoral or subpectoral biceps tenodesis. They reported that the tenodesis site was significantly more proximal in the group with postoperative stiffness than in the collective without stiffness [117]. Taylor et al. reported that decompressing techniques result in significantly better postoperative Constant scores than non-decompressing techniques [67].

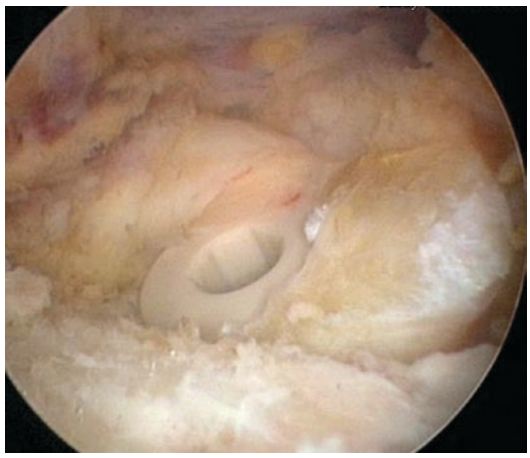

Fig. 10.5 Subacromial view, left shoulder. Interference screw fixation of the LHBT in the bicipital groove

Suprapectoral tenodesis is an all-arthroscopic technique that addresses lesions in zone 1 and 2 without interval opening. Before tenodesis, subacromial preparation is necessary. However, this technique fails to locate "hidden lesions" in zone 3. In our own patient population, we found "popeye" deformity in 7% of the patients. In a biomechanical evaluation of length-tension and mechanical strength comparing suprapectoral with subpectoral biceps tenodesis, Werner et al. showed a tendency toward overtension after arthroscopic suprapectoral tenodesis [118]. Comparative studies show consistently good to excellent outcomes and functional results after suprapectoral tenodesis [119–121].

Subpectoral tenodesis has recently been popularized because it allows full visualization of the proximal long head of the biceps tendon and removes the tendon from zones 1 and 2 (Fig. 10.6). A kinematic study evaluating the impact of subpectoral tenodesis on performing dynamic maneuvers under biplane fluoroscopy and comparing it to the contralateral side as an internal control showed that subpectoral tenodesis had little effect on glenohumeral kinematics [122]. A systematic review on comparative studies between supra- and subpectoral biceps tenodesis showed 98% good to excellent results for both methods, therefore revealing no identifiable difference [123]. Even though the subpectoral approach has very low complication rates [124], surgeons should consider the close neurovascular structures: to avoid contact with any neurovascular

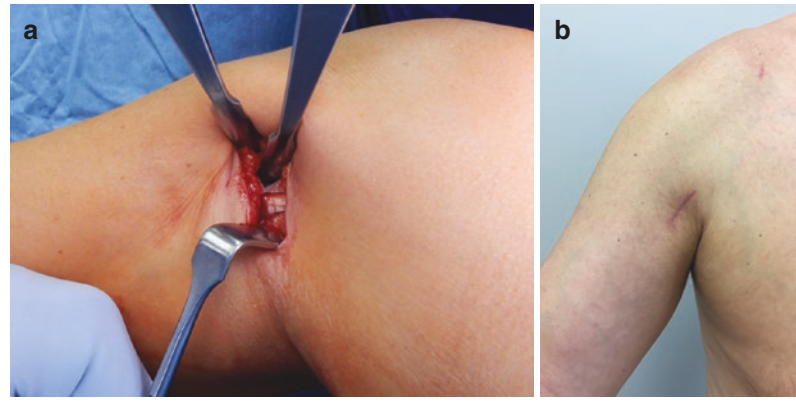

Fig. 10.6 Right shoulder: subpectoral LHBT tenodesis. (**a**) The tendon is clearly visible on the inferior border of the pectoralis major tendon. (**b**) No cosmetic impairment 3-month postoperative after subpectoral biceps tenodesis

structure, the arm should be positioned in external rotation to increase the distance to the musculocutaneous and radial nerve as well as the deep brachial artery [97].

Tenodesis site, fixation technique, and restauration of anatomic length-tension should be emphasized. While undertensioning may cause fatigue pain, muscle cramping, and cosmetic deformity, overtensioning may result in higher pullout forces at the tenodesis site and therefore theoretically increase the risk of failure. To reproduce normal length-tension, we perform tenotomy after an in situ subpectoral tenodesis.

References

1. Cooper DE, et al. Anatomy, histology, and vascularity of the glenoid labrum. An anatomical study. J Bone Joint Surg Am. 1992;74(1):46–52.
2. Ide J, Maeda S, Takagi K. Normal variations of the glenohumeral ligament complex: an anatomic study for arthroscopic Bankart repair. Arthroscopy. 2004;20(2):164–8.
3. Ilahi OA, Labbe MR, Cosculluela P. Variants of the anterosuperior glenoid labrum and associated pathology. Arthroscopy. 2002;18(8):882–6.
4. Pfahler M, et al. Age-related changes of the glenoid labrum in normal shoulders. J Shoulder Elb Surg. 2003;12(1):40–52.
5. Williams MM, Snyder SJ, Buford D Jr. The Buford complex--the "cord-like" middle glenohumeral ligament and absent anterosuperior labrum complex: a normal anatomic capsulolabral variant. Arthroscopy. 1994;10(3):241–7.
6. Williams MM, Snyder SJ. A sublabral foramen must not be confused with a Bankart lesion. Arthroscopy. 1994;10(5):586.
7. Taylor SA, O'Brien SJ. Clinically relevant anatomy and biomechanics of the proximal biceps. Clin Sports Med. 2016;35(1):1–18.
8. Vangsness CT Jr, et al. The origin of the long head of the biceps from the scapula and glenoid labrum. An anatomical study of 100 shoulders. J Bone Joint Surg Br. 1994;76(6):951–4.
9. Pal GP, Bhatt RH, Patel VS. Relationship between the tendon of the long head of biceps brachii and the glenoidal labrum in humans. Anat Rec. 1991;229(2):278–80.
10. Kim KC, Rhee KJ, Shin HD. A long head of the biceps tendon confluent with the intra-articular rotator cuff: arthroscopic and MR arthrographic findings. Arch Orthop Trauma Surg. 2009;129(3):311–4.
11. Kim KC, et al. Biceps long head tendon revisited: a case report of split tendon arising from single origin. Arch Orthop Trauma Surg. 2008;128(5):495–8.
12. Warner JJ, Paletta GA, Warren RF. Accessory head of the biceps brachii. Case report demonstrating clinical relevance. Clin Orthop Relat Res. 1992;280:179–81.
13. Gheno R, et al. Accessory head of biceps brachii muscle: anatomy, histology, and MRI in cadavers. AJR Am J Roentgenol. 2010;194(1):W80–3.
14. Nakatani T, Tanaka S, Mizukami S. Bilateral four-headed biceps brachii muscles: the median nerve and brachial artery passing through a tunnel formed by a muscle slip from the accessory head. Clin Anat. 1998;11(3):209–12.
15. Dierickx C, et al. Variations of the intra-articular portion of the long head of the biceps tendon: a classification of embryologically explained variations. J Shoulder Elb Surg. 2009;18(4):556–65.
16. Richards DP, Schwartz M. Anomalous intraarticular origin of the long head of biceps brachii. Clin J Sport Med. 2003;13(2):122–4.
17. Hyman JL, Warren RF. Extra-articular origin of biceps brachii. Arthroscopy. 2001;17(7):E29.
18. Andrews JR, Carson WG Jr, McLeod WD. Glenoid labrum tears related to the long head of the biceps. Am J Sports Med. 1985;13(5):337–41.

19. Snyder SJ, et al. SLAP lesions of the shoulder. Arthroscopy. 1990;6(4):274–9.
20. Morgan CD, et al. Type II SLAP lesions: three subtypes and their relationships to superior instability and rotator cuff tears. Arthroscopy. 1998;14(6):553–65.
21. Maffet MW, Gartsman GM, Moseley B. Superior labrum-biceps tendon complex lesions of the shoulder. Am J Sports Med. 1995;23(1):93–8.
22. Nord K, Ryu RKN. Further refinement of SLAP classification. In: AANA Annual Meeting. Orlando, FL; 2004.
23. Snyder SJ, Banas MP, Karzel RP. An analysis of 140 injuries to the superior glenoid labrum. J Shoulder Elb Surg. 1995;4(4):243–8.
24. Kim TK, et al. Clinical features of the different types of SLAP lesions: an analysis of one hundred and thirty-nine cases. J Bone Joint Surg Am. 2003;85-A(1):66–71.
25. Rhee YG, Lee DH, Lim CT. Unstable isolated SLAP lesion: clinical presentation and outcome of arthroscopic fixation. Arthroscopy. 2005;21(9):1099.
26. Sayde WM, et al. Return to play after Type II superior labral anterior-posterior lesion repairs in athletes: a systematic review. Clin Orthop Relat Res. 2012;470(6):1595–600.
27. Fedoriw WW, et al. Return to play after treatment of superior labral tears in professional baseball players. Am J Sports Med. 2014;42(5):1155–60.
28. Alpert JM, et al. The effect of age on the outcomes of arthroscopic repair of type II superior labral anterior and posterior lesions. Am J Sports Med. 2010;38(11):2299–303.
29. Brockmeier SF, et al. Outcomes after arthroscopic repair of type-II SLAP lesions. J Bone Joint Surg Am. 2009;91(7):1595–603.
30. Coleman SH, et al. Arthroscopic repair of type II superior labral anterior posterior lesions with and without acromioplasty: a clinical analysis of 50 patients. Am J Sports Med. 2007;35(5):749–53.
31. Kim SH, et al. Results of arthroscopic treatment of superior labral lesions. J Bone Joint Surg Am. 2002;84-A(6):981–5.
32. Ide J, Maeda S, Takagi K. Sports activity after arthroscopic superior labral repair using suture anchors in overhead-throwing athletes. Am J Sports Med. 2005;33(4):507–14.
33. Boileau P, et al. Arthroscopic treatment of isolated type II SLAP lesions: biceps tenodesis as an alternative to reinsertion. Am J Sports Med. 2009;37(5):929–36.
34. Ek ET, et al. Surgical treatment of isolated type II superior labrum anterior-posterior (SLAP) lesions: repair versus biceps tenodesis. J Shoulder Elb Surg. 2014;23(7):1059–65.
35. Denard PJ, et al. Arthroscopic biceps tenodesis compared with repair of isolated type II SLAP lesions in patients older than 35 years. Orthopedics. 2014;37(3):e292–7.
36. Gottschalk MB, et al. Subpectoral biceps tenodesis for the treatment of type II and IV superior labral anterior and posterior lesions. Am J Sports Med. 2014;42(9):2128–35.
37. Gupta AK, et al. Subpectoral biceps tenodesis for bicipital tendonitis with SLAP tear. Orthopedics. 2015;38(1):e48–53.
38. Brockmeyer M, et al. SLAP lesions: a treatment algorithm. Knee Surg Sports Traumatol Arthrosc. 2016;24(2):447–55.
39. Claessens H, Snoeck H. Tendinitis of the long head of the biceps brachii. Acta Orthop Belg. 1972;58(1):124–8.
40. Alpantaki K, et al. Sympathetic and sensory neural elements in the tendon of the long head of the biceps. J Bone Joint Surg Am. 2005;87(7):1580–3.
41. Boileau P, Ahrens PM, Hatzidakis AM. Entrapment of the long head of the biceps tendon: the hourglass biceps--a cause of pain and locking of the shoulder. J Shoulder Elb Surg. 2004;13(3):249–57.
42. Taylor SA, et al. Diagnostic glenohumeral arthroscopy fails to fully evaluate the biceps-labral complex. Arthroscopy. 2015;31(2):215–24.
43. Hussain WM, et al. The longitudinal anatomy of the long head of the biceps tendon and implications on tenodesis. Knee Surg Sports Traumatol Arthrosc. 2015;23(5):1518–23.
44. Denard PJ, et al. Anatomy of the biceps tendon: implications for restoring physiological length-tension relation during biceps tenodesis with interference screw fixation. Arthroscopy. 2012;28(10):1352–8.
45. Lafrance R, et al. Relevant anatomic landmarks and measurements for biceps tenodesis. Am J Sports Med. 2013;41(6):1395–9.
46. Cheng NM, et al. The arterial supply of the long head of biceps tendon: anatomical study with implications for tendon rupture. Clin Anat. 2010;23(6):683–92.
47. Koltz I, Tillmann B, Lullmann-Rauch R. The structure and vascularization of the biceps brachii tendon. Ann Anat. 1994;176:75–80.
48. Pfahler M, Branner S, Refior HJ. The role of the bicipital groove in tendopathy of the long biceps tendon. J Shoulder Elb Surg. 1999;8(5):419–24.
49. Depalma AF, Callery GE. Bicipital tenosynovitis. Clin Orthop. 1954;3:69–85.
50. Hitchcock HH, Bechtol CO. Painful shoulder; observations on the role of the tendon of the long head of the biceps brachii in its causation. J Bone Joint Surg Am. 1948;30A(2):263–73.
51. Refior HJ, Sowa D. Long tendon of the biceps brachii: sites of predilection for degenerative lesions. J Shoulder Elb Surg. 1995;4(6):436–40.
52. Gilmer BB, et al. Arthroscopic versus open comparison of long head of biceps tendon visualization and pathology in patients requiring tenodesis. Arthroscopy. 2015;31(1):29–34.
53. Festa A, et al. Visualization of the extra-articular portion of the long head of the biceps tendon during intra-articular shoulder arthroscopy. Arthroscopy. 2014;30(11):1413–7.

54. Moon SC, Cho NS, Rhee YG. Analysis of "hidden lesions" of the extra-articular biceps after subpectoral biceps tenodesis: the subpectoral portion as the optimal tenodesis site. Am J Sports Med. 2015;43(1):63–8.
55. Braun S, et al. Biomechanical evaluation of shear force vectors leading to injury of the biceps reflection pulley: a biplane fluoroscopy study on cadaveric shoulders. Am J Sports Med. 2010;38(5):1015–24.
56. Braun S, et al. Lesions of the biceps pulley. Am J Sports Med. 2011;39(4):790–5.
57. Bennett WF. Visualization of the anatomy of the rotator interval and bicipital sheath. Arthroscopy. 2001;17(1):107–11.
58. Bennett WF. Arthroscopic repair of anterosuperior (supraspinatus/subscapularis) rotator cuff tears: a prospective cohort with 2- to 4-year follow-up. Classification of biceps subluxation/instability. Arthroscopy. 2003;19(1):21–33.
59. Walch G, et al. Tears of the supraspinatus tendon associated with "hidden" lesions of the rotator interval. J Shoulder Elb Surg. 1994;3(6):353–60.
60. McClelland D, Bell SN, O'Leary S. Relocation of a dislocated long head of biceps tendon is no better than biceps tenodesis. Acta Orthop Belg. 2009;75(5):595–8.
61. Bennett WF. Arthroscopic bicipital sheath repair: two-year follow-up with pulley lesions. Arthroscopy. 2004;20(9):964–73.
62. Taylor SA, et al. The anatomy and histology of the bicipital tunnel of the shoulder. J Shoulder Elb Surg. 2015;24(4):511–9.
63. Cone RO, et al. The bicipital groove: radiographic, anatomic, and pathologic study. AJR Am J Roentgenol. 1983;141(4):781–8.
64. Hsu AR, et al. Biceps tenotomy versus tenodesis: a review of clinical outcomes and biomechanical results. J Shoulder Elb Surg. 2011;20(2):326–32.
65. Slenker NR, et al. Biceps tenotomy versus tenodesis: clinical outcomes. Arthroscopy. 2012;28(4):576–82.
66. Frost A, Zafar MS, Maffulli N. Tenotomy versus tenodesis in the management of pathologic lesions of the tendon of the long head of the biceps brachii. Am J Sports Med. 2009;37(4):828–33.
67. Taylor SA, et al. The clinical impact of bicipital tunnel decompression during long head of the biceps tendon surgery: a systematic review and meta-analysis. Arthroscopy. 2016;32(6):1155–64.
68. Romeo AA, Mazzocca AD, Tauro JC. Arthroscopic biceps tenodesis. Arthroscopy. 2004;20(2):206–13.
69. Sekiya JK, Elkousy HA, Rodosky MW. Arthroscopic biceps tenodesis using the percutaneous intra-articular transtendon technique. Arthroscopy. 2003;19(10):1137–41.
70. Provencher MT, LeClere LE, Romeo AA. Subpectoral biceps tenodesis. Sports Med Arthrosc. 2008;16(3):170–6.
71. Lo IK, Burkhart SS. Arthroscopic biceps tenodesis using a bioabsorbable interference screw. Arthroscopy. 2004;20(1):85–95.
72. Kim SH, Yoo JC. Arthroscopic biceps tenodesis using interference screw: end-tunnel technique. Arthroscopy. 2005;21(11):1405.
73. Gartsman GM, Hammerman SM. Arthroscopic biceps tenodesis: operative technique. Arthroscopy. 2000;16(5):550–2.
74. Checchia SL, et al. Biceps tenodesis associated with arthroscopic repair of rotator cuff tears. J Shoulder Elb Surg. 2005;14(2):138–44.
75. Boileau P, et al. Arthroscopic biceps tenodesis: a new technique using bioabsorbable interference screw fixation. Arthroscopy. 2002;18(9):1002–12.
76. Gill TJ, et al. Results of biceps tenotomy for treatment of pathology of the long head of the biceps brachii. J Shoulder Elb Surg. 2001;10(3):247–9.
77. Kelly AM, et al. Arthroscopic release of the long head of the biceps tendon: functional outcome and clinical results. Am J Sports Med. 2005;33(2):208–13.
78. 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 Elb Surg. 2005;14(3):238–46.
79. Berlemann U, Bayley I. Tenodesis of the long head of biceps brachii in the painful shoulder: improving results in the long term. J Shoulder Elb Surg. 1995;4(6):429–35.
80. Wittstein JR, et al. Isokinetic strength, endurance, and subjective outcomes after biceps tenotomy versus tenodesis: a postoperative study. Am J Sports Med. 2011;39(4):857–65.
81. Shank JR, et al. A comparison of forearm supination and elbow flexion strength in patients with long head of the biceps tenotomy or tenodesis. Arthroscopy. 2011;27(1):9–16.
82. Drakos MC, et al. Arthroscopic transfer of the long head of the biceps tendon: functional outcome and clinical results. Arthroscopy. 2008;24(2):217–23.
83. Nord KD, Smith GB, Mauck BM. Arthroscopic biceps tenodesis using suture anchors through the subclavian portal. Arthroscopy. 2005;21(2):248–52.
84. Post M, Benca P. Primary tendinitis of the long head of the biceps. Clin Orthop Relat Res. 1989;246:117–25.
85. Busconi BB, DeAngelis N, Guerrero PE. The proximal biceps tendon: tricks and pearls. Sports Med Arthrosc. 2008;16(3):187–94.
86. Edwards TB, et al. Repair of tears of the subscapularis. Surgical technique. J Bone Joint Surg Am. 2006;88(Suppl 1 Pt 1):1–10.
87. Brady PC, et al. Arthroscopic proximal biceps tenodesis at the articular margin: evaluation of outcomes, complications, and revision rate. Arthroscopy. 2015;31(3):470–6.
88. Wolf RS, Zheng N, Weichel D. Long head biceps tenotomy versus tenodesis: a cadaveric biomechanical analysis. Arthroscopy. 2005;21(2):182–5.
89. Franceschi F, et al. To detach the long head of the biceps tendon after tenodesis or not: outcome analysis at the 4-year follow-up of two different techniques. Int Orthop. 2007;31(4):537–45.

90. Randelli P, et al. Current practice in shoulder pathology: results of a web-based survey among a community of 1,084 orthopedic surgeons. Knee Surg Sports Traumatol Arthrosc. 2012;20(5):803–15.
91. Duff SJ, Campbell PT. Patient acceptance of long head of biceps brachii tenotomy. J Shoulder Elb Surg. 2012;21(1):61–5.
92. Galdi B, et al. Patients have strong preferences and perceptions for biceps tenotomy versus tenodesis. Arthroscopy. 2016;32(12):2444–50.
93. Patzer T, et al. Suprapectoral or subpectoral position for biceps tenodesis: biomechanical comparison of four different techniques in both positions. J Shoulder Elb Surg. 2012;21(1):116–25.
94. Froimson AI, Oh I. Keyhole tenodesis of biceps origin at the shoulder. Clin Orthop Relat Res. 1975;112:245–9.
95. Elkousy HA, et al. Arthroscopic biceps tenodesis using the percutaneous, intra-articular transtendon technique: preliminary results. Orthopedics. 2005;28(11):1316–9.
96. Dines D, Warren RF, Inglis AE. Surgical treatment of lesions of the long head of the biceps. Clin Orthop Relat Res. 1982;164:165–71.
97. Dickens JF, et al. Subpectoral biceps tenodesis: an anatomic study and evaluation of at-risk structures. Am J Sports Med. 2012;40(10):2337–41.
98. David TS, Schildhorn JC. Arthroscopic suprapectoral tenodesis of the long head biceps: reproducing an anatomic length-tension relationship. Arthrosc Tech. 2012;1(1):e127–32.
99. Castagna A, et al. Arthroscopic biceps tendon tenodesis: the anchorage technical note. Knee Surg Sports Traumatol Arthrosc. 2006;14(6):581–5.
100. Boileau P, Neyton L. Arthroscopic tenodesis for lesions of the long head of the biceps. Oper Orthop Traumatol. 2005;17(6):601–23.
101. Becker DA, Cofield RH. Tenodesis of the long head of the biceps brachii for chronic bicipital tendinitis. Long-term results. J Bone Joint Surg Am. 1989;71(3):376–81.
102. Ahmad CS, ElAttrache NS. Arthroscopic biceps tenodesis. Orthop Clin North Am. 2003;34(4):499–506.
103. Werner BC, et al. Biceps tenodesis is a viable option for salvage of failed SLAP repair. J Shoulder Elb Surg. 2014;23(8):e179–84.
104. McCormick F, et al. The efficacy of biceps tenodesis in the treatment of failed superior labral anterior posterior repairs. Am J Sports Med. 2014;42(4):820–5.
105. Werner BC, Brockmeier SF, Gwathmey FW. Trends in long head biceps tenodesis. Am J Sports Med. 2015;43(3):570–8.
106. Lopez-Vidriero E, et al. Biomechanical evaluation of 2 arthroscopic biceps tenodeses: double-anchor versus percutaneous intra-articular transtendon (PITT) techniques. Am J Sports Med. 2010;38(1):146–52.
107. Franceschi F, et al. Soft tissue tenodesis of the long head of the biceps tendon associated to the Roman Bridge repair. BMC Musculoskelet Disord. 2008;9:78.
108. Mazzocca AD, et al. The biomechanical evaluation of four fixation techniques for proximal biceps tenodesis. Arthroscopy. 2005;21(11):1296–306.
109. Verma NN, Drakos M, O'Brien SJ. Arthroscopic transfer of the long head biceps to the conjoint tendon. Arthroscopy. 2005;21(6):764.
110. Koh KH, et al. Treatment of biceps tendon lesions in the setting of rotator cuff tears: prospective cohort study of tenotomy versus tenodesis. Am J Sports Med. 2010;38(8):1584–90.
111. Mazzocca AD, et al. Subpectoral biceps tenodesis with interference screw fixation. Arthroscopy. 2005;21(7):896.
112. Arora AS, Singh A, Koonce RC. Biomechanical evaluation of a unicortical button versus interference screw for subpectoral biceps tenodesis. Arthroscopy. 2013;29(4):638–44.
113. Cameron JS, Skofronick JG, Grant RM. Physics of the body, Medical physics, vol. 2. King of Prussia: Rittenhouse Bk; 1999.
114. Ozalay M, et al. Mechanical strength of four different biceps tenodesis techniques. Arthroscopy. 2005;21(8):992–8.
115. Richards DP, Burkhart SS. A biomechanical analysis of two biceps tenodesis fixation techniques. Arthroscopy. 2005;21(7):861–6.
116. Buchholz A, et al. Biomechanical comparison of intramedullary cortical button fixation and interference screw technique for subpectoral biceps tenodesis. Arthroscopy. 2013;29(5):845–53.
117. Werner BC, et al. Increased incidence of postoperative stiffness after arthroscopic compared with open biceps tenodesis. Arthroscopy. 2014;30(9):1075–84.
118. Werner BC, et al. Arthroscopic suprapectoral and open subpectoral biceps tenodesis: a comparison of restoration of length-tension and mechanical strength between techniques. Arthroscopy. 2015;31(4):620–7.
119. Werner BC, et al. Arthroscopic suprapectoral and open subpectoral biceps tenodesis: a comparison of minimum 2-year clinical outcomes. Am J Sports Med. 2014;42(11):2583–90.
120. Green JM, et al. All-arthroscopic suprapectoral versus open subpectoral tenodesis of the long head of the biceps brachii without the use of interference screws. Arthroscopy. 2017;33(1):19–25.
121. Gombera MM, et al. All-arthroscopic suprapectoral versus open subpectoral tenodesis of the long head of the biceps brachii. Am J Sports Med. 2015;43(5):1077–83.
122. Giphart JE, et al. The long head of the biceps tendon has minimal effect on in vivo glenohumeral kinematics: a biplane fluoroscopy study. Am J Sports Med. 2012;40(1):202–12.
123. Abraham VT, Tan BH, Kumar VP. Systematic review of biceps tenodesis: arthroscopic versus open. Arthroscopy. 2016;32(2):365–71.
124. Nho SJ, et al. Complications associated with subpectoral biceps tenodesis: low rates of incidence following surgery. J Shoulder Elb Surg. 2010;19(5):764–8.

Acromioclavicular Joint Instability: When and How to Operate

11

Theresa Diermeier, Felix Dyrna, Andreas B. Imhoff, and Knut Beitzel

11.1 Introduction

Acromioclavicular (AC) joint separations are often caused by a direct impact to the lateral aspect of the shoulder and are common sport-related injuries. AC joint injuries are classified according to Rockwood grades I–VI [1]. This subdivision encompasses mild sprains to the AC capsule with preservation of the coracoclavicular (CC) ligaments to complete tearing of both the AC capsule complex and CC ligaments. The classification is based on a standard AP X-ray and does not include any dynamic aspects. In general Rockwood type I and type II injuries are treated nonsurgical without any major residuals [2]. Type III lesions are still discussed controversial, but surgical treatment is recommended for high-functional patients or overhead-throwing athletes. In case of severe ACJ instability (type IV or higher according to Rockwood classification), surgical treatment is proposed to avoid painful chronic instability and resultant scapular dyskinesia.

To the present date, the optimal utility and timing of surgery in the treatment of acromioclavicular joint injuries continues to be unidentified with more than 150 surgical reconstruction techniques [3].

Regardless of the technique, the main goal remains to obtain a pain-free shoulder with unrestricted range of motion and full strength. To achieve this, a set of crucial elements should be addressed during operative treatment of AC injuries:

1. Correction of the superior displacement and anterior-to-posterior translation of the clavicle through anatomic reduction of the acromioclavicular joint
2. Maintenance of AC joint stability during acute healing by supplementation of the CC ligament reconstruction with a synthetic material or rigid implant or biological reconstruction of the CC ligaments in a chronic case
3. Meticulous deltotrapezial fascia closure
4. Reconstruction of the AC capsular ligaments to address severe horizontal stabilization of the AC joint

The wide range of possible surgical procedures is also accompanied by a variable rate of technical errors and clinical failures. Such failures have already been reported in recent literature with high complication rates up to 80% [4]. In order to treat failed reconstructions, the reason for failure has to be clearly identified. Patient's history needs to be analyzed if a new adequate trauma or other factors like technical errors or

T. Diermeier · F. Dyrna · A. B. Imhoff · K. Beitzel (✉)
Department of Orthopaedic Sports Medicine, Technical University, Munich, Germany
e-mail: theresa.diermeier@tum.de; dyrna@tum.de; imhoff@tum.de

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_11

insufficient healing response were more likely predisposing factors for treatment failure. Knowledge about previous surgical procedure, tunnel placement, size of tunnels, previous fixation, and additional injuries is elementary to enter a revision case setting. Established on this knowledge, four clinical relevant situations are commonly found when revising a failed AC joint:

1. Failure of treatment due to wrong patient selection
2. Failure of treatment due to insufficient fixation with or without new trauma
3. Failure due to persisting instability (mainly in the horizontal plane) and/or excessive bone loss of the lateral clavicle
4. Failure combined with fractures of osseous structures

In current literature, only low evidence exists for the right treatment in case of failed ACJ reconstruction. Therefore, the following paragraphs cover possible failure reasons and adequate revision strategies as a specific guideline focusing on clinically relevant settings.

11.2 Failure of Treatment Due to Wrong Patient Selection

There is a consensus that in low-grade injuries (type I–II) a conservative treatment is favored, whereas high-grade injuries (types IV, V, and VI) should be treated operatively. Anyhow, a lack of evidence exists in regard to the treatment of acute Rockwood grade III injuries. The current approach leans toward initial non-operative management with clinicians opting for conservative treatment with good-to-excellent results in pain score, lower complication, and return to activity rates. However, up to 20% of patients with acute grade III AC injuries treated non-operatively show persistent pain and residual instability and are at risk for altered scapulohumeral kinematics.

In these patients, chronic AC pain and instability after non-operative treatment for grade III AC injuries results in a delayed surgical intervention. To better filter out those patients at risk, a more detailed addendum to the Rockwood classification has been requested in regard to grade III injuries [5].

Therefore, the grade III injuries had been further subclassified as grade IIIA and grade IIIB based on the presence of horizontal instability of the AC joint. While grade IIIA injuries demonstrate horizontal stability, grade IIIB injuries demonstrate overriding of the clavicle on a cross-body adduction radiograph or increased AP translation under clinical examination. This refinement should help surgeons in their decision making with a more patient-centered treatment algorithm for grade III injuries including this dynamic aspect of stability. Patients with a significant horizontal instability (IIIB or IV) should be adequately treated with an acute surgical repair by addressing the increased AP translation, for example, with a high strain suture brace and an additional CC ligament augmentation. This way a delayed surgical intervention with the need for biologic augmentation can be avoided.

For correct classification of the injury, the surgeon should always complete a detailed physical examination with a focus on instability testing including the horizontal AC stability. In a painful acute setting, a local infiltration can be helpful to ensure a sufficient exam. In addition, the scapula position and tracking should be evaluated to rule out pre-existing scapula dyskinesia. A thorough evaluation for additional lesions within the glenohumeral joint should be performed to exclude additional injuries, as high comorbidities with the long head of the biceps exist. Clinical examination should be supplemented by multiple imaging modalities. For radiologic evaluation a bilateral Zanca view and an additional Alexander projection are favored to allow an objective measure of the vertical and horizontal instability. According to the classification and additional lesions, the most appropriated therapy should be performed.

If a patient presents with a grade III lesion within 14 days of injury, treatment may additionally be guided by individual patient consid-

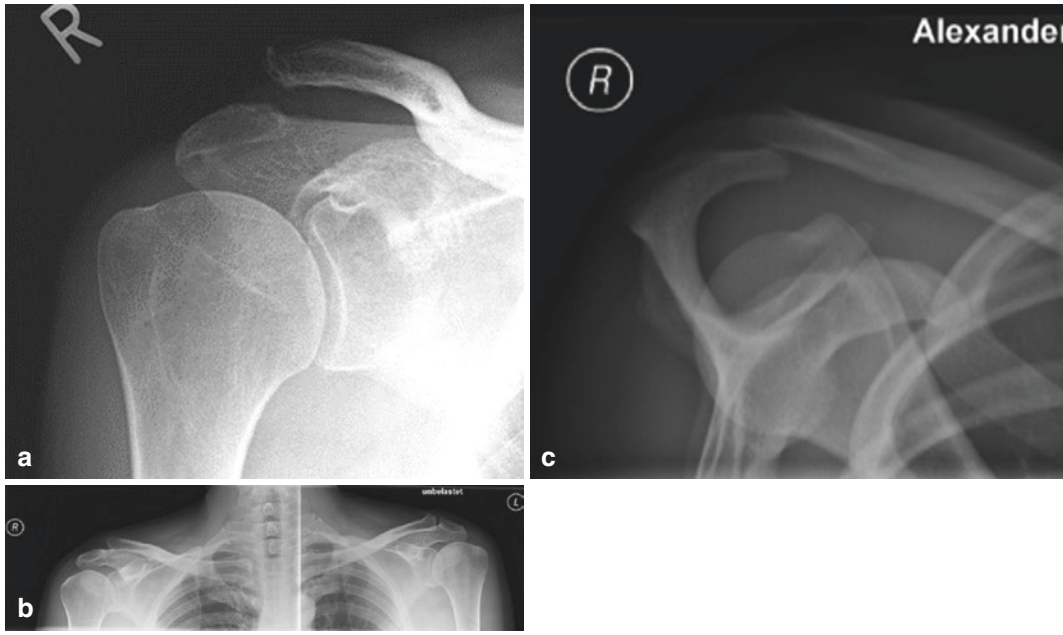

Fig. 11.1 X-Ray in three planes ((**a**) AP view; (**b**) bilateral Zanca view; (**c**) Alexander view) of a patient presenting with Rockwood type IV instability. Note the different judgment of vertical instability seen in the AP view compared to the bilateral Zanca view. Clear horizontal instability is demonstrated in the Alexander view

erations. This includes such factors as hand dominance, job or sport requirements, in-season status, risk of re-injury, and patient motivation to return to pre-injury activity. High-demand patients, such as laborers and athletes (i.e., throwing, contact sports), should thus be considered for acute surgical intervention. In low-demand patients, nonsurgical management focused on regaining scapular strength remains a feasible option. If initial nonsurgical treatment is favored in a type III situation, a stepwise treatment is recommended with a close follow-up to monitor the success of the procedure. Patients could undergo treatment with 3–4 weeks of nonsurgical management. A defined second evaluation for type III lesions should be performed at 3–6 weeks after the injury. Those demonstrating significant pain reduction and improved range of motion should continue with rehabilitation for another 3–6 weeks, while some of these conservatively treated patients will have persistent pain and loss of function interfering with return to previous activity or sports performance. These patients should be thoroughly reevaluated clinically and radiographically. If the patient presents with continued abnormal scapular movement and radiographic images in the Alexander view show an overriding of the clavicle over the acromion, subsequent surgical stabilization is needed. In those cases a biologic augmentation with a soft tissue graft (e.g., free tendon graft) will still allow eventual return to sport or work (Fig. 11.1).

11.3 Failure Due to Insufficient Fixation with or Without New Trauma

Depending on the surgical procedure, loss of reduction is described in 12–50% [6]. The risk of recurrent instability with insufficient fixation after surgical ACJ stabilizations is elevated in chronic high-grade (Rockwood type V) injuries [7]. If the initial stabilizing procedure failed due to insufficient primary fixation, insufficient biologic healing, or following an

adequate new trauma, an anatomical stabilization of the CC and AC ligaments using a free tendon graft is suggested. Different technical procedures are available, and it can be performed with or without addition of a non-resorbable suture utilized as internal bracing (e.g., fiber tape and cortical button) to increase primary stability [8, 9].

It has been shown biomechanically that tunnels can be re-utilized for revision with adequate primary stability [10]. Therefore, bone tunnels and fixation points of the initial procedure should be rechecked on a CT scan for position and size. Tunnel enlargement should be detected to confirm the correct tunnels position and assess proper tunnel diameter to allow new fixation. Costic et al. [11] showed that the use of a free soft tissue hamstring graft results in similar load to failure values and elongations compared to the native CC ligaments while being a less stiff construct. To further stabilize the construct, a fixation with tenodesis screws and an additional cerclage could be added [12]. This can be combined with a suture button device to increase the primary stability. In addition, biomechanical testing of suture button devices demonstrated superior biomechanical stability in all directions when compared to techniques like modifications of the Weaver-Dunn technique [13]. Besides graft choice and fixation technique, positioning is crucial to restore native anatomy and furthermore prevent complications. Geaney et al. [14] showed higher BMD values at anatomic CC ligament insertion resulting in higher fixation strength.

11.4 Failure Due to Persisting Instability (Mainly on the Horizontal Plane) and/or Excessive Bone Loss at the Lateral Clavicle

Failure due to persisting instability is mainly due to horizontal instability. Klimkiewicz et al. [15] clearly showed that the posterior and superior aspects of the AC capsule contribute significantly to horizontal and rotational stability. Other authors demonstrated [16, 17] approximately native horizontal and improved rotational stability with reconstructing the AC capsule. In cases of additional horizontal instability (Rockwood type IIIB–VI), surgical procedures aiming at anatomic reconstruction should be preferred; likewise it has been shown in recent biomechanical and clinical studies [18]. According to these results, current literature focuses more and more on incorporating the AC capsule into the repair. As consequence over time, a trend was seen for a combined stabilization of the AC capsule and the CC ligaments.

In case of persisting horizontal instability after AC joint stabilization, its clinical impact has to be evaluated very carefully. Previous studies have shown that persisting slight horizontal instability has no effect on the clinical outcomes [19]. However, if the horizontal instability is responsible of persisting pain or scapula dysfunction, surgical treatment should be considered. An anatomic technique using a free tendon graft to reconstruct the CC as well as the AC ligaments should be performed in these cases. In addition, the deltotrapezial fascia needs to be thoroughly adapted meticulously closed to regain its contribution to stability. Multiple techniques to achieve this goal have been reported. In our hands, an arthroscopically assisted technique or an open procedure can be chosen. No matter which technique is chosen, it is important to clearly evaluate the AC joint intraoperatively, reconstruct the AC ligaments biologically, and pay attention to a thorough closure of the fascia. To reconstruct the AC ligaments and capsule, multiple techniques can be used. Grafts can be sutured to and incorporated into the repaired capsule and fascia. For stronger fixation, bone tunnels can be placed through the lateral clavicle and the acromion (Fig. 11.2).

11.5 Failure Combined with Fractures of Osseous Structures

Loss of reduction and fractures were reported to be the most relevant mechanism of failure that require revision surgery [20]. The risk of fracture was highly correlated with the use of bone tunnels

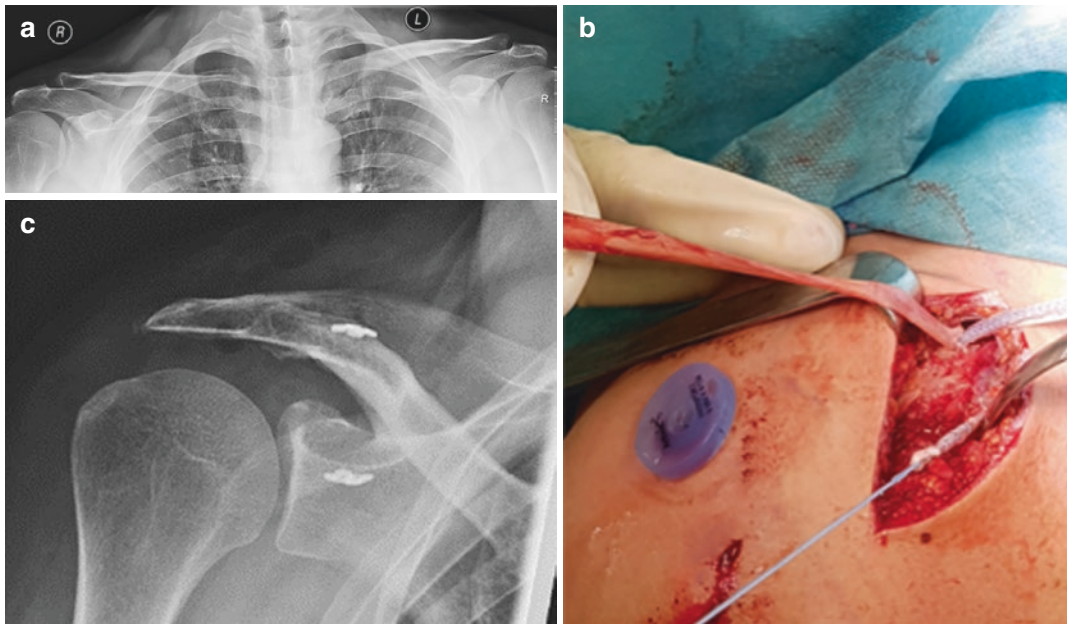

Fig. 11.2 (**a**) Bilateral Zanca view of a patient with persisting type IV instability and severe pain after failed treatment with hook plate for acute AC instability and additional resection of the lateral clavicle due to persisting pain. (**b**) Intraoperative picture of the patient treated with anatomic reconstruction of the CC and AC ligaments with gracilis tendon and internal bracing by fiber tapes and dog bones. (**c**) Postoperative X-ray

and in these circumstances to the size and position of the tunnels [7]. Technical errors and a steep learning curve were also identified to increase the risk for such failures [4]. This was especially the case of tunnels wider than 8 mm. Spiegl et al. [21] demonstrated that the size of clavicular drill holes can significantly weaken the bone and cause increased risk of fractures. Similar observations have been shown for coracoid drill holes as reported by Martetschlager et al. [22]. This data suggest a minimization of tunnel diameter and limitation of the number of bone tunnels in the area at risk for fracture. Technical tips (e.g., use of additional anterolateral portal for visualization of coracoid tunnel) can also help to optimize the tunnel placement and further reduce the risk of such technical problems [23].

In case of clavicular fractures, conservative treatment may represent an option, when the fracture does not lead to significant dislocation of the clavicle or secondary loss of reduction of the reconstructed AC joint. If the reduction is lost or significant dislocation recurs, surgical options may include an open reduction and internal fixation (ORIF) of the clavicle in combination with a CC ligaments reconstruction either with a suture pulley system or with addition of a biologic tendon graft (Fig. 11.3).

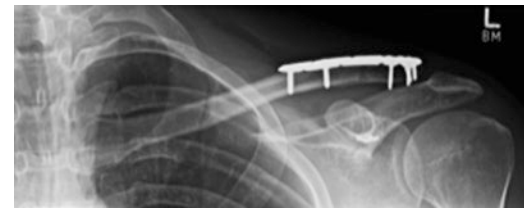

Fig. 11.3 Postoperative X-ray of a patient treated with clavicle plate combined with CC reconstruction with dog bone/fiber tape construct following fracture of the lateral clavicle

Coracoid drill holes have been shown to affect the stability and increase the risk of fracture. This has been shown to correlate with the number of holes as well as their size [22]. Most coracoid fractures can be handled conservatively. If the fracture results in a secondary instability, a thorough analysis of the fracture is needed. If the tunnel was placed too far anterior or a blowout occurred

medially or laterally due to wrong tunnel placement, a new tunnel cannot be placed in the correct anatomic position close to the base of the coracoid. If this is not intended, a graft can be guided around the coracoid. Usually the fracture at the side of the coracoid does not need direct fixation.

References

1. Williams G, Nguyen V, Rockwood C. Classification and radiographic analysis of acromioclavicular dislocations. Appl Radiol. 1989;18:29–34.
2. Dias JJ, et al. The conservative treatment of acromioclavicular dislocation. Review after five years. J Bone Joint Surg Br. 1987;69(5):719–22.
3. Beitzel K, et al. Current concepts in the treatment of acromioclavicular joint dislocations. Arthroscopy. 2013;29(2):387–97.
4. Milewski MD, et al. Complications related to anatomic reconstruction of the coracoclavicular ligaments. Am J Sports Med. 2012;40(7):1628–34.
5. Beitzel K, et al. ISAKOS upper extremity committee consensus statement on the need for diversification of the Rockwood classification for acromioclavicular joint injuries. Arthroscopy. 2014;30(2):271–8.
6. Kibler WB, Sciascia A. Current concepts: scapular dyskinesis. Br J Sports Med. 2010;44(5):300–5.
7. Martetschlager F, et al. Complications after anatomic fixation and reconstruction of the coracoclavicular ligaments. Am J Sports Med. 2013;41(12):2896–903.
8. Tauber M, et al. Arthroscopic stabilization of chronic acromioclavicular joint dislocations: triple-versus single-bundle reconstruction. Am J Sports Med. 2016;44(2):482–9.
9. Mazzocca AD, et al. A biomechanical evaluation of an anatomical coracoclavicular ligament reconstruction. Am J Sports Med. 2006;34(2):236–46.
10. Voss A, et al. A biomechanical analysis of different clavicular tunnel diameters in anatomic acromioclavicular ligament reconstruction. Arthroscopy. 2016;32(8):1551–7.
11. Costic RS, et al. Biomechanical rationale for development of anatomical reconstructions of coracoclavicular ligaments after complete acromioclavicular joint dislocations. Am J Sports Med. 2004;32(8):1929–36.
12. Erak S, et al. Acromioclavicular reconstructions with hamstring tendon grafts: a comparative biomechanical study. J Shoulder Elb Surg. 2008;17(5):772–8.
13. Beitzel K, et al. Biomechanical comparison of arthroscopic repairs for acromioclavicular joint instability suture button systems without biological augmentation. Am J Sports Med. 2011;39(10):2218–25.
14. Geaney LE, et al. Graft fixation is highest with anatomic tunnel positioning in acromioclavicular reconstruction. Arthroscopy. 2013;29(3):434–9.
15. Klimkiewicz JJ, et al. The acromioclavicular capsule as a restraint to posterior translation of the clavicle: a biomechanical analysis. J Shoulder Elb Surg. 1999;8(2):119–24.
16. Beitzel K, et al. Rotational and translational stability of different methods for direct acromioclavicular ligament repair in anatomic acromioclavicular joint reconstruction. Am J Sports Med. 2014;42(9):2141–8.
17. Grantham C, et al. A biomechanical assessment of a novel double endobutton technique versus a coracoid cerclage sling for acromioclavicular and coracoclavicular injuries. Knee Surg Sports Traumatol Arthrosc. 2016;24(6):1918–24.
18. Saier T, et al. Value of additional acromioclavicular cerclage for horizontal stability in complete acromioclavicular separation: a biomechanical study. Knee Surg Sports Traumatol Arthrosc. 2015;23(5):1498–505.
19. Scheibel M, et al. Arthroscopically assisted stabilization of acute high-grade acromioclavicular joint separations. Am J Sports Med. 2011;39(7):1507–16.
20. Woodmass JM, et al. Complications following arthroscopic fixation of acromioclavicular separations: a systematic review of the literature. Open Access J Sports Med. 2015;6:97.
21. Spiegl UJ, et al. Biomechanical consequences of coracoclavicular reconstruction techniques on clavicle strength. Am J Sports Med. 2014;42(7):1724–30.
22. Martetschläger F, et al. Effect of coracoid drilling for acromioclavicular joint reconstruction techniques on coracoid fracture risk: a biomechanical study. Arthroscopy. 2016;32(6):982–7.
23. Imhoff AB, Braun S, Beitzel K. Comments on complications after arthroscopic coracoclavicular reconstruction using a single adjustable loop length suspensory fixation device. Arthroscopy. 2015;31(6):1031–3.

Failed Slap Repair: Case Example

12

Antonio Cartucho

12.1 Case Presentation

A 33-year-old male suffered an indirect trauma to his right shoulder, described as forced forward traction of the limb. There was no sensation of dislocation, but the days after trauma he had pain even at rest. After this initial acute phase, the patient referred mechanical symptoms worsened by abduction and by strenuous activities. No relief of symptoms was obtained with rehabilitation.

At the clinical examination, range of motion (ROM) consisted of 150° of forward flection, 70° of abduction, and 60° of external rotation and internal rotation to the T12 vertebra. He had no signs of subacromial impingement, major joint instability, nor rotator cuff weakness. Only the O'Brien test and the biceps load test were positive [1, 2]. Magnetic resonance imaging (MRI) showed a small change of humeral head morphology, although not compatible with Hill-Sachs lesion (Fig. 12.1). A SLAP lesion was suspected, but could not be confirmed due to poor image quality. The patient underwent conservative treatment with rehabilitation and subacromial corticoid injection. After 1.5 months, there was only mild residual pain, no night pain, and normal mobility and strength. The patient was discharged and returned to his normal activities of daily living.

Two months later, the patient returned to the office due to a new trauma to his right shoulder. He suffered from pain with overhead movements and complained of limb paresthesia. The patient rated his pain as eight in the visual analogue score (VAS) and 50% in the subjective shoulder value (SSV). American Shoulder and Elbow Surgeons (ASES) score was 23 and Constant score was 42. ROM was 160° in forward flection and 90° in external rotation and internal rotation to the T4 vertebra. He had positive O'Brien and biceps load tests. Second MRI (Fig. 12.2) showed a type II SLAP lesion and no rotator cuff tears.

The patient was operated on and an arthroscopic SLAP repair was performed using two suture anchors, one anterior and one posterior to the biceps anchor (Fig. 12.3). After postoperative immobilization for 3 weeks, a standard rehabilitation protocol was prescribed. Four months after surgery, the patient presented with a pain VAS of 1, SSV of 90%, ASES score of 92, and Constant score of 90. ROM was 160° in forward flexion and 70° in external rotation and internal rotation to the T12 vertebra. Shoulder strength was normal but O'Brien and biceps load tests were still positive. There were no signs of subacromial impingement.

Nine months after surgery, the patient returned to office due to pain on his right shoulder. There was no history of new trauma. No night pain was

A. Cartucho
Shoulder and Elbow Department, Hospital Cuf Descobertas-José de Mello Saúde, Lisboa, Portugal

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_12

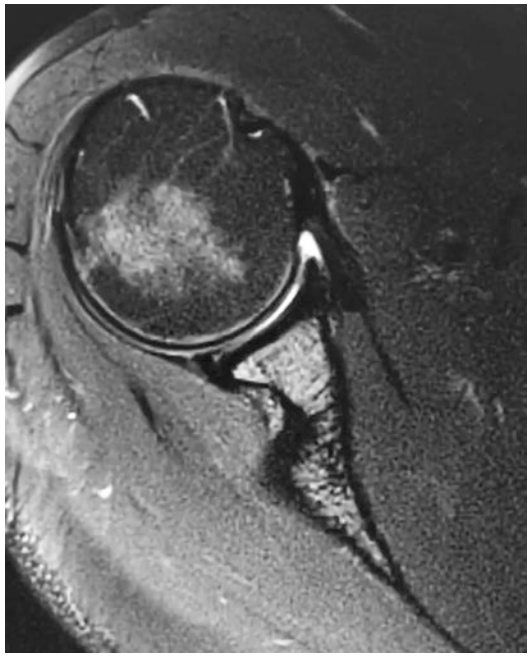

Fig. 12.1 Preoperative MRI

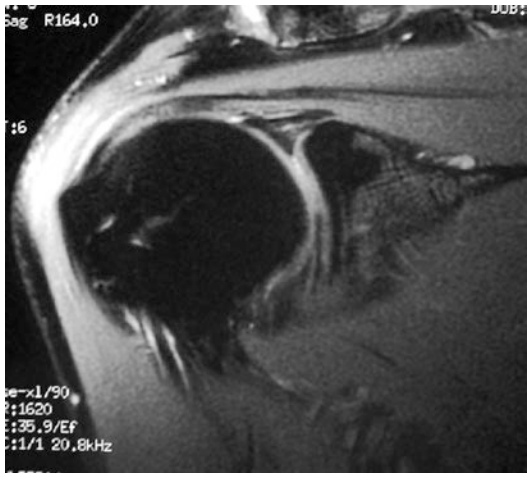

Fig. 12.2 Type 2 SLAP lesion

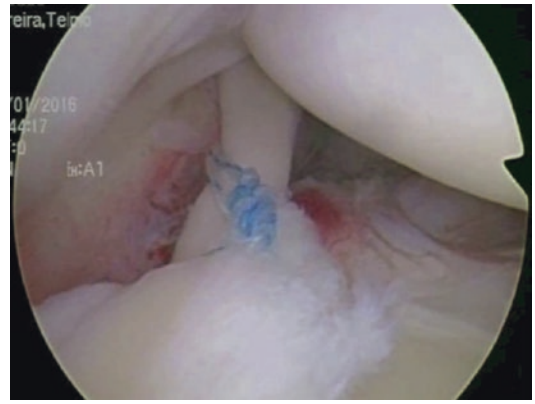

Fig. 12.3 Anterior and posterior anchor

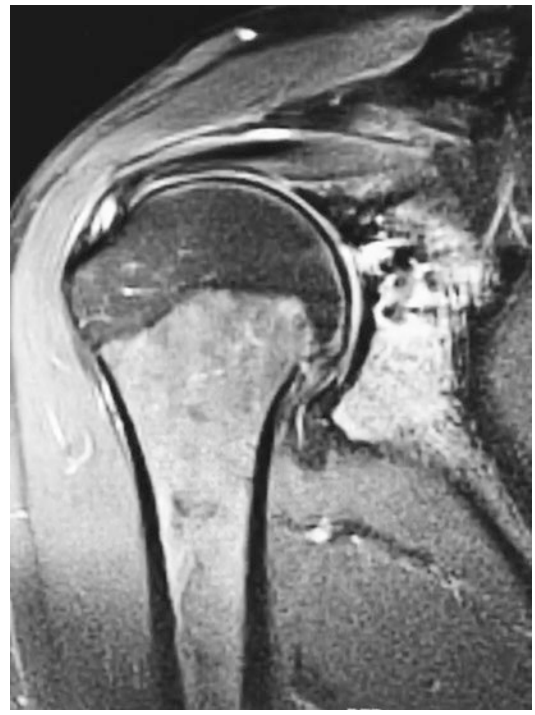

Fig. 12.4 Postoperative MRI

present and ROM was similar to that reported at discharge. The O'Brien and biceps load tests were positive. There was also an audible click in the shoulder in abduction and external rotation. A shoulder MRI was performed and an interstitial supraspinatus tear was present, no synovitis of the long head of the biceps (LHB) tendon and no signs of retear of the biceps anchor (Fig. 12.4).

Physiotherapy was prescribed, but due to a progressive worsening of pain, loss of strength, and positive impingement signs, a shoulder arthroscopy was scheduled.

Surgery was performed in beach chair position using a standard posterior portal for intra-articular inspection. The superior labrum showed degenerative changes and non-healing of the SLAP lesion, with prominent and loose sutures leading to superior labrum and biceps anchor instability (Fig. 12.5). After a complete diagnostic arthroscopy, the LHB tendon was tenotomized as close as possible to the labrum insertion, and the rotator interval was debrided in order to facilitate LHB tendon identification from the subacromial space. A debridement of the superior labrum was done with concomitant removal of the loose sutures. The arthroscope was passed into the subacromial space using the same posterior portal, and a lateral portal was established in line with the posterior aspect of the acromioclavicular joint and 2 cm lateral to the acromion. With the arthroscope in this portal, an anterior portal was created in line with the LHB tendon. The bicipital groove was prepared until bleeding subchondral bone was obtained (Fig. 12.6), and a suprapectoral LHB tenodesis with an interference screw was performed (Fig. 12.7). The supraspinatus showed a partial-thickness articular-side tear with no indication for repair.

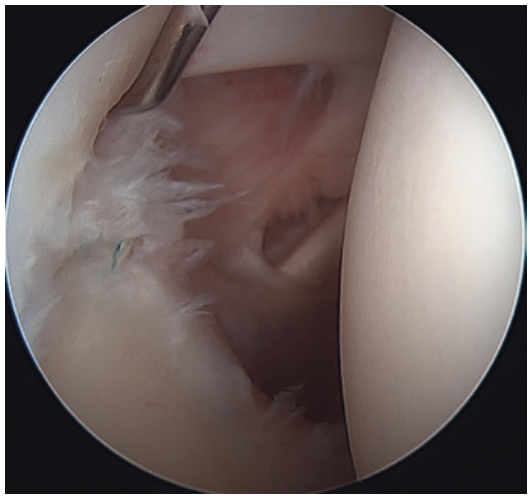

Fig. 12.5 Degenerative changes and prominent sutures

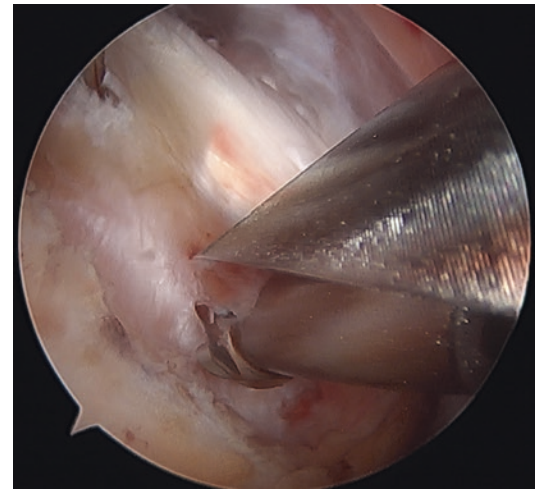

Fig. 12.6 Bicipital groove preparation

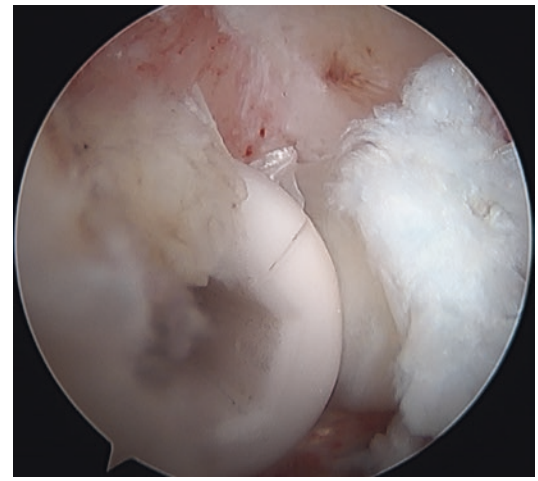

Fig. 12.7 Biceps tenodesis with interference screw

12.2 Discussion

There were two traumatic episodes separated in time. History of an indirect trauma to the shoulder in a young patient that after the acute period was relatively well-tolerated, and mostly interfered with overhead or repetitive activities, should raise the suspicion of a SLAP lesion. The presence of positive O'Brien and biceps load tests strengthens this hypothesis, and then a MR

arthrography (MRA) should be considered to confirm the diagnosis. Unfortunately, only standard MRI was available and diagnostic value of the exam was rather limited. Nevertheless, the patient became asymptomatic after a period of rehabilitation. This finding does not rule out the lesion, but proves that it is possible to balance the shoulder complex even in presence of an injury.

The second episode probably aggravated the lesion, and a new MRI confirmed the diagnosis, which led to surgery. Depending on the energy of this second traumatic event, a new set of rehabilitation sessions should be considered. Indeed, there is a consistent risk of overestimation and overtreatment of these lesions, as demonstrated by the substantial increment (150%) of SLAP repairs between the years 2004 and 2009 [3]. Weber et al. [4] also analyzed 4975 cases of SLAP repairs and found a three-times higher than expected, with an average patients' age of 37 years. Surgeons must be aware of these facts and carefully select their patients.

Failure of SLAP repair may be determined by missed treatment of concomitant pathology, development of new lesions, technique-related aspects, and non-healing (biologic failure). Development of postoperative stiffness and implant-related complications are also causes of failure. Frank et al. [5] also found an association between ASES score less than 50 and age greater than 40 years; alcohol/tobacco use; coexisting diabetes; pain in the bicipital groove on examination; positive O'Brien, Speed's, and/or Yergason's tests; and high levels of lifting required at work. Other authors [6] identified other causes that led to persistent pain such as chondral injuries, stiffness, and instrumentation problems. Byram et al. [7] associated chondral wear of the humeral head with overtensioning of the biceps, which is distinct from implant-related chondral injury. Katz et al. [8] found that stiffness was the primary cause of symptoms. Although adequate ROM was achieved with nonsurgical measures, patients continued to have symptoms that required additional surgery. Although my patient reached almost full ROM, persistent pain led to revision surgery.

Taking into consideration the structural lesions found at the second arthroscopy, the causes of failure might be due to non-healing of the superior labrum reconstruction with consequent instability of the biceps anchor. Nevertheless, the supraspinatus tear may also be responsible for symptoms due to a dynamic impingement secondary to supraspinatus weakness. There is a possibility that this lesion was missed at the first procedure, but most likely it is secondary to shoulder imbalance due to labrum non-healing. This hypothesis would explain the difference in the supraspinatus features at the two MRI exams.

Options for patients with a failed SLAP repair are rather limited. A new repair of the labrum was described by Park et al. [9]. At average 50 months of follow-up, the mean ASES score was 72.5, mean return to work was 57.8% of previous level, and mean return to sports was 42.2% of previous level. In order to optimize results, biceps tenodesis has been suggested. A suprapectoral tenodesis may be performed using suture anchors or interference screws, as in the present case. Gupta et al. [10] presented the results of biceps tenodesis and reported mean ASES score slightly exceeding that by Park et al. [9] in revision SLAP repairs. Suprapectoral tenodesis may be performed open or arthroscopic, while subpectoral fixation requires an open approach. Mazzocca et al. [11] found no biomechanical difference between four tenodesis techniques (open subpectoral with bone tunnel, arthroscopic with interference screw, open subpectoral with interference screw, and arthroscopic with suture anchor). Brady et al. [12] in a clinical study concluded that arthroscopic biceps tenodesis performed at the articular margin results in a low surgical revision rate, low rate of residual pain, and significant improvement in objective shoulder outcome scores. Nho et al. [13], in their study on patients who underwent open subpectoral biceps tenodesis, found a complication rate of only 2%. In a recent systematic review, Abraham et al. [14] concluded that both open and arthroscopic biceps tenodesis provide satisfactory outcomes in most patients.

Indeed, suprapectoral biceps tenodesis may offer an acceptable, if not better, alternative to primary SLAP repair, as described by several authors that reported excellent subjective results

with patients' return to a presurgical level of activity and sports participation [10, 15–17]. Similarly, good-to-excellent results were reported with primary treatment of SLAP tears by open subpectoral tenodesis [10, 17].

Conclusion

There has been a constant increase in diagnosis and treatment of SLAP lesions, and surgeons must be aware of the possibility of overestimation and overtreatment. Some clinical studies reported a revision rate for SLAP repair as high as 47%. Biceps tenodesis may be a good alternative for primary treatment of SLAP lesions. There is no evidence about superiority of arthroscopic suprapectoral tenodesis over open subpectoral tenodesis.

References

1. O'Brien SJ, Pagnani MJ, Felay S, et al. The active compression test: a new and effective test for diagnosing labrum tears and acromioclavicular joint abnormality. Am J Sports Med. 1998;26:610–3.
2. Seoung-H K, Kwon-Ick H, Kye-Young H. Biceps load test: a clinical test for SLAP lesions in shoulders with recurrent anterior dislocations. Am J Sports Med. 1999;27(3):300–3.
3. Zhang AL, Kreulen C, Ngo SS, Hame SL, Wang JC, Gamradt SC. Demographic trends in arthroscopic SLAP repair in the United States. Am J Sports Med. 2012;40:1144–7. https://doi.org/10.1177/0363546512436944.
4. Weber SC, Payvandi S, Martin DF, Harrast JJ. SLAP lesions of the shoulder: incidence rates, complications, and outcomes as reported by ABOS part II candidates (SS-19). Arthroscopy. 2010;26(Suppl 6):e9–e10.
5. Frank R, Nho S, McGil K, Grumet R, Cole B, Verma N, Romeo A. Retrospective analysis of arthroscopic superior labrum anterior to posterior repair: prognostic factors associated with failure. Adv Orthop. 2013;2013:125960. https://doi.org/10.1155/2013/125960.
6. Weber SC. Surgical management of the failed SLAP repair. Sports Med Arthrosc. 2010;18(3):162–6.
7. Byram IR, Dunn WR, Kuhn JE. Humeral head abrasion: an association with failedsupe rior labrum anterior posterior repairs. J Shoulder Elb Surg. 2011;20(1):92–7.
8. Katz LM, Hsu S, Miller SL, et al. Poor outcomes after SLAP repair: descriptive analysis and prognosis. Arthroscopy. 2009;25(8):849–55.
9. Park S, Glousman R. Outcomes of revision arthroscopic type II superior labral anterior posterior repairs. Am J Sports Med. 2011;39:1290–4.
10. Gupta AK, Chalmers PN, Klosterman EL, et al. Subpectoral biceps tenodesis for bicipital tendonitis with SLAP tear. Orthopedics. 2015;38:48–53.
11. Mazzocca AD, Bicos J, Santangelo S, Romeo AA, Arciero RA. The biomechanical evaluation of four fixation techniques for proximal biceps tenodesis. Arthroscopy. 2005;21:1296–306.
12. Brady PC, Narbona P, Adams CR, et al. Arthroscopic proximal biceps tenodesis at the articular margin: evaluation of outcomes, complications, and revision rate. Arthroscopy. 2015;31:470–6.
13. Nho SJ, Reiff SN, Verma NN, Slabaugh MA, Mazzocca AD, Romeo AA. Complications associated with subpectoral biceps tenodesis: low rates of incidence following surgery. J Shoulder Elb Surg. 2010;19:764–8.
14. Abraham V, Tan B, Kuma V. Systematic review systematic review of biceps tenodesis: arthroscopic versus open. Arthroscopy. 2016;32(2):365–71.
15. Boileau P, Parratte S, Chuinard C, Roussanne Y, Shia D, Bicknell R. Arthroscopic treatment of isolated type II SLAP lesions: biceps tenodesis as an alternative to reinsertion. Am J Sports Med. 2009;37:929–36.
16. Denard PJ, Lädermann A, Parsley BK, Burkhart SS. Arthroscopic biceps tenodesis compared with repair of isolated type II SLAP lesions in patients older than 35 years. Orthopedics. 2014;37:292–7.
17. Gottschalk MB, Karas SG, Ghattas TN, Burdette R. Subpectoral biceps tenodesis for the treatment of type II and IV superior labral anterior and posterior lesions. Am J Sports Med. 2014;42:2128–35.

Failed AC Joint Treatment: Case Examples

13

Francesco Franceschi and Marco Spoliti

13.1 Introduction

The most important lessons during our surgical activities result from the evaluation of our failures. In acromioclavicular (AC) joint disease, several factors should be evaluated before deciding on the best treatment. Patient's age, type of injury, and timing of injury (acute or chronic) are all factors that influence the type of treatment and then treatment outcome. Nowadays various surgical techniques are used, and in recent years, the role of arthroscopy has taken a leading role with the aid of devices that allow us, if used correctly, to achieve a stable fixation.

13.2 Case Presentation 1

This first case concerns a 32-year-old man, engaged in sports activity (horse riding), who reported a grade III AC joint dislocation to his left shoulder according to the Rockwood's classification [1], following a direct trauma (Fig. 13.1).

After an initial attempt to conservative treatment with bracing and corticosteroid injection, the patient underwent an arthroscopic stabilization using the AC TightRope system (Arthrex, Naples FL, USA) 1 month after the injury (Fig. 13.2).

Six months after surgery, the patient reported recurrence of cosmetic deformity with onset of pain in the absence of new trauma. The radiographic examination showed mobilization of the clavicular button (Fig. 13.3). After a careful evaluation of the immediate postoperative X-rays, it can be seen that the clavicular and coracoid tunnels were not coaxial (Fig. 13.4).

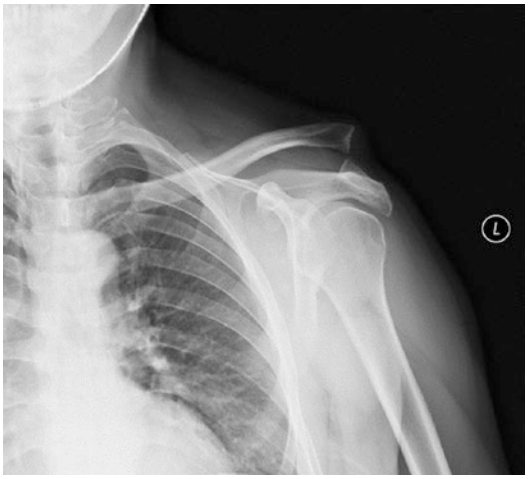

Fig. 13.1 Left shoulder, type III AC joint dislocation

F. Franceschi (✉)
Department of Orthopaedic and Trauma Surgery, Campus Bio-Medico University, Rome, Italy
e-mail: F.Franceschi@unicampus.it

M. Spoliti
Department of Orthopaedics and Traumatology, San Camillo-Forlanini Hospital, Rome, Italy

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_13

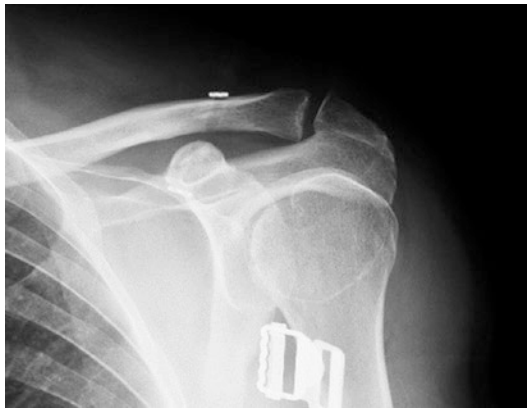

Fig. 13.2 Left shoulder, arthroscopic stabilization using the AC TightRope system (Arthrex)

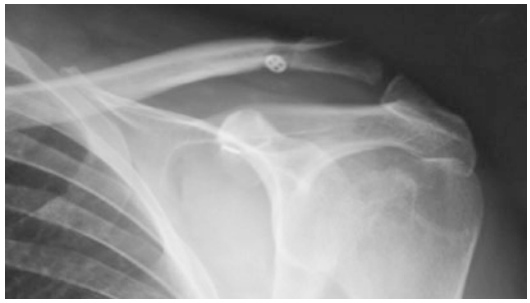

Fig. 13.3 Left shoulder, failure and migration of the clavicular button

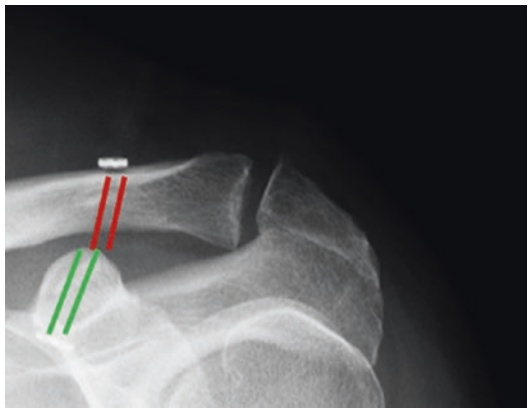

Fig. 13.4 Left shoulder, careful evaluation of postoperative X-rays showing that the clavicular and coracoid tunnels are not coaxial

13.3 Case Presentation 2

The second case concerns a 27-year-old female, sedentary, who reported a grade III AC joint dislocation to her right shoulder following an accidental fall. She arrived at our observation 3 months after the trauma complaining of pain and functional impairment (Fig. 13.5).

The patient underwent to arthroscopic stabilization using AC TightRope system (Arthrex) (Fig. 13.6).

Three months after surgery, the patient suffered from recurrence of pain, without cosmetic deformity (Fig. 13.7). Radiographic exams showed enlargement of the clavicular tunnel with slight migration of the clavicular button. In that case, due to the young age of the patient and the absence of cosmetic concern (Fig. 13.8), the

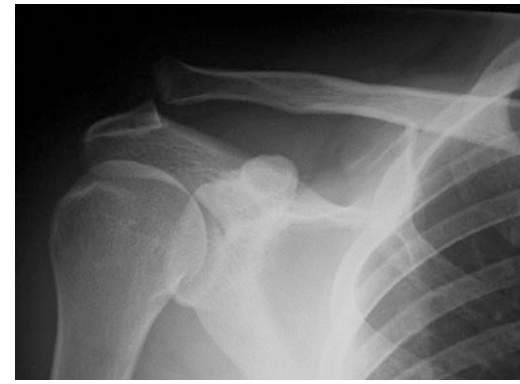

Fig. 13.5 Right shoulder, type III AC joint dislocation

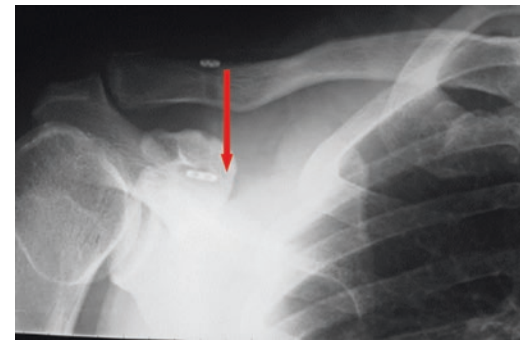

Fig. 13.6 Right shoulder, arthroscopic stabilization using the AC TightRope system (Arthrex)

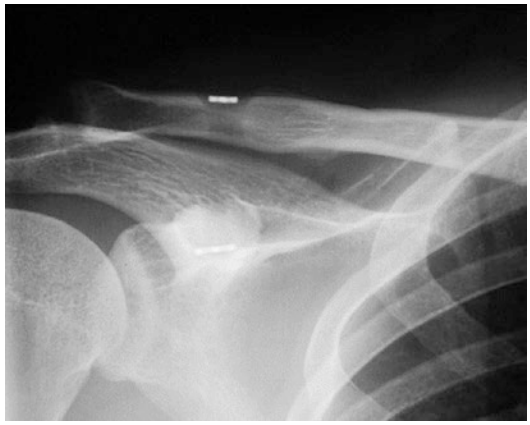

Fig. 13.7 Right shoulder, enlargement of the clavicular tunnel with slight migration of the clavicular button

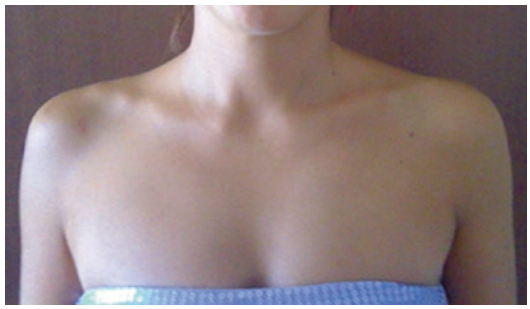

Fig. 13.8 Right shoulder, the patient does not show cosmetic disorders

treatment was conservative, consisting of a corticosteroid injection inside the AC joint with good results.

13.4 Case Presentation 3

The third case concerns a 30-year-old man, sportive, who reported a type IV AC joint dislocation following a sport trauma (ski). He arrived at our observation 5 months after the trauma suffering from pain, functional deficit, and severe cosmetic deformity. During the previous 5 months, the patient had been treated conservatively (injections, bracing, and anti-inflammatory drugs). Radiographic exams showed a type IV AC joint dislocation (Fig. 13.9).

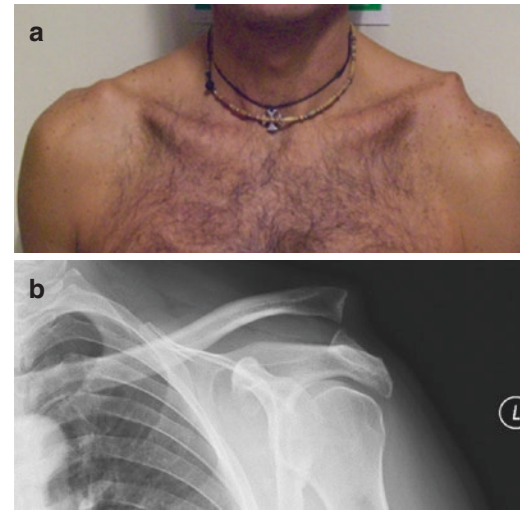

Fig. 13.9 (**a, b**) Left shoulder, type IV AC joint dislocation

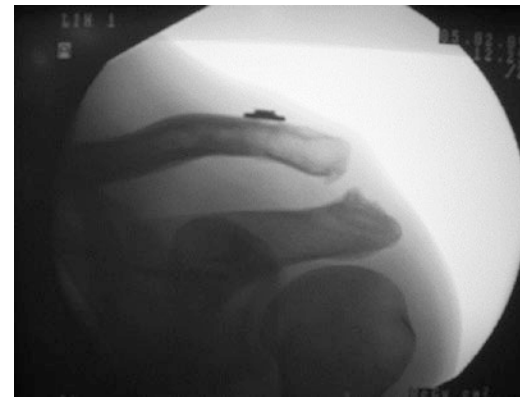

Fig. 13.10 Left shoulder, X-ray showed a recurrence of the dislocation

The patient underwent an arthroscopic stabilization using AC TightRope system (Arthrex). Three months after surgery, during sports activity, the patient had a sudden onset of pain with recurrence of cosmetic deformity (Fig. 13.10).

In this case, the treatment was to remove the previous means of fixations (Fig. 13.11), and subsequently, the GraftRope system (Arthrex) with a hamstring tendon allograft was used to reconstruct the coracoclavicular (CC) ligaments (Fig. 13.12).

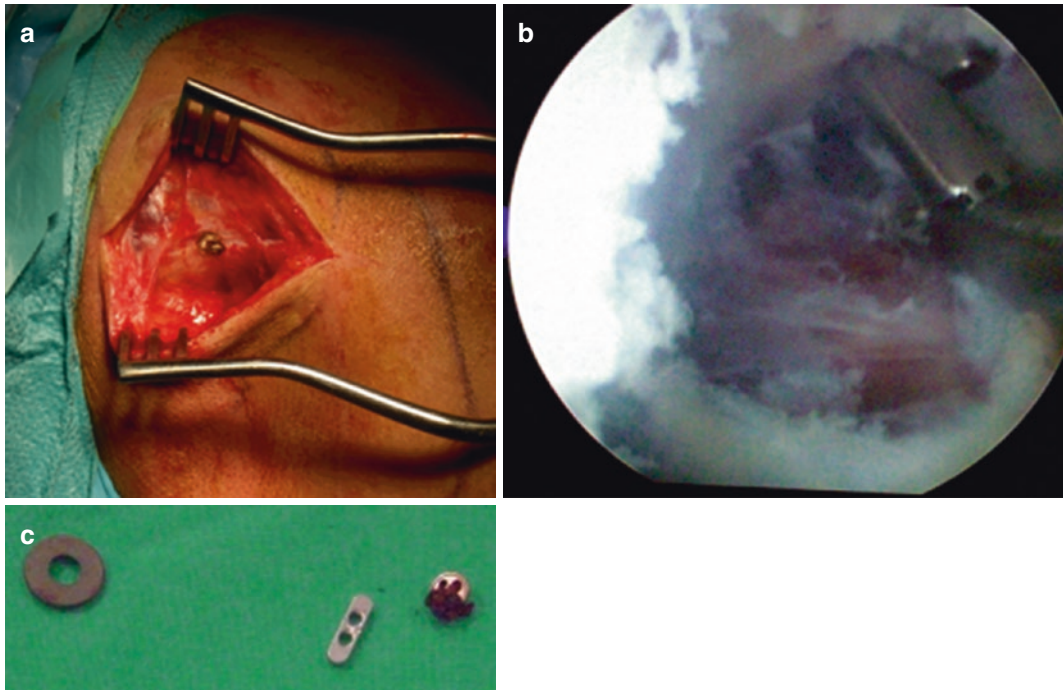

Fig. 13.11 (**a, b, c**) Left shoulder, removal of the fixation devices

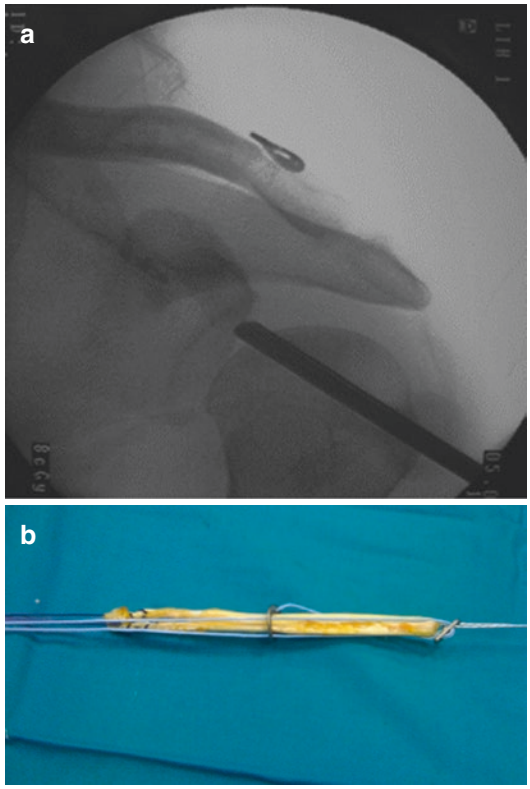

Fig. 13.12 (**a, b**) Left shoulder, revision surgery with GraftRope system (Arthrex)

13.5 Discussion

In the first case, failure was related to a technical error during surgery. Evidently, stabilization with Kirchner wire that precedes the final one with the TightRope was not stable enough or the Kirchner wire was removed before giving the final tension to the system. Indeed, the reduction obtained by positioning of the Kirchner wire must be the final one. An increase or a decrease in tension on the system can result in non-coaxial tunnel and cause the subsequent system failure.

From the last two cases, we learned that in patients with chronic dislocations, the treatment of choice should be from the beginning the use of a system with biologic graft because in the absence of healing of the CC ligaments all the tension is released on the fixation device, thus causing the failure or breakdown of the latter.

Reference

1. Rockwood CA Jr, Williams GR, Young D. Disorders of the acromioclavicular joint. In: Rockwood Jr CA, Matsen F, editors. The shoulder. Philadelphia: Saunders; 1998. p. 483–553.

Part III

Failed Anatomical Shoulder Arthroplasty

14 Anatomical Shoulder Arthroplasty: How It Works

Thierry Joudet, Christophe Charousset, and The Shoulder Friends Group

14.1 Introduction

The most common causes of failure of anatomical shoulder arthroplasty, regardless of design and surgical technique, are infection, allergy, instability, stiffness, rotator cuff tears, peri-prosthetic fractures, and glenoid erosions [1]. Indeed, all of these complications, except infection, are related to the implant. Moreover, the balance of the peri-prosthetic soft tissues is influenced by the prosthetic bulk, while the bone stock around the implant depends on its design and method of implantation [2]. As we would like to propose to our patients the best option in terms of implant survival and risk of revision, the future will be focused on improvement of implants design and surgical techniques. Whenever we try to envisage new solutions to improve outcomes, we must analyze the background in order to understand errors made in the past and to prevent them in the future.

T. Joudet (✉)
Centre JFK, Libourne, France
e-mail: thierry@joudet.fr

C. Charousset
IOAPC, Paris, France

The Shoulder Friends Group
Clinique du Cambresis, Cambrai, France

14.2 Humeral Component

The most marked evolution in shoulder prosthesis design over the last 50 years concerns the humeral side. The history of the humeral prosthesis began in the 1950s with Neer, who designed a Vitallium humeral stem intended to treat humeral fractures [3, 4]. This prosthesis was successfully adapted to fracture cases, but when treating cases of osteoarthritis (OA), the monobloc design used by Neer showed its limits, because its implantation did not respect the anatomy. In fact, each humeral stem size of the monobloc design corresponded to a fixed diameter and thickness of the humeral head, and for this reason the prosthesis was most often implanted in varus, too high and too bulky [5].

The concept of modularity between the humeral stem and the head was proposed in 1990 by Rockwood and Matsen [6]. These two eminent surgeons well understood the need to choose a stem size adapted to the humeral diaphysis and a humeral head size corresponding to the anatomy. However, this solution was not sufficient to perfectly reproduce the anatomy of the upper part of the humerus, as detailed in the studies of Boileau and Walch, who pointed out the concept of medial and posterior offset by showing that the axis of the humeral diaphysis is offset with respect to the center of rotation of the humeral head [7].

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_14

The third-generation prostheses appeared in the years 2000 and allowed perfect matching of the implant with the proximal humerus. The humeral stem approximately 12 cm in length could be fixed with or without cement, and the cephalic cup (humeral head) was adapted to the cut of the anatomical neck by respecting the medial offset and the posterior offset. However, these long humeral stems showed their limits. Favard et al. reported between 1% and 22% of humeral component loosening [8]. Problems already known in hip replacement surgery emerged, such as peri-prosthetic fractures and stress shielding [9]. The Australian register [10] showed almost 70% of bone resorption around the humeral stem for the SMR implant (Lima Corp., San Daniele del Friuli, Italy). In fact, the use of uncemented humeral stems with excessive distal prosthetic bulk leads to a diaphyseal blockage and stem fixation on its distal part. Hence, shear forces mainly pass through the most rigid distal region and the metaphyseal bone becomes less solicited. Still, a large difference in the number of revisions between uncemented stems (4.5%) and cemented stems (0.77%) has been reported [10]. Even if these third-generation anatomical prostheses represented the gold standard since the 2000s, some surgeons avoided using humeral stem and opted for a metaphyseal fixation [11].

Resurfacing was firstly promoted by Copeland [12] and was initially used as hemiarthroplasty (HA). This type of prosthesis intended simply to cover the worn humeral head; however, it was frequently too bulky and increased the tension effect on the soft tissues [13]. This effect was increased with the use of a glenoid implant. In addition, access to the glenoid was made difficult by the presence of the humeral head. Radiographic studies confirmed that resurfacing prostheses did not respect anatomy [14].

The so-called "stemless" and short-stem prosthesis (about 80 mm) humeral components appeared on the market as competing solutions and as compromise between resurfacing and long humeral stems, albeit they represent two completely different and opposite philosophies [15–17].

Stemless prostheses seek a purely metaphyseal fixation. They are intended as anatomic replacement and offer no possibility of modularity and conversion to an inverse prosthesis without removing the metaphyseal fixation (Fig. 14.1). The main advantage of this design is related to preservation of the metaphyseal bone stock with good bone fixation. Moreover, primary implant can be considered in case of surgical revision [18] (Fig. 14.2).

Contrasting to stemless prostheses, short-stem implants make modularity possible and allow for use with an anatomical configuration or conversion to a reverse shoulder arthroplasty (RSA) [19] (Fig. 14.3). In fact, this implant allows maintaining the humeral stem in case of revision. There are two different designs of short-stem prostheses. The first one has an "onlay" fixation of the humeral head, while the second one has an "inlay" configuration. This difference is very important when converting an anatomical

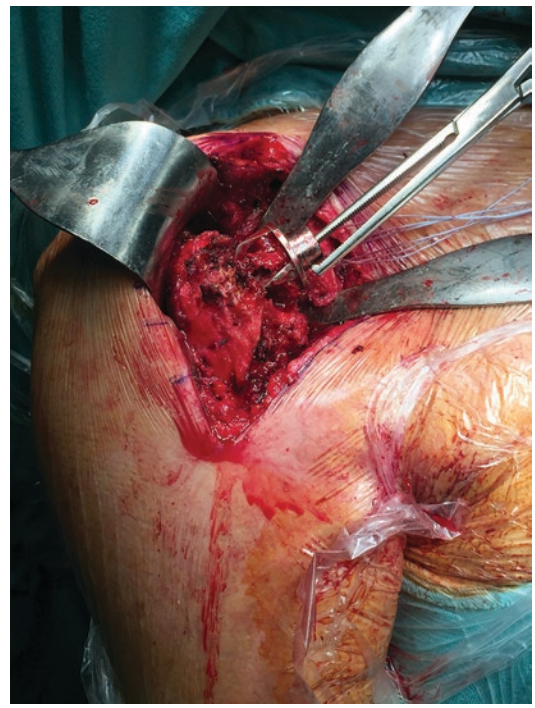

Fig. 14.1 Revision of a stemless anatomical arthroplasty. Conversion to an inverse prosthesis is impossible without removing the metaphyseal fixation

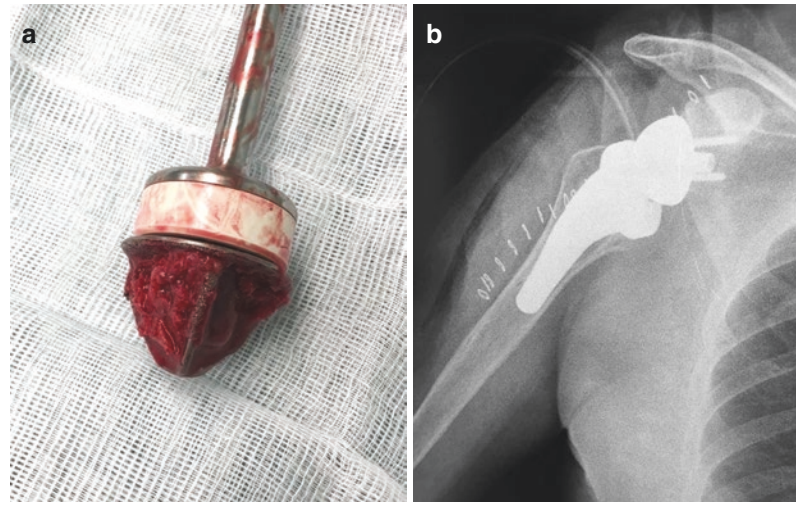

Fig. 14.2 Revision of a stemless anatomical arthroplasty. (**a**) The main advantage of this design is related to preservation of the metaphyseal bone stock. (**b**) Primary implant can be used in case of revision

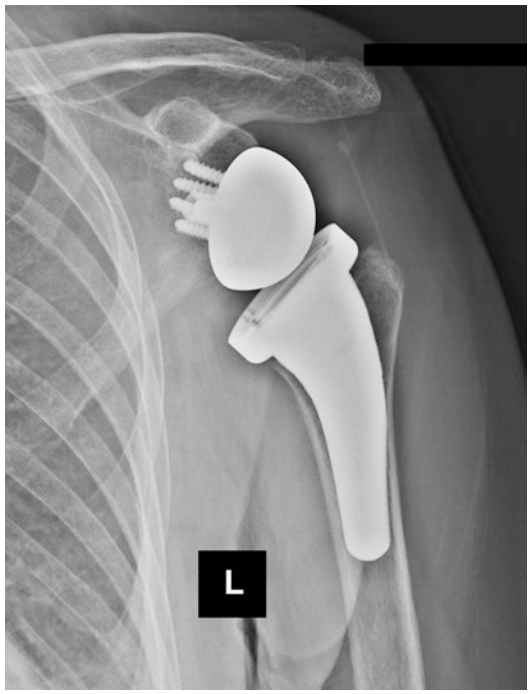

Fig. 14.3 Modular short-stem implants allow conversion from anatomical to a reverse shoulder arthroplasty without replacing the humeral stem

prosthesis to a RSA, as the metaphyseal part greatly increases the prosthetic bulk.

Although difficulty in conversion of a short-stem prostheses was noted by advocates of the stemless concept, finite element studies showed that it is difficult to expect a reliable metaphyseal fixation of a reverse stemless prosthesis. In fact, the lever arm is too large when the center of rotation of the joint is translated at the level of the glenoid. Nonetheless, Teissier et al. [20] evaluated the survival rate of the stemless TESS system (Zimmer-Biomet, Warsaw, IN, USA) and demonstrated an extremely limited number of disassembly.

14.3 Glenoid Component

Improvements in glenoid implant designs was less marked during the same period, even though the long-term survival of a total shoulder arthroplasty (TSA) depends mainly on its evolution. Several studies confirmed that TSA is preferable to HA because the functional results are better [21]. However, many authors reported contrasting midterm and long-term results due to the risk of erosion of the glenoid implant over time, thus suggesting alternative techniques, such as "rim and run" or biological glenoid resurfacing. Indeed, replacing the glenoid or not in anatomical shoulder arthroplasty is still controversial. Glenoid preservation exposes to the occurrence of glenoid pain by premature wear of cartilage and subchondral bone. Untreated glenoids show erosion that gradually increases with time, and the same phenomenon has been reported with biological resurfacing with fascia lata and with

the "ream and run" technique. However, the use of a glenoid implant does not avoid this progressive erosion [22]. In the case of a TSA, it is the softest component and the friction torque will wear it out. The particles (micro or macro) will then lead to a macrophage inflammatory reaction, which progressively leads to disassembly of the implant.

Technological advances in polyethylene (PE) design have evolved toward greater stability of the implant. The first-generation PE was oxidized, resulting in delamination and fast wear. The cross-linked PE became more stable over time. Finally, the addition of vitamin E made it possible to obtain a high stability of the material, thus reducing PE wear [23].

Two types of glenoid component design are present on the market: all-PE cemented implants and uncemented implants with metal back.

The uncemented metal-back glenoids are rigid and thick; they showed their limits according to the analysis of the results of different case series [24]. Indeed, the thickness of the metal back increases the prosthetic bulk, the implant is stiffer, the PE is thinner, and the risk of fast PE wear is higher. Moreover, the presence of fixing screws will be a pathway for the inflammatory granuloma which is a source of long-term failure [25]. For this reason, the all-PE cemented glenoid implant remains the gold standard.

Fixation of all-PE glenoids can be achieved by pegs, whose number is variable, or keel. The results of mechanical ex vivo studies indicated that fixation of pegged implants seems to be better [26]. Glenoid loosening can be monitored by the radiolucent lines [27].

Another aspect that can affect the implant stability is the shape of the back surface of the glenoid [28]. Anglin et al. [29] showed that a convex-backed implant performed better than a flat-backed glenoid, the latter having deformed the surface of the bone substitute.

New solutions with PE hybrid implants, whose posterior surface is covered with fine metal particles, have been proposed [30]. This design should promote osteointegration and thus should reinforce uncemented fixation while retaining elasticity of the PE implant. Despite the expected solid and durable fixation of these implants, the risk of a possible metal-on-metal contact after posterior-superior PE wear should be considered, resulting in metallosis and probable fast loosening. This safety issue confirms that any new technological advance requires evaluation by long-term follow-up studies.

14.4 Biomechanics

After considering each component individually, it is now necessary to look at the biomechanics of a TSA. Each component interacts with the other. We will talk here about basic concepts that are essential to understand how to make the right choices in preoperative planning.

Congruence is the comparison between the diameter of the prosthetic humeral head and the radius of the glenoid. A prosthesis is congruent when the two diameters are identical. Should there be perfect congruence between the humeral head and the glenoid, or is it better to have mismatch in order to decrease the rocking horse effect? And if so, how much mismatch can be tolerated? The debate remains open, as some authors prefer a perfect matching [31], while others recommend a mismatch of 4–6 mm [18]. A laboratory-based study [32] showed that exact matches between glenoid and humeral radii provided significantly better results in terms of rim displacement, but not for shear-out strength.

The notion of constraint is added to this reflection. At this aim, we have to compare the glenoid depth and the sphericity of the humeral head. A constrained prosthesis will have a very deep glenoid with maybe a non-spherical head. In this case, the forces transmitted by the humerus onto the glenoid will increase. This may be beneficial for increasing the prosthetic stability, but conversely can lead to early wear and rapid loosening [33].

Other principles are also well established. An oversized glenoid implant from front to back, or a glenoid implant improperly positioned, will entail a mechanical conflict with the metaphyseal

bone. An oversized prosthetic humeral head increases the lateral offset, lever arm of the rotator cuff, soft tissue tension, and shear forces on the glenoid implant, while it decreases the range of motion. Conversely, an undersized prosthetic humeral head decreases the lateral offset, lever arm of the rotator cuff and deltoid, and increases the risk of instability.

14.5 Indications

Indication to TSA concerns patients who retain a functional rotator cuff. For the concentric OA of a patient more than 50 year old, the reasonable choice is a TSA with a stemless humeral implant, a metallic or ceramic head, and a cemented all-PE glenoid implant. The prosthetic ceramic head may be preferred in the case of a semi-prosthesis because it may reduce the risk of glenoiditis. The use of such stemless implants in this case results in easier and less aggressive surgery. The humeral cut follows the anatomical neck of the humerus. The pure metaphyseal fixation does not require addressing the medial and posterior offset. The implant is fixed in the middle of the cut and the prosthetic humeral head has a centered Morse cone. The humeral implant without a support collar on the bone cut seems to give less resorption because the fixation is electively in cancellous stimulated bone. For glenoid PE implants, the trend is to reduce the size of the implants. Some midterm studies (8-year follow-up) reported higher loosening rates on large-sized glenoids. We therefore recommend implants with a mild mismatch. Finally, in case of revision of this type of prosthesis, the removal of the humeral implant will be easily carried out and will allow the surgeon to replace it with a primary implant (anatomical or reverse).

For concentric OA of a patient less than 50 years old, the reasonable choice is HA with a humeral stemless implant and a ceramic head. The ceramic implant should erode less cartilage and should prevent early glenoiditis.

In a young patient (less than 50 years old) with a rheumatic disease and poor bone quality with large bone changes, it will be difficult to retain the recommendation to use a stemless prosthesis. In this instance, the natural choice would be toward a conventional stemmed or short-stem implant. This would make it possible to envisage a later easy revision. Whatever the choice, it is recommended to use cemented implants in this case for the best primary fixation. The question remains whether to replace the glenoid or not. On considering that functional results are better for TSA, we recommend implanting the glenoid in patients over 50.

The use of a cemented PE implant remains the "gold standard." In young patients, the choice of performing a HA may be wise. In this case, regular check of the glenoid wear will allow to estimate the right time to convert HA to TSA.

With a proximal humerus fracture that requires a prosthesis, dedicated humerus fracture stems allow reconstruction of the tuberosities around the implant. Using the glenoid implant or not depends on the condition of the glenoid and follows the abovementioned rules.

In the case of a fracture sequelae of the proximal humerus, the humeral head is often malunited. Also, the posterior offset is increased, making it very difficult to use the conventional humeral stem. As a result, the choice of a stemless prosthesis takes on its full meaning. In this case, it will be necessary to be cautious, because the bone is often very dense and the preparation of the humeral metaphysis more difficult. Frequently, fracture sequelae lead to rapid glenoid wear and TSA could be necessary.

Conclusion

Survival of the anatomical shoulder arthroplasty depends on the implant and on the technique of implantation. Even if great effort is made to improve the design and quality of the materials of the prostheses, methods of implantation of a TSA remain of paramount importance. However, it should be reminded that prosthetic shoulder surgery is a soft tissue surgery. Surgical approach is important to restore the anatomy. Soft tissue balance is the key to success.

References

1. Fevang BT, Nystad TW, Skredderstuen A, Furnes ON, Havelin LI. Improved survival for anatomic total shoulder prostheses. Acta Orthop. 2015;86(1):63–70.
2. Walch G, Boileau P, Noël E. Shoulder arthroplasty: evolving techniques and indications. Joint Bone Spine. 2010;77(6):501–5.
3. Neer CS. The classic: articular replacement for the humeral head. 1955. Clin Orthop Relat Res. 2011;469(9):2409–21.
4. Bigliani LU, Cofield RH, Flatow EL, Fukuda HA, Hawkins RJ, Matsen FA, Morrison DS, Rockwood CA Jr, Warren RF. Charles Neer: on the giant of the shoulder. J Shoulder Elb Surg. 2009;18(3):333–8.
5. Sperling JW, Cofield RH, Rowland CM. Neer hemiarthroplasty and Neer total shoulder arthroplasty in patients fifty years old or less. Long-term results. J Bone Joint Surg Am. 1998;80(4):464–73.
6. Churchill RS, Kopjar B, Fehringer EV, Boorman RS, Matsen FA. Humeral component modularity may not be an important factor in the outcome of shoulder arthroplasty for glenohumeral osteoarthritis. Am J Orthop (Belle Mead NJ). 2005;34(4):173–6.
7. Boileau P, Walch G. The three-dimensional geometry of the proximal humerus. Implications for surgical technique and prosthetic design. J Bone Joint Surg Br. 1997;79(5):857–65.
8. Favard L, Katz D, Colmar M, Benkalfate T, Thomazeau H, Emily S. Total shoulder arthroplasty – arthroplasty for glenohumeral arthropathies: results and complications after a minimum follow-up of 8 years according to the type of arthroplasty and etiology. Orthop Traumatol Surg Res. 2012;98(4 Suppl):S41–7.
9. Schmidutz F, Agarwal Y, Müller PE, Gueorguiev B, Richards RG, Sprecher CM. Stress-shielding induced bone remodeling in cementless shoulder resurfacing arthroplasty: a finite element analysis and in vivo results. J Biomech. 2014;47(14):3509–16.
10. Australian orthopaedic association national joint replacement registry. Annual report. From: https://www.aoanjrr.dmac.adelaide.edu.au/en/annual-reports.
11. Gregory T, Hansen U, Emery RJ, Augereau B, Amis AA. Developments in shoulder arthroplasty. Proc Inst Mech Eng H. 2007;221(1):87–96.
12. Levy O, Copeland SA. Cementless surface replacement arthroplasty (Copeland CSRA) for osteoarthritis of the shoulder. J Shoulder Elb Surg. 2004;13(3):266–71.
13. Thomas SR, Wilson AJ, Chambler A, Harding I, Thomas M. Outcome of Copeland surface replacement shoulder arthroplasty. J Shoulder Elb Surg. 2005;14(5):485–91.
14. Hammond G, Tibone JE, McGarry MH, Jun BJ, Lee TQ. Biomechanical comparison of anatomic humeral head resurfacing and hemiarthroplasty in functional glenohumeral positions. J Bone Joint Surg Am. 2012;94(1):68–76.
15. Maier MW, Lauer S, Klotz MC, Bülhoff M, Spranz D, Zeifang F. Are there differences between stemless and conventional stemmed shoulder prostheses in the treatment of glenohumeral osteoarthritis? BMC Musculoskelet Disord. 2015;16:275.
16. Routman HD, Becks L, Roche CP. Stemless and short stem humeral components in shoulder arthroplasty. Bull Hosp Jt Dis. 2015;73(Suppl 1):S145–7.
17. Berth A, Pap G. Stemless shoulder prosthesis versus conventional anatomic shoulder prosthesis in patients with osteoarthritis: a comparison of the functional outcome after a minimum of two years follow-up. J Orthop Traumatol. 2013;14(1):31–7.
18. Habermeyer P, Lichtenberg S, Tauber M, Magosch P. Midterm results of stemless shoulder arthroplasty: a prospective study. J Shoulder Elb Surg. 2015;24(9):1463–72.
19. Loew M. Short stem shoulder prosthesis: concept and first results. Orthopade. 2013;42(7):501–6.
20. Teissier P, Teissier J, Kouyoumdjian P, Asencio G. The TESS reverse shoulder arthroplasty without a stem in the treatment of cuff-deficient shoulder conditions: clinical and radiographic results. J Shoulder Elb Surg. 2015;24(1):45–51.
21. Bell SN, Gschwend N. Clinical experience with total arthroplasty and hemiarthroplasty of the shoulder using the Neer prosthesis. Int Orthop. 1986;10(4):217–22.
22. Matsen FA, Warme WJ, Jackins SE. Can the ream and run procedure improve glenohumeral relationships and function for shoulders with the arthritic triad? Clin Orthop Relat Res. 2015;473(6):2088–96.
23. Affatato S, De Mattia JS, Bracco P, Pavoni E, Taddei P. Wear performance of neat and vitamin E blended highly cross-linked PE under severe conditions: the combined effect of accelerated ageing and third body particles during wear test. J Mech Behav Biomed Mater. 2016;64:240–52.
24. Papadonikolakis A, Neradilek MB, Matsen FA. Failure of the glenoid component in anatomic total shoulder arthroplasty: a systematic review of the English-language literature between 2006 and 2012. J Bone Joint Surg Am. 2013;95(24):2205–12.
25. Boileau P, Moineau G, Morin-Salvo N, Avidor C, Godenèche A, Lévigne C, Baba M, Walch G. Metal-backed glenoid implant with polyethylene insert is not a viable long-term therapeutic option. J Shoulder Elb Surg. 2015;24(10):1534–43.
26. Lacroix D, Murphy LA, Prendergast PJ. Three-dimensional finite element analysis of glenoid replacement prostheses: a comparison of keeled and pegged anchorage systems. J Biomech Eng. 2000;122(4):430–6.
27. Glennie RA, Giles JW, Johnson JA, Athwal GS, Faber KJ. An in vitro study comparing limited to full cementation of polyethylene glenoid components. J Orthop Surg Res. 2015;10:142.
28. Karelse A, Leuridan S, Van Tongel A, Debeer P, Van Der Sloten J, Denis K, De Wilde LF. Consequences of reaming with flat and convex reamers for bone

volume and surface area of the glenoid; a basic science study. J Orthop Surg Res. 2015;10:181.
29. Anglin C, Wyss UP, Nyffeler RW, Gerber C. Loosening performance of cemented glenoid prosthesis design pairs. Clin Biomech (Bristol, Avon). 2001;16(2):144–50.
30. Grey SG. Use of a caged, bone ingrowth, glenoid implant in anatomic total shoulder arthroplasty technique and early results. Bull Hosp Jt Dis. 2013;71(Suppl 2):S41–5.
31. Hertel R, Knothe U, Ballmer FT. Geometry of the proximal humerus and implications for prosthetic design. J Shoulder Elb Surg. 2002;11(4):331–8.
32. Walch G, Edwards TB, Boulahia A, Boileau P, Mole D, Adeleine P. The influence of glenohumeral prosthetic mismatch on glenoid radiolucent lines: results of a multicenter study. J Bone Joint Surg Am. 2002;84-A(12):2186–91.
33. Oosterom R, Rozing PM, Verdonschot N, Bersee HE. Effect of joint conformity on glenoid component fixation in total shoulder arthroplasty. Proc Inst Mech Eng H. 2004;218(5):339–47.

15 Anatomical Shoulder Arthroplasty: Why It Fails

Pascal Gleyze, Nikolaos Tzanakakis, and Constantina Moraiti

15.1 Introduction

The procedure of total shoulder arthroplasty (TSA) is currently increasing giving excellent results in terms of pain relief and shoulder function. Complications and failures are rare in expert hands, but we can have a high prevalence if the surgeon rarely performs this procedure.

Most of these complications are not the result of specific surgical errors, but rather a sum of pre-, intra-, and postoperative details that, when not addressed, accumulate and may result in problems difficult to solve and, finally, may lead to failure.

Experience is the sum of our mistakes, and the goal of this chapter is to share the key points that lead to success or failure of TSA, starting from selecting the right indication to the postoperative rehabilitation. Based on personal experiences added to a review of the literature, we present these key points and emphasize, at the same time, pitfalls and pearls to achieve a successful procedure.

P. Gleyze (✉) · N. Tzanakakis · C. Moraiti
Department of Orthopaedics and Sports Traumatology, Hôpital Nord Franche Comté, Trévenans, France

15.2 Analysis of the Causes of Failure

The failure rate after anatomic TSA is estimated to be approximately 13% (10–16%) with a 6–7% revision rate, which is quite high for a programmed surgical procedure (Table 15.1). The most common causes of failure are glenoid loosening (8%), instability (4%), periprosthetic fractures (1.5–3%), rotator cuff tears (1.3%), nerve injuries (0.8%), infections (0.7%), and deltoid muscle dysfunction (0.1%) [1–11].

15.2.1 Glenoid Loosening

Glenoid loosening can occur because of malpositioning of the implant, inadequate cementing, oversized drilling holes or poor fixation of the metal back.

15.2.2 Fractures

Excessive external rotation during the preparation of the humerus, aggressive preparation of the humerus with oversized rasps (humerus is a fragile bone), and mistake in the choice of the entry point and the orientation of the rasps at the level of the tuberosities, which implies working in the wrong axis, are all risk factors for fracture of the humerus or cortical damage (Fig. 15.1).

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_15

Table 15.1 Published complication rate of TSA

Author	Year	F-U (years)	Cases (N)	Complications (N)	Complication rate (%)
Wirth et al. [3]	1996	3.5	1459	204	14
Walch et al. [8]	2000	3.6	766	93	13
Boshali et at. [2]	2006	5	2810	414	14.7
Chin et al. [6]	2006	5	431	53	12

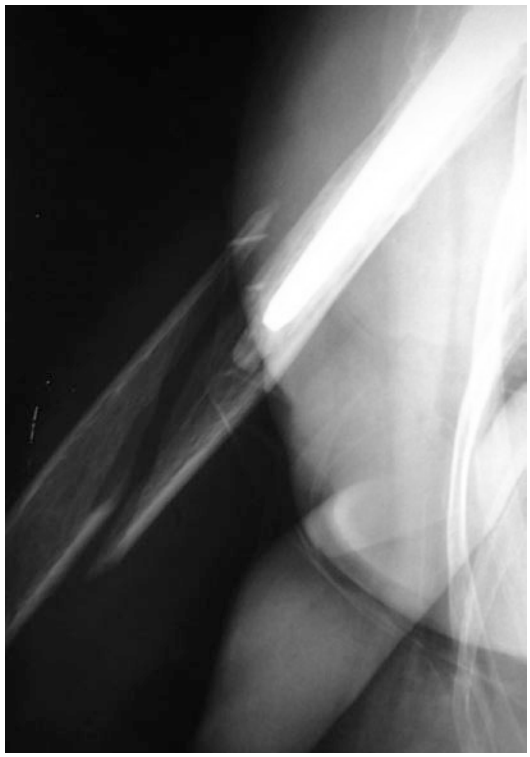

Fig. 15.1 Humeral fracture

15.2.3 Nerve Injuries

They are rare and the recovery is spontaneous in most of the cases (90%). They are mostly due to an improper dissection through the deltopectoral approach. The axillary nerve is at risk if the dissection is extended carelessly below the glenoid and the inferior border of the subscapular muscle (Fig. 15.2). Moreover, the axillary nerve is at risk of injury when we extend the dissection too distally during the anterolateral approach. On the other hand, the musculocutaneous nerve is at risk of harm when the release of the lateral border of the conjoint tendon is imprecise (Fig. 15.3).

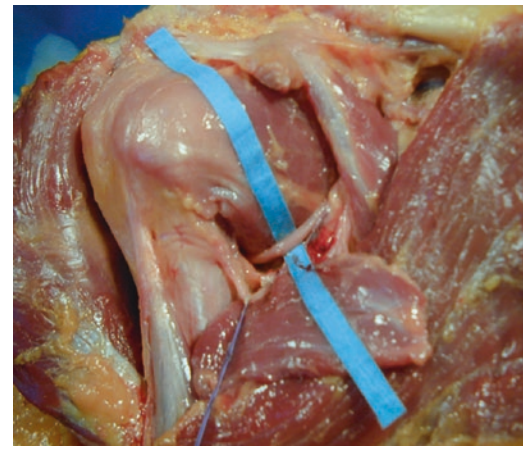

Fig. 15.2 Axillary nerve

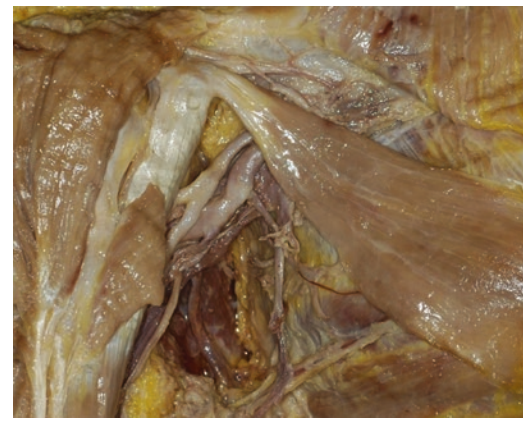

Fig. 15.3 Musculocutaneous nerve

A brachial plexus injury is possible under vigorous traction of the upper limb. It is essential knowledge that during the whole procedure, especially during "dangerous" movements (dislocation, working at glenoid, hyperextension) a nerve injury is possible, especially when the patient is fully relaxed after administration of myorelaxant drugs. Most of nerve injuries are associated with surgeon's technical errors.

15.2.4 Instability

In most cases, it is an anterior instability due to insufficiency or rupture of subscapularis. However, in some cases it can be a posterior instability and rarely superior or inferior [12].

Anterior instability can be due to the wrong placement of the glenoid component (Fig. 15.4). Specifically, the glenoid component may be in great anteversion after an excessive reaming of the anterior part or the glenoid, which will lead to anterior subluxation and ultimately to anterior dislocation of the humeral component. Thus, it is very important to calculate preoperatively the correct angle for the reaming of the glenoid, on computed tomography (CT) scans (Fig. 15.5). Intraoperatively, we must ensure the reproduction of this angle and not rely on our estimation on the version of the glenoid just visually as it could be entirely false.

In rare cases, it can be due to excessive muscle atrophy, because of pre- and/or postoperative immobilization of the shoulder or because of neurological problems.

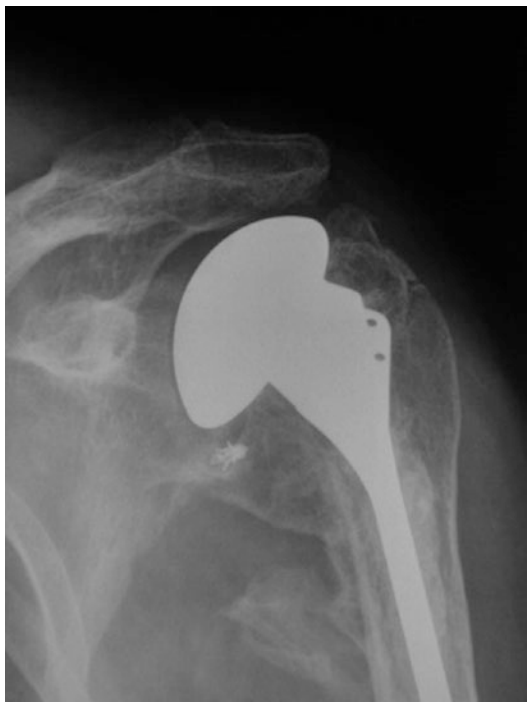

Fig. 15.4 Anterior instability

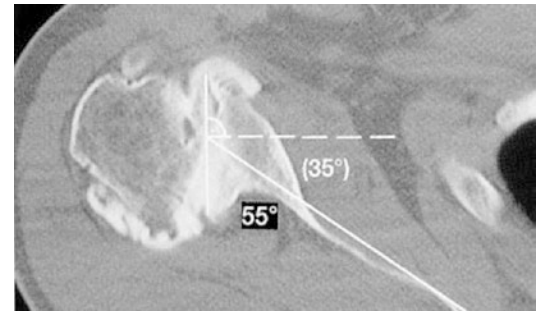

Fig. 15.5 Preoperative CT scan

Preoperative instability is a major factor of postoperative instability. Preoperative anterior instability implies laxity in the soft tissue, and a chronic weakness of the subscapularis muscle, which will influence the stability of the prosthesis. This is extremely important for anatomical TSA. Essential factors to achieve stability are perfect placement of the implant, increasing retroversion of the numeral component by a few degrees, repairing the anterior structures, and particularly reactivating the subscapularis muscle as early as possible postoperatively.

The rupture of the subscapularis is the most frequent but also the most avoidable cause of postoperative anterior instability. It is essential that the subscapularis is largely released and being functional at the end of the operation. Meticulous reinsertion is of paramount importance. When surgical management of subscapularis is inadequate, with the resumption of active mobilization, the tendon will stretch and finally fail. Thus, we will face the unpleasant situation, unfortunately so frequent, of a prosthesis with the implants perfectly positioned on the standard X-rays and with excellent results but only in short term, as progressively mechanical pain is developed. Initially this pain is minimal but it keeps increasing and functional failure with painful anterior instability will eventually occur.

Posterior instability is usually the result of a technical error, due to excessive retroversion of the humeral or/and glenoid implants. Concerning the humeral implant, the error often occurs during the preparation of the humeral shaft when the guide is not adequate or not properly set. The

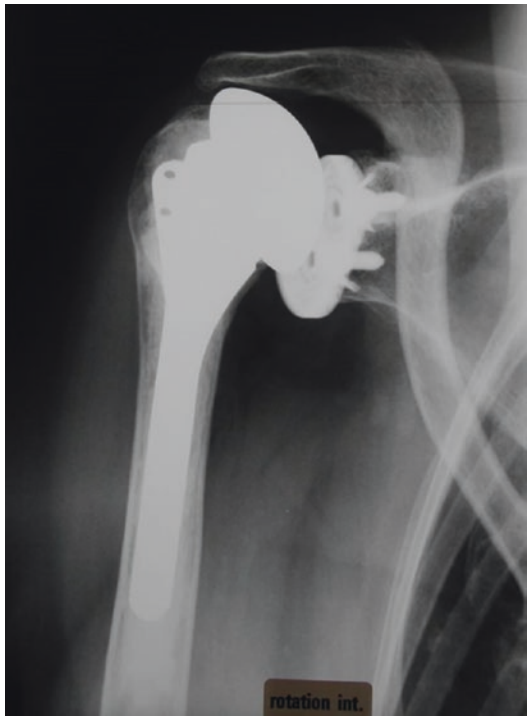

Fig. 15.6 Superior instability

surgeon must be careful when dealing with a B2 type of glenoid, according to the Walch's classification [13], as a nonsymmetric reaming may at the end increase the retroversion and therefore lead to posterior instability.

Preoperative hyperlaxity and posterior instability (both rare) are also factors of postoperative instability due to laxity of the posterior capsule.

Superior instability is, in most cases, the result of a massive rotator cuff tear. Preoperative and intraoperative assessment of the quality of the rotator cuff is important to avoid this complication. Rotator cuff is at risk of failure when fatty degeneration is present, still appearing in continuity during surgery [14]. Superior instability may also occur when rotator cuff is not reattached at the end of the procedure (Fig. 15.6).

Coracoacromial ligament release and upper tilt of the glenoid implant are considered as risk factors for superior instability [1, 2].

Implanting the humeral component in a low position (Fig. 15.7), usually due to a wrong humeral cut or in the base of bone loss when

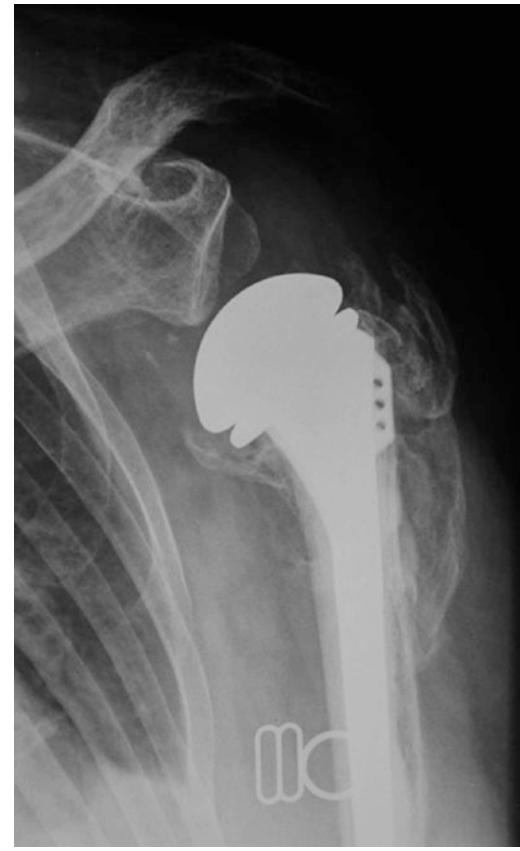

Fig. 15.7 Inferior instability

performing the prosthesis on a malunion, may lead to inferior instability. Congenital hyperlaxity as well as deltoid dysfunction may also be responsible for inferior instability [1, 2].

15.2.5 Infection

An infection is rarely intrinsic [3]; it results, in the vast majority of cases, from faults when taking care of the patient. These faults are always under the responsibility of the surgeon, even if he is cautious and paradigmatic in his operating procedure (dressing, safety distance, control of personnel and materials, etc.). The infection is due to an accumulation of mistakes and risk factors, which unfortunately are not always under the control of the surgeon (i.e., careless passage of a non-sterile staff and contact with the sterile field, grasping the handle of the operating theater

light, which is no more sterile as it had been touched by the surgeon's or the nurse's hat, etc.). Whatever the cause, it should not be perceived as a fatality by a surgeon worthy of his ethics and responsibilities. Each one of us is responsible for the acts of the entire staff that participates in the medical care of the patient. It is not only a matter of operating properly, but it is also of assuming the trust that the patient has in us.

15.3 How to Prevent Failure: From Indication to Postoperative Rehabilitation

In this subheading, we will focus on the key points that assure the success of the shoulder arthroplasty. Rather than describing a faultless procedure, we will emphasize on the points that may hide some traps if they are not deeply understood and correctly performed.

15.3.1 Indication to Surgery and Patient Information

The goal is to explain to the patient the limitations of the surgery and verify that the patient has well understood what the expectations from this procedure are. Informing the patient is of paramount importance. The patient always seeks for an improvement in her/his shoulder function and relief of pain; we should be able achieve these goals. We know that muscle atrophy and fatty infiltration and/or preoperative shoulder stiffness may have a negative role on the outcome, and this should be clearly explained to the patient. It is also important to explain that apart from the surgical procedure, her/his postoperative activity regimen is equally of great importance for a functional result. Even the best shoulder arthroplasty performed by the best surgeon in the world will not have good results if the patient does not show any will or any motivation or if she/he limits his activities. Pain is usually diminished after surgery, but functional recovery depends largely on muscle atrophy, shoulder stiffness, and activity. This information will help the patient to accept the outcome of the procedure when it is not as excellent as expected!

15.3.2 Preoperative Planning

The orthopedic surgeon is a "real time 3D receptor-analyst." He must incorporate and calculate, continuously, during the whole procedure, the 3D positioning of all the anatomical structures for being himself accurate and appropriate in his gestures. This is of vital importance in TSA for the integration of every information relative to the orientation of the glenoid and the malformations/retroversion of the humeral head. Thus, the surgeon must study and integrate in his preoperative planning all anatomical details for ensuring a good result.

On standard X-rays, we must evaluate bone quality and the grade of osteoarthritis (OA) and identify any osteophytes. OA can be assessed with the classification of Samilson [15], Walch [11], or Sirveaux [16] and with radiographs of the contralateral shoulder which may be useful in elderly patients.

CT-arthrography (CTA) or MRI must be performed in all patients to evaluate the rotator cuff for tendon tears and fatty infiltration of the muscles, particularly the subscapularis. Imaging studies should be used to evaluate the shape of the glenoid (e.g., B2 type) and its bone density. Preoperative and intraoperative assessment of the axis of the glenoid relatively to the articular surface are of paramount importance for the right preparation of the glenoid.

15.3.3 Patient Positioning

Checking and bearing in mind the patient positioning is very important to avoid intraoperative fractures, nerve lesions, misunderstanding of the orientation and thus wrong positioning of the implants, dislocations, etc. Intraoperative comfort and security are imperative; thus, the following criteria seem extremely important to us.

A good positioning of the trunk of the patient allows us to place the arm in extension. Placing the elbow behind the body allows sufficient exposition of the humerus and is one of the key points for the good visualization of the glenoid. By using a special orthopedic table, which offers the possibility to remove its posterolateral part exposing the posterior surface of the shoulder, we avoid placing the patient in an unstable position on the lateral border of the table. In this phase, while inspecting the positioning of the patient, the surgeon must also examine the orientation of the trunk relative to the ground. This could be useful while preparing the glenoid, for the definition of the axes and the center.

Maximum muscle relaxation is of paramount importance, especially when performing the deltopectoral approach, which demands the retraction of the deltoid. An inappropriate regional anesthesia and insufficient muscle relaxation could make surgery more difficult or, the worst, dangerous.

Systolic blood pressure should be 100 mmHg or below, to obtain a precise dissection and a bloodless operating field.

15.3.4 Surgical Approach

This depends on surgeon's preference. Deltopectoral approach is usually used for anatomic shoulder arthroplasty while the anterior-superior approach for reverse shoulder arthroplasty (RSA).

When performing the anterior-superior approach, it is necessary to find the space between the anterior and the middle part of the deltoid. If not, the dissection it will be through the fibers of the deltoid, making that too aggressive. The distance between the lateral border of the acromion and the distal part of the dissection should not exceed "three finger widths," meaning 3 cm, to avoid injuring the axillary nerve. The "safe zone" to avoid the axillary nerve is traditionally defined between 5 and 7 cm distally to the lateral border of the acromion. However, in 20% of the cases, the axillary nerve lies 3–5 cm [17, 18] distally to the acromion. Thus, it is imperative to identify the axillary nerve when we should extend the approach further distally. Injuring the axillary nerve will result in a deficit in flexion, abduction and external rotation of the superior limb, deltoid atrophy, and sensory deficit at the lateral surface of the shoulder and the anterior surface of the arm. The lesion may be minimal, such as neurapraxia, presenting mild clinical signs that may not be perceived postoperatively as there is always pain and limitation in shoulder function. We must always search for these signs, and if in doubt, we should not hesitate to perform an electromyography, which in most cases will be reassuring.

Deltopectoral approach is usually used for anatomical shoulder arthroplasty. It allows for the distal extension of the dissection but can be very aggressive for the muscles if there is insufficient muscle relaxation. A good exposure without injuring the musculocutaneous and the axillary nerves requires careful dissection of the lateral border of the conjoint tendon and the proximal two thirds of the pectoralis major on the humerus. The axillary nerve lies approximately 2 cm from the inferior border of the glenoid. For this reason, the preparation of the subscapularis should be performed with great caution, with the arm in internal or neutral rotation (no more than 0° external rotation). With the arm in external rotation, the axillary nerve is under tension and comes closer to the subscapularis and the inferior border of the glenoid.

15.3.5 Subscapularis Release

We have already discussed the importance of the subscapularis on the functional outcome and the risk of anterior instability if this muscle is not efficient after surgery [19]. We must understand that the subscapularis is already in a nonoptimal condition in a shoulder that needs an arthroplasty. This is because the shoulder is usually stiff, with a deficit in external rotation, which creates the condition of atrophy or even fatty infiltration of the muscle. Therefore, we must be very cautious in dissecting and reinserting the subscapularis.

Subscapularis can be released by either tenotomy or osteotomy of the lesser tuberosity. The osteotomy leaves the tendon intact but necessitates a solid fixation to avoid failure or nonunion. In addition, the sutures put in the subscapularis as

landmarks allow for an estimation of its quality and its stiffness. The preparation and the dissection of the subscapularis must be performed from lateral to medial and from superior to inferior, trying to avoid its inferior border. The techniques of "freeing" the subscapularis [19] are dangerous and in most cases useless. The best is to pass and slide a finger over the posterior surface of the subscapularis and cut the adhesions to the anterior glenoid rim and the neck of the glenoid. Usually there are osteophytes at the anterior border of the glenoid that need to be excised. We can use an elevator to liberate the anterior surface of the glenoid neck, but we should be very careful. Subscapularis release is completed by sectioning the coracohumeral ligament and by partially resection of the anterior capsule. At the end of the procedure, the subscapularis should be reinserted without tension with the arm in external rotation at 0 degrees of abduction.

15.3.6 Intraoperative Assessment of the Rotator Cuff

CTA or MRI scans allow for an adequate preoperative evaluation of the rotator cuff. Intraoperative visualization and tactile evaluation are very important. Good-quality tissue without fatty infiltration suggests that in the case of a rupture of the supraspinatus, the surgeon could perform anatomic TSA and rotator cuff repair. This will be more beneficial to the shoulder than a RSA. On the contrary, when rotator cuff shows anatomical integrity and good-quality tissue on the preoperative imaging studies, but looks fragile during surgery, a RSA should be implanted, especially in elderly patients. This is because a weak rotator cuff would eventually fail in a few months after surgery, thus necessitating an early revision surgery to RSA. Biological age of patient (tissue quality) and surgeon's experience are crucial to decide the best option for the fate of the implant.

15.3.7 Adequate Capsulotomy

This is one of the most important points for the optimal exposure of the glenoid, which is vital for the good positioning of the glenoid implants. Moreover, an adequate capsulotomy is a prerequisite for achieving full range of motion postoperatively in all stiff osteoarthritic shoulders. This capsulotomy is usually neglected, as the surgeon after liberating the subscapularis and dislocating the humerus goes on with preparing the humerus without realizing the difficulties he will encounter when preparing the glenoid if the capsulotomy is not correct. We must understand that to prepare the glenoid and to position and orient correctly the implants, we must expose the entire articular surface of the glenoid and be able to place the Kirchner wire, the reamer, and all other instruments perpendicular to that. This is possible only if the capsulotomy is carried beyond the inferior pole of the glenoid. Ideally, it should be extended at least up to 7 o'clock position which will allow for a sufficient "opening" of the joint and exposure of the articular surface. This step may be difficult and dangerous, as it requires dissection of the fibrous and sometimes thick capsular tissue with adhesions to the inferior border of the glenoid. The degree of difficulty increases in cases of bloody fields or obese patients.

Once the subscapularis is dissected (upper two thirds), traction sutures are put at the muscle-tendon junction, and a retractor is placed on the posterior border of the glenoid. Thus, the anterior capsule is in tension, allowing for its identification and its resection from the anterior border at the glenoid at 5 o'clock position, starting from the correspondent level on the posterior part of the subscapularis (Fig. 15.8). While advancing with the resection, the head is progressively driven away from the glenoid. Once the section

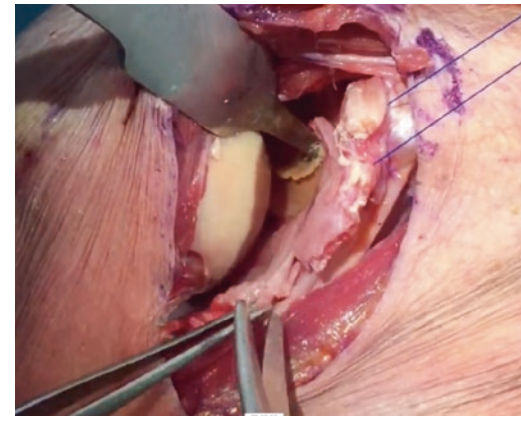

Fig. 15.8 Capsulotomy

of the capsule at 5 o'clock position is achieved, the rest of the resection should be done with extreme caution, in contact with the bone, using scissors, up to the 7 o'clock position. This will expose the inferior part of the glenoid neck. When the capsulotomy is completed, the humeral head can be dislocated and good glenoid exposure can be achieved.

15.3.8 Preparation of the Humerus

The appropriate cut of the humeral neck is still under debate. The angle of the cut is related to many biomechanical elements that interfere with the functional outcome such as lateral offset, the distance between the tip of the head and the great tuberosity (which influences the range of motion and strength), etc. [20]. There are two tendencies: making a cut in fix angle or in a variable angle. We think that a cut in a variable angle counteracts the benefit of an off-centered humeral head. The off-centered heads, de facto, compensate for the cut. Thus, we propose using the off-centered heads and avoid malpositioning.

The preparation of the shaft must be meticulous, progressive. The entry point for the rasp (before cutting the head) must be perfectly centered. We should not exaggerate in our effort to seek a perfect contact between the implant with the cortical, as we might provoke fractures and false routes.

15.3.9 Preparation and Positioning of the Glenoid Implant

Malpositioning of the glenoid implant will lead to an acute failure. This complication is the most common cause for revision surgery [21, 22]. Adequate positioning of the glenoid component is possible if we perform an extended capsulotomy further than 6 o'clock position on the glenoid. It is important to check if there is any osseous border left around the glenoid after the reaming, which could prevent the impaction of the glenoid implant.

15.3.10 Positioning of the Humeral Implant

The humeral implant must be in the right axis, even in the case of "stemless" prostheses. The axis of the shaft must be respected to avoid humeral loosening and even glenoid loosening. The percentage of the remplissage must be according to the manufacturer's guidelines.

The distance between the tip of the great tuberosity and the humeral head should be, ideally, 6 mm. The axis of the humeral shaft should be 30 mm away from the articular surface of the glenoid [20].

15.3.11 Mismatch

The recommendations of the manufacturers are very precise and must be respected. A mismatch between 2 and 4 mm corresponds to the guidelines for most the implants. A mismatch greater than 4 mm increases the risk of a secondary rupture of the subscapularis while a mismatch inferior to 2 mm increases the risk of failure of the glenoid implant.

15.3.12 Reinsertion of the Rotator Cuff and of the Subscapularis

We must not hesitate to dedicate the time needed to assure that the reinsertion of the subscapularis is well adapted to the rotation and the rotator cuff interval is closed. Closing the rotator cuff interval solidifies the reinsertion of the subscapularis as the supraspinatus becomes a part of it.

15.3.13 Postoperative Care

The outcome of TSA equally depends on the patient's ability to move the arm and on the quality of the procedure. Thus, it is essential to verify that postoperative rehabilitation is correctly performed, especially for restriction of external rotation. Passive and active ROM exercises are allowed

immediately after surgery only if the patient respects the restriction of the external rotation (0°). Only passive ROM exercises are allowed for the first 6 weeks after surgery in case of metal-backed glenoid and if rotator cuff repair was performed.

Conclusion

Anatomical TSA is a common surgical procedure that provides satisfactory results. Preservation of anatomical structures and in particular of the rotator allows a good functional recovery and an almost complete pain relief. However, sometimes this is not the case, because of small technical deficiencies or errors that, when accumulated, can increase difficulty of the surgical procedure and the risk of complications and failures. The best and the worst are always possible in the field of shoulder arthroplasty and one can switch very easily from one to the other. In this chapter, we provided some practical and useful tips and pearls to shoulder surgeons based on our experience, which is the fruit of past errors and is always beneficial to share.

References

1. Bohsali KI, Bois AJ, Wirth MA. Complications of shoulder arthroplasty. J Bone Joint Surg Am. 2017;99(3):256–69.
2. Bohsali KI, Wirth MA, Rockwood CA Jr. Complications of total shoulder arthroplasty. J Bone Joint Surg Am. 2006;88(10):2279–92.
3. Wirth MA, Rockwood CA Jr. Complications of total shoulder-replacement arthroplasty. J Bone Joint Surg Am. 1996;78(4):603–16.
4. Affonso J, Nicholson GP, Frankle MA, Walch G, Gerber C, Garzon-Muvdi J, McFarland EG. Complications of the reverse prosthesis: prevention and treatment. Instr Course Lect. 2012;61:157–68.
5. Mattei L, Mortera S, Arrigoni C, Castoldi F. Anatomic shoulder arthroplasty: an update on indications, technique, results and complication rates. Joints. 2015;3(2):72–7.
6. Chin PY, Sperling JW, Cofield RH, Schleck C. Complications of total shoulder arthroplasty: are they fewer or different? J Shoulder Elb Surg. 2006;15(1):19–22.
7. Wiater BP, Moravek JE Jr, Wiater JM. The evaluation of the failed shoulder arthroplasty. J Shoulder Elb Surg. 2014;23(5):745–58.
8. Walch G, Boileau P. Prosthetic adaptability: a new concept for shoulder arthroplasty. J Shoulder Elbow Surg. 1999;8:443.
9. Walch G, Edwards TB, Boulahia A, Boileau P, Mole D, Adeleine P. The influence of glenohumeral prosthetic mismatch on glenoid radiolucent lines: results of a multicenter study. J Bone Joint Surg Am. 2002;84-A(12):2186–91.
10. Westphal T, Woischnik S, Adolf D, Feistner H, Piatek S. Axillary nerve lesions after open reduction and internal fixation of proximal humeral fractures through an extended lateral deltoid-split approach: electrophysiological findings. J Shoulder Elb Surg. 2017;26(3):464–71.
11. Aibinder WR, Schoch B, Schleck C, Sperling JW, Cofield RH. Revisions for aseptic glenoid component loosening after anatomic shoulder arthroplasty. J Shoulder Elb Surg. 2017;26(3):443–9.
12. Padegimas EM, Zmistowski BM, Restrepo C, Abboud JA, Lazarus MD, Ramsey ML, Williams GR, Namdari S. Instability after reverse total shoulder arthroplasty: which patients dislocate? Am J Orthop (Belle Mead NJ). 2016;45(7):E444–50.
13. Walch G, Badet R, Boulahia A, Khoury A. Morphologic study of the glenoid in primary glenohumeral osteoarthritis. J Arthroplast. 1999;14(6):756–60.
14. Goutallier D, Postel JM, Bernageau J, et al. Fatty muscle degeneration in cuff ruptures. Pre- and postoperative evaluation by CT scan. Clin Orthop Relat Res. 1994;304:78–83.
15. Samilson RL, Watkins MB, Winters DM. Acute suppurative arthritis. J Bone Joint Surg Am. 1956;38-A(6):1313–20.
16. Molé D, Wein F, Dézaly C, Valenti P, Sirveaux F. Surgical technique: the anterosuperior approach for reverse shoulder arthroplasty. Clin Orthop Relat Res. 2011;469(9):2461–8. Published online 2011 Mar 30.
17. Gill TJ, Hawkins RJ. Complications of shoulder surgery: treatment and prevention. New York: Lippincott; 2008. p. 88.
18. Valenti P, Sirveaux F. Surgical technique: the anterosuperior approach for reverse shoulder arthroplasty. Clin Orthop Relat Res. 2011;469(9):2461–8. Published online 2011 Mar 30.
19. Scheibel M, Habermeyer P. Subscapularis dysfunction following anterior surgical approaches to the shoulder. J Shoulder Elb Surg. 2008;17(4):671–83.
20. Katz D, Kany J, Valenti P, Sauzières P, Gleyze P, El Kholti K. New design of a cementless glenoid component in unconstrained shoulder arthroplasty: a prospective medium-term analysis of 143 cases. Eur J Orthop Surg Traumatol. 2013;23(1):27–34.
21. Sabesan VJ, Ackerman J, Sharma V, Baker KC, Kurdziel MD, Wiater JM. Glenohumeral mismatch affects micromotion of cemented glenoid components in total shoulder arthroplasty. J Shoulder Elb Surg. 2015;24(5):814–22.
22. Papadonikolakis A, Neradilek MB, Matsen FA. Failure of the glenoid component in anatomic total shoulder arthroplasty: a systematic review of the English-language literature between 2006 and 2012. J Bone Joint Surg Am. 2013;95(24):2205–12.

Total Shoulder Arthroplasty: How to Prevent Failure

16

Philipp R. Heuberer and Leo Pauzenberger

16.1 Introduction

Total shoulder arthroplasty (TSA) has evolved significantly over the last decades starting in 1955 with the original Neer monoblock prosthesis, over the second-generation Neer prosthesis at the end of the 1980s, followed by the third-generation prosthesis developed by Walch that allowed to adjust the offset, and finishing with the anatomical humeral head replacement developed by Gerber at the end of the 1990s (fourth-generation implant, additionally adjustable inclination). At the same time, the humeral head resurfacing developed by Copeland has been successfully implanted since the early 1990s, allowing a bone-sparing surgery and, thus, giving more options in revision situations. At the beginning of the new millennium, stemless humeral head implants with metaphyseal fixation were developed that combined the advantages of bone stock preservation and facilitating glenoid access at the same time.

Over the years, TSA evolved into a very successful surgery. Two factors led to success. First, the large choice of implant systems provided options for all kinds of surgical situations; second, improved surgeons' skills and experience made improved the outcome of the procedure.

P. R. Heuberer (✉) · L. Pauzenberger
St. Vincent Shoulder & Sports Clinic, Vienna, Austria
e-mail: philipp@heuberer.at

16.2 Causes of Failure of Total Shoulder Arthroplasty

Arthroplasty registries can teach a lot when it comes to survival and revision rates. Overall, it is expected that the cumulative percentage of revision surgery for conventional total shoulder replacements for osteoarthritis is 10.1% after 7 years [1]. In general, the age of the patient is a risk factor for revision. Patients aged less than 55 years have a higher revision rate compared to those aged 65–75 years and older. Hence, the younger the patient, the higher the risk of revision. Regarding the implants, the question arises, as in all joint replacements, whether components should be cemented or one should rely on bone ingrowth. Looking strictly at registries, the message is quite clear: Revision rates are considerably higher if the glenoid component is not cemented. Regarding the humeral shaft, it does not matter if a cemented or cementless technique is used.

On the glenoid side, there are mainly two possibilities with an all-polyethylene or a metal-backed glenoid component. Literature and arthroplasty registries clearly show that initial revision rates can be lowered by the use of all-polyethylene glenoid components, but are likely to fail as well over time. Metal-backed glenoid components with a modular insert are known to be prone to loosening of the polyethylene insert, whereas metal-backed glenoid

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_16

components with a fixed insert seem to last longer. Nevertheless, metal-backed glenoid components in general seem to have some inherent potential problems: First, the thickness of combined metal and polyethylene components increases the risk of joint overstuffing. Second, given the ball-and-socket configuration of the glenohumeral joint, the main forces of the humeral head on the glenoid component are translational, not compressive forces, and, therefore, there is an imminent risk of glenoid loosening due to the so-called rocking horse phenomenon.

Polyethylene glenoid components come as pegged or keeled versions. Revision rates do not differ much between the two, although it seems that bone stock defects left by pegged glenoid components are somewhat easier to handle than those of keeled components in the revision setting.

Overall, the impact of implant design shows improved survival for newer generations of shoulder prostheses [2].

Reasons for revision surgery in TSA are manifold. The two predominant reasons are secondary rotator cuff insufficiency and instability or dislocation, which are mostly contributed to a rotator cuff insufficiency. Both together account for nearly 50% of revision surgeries. In comparison, component loosening and postoperative infections are relatively infrequent causes.

There are three possibilities where the rotator cuff can fail: anteriorly, posteriorly and superiorly. Causes for anterior rotator cuff failure, or secondary subscapularis tears, include inadequate soft tissue releases or overstuffing of the joint with continuous anterior soft tissue contact and irritation.

Posterior rotator cuff failure can often be attributed to either component malpositioning or glenoid malcorrection, especially in a glenoid type B2 or C according to Walch's classification [3]. Superior cuff failures occur in cases of bad tendon qualities, also known as rotator cuff at risk, or implant overstuffing [4].

16.3 How to Prevent Failure

Taking into account the main causes of failure of TSAs, surgical technique has a major role. Several key steps have to be respected to achieve a good outcome in TSA. First, accurate preoperative planning is essential for a successful surgery. Therefore, the routine acquisition of preoperative computed tomography (CT) or magnetic resonance imaging (MRI) scans is highly encouraged. Glenoid morphology according to Walch's classification [3], glenoid version and posterior subluxation of the humeral head need to be evaluated preoperatively. Glenoid wear according to Sirveaux's classification [5] and available bone stock do further dictate guidewire placement, so that the glenoid can be corrected anatomically. Finally, muscle volume and tendon status guide the decision on which implant to choose.

A deltopectoral approach is the gold standard for TSA. The skin incision is leading inferiorlaterally from the coracoid with the possibility of simple extension, if necessary together with a clavicular osteotomy [6]. After splitting the deltopectoral interval, the long head of the biceps (LHB) tendon is approached, which should be routinely sacrificed, whereas data show that a biceps tenodesis provides a more favourable postoperative outcome than a tenotomy (Fig. 16.1).

The next critical step is the takedown of the subscapularis tendon. There are mainly three options for successive subscapularis repair, depending on the technique of detachment: tendon-to-tendon, tendon-to-bone or bone-to-bone detachment. Cumulative data in the literature suggests that an osteotomy presents equal clinical results to peeling the tendon from its bony attachment, whereas a mid-tendon tenotomy seems to present slightly inferior results in comparison [7–9]. However, in biomechanical cadaver studies, no method showed clear superiority over the others [10, 11]. Regardless of the used technique, after detachment of the subscapularis tendon, there are several risks that need to be considered: First, fatty infiltration of the

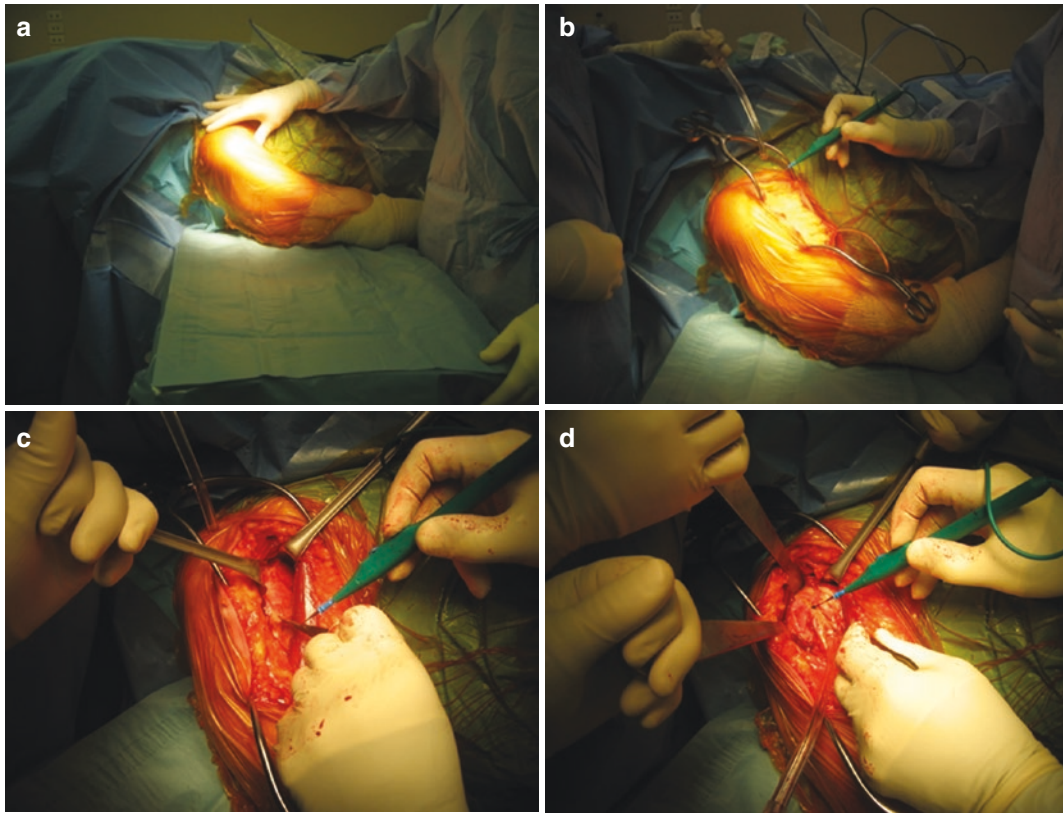

Fig. 16.1 Standard deltopectoral approach for anatomic TSA. The patient is placed in beach chair position (**a**). An oblique skin incision approximately from the tip of the coracoid following the deltoid muscle to lateral of the axilla is made (**b**). The deltopectoral interval is opened (**c**) and dissection continued down to the rotator cuff muscles. By following the LHB tendon proximally, the rotator cuff interval can be opened and the superior edge of the subscapularis muscle is defined (**d**)

subscapularis muscle progresses after tendon detachment and reattachment [12]. Second, fatty infiltration of the subscapularis muscle higher than grade II is associated with worse outcome after TSA [13]. Postoperatively, 25% of the patients show a decreased subscapularis function and 30% show a partial subscapularis tendon tear [14]. The overall revision rate for subscapularis tendon insufficiency has been reported to be at least 4% [15]. Therefore, subscapularis detachment is one of the most critical steps of the deltopectoral approach with pivotal implications for further revision surgery (Fig. 16.2).

Next, an adequate soft tissue release is mandatory, including the release of the subscapularis tendon from the joint capsule together with a release of the inferior joint capsule from the humeral head under external rotation and abduction until the teres minor muscle insertion is reached. It is essential to carefully protect the axillary nerve during these releases.

Only after sufficient soft tissue release, an adequate humeral head dislocation and subsequent glenoid exposure can be achieved. Humeral head resection should be performed along the anatomical neck and under careful preservation of the rotator cuff insertion. A recent study suggested an inferio-superior rather than an anterio-posterior resection, matching the native humeral head retroversion more closely than with an antero-superior resection

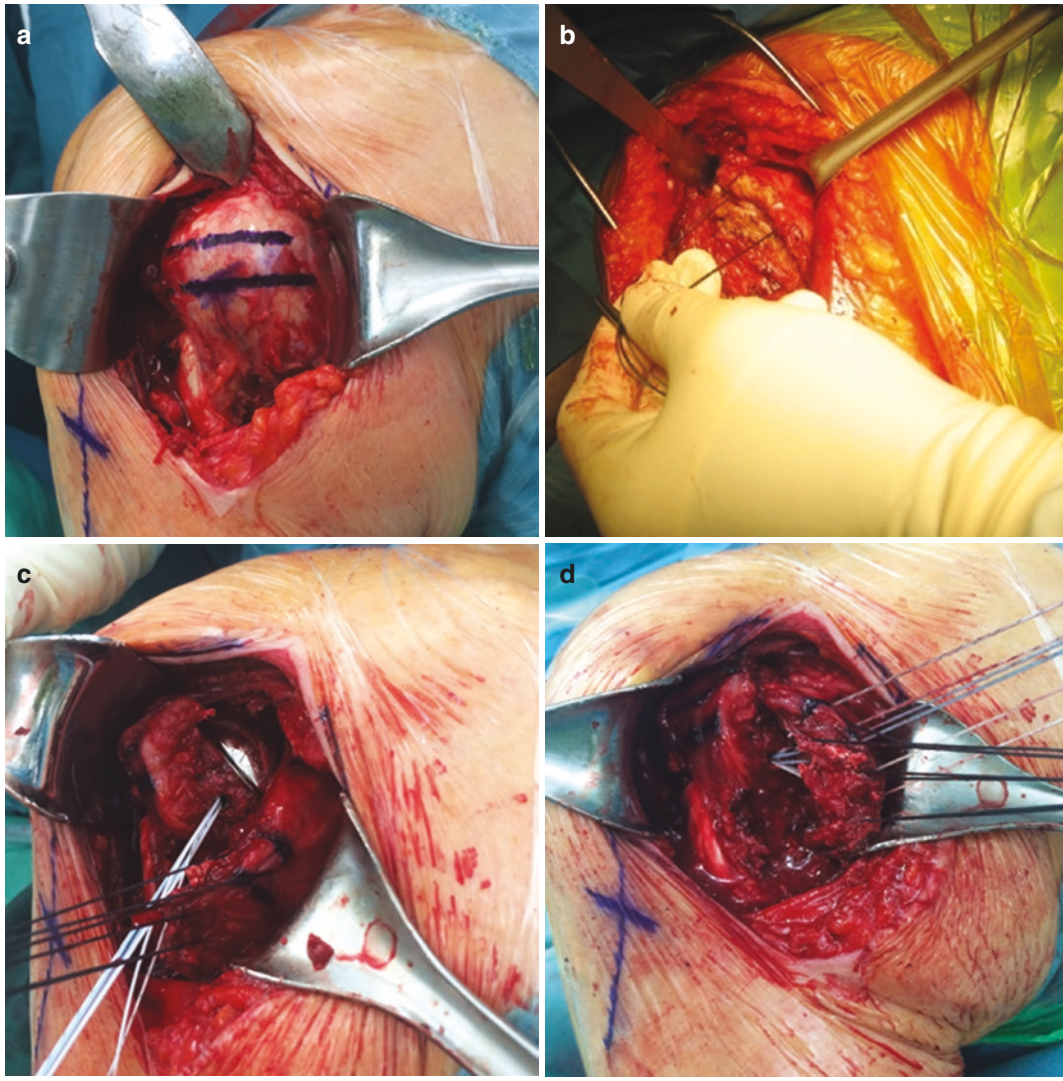

Fig. 16.2 Combined technique for subscapularis detachment. The subscapularis tendon is marked to help later anatomically correct refixation (**a**). Detachment is performed by peeling off the medial two thirds of the tendon, leaving the lateral third of the tendon intact for side-to-side reconstruction (**b**). A combination of a triple-loaded suture anchor with FiberWire (Arthrex, Inc.) and permanent sutures is used (**c**). The FiberWire sutures are passed through the tendon in a mattress stitch fashion to provide a secure medial row fixation, whereas the permanent sutures are used for side-to-side fixation with the left-intact lateral third of the tendon (**d**)

technique [16]. Inferior osteophytes should be completely removed before progressing further.

Glenoid exposure is achieved by leaving the humeral head posteriorly with a retractor so a full circumferential release around the glenoid can be carried out. The goal is to have an "en face" view of the glenoid, which is further facilitated by full muscle relaxation.

The most crucial step then is to locate the glenoid centre. Although, various guiding instruments have been developed, it is important to always consider individual glenoid morphology, how much eccentric bone needs to be removed and if any bone graft is needed. Correction of glenoid version should be planned before surgery, ideally on multiplanar CT or 3D reconstruc-

tions. When introducing the guidewire into the supposed glenoid centre, palpation of the glenoid rim with the finger and drilling under triangulation can be helpful to ensure correct drilling direction. Reaming of the glenoid bone bed should not exceed the subchondral bone area and be carried out until flush seating of the trial component is reached.

As discussed before, it might be beneficial to use a cemented glenoid implant. However, only minimal cement should be used, mainly filling the peg or keel holes. Once the cement has hardened, the humeral head is exposed again, and in case of a stemmed implant, the humeral shaft is prepared according to the implant's surgical technique.

Regarding retroversion of the humeral component, restoration of an anatomical version is recommended. Again, the definitive humeral prosthesis can be either cemented or cementless, with the literature not clearly favouring one fixation technique over the other.

Some surgical pearls in TSA can improve the success of the procedure. When testing the stability of the glenohumeral joint, the humeral head should pop back into position energetically when pushed posteriorly, indicating adequate rotator cuff tension. The subscapularis tendon needs to be reattached firmly. At this aim, we use a combination of transosseous and side-to-side sutures following tendon peel detachment with a small humeral tendon stump. Non-absorbable sutures can be used when closing the deltopectoral interval, so it is easier to find in revision surgery.

16.4 Future Directions

TSA is still evolving. New designs, such as stemless humeral head implants, have already proven to work as intended and are now approaching the 10-year follow-up mark. Furthermore, they have successfully simplified revision surgery already since their introduction.

Subscapularis-sparing surgical techniques have been developed, which could prevent the downsides associated with detachment of the subscapularis tendon. However, these techniques are very demanding and not applicable in every patient.

As component positioning in TSA is a crucial step, patient-specific instrumentation (PSI) has been recently introduced. PSI could improve surgical accuracy and support optimal component positioning with consistent correction of version and inclination. How this will influence the long-term clinical outcome and survival of TSA will be object of future studies.

Conclusion

Total shoulder arthroplasty has come a long way since its beginnings and developed into a very successful surgery over the last decades. Nonetheless, compared to total knee and hip reconstruction, shoulder arthroplasty still provides inferior long-term clinical outcome and survival rates. With the introduction of reverse shoulder arthroplasty, some major problems could be addressed, but also at the cost of creating new issues. Further development of arthroplasty systems and surgical techniques will be necessary in the times to come, to make shoulder replacement as uniformly successful as knee and especially hip arthroplasty.

References

1. NJRR A. Australian orthopaedic association national joint replacement registry. Demographics and outcomes of shoulder arthroplasty. Adelaide: AOANJRR; 2013, 2014, 2015.
2. Fevang BT, Nystad TW, Skredderstuen A, Furnes ON, Havelin LI. Improved survival for anatomic total shoulder prostheses. Acta Orthop. 2015;86(1):63–70.
3. Walch G, Badet R, Boulahia A, Khoury A. Morphologic study of the glenoid in primary glenohumeral osteoarthritis. J Arthroplast. 1999;14(6):756–60.
4. Sperling JW, Hawkins RJ, Walch G, Mahoney AP, Zuckerman JD. Complications in total shoulder arthroplasty. Instr Course Lect. 2013;62:135–41.
5. 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.
6. Redfern TR, Wallace WA, Beddow FH. Clavicular osteotomy in shoulder arthroplasty. Int Orthop. 1989;13(1):61–3.

7. Scalise JJ, Ciccone J, Iannotti JP. Clinical, radiographic, and ultrasonographic comparison of subscapularis tenotomy and lesser tuberosity osteotomy for total shoulder arthroplasty. J Bone Joint Surg Am. 2010;92(7):1627–34.
8. Jandhyala S, Unnithan A, Hughes S, Hong T. Subscapularis tenotomy versus lesser tuberosity osteotomy during total shoulder replacement: a comparison of patient outcomes. J Shoulder Elb Surg. 2011;20(7):1102–7.
9. Lapner PL, Sabri E, Rakhra K, Bell K, Athwal GS. Comparison of lesser tuberosity osteotomy to subscapularis peel in shoulder arthroplasty: a randomized controlled trial. J Bone Joint Surg Am. 2012;94(24):2239–46.
10. Van den Berghe GR, Nguyen B, Patil S, D'Lima DD, Mahar A, Pedowitz R, Hoenecke HR. A biomechanical evaluation of three surgical techniques for subscapularis repair. J Shoulder Elb Surg. 2008;17(1):156–61.
11. Van Thiel GS, Wang VM, Wang FC, Nho SJ, Piasecki DP, Bach BR Jr, Romeo AA. Biomechanical similarities among subscapularis repairs after shoulder arthroplasty. J Shoulder Elb Surg. 2010;19(5):657–63.
12. 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.
13. Edwards TB, Boulahia A, Kempf JF, Boileau P, Nemoz C, Walch G. The influence of rotator cuff disease on the results of shoulder arthroplasty for primary osteoarthritis: results of a multicenter study. J Bone Joint Surg Am. 2002;84-A(12):2240–8.
14. Liem D, Kleeschulte K, Dedy N, Schulte TL, Steinbeck J, Marquardt B. Subscapularis function after transosseous repair in shoulder arthroplasty: transosseous subscapularis repair in shoulder arthroplasty. J Shoulder Elb Surg. 2012;21(10):1322–7.
15. Scheibel M, Habermeyer P. Subscapularis dysfunction following anterior surgical approaches to the shoulder. J Shoulder Elb Surg. 2008;17(4):671–83.
16. Suter T, Kolz CW, Tashjian RZ, Henninger HB, Gerber Popp A. Humeral head osteotomy in shoulder arthroplasty: a comparison between anterosuperior and inferoanterior resection techniques. J Shoulder Elb Surg. 2017;26(2):343–51.

17 Anatomical Shoulder Arthroplasty: How to Manage Failure

Friso A. de Boer and Pol E. Huijsmans

17.1 Introduction

Over the last decade, there has been an increased use of anatomical total shoulder arthroplasty (TSA) for the treatment of glenohumeral osteoarthritis (OA). In the United States, 29,359 anatomical TSA were implanted in 2011 versus 21,692 reverse shoulder arthroplasty (RSA) [1]. Reported complication rate ranges from 10 to 23% [2]. Revision surgery is performed in 8–11% at long-term follow-up [3–5]. An annualized risk of 1.1–1.4% has been reported [6].

In this chapter, we first describe the reasons of failure of anatomical total shoulder arthroplasty, such as instability, rotator cuff failure, component loosening, fractures, and infections. Second, we discuss the most common solution for a failed anatomical prosthesis: conversion to RSA.

17.2 Instability

The reported incidence of instability (both dislocation and subluxation) after TSA is around 5% [2, 4]. Dislocation occurs most often anteriorly [2]. If an acute, complete dislocation occurs, reduction can be achieved by traction with the arm in neutral position. Generally, the patient should be put under general anesthesia to facilitate reduction. When reduction is confirmed and tested under fluoroscopy, the shoulder should be immobilized.

The success rate of closed reduction in cases of dislocation of anatomical TSA is unknown. However, after closed reduction of RSA followed by 6 weeks of immobilization, 62% remained stable after a mean follow-up of 2.3 years [7]. In the case of recurrent dislocations, revision surgery should be considered. Before revision surgery is performed, the cause of dislocation should be diagnosed. Possible causes could be divided into component related or soft tissue related. A differentiation between anterior and posterior instability can be made.

Radiographs or computed tomography (CT) scan can be used to evaluate loosening or malposition of the components; however, the latter could be difficult to interpret due to metal artifacts. Radiographs can show an indirect mark for rotator cuff lesions when anterior (subscapularis tear) or superior (supra-/infraspinatus tear) migration is present. Superior migration is classified according to the Torchia classification [8] as mild (>25% of the diameter of the humeral component), moderate (25–50%), or severe (>50%). Ultrasonography could be a more reliable imaging technique to assess the rotator cuff postoperatively, but shoulder ultrasonography depends highly on the experience of the interpreter [9–11]. Recent developments in metal reduction protocols

F. A. de Boer
Haga Hospital, The Hague, The Netherlands

P. E. Huijsmans (✉)
Bergman Clinics, Rijswijk, The Netherlands
e-mail: p.huijsmans@bergmanclinics.nl

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_17

enable the use of magnetic resonance imaging (MRI) to detect rotator cuff pathology [10].

Anterior instability is caused by humeral implant malrotation; glenoid malposition, wear, or loosening; oversized humeral head; soft-tissue imbalance (with the subscapularis being the principle cause); or deltoid dysfunction [12].

Posterior instability is generally caused by excessive retroversion of the prosthesis, due to either suboptimal placement or loosening, posterior capsule lesions, or severe type B or C glenoid wear according to the Walch's classification [13]. Intraoperatively, posterior grafting could be used to improve the version of the glenoid [2].

Physical therapy generally does not improve stability [2]. Revision using anatomic components is not a good option: 28% of success rate after revision (measured by recurrence of instability), with anterior instability having worse outcome than posterior instability [14]. When soft tissues are in good condition, neurological causes are excluded, and the instability is caused by malposition of a single component, revision of anatomical TSA could be considered. In any other case, for both anterior or posterior instability, revision by conversion to RSA is the most common solution [2, 4]. Successful outcome (measured by recurrence of instability) was reported in about 94% of the cases [15]. In case of persistent severe instability, arthrodesis could be a salvage option (Fig. 17.1).

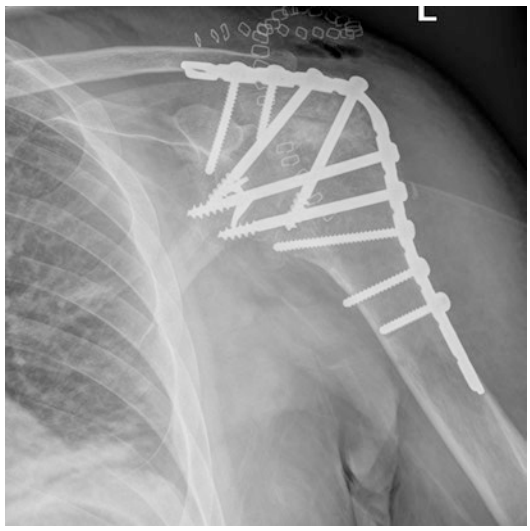

Fig. 17.1 Shoulder arthrodesis

17.3 Rotator Cuff Failure

The incidence of rotator cuff tears after TSA is 1.3–14.3%, most frequently involving the supraspinatus and/or infraspinatus tendon. It is usually a slowly progressive complication, more frequently found in series with long-term follow-up [2, 4, 16]. It should be noted that only a small part of patients with rotator cuff tears after TSA express clinical symptoms: in the above-cited series, revision rate because of cuff tears was only 1.2%. Symptoms can consist of pain, instability, and/or glenoid loosening. Subscapularis tears can cause anterior instability. Reporting on a cohort of patients with an anatomical TSA without symptoms, 74% had an intact subscapularis, and only 3% had a full-thickness tear on ultrasound examination after a minimum follow-up of 2 years [11]. In contrast, in patients who had revision arthroplasty for anterior instability, the subscapularis tendon was attenuated in all patients, and 79% had a full-thickness subscapularis tear [14]. Tears of the supraspinatus and/or infraspinatus can cause anterosuperior instability, which causes a changed biomechanical situation in the contact area between the glenoid and humeral components due to proximal migration of the humerus, which in turn accelerates insert wear and glenoid loosening through the so-called rocking-horse phenomenon [15, 16].

As described above, radiographs, ultrasonography, or MRI with metal reduction protocol can be used for assessment of the rotator cuff.

Secondary rotator cuff repair or reconstruction after TSA could be considered dependent on the type of lesion; however, reported results were not good [17, 18]. The only suitable solution is conversion to RSA [2, 4].

17.4 Component Loosening

17.4.1 Glenoid Loosening

Glenoid loosening is a relatively frequent complication of TSA [6] (Fig. 17.2). Reported survivorship of the glenoid component with revision for glenoid loosening as an endpoint was 98%

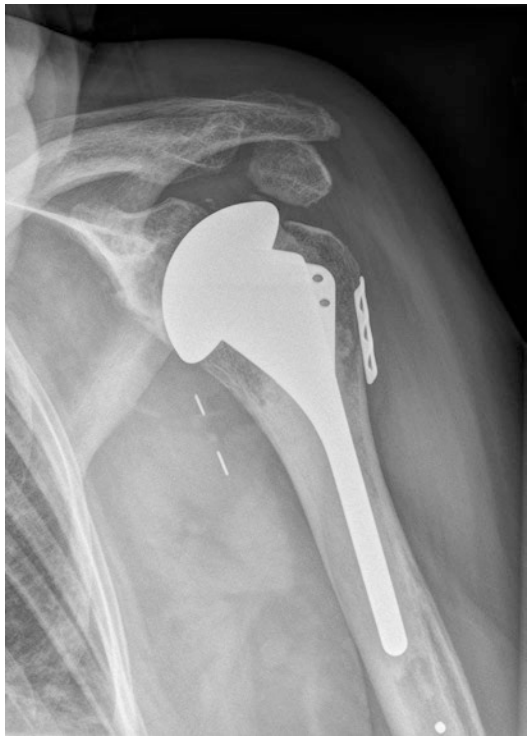

Fig. 17.2 Glenoid loosening with migration

after 5 years, but only 62.5% after 10 years [19]. The reported annualized rate of asymptomatic glenoid loosening on radiographs was 7.3% per year; 1.2% per year has symptomatic loosening, and 0.8% annually requires revision [6]. Another study described an incidence of 14.3% of glenoid loosening. Revision was performed in 28.5% of all loose implants [2].

Metal-backed implants have a higher risk of glenoid loosening and revision than all-polyethylene (PE) components [20]. Other reported complications associated with metal-backed implants are glenoid implant fracture, dissociations between PE and metal, and wear of both PE and metal (Fig. 17.3) [2]. The rate of symptomatic loosening is higher in female than in male [6]. Keeled components are more at risk for asymptomatic radiolucent lines than pegged components; however no difference in clinical outcome was found [6, 21].

Revision using anatomic components can only be considered when the cuff is sound and the head is centered on X-rays. It is important to

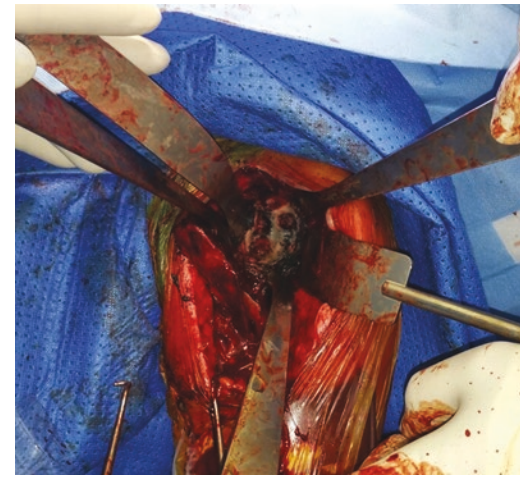

Fig. 17.3 Metal wear

assess the amount of bone loss. If the amount of bone loss is high, reimplantation of a glenoid component should be complemented by reconstruction of the glenoid using a bone graft. In cases of severe bone loss, it could be necessary to perform a two-stage revision with component removal and bone grafting performed in the first stage. After bone ingrowth, the final procedure takes place. With cuff deficiency or non-centered humeral head (suggesting cuff deficiency), RSA should be used for revision, also supported by a bone graft if necessary [4].

17.4.2 Humeral Loosening

The radiological diagnosis of humeral loosening is defined by a radiolucent line of more than 2 mm around the stem or migration of the implant. Humeral loosening is reported for 6–7% of shoulder arthroplasties, reporting both clinical and asymptomatic radiological cases [2, 22]. There is a greater risk of humeral loosening in cementless stems compared to cemented stems [2].

As for glenoid loosening, the choice between revisions with anatomical or reverse components depends on the condition of the rotator cuff and the centering of the humeral head on X-rays [4]. After removal of the humeral stem, the amount of bone stock and possible fractures

determine the revision options. Generally, the reimplanted stem should be cemented. A reversed prosthesis combined with a bone graft is necessary if the integrity of the metaphyseal bone is affected.

17.5 Fractures

17.5.1 Intraoperative Fractures

Reported incidence of intraoperative fractures is 2% [2]. This mostly concerns humerus fractures. Sutures can be used for isolated tuberosity fractures, cerclage wire for metaphyseal fractures around the stem, and plating for fractures distal to the humeral stem. Intraoperative glenoid fractures can be treated with screw or pin fixation, stabilization with the use of the glenoid implant or a bone graft, depending on the type of fracture. A preoperative CT scan should be performed for the evaluation of the bone stock and shape of the glenoid in order to prevent the occurrence of glenoid fractures due to poor drill orientation or placement [2]. Patient-specific drill guide might further improve glenoid preparation and prevent intraoperative glenoid-related complications [23].

17.5.2 Postoperative Fractures

Reported incidence of postoperative humerus fractures is 1% [2]. The choice between operative and nonoperative treatment is highly dependent on the type of fracture. Because of the risk of complications with operative treatment, this should be considered for fractures that are unstable or in the case of nonunion. The Wright and Cofield [24] classification differentiates three types of periprosthetic humerus fractures: at the tip of the component with proximal extension (type A), at the tip (type B), and distal to the tip (type C). According to Andersen et al. [25], the fixation of the stem is more important in decision-making than the fracture classification. They recommend operating displaced fractures, which could be treated with plate-and-screw fixation, or fractures around a loose stem, which could be treated by revision arthroplasty using a long humeral stem to bypass the fracture [25].

17.6 Infection

Similar to infections in other orthopedic implants, infections of TSA can be divided into early (<2 months), subacute (2 months to 1 year), or late infections (>1 year). In the shoulder, early infections are less common [26]. More frequently, late infections occur with a reported incidence of 0.9–4% [2, 4, 26, 27]. Infection of TSA is difficult to manage, due to limited bone stock in the shoulder, and treatment has inferior success rates than infections of hip or knee prostheses [26]. Most common microorganism species causing infections in shoulder arthroplasty are *Staphylococcus aureus*, coagulase-negative staphylococcus, and *Propionibacterium acnes* [2].

Early infections should be treated by debridement, antibiotics, and implant retention (DAIR). Changeable components should be changed. Subacute or late infections should be treated by implant revision in one stage or two stages bridged by temporary gentamicin beads or cement spacer (Fig. 17.4). Treatment of patients in coordination with an infectologist or microbiologist is important to optimize the choice of antibiotic therapy.

It is important to take into account the condition and demands of the patient. In elderly patients, whose condition does not allow large revision surgery, and in the absence of systemic infection, it could be considered to create a fistula to drain the shoulder with the implant left in situ, supported by lifelong antibiotic suppression therapy. It was also reported that a definitive treatment with a cement spacer leads to good or fair results in patients that do not tolerate additional surgery [28]. Different to hip arthroplasty,

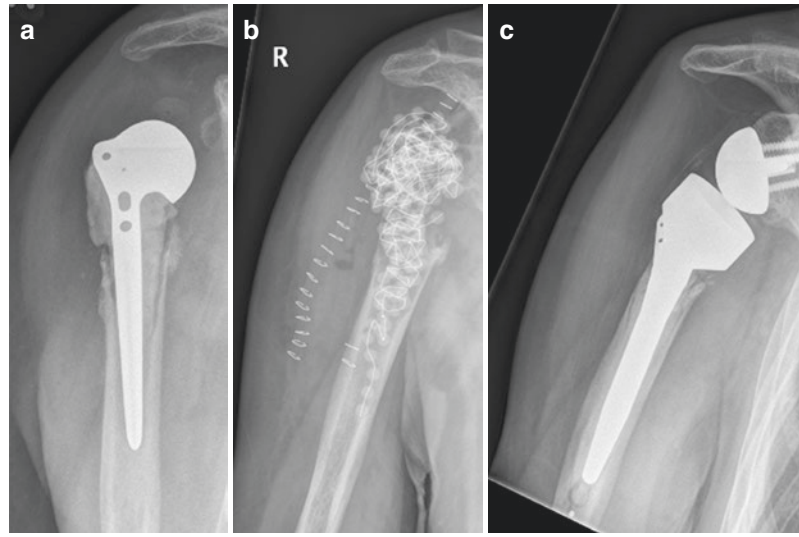

Fig. 17.4 (**a**–**c**) Late infection treated with two-stage procedure

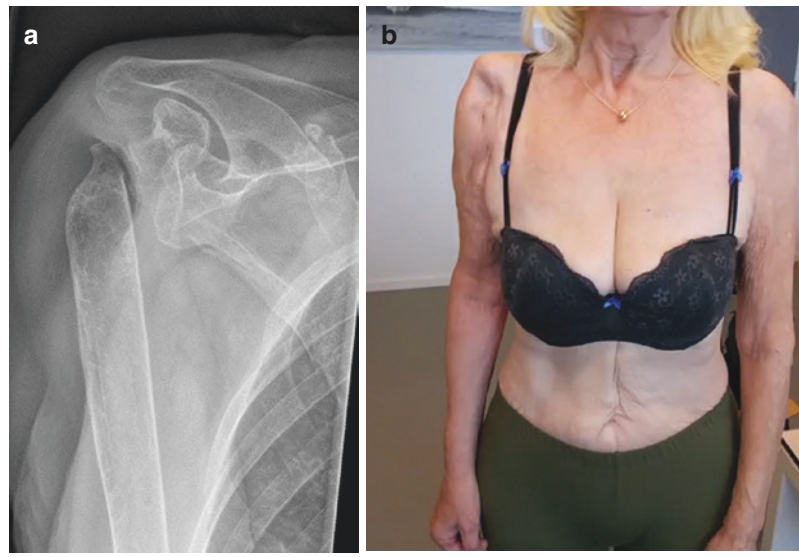

Fig. 17.5 (**a**, **b**) Girdlestone of the shoulder

removal of the implant ("Girdlestone" of the shoulder), even as salvage procedure, is an unattractive option and creates a very disabling condition (Fig. 17.5).

17.7 Neurological Injuries

Neurologic injuries were reported to have an incidence of 1.8%, mostly to the brachial plexus. The majority of cases comprise neurapraxia, which resolves spontaneously. The most important way to prevent is obviously to be careful during surgery and to avoid excessive traction and external rotation of the arm [29]. Damage to the axillary nerve leads to deltoid dysfunction, which causes bad results of the arthroplasty. It is a rare complication, reported with an incidence of 0.1% [2].

In the case of persistent severe neurologic deficit, arthrodesis could be the only surgical option.

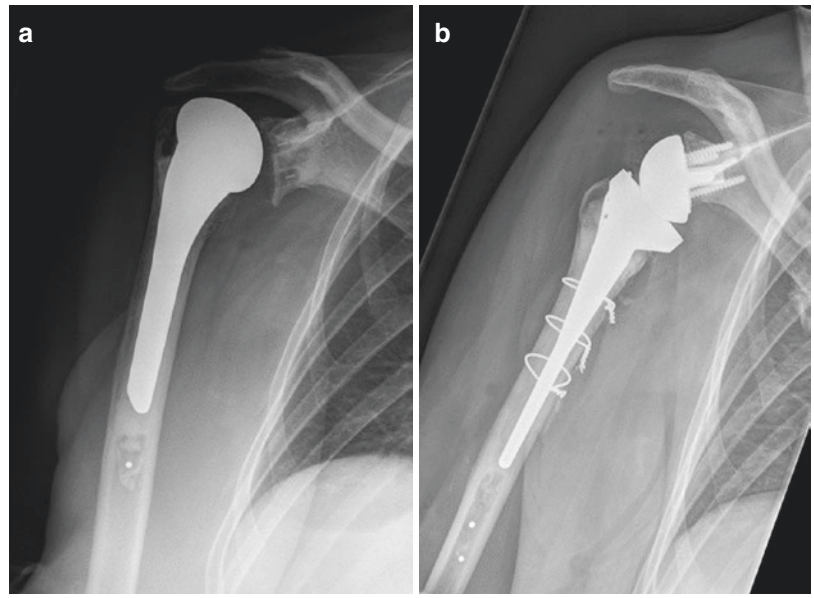

Fig. 17.6 (**a**, **b**) Revision using reverse prosthesis

17.8 Revision to RSA

Revision of TSA is associated to a higher complication rate and deterioration of clinical outcome. Patient-reported outcome after revision to RSA was significantly worse when initial indication for primary anatomic arthroplasty was a humerus fracture, compared to OA. Moreover, complication rate is higher for patients initially treated for fractures [30]. Having said that, improvement in range of motion, pain relief, and high patient satisfaction were reported after revision shoulder arthroplasty using a RSA [31–33]. Melis et al. [34] described a patient satisfaction of 86% and a mean Constant score of 55 in 37 cases of revision to RSA because of glenoid loosening in the presence of rotator cuff tears and/or instability. However, re-revision rate was quite high (21%). As noted before, the use of anatomic components in revision surgery usually leads to unsatisfactory results, and is indicated in selected cases only.

A major difficulty in revision surgery, whether using anatomic or reverse components, is removal of the humeral stem. Wall et al. [35] reported a 24% rate of humerus fractures during procedure. A window in the humeral cortex could aid in the removal of the stem. For coated proximal stems, a medial window can be used, an anterior window for other types of humeral stems [36]. The window should be closed by using cerclage wire (Fig. 17.6).

Over the last decade, prosthetic design has evolved to modular systems (i.e., SMR (Lima Corp, San Daniele del Friuli, Italy) and Ascend (Wright Medical Group, Memphis, TN, USA)) where a primary anatomical prosthesis can be converted to a reversed prosthesis without removal of the humeral stem (Fig. 17.7). With the modular systems, it is possible to replace the humeral head sphere with a reversed component when revision surgery is necessary, which is an easier procedure for the surgeon and for the patient. Good clinical outcomes were reported at medium-term follow-up [3].

Removal of the glenoid can be difficult; therefore, sometimes it can be easier to divide the component in parts using an osteotome. Care must be taken at all times to preserve the bone of the glenoid and to remove the cement as much as possible.

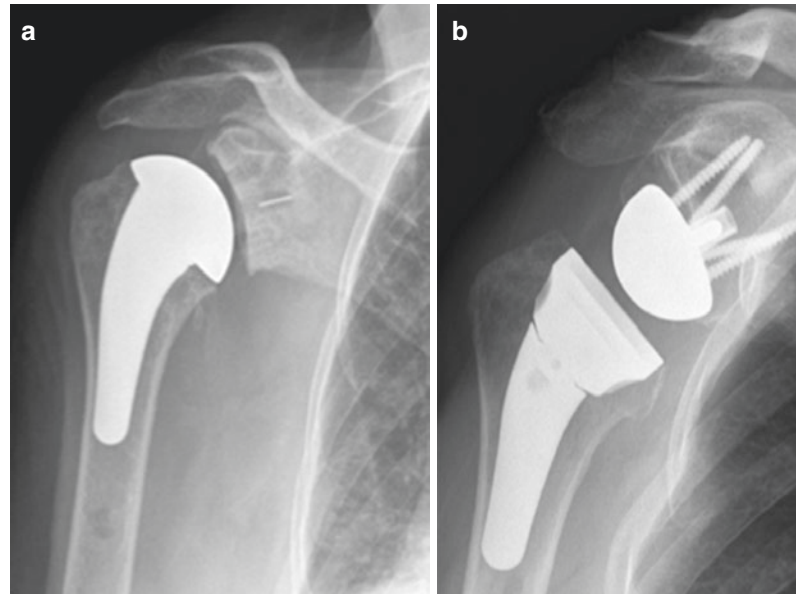

Fig. 17.7 (**a**, **b**) Modular system (Ascend)

References

1. Westermann RW, Pugely AJ, Martin CT, Gao Y, Wolf BR, Hettrich CM. Reverse shoulder arthroplasty in the United States: a comparison of National Volume, patient demographics, complications, and surgical indications. Iowa Orthop J. 2015;35:1–7.
2. Gonzalez JF, Alami GB, Baque F, Walch G, Boileau P. Complications of unconstrained shoulder prostheses. J Shoulder Elb Surg. 2011;20(4):666–82.
3. Castagna A, Delcogliano M, De Caro F, Ziveri G, Borroni M, Gumina S, Postacchini F, De Biase CF. Conversion of shoulder arthroplasty to reverse implants: clinical and radiological results using a modular stem. Int Orthop. 2013;37(7):1297–305.
4. Favard L. Revision of total shoulder arthroplasty. Orthop Traumatol Surg Res. 2013;99(1 Suppl):S12–21. https://doi.org/10.1016/j.otsr.2012.11.010.
5. Habermeyer P, Magosch P. Strategies in revision shoulder arthroplasty. Orthopade. 2013;42(7):542–51. https://doi.org/10.1007/s00132-012-2025-5.
6. Papadonikolakis A, Neradilek MB, Matsen FA. Failure of the glenoid component in anatomic total shoulder arthroplasty: a systematic review of the English-language literature between 2006 and 2012. J Bone Joint Surg Am. 2013;95(24):2205–12.
7. Teusink MJ, Pappou IP, Schwartz DG, Cottrell BJ, Frankle MA. Results of closed management of acute dislocation after reverse shoulder arthroplasty. J Schoulder Elbow Surg. 2015;24(4):621–7.
8. Torchia ME, Cofield RH, Settergren CR. Total shoulder arthroplasty with the Neer prosthesis: long-term results. J Shoulder Elb Surg. 1997;6(6):495–505.
9. Fischer CA, Weber MA, Neubecker C, Bruckner T, Tanner M, Zeifang F. Ultrasound vs. MRI in the assessment of rotator cuff structure prior to shoulder arthroplasty. J Orthop. 2015;12(1):23–30.
10. Gyftopoulos S, Rosenberg ZS, Roberts CC, Bencardino JT, Appel M, Baccei SJ, et al. ACR appropriateness criteria imaging after shoulder arthroplasty. J Am Coll Radiol. 2016;13(11):1324–36.
11. Scalise JJ, Ciccone J, Iannotti JP. Clinical, radiographic, and ultrasonographic comparison of subscapularis tenotomy and lesser tuberosity osteotomy for total shoulder arthroplasty. J Bone Joint Surg Am. 2010;92(7):1627–34.
12. Moeckel BH, Altchek DW, Warren RF, Wickiewicz TL, Dines DM. Instability of the shoulder after arthroplasty. J Bone Joint Surg Am. 1993;75(4):492–7.
13. Walch G, Boulahia A, Boileau P, Kempf JF. Primary glenohumeral osteoarthritis: clinical and radiographic classification. The Aequalis Group. Acta Orthop Belg. 1998;64S2:46–52.
14. Sanchez-Sotelo J, Sperling JW, Rowland CM, Cofield RH. Instability after shoulder arthroplasty: results of surgical treatment. J Bone Joint Surg Am. 2003;85-A(4):622–31.
15. Levy DM, Abrams GD, Harris JD, Bach BR Jr, Nicholson GP, Romeo AA. Rotator cuff tears after total shoulder arthroplasty in primary osteoarthritis: a systematic review. Int J Shoulder Surg. 2016;10(2):78–84.

16. Franklin JL, Barrett WP, Jackins SE, Matsen FA. Glenoid loosening in total shoulder arthroplasty. Association with rotator cuff deficiency. J Arthroplast. 1988;3(1):39–46.
17. Miller BS, Joseph TA, Noonan TJ, Horan MP, Hawkins RJ. Rupture of the subscapularis tendon after shoulder arthroplasty: diagnosis, treatment, and outcome. J Shoulder Elb Surg. 2005;14(5):492–6.
18. Shields E, Ho A, Wiater JM. Management of the subscapularis tendon during total shoulder arthroplasty. J Shoulder Elb Surg. 2017;26(4):723–31.
19. Denard PJ, Raiss P, Sowa B, Walch G. Mid- to long-term follow-up of total shoulder arthroplasty using a keeled glenoid in young adults with primary glenohumeral arthritis. J Shoulder Elb Surg. 2013;22(7):894–900.
20. Papadonikolakis A, Matsen FA. Metal-backed glenoid components have a higher rate of failure and fail by different modes in comparison with all-polyethylene components: a systematic review. J Bone Joint Surg Am. 2014;96(12):1041–7.
21. Throckmorton TW, Zarkadas PC, Sperling JW, Cofield RH. Pegged versus keeled glenoid components in total shoulder arthroplasty. J Shoulder Elb Surg. 2010;19(5):726–33.
22. Bohsali KI, Wirth MA, Rockwood CA Jr. Complications of total shoulder arthroplasty. J Bone Joint Surg Am. 2006;88(10):2279–92.
23. Iannotti JP, Weiner S, Rodriguez E, Subhas N, Patterson TE, Jun BJ, Ricchetti ET. Three-dimensional imaging and templating improve glenoid implant positioning. J Bone Joint Surg Am. 2015;97(8):651–8.
24. Wright TW, Cofield RH. Humeral fractures after shoulder arthroplasty. J Bone Joint Surg Am. 1995;77(9):1340–6.
25. Andersen JR, Williams CD, Cain R, Mighell M, Frankle M. Surgically treated humeral shaft fractures following shoulder arthroplasty. J Bone Joint Surg Am. 2013;95(1):9–18.
26. 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.
27. Weber P, Utzschneider S, Sadoghi P, Andress HJ, Jansson V, Müller PE. Management of the infected shoulder prosthesis: a retrospective analysis and review of the literature. Int Orthop. 2011;35(3):365–73.
28. Mahure SA, Mollon B, Yu S, Kwon YW, Zuckerman JD. Definitive treatment of infected shoulder arthroplasty with a cement spacer. Orthopedics. 2016;39(5):e924–30.
29. Parisien RL, Yi PH, Hou L, Li X, Jawa A. The risk of nerve injury during anatomical and reverse total shoulder arthroplasty: an intraoperative neuromonitoring study. J Shoulder Elb Surg. 2016;25(7):1122–7.
30. Holschen M, Franetzki B, Witt KA, Liem D, Steinbeck J. Conversions from anatomic shoulder replacements to reverse total shoulder arthroplasty: do the indications for initial surgery influence the clinical outcome after revision surgery? Arch Orthop Trauma Surg. 2017;137(2):167–72.
31. Abdel MP, Hattrup SJ, Sperling JW, Cofield RH, Kreofsky CR, Sanchez-Sotelo J. Revision of an unstable hemiarthroplasty or anatomical total shoulder replacement using a reverse design prosthesis. Bone Joint J. 2013;95-B(5):668–72.
32. Kelly JD, Zhao JX, Hobgood ER, Norris TR. Clinical results of revision shoulder arthroplasty using the reverse prosthesis. J Shoulder Elb Surg. 2012;21(11):1516–25.
33. Patel DN, Young B, Onyekwelu I, Zuckerman JD, Kwon YW. Reverse total shoulder arthroplasty for failed shoulder arthroplasty. J Shoulder Elb Surg. 2012;21(11):1478–83.
34. Melis B, Bonneviale N, Neyton L, Lévigne C, Favard L, Walch G, Boileau P. 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(3):342–9.
35. 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.
36. Sperling JW, Cofield RH. Humeral windows in revision shoulder arthroplasty. J Shoulder Elb Surg. 2005;14(3):258–63.

18 Failed Anatomical Shoulder Arthroplasty: Case Example 1

Radovan Mihelic and Zdravko Jotanovic

18.1 Introduction

Four-part fractures in elderly patients are often treated by shoulder arthroplasty. Depending on the status of the greater tuberosity, anatomical or reverse shoulder prosthesis can be used. If the anatomical prosthesis is used, the greater tuberosity must be fixed to the implant or to the humeral shaft in order to enable normal function of the rotator cuff. Non-healing of the greater tuberosity will signify failed function of the shoulder.

18.2 Case Presentation

An 85-year-old female came to our attention with a four-part proximal humerus fracture in June 2010 (Fig. 18.1). After the clinical exam and X-ray imaging, cemented humeral hemiarthroplasty (HA) was indicated. At that time, our hospital had a special HA equipped with a small plate, which was added to the implant for the fixation of the greater tuberosity (Fig. 18.2). At the time of surgery, the supraspinatus insertion was intact; thus, after cementing the prosthetic stem into the humerus, the greater tuberosity was fixed with the plate and nonabsorbable sutures. The lesser tuberosity was removed and biceps tenotomy was performed. After surgery, arm immobilization into a sling was maintained for 6 weeks followed by a cautious rehabilitative program.

Clinical evaluation at 9 months revealed a poor function of the operated shoulder, with 45° of abduction and no internal and external rotation.

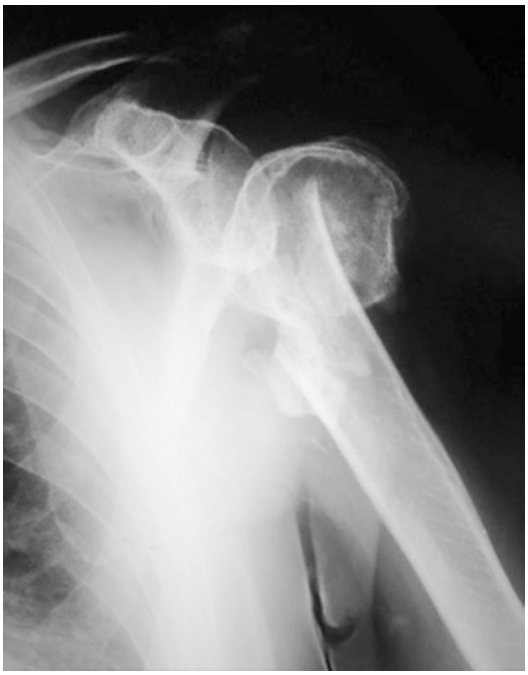

Fig. 18.1 Four-part fracture of the proximal humerus

R. Mihelic (✉) · Z. Jotanovic
Orthopaedic Clinic, University of Lovran,
Lovran, Croatia

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_18

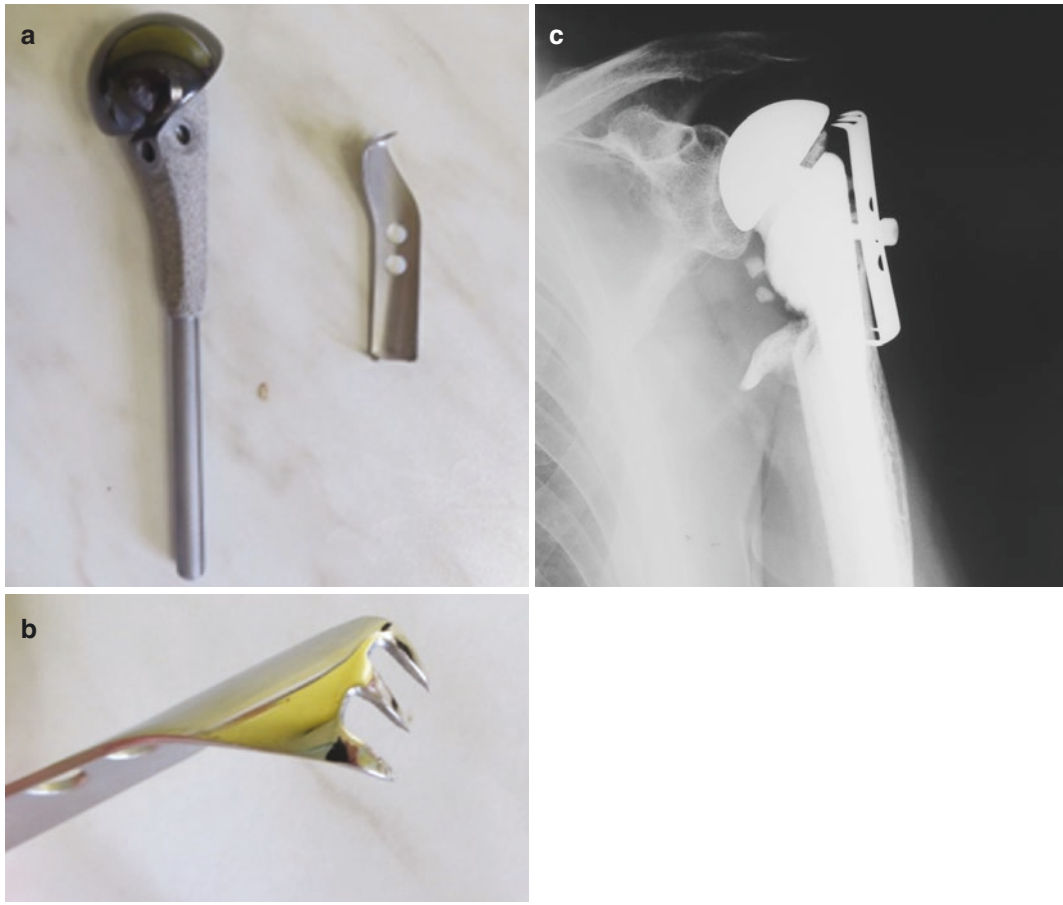

Fig. 18.2 Humeral hemiarthroplasty (**a**). Dorsal plate for the greater tuberosity fixation (**b**). Postoperative X-ray (**c**)

The patients suffered from severe pain to the shoulder irradiated to the cervical region. Radiographic exams revealed superior migration of the prosthesis and resorption of the greater tuberosity. It was evident that the plate caused the rupture of rotator cuff.

In March 2011, revision surgery was performed to remove the plate. We found rupture of the supraspinatus and subscapularis tendons and anterior-superior dislocation of the implant. The deltoid muscle was absolutely atrophic and scarred.

A surgical option was to perform HA removal by an extensive humerus split and implantation of a reverse shoulder arthroplasty (RSA). However, in considering the potential morbidity of the planned surgical procedure, the patient's general health status, and the unpredictability of surgical outcome due to the atrophy of deltoid muscle, we opted for conservative treatment. The patient's accepted loss of function of her shoulder and symptoms were managed with painkillers. The prosthesis remained anteriorly dislocated (Fig. 18.3).

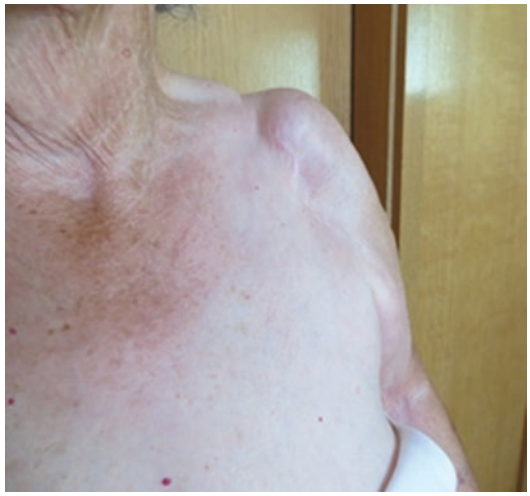

Fig. 18.3 Displaced prosthesis at the anterior aspect of the shoulder

Conclusion

This case illustrates that although the supraspinatus was intact at the time of first surgery, its late rupture caused HA failure. We should consider immediate implantation of a RSA would have probably given better and more durable results.

19 Failed Anatomical Shoulder Arthroplasty: Case Example 2

Ladislav Kovacic

19.1 Introduction

Postoperative rotator cuff tear (RCT) is recognized as the fourth most frequent complication of total shoulder arthroplasty (TSA), its prevalence ranging between 1.3 and 7.8% [1–4]. Symptomatic anterosuperior and posterosuperior RCTs after anatomic TSA may require conversion to reverse shoulder arthroplasty (RSA), as studies have demonstrated unsatisfactory results when rotator cuff repair after the index procedure has been attempted [3, 5, 6].

The problem of rotator cuff dysfunction is even more important in fracture hemiarthroplasty (HA), because the results of treatment are inferior to those reported for TSA in osteoarthritis. Complication rate in fracture HA rises up to 64% [7–10]. Such outcome is mainly due to complications related to healing of the tuberosities such as nonunion and malunion accompanied with rotator cuff dysfunction.

Shoulder replacement fails when it does not achieve the expectations of the patient and the surgeon. Painful pseudoparalysis of the shoulder may be a reason to consider revision surgery with RSA. In some instances, modular shoulder prostheses allow removal of the humeral head component and, by gaining access to the glenoid, give the chance for replacement with reverse components. However, revision often requires removal of the entire humeral component, which can be extremely challenging. If longitudinal humeral osteotomy along the bicipital groove does not dislodge the prosthesis, pectoralis major pedicled cortical bone window osteotomy can be used. This is the approach used in the case of the present chapter.

19.2 Case Presentation

A 50-year-old female had a comminuted proximal humerus fracture in her right dominant shoulder 1.5 years ago and was treated with a HA. Since the operative procedure, she experienced pain and limited shoulder function. She had difficulties in performing activities of daily living and she was not able to return to work. Passive range of motion (ROM) was substantially limited. External rotation with the arm at the side was 0°, and passive abduction and forward flexion were 40°. Active ROM was limited as well. Active abduction reached 30° and active forward flexion was 40°. She was not able to reach the head with the affected hand.

X-ray of the shoulder revealed superior migration of the prosthesis and reduced acromiohumeral distance (Fig. 19.1). Partial resorption of the greater tuberosity was also seen. Computed

L. Kovacic
Department of Traumatology, University Medical Centre Ljubljana, Ljubljana, Slovenia

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_19

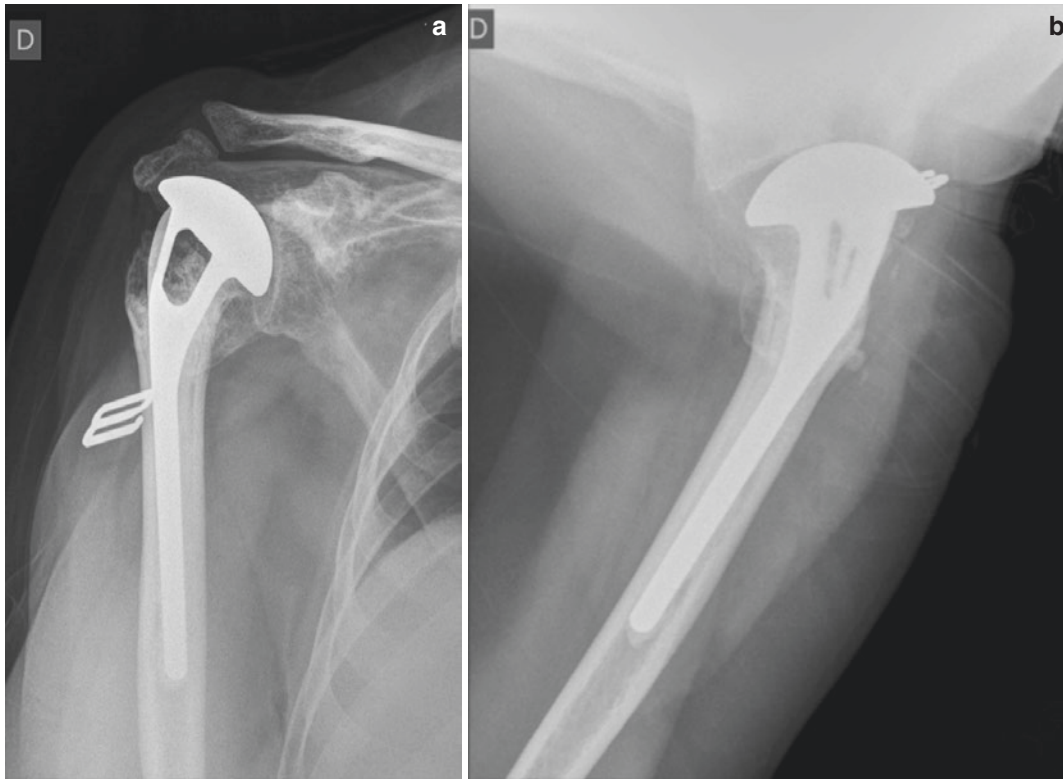

Fig. 19.1 Anteroposterior (**a**) and axillary (**b**) radiographic views of shoulder HA. Images reveal superior migration of the prosthesis, reduced acromio-humeral distance, and partial resorption of the greater tuberosity

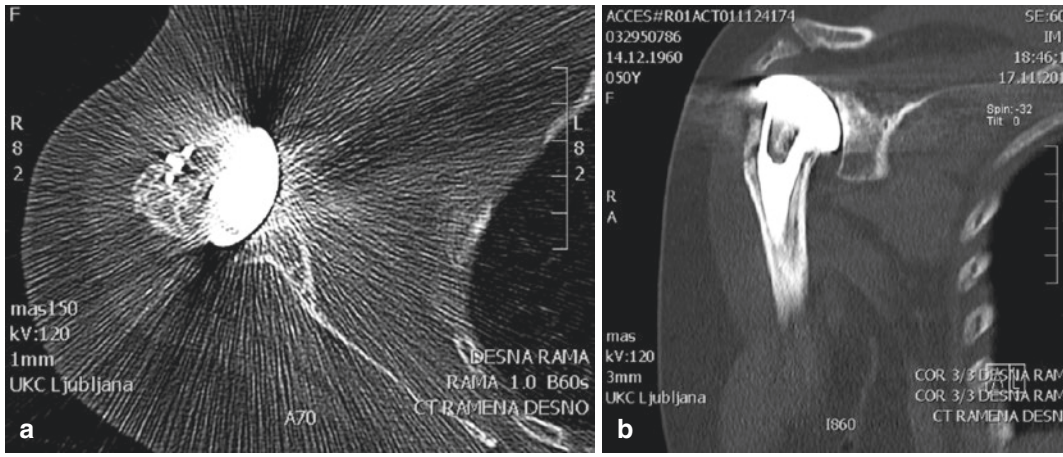

Fig. 19.2 Axial (**a**) and coronal (**b**) CT scans of shoulder HA. Remnants of the tuberosities are in nonanatomical position

tomography (CT) scan revealed the remnant of the greater and lesser tuberosity in nonanatomical position (Fig. 19.2).

As non-operative treatment was unsuccessful and ROM remained limited despite a long rehabilitation program, it was proposed to improve

her shoulder function with surgical procedure. Because of her age, arthroscopic capsulotomy was planned and performed. Surgical procedure revealed very narrow glenohumeral joint space and contracted rotator cuff with scar tissue in the subacromial space. Anterior and posterior rotator cuff was present, but the supraspinatus tendon showed significant atrophy and a full-thickness tear. Arthroscopic release resulted in improved passive ROM. Passive abduction and forward flexion were restored to 80° after surgical procedure, but as expected there was no improvement in active ROM, and the patient was not able to maintain the arm in abducted position. Furthermore, strength in abduction and external rotation was diminished.

Because of the moderate pain and functional deficit in the shoulder with absent active ROM, further surgical treatment was proposed. Thus, it was decided to treat rotator cuff insufficiency with RSA.

19.2.1 Surgical Technique

Patient was positioned in standard beach-chair position with the arm freely mobile at the edge of the operating table. The arm was draped in such a way to allow distal extension of the surgical approach if needed in the case of intraoperative complications. Deltopectoral approach was performed. Identification of deltopectoral groove is sometimes difficult due to changed anatomy following previous surgery. The cephalic vein may not be present in every case. Coracoid process and conjoined tendon serve as a landmark. Careful dissection and scar tissue release are necessary. Deltoid muscle was mobilized and proximal part of the humerus was exposed. Subacromial space was carefully released and rotator cuff remnants were exposed. Any rotator cuff residues should be preserved if possible. Teres minor tendon is frequently still present and intact. When dissection is performed medially, axillary nerve should be exposed and protected. Performing the dissection, several tissue samples are taken for microbiology examination.

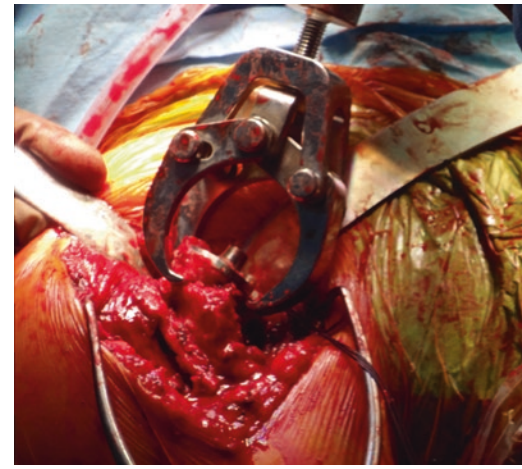

Fig. 19.3 Attempt to extract the humeral stem with extraction set

After dissection of the subacromial space and rotator cuff, dislocation of the proximal humerus with prosthesis in situ was possible. Further exposure of the head was necessary to be able to remove the prosthetic head component. Accessible cement at the level of former metaphysis was extracted. Interface between the proximal implant and bone was dissected with small chisels. An attempt to extract the implant with extraction set was then performed (Fig. 19.3). As the shoulder prosthesis could not be extracted, the transhumeral approach was necessary.

To perform a humeral window, further dissection of the humeral diaphysis in distal direction was achieved. Humerus was exposed subperiosteally along the intertubercular groove. Pectoralis major insertion was preserved, but blunt dissection around the insertion was done, and pectoralis muscle was retracted with a suture loop. Anterior humeral window was performed on the lateral aspect of the humerus. The cutting line was marked with drill holes using a 2-mm K wire. At the level of the tip of the stem, there was no metal resistance, thus indicating sufficient length of osteotomy. The bone was cut laterally with an oscillating saw, and the medial cut was then performed parallel to the previous one. Both cuts were connected at the distal level. Thereafter, the pectoralis major pedicled cortical bone window was loosened with chisels and retracted medially

(Fig. 19.4). The prosthesis was further loosened by chiseling the cement and finally extracted (Fig. 19.5). Cement remnants were removed from the bone as well as residual cement plug. Care was taken to avoid violating the thin humeral cortex.

Osteotomy of the humerus gives short anchorage for the revision implant; therefore, stems longer than 150 mm are often necessary. Appropriate planning and sufficient collection of the available stems are important. In the revision cases, where soft tissues are frequently inadequate, positioning of the implant is of outmost importance to reduce possible complications. Reconstruction of the humeral height, appropriate tension of the soft tissue, and correct retroversion are the issues that should be addressed very carefully. Humeral diaphysis and proximal humerus should be reconstructed as much as possible.

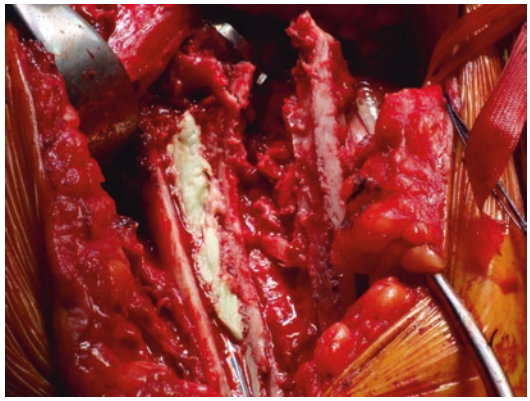

Fig. 19.4 Pectoralis major pedicled cortical bone window opened and retracted medially revealing cemented humeral stem in the humeral diaphysis

Wires and strong sutures were placed around the humeral shaft and proximal humerus for refixation of the bone window. At this stage, humerus was protected with provisional stem allowing safe glenoid preparation. The glenoid was exposed, bone stock quality was evaluated, and contact surface was reamed and prepared to receive the glenoid baseplate, and the glenosphere was then fixed.

Next step was cementing the humeral component. Due to usually very narrow medullar canal, coating of the humeral component with cement is advisable to have good distribution of the cement through the whole length. The humeral implant

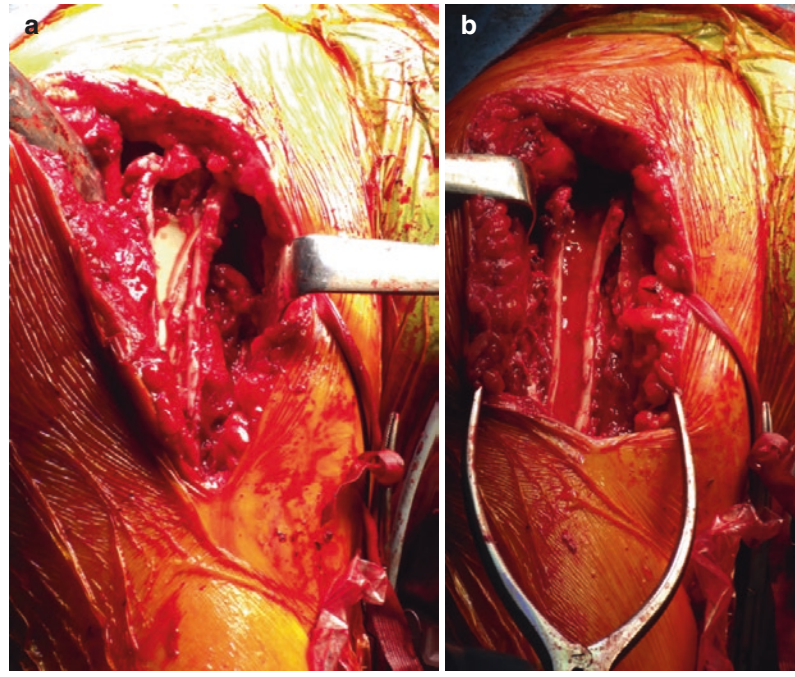

Fig. 19.5 Humeral diaphysis with extracted humeral prosthetic stem. (**a**) Remnants of cement are still present in the humeral canal. (**b**) All the remnants of the cement are extracted

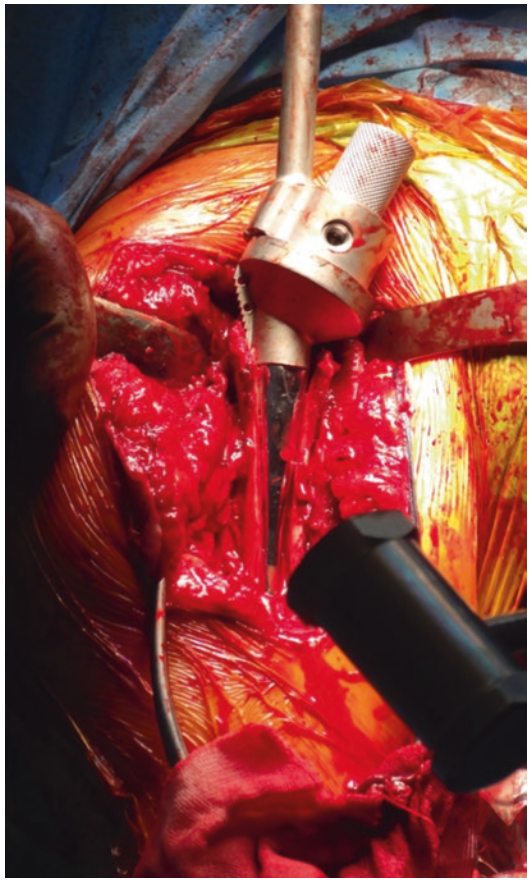

Fig. 19.6 Cemented humeral stem implanted at the position respecting humeral height and version

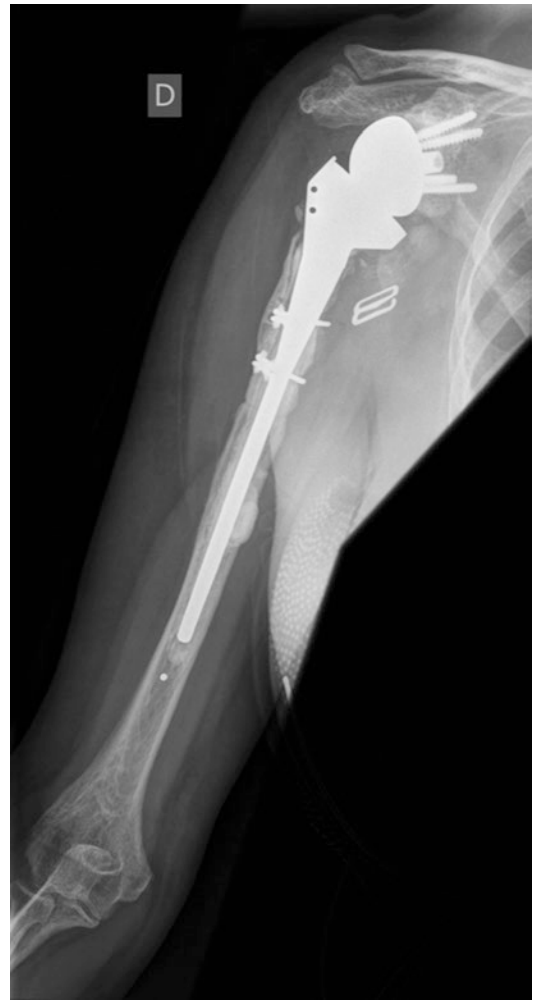

Fig. 19.7 Revision RSA. Long cemented stem is used to achieve adequate anchorage of the prosthesis in the humeral diaphysis. Cortical bone window is reduced and closed with cerclage wires

was cemented by respecting the planned height and version (Fig. 19.6). The humeral window was closed using reduction forceps and previously positioned wires and sutures cerclages. The stability of prosthesis was judged by trial components of the final liner. Often, stability of the prosthesis should be determined before the bone window closure. Fixation of the stem was reinforced by cement coating around the proximal part of the prosthetic stem. The residual rotator cuff was reattached and final intraoperative ROM and stability were tested. Postoperative X-ray was performed to confirm adequate implant placement, stability of the prosthesis and closure of the humeral window (Fig. 19.7).

After postoperative rehabilitation, functional result was satisfactory. The shoulder was stable and active ROM increased (Fig. 19.8). The patient had no pain in the shoulder, and she was able to use her arm for majority of daily activities. However, some limitations were still present including lifting and holding heavy objects.

19.3 Discussion

Revision RSA for the treatment of failed anatomic shoulder arthroplasty has become relatively frequent only in recent years. Only a few articles describing results of such a procedure are available [11–14]. The primary indication for

Fig. 19.8 Patient with revision RSA on her right dominant shoulder and range of motion gained with rehabilitation in abduction (**a**), forward flexion (**b**), and external rotation (**c**)

revision surgery is painful shoulder with loss of function after hemi- or total shoulder arthroplasty. To date, there are no other satisfactory surgical treatment options available for those patients. Available data suggest that this revision surgery is associated with reduction of pain and improvement of functional outcomes. However, improvement is at best approximately 65% of the normal ROM, and the mean Constant scores are considerably lower than those observed after primary RSA and below the normative scores, ranging from 44.2 to 56 [11–14]. Moreover, revision rate ranged from 13 to 42%, and the overall complication rate after revision RSA has been reported to be as high as 62%, which is much higher than that of primary RSA [15].

The complexity of shoulder revision arthroplasty is related to several factors, such as dealing with osteopenic bone, determining appropriate length and version of the prosthetic components, and gaining sufficient soft tissue tension in order to provide adequate stability and function of the implant.

Extraction of the humeral stem is rarely straightforward and can be very challenging. Usually, the bone of the humerus is thin, osteopenic and extremely fragile, so there is a great risk of fracturing humeral shaft and loosing bone substance. Modular systems may permit retention of humeral stem and help to minimize complications related to extraction of the stem. However, this is not always the case. Different techniques of stem

removal have been described in the literature. It is reasonable to try with the simplest method like axial blows and in the case of well-fixed humeral stem progress to complex procedure, such as axial osteotomy and even pectoralis major pedicled cortical window osteotomy [16]. The main objective is to avoid intraoperative fracture and maintain appropriate fit of revision stem.

The second major problem is appropriate positioning of the revision prosthesis regarding the height and version, thus achieving correct tensioning of the deltoid and soft tissue balancing. Excessive lengthening of the humerus may result in pain, neurologic complication, abduction contractures, early baseplate loosening, scapular spine fatigue fractures, and even instability. On the contrary, improper tensioning of the deltoid may lead to prosthetic instability. Furthermore, when choosing the right retroversion, an acceptable balance must be found between the need for stable joint closure and abutment at the glenoid rim in full rotation. If in doubt, greater retroversion should be preferred where there is a risk of dislocations due to contractures and muscle defects, keeping in mind that this will reduce internal rotation.

In every case of revision arthroplasty, tissue biopsies and joint fluid aspiration are recommended to rule out infection. Cultures taken at revision surgery are often positive even if the patients are presumed to be uninfected. The most common pathogen is *Propionibacterium acnes*, which is of indolent nature and not always easy to identify [17].

Conclusion

Failed shoulder arthroplasty is a challenging clinical condition, which can be solved with revision RSA. Its use can reduce pain and improve function, thus improving patients' quality of life. However, clinical outcomes are clearly less predictable, and complications and revision rates are higher than in patients who undergo a primary shoulder replacement procedure. When a well-fixed humeral stem must be replaced, it can be safely extracted using pectoralis major pedicled cortical window, thus reducing the possibility for intraoperative complications.

References

1. Bohsali KI, Wirth MA, Rockwood CA Jr. Complications of total shoulder arthroplasty. J Bone Joint Surg Am. 2006;88:2279–92.
2. Chin PY, Sperling JW, Cofield RH, Schleck C. Complications of total shoulder arthroplasty: are they fewer or different? J Shoulder Elb Surg. 2006;15:19–22.
3. Young AA, Walch G, Pape G, Gohlke F, Favard L. Secondary rotator cuff dysfunction following total shoulder arthroplasty for primary glenohumeral osteoarthritis: results of a multicenter study with more than five years of follow-up. J Bone Joint Surg Am. 2012;18:685–93.
4. Bohsali KI, Bois AJ, Wirth MA. Complications of shoulder arthroplasty. J Bone Joint Surg Am. 2017;99:256–69.
5. Hattrup SJ, Cofield RH, Cha SS. Rotator cuff repair after shoulder replacement. J Shoulder Elb Surg. 2006;15:78–83.
6. Abdel MP, Hattrup SJ, Sperling JW, Cofield RH, Kreofsky CR, Sanchez-Sotelo J. Revision of an unstable hemiarthroplasty or anatomical total shoulder replacement using a reverse design prosthesis. Bone Joint J. 2013;95-B:668–72.
7. Boileau P, Krishnan SG, Tinsi L, Walch G, Coste JS, Mole D. Tuberosity malposition and migration: reasons for poor outcomes after hemiarthroplasty for displaced fractures of proximal humerus. J Shoulder Elb Surg. 2002;11:401–12.
8. Kralinger F, Schweiger R, Wambacher M, Farrell E, Menth-Chiari W, Lajtai G, Hübner C, Resch H. Outcome after primary hemiarthroplasty for fracture of the head of the humerus. A retrospective multicenter study of 167 patients. J Bone Joint Surg Br. 2004;86:217–9.
9. Gronhagen CM, Abbaszadegan H, Revay SA, Adolphson PY. Medium-term results after primary hemiarthroplasty for comminuted proximal humerus fractures: a study of 46 patients followed up for an average of 4.4 years. J Shoulder Elb Surg. 2007;16:766–73.
10. Kovacic L, Stefanic F, Senekovic V. Clinical and radiological outcome in low profile metaphyseal shoulder fracture prosthesis design. In: 11th European Congress of Trauma and Emergency Surgery. Brussels, Belgium; 2010.
11. Levy J, Frankle M, Mighell M, Pupello D. The use of the reverse shoulder prosthesis for the treatment of failed hemiarthroplasty for proximal humeral fracture. J Bone Joint Surg Am. 2007;89:292–300.
12. Walker M, Willis MP, Brooks JP, Pupello D, Mulieri PJ, Frankle MA. The use of the reverse shoulder arthroplasty for treatment of failed shoulder arthroplasty. J Shoulder Elb Surg. 2012;21:514–22.
13. Patel DN, Young B, Onvekwelu I, Zuckerman JD, Kwon YW. Reverse total shoulder arthroplasty for failed shoulder arthroplasty. J Shoulder Elb Surg. 2012;21:1478–83.

14. Ortmaier R, Resch H, Matis N, Blocher M, Auffarth A, Mayer M, Hitzl W, Tauber M. Reverse shoulder arthroplasty in revision of failed shoulder arthroplasty – outcome and follow-up. Int Orthop. 2013;37:67–75.
15. Randelli P, Randelli F, Compagnoni R, Cabitza P, Ragone V, Pulici L, Banfi G. Revision reverse shoulder arthroplasty in failed shoulder arthroplasties for rotator cuff deficiency. Joints. 2015;3:31–7.
16. Gohlke F, Rolf O. Revision of failed fracture hemiarthroplasties to reverse total shoulder prosthesis through the transhumeral approach: method incorporating a pectoralis-major-pedicled bone window. Oper Orthop Traumatol. 2007;19:185–208.
17. Kelly JD 2nd, Hobgood ER. Positive culture rate in revision shoulder arthroplasty. Clin Orthop Relat Res. 2009;467:2343–8.

Failed Anatomical Shoulder Arthroplasty: Case Example 3

20

Pietro Simone Randelli, Chiara Fossati, and Alessandra Menon

20.1 Case Presentation

A 69-year-old man reported a right shoulder injury after an accidental fall from a standing height in November 2015.

Initial standard radiological examination (Fig. 20.1) and computed tomography (CT) scan revealed a multifragmentary four-part proximal humerus fracture.

The patient was right dominant, retired, and not involved in any manual labour. He had received a pacemaker implantation as only previous surgical procedure. Neither smoking habits nor other comorbidities as diabetes mellitus or neurological diseases were reported.

The patient initially referred to another institution, where a modular reverse shoulder arthroplasty (RSA) (SMR™, Lima Corporate, Villanova di San Daniele, UD, Italy) was implanted through a standard deltopectoral approach (Fig. 20.2). No intraoperative complications were reported, and the patient started a regular rehabilitation according to institution's internal protocol the day after surgery.

One week after surgery, the patient referred sudden onset of acute right shoulder pain after a trivial movement of the operated shoulder, followed by a complete loss of shoulder function. Standard radiographs taken in the emergency department revealed an anterior dislocation of the implant. Three sequential attempts of closed reduction without anaesthesia were performed, but implant stability was reached for no longer than few minutes after every attempt.

The patient was discharged and he referred to our institution 2 months after the index procedure. Local findings at physical examination included a visible deformity of the anterior and lateral shoulder profile, diffuse swelling and warmth, erythema overlying the anterior part of the right shoulder, tenderness to palpation and pain provoked by passive mobilization of the glenohumeral joint (Fig. 20.3). Active and passive range of motion were limited to minimal abduction and flexion.

The diagnosis of a chronic painful unreduced dislocation of the RSA was confirmed by standard radiological examination.

On considering the patient's history and the local inflammatory findings, the diagnostic workup included also a CT scan and a bone scan. The latter revealed accumulation of the radionu-

P. S. Randelli (✉) · C. Fossati
Laboratory of Applied Biomechanics, Department of Biomedical Sciences for Health, University of Milan, Milan, Italy

1° Clinica Ortopedica, ASST Centro Specialistico Ortopedico Traumatologico Gaetano Pini-CTO, Milan, Italy
e-mail: pietro.randelli@unimi.it

A. Menon (✉)
1° Clinica Ortopedica, ASST Centro Specialistico Ortopedico Traumatologico Gaetano Pini-CTO, Milan, Italy
e-mail: Alessandra.Menon@asst-pini-cto.it

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_20

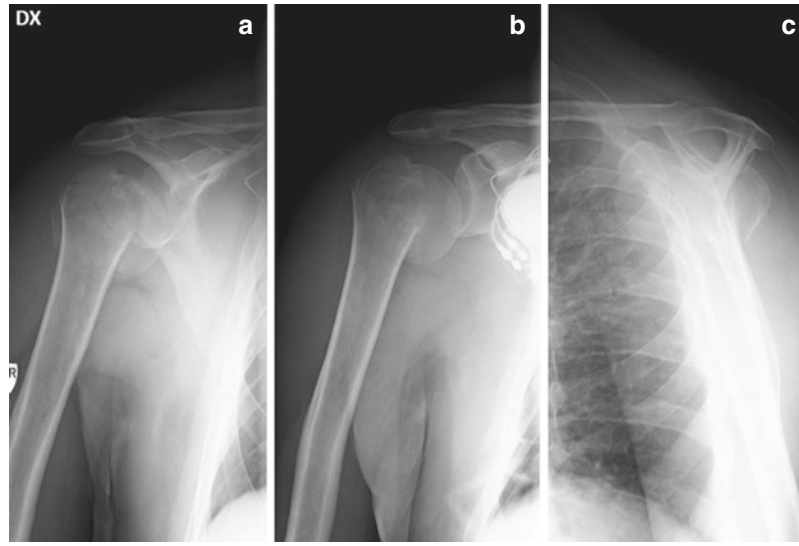

Fig. 20.1 Shoulder trauma series performed at another institution, showing the multifragmentary four-part fracture of the right proximal humerus. (**a**) Anteroposterior shoulder internal rotation view. (**b**) Anteroposterior shoulder external rotation view. (**c**) Shoulder outlet view

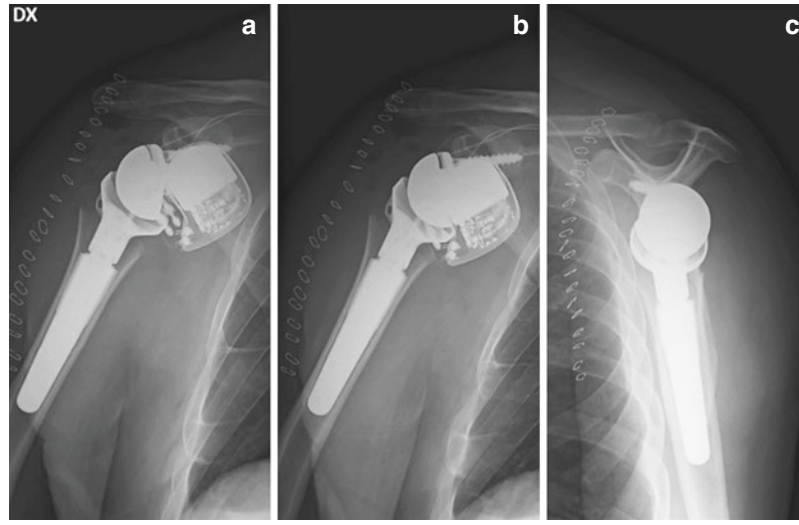

Fig. 20.2 Postoperative radiographic control performed the day after surgery at another institution, showing the implant of the reverse total shoulder arthroplasty. (**a**) Anteroposterior shoulder internal rotation view. (**b**) Anteroposterior shoulder external rotation view. (**c**) Shoulder outlet view

clide tracer in the periprosthetic area, suggesting a periprosthetic joint infection.

A joint puncture was performed, but the collected fluid was insufficient for routine bacteriologic culture methods to isolate any bacterial agent. A thorough clinical investigation did not reveal any finding suggestive of systemic infection or infection localized in other primary *foci*.

According to the clinical and radiological findings and the absence of a certain microbial aetiological agent, we decided for a two-stage revision procedure. In March 2016, in the first intervention, the RSA was removed and exchanged with an antibiotic-loaded (gentamicin + clindamycin + vancomycin) acrylic bone cement spacer (Copal G+C with addition of vancomycin; Heraeus Holding GmbH, Heraeusstraße 12-14, Hanau, Germany) (Fig. 20.4).

After collection of intraoperative cultures, the patient received a single dose of intravenous (iv) antibiotic therapy with 800 mg of teicoplanin and 200 mg of ciprofloxacin.

Two different germs were identified from the intraoperative cultures, *Staphylococcus xylosus* and *Staphylococcus epidermidis*, both susceptible to two out of the three antibiotics used in the

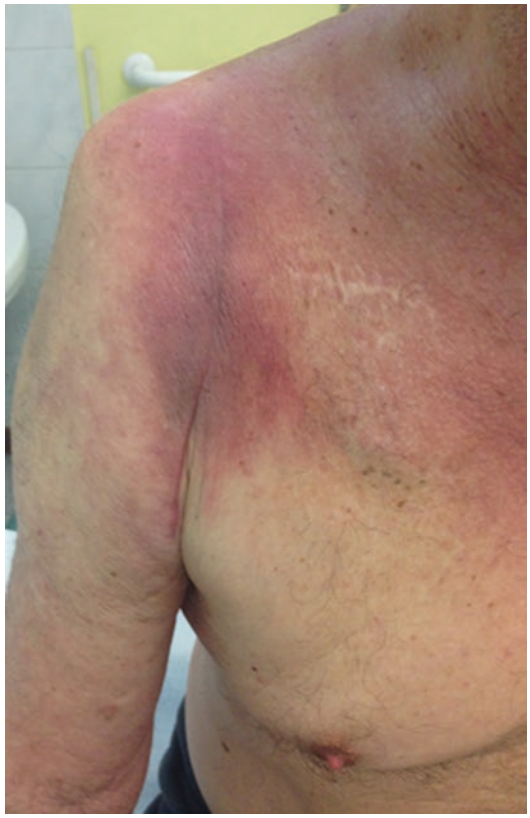

Fig. 20.3 Local findings at clinical examination. Note the erythema along the surgical scar, suggesting a periprosthetic joint infection

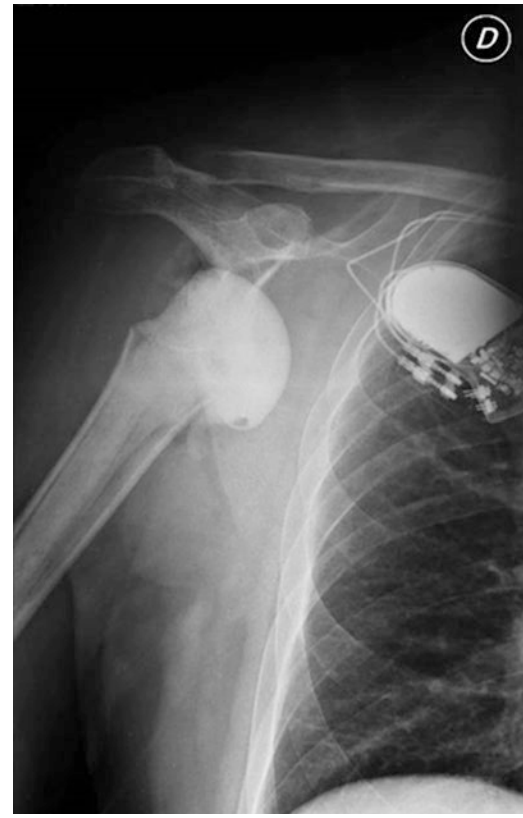

Fig. 20.4 Postoperative radiographic control performed at our institution, showing the antibiotic-loaded acrylic bone cement spacer

spacer. Postoperative antibiotic administration was targeted on culture results and continued orally (amoxicillin/clavulanic acid 825/125 mg, twice a day), under close monitoring of laboratory parameters, for approximately 1 month after patient's discharge.

Once C-reactive protein levels returned into the normal range, in July 2016, the patient underwent a new revision surgery with removal of the cement spacer and implantation of a reverse total shoulder arthroplasty cementing the glenoid with antibiotic-loaded cement (Delta Xtend™, DePuy, Warsaw, IN, USA) (Fig. 20.5). In both revision procedures, a deltopectoral approach was used.

On the first postoperative day, a radiographic control revealed a dislocated implant (Fig. 20.6).

A new revision surgery was hence performed soon afterwards to restore glenohumeral joint stability: the humeral polyethylene cup was changed and a 9 mm spacer was added to augment the implant stability (Fig. 20.7).

Postoperative rehabilitation included shoulder rest in a 10° abduction sling (UltraSling, DJO Global, Vista, CA, USA) for the first 4 weeks; in this period active elbow flexion and extension and scapular exercises were encouraged. Passive mobilization and assisted active exercises were continued up to 3 months after surgery. No other dislocations occurred.

At 6-month follow-up, the patient underwent a thorough clinical examination. Active right shoulder range of motion, including external rotation at side, internal rotation and forward elevation were collected. Pain and quality of life were evaluated with validated outcomes scores: visual analogue scale (VAS) and Simple Shoulder Test (SST).

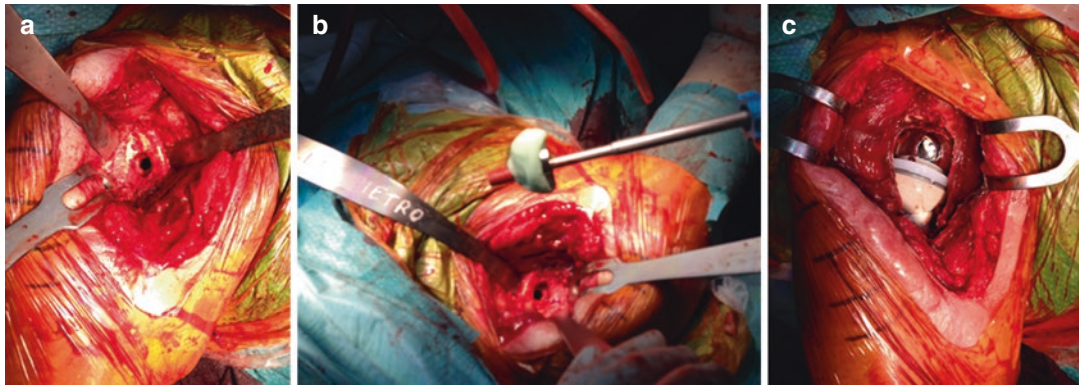

Fig. 20.5 Intraoperative images showing the reimplantation of the reverse shoulder arthroplasty. (**a**) Good glenoid bone stock after removal of the cement spacer. (**b**) Implant of the glenoid with antibiotic-loaded cement. (**c**) Final reduction of an apparently stable implant at the end of the surgical procedure

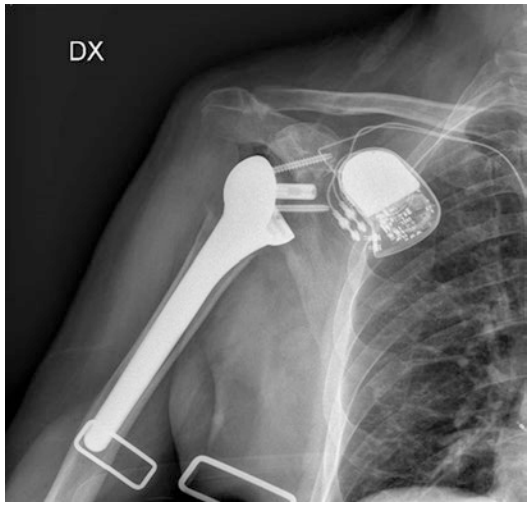

Fig. 20.6 Postoperative radiographic control performed the day after surgery at our institution, showing the dislocation of the new implant

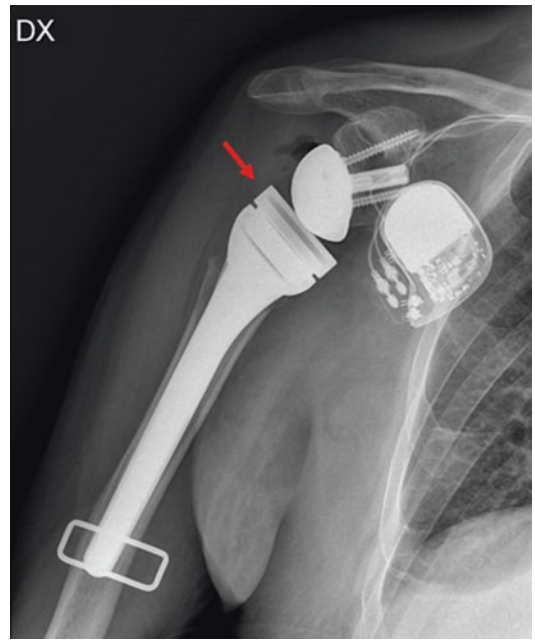

Fig. 20.7 Postoperative radiographic control performed at our institution, showing correct position of the prosthetic components. Note the 9 mm spacer used as humeral augmentation (red arrow)

The patient showed satisfactory clinical outcomes with a stable implant. He did not refer any shoulder pain (VAS = 0), and his range of motion allowed him to carry out the majority of daily activities (SST = 9/12) (Fig. 20.8).

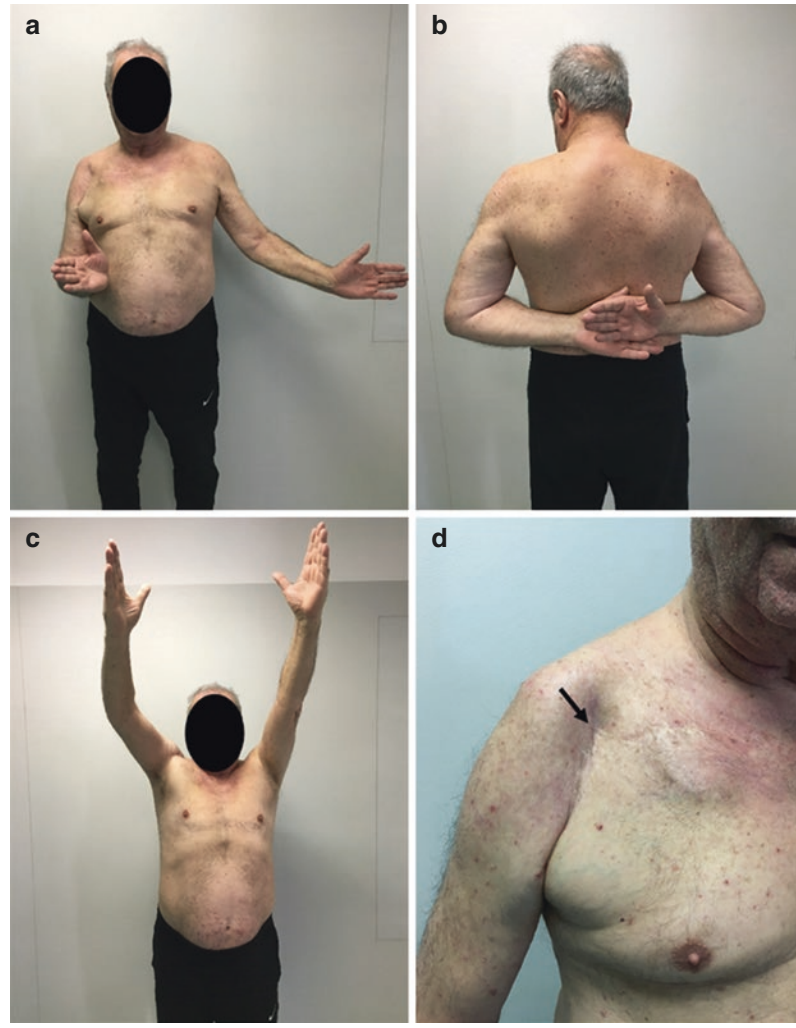

Fig. 20.8 Clinical evaluation at the final follow-up. (**a**) External rotation. (**b**) Internal rotation. (**c**) Forward flexion. (**d**) Surgical scar without signs of local infection (arrow)

Part IV

Failed Reverse Shoulder Arthroplasty

Reverse Shoulder Arthroplasty: How It Works

21

Bruno Toussaint and Jérôme Bahurel

21.1 Introduction

The semi-constrained reverse shoulder arthroplasty (RSA) designed by incorporating Grammont's work allows us to treat complex shoulder conditions, such as irreparable rotator cuff tears, eccentric shoulder osteoarthritis (OA), rheumatic diseases, and trauma in elderly patients. In other words, all those conditions where all other shoulder arthroplasty designs had failed.

The rationale of RSA has been supported by various biomechanical studies and by the development of several implant designs. Scapular notching is an unfavorable effect of this type of prosthesis, albeit it does not necessarily compromise the longevity of the implant. Understanding of this phenomenon has opened the way to its prevention.

Recent studies showed improved survival of RSA. This is promising and allows the range of indications to be widened.

21.2 History

Functional impairment of the shoulder and glenohumeral OA have both contributed to the development of shoulder prostheses. The first shoulder prostheses, developed by Gluck in 1800 and then improved by the French surgeon Jean Emile Péan in 1893 [1, 2], were constrained and combined a platinum and leather stem articulating with a paraffin-coated rubber head. Following experiences to address the insufficiency of rotator cuff were based on constrained designs [3, 4].

After developing an unconstrained prosthesis initially indicated in fractures, Neer conceived the first reverse prosthesis in 1970, and with it established the concept of replacing the irreparable rotator cuff tear with the deltoid muscle. However, the sphere size was one of the elements limiting the effectiveness of the prosthesis [5].

In 1972, Reeves designed a prosthesis with a center of rotation identical to the anatomical center of rotation. At the same time, Gérard and Lannelongue developed their own prosthesis model, which however resulted in a high number of dislocations and for this reason it was quickly abandoned.

Kobel and Kessel respectively worked on glenoid component fixation by means of a central screw and lateralization of the center of rotation and observed bone lysis (radiolucent lines) around the glenoid [6]. Simultaneously, Bayley and Walker were using glenoid component fixation with a hydroxyapatite-coated screw, but with a medialized center of rotation.

In 1975, Fentin used the Jefferson prosthesis with a large-diameter glenosphere with a metal-metal bearing surface and lateralized center of

B. Toussaint (✉) · J. Bahurel
Clinique Générale, Annecy, France

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_21

rotation. However, there were numerous complications. At the same time, Beddow used a prosthesis similar in design to the Charnley prosthesis, with the stem fixated in the pillar of the scapula.

In 1978, Buechel, Pappas, and De Palma adopted the concept of dual mobility after Bickel and Stanmore failed in their attempt to design anatomically constrained prostheses. This floating socket concept was then adopted by Swanson and Worland [7].

In 1985, Grammont designed his first inverted prosthesis. The concave polyethylene humeral part, articulating with the convex glenoid part, is based on four principles: the intrinsic stability of the prosthesis, the fixed part being convex and the moving part being concave, the medialization and lowering of the center of rotation, and the center of rotation being on the surface of the glenoid rim [8]. Unfortunately, the first glenoid component was lateralized and responsible for disassembling.

In 1989, the second Delta III prosthesis finalized the reverse prosthesis concept. The center of rotation is at the interface between the bone and the prosthetic metaglene component and requires medialization and lowering. Its stability must be ensured by the prosthetic design. The recommended humeral cut angle is 155° as it offers the greatest stability to the prosthesis. These principles dominated the evolution and development of the different models of inverted prostheses that followed.

In 1998, Frankle designed a prosthesis with a center of rotation lateralized with respect to the bone-prosthesis interface and using metaglene screw fixation. Other designs modified the baseplate fixation of the metaglene by adding divergent screws (Equalis; Tornier, now Wright Medical, Memphis, TN, USA).

The modularity of the humeral implant made it possible to convert an anatomical replacement to a RSA without removing the humeral stem. The Bigliani–Flatow prosthesis and the Anatomical prosthesis developed by Gerber (Zimmer-Biomet; Warsaw, IN, USA) and Habermeyer's Universe prosthesis (Arthrex, Naples, FL, USA) are a response to this requirement for modularity. Other models such as the Universal Arrow System (FH Ortho, Heimsbrunn, France) aimed to reduce the risk of notching.

In 2004, the first stemless RSA was conceived, with fixation on the metaphyseal region (TESS; Zimmer-Biomet; and Verso; IDO, reading, UK). More recently, short-stem RSA (Ascend; Wright) has been introduced. Indeed, this implant design allows filling the width of the diaphysis and then it is still exposed to the same risk of fracture of standard stems, as it is simply placed higher on the shaft.

21.3 Biomechanics

The fundamentals summarized by Grammont were the medialization of the neo-articulation center of rotation, located at the glenoidal bone-prosthesis (metaglene) interface, an inclination at 155° of the humeral part to ensure stability, and the lowering of the humeral part to increase the efficiency of the deltoid muscle [9, 10]. At first, these points appeared essential, as they would guarantee the correct function of RSA. Initially studied by the company that manufactured the Grammont prosthesis, these principles were further confirmed by cadaveric and finite element studies. However, the Frankle prosthesis separated the bone-prosthesis interface from the center of rotation and lateralized it with respect to the Grammont prosthesis [11]. Several authors then began to challenge the Grammont principles and demonstrated that decreasing the inclination angle of the humeral component improved mobility of the RSA without significantly decreasing its stability [12, 13]. These studies compared the inclination angles at 155°, 145°, and 135° and showed that decreasing the inclination angle increased mobility in adduction while reducing abduction or increasing forces necessary for abduction [12, 13].

Stability of the RSA also depends on the size of the glenosphere; the greater the size of the glenosphere, the more stable the prosthesis, in the absence of muscular stabilization. Conversely, the size of the glenosphere does not significantly affect joint mobility. The polyethylene humeral cup reduces stability while seeking to increase the articular range of motion [14, 15].

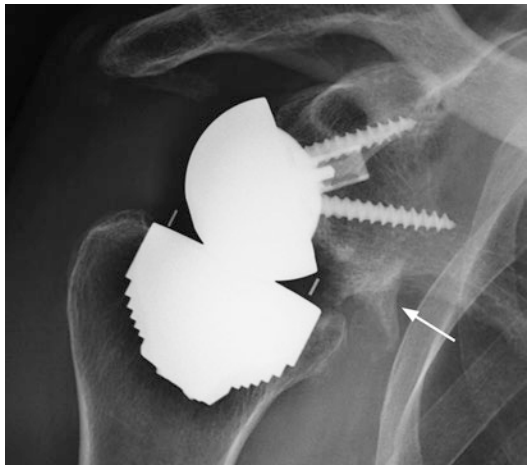

Fig. 21.1 Scapular spur (arrow) due to notching mechanism

The discovery of the scapular notching (Fig. 21.1) led to a new though process and to new anatomical and biomechanical studies, which showed that the retroversion of the humeral implant reduced the risk of notching, as did reducing the inclination angle. Moreover, the low position of the glenosphere and the use of large-diameter glenosphere further reduce the risk of notching [16].

A bone graft under the baseplate does not seem to modify the risk of notching but lateralize the glenosphere and notably correct the medialization of the glenohumeral joint with regard to potential concentric erosion of the glenoid [17].

The position of the glenoid is important. It should be parallel with the glenoid surface at a slight inferior tilt, but no superior inclination, which favors the dislocation of the glenoid implant and exposes the bone of the lower pillar of the scapula to be in contact with the humeral implant. Conversely, inferior inclination is supported by the lower pillar of the scapula, one of the stronger points of fixation for the glenoid implant. Moreover, the glenosphere is a section of sphere, and when rotating around its center it does not alter its position in relation to the humeral component [18, 19]. Elongating the upper limb by lowering the position of the glenoid and of the humeral metaphysis combined with lateralization of the humeral implant and increasing the size of the glenosphere will increase the deltoid force moment and power [20–23]. As it stands, biomechanical studies redefined a certain number of basic principles for the kinematics and kinetic of RSA:

- The lateralization of the center of rotation does not modify the fixation strength of the glenoid component, which should be uncemented.
- The position of the glenosphere should be as low as possible with fixation by a central peg or screw and at least two diverging screws fixed in the lower pillar of the scapula and into the base of the coracoid process.
- The size of the glenosphere improves the stability of the implant without significantly altering its mobility [24–26].
- Metaphyseal lowering and lateralization of the humerus can improve the function of the deltoid muscle.
- Humeral implant retroversion is a factor that reduces notching.
- The inclination angle of the humeral component can be varied from 155° to 135°, improving mobility in adduction but increasing stress in abduction and reducing the risk of notching [27, 28].
- The lateralization of the center of rotation is a decisive factor in the restoration of rotations by recruitment of the anterior and posterior parts of the deltoid muscle, which could then act as internal or external rotator muscles.
- Increased glenoid bone volume under the metaglene and the glenosphere limits the excursion of the humeral part around the glenosphere [29, 30].

All these modifications seem to have no effect on internal polyethylene wearing (Fig. 21.2). Indeed, wearing of the polyethylene releases polyethylene particles with a deleterious biological effect, promoting bone resorption at the bone-prosthesis interface [31, 32]. This explains why prosthetic designs with a reversal of the metal-polyethylene bearing surface are under evaluation.

Fig. 21.2 Polyethylene wearing

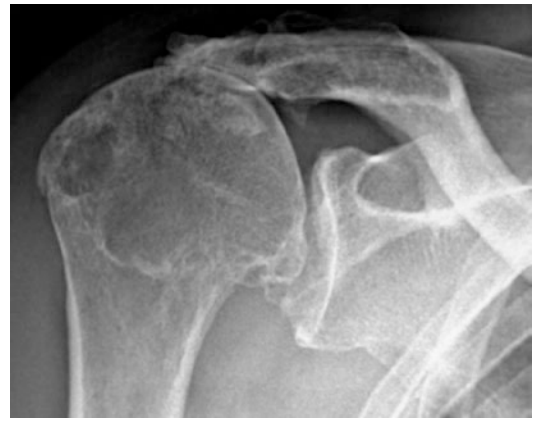

Fig. 21.3 Eccentric glenohumeral osteoarthritis

21.4 Indications

Reverse prostheses were initially designed for pseudoparalytic massive rotator cuff tears, with or without associated eccentric glenohumeral OA [33–37] (Fig. 21.3). After 2001, the 70–75-year age limit was introduced in light of the survivorship rate at 10 years [38].

The indications have been extended to young patients with massive rotator cuff tears where other therapeutic options, such as latissimus dorsi transfer or superior capsule reconstruction, cannot be considered because of irreparable subscapularis and/or infraspinatus tendon [38–41]. Indication to RSA has been also extended to rheumatoid arthritis with or without cuff tear [42, 43]. Other indications are revision of anatomical shoulder arthroplasty [44–47], bone tumors [48], fractures [49–51] or fracture sequelae [52], and, more recently, complex fractures or fracture-dislocations in elderly patients [53].

Common requirements to all the RSAs are adequate glenoid bone stock [54–56] for the implantation of the metaglene and the glenosphere, and good deltoid function, although some authors suggested muscular transfers to counter any insufficiency of the deltoid muscle [57]. Severe glenoid bone losses should be addressed by bone graft or customized metaglene. Image processing software help calculating the volume and shape of the bone graft or designing custom implants to compensate for such loss of bone substance [58, 59].

Combining a latissimus dorsi transfer with RSA allows recovery of external rotation in cases of combined deficiency of the infraspinatus and teres minor muscles [60, 61].

21.5 Results

First results reported in 2001 and, then in 2011 [62], demonstrated very good results with 90% survivorship at 5 years, but with a worsening of survivorship rate from 8 years onward. At 10 years, 72% had lost 30 points on the Constant-Murley score scale. However, the oldest RSA designs were not compliant with the positioning criteria currently recognized by the majority of authors, thus affecting the results.

Similar results reported by other authors [63–65] led to a certain amount of caution in the indication for RSA. Early complications during the first 3 years were frequent and were dominated by infections, which are difficult to manage and require revision surgery with unpredictable results. The abovementioned series reported 107 complications in 489 patients, infection and mechanical problems with the glenoid being the most frequent with 27 cases each, 19 cases of instability, 14 hematomas, 11 humeral complications, 6 neurological complications, and 3 fractures of the scapula. The survivorship rate differed according to etiology, with massive cuff tears having the best 5- and 10-year survivorship rates, followed by

cuff tear arthropathy. Survivorship of RSA which had been converted to hemiarthroplasty was 89% after 10 years. It was concluded that the outcome becomes significantly worse after 8 years.

Melis et al. [66] reported on a series of 68 patients reviewed after 8 years on average and showed that the rate of notching was 88%, but with no significant effect on the clinical outcome. This result is similar to that of other studies [62, 63].

Recently, Bacle et al. [67] reported on a series of 109 patients with a mean follow-up of 150 months (121–241 months). The Kaplan–Meier survival curve showed a stable curve with 93% survivorship at 10 years, albeit few implants aged more than 10 years. This study demonstrated that the survival of RSA is longer than was initially thought. This encourages an extension of the indications. At the same time, series of stemless RSA reported very similar results, even if with shorter follow-up, which is encouraging for the preservation of bone stock [68, 69]. The recently designed short-stem prostheses have an insufficient follow-up.

Conclusion

RSA is the solution to deal with irreparable rotator cuff tears where all therapeutic options have been exhausted, as well as to the associated eccentric glenohumeral OA. RSA can also be indicated to avoid problems caused by revision surgery on anatomical prostheses, such as rheumatoid disease and proximal humerus fractures in elderly patients.

The use of RSA is now well defined by following the principles of medialization of the center of rotation of the shoulder, and lateralization of the humerus, which increases the efficacy and power of the deltoid muscle. The angle of the humeral neck appears to be less important, and reducing it may improve mobility, particularly in rotation. The position of the metaglenoid and glenosphere must be as low as possible without superior tilt. The length of the humeral stem must ensure lengthening of the arm, which provides stability.

Recent 10-year follow-up studies are more optimistic than the initial results, and this allowed to extend indications to RSA. Nonetheless, the surgeon must be aware that revision of RSA is an extremely difficult procedure with less predictable results than primary implant.

References

1. Hawk AJ. ArtiFacts: the case of Jules Pedoux. Clin Orthop Relat Res. 2016;474(2):302–3.
2. Katz D, O'Toole G, Cogswell L, Sauzieres P, Valenti P. A history of the reverse shoulder prosthesis. Int J Shoulder Surg. 2007;1:108–13.
3. Post M, Haskell SS, Jablon M. Total shoulder replacement with a constrained prosthesis. J Bone Joint Surg Am. 1980;62(3):327–35.
4. Lettin AW, Copeland SA, Scales JT. The Stanmore total shoulder replacement. J Bone Joint Surg Br. 1982;64(1):47–51.
5. Coughlin MJ, Morris JM, West WF. The semiconstrained total shoulder arthroplasty. J Bone Joint Surg Am. 1979;61(4):574–81.
6. Flatow EL, Harrison AK. A history of reverse total shoulder arthroplasty. Clin Orthop Relat Res. 2011;469(9):2432–9.
7. Worland RL, Jessup DE, Arredondo J, Warburton KJ. Bipolar shoulder arthroplasty for rotator cuff arthropathy. J Shoulder Elbow Surg. 1997;6(6):512–5.
8. 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.
9. Baulot E, Sirveaux F, Boileau P. Grammont's idea: the story of Paul Grammont's functional surgery concept and the development of the reverse principle. Clin Orthop Relat Res. 2011;469(9):2425–31.
10. Gutiérrez 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.
11. 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.
12. 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 Elbow Surg. 2016;25(4):589–97.
13. 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 Elbow Surg. 2014;23(8):1091–8.

14. 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 Elbow Surg. 2016;25(5):763–71.
15. 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 Elbow Surg. 2015;24(9):1359–66.
16. 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(3):288–97. https://doi.org/10.4055/cios.2016.8.3.288.
17. Greiner S, Schmidt C, Herrmann S, Pauly S, Perka C. Clinical performance of lateralized versus nonlateralized reverse shoulder arthroplasty: a prospective randomized study. J Shoulder Elbow Surg. 2015;24(9):1397–404.
18. Ackland DC, Patel M, Knox D. Prosthesis design and placement in reverse total shoulder arthroplasty. J Orthop Surg Res. 2015;10:101.
19. Chae SW, Lee J, Han SH, Kim SY. Inferior tilt fixation of the glenoid component in reverse total shoulder arthroplasty: a biomechanical study. Orthop Traumatol Surg Res. 2015;101(4):421–5.
20. 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.
21. Giles JW, Langohr GD, 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(11):3615–26.
22. 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 Elbow Surg. 2016;25(6):998–1004.
23. Fischer C, Krammer D, Hug A, Weber MA, Kauczor HU, 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 Elbow Surg. 2017;26(1):108–17.
24. Feeley BT, Zhang AL, Barry JJ, Shin E, Ho J, Tabaraee E, Ma CB. 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.
25. 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 HR, Ullrich O, Rühli FJ, 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(1):9.
26. Nyffeler RW, Werner CM, Gerber C. Biomechanical relevance of glenoid component positioning in the reverse delta III total shoulder prosthesis. J Shoulder Elbow Surg. 2005;14(5):524–8.
27. 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.
28. Wright J, Potts C, Smyth MP, Ferrara L, Sperling JW, Throckmorton TW. A quantitative analysis of the effect of baseplate and glenosphere position on deltoid lengthening in reverse total shoulder arthroplasty. Int J Shoulder Surg. 2015;9(2):33–7.
29. Carpenter S, Pinkas D, Newton MD, Kurdziel MD, Baker KC, Wiater JM. Wear rates of retentive versus nonretentive reverse total shoulder arthroplasty liners in an in vitro wear simulation. J Shoulder Elbow Surg. 2015;24(9):1372–9.
30. 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.
31. de Wilde LF, Poncet D, Middernacht B, Ekelund A. Prosthetic overhang is the most effective way to prevent scapular conflict in a reverse total shoulder prosthesis. Acta Orthop. 2010;81(6):719–26.
32. Walker M, Brooks J, Willis M, Frankle M. How reverse shoulder arthroplasty works. Clin Orthop Relat Res. 2011;469(9):2440–51.
33. 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.
34. 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 Elbow Surg. 2015;24(11):1698–706.
35. Laurence M. Replacement arthroplasty of the rotator cuff deficient shoulder. J Bone Joint Surg Br. 1991;73(6):916–9.
36. Neer CS 2nd, Craig EV, Fukuda H. Cuff-tear arthropathy. J Bone Joint Surg Am. 1983;65(9):1232–44.
37. Neri BR, Chan KW, Kwon YW. Management of massive and irreparable rotator cuff tears. J Shoulder Elbow Surg. 2009;18(5):808–18.
38. Arntz CT, Jackins S, Matsen FA 3rd. Prosthetic replacement of the shoulder for the treatment of defects in the rotator cuff and the surface of the glenohumeral joint. J Bone Joint Surg Am. 1993;75(4):485–91.
39. Kircher J. Shoulder endoprosthesis in the elderly: Hemiarthroplasty or total shoulder arthroplasty? Anatomic or reverse? Orthopade. 2017;46(1):40–7.
40. Statz JM, Wagner ER, Houdek MT, Cofield RH, Sanchez-Sotelo J, Elhassan BT, Sperling JW. Outcomes of primary reverse shoulder arthroplasty

in patients with morbid obesity. J Shoulder Elbow Surg. 2016;25(7):e191–8.
41. Samuelsen BT, Wagner ER, Houdek MT, Elhassan BT, Sánchez-Sotelo J, Cofield R, Sperling JW. Primary reverse shoulder arthroplasty in patients aged 65 years or younger. J Shoulder Elbow Surg. 2017;26(1):e13–7.
42. Rittmeister M, Kerschbaumer F. Grammont reverse total shoulder arthroplasty in patients with rheumatoid arthritis and nonreconstructible rotator cuff lesions. J Shoulder Elbow Surg. 2001;10(1):17–22.
43. Gee EC, Hanson EK, Saithna A. Reverse shoulder Arthroplasty in rheumatoid arthritis: a systematic review. Open Orthop J. 2015;9:237–45.
44. Chuinard C. CORR Insights®: conversion of stemmed hemi- or total to reverse total shoulder arthroplasty: advantages of a modular stem design. Clin Orthop Relat Res. 2015;473(2):661–2.
45. Cisneros LG, Atoun E, Abraham R, Tsvieli O, Bruguera J, Levy O. Revision shoulder arthroplasty: does the stem really matter? J Shoulder Elbow Surg. 2016;25(5):747–55.
46. Hartel BP, Alta TD, Sewnath ME, Willems WJ. Difference in clinical outcome between total shoulder arthroplasty and reverse shoulder arthroplasty used in hemiarthroplasty revision surgery. Int J Shoulder Surg. 2015;9(3):69–73.
47. Hurwit DJ, Liu JN, Garcia GH, Mahony G, Wu HH, Dines DM, Warren RF, Gulotta LV. A comparative analysis of work-related outcomes after humeral hemiarthroplasty and reverse total shoulder arthroplasty. J Shoulder Elbow Surg. 2017;26(6):954–9.
48. Guven MF, Aslan L, Botanlioglu H, Kaynak G, Kesmezacar H, Babacan M. Functional outcome of reverse shoulder tumor prosthesis in the treatment of proximal humerus tumors. J Shoulder Elbow Surg. 2016;25(1):e1–6.
49. Dean BJ, Jones LD, Palmer AJ, Macnair RD, Brewer PE, Jayadev C, Wheelton AN, Ball DE, Nandra RS, Aujla RS, Sykes AE, Carr AJ. A review of current surgical practice in the operative treatment of proximal humeral fractures: does the PROFHER trial demonstrate a need for change? Bone Joint Res. 2016;5(5):178–84.
50. Dezfuli B, King JJ, Farmer KW, Struk AM, Wright TW. Outcomes of reverse total shoulder arthroplasty as primary versus revision procedure for proximal humerus fractures. J Shoulder Elbow Surg. 2016;25(7):1133–7.
51. Shukla DR, McAnany S, Kim J, Overley S, Parsons BO. Hemiarthroplasty versus reverse shoulder arthroplasty for treatment of proximal humeral fractures: a meta-analysis. J Shoulder Elbow Surg. 2016;25(2):330–40.
52. Grubhofer F, Wieser K, Meyer DC, Catanzaro S, Schürholz K, Gerber C. Reverse total shoulder arthroplasty for failed open reduction and internal fixation of fractures of the proximal humerus. J Shoulder Elbow Surg. 2017;26(1):92–100.
53. Kurowicki J, Triplet JJ, Momoh E, Moor MA, Levy JC. Reverse shoulder prosthesis in the treatment of locked anterior shoulders: a comparison with classic reverse shoulder indications. J Shoulder Elbow Surg. 2016;25(12):1954–60.
54. Donohue KW, Ricchetti ET, Iannotti JP. Surgical management of the biconcave (B2) glenoid. Curr Rev Musculoskelet Med. 2016;9(1):30–9.
55. Formaini NT, Everding NG, Levy JC, Santoni BG, Nayak AN, Wilson C, Cabezas AF. The effect of glenoid bone loss on reverse shoulder arthroplasty baseplate fixation. J Shoulder Elbow Surg. 2015;24(11):e312–9.
56. Jones RB, Wright TW, Roche CP. Bone grafting the Glenoid versus use of augmented Glenoid baseplates with reverse shoulder Arthroplasty. Bull Hosp Jt Dis (2013). 2015;73(Suppl 1):S129–35.
57. Marinello PG, Amini MH, Peers S, O'Donnell J, Iannotti JP. Reverse total shoulder arthroplasty with combined deltoid reconstruction in patients with anterior and/or middle deltoid tears. J Shoulder Elbow Surg. 2016;25(6):936–41.
58. Holcomb JO, Cuff D, Petersen SA, Pupello DR, Frankle MA. Revision reverse shoulder arthroplasty for glenoid baseplate failure after primary reverse shoulder arthroplasty. J Shoulder Elbow Surg. 2009;18(5):717–23.
59. 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):13–8.
60. Puskas GJ, Germann M, Catanzaro S, Gerber C. Secondary latissimus dorsi transfer after failed reverse total shoulder arthroplasty. J Shoulder Elbow Surg. 2015;24(12):e337–44.
61. Shi LL, Cahill KE, Ek ET, Tompson JD, Higgins LD, Warner JJ. 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(10):3212–7.
62. 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(8):1742–7.
63. Nyffeler RW, Werner CM, Simmen BR, Gerber C. Analysis of a retrieved delta III total shoulder prosthesis. J Bone Joint Surg Br. 2004;86(8):1187–91.
64. Werner BS, Abdelkawi AF, Boehm D, Hudek R, Plumhoff P, Burkhart KJ, Gohlke F. Long-term analysis of revision reverse shoulder arthroplasty using cemented long stems. J Shoulder Elbow Surg. 2017;26(2):273–8.
65. 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.

66. 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 Br. 2011;93(9):1240–6.
67. 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(6):454–61.
68. Giannotti S, Bottai V, Dell'Osso G, Bugelli G, Guido G. Stemless humeral component in reverse shoulder prosthesis in patient with Parkinson's disease: a case report. Clin Cases Miner Bone Metab. 2015;12(1):56–9.
69. 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 Elbow Surg. 2016;25(8):1362–70.

Reverse Shoulder Arthroplasty: Why It Fails

22

Vladimir Senekovič

22.1 Introduction

Reverse shoulder arthroplasty (RSA) is a successful treatment for elderly patients with large rotator cuff tears or painful rotator cuff arthropathy [1–5]. Four-part fractures, failed anatomical shoulder replacements, and failed fracture treatments with other methods are other indications to RSA [6–8].

Some problems are unique to the RSA. The most important ones are neurologic problems, bone fracture around the implant, hematoma postoperative infection, instability (dislocation), baseplate failure, and scapular notching. The best way to handle these problems has not yet been determined. The long-term results in patients who have a RSA show about 20% complication rate [9, 10]. Improvement in implant design, knowledge of shoulder biomechanics, and management of common complications will help the surgeon avoiding failures of RSA.

22.2 Neurologic Complications

The most important complication of RSA is neurologic injury, which can be preoperative or intraoperative. If we have a chronic neurologic deficit (i.e., plexus brachialis palsy, axillary nerve palsy, or suprascapular nerve palsy), preoperative electromyography (EMG) investigation is always necessary [11].

Traction injuries during surgery can cause sensory damage to the arm, hand, and/or fingers [12]. Sometimes, the RSA implant causes strain to the brachial plexus. In other cases, the implant can displace (push aside) the brachial plexus, thus creating loss of sensory and/or motor function to the arm. Nerve stretch injury can be also very painful. Another cause of nerve injury is scar tissue that can compress or impinge nerves of the brachial plexus, thus causing impairment of sensory and/or motor function and pain [9, 10].

The diagnosis of such problems is precise neurologic examination and ultrasound (US) investigations. EMG can be helpful after 3–4 weeks from the onset of symptoms. In acute motor deficit and very painful shoulder after operation, revision surgery is indicated in the acute phase.

22.3 Periprosthetic Fracture

Most of these fractures occur during the surgical procedure as the surgeon prepares the bone to receive the implant. Preoperative computed

V. Senekovič
Faculty of Medicine, University of Ljubljana, Ljubljana, Slovenia

Clinic Arbormea Medical Ljubljana, Ljubljana, Slovenia
e-mail: vladimir.senekovic@arbormea.com

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_22

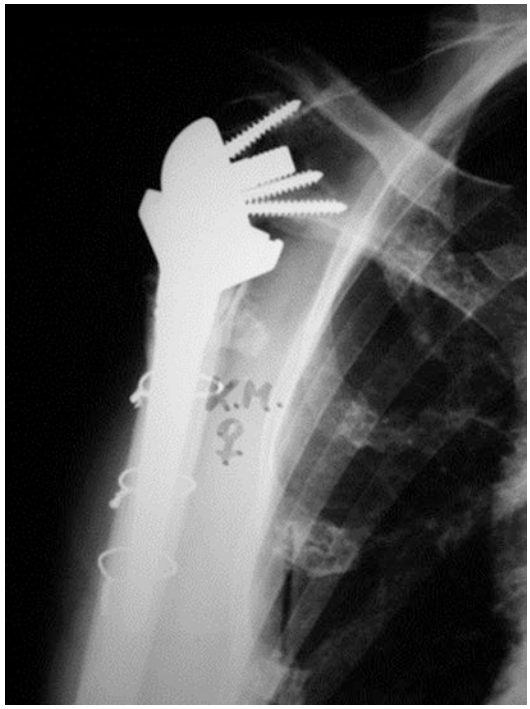

Fig. 22.1 Periprosthetic fracture of the shaft fixed with cerclages

tomography (CT) of the shoulder is useful to evaluate the bone quality and to estimate if the bone is hard enough for implantation of RSA.

If periprosthetic fracture of the shaft occurs during surgery, we have to fix the fracture with cerclages and/or plates [13] (Fig. 22.1).

When glenoid breaks during the operation, we can try to fix it [14]. If this is not possible, hemiarthroplasty with large humeral head can be considered.

The surgeon must be very familiar with the implant itself, its design and how it is supposed to work, as well as the best way to hold the implant in place [15–17]. Care must be taken when handling the patient's arm during the procedure. Extreme shoulder positions in a patient with weak or brittle bones can contribute to bone fractures [18].

22.4 Hematoma

Hematoma is another common complication of RSA. Proper placement of the implant is necessary to avoid fluid collection in empty areas (dead spaces). Sometimes patients develop pathways of drainage called sinus tracts at the incision site where blood and fluid can pool causing a hematoma. Studies have also shown that infection and hematoma are linked [19].

22.5 Infection

Up to 10% of all patients receiving a RSA develop a serious infection. Risk factors include multiple previous surgeries, a large-sized dead space, poor sterile technique, and revision surgeries on the reverse implant. The most common cause of low-grade infection in shoulder surgery is *Propionibacterium* acnes which is very difficult to isolate. Surgery has to be very precise and non-traumatic to avoid this complication. Moreover, broad-spectrum antibiotics should be administered before the procedure [20].

When the patient develops an infection after RSA, surgical treatment and targeted antibiotics are the treatment of choice. Irrigation and debridement is the first-line treatment in acute phase. When this fails, we have to remove the implant. Antibiotic-impregnated polymethylmethacrylate (cement) beads are used to fill the empty space of the shoulder. Revision RSA can be considered when infection has been eradicated [21, 22] (Fig. 22.2).

22.6 Instability

Arm placed in extension, adduction, and internal rotation can dislocate a reverse shoulder implant. Any imbalance in the muscle tension around the shoulder and mismatch of the prosthetic components are the most frequent causes of instability [23–28]. Because of this, bigger implant components should be used to avoid the risk of instability, and muscle tension should be assessed during surgery during implantation of the trial components [29, 30]. Humeral rotation must be checked during surgery to evaluate the risk of instability [31].

RSA is a valid treatment option for shoulder fractures. My indications for RSA are three- and four-part fractures, head splitting, and complex fracture-dislocations with severe dislocation

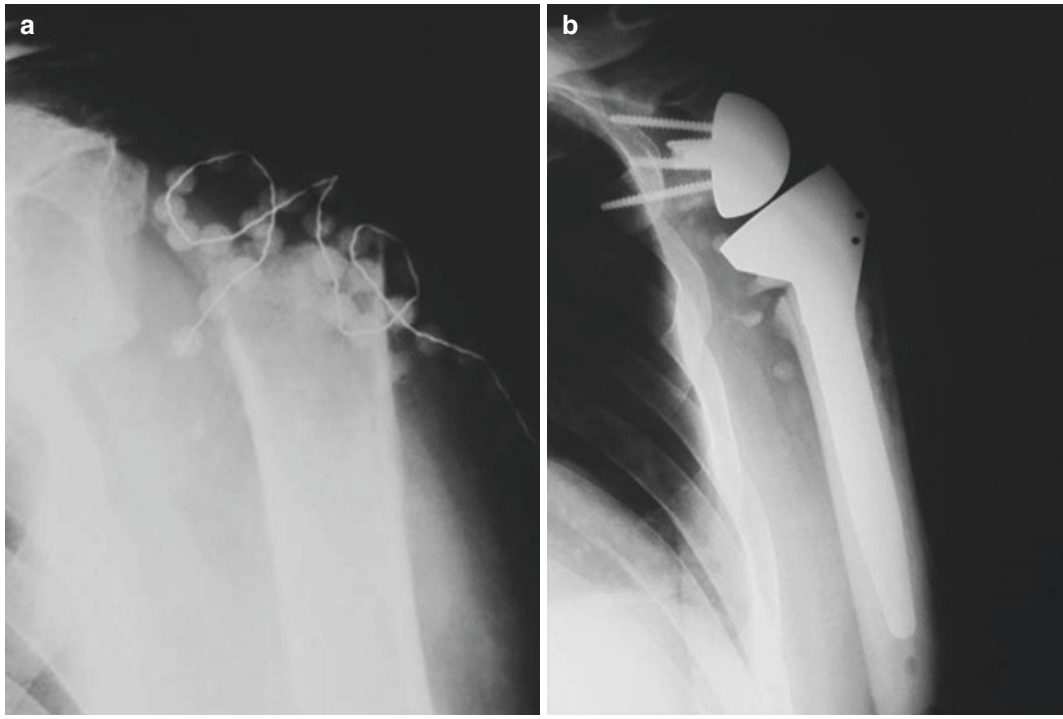

Fig. 22.2 (**a**) The whole implant has been removed because of the infection. The empty space is filled with antibiotic-impregnated polymethylmethacrylate (cement) beads. (**b**) Revision RSA after eradication of infection

(varus/valgus and retroversion-dislocation of the head more than 45°, dislocation of the tuberosities more than 1 cm and dislocation between the head and the shaft of more than half of the thickness) and age (older than 75 years). Special designs of RSA allow fixation of tuberosities close to the shaft (Fig. 22.3).

When dislocation of the prosthesis occurs, the surgeon will try putting the shoulder back in the socket and then placing the patient in a sling for 3–6 weeks. Patients are cautioned to avoid shoulder extension, adduction, and internal rotation (those motions that can flip the shoulder out of joint) until fully healed. If conservative treatment fails, revision surgery is the only treatment option (Fig. 22.4). At surgery, polyethylene insert must be replaced with a thicker one.

Fig. 22.3 Special fracture design of RSA that allows fixation of tuberosities close to the shaft

22.7 Baseplate Failure

The baseplate (metaglene) is the part of the prosthesis that attached the glenosphere to the scapula. If bone ingrowth does not occur around the

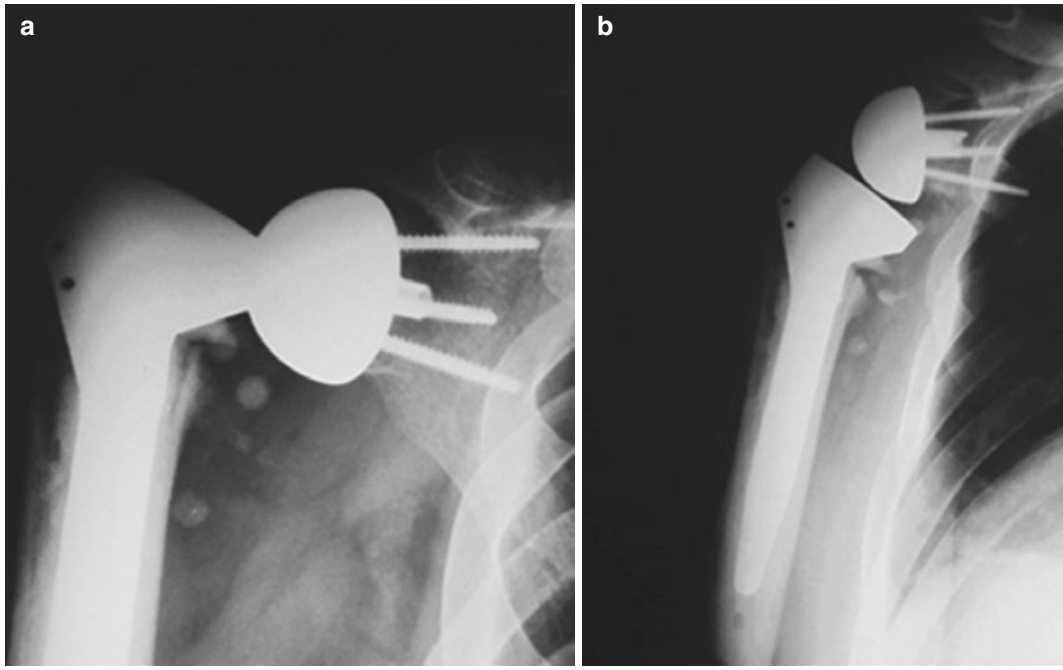

Fig. 22.4 (**a**) Dislocation of the prosthesis. (**b**) Revision case of dislocated RSA. After change of the polyethylene insert for a thicker one, stability of the prosthesis was restored

baseplate, the implant may not be secure. Implant manufacturers provided locking screws that can be angled into the denser bone to help prevent the problem of baseplate failure. A central screw (right through the middle of the bone) also helps anchoring the implant. Other design features under investigation include using thicker screws, a tilted baseplate, and offsetting the center of rotation [32, 33].

22.8 Scapular Notching

Scapular notching is the erosion of the scapular neck related to impingement by the medial rim of the humeral cup during adduction. It is a radiographic sign specific to RSA. Several studies showed decreased range of motion, decreased strength, lower Constant scores, and more pain because of scapular notching [34, 35]. It appears that lowering the glenosphere on the glenoid decreases contact between the humeral component and the inferior bony pillar, thus decreasing the rate and grade of notching [36–39] (Fig. 22.5). Once notching occurs after RSA, we have to follow the patient. Revisions in symptomatic patients require debridement, bone grafting, and baseplate augmentation.

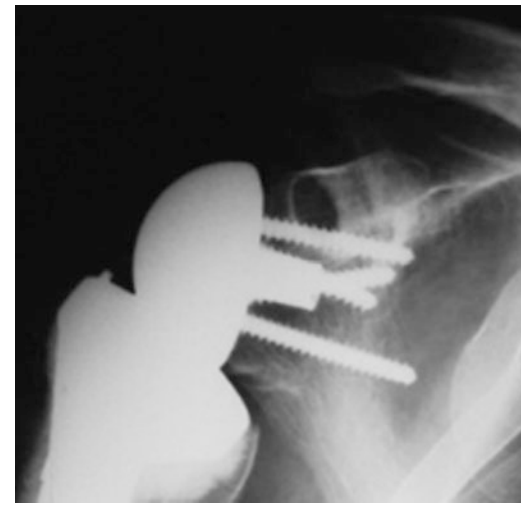

Fig. 22.5 Glenosphere can be put 1–2 cm below the inferior border of the glenoid in order to reduce the risk of scapula notching

Conclusion

RSA is primarily indicated in elderly patients with large rotator cuff tears and rotator-cuff arthropathy. Fractures and failed anatomical shoulder replacements are two other reasons and indications to RSA. As with any surgery (and especially joint replacement procedures), there are potential problems and complications that can develop. Accurate neurologic examination before and after surgery, evaluation of the bone quality, proper placement of the implant, and care to avoid fluid collection in empty areas where hematoma can develop are crucial to reduce the risk of failure of RSA. To avoid infections, surgery has to be very precise and non-traumatic, and broad-spectrum antibiotics must be used just before the procedure. To avoid baseplate failure, we should use baseplate with locking screws that can be angled into the denser bone. Scapula notching can be avoided by lowering the glenosphere.

Although progresses in implant designs will continue to improve outcomes, we have to be aware that 6-year survival rate of RSA is only 80% [2]. For this reason, we have to know why RSA fails. Studies following patients over 10, 15, and 20 years will give us the proper feedback needed to improve survival rates and prevent problems associated with this procedure.

References

1. Baulot E, Chabernaud D, Grammont PM. Results of Grammont's inverted prosthesis in omarthritis associated with major cuff destruction. Apropos of 16 cases. Acta Orthop Belg. 1995;61(Suppl 1):112–9.
2. Wall B, Nové-Josserand L, O'Connor DP, et al. Reverse total shoulder arthroplasty: a review of results according to etiology. J Bone Joint Surg Am. 2007;89:1476–85. https://doi.org/10.2106/JBJS.F.00666.
3. Frankle M, Siegal S, Pupello D, et al. 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. https://doi.org/10.2106/JBJS.D.02813.
4. 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.
5. Levy JC, Virani N, Pupello D, Frankle M. Use of the reverse shoulder prosthesis for the treatment of failed hemiarthroplasty in patients with glenohumeral arthritis and rotator cuff deficiency. J Bone Joint Surg Br. 2007;89:189–95. https://doi.org/10.1302/0301-620X.89B2.18161.
6. 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. https://doi.org/10.1016/j.jse.2006.01.003.
7. Patel M, Nara K, Nara N, Bonato L. Primary reverse total shoulder arthroplasty for proximal humerus fractures in patients over the age of seventy. Orthop Proc. 2012;94–B:161.
8. Boileau P, Trojani C, Walch G, et al. Shoulder arthroplasty for the treatment of the sequelae of fractures of the proximal humerus. J Shoulder Elb Surg. 2001;10:299–308. https://doi.org/10.1067/mse.2001.115985.
9. Bohsali KI, Bois AJ, Wirth MA. Complications of shoulder arthroplasty. J Bone Joint Surg Am. 2017;99:256–69. https://doi.org/10.2106/JBJS.16.00935.
10. Wierks C, Skolasky RL, Ji JH, McFarland EG. Reverse total shoulder replacement: intraoperative and early postoperative complications. Clin Orthop. 2009;467:225–34. https://doi.org/10.1007/s11999-008-0406-1.
11. Kronberg M, Németh G, Broström LA. Muscle activity and coordination in the normal shoulder. An electromyographic study. Clin Orthop. 1990:76–85.
12. Lenoir H, Dagneaux L, Canovas F, et al. Nerve stress during reverse total shoulder arthroplasty: a cadaveric study. J Shoulder Elb Surg. 2017;26:323–30. https://doi.org/10.1016/j.jse.2016.07.020.
13. García-Fernández C, Lópiz-Morales Y, Rodríguez A, et al. Periprosthetic humeral fractures associated with reverse total shoulder arthroplasty: incidence and management. Int Orthop. 2015;39:1965–9. https://doi.org/10.1007/s00264-015-2972-7.
14. Kennon JC, Lu C, McGee-Lawrence ME, Crosby LA. Scapula fracture incidence in reverse total shoulder arthroplasty using screws above or below metaglene central cage: clinical and biomechanical outcomes. J Shoulder Elb Surg. 2017;26:1023–30. https://doi.org/10.1016/j.jse.2016.10.018.
15. Hsu SH, Greiwe RM, Saifi C, Ahmad CS. Reverse total shoulder arthroplasty—biomechanics and rationale. Oper Tech Orthop. 2011;21:52–9. https://doi.org/10.1053/j.oto.2010.10.006.
16. Molé D, Wein F, Dézaly C, et al. Surgical technique: the anterosuperior approach for reverse shoulder arthroplasty. Clin Orthop. 2011;469:2461–8. https://doi.org/10.1007/s11999-011-1861-7.
17. Ackland DC, Richardson M, Pandy MG. Axial rotation moment arms of the shoulder musculature after reverse total shoulder arthroplasty. J Bone Joint Surg Am. 2012;94:1886–95. https://doi.org/10.2106/JBJS.J.01861.

18. Hamid N, Connor PM, Fleischli JF, D'Alessandro DF. Acromial fracture after reverse shoulder arthroplasty. Am J Orthop (Belle Mead NJ). 2011;40:E125–9.
19. Zhou HS, Chung JS, Yi PH, et al. Management of complications after reverse shoulder arthroplasty. Curr Rev Musculoskelet Med. 2015;8:92–7. https://doi.org/10.1007/s12178-014-9252-9.
20. Bonnevialle N, Dauzères F, Toulemonde J, et al. Periprosthetic shoulder infection: an overview. EFORT Open Rev. 2017;2:104–9. https://doi.org/10.1302/2058-5241.2.160023.
21. Assenmacher AT, Alentorn-Geli E, Dennison T, et al. Two-stage reimplantation for the treatment of deep infection after shoulder arthroplasty. J Shoulder Elb Surg. 2017;26:1978–83. https://doi.org/10.1016/j.jse.2017.05.005.
22. Stone GP, Clark RE, O'Brien KC, et al. Surgical management of periprosthetic shoulder infections. J Shoulder Elb Surg. 2017;26:1222–9. https://doi.org/10.1016/j.jse.2016.11.054.
23. Clouthier AL, Hetzler MA, Fedorak G, et al. Factors affecting the stability of reverse shoulder arthroplasty: a biomechanical study. J Shoulder Elb Surg. 2013;22:439–44. https://doi.org/10.1016/j.jse.2012.05.032.
24. Routman HD. The role of subscapularis repair in reverse total shoulder arthroplasty. Bull Hosp Jt Dis. 2013;71(Suppl 2):108–12.
25. Edwards TB, Williams MD, Labriola JE, et al. Subscapularis insufficiency and the risk of shoulder dislocation after reverse shoulder arthroplasty. J Shoulder Elb Surg. 2009;18:892–6. https://doi.org/10.1016/j.jse.2008.12.013.
26. Gulotta LV, Choi D, Marinello P, et al. Humeral component retroversion in reverse total shoulder arthroplasty: a biomechanical study. J Shoulder Elb Surg. 2012;21:1121–7. https://doi.org/10.1016/j.jse.2011.07.027.
27. Berhouet J, Garaud P, Favard L. Influence of glenoid component design and humeral component retroversion on internal and external rotation in reverse shoulder arthroplasty: a cadaver study. Orthop Traumatol Surg Res. 2013;99:887–94. https://doi.org/10.1016/j.otsr.2013.08.008.
28. Hamilton MA, Roche CP, Diep P, et al. Effect of prosthesis design on muscle length and moment arms in reverse total shoulder arthroplasty. Bull Hosp Jt Dis. 2013;71(Suppl 2):S31–5.
29. 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. https://doi.org/10.1016/j.jse.2009.11.044.
30. Randelli P, Randelli F, Arrigoni P, et al. Optimal glenoid component inclination in reverse shoulder arthroplasty. How to improve implant stability. Musculoskelet Surg. 2014;98(Suppl 1):15–8. https://doi.org/10.1007/s12306-014-0324-1.
31. Chalmers PN, Hammond J, Juhan T, Romeo AA. Revision posterior shoulder stabilization. J Shoulder Elb Surg. 2013;22:1209–20. https://doi.org/10.1016/j.jse.2012.11.019.
32. Gutiérrez S, Walker M, Willis M, et al. Effects of tilt and glenosphere eccentricity on baseplate/bone interface forces in a computational model, validated by a mechanical model, of reverse shoulder arthroplasty. J Shoulder Elb Surg. 2011;20:732–9. https://doi.org/10.1016/j.jse.2010.10.035.
33. Stephens BF, Hebert CT, Azar FM, et al. Optimal baseplate rotational alignment for locking-screw fixation in reverse total shoulder arthroplasty: a three-dimensional computer-aided design study. J Shoulder Elb Surg. 2015;24:1367–71. https://doi.org/10.1016/j.jse.2015.01.012.
34. Lévigne C, Boileau P, Favard L, et al. Scapular notching in reverse shoulder arthroplasty. J Shoulder Elb Surg. 2008;17:925–35. https://doi.org/10.1016/j.jse.2008.02.010.
35. Simovitch RW, Zumstein MA, Lohri E, et al. Predictors of scapular notching in patients managed with the Delta III reverse total shoulder replacement. J Bone Joint Surg Am. 2007;89:588–600. https://doi.org/10.2106/JBJS.F.00226.
36. Mizuno N, Denard PJ, Raiss P, Walch G. The clinical and radiographical results of reverse total shoulder arthroplasty with eccentric glenosphere. Int Orthop. 2012;36:1647–53. https://doi.org/10.1007/s00264-012-1539-0.
37. Poon PC, Chou J, Young SW, Astley T. A comparison of concentric and eccentric glenospheres in reverse shoulder arthroplasty: a randomized controlled trial. J Bone Joint Surg Am. 2014;96:e138. https://doi.org/10.2106/JBJS.M.00941.
38. 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. https://doi.org/10.1016/j.jse.2004.09.010.
39. de Wilde LF, Poncet D, Middernacht B, Ekelund A. Prosthetic overhang is the most effective way to prevent scapular conflict in a reverse total shoulder prosthesis. Acta Orthop. 2010;81:719–26. https://doi.org/10.3109/17453674.2010.538354.

Reverse Shoulder Arthroplasty: How to Prevent Failure

23

Eric Petroff and Johnathan Edwards

23.1 Introduction

The indications for shoulder arthroplasties have expanded in the last 10 years. In France, the total number of shoulder arthroplasties performed was 7022 in 2006 to 15,684 in 2015. In many European countries, reverse shoulder arthroplasties (RSA) are used more often than traditional anatomical designed arthroplasties. In the USA, there were 21,940 RSA procedures performed in 2011.

RSA has been successful at minimizing pain and maximizing function for many patients with rotator cuff-deficient shoulders. Because of the success of RSA, the number of complications and revisions has increased as well. Complication rates for RSA are reported as high as 68% with substantially higher complication rates observed in revision surgery [1]. Management of complications associated with RSA is challenging. Surgical revision of a failed or complicated RSA is a high-risk surgery; Boileau showed that 30% of such patients had subsequent complications after reoperation and needed further surgical interventions [2].

Increased use of primary RSA has led to reports of associated problems unique to the procedure. The most common complications include scapular notching, glenohumeral dislocation, mechanical baseplate failure, scapular fracture, loss of external rotation, nerve injury, and infection [3].

Grammont developed his prototype of the RSA in 1985. The Grammont reverse shoulder prosthesis is a semi-constrained traditional implant used in RSA. Complication rates associated with the original Grammont prosthesis were higher than prosthesis of conventional anatomic replacement. Significant efforts have been made to refine surgical implantation method and prosthesis design to decrease complication rates. Several RSA systems are available from various manufacturers with their own specifications. Variables such as neck-shaft angle of the humerus, glenosphere diameter, eccentricity and lateral offset, glenoid baseplate tilt, and component fixation are known to influence clinical outcome and can vary significantly in different implant designs and surgical approaches [1]. Knowledge of these various designs is an important factor in the management of complications and revisions.

23.2 Scapular Notching: How to Avoid It?

Scapular notching is a recognized consequence of RSA and is a mechanical impingement between the humeral component and the lateral pillar of the scapula. Inferior scapular notching is a well-documented complication that is

E. Petroff (✉) · J. Edwards
Clinique de Flandre, Coudekerque Branche, France
e-mail: petroff.eric@neuf.fr

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_23

observed on 16–96% of postoperative radiographs and has a potential impact on clinical outcomes. Scapular notching is commonly classified using the Nerot-Sirveaux grading system [4], which quantifies the loss of inferior glenoid neck bone based on the Grammont design. Several design modifications to the Grammont design have been proposed to decrease scapular notching.

Polyethylene (PE) wear due to scapular conflict is a common finding in RSA and may explain the variability observed in notching evolution. Chemical osteolysis could be related to PE wear particles because of this mechanical impingement. Progression is controversial: some authors have reported that it appears during the first year and then stabilizes [5], whereas for others the frequency and the severity of notching have been observed to increase with time [6].

Surgical technique is an important factor in RSA and greatly influences long-term postoperative clinical outcomes. Factors such as glenoid position and erosion and tilt of the sphere also affect the outcome.

The deltopectoral approach allows for better exposure of the inferior part of the glenoid and better positioning of the baseplate. It is well recognized that it is more difficult to implant the baseplate inferiorly with downward tilt using a superolateral approach [7].

Inferior glenoid positioning is perhaps the most important factor in influencing surgical outcome [6]. The ideal location of the drill hole for the baseplate post is 11.5 ± 1.0 mm above the inferior glenoid rim. Kelly et al. [8] concluded that drilling the baseplate posthole 12 mm above the inferior glenoid rim (the 12-mm rule) results in excellent glenosphere position in most cases (Fig. 23.1).

Preoperative superior erosion of the glenoid may also influence surgical outcome in RSA. The preoperative type of the glenoid erosion (types E2 and E3 according to Favard) influences surgical positioning of the baseplate. Spontaneous, upward rotation of the scapula in the coronal plane has also been recognized as a risk factor for notching because of the resulting inappropriate superior tilt of the glenoid side [4, 9].

An inferior tilt of the glenosphere (10°) prevents contact between humerus and scapula in adduction. Some authors do not recognize a biomechanical or clinical influence of inferior tilt. Although inferior glenoid positioning is the most important factor, inferior tilting helps to prevent notching; moreover inferior tilting helps to prevent superior tilting, which is always detrimental for notching and baseplate stability [10].

23.2.1 Glenosphere Design

The lateralization of the glenosphere may be dependent on three factors: the sphere design itself, the baseplate component, or the bony increased-offset (BIO-RSA), as described by Boileau et al. [11] (Fig. 23.2). It seems that the frequency of notching decreases with the amount of lateralization. Designs with prosthetic lateralization have lower rates of notching compared to traditional Grammont designs. However, excessive lateralization may cause glenoid loosening due to the lever arm that is created upon anchoring of the glenoid.

Eccentric spheres are another option to lower the position of the sphere and clear the lateral scapular pillar [12, 13]. Glenosphere eccentricity may be achieved by shifting the glenosphere center of rotation without altering the position of the baseplate. Inferior eccentricity of the glenosphere may mitigate adduction impingement by shifting the glenohumeral joint center of rotation inferiorly.

Prosthetic overhang is the most effective way to prevent scapular conflict in a RSA. An inferior prosthetic overhang of 5 mm resulted in a gain in notch angle of 39°. A prosthetic overhang therefore seems to be an important

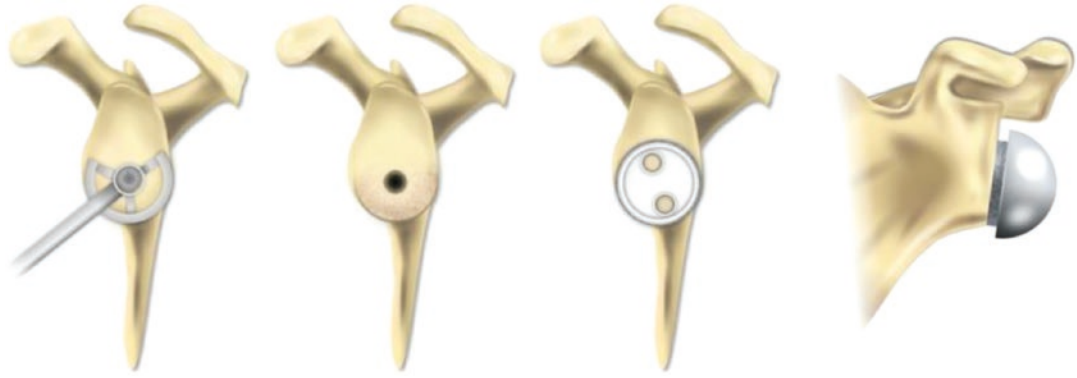

Fig. 23.1 Inferior glenoid placement of the baseplate

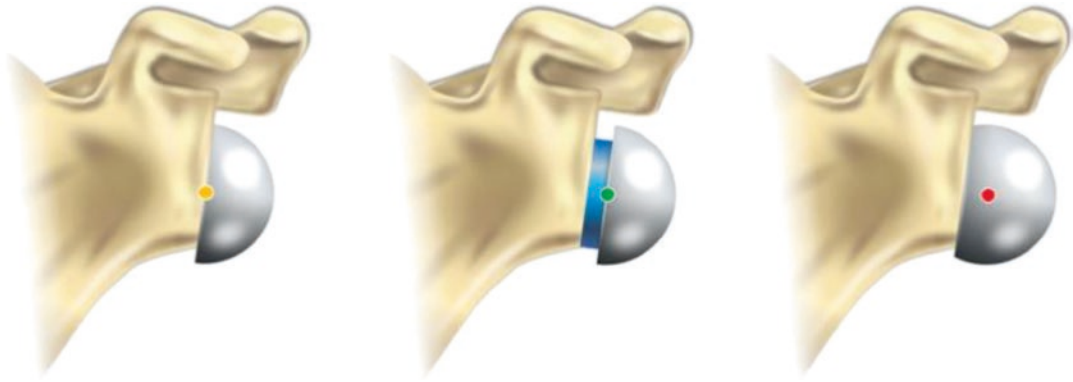

Fig. 23.2 Lateralization may be part of the sphere design or related to baseplate component or via bony increased-offset

factor to consider when performing a RSA (Fig. 23.3) [7, 12, 14].

Larger glenosphere (42 mm diameter) has been described by Guttierez et al. [13]. They showed in a computerized model that increased glenosphere diameter resulted in a greater "impingement-free" range of motion. A decreased inclination angle of 155° of the humeral cup may also decrease the risk of scapular conflict. The Grammont design increases the potential for contact between the humerus and scapula. While, a lower inclination angle significantly decreases the risk of scapular conflict, but it also increases the risk of prosthetic instability [13, 15].

23.3 Factors Affecting Initial Fixation of the Glenoid Component

23.3.1 The Glenoid Shape

Walch et al. [16] have previously classified glenoid morphology in cases of advanced glenohumeral osteoarthritis based on the preoperative computed tomography scans of individuals undergoing shoulder arthroplasty. Reconstruction of the B2, B3, or C glenoid presents a challenging clinical problem that has been associated with poor clinical outcomes and implant survivorship.

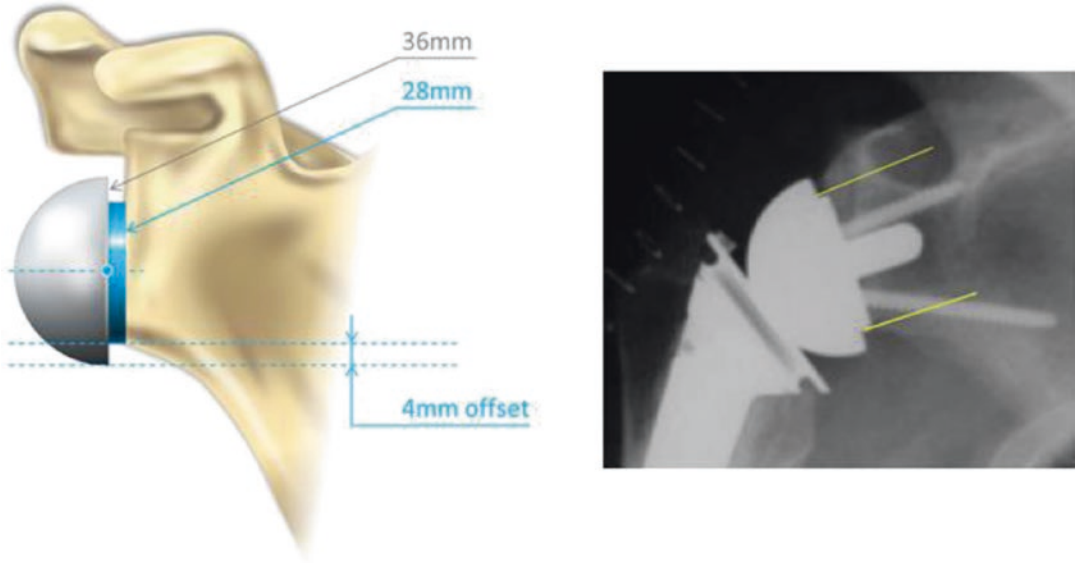

Fig. 23.3 Prosthetic overhang is the most effective way to prevent scapular conflict in a RTSA

Careful preoperative planning is essential for accurate preparation and execution of the optimal surgical plan. The more severe deformities may require posterior glenoid bone grafting and/or augmented implants to restore native anatomy. In primary RSA with glenoid bone loss, humeral head autograft can be used in the majority of cases with good results. The bone defect can be corrected with humeral head autograft as described by Boileau with a modified BIO technique to provide a larger asymmetric graft [17].

23.3.2 Bioactive Metal

The development of porous metals and coatings has revolutionized the field of orthopedics. However, most implants are fabricated utilizing traditional materials (i.e., sintered beads, fiber metal, plasma spray), which have several inherent limitations. Several new porous metals have been recently introduced to improve upon the biomaterial properties of these traditional metals. Tritanium (Stryker, Mahwah, NJ), Regenerex (Biomet, Warsaw, IN), Stiktite (Smith and Nephew, Memphis, TN), and Trabecular Metal (Zimmer, Warsaw, IN) are currently available for use in orthopedic surgery, all with a characteristic appearance similar to cancellous bone. The open-cell structure of these materials affords several intriguing properties, including high volumetric porosity (60–80%), low modulus of elasticity, and high frictional characteristics. The following represents a review of the biomaterial properties and applications in orthopedic surgery for this new class of highly porous metals (Fig. 23.4) [18].

23.3.3 Post or Central Screw?

The baseplate is typically stabilized to the glenoid via a press-fit central post or a central screw. A central bi-cortical cancellous screw can be used for added compressive fixation strength. Frankle et al. [19] demonstrated that glenoid baseplates with a central screw facilitated compression (tenfold) and had an increase in load to failure (2.3-fold) over those implants with only a post. The goal is osseous ingrowth into baseplate (AO principle).

23.4 Evolution of Humeral Stems

The design of humeral implants for shoulder arthroplasty has evolved over the years. The new-generation modular shoulder prostheses have an anatomical humeral stem that replicates the three-dimensional parameters of the proximal humerus.

Fig. 23.4 Trabecular Metal on the contact surface of the glenoid baseplate

An anatomical reconstruction is the best way to restore stability and mobility of the prosthetic shoulder and improve implant durability.

Complications relating to the humeral component (such as loosening and stress shielding of the humeral stem or periprosthetic fractures) are much less common (having an incidence of approximately 1% according to the literature). Regarding the long-term outcome of shoulder replacement, the failure rate increases over time and is directly in line with failure rates of hip and knee replacements.

A well-fixed humeral component may need to be removed for several reasons including infection, component malposition, humeral fracture, and glenoid exposure. The removal of a well-fixed humeral component during the course of revision shoulder arthroplasty is a significant challenge. In order to reduce these complications, many manufacturers progressively shortened humeral stem implants. For example, the Wright Ascend (Wright, Edina, MN, USA) has stem lengths ranging from 66 to 98 mm, and the Biomet Mini (Biomet, Warsaw, IN, USA) has a stem length of 70 mm. Stem shortening, coupled with elimination of humeral cement, potentially allows easier stem removal and improved bone quality in the event of subsequent revision. Stemless arthroplasty, with complete humeral stem elimination and reliance on metaphyseal fixation, provides even greater bone preservation for possible revision. In 2015, Teissier et al. [20] reported the results of Biomet TESS implants with a minimum follow-up of 2 years. He concluded that TESS RSA provided encouraging midterm results with favorable outcomes and a low rate of complications. The stemless TESS with a reverse corolla is a reliable, less invasive system.

23.5 New Methods of Preoperative Planning for Management of Glenoid Bone Loss

Shoulder arthroplasty has evolved over the last decade with improvements in implant design and surgical instrumentation. Despite these advances, glenoid positioning in shoulder arthroplasty continues to be a difficult problem. Recent advances in three-dimensional imaging techniques and the use of computer planning software may potentially address some of the common difficulties encountered by surgeons (Fig. 23.5). The addition of patient-specific instrumentation (PSI) and guides provide an option for patients with significant glenoid deformity that may allow improved accuracy of glenoid component implantation compared to using standard instrumentation.

The concept of using three-dimensional imaging developed into planning software to simulate glenoid component implantation using three-dimensional computer software. Iannotti et al. [21] demonstrated improved glenoid orientation with the use of preoperative planning software to guide implantation of the glenoid component. These authors were able to better place the glenoid component within 5° of desired inclination and 10° of desired version with three-dimensional templating and computer planning, compared to standard techniques in a randomized controlled trial of 46 patients.

The development of three-dimensional imaging techniques and computerized planning for shoulder arthroplasty can greatly assist with preoperative surgical planning; however, the development of PSI has been introduced to allow improved surgical implementation of the preop-

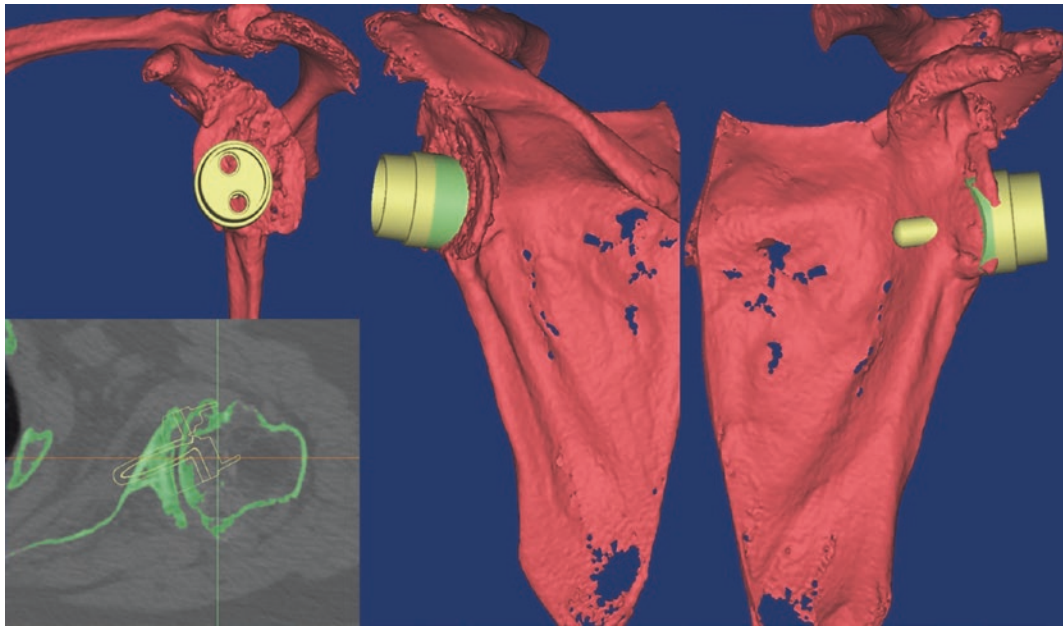

Fig. 23.5 Preoperative 3D planning management of glenoid bone loss

erative plan. The premise of PSI to guide surgeons in implanting the glenoid component is using three-dimensional reconstructions of CT scans to build custom-made jigs for guide pin placement determined by the patient's bony anatomy. Typical landmarks for guide placement include the peripheral glenoid rim or base of the coracoid. Recently, Walch et al. [22] showed excellent clinical correlation of the guide pin position using preoperative patient-specific planning to those placed in 18 cadaveric scapulae using PSI. Currently, there are several companies that provide the software and tools for preoperatively planning.

The software creates a guide to direct implantation of the prosthesis that is utilized during surgery [23]. Each system has slight differences in the planning tool as well as the shape of the guide created and landmarks used to reference the position of the guide at the time of surgery. Most systems require a CT scan of certain aspects of the shoulder joint that can then be used to create three-dimensional reconstructions for surgical planning. Most systems allow direct surgeon planning in some form as opposed to having an engineer and create the plan for the surgeon. Guides are then created based upon these plans and shipped for use during surgery with a typical delay of 3–4 weeks (Fig. 23.6). Further data is required to determine if the improved accuracy of cases guided by patient-specific instrumentation will lead to improved clinical outcomes and implant survivorship since at this point it is only speculative [20, 22, 23].

Conclusion

RSA is an evolving technique. Indications for surgery, operative technique, implant design, and the avoidance of complication are dependent on fundamental principles of biomechanics. Surgical technique and prosthesis design have a significant influence on clinical outcome of RSA and implant longevity. Scapular notching and external rotation deficit are predominantly influenced by joint center of rotation position and postoperative muscle leverage, respectively. These factors can vary substantially with implant design.

Future research and prosthetic design development will give a better understanding of many design parameters: the influence of optimum bearing surfaces, glenoid diameters, implant version/anatomy, inclination and off-

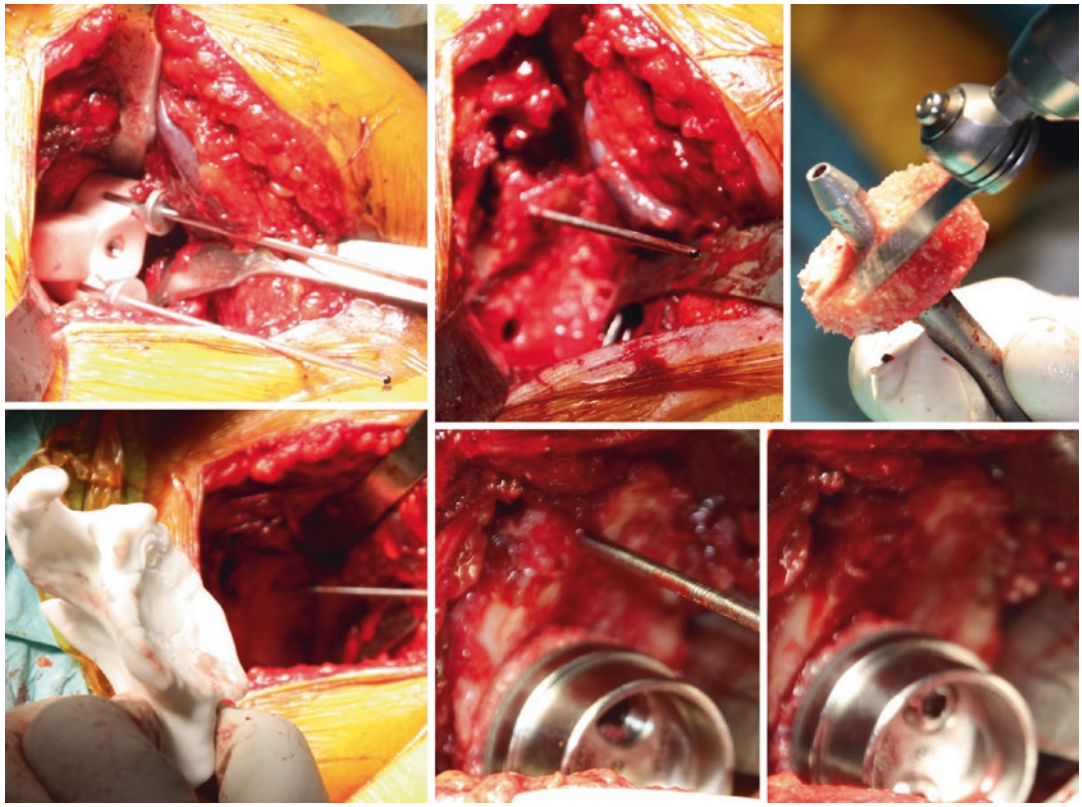

Fig. 23.6 Placement of the patient-specific guide is verified by aligning it in the correct orientation on the surrogate glenoid

set, and their effect on muscle and joint function. These design parameters are highly relevant to clinical outcome. Recent cadaveric and clinical studies using preoperative computer planning and patient-specific instrumentation report improved anatomic placement of glenoid components in TSA. This ever-evolving technology demonstrates the reliability and precision of preoperative planning software and patient-specific guides for glenoid component in total arthroplasty.

References

1. Ackland DC, Patel M, Knox D. Prosthesis design and placement in reverse total shoulder arthroplasty. J Orthop Surg Res. 2015;10:101.
2. 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.
3. 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(3):288–97.
4. Lévigne C, Nérot C, Boileau P, Sirveaux F, Molé D, Favard L. Scapular notching. Rev Chir Orthop Reparatrice Appa Mot. 2007;93(3):S74–81.
5. Simovitch R, Zumstein M, Lohri E, Helmy M, Gerber C. Predictors of scapular notching in patients managed with the Delta III reverse shoulder replacement. J Bone Joint Surg Am. 2007;89:588–600.
6. Lévigne C, Garret J, Boileau P, Alami G, Favard L, Walch G. Scapular notching in reverse shoulder arthroplasty: is it important to avoid it and how? Clin Orthop Relat Res. 2011;469:2512–20.
7. Lévigne C, Boileau P, Favard L, Garaud P, Molé D, Sirveaux F, Walch G. Scapular notching in reverse shoulder arthroplasty. J Shoulder Elb Surg. 2008;17:925–35.
8. Kelly JD, Humphrey CS, Norris TR. Optimizing glenosphere position and fixation in reverse shoulder arthroplasty. J Shoulder Elb Surg. 2008;17:589–94.
9. Favard L, Lautmann S, Sirveaux F, Oudet D, Kerjean Y, Huguet D. Hemiarthroplasty versus reverse arthroplasty in the treatment of osteoarthritis with massive rotator cuff tear. In: Walch G, Boileau P, Molé D, edi-

tors. 2000 Prothèses d'épaule… recul de 2 à 10 ans. Paris: Sauramps Medical; 2001. p. 261–8.
10. Denard PJ, Lederman E, Parsons BO, Romeo AA. Finite element analysis of glenoid-sided lateralization in reverse shoulder arthroplasty. J Orthop Res. 2016;17:1–8.
11. Boileau P, Moineau G, Roussanne Y, O'Shea K. Bony increased-offset reversed shoulder arthroplasty: minimizing scapular impingement while maximizing glenoid fixation. Clin Orthop Relat Res. 2011;469(9):2558–67.
12. de Wilde LF, Poncet D, Middernacht B, Ekelund A. Prosthetic overhang is the most effective way to prevent scapular conflict in a reverse total shoulder prosthesis. Acta Orthop. 2010;81(6):719–26.
13. Guttierrez S, Comiskey CA IV, Luo ZP, Pupello DR, Franckle MA. Range of impingement-free abduction and adduction deficit after reverse shoulder arthroplasty. Hierarchy of surgical and implant design-related factors. J Bone Joint Surg Am. 2008;90:2606–15.
14. Cuff D, Clark R, Pupello D, Frankle M. Reverse shoulder arthroplasty for the treatment of rotator cuff deficiency: a concise follow-up, at a minimum of five years, of a previous report. J Bone Joint Surg Am. 2012;94:1996–2000.
15. Kempton LB, Balasubramamiam M, Ankerson E, Wiater JM. A radiographic analysis of the effects of prosthesis design on scapular notching following reverse total shoulder arthroplasty. J Shoulder Elb Surg. 2011;20:571–6.
16. Walch G, Badet R, Boulahia A, Khoury A. Morphologic study of the glenoid in primary glenohumeral osteoarthritis. J Arthroplast. 1999;14(6):756–60.
17. Hendel MD, Werner BC, Camp CL, Gulotta LV, Walch G, Dines DM, Dines JS. Management of the biconcave (B2) glenoid in shoulder arthroplasty: technical considerations. Am J Orthop (Belle Mead NJ). 2016;45(4):220–7.
18. Levine B. A new era in porous metals: applications in orthopaedics. Adv Eng Mater. 2008;10(9):788–92.
19. Frankle MA, Pupello D, Cuff D. Treating the rotator cuff-deficient shoulder: the Florida Orthopedic Institute experience. In: Frankle MA, editor. Rotator cuff deficiency of the shoulder 1. New York: Thieme Medical Publishers; 2008. p. 147–70.
20. Teissier P, Teissier J, Kouyoumdjian P, Asencio G. TESS RSA provided encouraging midterm results with favorable outcomes and a low rate of complications. The stemless TESS with a reverse corolla is a reliable, less invasive system. J Shoulder Elb Surg. 2015;24(1):45–51.
21. Iannotti JP, Weiner S, Rodriguez E, Subhas N, Patterson TE, Jun BJ, et al. Three-dimensional imaging and templating improve glenoid implant positioning. J Bone Joint Surg Am. 2015;97:651–8.
22. Walch G, Vezeridis PS, Boileau P, Deransart P, Chaoui J. Three- dimensional planning and use of patient-specific guides improve glenoid component position: an in vitro study. J Shoulder Elb Surg. 2015;24:302–9.
23. Wylie JD, Tashjian RZ. Planning software and patient-specific instruments in shoulder arthroplasty. Curr Rev Musculoskelet Med. 2016;9:1–9.

Reverse Shoulder Arthroplasty: How to Manage Failure

24

Srinath Kamineni

24.1 Introduction

Since its introduction as a treatment for the unreconstructable rotator cuff tear and cuff tear arthropathy in the early 1970s by pioneers like Lipmann Kessel and Ian Bayley [1] in England and Paul Grammont [2] in France, the reverse shoulder arthroplasty (RSA) has become a mainstay of treating these pathologies. With increasing success has followed increasing usage, expansion of indications, and the inevitable increase in complications and revision surgeries [3]. Zumstein et al., in their meta-analysis of current literature, reported an overall complication rate of 20% and that 13% of RSA operations either needed surgical revision of the implants or reoperations [3]. They also concluded that primary RSA was much more successful (fewer complications) than a conversion from an anatomic total shoulder arthroplasty (more complications) by a factor of 1:3.

There appears to be several reasons why the RSA, which represents a true technological breakthrough, has become so popular, which include the changing education of surgeons in training, fee for service revenue models, the "ease" of the surgical technique, and external influences by corporations and the litigation system. Although many of these issues are outside the scope of this chapter, some of these common themes will be mentioned. But, before embarking on a management strategy for a failed operation, it is worth considering whether the failure was due to a surgeon-related technical error, an implant design-related failure, or a general failure related to any arthroplasty. It should however be noted that many of these factors coexist when an operation fails, but it is equally important to identify such components of the failure. Several papers document long lists of causes for the failure of a RSA. However, the vast majority of complications can be grouped into six major categories (Table 24.1) and will be discussed individually.

Table 24.1 Commonest reasons for RSA failure

Instability
Septic complications
Component loosening
Component disassembly
Fractures
General complications

24.2 Instability

The single commonest cause for a revision procedure following a RSA is prosthetic instability (Fig. 24.1). Many causes have been identified as factors and often may coexist. Factors thought to be responsible include loss of articular constraint, previous surgeries, surgical approach, implant

S. Kamineni
University of Kentucky, Shoulder Elbow Research Centre, Orthopaedics and Sports Medicine, Lexington, KY, USA

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_24

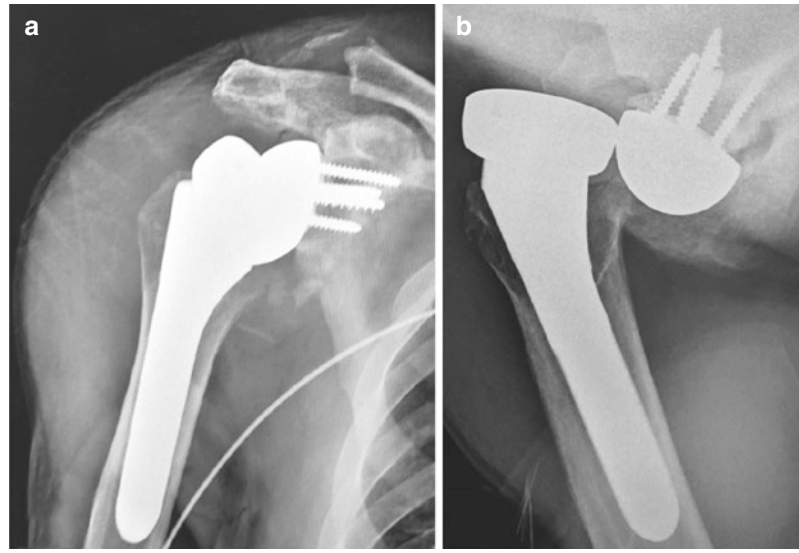

Fig. 24.1 Anteriorly dislocated RSA. (**a**) AP radiograph, (**b**) axillary radiograph

malposition, and causes of altered kinematics. The surgical approach can affect the orientation of component implantation, with a direct superolateral approach, affording a more direct end-on glenoid view. This more direct view may be the reason why this approach has a lesser association with instability (0%) compared to the deltopectoral approach (10%) [4].

Previous surgeries, e.g., failed ORIF/arthroplasty, that are converted to RSA, when compared to a primary RSA, have a three times greater instability rate [4]. These previous surgeries are associated with many important features, notably scar tissues and altered quality of remaining muscle and bone. These altered tissues may well play a significant role in the constraint and behavior of the RSA, thereby influencing instability. Constraint is also affected due to humeral axis shortening, loss of humeral bone stock, distal malposition of the stem, humeral component subsidence or proximally malpositioned (Fig. 24.2), or when the deltoid lever arm is too medialized (due to glenoid bone loss or a smaller glenosphere) [5, 6]. Constraint can also be decreased if the humeral tray is malrotated, which can be readily resolved by component reorientation (Fig. 24.3). Loss of anterior stability can also be the result of subscapularis failure and anterior unipennate deltoid atrophy [6].

When attempting to manage an unstable RSA, understanding the cause of instability is paramount and can broadly be separated into early (<12 weeks) or late (>12 weeks) instability. Moreover, it should be noted that instability is not only a frank dislocation but also when the patient complains of clunking, and apprehension, suggestive of subluxation or maltracking in certain directions.

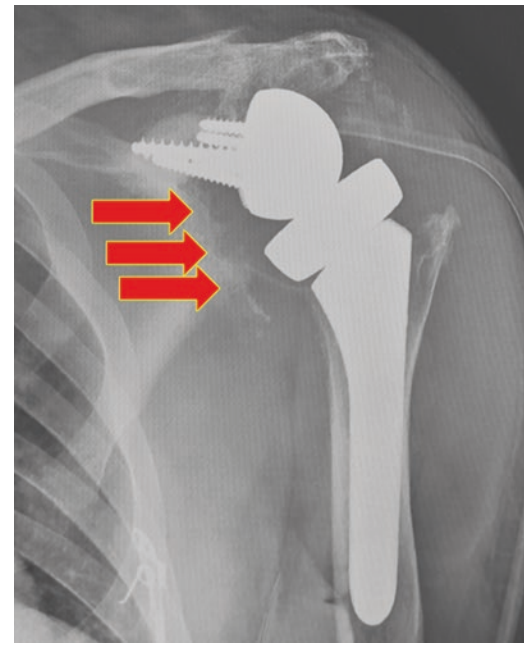

Fig. 24.2 Technical error with a superiorly placed glenosphere leading to notching, and pain, requiring a revision surgery

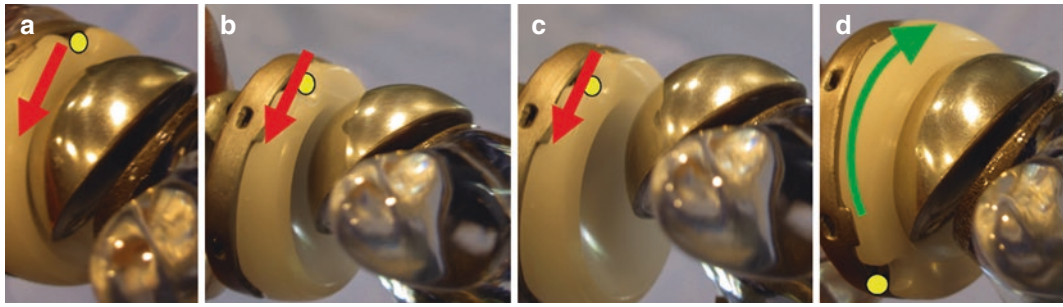

Fig. 24.3 (**a**–**c**) A model demonstration of anterior instability due to component malposition, (**d**) resolved with component reorientation

24.2.1 Management of Early (<12 Week) Instability

An issue with early instability is how aggressive to investigate for bone loss or component malposition. If plain radiographs do not show any obvious deficit, it is reasonable to reduce a dislocated RSA under sedation-anesthesia. A vital part of a reduction procedure is to reduce the joint without excessive force, especially axial twisting, for fear of creating a peri-prosthetic fracture. An equally important and often undermentioned aspect is the subsequent examination under anesthesia (EUA) of the reduced joint to understand the forces and direction required to dislocate/sublux the joint. The findings from the EUA will help to guide the position in which the shoulder needs to be maintained for the next 4–6 weeks, while the soft tissues heal and stabilize the joint. It should be borne in mind that although there may be some real mechanical issues (bone loss or component malpositioning) that could in other circumstance be revised, the majority (59–62%) of cases can stabilize and function well with conservative measures [6, 7].

24.2.2 Management of Late (>12 Week) Instability

If a dislocation occurs after 3 months, or remains unstable with recurrent subluxations, the likelihood of resolving this situation without further surgery is minimal. The two major components to be factored into a subsequent surgical solution are humeral length and/or medialization of the glenosphere. When considering the next step, regardless if the bony anatomy is preserved or not, it is worth acquiring comprehensive imaging studies to assess the position of the greater tuberosity and the total humeral length, as measured by the contralateral limb. These parameters need to be accounted in relation to the deltoid tension.

If the predominant issue is a loss of humeral length, the humeral axis can be lengthened by increasing the polyethylene liner thickness and or using a larger and eccentrically placed glenosphere as inferior as possible, both of which, individually or in combination, may be sufficient to gain stability without the need for humeral component revision. This strategy tends be more useful for less severe humeral bone loss cases.

For cases with greater instability, and significant humeral length deficits, the humeral stem may need to be revised and lengthened, or for massive bone loss cases, a tumor prosthesis and an allograft-prosthetic composite are viable options.

When the issue is determined to be an excessively medialized glenosphere, which presents as continued instability despite correcting humeral length, the strategy has to lateralize the glenosphere. The lateralization allows the deltoid to improve its "wrapping angle," thereby improving stability. Depending on the severity of the case, switching a smaller glenosphere for a larger laterally offset glenosphere may suffice in minor cases, progressing to replacing the proximal humerus bone stock and further lateralizing the glenosphere

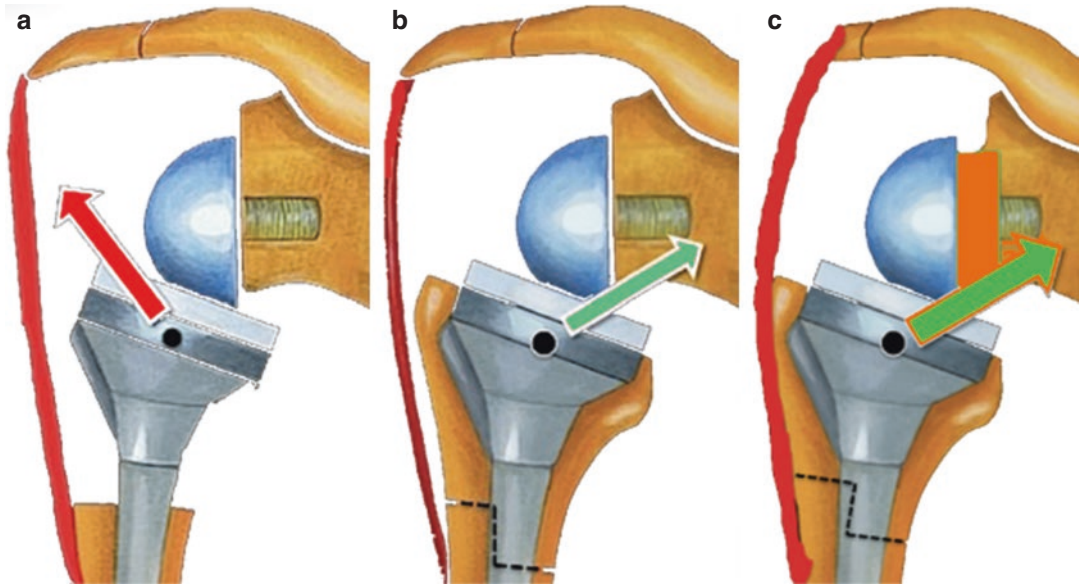

Fig. 24.4 Effect of the deltoid wrapping angle, in relation to the lateralization of the glenosphere and replacing the proximal humeral bone loss (modified from Ref. [10]). (**a**) Deltoid vector directed to cause instability, (**b**) replacement of the proximal humeral bone loss aids to improve the wrapping angle, (**c**) lateralization by using a larger glenosphere and structural bone graft further improves the deltoid wrapping angle and stability

using structural bone graft (Fig. 24.4). Deltoid tension is an important feature of instability, but it is more difficult to correctly gauge when muscle relaxation is used during anesthesia, although muscle relaxation improves surgical exposure. Additionally, the lateralization of the glenosphere allows the deltoid to improve its tension and its coaptation force, thereby aiding stability [8, 9].

Postoperatively, the arm should be maintained in a position of least vulnerability in abduction for a minimum of 4–6 weeks, allowing the deltoid to shrink to its new length.

24.3 Septic Complications

Infection is one of the most devastating causes of RSA failure and is the second commonest reason for revision surgery. Common risk factors for infection include previous surgeries of the shoulder, with the risk increasing with the number of surgeries [6, 11]. Although *Staphylococcus aureus* and *Staphylococcus epidermidis* are common pathogens, propionibacterium acnes has a special predilection for the shoulder as a pathogen [12].

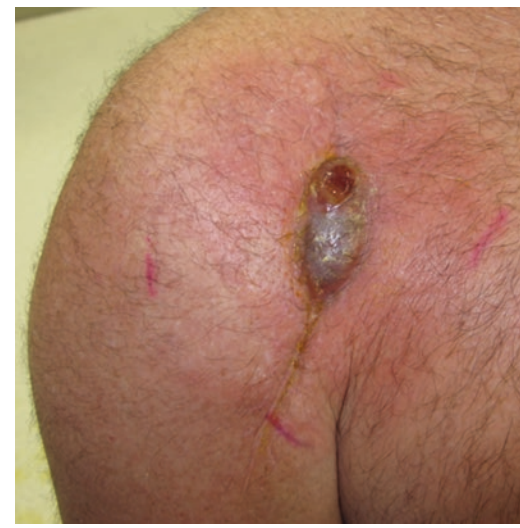

Fig. 24.5 A discharging anterior wound which was treated by a medical team with antibiotics for a month prior to an orthopedic referral. The organism was cultured from a wound swab

Whereas obtaining the diagnosis may be straightforward in some cases (Figs. 24.5 and 24.6), in others a tissue sample is required from the operative intervention. The strategy for managing RSA

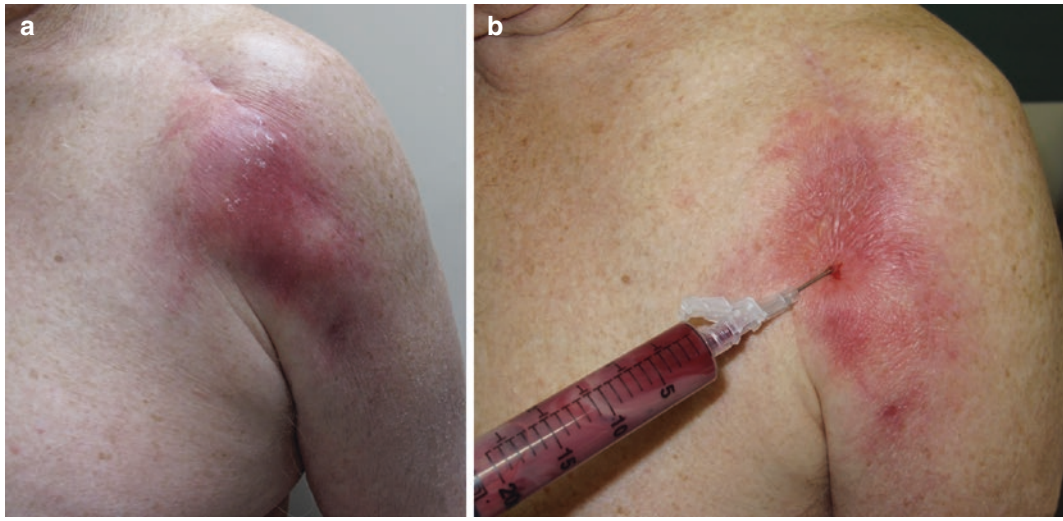

Fig. 24.6 (**a**) Clinical presentation of an infected hematoma after shoulder arthroplasty in a warfarinized patient, (**b**) aspiration and culture revealed *Staphylococcus aureus*, successfully treated with early aggressive wound debridement and antibiotics, without the need for component explantation

infections is based on timing of the infective process, specific pathogen, and the general state of the patient's health and function.

In broad terms, for infections diagnosed and treated within 3 months (acute infections), the overall goal is to preserve the implant (which is well fixed), but to aggressively decrease the infection load. This would entail extensive debridement of soft tissues and exchange of the polyethylene liner and the glenosphere and irrigation of the site (Fig. 24.7). Chronic infections (present for greater than 3 months) in general are not expected to retain the implants and have two widely accepted pathways for treatment. The implant can be removed and the wound derided, thereby resulting in a resection arthroplasty, or the implant can be initially removed, the wound derided and then secondarily re-implanted when the risk of infection is eliminated. The latter option is currently thought to be the gold standard for chronic infections, although a resection arthroplasty is to be considered if that patient is frail and not able to undergo multiple procedures or if the pathogen is resistant. The outcomes of a resection arthroplasty are poor, with a shoulder that is unstable and telescopes with muscle activation [12], but can result in a shoulder that is able to perform basic activities [13]. A third option of a simple surgical debridement and irrigation of the chronic infective site while retaining the implant in situ similar to the management of an acute infection, although not widely recommended, does have proponents [14].

When dealing with a chronic RSA infection, there is some controversy regarding whether the debridement of the infection and reimplantation of the prosthesis should be carried out in a single or multiple, commonly a two-stage, procedures.

While the primary purpose of the intervention is to eradicate infection, an important consideration is the preservation of function. Performing a one-stage procedure results in a better functional outcome, but risks a higher recurrence of infection. A one-stage procedure can be considered in medically compromised patients with a preoperatively known and treatable pathogen, which is often a less common circumstance [12, 15, 16]. However, the two-stage procedure is still considered the gold standard, and this is my recommended approach, while using the normalization of the ESR/CRP/white cell count as adjunctive parameters in deciding that the second stage is definitive. It should be noted that the two-stage procedure has a lesser functional outcome and a higher complication rate, but is more predictable for the eradication of deep infection.

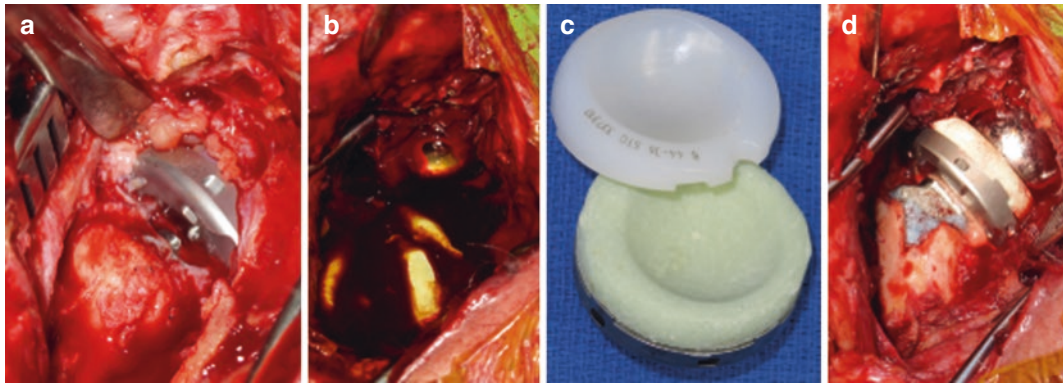

Fig. 24.7 Early infection was identified in a previously infected contralateral shoulder demonstrating a hybrid technique. (**a**) Thorough debridement, (**b**) 10-min agitated Betadine soak with cement rods impregnated with vancomycin and tobramycin, (**c**) the polyethylene is replaced with a custom cement liner impregnated with vancomycin and tobramycin, (**d**) the joint re-articulated with cement spacer

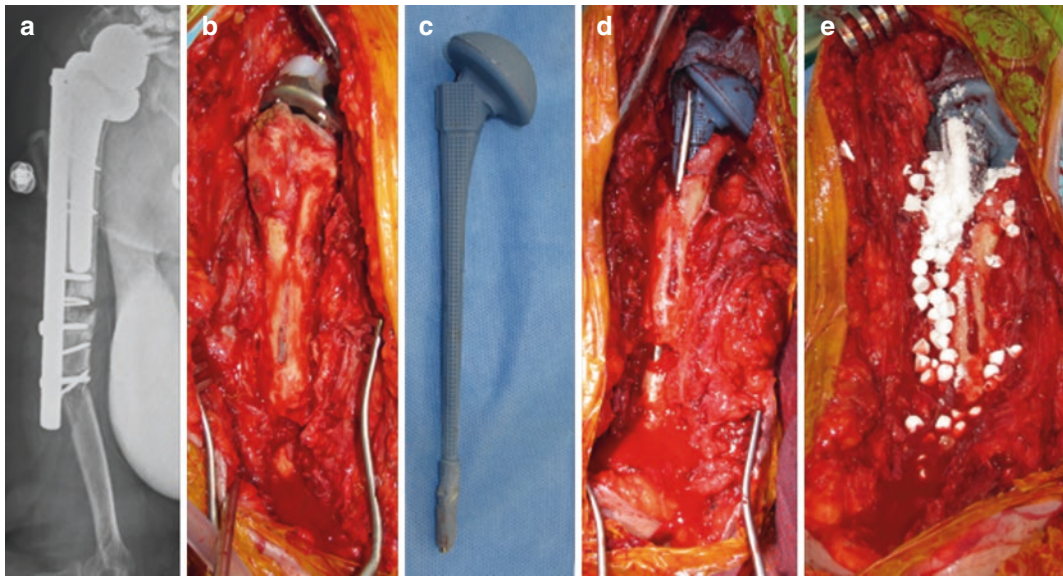

Fig. 24.8 (**a**) Infected peri-prosthetic fracture of a multiply operated RSA, (**b**) cerclage wires removed, cloacae visible, (**c**) a modified vancomycin and tobramycin impregnated ZB Prostalac spacer, (**d**) Prostalac replacement with Steinman pin augmentation for fracture stabilization, (**e**) calcium phosphate beads impregnated with vancomycin placed in surgical site prior to closure

Often, with a commonly used two-stage procedure, when an extensive debridement is undertaken, and the original prosthesis is removed, a cement spacer is employed to maintain the soft tissue space. These spacers are often antibiotic loaded, but the literature does not support their use to improve clinical outcomes, but they appear to improve infection control [17, 18] (Fig. 24.8).

The removal of a well-fixed implant can pose some difficulties but, with the right approach and equipment, tends to be a very achievable challenge. After an extensive wound debridement, and scar release, the joint can be dislocated, and depending on the modularity of the system being revised, modular components can be sequentially removed (e.g., humeral polyethylene tray, followed by the glenosphere). Removing a

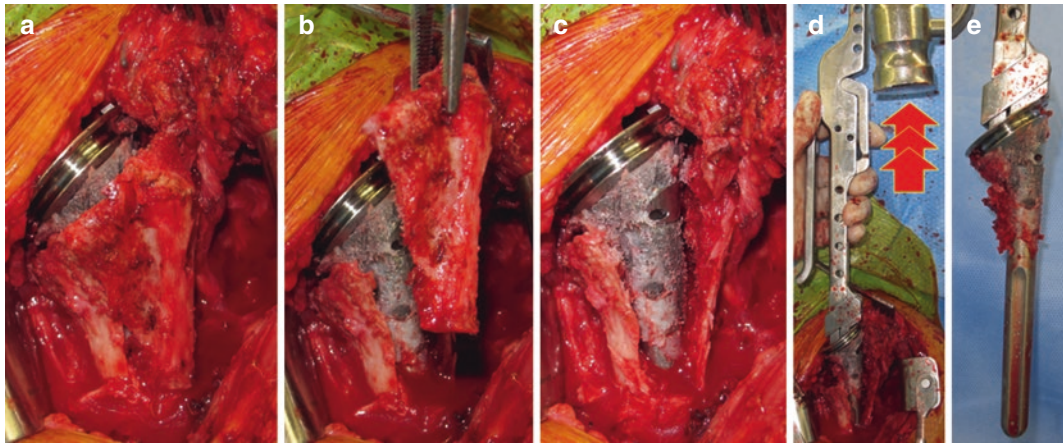

Fig. 24.9 When a stem is well fixed, but needs to be removed during an infected RSA revision, a planned humeral window is useful for assistance with stem extraction, minimizing the risk of iatrogenic fracture. (**a**) A well-fixed humeral stem, (**b**) a planned anterior humeral window is created, (**c**) the humeral window is removed allowing osteotome access to the ingrowth surface of the stem, (**d**) the loosened stem is grasped with a specific handle and a back-slap hammer is used to extract it from the humerus, (**e**) the extracted humeral stem

well-cemented or biologically fixed humeral stem may require flexible osteotomes, ultrasonic cement removal, or a controlled humeral window [19] (Fig. 24.9). A glenosphere baseplate is more problematic since those designs with a significant ingrowth/ongrowth keel/post will potentially cause collateral extraction of glenoid bone stock, and great care should be paid to minimize this bone loss.

24.4 Aseptic Component Loosening

24.4.1 Humeral Component Loosening

Aseptic component loosening is not unique to RSA, and hence is covered elsewhere, where it pertains to the generic loosening and bone loss in relation to shoulder arthroplasty. When the humerus is involved, the same strategies that are employed during a TSA revision are utilized, notably stem and cement extraction (with flexible osteotomes, ultrasonic cement removers, humeral windows), canal preparation, and impaction grafting to augment thinned cortices (Fig. 24.10), allograft/metal plate struts if cortical augmentation is desirable. Finally, long stems are reintroduced to bypass the weakened cortex, or a custom/cortical substitution stem if significant cortical loss exists, and an allograft-prosthetic composite [9] is not suitable.

A reason to reconstruct the proximal bone loss is to improve the coaptation force transmission, by improving the deltoid wrapping angle. A further consideration in the revision case is to augment the power of external rotation, since such cases often, due to the loss of the external rotator musculature, lack the ability to reach the back of their head, etc. Hence, a latissimus dorsi transfer can significantly improve functionality, even in revision cases, but is dependent on proximal bone stock for reattachment [20–22].

24.4.2 Glenoid Component Loosening

An original concern about the longevity of the RSA concept, glenoid component loosening, was because of the expected significant shear stresses predicted at the component-bone interface. Interestingly, glenoid component aseptic loosening has not been as common as feared and can mostly be ascribed to the medialized glenosphere concept championed by Grammont [2]. Although, with more lateralized designs, this

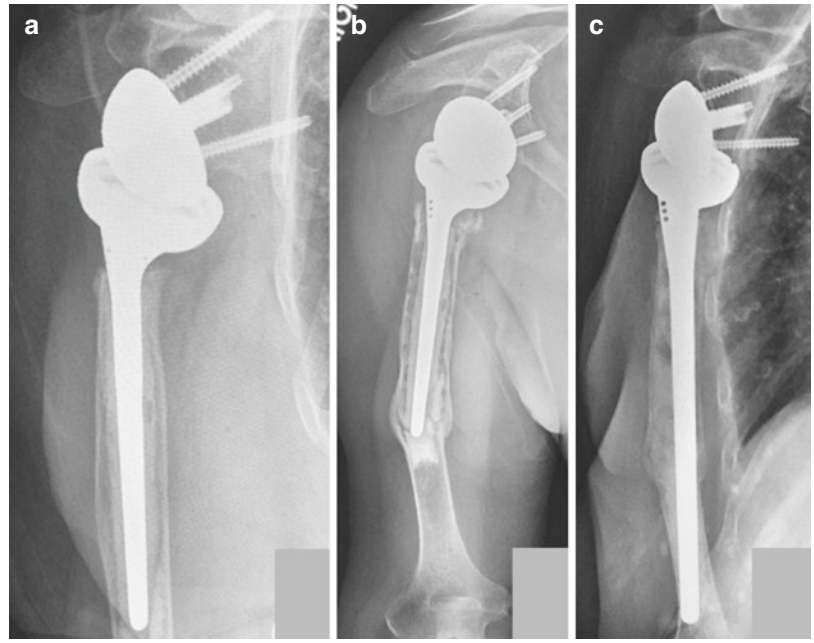

Fig. 24.10 (**a**) A radiographically loose and symptom-free humeral component, presents with acute pain after a fall. (**b**) Peri-prosthetic humeral fracture. The fracture was treated conservatively until union. (**c**) The humeral component was revised with a long stem and a combination of impaction grafting proximally and cementation for early stability distally

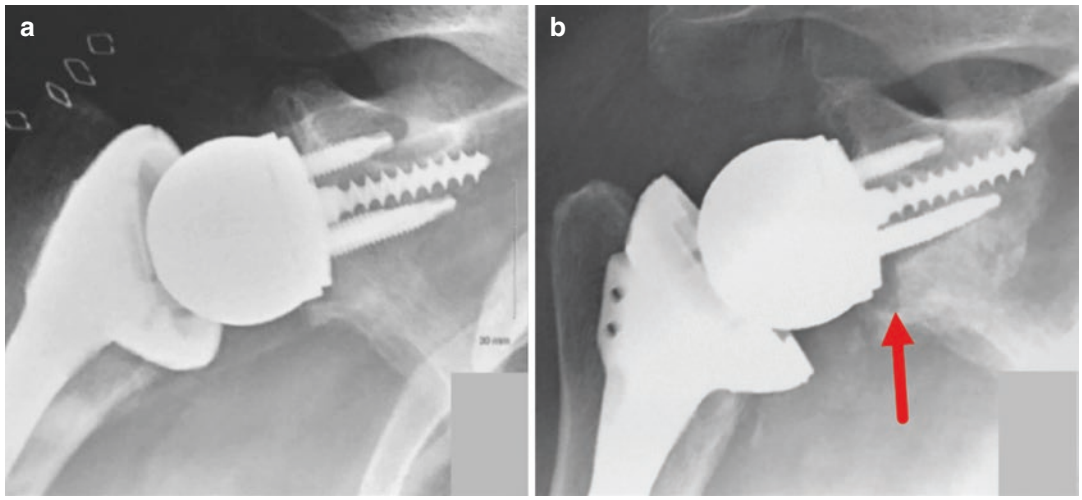

Fig. 24.11 (**a**) Immediate postoperative radiograph of a lateralized RSA. (**b**) early component loosening at 6-week follow-up in a large patient, with a well-placed glenosphere, but a lateralized design

problem can still persist (Fig. 24.11) [23], the majority of aseptic loosening is likely due to technical errors, e.g., superior inclination and placement [24], with trauma as another possibility.

When approaching a loose glenoid component, the first priority is to exclude infection by performing preoperative infection screens and sending intraoperative tissue samples for microscopy. The second most important consideration is the remaining glenoid bone stock after component removal, and hence great care and attention should be paid during this step, in order to minimize unnecessary bone loss. The remaining glenoid bone stock can be categorized as contained or uncontained (partially or complete).

Contained Defects As a general rule, when dealing with bone loss, the best way to reconstitute bone stock is with a structural graft where possible. Small contained defects can adequately be

filled with impacted cancellous bone, with stability achieved by a central screw/peg and peripheral screws that predominantly engage good host bone. For larger contained cavitary defects, a femoral head allograft, cancellous core, is shaped to fit the defect, which achieves some structural stability, and fixed into place with the baseplate central screw/peg and peripheral screws.

Uncontained Defects Whether partially or circumferentially uncontained, structural bone graft with good healing potential is required to reconstruct these glenoids, the optimal graft being a tricortical autologous iliac crest. The defect to be reconstructed can often be predicted from preoperative CT scans. Depending on the surgeon, a decision regarding a custom-made baseplate, incorporating substitution for the defect, versus a biological option, can be made preoperatively. If a biological option is chosen, often in the younger patient, the host fixation bone is used to anchor the baseplate-tricortical graft construct using screws going through the baseplate and the structural graft. The position of the baseplate should be inferior with an inferior inclination.

If the graft fixation is assured and stable, e.g., impaction with a contained defect and good fixation into host bone, without undue force exerted by the humeral component on the glenosphere, upon reduction, then the whole procedure can be completed in a single stage. If fixation into host bone is suboptimal, uncontained defect is being reconstructed, moderate force exerted on the glenoid component by the reduced humeral component, the safer option would a two-stage procedure. The second stage, reimplantation of the humeral component, would be undertaken when there is bony incorporation of the glenoid component and bone stock, e.g., after 3–6 months or after CT evidence.

24.5 Component Dissociation

Although relatively uncommon, the disassembly of an implanted prosthesis dictates a reoperation to reassemble the components. However, the reason for the dissociation has to be understood and rectified (Fig. 24.12). The reason is often multifactorial and can range from surgical technical error (non-congruous placement of glenosphere on baseplate, cross-threading of glenosphere retaining screw, interposed tissue between glenosphere and baseplate or between polyethylene liner and tray or between tray and stem, etc.) [25], implant design, or a traumatic event. When considering the revision option, the cause should be clearly defined, and if purely a traumatic event, ensure correct surgical technique during revision. If technical error is the culprit, the solution is simpler, greater diligence

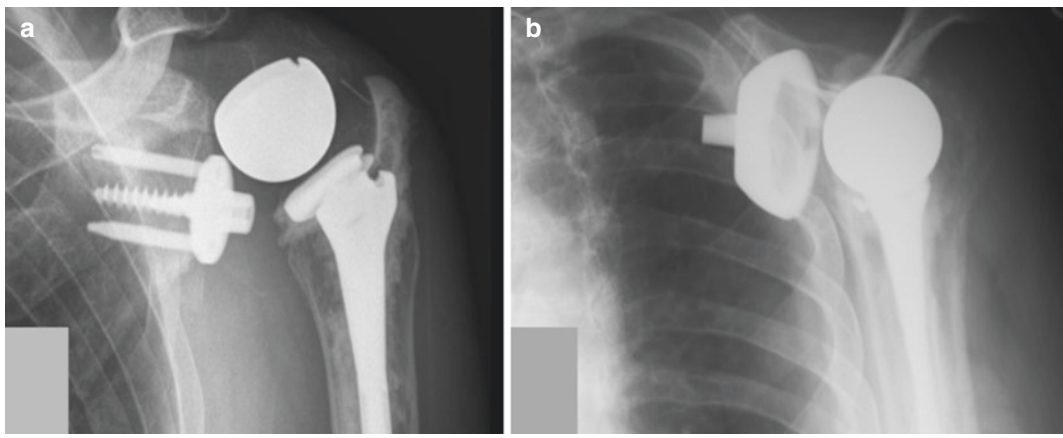

Fig. 24.12 Component failure, (**a**) glenosphere dissociation, (**b**) humeral tray dissociation (with permission from Mark Frankle, MD)

to the procedure. If however the prosthetic design/size allows edge impingement of the components, simply choosing a smaller glenosphere may transfer component impingement to a scapular notching problem. Hence, with larger glenosphere sizes, implicated in glenosphere dissociation [26], we recommend downsizing the glenosphere diameter and performing a controlled notchplasty.

24.6 Acromial Fracture

Acromial fractures are a recognized complication after RSA, generally thought to be a fatigue fracture mechanism, and can occur at any stage in the postoperative course. Risk factors include previous acromioplasty, osteoporosis [27], excessive deltoid tensioning, and direct trauma. The clinical outcomes are adversely affected by some acromial fractures, but not all. The literature is non-conclusive regarding the management, and although conservatively managed acromial fractures result in diminished outcomes, they are improved compared to the post-injury state [28]. Fracture fixation, although an option, should be weighed against the ability to predictably gain a stable fixation. However, displaced scapular spine fractures benefit from ORIF, while anterior acromial avulsions may consistently be treated with nonoperative management [29]. It should be noted that operatively treated acromial base fractures are unpredictable with respect to the final outcome and not significantly different to nonoperatively treated cases [30].

24.7 Scapular Notching

Scapular notching is a unique problem of RSA and occurs with medial and posteromedial contact between the prosthetic humeral component rim and bony scapular neck. While symptomatic, with a presentation of pain, and radiographically visible, the clinical significance of notching has been poorly understood. More recently there has been an association between scapular notching and poorer clinical outcomes [31]. Avoidance of this issue is based on a combination of prosthetic design (more lateralized glenosphere) [32] and an inferior baseplate placement [33]. However, there is no consensus regarding the management for a patient who presents with notching, with or without pain. On two (personal experience) occasions with the presentation of a well-fixed but painful scapular notching, a surgical notchplasty was performed, after optimization of the modular components (Figs. 24.13 and 24.14). When the notching is severe, not only is the polyethylene liner damaged by the contact, it generates polyethylene wear debris and possibly even metallosis with contact with the inferior glenosphere screws and/or metal rim of the humeral component. At a revision surgery, the metallosis debris is seen as a blackened synovitis which should be derided.

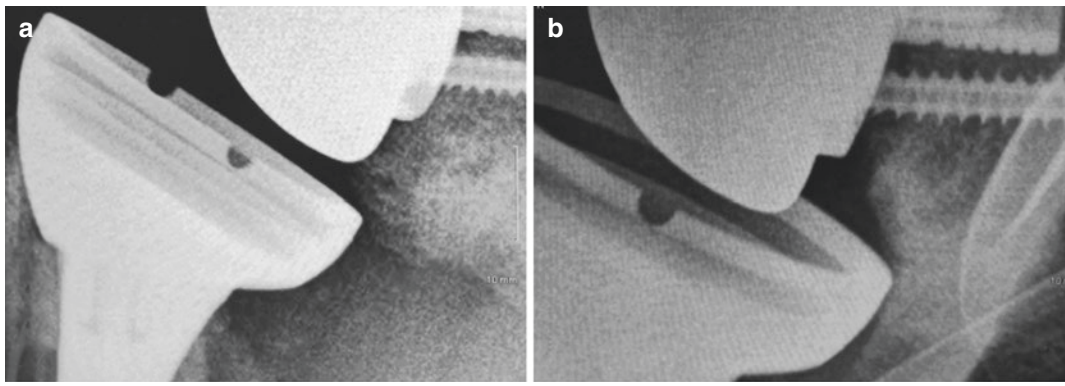

Fig. 24.13 (**a**) Medialized RSA without inferiorization to the margin of the glenoid. (**b**) After 3 years, the patient returned with painful scapular notching and grinding

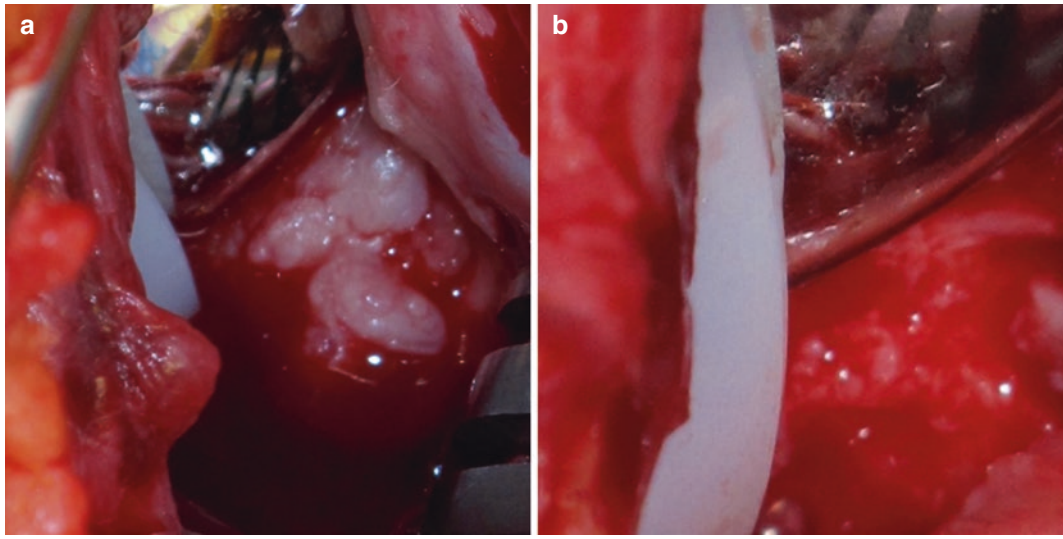

Fig. 24.14 (**a**) Painful inferior scapular notching. (**b**) Surgical "notchplasty" of inferior scapular bone. The extent of notchplasty was gauged by lack of humerus-scapular neck contact with maximal intraoperative adduction and internal-external rotation

Conclusion

Always think of infection when assessing a failed RSA, loose component(s), and performing a revision surgery, and act by obtaining preoperative infection indices (for absolute and tracking purposes).

Recommendation Always send intraoperative tissue samples from the soft tissues and from within bony cavities for microscopy, culture, and sensitivities.

Assess bone stock with respect to the pathological process and the surgical process of revision. Whereas the pathological process can diminish bones stock due to septic or aseptic loosening, the surgical process may, by necessity, further diminish the bone stock, thereby complicating the revision surgery. For example, when revising a glenoid component with and in growth trabecular metal post, an over-coring drill helps to remove ingrowth component, at the expense of added glenoid bone stock.

Recommendation Respect and preserve bone stock, especially the glenoid, and consider using a glenoid component that does not have a sizable ingrowth glenoid post.

Since the deltoid is the main motor driver for the reverse construct arthroplasty, all efforts during surgical exposure and subsequent tensioning of the implant should focus on minimizing trauma to this muscle. This includes avoiding deltoid-splitting approaches and overtensioning to construct.

Recommendation Regardless of the previous approaches utilized in a failed case, approach the revision scenario with a deltopectoral approach, and take extra care to gain stability without overstuffing the joint.

References

1. Kessel L, Bayley I. Prosthetic replacement of shoulder joint: preliminary communication. J R Soc Med. 1979;72(10):748–52.
2. Grammont PM, Baulot E. Delta shoulder prosthesis for rotator cuff rupture. Orthopedics. 1993;16(1):65–8.
3. Zumstein MA, et al. Problems, complications, reoperations, and revisions in reverse total shoulder arthroplasty: a systematic review. J Shoulder Elbow Surg. 2011;20(1):146–57.
4. Wall BT, Mottier F, Walch G. Complications and revision of the reverse pros-thesis: a multicenter study of 457 cases. Reverse shoulder arthroplasty. Montpellier: Sauramps Médical; 2006.
5. Boileau P, Rumian AP, Zumstein MA. Reversed shoulder arthroplasty with modified L'Episcopo for combined loss of active elevation and external rotation. J Shoulder Elb Surg. 2010;19(2 Suppl):20–30.

6. Boileau P, et al. Revision surgery of reverse shoulder arthroplasty. J Shoulder Elb Surg. 2013;22(10):1359–70.
7. Teusink MJ, et al. Results of closed management of acute dislocation after reverse shoulder arthroplasty. J Shoulder Elb Surg. 2015;24(4):621–7.
8. Boileau P, et al. Neer Award 2005: The Grammont reverse shoulder prosthesis: results in cuff tear arthritis, fracture sequelae, and revision arthroplasty. J Shoulder Elb Surg. 2006;15(5):527–40.
9. Chacon A, et al. Revision arthroplasty with use of a reverse shoulder prosthesis-allograft composite. J Bone Joint Surg Am. 2009;91(1):119–27.
10. Boileau P. Complications and revision of reverse total shoulder arthroplasty. Orthop Traumatol Surg Res. 2016;102(1 Suppl):S33–43.
11. Morris BJ, et al. Risk factors for periprosthetic infection after reverse shoulder arthroplasty. J Shoulder Elb Surg. 2015;24(2):161–6.
12. Jacquot A, et al. Surgical management of the infected reversed shoulder arthroplasty: a French multicenter study of reoperation in 32 patients. J Shoulder Elb Surg. 2015;24(11):1713–22.
13. Braman JP, et al. The outcome of resection shoulder arthroplasty for recalcitrant shoulder infections. J Shoulder Elb Surg. 2006;15(5):549–53.
14. Zavala JA, et al. Management of deep infection after reverse total shoulder arthroplasty: a case series. J Shoulder Elbow Surg. 2011;21(10):1310–5.
15. Ghijselings S, Stuyck J, Debeer P. Surgical treatment algorithm for infected shoulder arthroplasty: a retrospective analysis of 17 cases. Acta Orthop Belg. 2013;79(6):626–35.
16. Beekman PD, et al. One-stage revision for patients with a chronically infected reverse total shoulder replacement. J Bone Joint Surg Br. 2010;92(6):817–22.
17. Verhelst L, et al. 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(8):1224–33.
18. Jawa A, et al. Prosthesis of antibiotic-loaded acrylic cement (PROSTALAC) use for the treatment of infection after shoulder arthroplasty. J Bone Joint Surg Am. 2011;93(21):2001–9.
19. Sahota S, Sperling JW, Cofield RH. Humeral windows and longitudinal splits for component removal in revision shoulder arthroplasty. J Shoulder Elb Surg. 2014;23(10):1485–91.
20. Gerber C, et al. Reverse Delta-III total shoulder replacement combined with latissimus dorsi transfer. A preliminary report. J Bone Joint Surg Am. 2007;89(5):940–7.
21. Favre P, et al. Latissimus dorsi transfer to restore external rotation with reverse shoulder arthroplasty: a biomechanical study. J Shoulder Elbow Surg. 2008;17(4):650–8.
22. Puskas GJ, et al. Secondary latissimus dorsi transfer after failed reverse total shoulder arthroplasty. J Shoulder Elb Surg. 2015;24(12):e337–44.
23. Elwell J, Choi J, Willing R. Quantifying the competing relationship between adduction range of motion and baseplate micromotion with lateralization of reverse total shoulder arthroplasty. J Biomech. 2017;52:24–30.
24. Gutierrez S, et al. Biomechanical comparison of component position and hardware failure in the reverse shoulder prosthesis. J Shoulder Elbow Surg. 2007;16(3 Suppl):S9–S12.
25. Garberina MJ, Williams GR Jr. Polyethylene dissociation after reverse total shoulder arthroplasty: the use of diagnostic arthroscopy. J Shoulder Elbow Surg. 2008;17(1):e16–8.
26. Cusick MC, et al. Glenosphere dissociation after reverse shoulder arthroplasty. J Shoulder Elb Surg. 2015;24(7):1061–8.
27. Otto RJ, et al. Scapular fractures after reverse shoulder arthroplasty: evaluation of risk factors and the reliability of a proposed classification. J Shoulder Elb Surg. 2013;22(11):1514–21.
28. Hattrup SJ. The influence of postoperative acromial and scapular spine fractures on the results of reverse shoulder arthroplasty. Orthopedics. 2010;33(5):302.
29. Crosby LA, Hamilton A, Twiss T. Scapula fractures after reverse total shoulder arthroplasty: classification and treatment. Clin Orthop Relat Res. 2011;469(9):2544–9.
30. Wahlquist TC, Hunt AF, Braman JP. Acromial base fractures after reverse total shoulder arthroplasty: report of five cases. J Shoulder Elbow Surg. 2011;20(7):1178–83.
31. Mollon B, et al. Impact of scapular notching on clinical outcomes after reverse total shoulder arthroplasty: an analysis of 476 shoulders. J Shoulder Elb Surg. 2017;26(7):1253–61.
32. Katz D, et al. Does lateralisation of the centre of rotation in reverse shoulder arthroplasty avoid scapular notching? Clinical and radiological review of one hundred and forty cases with forty five months of follow-up. Int Orthop. 2016;40(1):99–108.
33. Li X, et al. Inferior glenosphere placement reduces scapular notching in reverse total shoulder arthroplasty. Orthopedics. 2015;38(2):e88–93.

25 Failed Reverse Shoulder Arthroplasty: Case Example 1

Berte Bøe and Tom C. Ludvigsen

25.1 Introduction

The number of primary total shoulder arthroplasties (TSA) has increased exponentially in recent years, with a corresponding increase in the number of revision procedures. The infection rate after primary shoulder arthroplasties has a reported incidence of 0.4–2.9% [1, 2].

Reverse arthroplasties, young age, male gender and trauma-related arthroplasties all have greater risks of infection [3]. The infection rate also increases in incidence with every subsequent revision [4].

The treatment is challenging due to the increasing resistance of infectious organisms and the burden of the patients. The numbers of shoulder arthroplasties are few compared to hip and knee arthroplasties, and surgeons' experience remains limited. However, the management of periprosthetic joint infection (PJI) can to some extent be compared regardless of which joint is affected.

Unlike hip and knee infections, revision shoulder arthroplasties are often culture-positive for *Propionibacteria*. The functional outcomes of revising *Propionibacteria* culture-positive failed arthroplasties with a single-stage revision and immediate antibiotic therapy are not necessarily inferior to the clinical outcomes of revising failed shoulder arthroplasties that are not culture-positive [5].

For an early PJI the recommendations in general would be a soft tissue debridement with change of head/glenosphere and/or polyethylene components.

For a delayed PJI, more than 3 months after primary arthroplasty, the most common treatment is a two-staged revision of the arthroplasty. Time window between the two surgeries depends on microorganism and blood samples. Some surgeons prefer one-stage revision. Irrigation and debridement with component retention and chronic antibiotic suppression is another alternative for the management of acute or late hematogenous deep periprosthetic shoulder infection. Recently, Dennison et al. [6] reported 70% component retention after irrigation and debridement. Most patients were prescribed chronic antibiotic suppression therapy, and reasonable motion was maintained.

Regarding antibiotic treatment, it is important to have an antibiotic-free interval before revision surgery. This will increase the likelihood of having positive cultures. The treatment with empiric antibiotics should be initiated immediately after sampling during revision surgery. The samples have to be cultured for at least 14 days in shoulder revisions because of the slow-growing propionibacterium. Involvement of an infectologist is recommended for all these patients. Intravenous

B. Bøe (✉) · T. C. Ludvigsen
Division of Orthopaedic Surgery,
Oslo University Hospital, Oslo, Norway
e-mail: berte2@mac.com; tomcl@getmail.no

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_25

antibiotics should be administered for at least 14 days and oral antibiotics subsequent at least 6 weeks.

25.2 Case Presentation

Our case is a male born 1939. He had been permanently out of work since 1983 due to back pain when first admitted to an orthopaedic surgeon for pain in his right shoulder. He had been a manual factory worker. This first visit was in 2000 and he complained of reduced range of motion in the shoulder. X-rays showed osteoarthritis in the glenohumeral joint with flattening of the humeral head and subchondral sclerosis on the glenoid side. The patient was 61 years old and the surgeon considered arthrodesis or TSA.

In 2002, he was operated with an uncemented TSA in his right shoulder. In 2011, he experienced increasing pain and was referred to a shoulder specialist. At this point, he had pain at rest and could only use the arm close to the body.

X-rays showed lucency around the glenoid peg and a strange contour on the metal backing. There was a broken screw in the glenoid and extensive wear of the polyethylene (PE) components. The head of the prosthesis was cranially migrated indicating that supraspinatus was not efficient (Fig. 25.1). At the clinical exam, he showed subscapularis weakness. Infraspinatus and teres minor were acceptable, and he could contract all three parts of the deltoid muscle.

The only possible solution was revision to a reverse shoulder arthroplasty (RSA). The patient was informed about possible complications in form of nerve injury and infection. Both the patient and the surgeon needed some time to decide for operation or not. After 3 more years, in 2014, the decision was made to operate.

In preoperative planning, we had to consider bone grafting and risk of fractures. We normally use autograft from iliac crest or frozen allografts from retained femoral heads. In revision cases, poor quality of glenoid bone may occur, and the surgeon should prepare for bone grafting to achieve good fixation of the metaglene component. To prevent fracture lines in the humerus, it is sometimes advisable to protect with a cerclage before chiselling along the stem. It is also advisable to make a controlled osteotomy rather than risk an uncontrolled fracture.

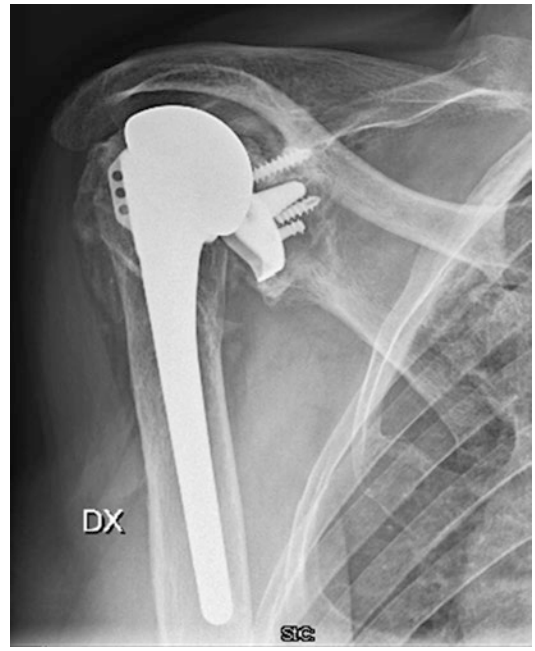

Fig. 25.1 X-rays of failed TSA show anterior-superior dislocation of the humerus. There was a broken screw in the metal-backed glenoid component

As in revision cases, the index operation was often performed long ago, and in another hospital, it is very important to acquire exact data on components implanted and to contact the implant provider to have the right equipment for component removal.

For revision surgery, we recommend deltopectoral approach. This approach can be extended in both directions.

In our patient, we found extensive metallosis. The tissue was sticky and grey/black. We tried to remove it as much as possible. The head of the screw was worn and broke when we tried to remove it. In spite of bone loss in the centre of the glenoid, the outer ring was intact, and we used it as a platform for the glenoid baseplate.

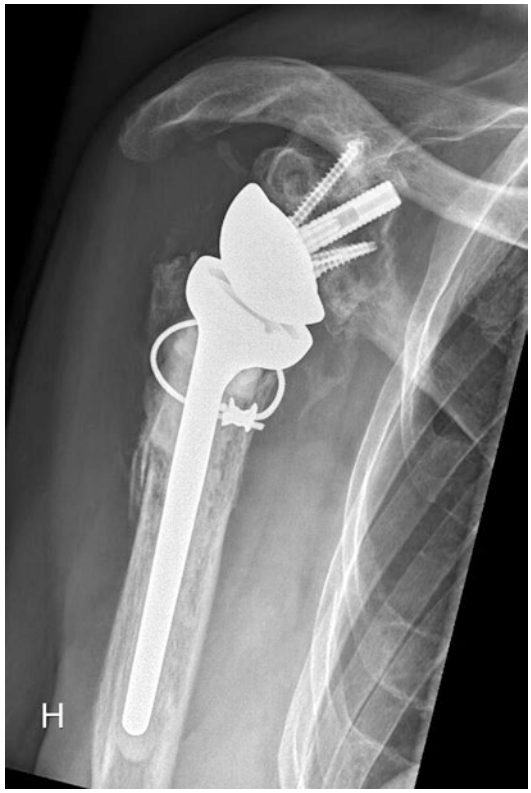

Fig. 25.2 Revision RSA was performed after removal of the primary implant. Glenoid baseplate was fixed with long central peg and screws. Proximal humerus was secured with a cerclage wire to prevent fracture

We put a femoral head allograft inside the intact ring of native glenoid. The glenoid baseplate of revision RSA is usually fixed with screws and a long central peg.

When removing a cemented humeral stem, there is always a high risk of fracturing the humerus. Gradually, it is possible to remove it by chiselling along the stem. The cement mantle can be left inside the bone in cases with no suspicion of infection. This reduces the risk of fractures. We secured the proximal humerus with a cerclage wire in fear of a threatening fracture (Fig. 25.2).

Six weeks after the revision, the patient came to his first follow-up visit. He felt tired and from the wound there had been some secretion of yellow fluid. His general practitioner had given him penicillin tablets. He did not have fever and the blood samples were nearly normal. We told him to quit antibiotics and 2 weeks later performed a soft tissue revision with change of polyethylene liner. There was a 15 mm fistula in the wound all the way in to the implant. Again, there was a lot of metallosis. No purulent secretion could be seen. Standard antibiotics after soft tissue revisions of arthroplasties in our department are intravenous (iv) Ekvacillin (cloxacillin) and vancomycin. Treatment with vancomycin requires careful monitoring to avoid kidney failure.

One week after revision, there was growth of *Propionibacterium avidum* in all seven samples, including bone biopsy. The bacterium was sensitive to penicillin, and the treatment was changed to iv penicillin for 2 weeks and thereafter ciprofloxacin tablets for 3 months.

The patient gradually felt better during the first months after the revision. He had been without antibiotics for 5 months when he showed up with an abscess in the wound. The abscess was drained and he was treated with iv penicillin for 2 weeks. The samples were once again positive for propionibacterium. At this time, the patient was not motivated for any more surgery, and we decided to try lifelong suppression treatment. Ciprofloxacin is not a drug of choice for lifelong treatment because of resistance. Our patient was treated with apocillin (phenoxymethylpenicillin).

Six months later the patient was suffering from fatigue and had red to violet discolouration around the wound. Blood samples were normal. Antibiotics were discontinued for 2 weeks and the patient was revised with insertion of a spacer. The stem was completely loose. There was still extensive grey-black discolouration of subcutaneous tissues as seen with metallosis. We tried to remove all cement from the humerus. The metaglene was completely fixed and had to be chiselled off the glenoid after removing the screws. The bone graft had healed and could be used for implanting a new glenoid component later. To have the option of later rearticulating, we implanted a custom-made spacer (Fig. 25.3).

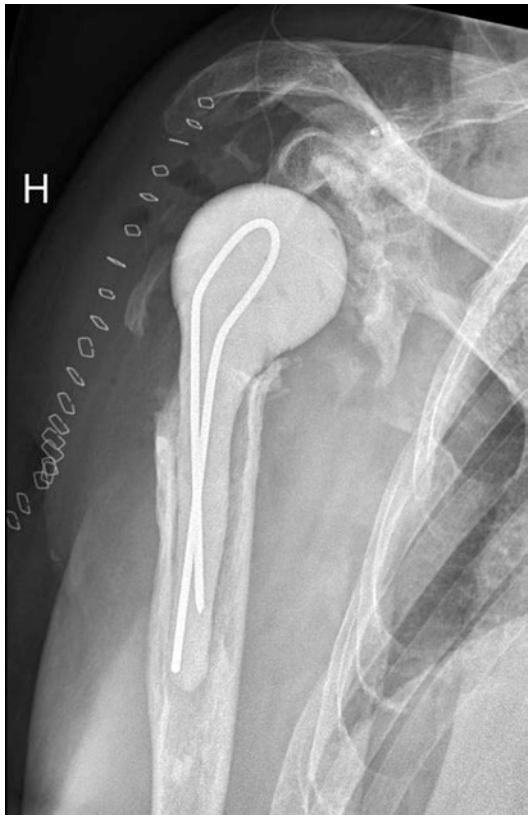

Fig. 25.3 A custom-made cement spacer was implanted after RSA removal

The patient was treated with penicillin and vancomycin in accordance with culture results. Six months later he felt good. Then, no antibiotics were administered for subsequent 3 months. As the patient was not motivated for further surgery, he was followed-up with repeated X-rays every 6 months. At the last follow-up visit, no colour changes were observed in the skin around the wound. He complained some residual pain. The spacer apparently allows limited function and range of motion consisted of approximately 30° of flexion, abduction and extension. At the X-rays there was no visible bone erosion, albeit erosion of the glenoid due to wear from the spacer head could be expected.

References

1. Trappey GJ, et al. What are the instability and infection rates after reverse shoulder arthroplasty? Clin Orthop Relat Res. 2011;469(9):2505–11.
2. Singh JA, et al. Periprosthetic infections after total shoulder arthroplasty: a 33-year perspective. J Shoulder Elb Surg. 2012;21(11):1534–41.
3. Richards J, et al. Patient and procedure-specific risk factors for deep infection after primary shoulder arthroplasty. Clin Orthop Relat Res. 2014;472(9):2809–15.
4. Buchalter DB, et al. Two-stage revision for infected shoulder arthroplasty. J Shoulder Elb Surg. 2016;26(6):939–47.
5. Hsu JE, et al. Single-stage revision is effective for failed shoulder arthroplasty with positive cultures for propionibacterium. J Bone Joint Surg Am. 2016;98(24):2047–51.
6. Dennison T, et al. Management of acute or late hematogenous infection after shoulder arthroplasty with irrigation, debridement, and component retention. J Shoulder Elb Surg. 2017;26(1):73–8.

Failed Reverse Shoulder Arthroplasty: Case Example 2

26

Maristella F. Saccomanno, Cristina Rossi, Carmine Latte, and Andrea Grasso

26.1 Introduction

Total shoulder arthroplasty (TSA) is an increasingly common procedure in the treatment of primary and secondary degenerative conditions of the shoulder [1]. This procedure has proven to be successful in decreasing pain, improving range of motion, and restoring function [2, 3]. Overall, the reported rate of complications after TSA is highly variable in literature, ranging from 12 to 39.8% [4–6]. Early revision surgery is mainly due to dislocation/instability of the implant most of the time linked to new trauma or surgical errors, whereas, in the long term, major causes of revision surgery are rotator cuff insufficiency and loosening of glenoid component due to osteolysis [7]. The osteolytic phenomenon seems to be linked to a biologic response to polyethylene wear debris [8]. Wear particles are released by the articulating surfaces. These particles may cause a cytokine-driven inflammatory response that depends on the material, size, dose, and morphology of the wear particles [9]. A recent study [10] on 165 patients, who underwent an anatomic TSA with uncemented metal-backed glenoid components, showed that the rate of the implant survival was 60% (100% CI, 44–71%) and 46% (100% CI, 32–54%) at 10 and 12 years, respectively, with a severe drop of the curve after the fourth year. Eighty percent of patients who underwent revision had evidence of polyethylene wear. Therefore, the study confirms that polyethylene wear is a long-term complication.

This is a case report of a patient who had an early polyethylene insert wear.

26.2 Case Presentation

A 68-year-old healthy female was referred to the senior author (AG) for a shoulder problem. The patient complained a progressive pain in her right shoulder started 5 years ago with no traumatic onset and worsened in the last 6 months so that daily activities were very limited. Pain was mainly exacerbated by forward flexion and internal rotation. At the examination, passive range of motion was complete, but painful over 90° of forward flexion and in maximal external rotation. Active range of motion showed forward flexion up to 110°, external rotation up to 50°, and internal rotation to the hip. Tests evaluating cuff integrity were negative.

X-rays and magnetic resonance imaging (MRI) were then performed. The patient underwent standard radiograph series, including anteroposterior, axillary, and Lamy view

M. F. Saccomanno (✉)
Department of Orthopaedics, Catholic University, Rome, Italy

"A. Gemelli" University Hospital, Rome, Italy

C. Rossi · C. Latte
Villa Valeria Clinic, Rome, Italy

A. Grasso
Department of Orthopaedics,
Casa di Cura Villa Valeria, Rome, Italy

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_26

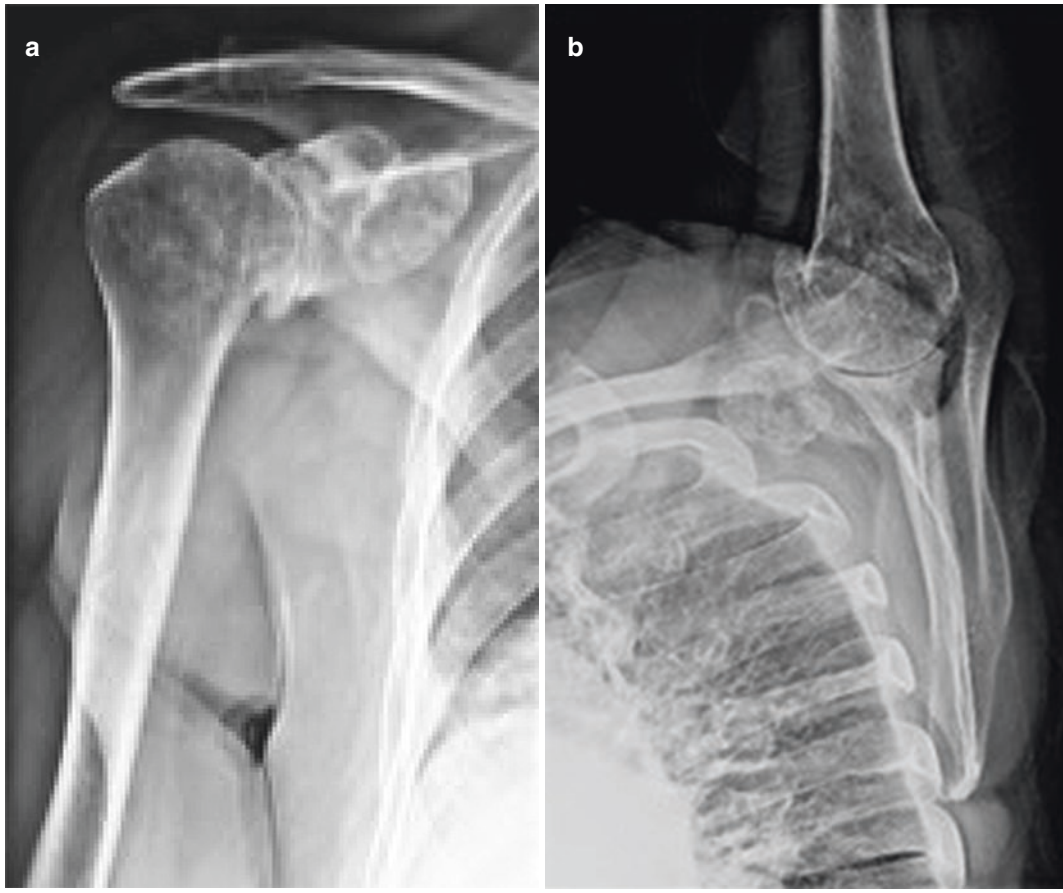

Fig. 26.1 Right shoulder. Preoperative X-rays: anteroposterior view (**a**) and axillary view (**b**)

(Fig. 26.1). Imaging showed primary concentric osteoarthritis (OA) with cuff integrity.

Therefore, the patient underwent an anatomic TSA (SMR, Lima LTO, Udine, Italy) on November 2010. The operative procedure was uncomplicated. The patient was immobilized in a sling for 4 weeks postoperatively. Ten days after the procedure, a standard rehabilitation protocol was started. At the clinical follow-up 6 months after surgery, minimal pain and no limitations in daily activities were reported. Active range of motion significantly improved forward flexion up to 170°, external rotation up to 60°, and internal rotation to the lower back. X-rays did not show relevant issues (Fig. 26.2).

Two years postoperatively, the patient came back to the clinic complaining of constant pain in her right shoulder, started 1 month ago without any sort of trauma, associated with slowly increasing swelling and severely restricted range of motion.

Physical examination revealed tenderness and swelling over the anterior region of the right shoulder. Passive range of motion was severely limited by pain. Active range of motion showed forward flexion up to 60°, external rotation up to 15°, and internal rotation to the hip.

X-rays showed upper migration of the humeral head in the AP view and a possible glenoid malposition in the axillary view (Fig. 26.3).

A joint aspiration was also performed in order to find out a possible explanation for the intense swelling and to simultaneously improve the pain. Forty milliliters of blood were obtained. Samples were sent to microbiology laboratory for routine cultures. No signs of infection were found.

Before performing a revision surgery, a diagnostic arthroscopy was performed. The arthros-

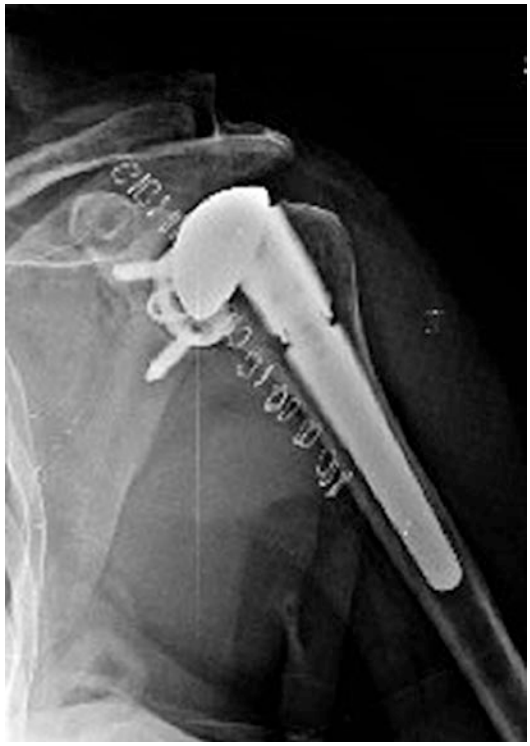

Fig. 26.2 Right shoulder. Postoperative X-rays after anatomic TSA

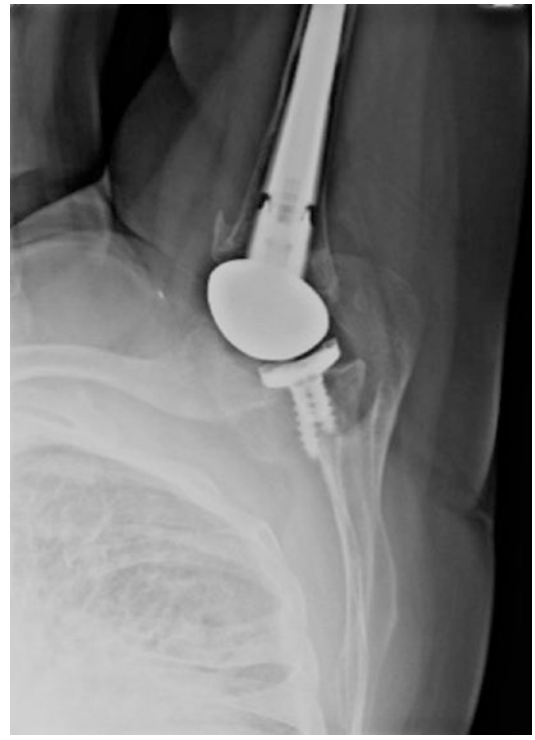

Fig. 26.3 Right shoulder. X-rays 2 years after surgery. The axillary view shows incomplete glenoid component seating and altered version

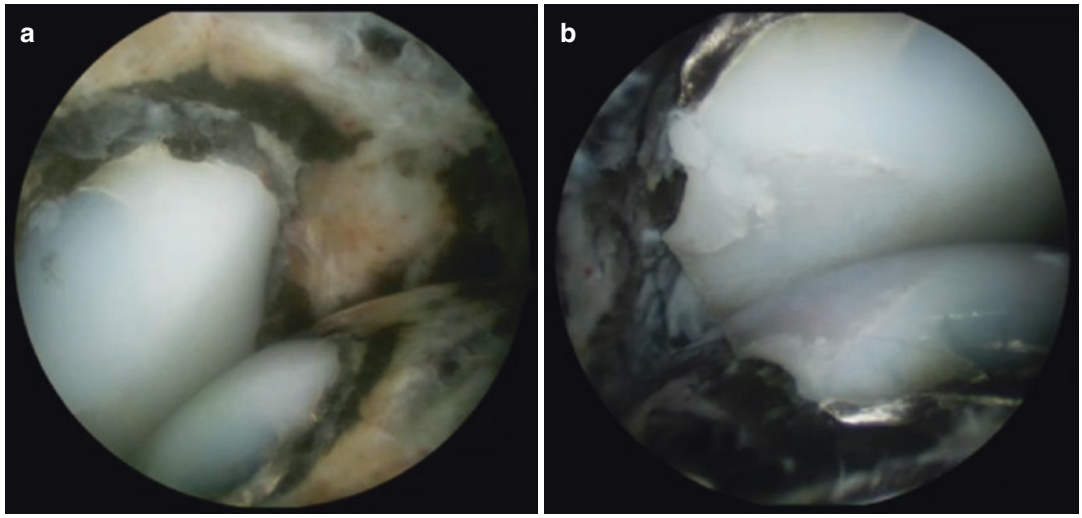

Fig. 26.4 Right shoulder. Arthroscopic view from posterior portal showed metallosis (**a**) and polyethylene wear (**b**)

copy showed superior polyethylene wear and diffuse signs of metallosis of the soft tissue around the glenoid component (Fig. 26.4). Based on these findings, it was decided to convert the implant to a reverse shoulder configuration. Thanks to the modularity of the implant chosen, the revision surgery consisted of polyethylene insert removal along with the humeral head and

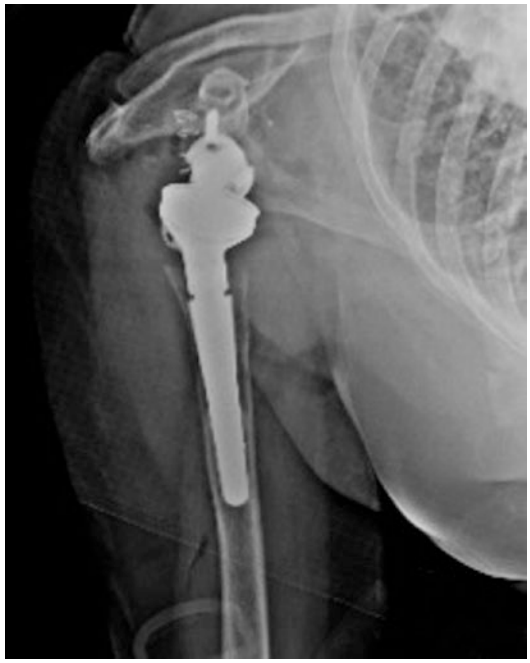

Fig. 26.5 Right shoulder. Postoperative X-ray after first revision with RSA

subsequent implantation of the reverse humeral body and the glenosphere. After reduction, the joint showed good stability and full passive range of motion. Postoperative X-rays showed good implant positioning (Fig. 26.5). Following a standard postoperative protocol, the arm was immobilized in a sling for 4 weeks, and physical therapy started 15 days after surgery. Unfortunately, after sling removal, the patient complained of acute shoulder pain and severely restricted range of motion. X-rays showed an anterosuperior dislocation of the humeral component (Fig. 26.6). A closed reduction under anesthesia in the operative room was attempted with no success. Therefore, the patient underwent a second revision procedure. Intraoperative findings showed an irreparable subscapularis tendon. A thicker inlay trial was initially tested with no success. Osteolysis around the metal glenoid baseplate was detected, and therefore the glenoid component was removed. The glenoid bone stock underneath was very poor. The implant was then converted to a hemiarthroplasty with a large humeral head (Fig. 26.7).

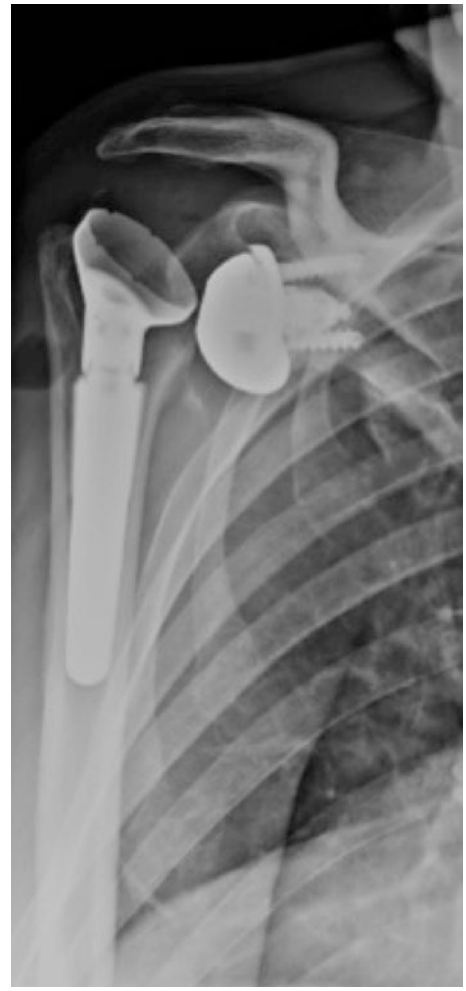

Fig. 26.6 Right shoulder. AP view shows RSA dislocation

A standard postoperative rehabilitation protocol was followed.

Five years after the revision procedure, the patient had no further complication and returned to normal daily activities.

26.3 Discussion

Polyethylene wear has been shown to be an important long-term complication after anatomic TSA [11–13]. It generates plastic wear debris that has several consequences: biologically, wear debris is responsible for osteolysis and severe

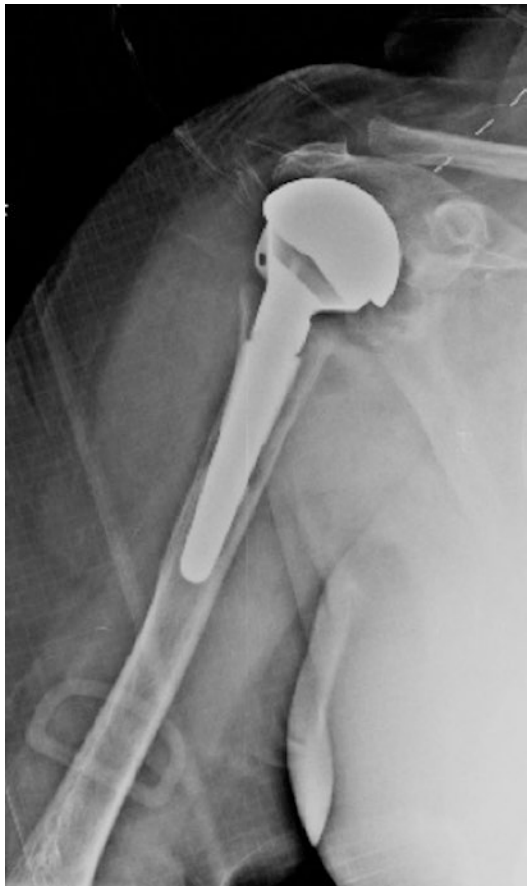

Fig. 26.7 Right shoulder. Postoperative X-ray after conversion to hemiarthroplasty

glenoid bone resorption; mechanically, bone resorption leads to glenoid component shift and loosening; and clinically, mobility of the glenoid component and synovitis are responsible for pain and progressive loss of motion.

This clinical report showed a singular case of accelerated polyethylene wear probably due to the glenoid component malposition. Glenoid component malposition caused increased eccentric loads which subsequently leads to higher stresses to the polyethylene [14].

Several studies have shown that glenoid component positioning is a critical factor for TSA survival [15, 16]. A cadaveric study suggested that glenoid version directly affects contact areas, contact pressures, and reaction forces of the prosthetic shoulder, thus altering TSA biomechanics [17]. The authors showed that retroverted glenoid components were associated with smaller contact areas and higher contact pressures and concluded that retroversion leads to eccentric loading on the posterior glenoid, increased stress at the bone-implant interface, and ultimately affect polyethylene wear and component stability. Therefore, it is clear that proper placement (inferior-superior position) and appropriate version of the glenoid component are crucial to ensure long-term function of the implant [18].

Although someone can argue that a computer tomography (CT) scan is mandatory for the preoperative assessment of glenoid wear and erosion in order to avoid glenoid component malposition, it must also be highlighted that intraoperative correction of glenoid version is anyways a challenging task in arthritic shoulders, and it is essentially based on surgeon experience. Several reasons make the intraoperative glenoid component placement difficult. First, the lack of visible scapular landmarks makes hard to identify the position of the scapular spine. Second, anatomic placement and secure implant fixation of the glenoid component may be limited by the size of the native glenoid and the availability of sufficient bone stock. Third, the orientation of the exposed glenoid articular surface can be misleading in predicting accurate positioning of the glenoid component with respect to the scapula, since posterior wear and erosion of the glenoid are a common finding in patients with glenohumeral OA.

A second complication occurred in the present case. The early dislocation after the sling removal can be a consequence of previous underestimate glenoid malpositioning and osteolysis around glenoid baseplate, even if clear signs of glenoid component loosening were not found at the time of the second surgery.

Differently from hip and knee, in shoulder surgery the correlation between component malposition and accelerated polyethylene wear has not been fully investigated yet. Therefore, this clinical case is important because this highlights the possible cascade of events which can follow glenoid malposition leading to an accelerated polyethylene wear and its related consequences.

References

1. Kim SH, Wise BL, Zhang Y, Szabo RM. Increasing incidence of shoulder arthroplasty in the United States. J Bone Joint Surg Am. 2011;93:2249–54. https://doi.org/10.2106/JBJS.J.01994.
2. Deshmukh AV, Koris M, Zurakowski D, Thornhill TS. Total shoulder arthroplasty: long-term survivorship, functional outcome, and quality of life. J Shoulder Elbow Surg. 2005;14:471–9. https://doi.org/10.1016/j.jse.2005.02.009.
3. Lo IKY, Litchfield RB, Griffin S, et al. Quality-of-life outcome following hemiarthroplasty or total shoulder arthroplasty in patients with osteoarthritis. A prospective, randomized trial. J Bone Joint Surg Am. 2005;87:2178–85. https://doi.org/10.2106/JBJS.D.02198.
4. Bohsali KI, Bois AJ, Wirth MA. Complications of shoulder arthroplasty. J Bone Joint Surg Am. 2017;99:256–69. https://doi.org/10.2106/JBJS.16.00935.
5. Chin PYK, Sperling JW, Cofield RH, Schleck C. Complications of total shoulder arthroplasty: are they fewer or different? J Shoulder Elbow Surg. 2006;15:19–22. https://doi.org/10.1016/j.jse.2005.05.005.
6. Gonzalez J-F, Alami GB, Baque F, et al. Complications of unconstrained shoulder prostheses. J Shoulder Elbow Surg. 2011;20:666–82. https://doi.org/10.1016/j.jse.2010.11.017.
7. Bansal A, Khatib ON, Zuckerman JD. Revision total joint arthroplasty: the epidemiology of 63,140 cases in New York State. J Arthroplasty. 2014;29:23–7. https://doi.org/10.1016/j.arth.2013.04.006.
8. Purdue PE, Koulouvaris P, Potter HG, et al. The cellular and molecular biology of periprosthetic osteolysis. Clin Orthop. 2007;454:251–61. https://doi.org/10.1097/01.blo.0000238813.95035.1b.
9. Hallab NJ, McAllister K, Brady M, Jarman-Smith M. Macrophage reactivity to different polymers demonstrates particle size- and material-specific reactivity: PEEK-OPTIMA(®) particles versus UHMWPE particles in the submicron, micron, and 10 micron size ranges. J Biomed Mater Res B Appl Biomater. 2012;100:480–92. https://doi.org/10.1002/jbm.b.31974.
10. Boileau P, Moineau G, Morin-Salvo N, et al. Metal-backed glenoid implant with polyethylene insert is not a viable long-term therapeutic option. J Shoulder Elbow Surg. 2015;24:1534–43. https://doi.org/10.1016/j.jse.2015.02.012.
11. Cheung EV, Sperling JW, Cofield RH. Polyethylene insert exchange for wear after total shoulder arthroplasty. J Shoulder Elbow Surg. 2007;16:574–8. https://doi.org/10.1016/j.jse.2006.12.009.
12. Gunther SB, Graham J, Norris TR, et al. Retrieved glenoid components: a classification system for surface damage analysis. J Arthroplasty. 2002;17:95–100.
13. Matsen FA, Clinton J, Lynch J, et al. Glenoid component failure in total shoulder arthroplasty. J Bone Joint Surg Am. 2008;90:885–96. https://doi.org/10.2106/JBJS.G.01263.
14. Carpenter SR, Urits I, Murthi AM. Porous metals and alternate bearing surfaces in shoulder arthroplasty. Curr Rev Musculoskelet Med. 2016;9:59–66. https://doi.org/10.1007/s12178-016-9319-x.
15. Nyffeler RW, Sheikh R, Atkinson TS, et al. Effects of glenoid component version on humeral head displacement and joint reaction forces: an experimental study. J Shoulder Elbow Surg. 2006;15:625–9. https://doi.org/10.1016/j.jse.2005.09.016.
16. Spencer EE, Valdevit A, Kambic H, et al. The effect of humeral component anteversion on shoulder stability with glenoid component retroversion. J Bone Joint Surg Am. 2005;87:808–14. https://doi.org/10.2106/JBJS.C.00770.
17. Shapiro TA, McGarry MH, Gupta R, et al. Biomechanical effects of glenoid retroversion in total shoulder arthroplasty. J Shoulder Elbow Surg. 2007;16:S90–5. https://doi.org/10.1016/j.jse.2006.07.010.
18. Iannotti JP, Spencer EE, Winter U, et al. Prosthetic positioning in total shoulder arthroplasty. J Shoulder Elbow Surg. 2005;14:111S–21S. https://doi.org/10.1016/j.jse.2004.09.026.

Part V

Failed Rotator Cuff Repair

Rotator Cuff Repair: Why It Fails

27

Olaf Lorbach

27.1 Introduction

Rotator cuff repair is a common cause of shoulder dysfunction and pain [1–4] with a reported incidence of 25% in the population of 50 years and an overall prevalence of 5–40% [5]. Mainly affecting the elderly patients [6], it is associated with high direct as well as indirect costs [7].

Although open as well as arthroscopic rotator cuff repair led to good clinical results [8–12], a failure rate is reported especially in chronic, massive tears [9, 10, 13, 14].

Reasons for failure of the construct are multifactorial and can be divided into two main groups: biomechanical and biological factors.

27.2 Biomechanical Factors

The ideal biomechanical rotator cuff repair should provide low gap formation under cyclic loading a well as a high ultimate failure strength [15]. These biomechanical properties are potentially influenced by the different factors like the anchor material and design, the suture material, the suture configuration, as well as the surgical technique (double-row vs. single-row repair).

Double-row repair was initially described with superior biomechanical results compared to single-row repairs [16–20]. However, these double-row constructs were compared to simple suture repairs. As the suture tendon interface seems to be the weakest part of the reconstruction, it seems reasonable to compare double-row constructs to single-row repairs using modified suture configurations.

When comparing single-row repairs using modified suture configurations to double-row repairs, no significant differences in ultimate load-to-failure [21–31] or cyclic displacement [22–27, 30, 31] were reported in the majority of studies.

The transosseous equivalent rotator cuff repair (TOE) was introduced as the second generation of double-row repairs and was described with several potential benefits as decreased knot impingement, improved footprint coverage, as well as superior biomechanical properties compared to single-row and simple double-row repairs. However, TOE rotator cuff repairs are also associated with several concerns about tendon strangulation and necrosis [32], the potential risk of over-tensioning the construct medially [33], and a reduced intra-tendinous blood flow [34]. Moreover, the technique seems to be highly associated with a different re-tear pattern where the construct fails medial to the footprint at the musculotendinous junction [32, 35–38].

O. Lorbach
Sporthopaedicum Berlin, Berlin, Germany
e-mail: olaf.lorbach@uks.eu

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_27

Furthermore, it may not be the best choice for every rupture as a decreased remnant tendon length <10 mm was significantly associated with a higher re-rupture rate for TOE double-row repairs compared to single-row repairs (46% vs. 6%) [39].

Despite any potential benefit in biomechanical studies, no significant differences were further seen in the majority of clinical studies neither in the clinical scores [6, 35, 40–49] nor in the reported re-rupture rate evaluated by CT-arthrogram 42 or MRI [6, 41, 43, 45]. 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 [12, 42, 43].

In summary, the evidence of a biomechanical as well as a clinical superiority for any type of double-row repairs compared to single-row repairs is, based on the current literature, at least questionable. The weakest point of the repair is the suture tendon interface. Therefore, the biomechanical performance is dependent on the number of sutures, which penetrate the tendon as well as the suture configuration and not on the number of anchors. 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 [50].

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

In spite of the dramatic increase in publications per year as well as the tremendous amount of new implants and technique modifications, there is little evidence that the results of rotator cuff repairs are improving with a mean re-tear rate of 27% at a 2-year follow-up [51].

Therefore, biology seems to be the most important factor affecting the healing rate of the construct.

27.3 Biological Factors

Several biological factors have been described to have a significant impact on healing of the rotator cuff. Factors which seem to impair healing are age [52–55], tendon quality (retraction, fatty atrophy) [53, 54], the number of tendons involved [54, 55], tear size [53, 54], the body mass index [53], as well as an associated biceps/AC joint pathology [54].

Fermont et al. [56] identified 12 prognostic factors which can be divided into different categories: demographic factors (age, gender), clinical factors (BMD, diabetes mellitus, level of sports activity, preoperative ROM, obesity), factors related to the integrity of the RC (size of the lesion, retraction, fatty infiltration, multiple tendon involvement, preop tendon length), factors related to the surgical procedure (concomitant biceps pathology or AC joint procedure), and postoperative factors (compliance, rehabilitation).

Nho et al. [54] investigated the influencing factors for a re-tear of the repaired rotator cuff in 129 patients who completed follow-up (67%). Several factors were identified to have a significant impact on healing.

The size of the tear and the number of tendons involved increase the relative risk for a tendon defect after arthroscopic rotator cuff repair 2.29 times for each centimeter of increase in tear size. Moreover, it was increased 8.92 times from a single to a multiple tendon tear. Age was also an influencing factor with increasing the risk of a re-tear approximately 1.08 times for every additional year. Concomitant biceps and/or AC joint pathology was reported with an 11 times higher risk of a tendon defect (biceps), respectively, four times higher risk of a tendon defect (AC joint procedure).

Finally, tendon quality also significantly influenced rotator cuff healing with a three times higher risk for a failure comparing bad to good tendon quality.

Structural failure, however, is not consistently associated with the clinical outcome [52, 57–60]. Namdari et al. [61] divided 70 patients with a structural failure of the rotator cuff repair assessed by ultrasound into two groups. Group one consisted of patients with a successful clinical outcome, whereas group two consisted of patients with an unsuccessful clinical outcome. Factors which were related to bad clinical results were

labor-intensive occupation, any claim (workers compensation claim, litigation claim, disability claim), low preoperative total ASES and SST scores, as well as a limited preoperative active external rotation. However, they could also identify several factors that did not have a negative influence on the results such as the dominant side, age, sex, medical comorbidities, smoking status, as well as previous surgery.

Conclusion

In summary, the most important factors which were associated with a structural failure of the repair were increasing age, the tear size and the number of tendons involved, the tendon quality (retraction, fatty infiltration, atrophy), as well as associated biceps or AC joint pathology.

Therefore, the best patient for a successful repair seems to be a young patient with a single tendon tear, good tendon quality, and no associated biceps or AC joint pathology.

References

1. Maffulli N, Longo UG, Spiezia F, Denaro V. Sports injuries in young athletes: long-term outcome and prevention strategies. Phys Sportsmed. 2010;38(2):29–34.
2. Maffulli N, Longo UG, Gougoulias N, Loppini M, Denaro V. Long-term health outcomes of youth sports injuries. Br J Sports Med. 2010;44(1):21–5.
3. Maffulli N, Longo UG, Gougoulias N, Caine D, Denaro V. Sport injuries: a review of outcomes. Br Med Bull. 2011;97:47–80.
4. Maffulli N, Longo UG, Spiezia F, Denaro V. Aetiology and prevention of injuries in elite young athletes. Med Sport Sci. 2011;56:187–200.
5. DePalma A, Callery G, Bennett G. Variational anatomy in degenerative lesions of the shoulder joint. Instr Cours Lect. 1949;6:255–81.
6. 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(8):1254–60.
7. Meislin RJ, Sperling JW, Stitik TP. Persistent shoulder pain: epidemiology, pathophysiology, and diagnosis. Am J Orthop (Belle Mead NJ). 2005;34(12 Suppl):5–9.
8. 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.
9. Gazielly DF, Gleyze P, Montagnon C. Functional and anatomical results after rotator cuff repair. Clin Orthop Relat Res. 1994;304:43–53.
10. 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.
11. Ide J, Maeda S, Takagi K. A comparison of arthroscopic and open rotator cuff repair. Arthroscopy. 2005;21(9):1090–8.
12. 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.
13. Bishop J, Klepps S, Lo IK, Bird J, Gladstone JN, Flatow EL. Cuff integrity after arthroscopic versus open rotator cuff repair: a prospective study. J Shoulder Elb Surg. 2006;15(3):290–9.
14. Postel JM, Goutallier D, Lavau L, Bernageau J. Anatomical results of rotator cuff repairs: study of 57 cases controlled by arthrography. J Shoulder Elb Surg. 1994;3:20.
15. Gerber C, Schneeberger AG, Beck M, Schlegel U. Mechanical strength of repairs of the rotator cuff. J Bone Joint Surg Br. 1994;76(3):371–80.
16. Baums MH, Spahn G, Steckel H, Fischer A, Schultz W, Klinger HM. 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(12):1466–72.
17. 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(3):407–14.
18. Meier SW, Meier JD. The effect of double-row fixation on initial repair strength in rotator cuff repair: a biomechanical study. Arthroscopy. 2006;22(11):1168–73.
19. Park MC, Idjadi JA, Elattrache NS, Tibone JE, McGarry MH, Lee TQ. The effect of dynamic external rotation comparing 2 footprint-restoring rotator cuff repair techniques. Am J Sports Med. 2008;36(5):893–900.
20. Waltrip RL, Zheng N, Dugas JR, Andrews JR. Rotator cuff repair. A biomechanical comparison of three techniques. Am J Sports Med. 2003;31(4):493–7.
21. Cummins CA, Appleyard RC, Strickland S, Haen PS, Chen S, Murrell GA. Rotator cuff repair: an ex vivo analysis of suture anchor repair techniques on initial load to failure. Arthroscopy. 2005;21(10):1236–41.
22. 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(8):1504–10.

23. 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(9 Suppl):S95–105.
24. 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.
25. Lorbach O, Pape D, Raber F, Busch LC, Kohn D, Kieb M. 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. 2012;20(11):2139–47.
26. 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.
27. 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.
28. 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(12):1265–70.
29. 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.
30. 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(8):941–8.
31. 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(11):2425–31.
32. 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.
33. Park MC, McGarry MH, Gunzenhauser RC, Benefiel MK, Park CJ, Lee TQ. Does transosseous-equivalent rotator cuff repair biomechanically provide a "self-reinforcement" effect compared with single-row repair? J Shoulder Elb Surg. 2014;23(12):1813–21.
34. Christoforetti JJ, Krupp RJ, Singleton SB, Kissenberth MJ, Cook C, Hawkins RJ. Arthroscopic suture bridge transosseous equivalent fixation of rotator cuff tendon preserves intratendinous blood flow at the time of initial fixation. J Shoulder Elb Surg. 2012;21(4):523–30.
35. 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(12):2777–85.
36. 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(5):983–91.
37. Trantalis JN, Boorman RS, Pletsch K, Lo IK. Medial rotator cuff failure after arthroscopic double-row rotator cuff repair. Arthroscopy. 2008;24(6):727–31.
38. 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(11):2440–7.
39. 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(10):2270–7.
40. Aydin N, Kocaoglu B, Guven O. Single-row versus double-row arthroscopic rotator cuff repair in small- to medium-sized tears. J Shoulder Elb Surg. 2010;19(5):722–5.
41. 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.
42. 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(8):1247–53.
43. Dehaan AM, Axelrad TW, Kaye E, Silvestri L, Puskas B, Foster TE. 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(5):1176–85.
44. Grasso A, Milano G, Salvatore M, Falcone G, Deriu L, Fabbriciani C. Single-row versus double-row arthroscopic rotator cuff repair: a prospective randomized clinical study. Arthroscopy. 2009;25(1):4–12.
45. 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(4):453–62.
46. Reardon DJ, Maffulli N. Clinical evidence shows no difference between single- and double-row repair for rotator cuff tears. Arthroscopy. 2007;23(6):670–3.
47. 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(3):732–42.
48. Wall LB, Keener JD, Brophy RH. Clinical outcomes of double-row versus single-row rotator cuff repairs. Arthroscopy. 2009;25(11):1312–8.
49. 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(2):343–8.
50. Wieser K, Rahm S, Farshad M, Ek ET, Gerber C, Meyer DC. Stitch positioning influences the suture hold in supraspinatus tendon repair. Knee Surg Sports Traumatol Arthrosc. 2013;21(7):1587–92.
51. 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(2):491–500.
52. 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.
53. 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(10):2099–107.
54. Nho SJ, Brown BS, Lyman S, Adler RS, Altchek DW, MacGillivray JD. Prospective analysis of arthroscopic rotator cuff repair: prognostic factors affecting clinical and ultrasound outcome. J Shoulder Elb Surg. 2009;18(1):13–20.
55. Tashjian RZ, Hollins AM, Kim HM, et al. Factors affecting healing rates after arthroscopic double-row rotator cuff repair. Am J Sports Med. 2010;38(12):2435–42.
56. Fermont AJ, Wolterbeek N, Wessel RN, Baeyens JP, de Bie RA. Prognostic factors for successful recovery after arthroscopic rotator cuff repair: a systematic literature review. J Orthop Sports Phys Ther. 2014;44(3):153–63.
57. 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(12):1899–905.
58. 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(7):1533–41.
59. Rimmke N, Maerz T, Cooper R, Yadavalli S, Anderson K. Arthroscopic suture bridge rotator cuff repair: functional outcome, repair integrity, and preoperative factors related to postoperative outcome. Phys Sportsmed. 2016;44(2):126–32.
60. 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.
61. 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.

How to Manage Failed Rotator Cuff Repair: Arthroscopic Revision Surgery

28

Alexander Otto, Knut Beitzel, and Andreas B. Imhoff

28.1 Introduction

With the development of new operative techniques and instrumentation, even massive rotator cuff tears can be addressed arthroscopically [1]. This paradigm change from open to arthroscopic surgery begun in the last decade and is based on conclusive biomechanical principles coupled with the development of reliable specific arthroscopic instrumentation [1]. Despite the technical evolution mentioned above, unsatisfactory postoperative outcome that leads to revision surgery still occurs in 6–8% of the cases [2].

There are multifactorial reasons for disappointing outcome in rotator cuff repair. In brief, main components of failure are diagnostic inaccuracies, technical errors, surgical complications, failed tendon healing, and traumatic postoperative events [3]. In our department, we performed rotator cuff revision surgery in 60 cases between 2006 and 2014. According to our unpublished data, the indication for revision surgery was tendon re-rupture in 58.93%, infection in 17.86%, shoulder stiffness in 8.93%, new tendon rupture in 3.57%, material failure in 3.57%, and tendinitis of the long head of the biceps (LHB) in 3.57% of the cases.

Especially in revision cases, all skills of the clinician are demanded to accomplish proper treatment with pain relief and improved functionality, since the situation is more complex than in a primary setting. Revision rotator cuff repair is strongly dependent on the (poor) tissue quality, retained hardware, patient factors, as well as his/her expectations and the sequelae of postoperative complications [3]. A successful revision repair requires a thorough understanding of shoulder anatomy, sufficient diagnosis of failed surgery, as well as comprehension of the causes of failure. Furthermore, well-grounded knowledge to confirm the indication for operative intervention and technical skills of surgical revision are needed [3].

One key point is to differentiate between a relevant and symptomatic rotator cuff re-tear and symptomatic comorbidities that have not been properly addressed or have been neglected in prior surgery. Essential is that a structural failure must not be identical with the cause of clinical failure and symptomatic comorbidities can be the leading reason for unsatisfactory outcome [3].

A. Otto · K. Beitzel · A. B. Imhoff (✉)
Department of Orthopaedic Sports Medicine, Hospital Rechts der Isar, Technical University of Munich, Munich, Germany
e-mail: alexander.otto@tum.de; beitzelknut@tum.de; imhoff@tum.de

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_28

28.2 Preoperative Assessment

Successful management of a failed rotator cuff repair strongly depends on a thorough assessment that leads to the therapeutic strategy. Evaluation of the affected shoulder consists of a detailed history, physical examination, and imaging [3].

An exact collection of patients' pre- and postoperative history combined with detailed knowledge of the prior surgical intervention through operative notes is the first important step. A review of the postoperative rehabilitation course can be the first hint on the etiology of the symptoms. The extent of pre- / postoperative physical therapy, the use of injections as well as medical comorbidities and nicotine use should be documented [3].

As a second crucial step, the clinical evaluation is not only focused on the affected shoulder. It is expanded to other potential pain generators and possible causes of referred pain (e.g., cervical spine) [2–4].

Especially in revision cases, a thorough radiological evaluation of the affected shoulder, using static (e.g., X-ray and MRI-, CT-scan) and dynamic examinations (e.g., ultrasound), is needed to verify the clinical suspicion [2–4].

28.3 Principles of Treatment

All gathered information through above mentioned assessment allow the clinician a reasoned treatment plan. Although a re-tear is present, certain cases are eligible for conservative treatment. According to pain or functional limitations in the postoperative course, the surgeon must determine whether revision repair is indicated or if a conservative treatment might be sufficient [2].

In general, the indications for revision rotator cuff repair are comparable to those of primary repair: Pain and limited shoulder function with the presence of a recurrent tear [3, 4]. Nevertheless, in the revision setting, rotator cuff status and patient-related factors need further diligent consideration [3, 4]. Although a sufficient preoperative evaluation was performed, the feasibility to repair the tendon defect adequately cannot be totally determined until the actual surgery [4]. In general, the postoperative outcome as well as the tendon healing are less predictable than in primary surgery [4]. Therefore, realistic expectations must be discussed [4]. Prior to surgery, the postoperative outcome can be optimized by physiotherapy [4].

Revision rotator cuff repair is indicated for recurrent tears in patients with a reasonable chance for tendon healing and a motivation for the straining postoperative protocol [2–4]. The ideal patient is less than 60–65 years of age, has an intact deltoid origin (no signs of atrophy), performs an active abduction >90° and a forward elevation >120° [2–4]. Furthermore, the muscles of the affected rotator cuff tendons should not possess advanced fatty infiltration (Goutallier ≤2°), the tendons itself should mandatorily have adequate quality on advanced imaging, and there should be no high-grade tear retraction (Patte ≤2°) [2–4].

Absolute contraindications for revision cuff repair are active infections, axillary nerve injuries, and cuff tear arthropathy (e.g., significant proximal humeral migration or arthritic changes in the glenohumeral joint) [4]. If the involved muscles of two or more torn tendons show advanced atrophy or fatty infiltration, revision cuff repair should not be performed, unless there is a high possibility to improve the patient's functionality through a partial repair [4].

In patients with unrepairable cuff tears showing significant loss of external rotation strength and no signs of advanced glenohumeral arthritis a tendon transfer may be indicated [3]. Alternatively, a superior capsular reconstruction for massive irreparable rotator cuff tears can be applied [5]. Here, a human dermal allograft or autograft fascia lata to restrain superior migration of the humeral head can be used [5]. In the presence of severe rotator cuff arthropathy, a reverse shoulder arthroplasty is indicated [3].

28.4 Operative Management

Starting with patient positioning and general approach, we would like to depict the concept to treat the main components of failed rotator cuff

repair as mentioned above. First, we focus on the sequelae of insufficient treatment through diagnostic inaccuracies and technical errors. Then, the management of postoperative adverse events, such as stiffness and infection, are presented. Finally, the arthroscopic surgery for recurrent or persistent lesions is depicted.

The general aim of rotator cuff surgery is the creation of a functional rotator cuff by restoring physiologic shoulder mechanics with either partial or complete repair [6]. Sufficient force transmission is restored through balanced force couples and a re-established suspension bridge system. Careful handling of the rotator cuff is extremely important since re-torn rotator cuff tissue can be of poor quality. Therefore, extensive tendon mobilization to achieve anatomic repair is often needed. The surgeon should not be bound to use portals from prior surgery, because reusing inappropriately placed portals can compromise surgical technique and outcome (e.g., intraoperative vision, anchor placement) [3].

28.4.1 Patient Positioning and General Approach

The authors prefer beach chair position using an arm holder that allows inferior traction to the humeral head and more additional internal or external rotation than in lateral decubitus. The common arthroscopic approach for revision rotator cuff repair begins at the anterior structures and moves to the posterior structures [6].

28.4.2 Insufficient Treatment

Persistent shoulder function impairment can occur due to prior diagnostic inaccuracies, missed intraoperative pathologies, as well as technical errors. The following sections focus on the treatment of those relevant pathologies. As the approach toward revision rotator cuff repair starts anteriorly [6], the treatment of LHB and subscapularis tendon is first presented. Then relevant acromioclavicular joint pathologies and material failures in rotator cuff revisions are brought into focus.

28.4.2.1 Long Head of Biceps Tendon

In our experience, a persistent shoulder affection through the long head of the biceps tendon occurs in 3.57% of revision cases. Therefore, a thorough assessment of the long head of the biceps tendon should be performed. Beginning at the superior labrum with its insertion, the intra-articular course is further followed, to rule out possible adhesions or (degenerative) signs for tendinopathy [2–4, 6]. Closer attention is brought to the medial and lateral pulley sling, if possible in external and internal rotation stress through the arm holder or by using a probe to provoke biceps instability. Biceps procedures should be performed in relevant SLAP lesions, signs of LHB tendinopathy, and to avoid late-onset biceps symptoms in the presence of a recurrent anterior supraspinatus tear, since an entrapment of the biceps tendon may occur after supraspinatus repair [4, 7]. The surgical technique is adapted to patient's age, body habitus, and physical demands as well as the surgeon's ability [4]. Generally, high-demanding patients, below 50 years and/or heavy laborer, should undergo a biceps tenodesis, whereas low-demanding patients, above 55 years, should receive a tenotomy [2].

Tenotomy of the LHB

Through an anterosuperior portal, using an arthroscopic scissor or electrothermal probe, the long head biceps tendon is transected close to the biceps anchor at the superior labrum (Fig. 28.1) [8]. Either spontaneously or following manual compression of the biceps muscle, a distal retraction is observed. A careful debridement of the biceps anchor and remaining LHB fibers must be performed with an electrothermal probe to avoid postoperative pain due to prominent remnants of the tendon.

Intra-Articular Tenodesis of the LHB

First, a holding suture is placed in the biceps tendon arthroscopically and followed by a biceps tenotomy. The biceps tendon is then pulled extracorporeal and a Krakow suture is applied. If subscapularis repair is needed, the biceps tenodesis will be performed afterwards. A guide wire is then placed and overdrilled at the top of the

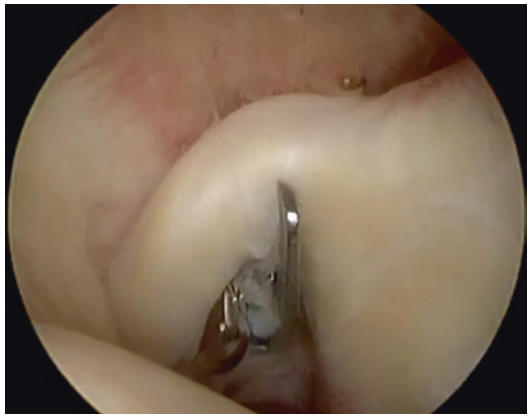

Fig. 28.1 Tenotomy of the LHB tendon using an arthroscopic scissor (Reproduced with permission from Springer (Ref. [8]))

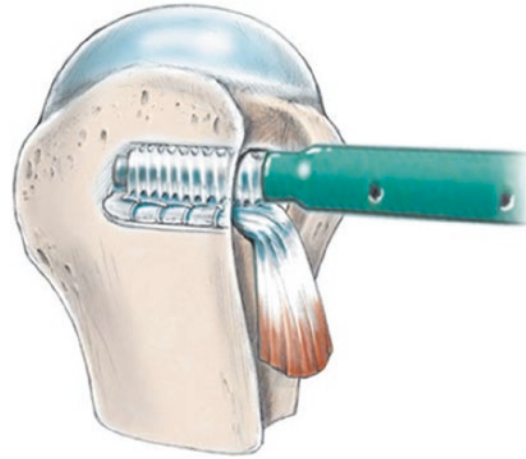

Fig. 28.2 Scheme of intra-articular tenodesis (Reproduced with permission from Springer (Ref. [8]))

bicipital groove. The biceps tendon is fixed using a biotenodesis screw or a suture anchor (Figs. 28.2 and 28.3). The thickness of the tendon is measured for the appropriate diameter of the drill hole. The length of the used device should be correlated to the depth of the tunnel.

Subpectoral Tenodesis of the LHB

Alternatively, a subpectoral tenodesis can be applied. Through an additional subpectoral approach, the biceps tendon is luxated and stitched with a nonabsorbable suture starting about 20 mm proximal to the musculotendinous junction (Fig. 28.4a). Proximal to the pectoralis major tendon and just distal to the bicipital groove, a guide wire is inserted perpendicular (unicortical) to the humeral shaft axis and overdrilled until reaching the opposite cortex (Fig. 28.4a). The tendon is fixed inside the bony tunnel using a biotenodesis screw or a cortical button (Figs. 28.4b and 28.5). The length of the used screw should be correlated to the depth of the tunnel to avoid any protrusion of the screw above the level of the humeral shaft [8].

28.4.2.2 Subscapularis Repair

If the patient shows a persistent affection of the subscapularis tendon, an accurate arthroscopic visualization should be performed. Even with an intact biceps pulley, a so-called hidden lesion of

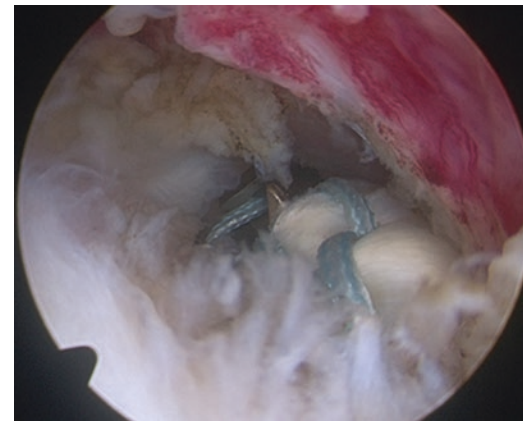

Fig. 28.3 Intra-articular vision after LHB tenodesis (while the used suture material is shortened)

the subscapularis was observed in 16.4% of the cases in open surgery for "isolated" supraspinatus tendon tears [9]. Especially in cases with the suspicion of a neglected lesion, a complete visualization of the subscapularis insertion, including both the intra-articular side as well as the bursal side of the tendon through debridement of the medial pulley and rotator interval is imperative [9]. From the medial pulley sling and the lesser tuberosity, the course of the subscapularis tendon is followed to the coracoid.

In some cases, the subscapularis might not be fully visible at first sight due to tendon retraction

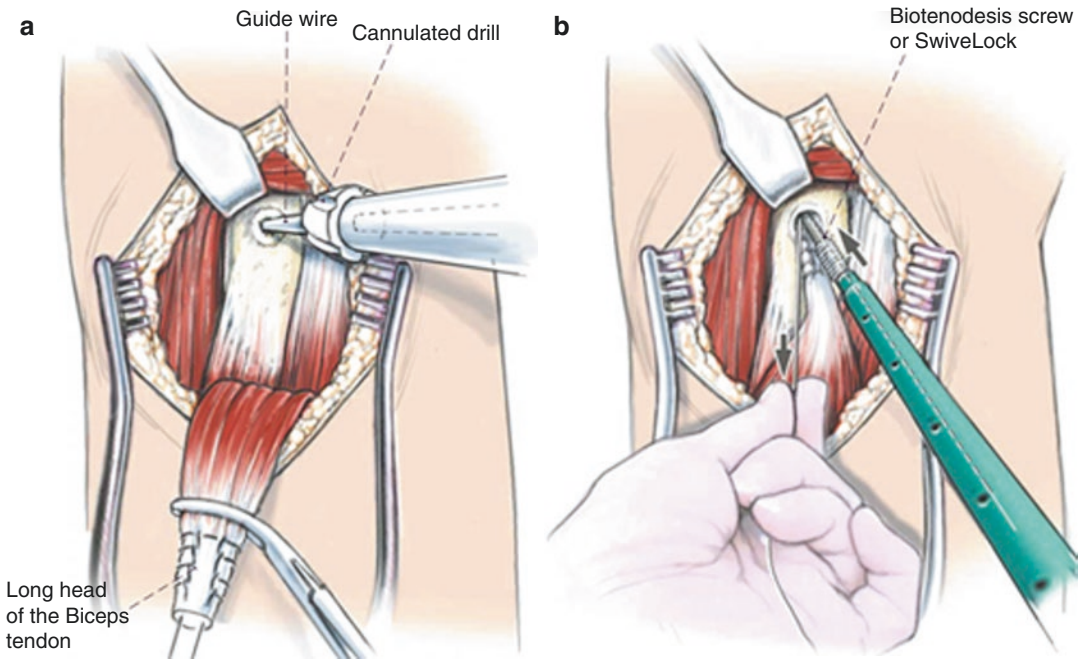

Fig. 28.4 Scheme of subpectoral tenodesis: drilling and reaming of the bone tunnel with a cannulated reamer over a guide wire (**a**), followed by a tenodesis screw fixation (**b**) (Reproduced with permission from Springer (Ref. [8]))

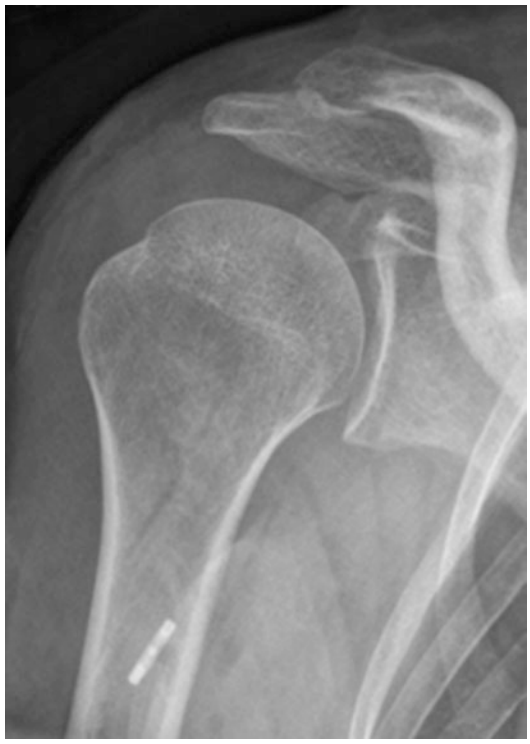

Fig. 28.5 Postoperative radiological control after subpectoral LHB tenodesis using a cortical button

or adhesions [6]. An additive anterolateral working portal is advantageous for addressing the subscapularis, the coracoid, and the lesser tuberosity in case of further needed dissection. A traction suture should be placed through the superior portion of the subscapularis to facilitate tendon identification (Fig. 28.6a). Now a three-sided release is performed with a shaver or electrothermic device beginning at the articular side. Then all scar tissue and adhesions between the glenoid and the subscapularis tendon are removed (Fig. 28.6b). Especially adhesive scar tissue connected to the medial glenohumeral ligament (MGHL) needs to be resected to reach unhindered tendon mobilization. Thus, an additional extensive reduction of MGHL itself is performed to prevent possible future adhesions that can limit function and outcome. Then the cranial and cranioventral subscapularis fibers are freed from adhesions, following the articular course to the coracoid arch. Further debridement of the coracoid neck and coracoid base has to be performed to fully free the subscapularis. In order to achieve better visualization, it can be helpful to switch

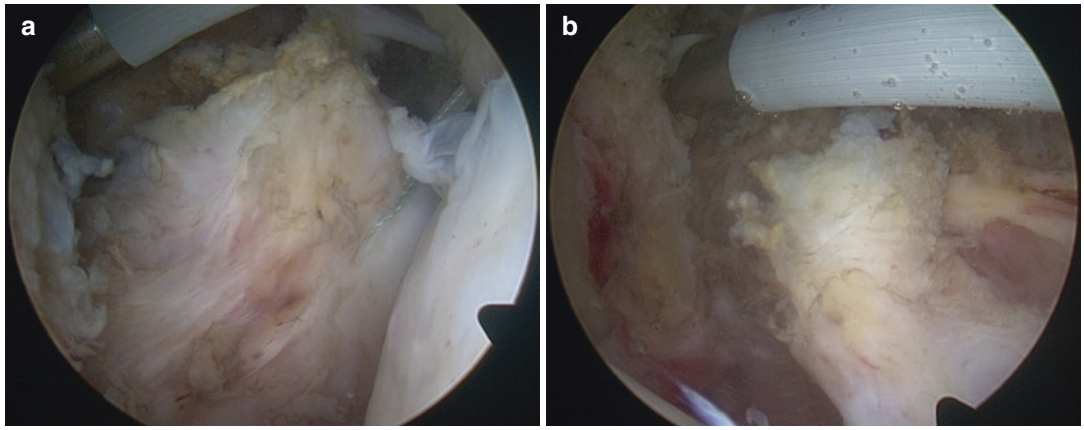

Fig. 28.6 (**a**, **b**) Tensioning of the tendon with a temporary traction suture and mobilization of the tendon through release of adhesions (Reproduced with permission from Springer (Ref. [8]))

portals for full vision of the coracoid. A release or dissection further medial to the coracoid is contraindicated due to the proximity of the neurovascular structures. After completion of the subscapularis release, a sufficient lateral mobilization to the lesser tuberosity through the used traction suture is usually obtained. Then the bone bed of the subscapularis is roughened with a shaver or burr to a bleeding base. If the tendon does not fully reach the anatomic position intraoperatively, the bone bed can be placed an additional 5–7 mm more medial without adversely affecting function. Depending on the amount of lateral mobilization of the tendon and loss of tendon structure in the revision setting, the repair of the subscapularis is performed through a single- or double-row suture bridge technique (Figs. 28.7 and 28.8) [6].

28.4.2.3 Arthroscopic Resection of the Acromioclavicular Joint

Even in severe revision cases, a standard acromioplasty should not regularly be performed. Instead a subacromial smoothing while preserving the coracoacromial ligament and the coracoacromial arch should be applied. In symptomatic cases and with radiological signs for acromioclavicular osteoarthritis, arthroscopic resection of the AC joint and co-planing can be performed (Fig. 28.9). However, it is crucial to avoid an overly excessive resection with following AC joint instability [10] or acromion fracture [3] (Fig. 28.10). An anterior portal in line of the AC joint is ideal to create a dome-shaped articular space under preservation of the superior and dorsal capsule [8].

28.4.2.4 Material Failure

As arthroscopic surgical technique evolved, so did fixation devices from non- to absorbable suture anchors [11]. Complications of the earlier used nonabsorbable anchors included loosening, migration, and incarceration of the metal implant within the joint, resulting in chondral damage. Although technological improvement takes place, even with nonabsorbable anchors, adverse events still occur (e.g., loosening, cystic resorption, osteolysis, and arthropathy) [11].

Regardless of the prior used anchor material, we observed material failure in 3.57% leading to revision rotator cuff repair. In order to avoid further inflammatory reactions and chondral defects, an arthroscopic removal should be performed. If prior used material can be assembled, a further microbiological assessment of the component is reasonable. Even if the patient describes the start of the symptoms 12 weeks after the operation and an appropriate tendon healing can be expected, a detailed assessment of the previous reconstructed tendon is crucial.

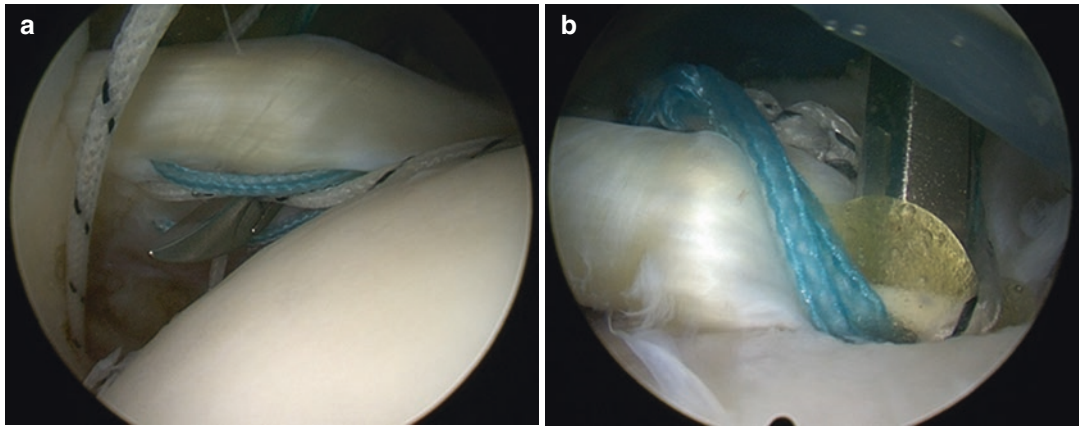

Fig. 28.7 (**a**, **b**) Suture passing through the subscapularis tendon and fixation in double-row technique

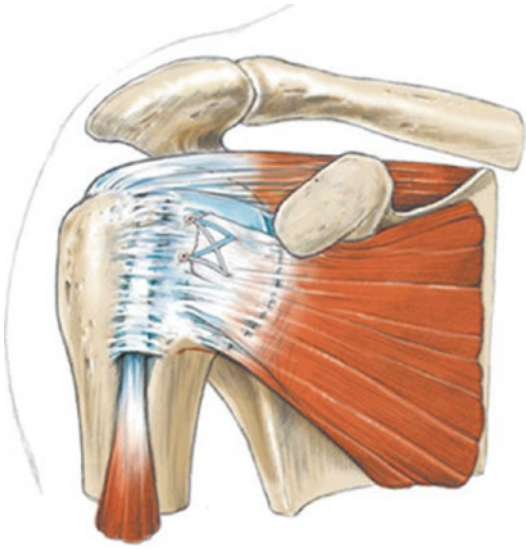

Fig. 28.8 Scheme of subscapularis repair in double row technique with suture bridge configuration (Reproduced with permission from Springer (Ref. [8]))

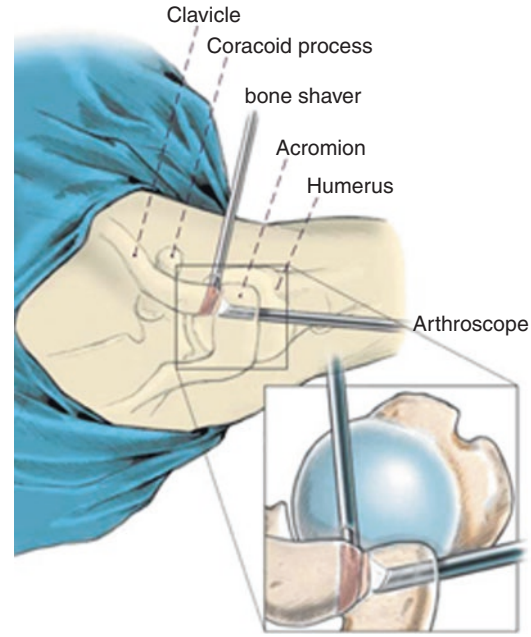

Fig. 28.9 Resection of the AC joint with a bone shaver through the anterior-superior portal (Reproduced with permission from Springer (Ref. [8]))

28.4.3 Shoulder Stiffness

An equal loss of passive and active motion with good strength may be the result of postoperative adhesions or capsular contractures [2, 4]. Limited passive external rotation is a sign for anterosuperior capsular contracture, whereas infringed internal rotation indicates posterior capsular contracture [2]. Huberty et al. observed the development of shoulder stiffness in 4.9% after arthroscopic rotator cuff repair [12].

If there is a persistent symptomatic motion deficit, despite intensive conservative treatment for more than 6 months with subsidence of inflammatory symptoms, an arthroscopic release is indicated [8]. Starting with the release of the rotator interval including, the superior as well as the medial glenohumeral and the coracohumeral

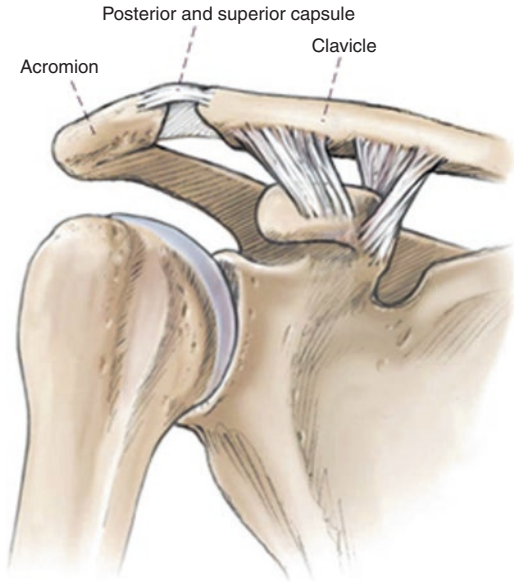

Fig. 28.10 Dome-shaped widening of the joint space with preservation of the superior and posterior parts of the joint capsule (Reproduced with permission from Springer (Ref. [8]))

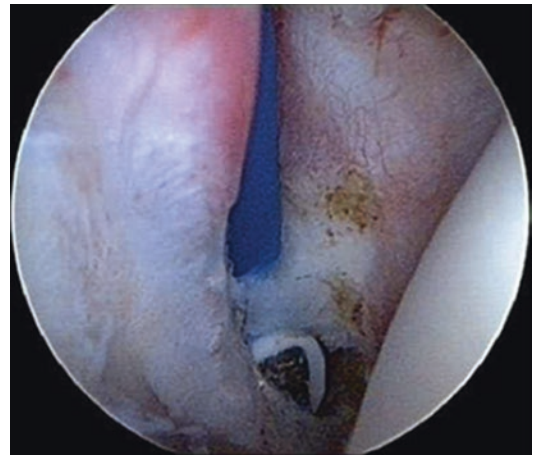

Fig. 28.11 Release of adhesions near the subscapularis tendon (Reproduced with permission from Springer (Ref. [8]))

ligament are transected (Fig. 28.11). Approximately 1 cm medial to the glenoid, the capsule is dissected clockwise to the 6 o'clock position. Especially at this position, the device should always point to the glenoid to protect the axillary nerve. The capsular release is completed posteroinferior through switching the arthroscope into the anterosuperior portal and performing the capsulotomy at the posterior glenoid (Fig. 28.12) [8].

28.4.4 Infection

Atwhal et al. observed after 4886 rotator cuff repairs 0.43% deep infections [13]. Propionibacterium acnes was found to be the most common isolated organism with 51% [13]. Coagulase-negative staphylococci species were identified in 31% and *Staphylococcus aureus* in 21% [13].

In our experience, 18% of rotator cuff revision cases showed deep infections. In order to eradicate the infection, surgical irrigation, debridement, and intravenous antibiotics are mandatory [3]. Even after successful eradication, the outcome is often affected by stiffness, adhesions, failure of the repaired tissue, and continuous pain [3]. If the surgeon has any doubts or signs for infection are visible in revision surgery, intraoperative samples for microbiological analysis need to be acquired.

28.4.5 Recurrent or Persistent Rotator Cuff Lesions

Both traumatic postoperative events and failed tendon healing can result in rotator cuff re-tears. Traumatic failure can occur in the early phase due to single traumatic events (e.g., fall on the abducted arm), overly aggressive rehabilitation, or noncompliance to postoperative limitations. Late traumatic failure is caused through acute injuries or repetitive trauma after complete rotator cuff healing [3].

The reasons for failed tendon healing are poor rotator cuff and major tuberosity vascularity, insufficient rotator cuff tissue, inferior bone quality, as well as advanced patient age. Failed rotator cuff repair is also associated with advanced muscle atrophy, fatty degeneration, and the affection of two or more rotator cuff tendons [3].

Due to postoperative adhesions and prior tendon sutures, an extensive subacromial prepara-

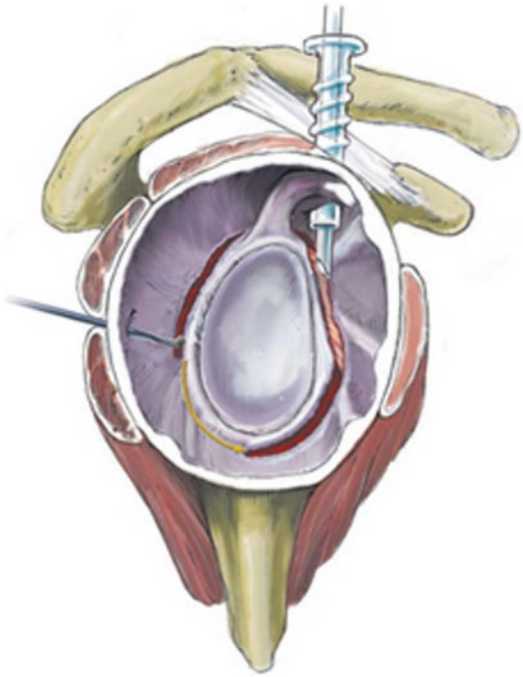

Fig. 28.12 Scheme of the capsular release with an electrocautery device (Reproduced with permission from Springer (Ref. [8]))

tion might be needed, in order to reconstruct the initial course of the tendon and to characterize the re-tear [3]. If an insufficient tendon mobility still is present, an anterior and/or posterior interval slide is indicated [6].

28.4.5.1 Subacromial Preparation

The arthroscope is placed subacromial to visualize the rotator cuff from above. Therefore, an accurate debridement of the subacromial space, under the premise to maintain as much functional tendon remnants as possible, is performed [6]. In order to protect the rotator cuff while removing scar tissue adherent to the acromion, the shaver or electrothermic device is orientated toward the acromion and moved from the scapular spine to the lateral edge of the acromion. An electrocautery probe and a shaver are alternately used to debride the undersurface of the acromion as far as to the AC joint and the scapular spine.

The subacromial preparation is further expanded until supra- and infraspinatus are fully visible and the tear can be inspected. Special effort is required for removing residual bursal layers while moving further lateral to the major tuberosity. Especially posterolateral adhesions need to be removed to avoid limited vision while repair. Scar tissue should be removed and adhesions should be circumferentially debrided at the bursal and articular sides of the rotator cuff [3]. If prior used sutures and anchor material are visible, they should be removed in order to reconstruct the initial course of the tendon and to assemble material for further microbiological assessment. While assessing the tear pattern (crescent shaped, U shaped, L shaped, or reverse L shaped) and size (small, medium, large), a grasper pulls the tendon laterally toward the insertion on the greater tuberosity (Figs. 28.13 and 28.14). An instant repair is possible, if the tendon can be mobilized to the footprint without inappropriate tension. Further soft tissue release is needed if the tendon does not reach the bone bed. As a crucial landmark, the scapular spine allows to differentiate between the fibers of supra- and infraspinatus. With traction sutures placed, the further soft tissue release and tendon mobilization can be performed under improved distinction of supra- and infraspinatus tendon [6].

28.4.5.2 Anterior and Posterior Interval Slide

An initial deep capsular release is followed by a thorough evaluation of the tendon mobility with an arthroscopic grasper of each portion from medial to lateral and anterior to posterior to the footprint. If an insufficient mobility can still be observed due to anterior or posterior boundaries, an anterior and/or posterior interval slide is indicated [6]. In general the surgeon must avoid to dissect more than 10–12 mm medial to the glenoid rim to minimize the risk for injury to the suprascapular nerve at the base of the scapular spine, while performing posterior interval slide [6].

An anterior interval slide is performed by a coracohumeral ligament release, beginning at the top of the biceps root toward the base of the coracoid using an electrothermic device while aiming 45° anteromedially. This is best achieved through a lateral portal, and the applied instrument

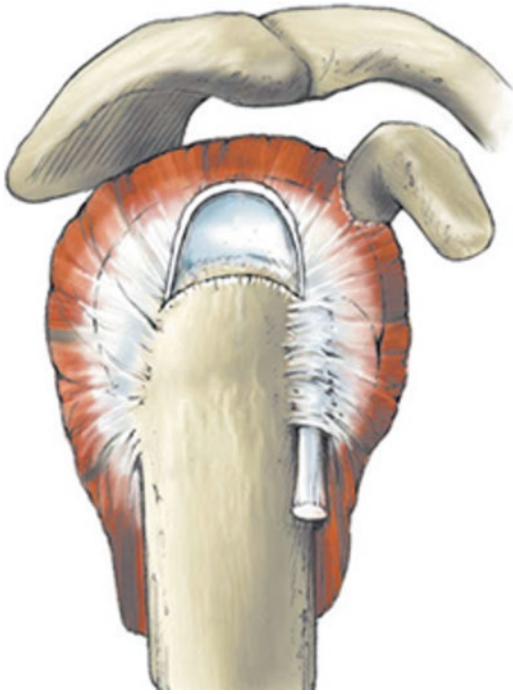

Fig. 28.13 U-shaped tear (Reproduced with permission from Springer (Ref. [8]))

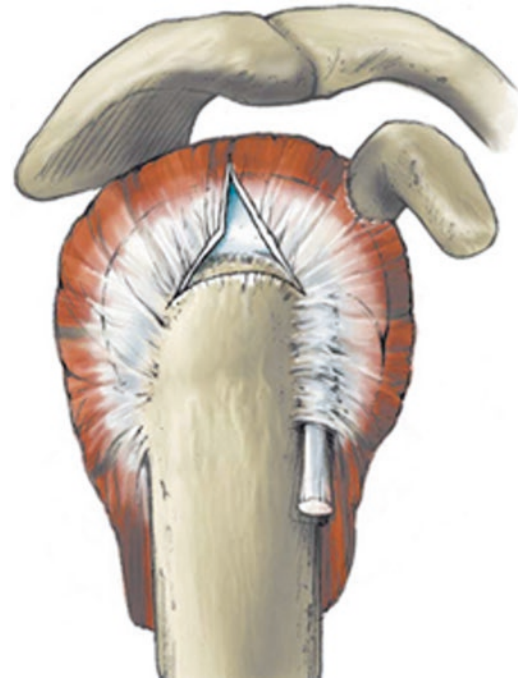

Fig. 28.14 L-shaped tear (Reproduced with permission from Springer (Ref. [8]))

should be placed safely toward the tissue/bone opposite to the tendon [6].

A posterior interval slide is best supported through two holding sutures at the posterior part of the supraspinatus tendon and at the anterior part of the infraspinatus tendon. Under lateral traction, a posterior interval slide is performed by separating the supra- and infraspinatus between the two sutures moving toward the base of the scapular spine with an arthroscopic scissor or electrothermic device. The incision continues medially until the perineural fat is approached to avoid injury to the suprascapular nerve. Enough additional lateralisation of the tendons is generally achieved through an interval slide to allow arthroscopic repair [6].

28.4.5.3 Supra-/Infraspinatus Revision Reconstruction

After accomplishing adequate tendon mobility and full preparation of the anatomic footprint, a supra- and/or infraspinatus repair can be performed. Before anchor placement, the greater tuberosity is roughened with a shaver or burr to create a bleeding bed to optimize tendon healing [4]. Prior to this step, instable or protuberant suture anchors with remaining suture material should be removed [3]. Although an excessive hardware removal may create bone defects that weaken the greater tuberosity and limit the fixation of the rotator cuff, a crowding of anchor material in the major tuberosity should be avoided [3]. Even with no visible hardware from prior surgeries, a tap should be used for anchor positioning to prevent suture anchor damage and subsequent failure from collision with embedded hardware. If new suture anchors are placed circumferential to prior suture anchor tracks, an oversized anchor should be considered to improve anchor stability [3].

If the needed tendon mobility is present, a double-row repair construct would be suggested, due to expected minor tendon quality and the superior construct strength (Figs. 28.15 and 28.16) [3, 14]. In tears with persistent retraction or in cases with tendon loss due to medial row

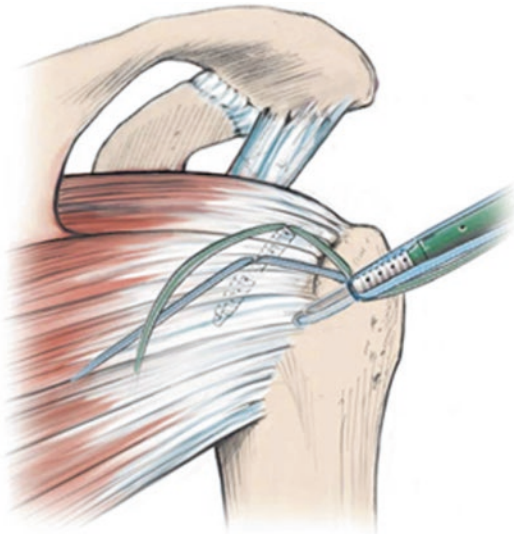

Fig. 28.15 First anchor of the lateral row (Reproduced with permission from Springer (Ref. [8]))

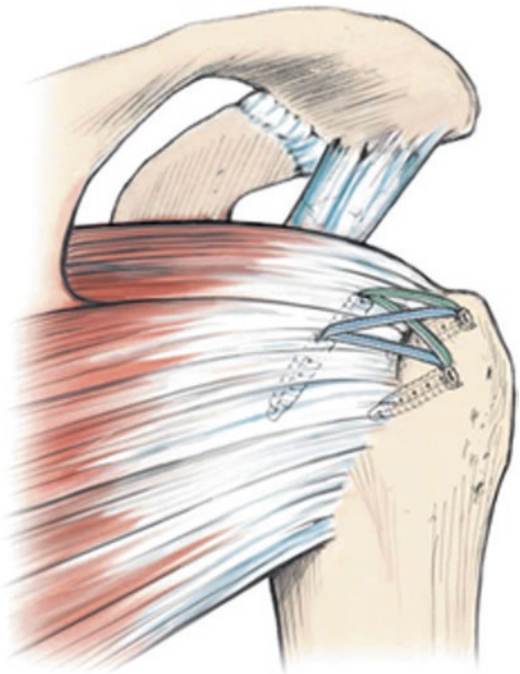

Fig. 28.16 The lateral row is completed with another anchor in similar technique (Reproduced with permission from Springer (Ref. [8]))

failure and the tendon does not fully cover the footprint, a single row repair is combined with side-to-side sutures (Fig. 28.17). Alternatively, in

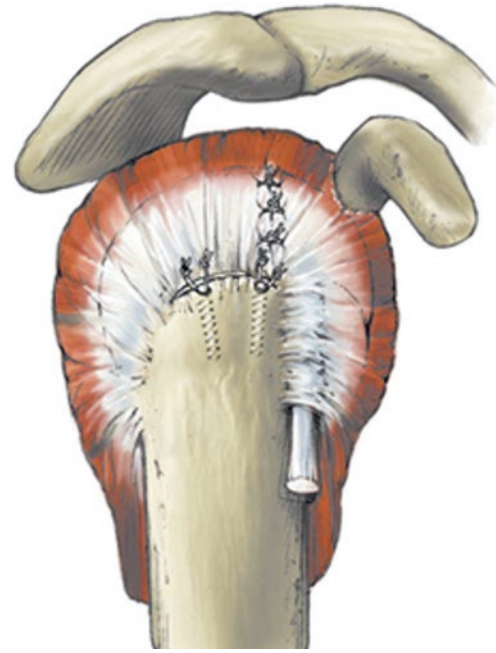

Fig. 28.17 Repairing a L-shaped tear through adaptation of the more mobile portion to the less mobile portion using side to side sutures and subsequent reinsertion to the bone (Reproduced with permission from Springer (Ref. [8]))

tears with restricted mobility and with greater retraction than sagittal plane width, a nonanatomic repair through a margin convergence can be achieved (Fig. 28.18) [3]. The margin convergence effectively lateralizes the edges of the torn tendon, which permits to close the defect near the footprint at the major tuberosity. The tendon edges are then repaired to the tuberosity with suture anchors either with a single-row construct or a double-row construct [4].

In some instances, only a partial tendon repair may be feasible. In order to reestablish as much of the rotator cuff force couple as possible, the subscapularis and infraspinatus tendon are repaired while the superior unrepairable defect is left aside. This may allow pain relief through humeral head depression and restoration of shoulder function [4].

28.4.5.4 Postoperative Rehabilitation

Our postoperative rehabilitation in revision cases is similar to the course after primary rotator cuff

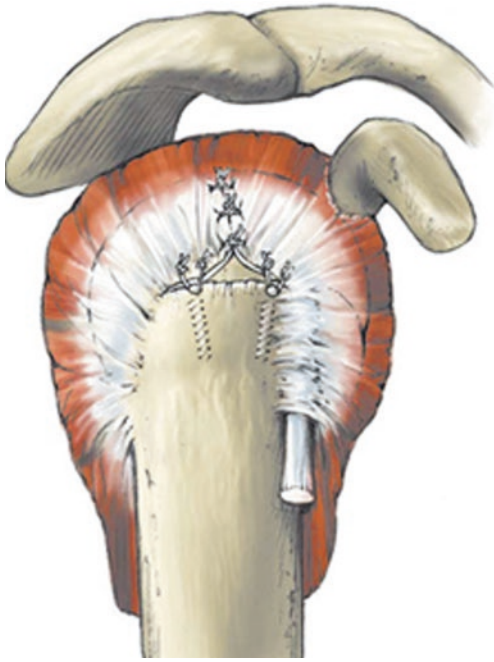

Fig. 28.18 Repairing a U-shaped tear using side-to-side sutures to decrease the size of the tear ("margin convergence") and consecutive reinsertion to the bone (Reproduced with permission from Springer (Ref. [8]))

repair. Definitely, a slower progression to active motion may be considered in cases with poor intraoperative tendon quality and partial repair. However, it should be avoided to excessively decelerate the progression of passive range of motion, which can consequently lead to postoperative stiffness and limited results. Therefore, we recommend the usage of a shoulder abduction pillow for 6 weeks after revision rotator cuff repair. Patients are permitted to remove the sling periodically for physiotherapy and range of motion activities of the elbow, forearm and wrist several times a day. The range of motion is limited for 6 weeks depending on the reconstructed tendons. In the first three postoperative weeks the patients are only allowed to be passively moved in the glenohumeral joint. Active-assisted movement is then performed until the sixth postoperative week. Free active-assisted movement is reached in the seventh week, and unlimited active range of movement is achieved in the ninth postoperative week.

References

1. Burkhart SS, Lo IK. Arthroscopic rotator cuff repair. J Am Acad Orthop Surg. 2006;14(6):333–46.
2. Strauss EJ, McCormack RA, Onyekwelu I, Rokito AS. Management of failed arthroscopic rotator cuff repair. J Am Acad Orthop Surg. 2012;20(5):301–9.
3. George MS, Khazzam M. Current concepts review: revision rotator cuff repair. J Shoulder Elb Surg. 2012;21(4):431–40.
4. Keener JD. Revision rotator cuff repair. Clin Sports Med. 2012;31(4):713–25.
5. Thorsness R, Romeo A. Massive rotator cuff tears: trends in surgical management. Orthopedics. 2016;39(3):145–51.
6. Ladermann A, Denard PJ, Burkhart SS. Revision arthroscopic rotator cuff repair: systematic review and authors' preferred surgical technique. Arthroscopy. 2012;28(8):1160–9.
7. Kim SJ, Lee IS, Kim SH, Woo CM, Chun YM. Arthroscopic repair of concomitant type II SLAP lesions in large to massive rotator cuff tears: comparison with biceps tenotomy. Am J Sports Med. 2012;40(12):2786–93.
8. Imhoff AB, Feucht MJ. Surgical atlas of sports orthopaedics and sports traumatology. Berlin: Springer; 2015.
9. Neyton L, Daggett M, Kruse K, Walch G. The hidden lesion of the subscapularis: arthroscopically revisited. Arthrosc Tech. 2016;5(4):e877–81.
10. Beitzel K, Sablan N, Chowaniec DM, et al. Sequential resection of the distal clavicle and its effects on horizontal acromioclavicular joint translation. Am J Sports Med. 2012;40(3):681–5.
11. Dhawan A, Ghodadra N, Karas V, Salata MJ, Cole BJ. Complications of bioabsorbable suture anchors in the shoulder. Am J Sports Med. 2012;40(6):1424–30.
12. 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(8):880–90.
13. Athwal GS, Sperling JW, Rispoli DM, Cofield RH. Deep infection after rotator cuff repair. J Shoulder Elb Surg. 2007;16(3):306–11.
14. 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(3):407–14.

How to Manage Failed Rotator Cuff Repair: Biologic Augmentation

29

Paolo Avanzi, Luca Dei Giudici, Antonio Gigante, and Claudio Zorzi

29.1 Introduction

Recurrent tearing or failure to heal of rotator cuff repair complicates up to 94% of the cases [1, 2] leading to a "failed rotator cuff syndrome," represented by continued pain, weakness, and limited active range of motion (ROM) [3]. Results of revision repairs tend to be similar to those of a primary procedure in the short-term follow-up. Mora et al. [4] recently reported on 51 cases after a mean 25 months, showing reliable improvement in shoulder function, pain, and satisfaction. However Shamsudin et al. [5] showed that those good results do not persist over the 2 years' time mark, with patients being twice as likely to have a retorn tendon, impaired ROM, strength, and residual pain when compared to a primary repair control group.

With the development of new technologies and bioengineered devices, more solutions are becoming available to fulfill the ultimate goal of a rotator cuff revision repair: a functional cuff that provides a normal biomechanics [6].

P. Avanzi · C. Zorzi
Department of Orthopaedics, Sacro Cuore – Don Calabria Hospital, Verona, Italy
e-mail: paolo.avanzi@sacrocuore.it

L. Dei Giudici (✉) · A. Gigante
Orthopaedic Unit, Casa di Cura "Villa Igea" Hospital, Ancona, Italy

29.2 The Concept of Biological Augmentation

Insufficient biological healing and tension overload are the two main factors involved in a failed repair and the limits that actual research tries to overcome.

Typical healing of a torn tendon does not regenerate the tendon-bone architecture formed during prenatal development but results in a weaker fibrovascular scar [1]. Incompletely covered repairs might result in less than optimal healing [7], while maximizing the contact area and contact pressure between the tendon and bone might enhance biological healing, strength, and tendon function [8]. Burkhart et al. [9] showed how a gap of at least 5 mm between the repaired tendon and its footprint has fewer chances to heal and that this gap most probably forms due to low-level loading of the rotator cuff repair with routine muscle contraction. For this reason an ideal repair needs a high initial fixation strength, the restoration of the anatomic footprint, the minimization of gap formations, and a mechanical stability until biologic healing occurs [10].

In the last decade, the concept of "biological augmentation" was applied to rotator cuff repairs, intended as a way to enhance the healing response and to provide a mechanical bridge for a tension-free repair. Several methods were proposed, classified according to mechanism of delivery and

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_29

cell type [10]. Vehicles range from in situ delivery to scaffold materials, while cell types include individual growth factors (GFs), stem cells, or a combination of both [10]. Goals of biologic augmentation are structural stability, improved biochemical environment, and complete biocompatibility [11]. Treatments are indicated in patients with massive and retracted tears but, with healthy muscles and poor tendon quality, conditions often seen in revision settings [12].

Biological augmentation can be also classified as "biomechanical" and "biochemical augmentation."

"Biomechanical augmentation" includes grafts of various types (auto-, xeno-, and allografts, synthetics), also called scaffolds or patches, that exert a direct predominantly mechanical effect of reinforcement on the repairs. These scaffolds were found associated to different degrees of secondary indirect biological effects that led to a better healing.

"Biochemical augmentation" includes all the procedures involving the addition of macromolecules like growth factors (GF), stem cells, and drugs; those will exert a predominantly biochemical effect on the healing, while the mechanical effect will vary depending on the vehicle used for their delivery (i.e., injections or scaffold coating).

29.3 Biomechanical Augmentation

Scaffold augmentation demonstrated promising outcomes with a low rate of failure [13] by protection of the repair during the immediate postoperative period, thus enhancing the rate and quality of the healing [14]. These augmentations provide a collagen-based structure to the repair area, and over time host cells populate the scaffold gradually remodeling it, providing for a better healing response and ultimately improving the quality of the resulting tissue [15]. Quality of tendon-to-bone fixation is also mechanically influenced, as scaffolds were shown to mitigate the reduction of mechanical properties of a difficult repair by bearing 45% of the total load [16].

Two main scaffold categories are available, biologicals and synthetics; the former includes autografts, xenografts, and allografts, and the latter includes bioengineered polymeric matrices. Biological scaffolds are providing compelling evidences. The addition of such a graft reduced gap formation from 40 to 3% at the tendon-bone repair site, markedly increased the load necessary to produce the critical gap of 5 mm, and have the capacity to protect the repair by supporting a mean of 35% of the global load applied to the repair [17]. Synthetic scaffolds hold increasing interest due to the good clinical outcomes, but several concern exist, such as limited in growth potential and issues related to foreign material reactions, with possible acute inflammatory response or chronic inflammation [18].

Up to date, there is no consensus whether to prefer one type of scaffold than another, as each has advantages and disadvantages, there are no long-term outcome studies, and randomized clinical trials are still too few. A recent review compared short-term results of allografts, xenografts, and synthetic scaffolds versus standard primary repair, advising for human dermal allografts [19]. Those were found associated with superior functional and structural outcome; no differences were found; instead, comparing xenograft augmentation with standard repairs and a possible correlation with worse rerupture rates and occasionally severe inflammatory reactions were suspected.

29.3.1 Basic Principles and Decision-Making

To apply successfully a biomechanical augmentation, every aspect that could predict a retear should be assessed and addressed. A recent analysis of 1000 consecutive rotator cuff repairs revealed that retears are multifactorial processes best predicted by tear measures (size, area, and thickness) [20]; a strong association was found between lesions bigger than 2 cm^2 and retear rate, increasing in a linear fashion [21]. Therefore, an extensive release of the soft tissues

must be performed during revision repairs. Other associations were found for muscle quality and tendon retraction. Muscle must be assessed for tropism according to Fuchs et al. [22] and for fatty infiltration according to Goutallier et al. [23]. Tendon retraction must be assessed according to Patte [24]. The combination of the several grades of the abovementioned factors dictates the reparability of the torn cuff and the indications for augmentation. The overall balance of the joint is the last tear-depending factor that must be considered; it depends on the involvement of the cable: if this structure is disrupted, it will create an unbalanced shoulder with pseudoparalysis and humeral head upper migration, translating in a repair with poorer outcomes [25]; if it is intact, even in a setting of a massive tear, the patient will retain good functionality and the repair will have more success. An indirect sign for the evaluation of the joint balance is the migration of the humeral head according to Fukuda et al. [26].

Besides tear-related factors, other aspects to account for are previous surgeries on the affected shoulder and the hardware used, as both metallic and resorbable anchors could create unforeseeable technical complications; suture techniques, as suture bridge was demonstrated to better preserve the repair at the insertion site [27]; and patient's activity demand. Patient's age at surgery is, instead, a strong independent factor related to retears [20].

Biomechanical augmentation is therefore indicated for a reparable tear, defined as a balanced shoulder with a Fuchs index up to neutral, a Goutallier index up to two, and a retraction type early or late, with minimal signs of osteoarthritis. Typical patient is young, presenting a superior or posterior-superior tear with an intact subscapularis and a preserved forward elevation. If a massive tear is present in such a patient, a latissimus dorsi transfer could be more suitable [28], but if every other criteria are in place, an augmentation is still indicated; it will not be indicated in elderly patients with massive tears, where a spacer or a joint replacement is more suitable, depending on the progression of the osteoarthritis.

29.4 Results

Biomechanical augmentation was performed using autografts, allografts, synthetic grafts, and xenografts.

Biceps tendon was one of the first autograft used for cuff repair biological augmentation [29], due to a very often concomitant biceps tendon pathology requiring tenotomy and/or tenodesis and therefore the excision of its proximal portion. It gives the advantage of a readily available tissue that does not create a donor-site morbidity, providing more collagen to the repair and, therefore, more potential healing [30]. Fascia lata was another promising graft for its properties, very similar to those of the rotator cuff tendons [22, 23]. A renewed interest arose in the recent years investigating the advantages of reinforcing fascia lata grafts with poly-L-lactic acid (PLLA) or PLLA/polyglycolic acid polymer braids and its application on rotator cuff repairs, in a cadaveric model [33]. It provided mechanical augmentation and minimized tendon retraction showing a gap formation reduced by 48% and showed significantly better mean postoperative scores with a lower retear rate, detected on MRI (8.3% vs 41.7%) after a mean follow-up of 35 months [34]. Other autografts were described. Coracoacromial ligament (CAL) [35] resulted in excellent outcomes in terms of subjective functionality scores and ROM evaluation, showing normal tendon signals on MRI and with no development of complications, after 2 years; iliotibial band [36] and periosteal flap [37] showed higher rates of complications.

Allografts are allogenic matrices produced by decellularization of cadaveric material from humans in order to reduce the risk of graft rejection [38]. These increase the strength of the repaired tendon [39] and the time zero failure loads, despite the method of application [39]; however the elastic moduli of allografts are less than that of autogenic tendon [40]. There have been concerns about the presence of residual DNA that could increase inflammatory response and degeneration [32]. A human dermal matrix was demonstrated to bring a significant contribution to tendon repairs, complete healing

on MRIs, and excellent postoperative scores [13, 44–47].

Synthetic patches were first investigated by Ozaki et al. [42] in 1986; after them several authors reported on the advantages offered by those materials. Gore-Tex patches showed high elongation values [43]; a woven poly-L-lactide graft, instead, showed up to 76% of increase in ultimate load increase [44]. Patches made of aligned nanofibers were found to have a higher elastic modulus, yield strength, and ultimate strength compared to unaligned ones and appeared able to affect the cellular response, with fibroblasts attaching neatly along the major axis [38]. Recent clinical studies presented promising results in terms of healing rate, biocompatibility, regenerated tendon area, ultimate load strength, ROM recovery, and retear rates [45–47]. Some concerns emerged over the degradation products of the polymers used to produce these grafts. High levels of lactic and glycolic acid can be produced, which impair osteoblast proliferation and inhibit mineralization of the matrix, whereas in nontoxic concentrations they decrease cellular proliferation and increase differentiation of osteoblasts [48].

Xenografts are extracellular matrices derived from xenogenic material, used as excellent 3D scaffolds for the regeneration of musculoskeletal, dermal, cardiovascular, and gastrointestinal tissues [49] treated with gamma irradiation and other techniques to be decellularized and to warrant immunogenicity but with enhanced molecules liberation. Two graft sources are commonly used: porcine small intestinal submucosa (SIS) and porcine dermis. SIS contains collagen (type I) and several growth factors (TGF-b, FGF-2, VEGF) [30, 31] and was applied as a biological scaffold to support cellular attachment, angiogenesis, and collagen formation, hoping to form a structure similar to the native enthesis [50]. Clinical applications revealed that it does not improve the healing rate nor the outcome scores [51], it shows several reruptures and persisting symptoms [52, 53], and the general advice is against its use.

Porcine acellular dermal patches are produced by decellularization of porcine dermal matrix accomplished by high-salt and high-detergent processes, maintaining architecture and biochemistry of the dermis [54]; they have shown repopulation and revascularization, minimal inflammatory response elicited by the host in animal models, and a propensity to remodel to a fascia-like architecture by 6 months [55] (Fig. 29.1). Integration with the adjacent tendon tissue was demonstrated at 24 months, without

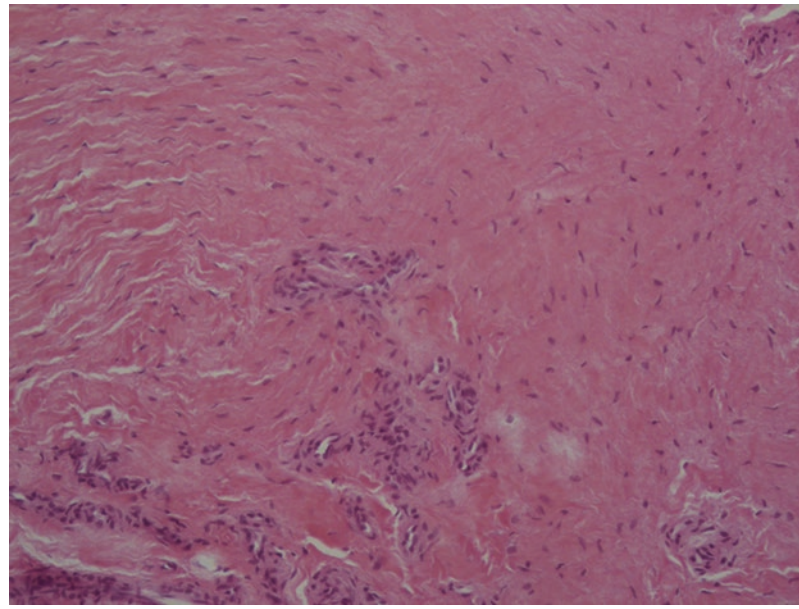

Fig. 29.1 Biopsy taken after 6 months of a supraspinatus tendon repair augmented with a porcine dermal scaffold. Hematoxylin and eosin staining. A proper spatial orientation of the collagen fibers (along the major axis and organized in crimps) is notable Some areas of vacuolar degeneration are noted, along with fibrotic tissue with monomorphonuclear cells

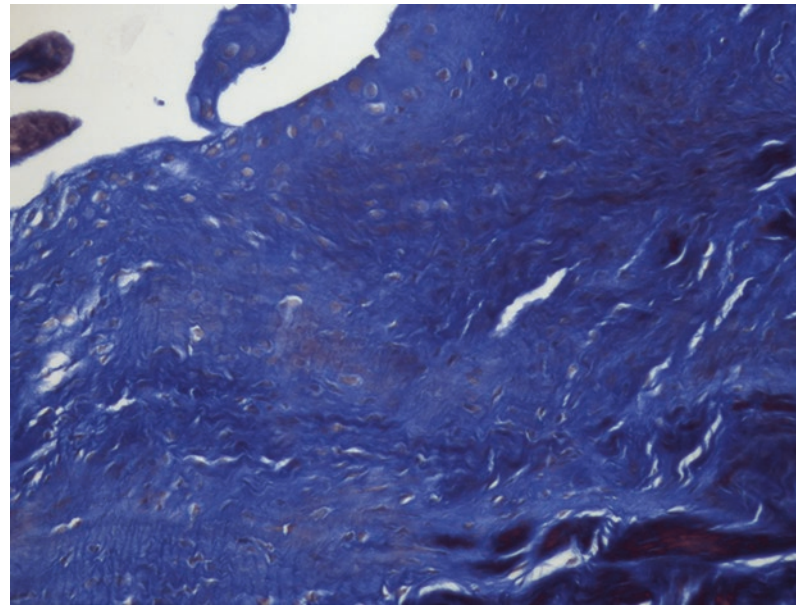

Fig. 29.2 Biopsy taken after 6 months of a supraspinatus tendon repair augmented with a porcine dermal scaffold. Trichromic Masson staining. More evident crimps are notable, with areas of neovascularization and chondrocyte- and tenocyte-like cells

macrophages and giant cells infiltration, without areas of calcification, fibrocartilage, and ectopic bone, and appearing similar to a mature tendon-bone insertion [56]. Moreover, if non-cross-linked, those patches demonstrated to promote the expression of collagen type I and III in the tenocytes, molecules responsible for tendon strength, healing, and fibrosis [57] (Fig. 29.2). These findings can explain a 50–60% increase in the maximum force at failure [57].

Clinical outcome showed promising results as well. Petri et al. [58] concluded that patch augmentation was a safe and effective treatment even for patients with massive, retracted rotator cuff tears providing a 9/10 satisfaction rate with no complications or problems associated with the graft itself and statistically significant improvements on all functionality scores. Badhe et al. [59], after 4.5 years from surgeries, reported excellent outcomes for chronic, extensive rotator cuff tears, without complication, allergic, toxic, or foreign body reactions. Similar results were obtained also by Giannotti et al. [60] that after 2.5 years of mean follow-up showed a great improvement in clinical scores, US imaging, and MRI. Other authors, although, reported worst outcomes when comparing a porcine dermal patch to other graft types, showing lower forward extension, abduction, and external rotation, and functional scores, with a retear rate of 44%, in comparison to 23% of allografts and 15% of synthetic grafts [61].

29.4.1 Authors Preferred Technique

The several possibilities available reflect that it is still an evolving field. Biomechanical augmentations were more widely applied both in vitro and in vivo; it seems more prudent, at this stage, to prefer this approach. Xenografts and allografts are the two types of scaffold with the best outcome; the differences are slight and the choice of one over another resides in the surgeon hands and experience. The authors of this chapter began to apply a porcine, non-cross-linked dermal scaffold since 2013 in an ongoing not yet published RCT, experiencing a healing rate of 96% versus 68% of their standard repair; extensive MRI evaluation also showed outstanding findings, along with ROM and strength recovery. The performed surgical technique (the Goal Post) was published recently [62] (Fig. 29.3).

Recent published comparisons also seem to indicate a slight advantage for the xenograft group. Beitzel et al. [63] demonstrated superior results in terms of cell adhesion, proliferation,

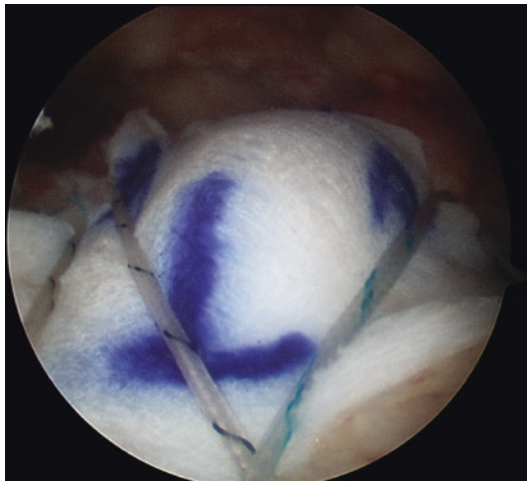

Fig. 29.3 Operative arthroscopic picture of a porcine dermal patch scaffold implantation according to the "Goal Post" technique [62]

and general histologic evaluation. Proctor [45] reported a 78% healing rate at 42 months after surgery and an improvement in all the scores submitted. Barber et al. [13] had similar results with an 85% healing rate at 14.5 months without complication.

The advantages of rotator cuff augmentation could have several explanations. First of all, the addition of a patch creates a "superior capsule reconstruction effect" [64]. Moreover, a self-reinforcing effect takes place as showed by Burkhart et al. [65], protecting the repair without increasing its stiffness, warranting an increased ultimate load to failure.

29.5 Biochemical Augmentation

Biochemical augmentations are those procedures that add beneficial molecules to the site of the repair. Those molecules can be autogenous or synthetized in a laboratory, and the enhancement relies almost exclusively on local metabolism. Several delivery systems are available and could add positive effects on the repairs, for example, the coating a synthetic scaffold with those molecules will add all the benefits aforementioned. Many applications are being studied, and even if in an early stage, some findings are promising.

Platelet-rich plasma (PRP) was seen with increasing interest in the last decades as a mean to locally deliver endogenous growth factors (GF) through a simple injection, and its use was described and evaluated for different pathologies as several in vitro studies have shown that growth factors promote the regeneration of bone, cartilage, and tendons [66]. The wide usage and the unstandardized preparation methods represent also a limit. Many authors investigated its feasibility for rotator cuff tear augmentation [67–71]. No difference between augmented and non-augmented was noted, and its application is not recommended.

A sustained release of growth factors was obtained by impregnation of a biodegradable gelatin hydrogel sheet (GHS) with bone morphogenetic protein 7 (BMP-7) [72] obtaining favorable collagen fiber orientation after 8 weeks along with more chondrocytes at the tendon-bone insertion, indicating a positive stimulation on the tenocyte matrix production. The same findings were not observed in the study group treated without the GHS carrier, suggesting that it acts as a GF reservoir. BMP-2 was applied on an acellular dermal patch [73] and showed new bone formation after 4 and 8 weeks, an increase of the ultimate failure load to the level of intact tissues, and with rich cell penetration at the tendon-bone interface that were induced along the chondrogenic line.

Advanced scaffolds made in nanofibers and processed through electrospinning showed the ability to aid in cell attachment and proliferation, obtaining a faster remodeling than dermal patches [74]. Zhao et al. [75] loaded such a scaffold with basic fibroblast growth factor (bFGF). Bioactivity of the bFGF was maintained during the first 3 weeks, and in the subsequent period, an increased extracellular matrix proliferation was observed, and at 8 weeks the scaffold was absorbed showing an improved collagen organization and a more mature enthesis compared to non-augmented group.

Mesenchymal stem cells (MSC) can also be delivered at the repair site. MSCs can be obtained by direct bone marrow (BM) stimulation or harvested from sites like the iliac crest. These were found to be metabolically active providing higher amount of fibrocartilage formation and better ori-

entation of fibrocartilage fibers [76]. In a comparison with a conventional repair, a lower retear rate was observed for the augmented cases, suggesting a positive influence on the repair [77].

Adipose tissue-derived stem cells (ASC) appeared able to differentiate into tenocytes and myocytes [78] and to release GFs and cytokines [79]. Oh et al. [80] published the first study in a rotator cuff model using ASCs comparing four groups for a rabbit subscapularis tendon suture, using saline, saline and ASCs, only ASCs, and only suture. They found better healing properties and a capacity of regeneration after fatty infiltration of the muscle.

29.6 Salvage Procedures

When an augmentation is contraindicated, salvage procedures for rotator cuff repair revision can be taken into consideration. The goal is to obtain a pain-free and balanced shoulder by reducing the humeral head translation, delaying the need for more demanding surgeries.

A bridging technique is performed filling the gap created between the retracted tendon and the humeral footprint by interposition of one of the abovementioned grafts [81]. Best results were obtained using human dermal allograft [41, 81], reporting significant improvements in pain, ROM, and strength at an average 3-year follow-up with a 75% healing rate [82]; however, concerns about the potential long-term medial pullout failures remain.

Technically similar, superior capsule reconstruction (SCR) requires the lateral fixation of a graft on the greater tuberosity and the medial fixation on the glenoid; it fully restores humeral translation, increasing joint stability and function and reducing pain [83].

Patients with massive irreparable tears, mild osteoarthritis, and an age that is too old for a tendon transfer but still young for a joint replacement could gain benefits from a subacromial spacer. This is a promising balloon-shaped biodegradable device to be inserted in the subacromial space, reducing the humeral translation and therefore pain, which could be used as a temporary treatment [84].

References

1. Weeks KD, Dines JS, Rodeo SA, Bedi A. The basic science behind biologic augmentation of tendon-bone healing: a scientific review. Instr Course Lect. 2014;63:443–50.
2. Baleani M, Schrader S, Veronesi CA, Rotini R, Giardino R, Toni A. Surgical repair of the rotator cuff: a biomechanical evaluation of different tendon grasping and bone suture fixation techniques. Clin Biomech (Bristol, Avon). 2003;18:721–9.
3. Strauss EJ, McCormack RA, Onyekwelu I, Rokito AS. Management of failed arthroscopic rotator cuff repair. J Am Acad Orthop Surg. 2012;20:301–9. https://doi.org/10.5435/JAAOS-20-05-301.
4. 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 Sport Traumatol Arthrosc. 2016;25(7):2157–63. https://doi.org/10.1007/s00167-016-4392-7.
5. Shamsudin A, Lam PH, Peters K, Rubenis I, Hackett L, Murrell GAC. Revision versus primary arthroscopic rotator cuff repair. Am J Sports Med. 2015;43:557–64. https://doi.org/10.1177/0363546514560729.
6. Lädermann A, Denard PJ, Burkhart SS. Revision arthroscopic rotator cuff repair: systematic review and authors' preferred surgical technique. Arthrosc J Arthrosc Relat Surg. 2012;28:1160–9. https://doi.org/10.1016/j.arthro.2012.01.006.
7. 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;25:1093–100. https://doi.org/10.1016/j.arthro.2009.07.010.
8. Tuoheti Y, Itoi E, Yamamoto N, Seki N, Abe H, Minagawa H, et al. Contact area, contact pressure, and pressure patterns of the tendon-bone interface after rotator cuff repair. Am J Sports Med. 2005;33:1869–74. https://doi.org/10.1177/0363546505278256.
9. Burkhart SS, Diaz Pagàn JL, Wirth MA, Athanasiou KA. Cyclic loading of anchor-based rotator cuff repairs: confirmation of the tension overload phenomenon and comparison of suture anchor fixation with transosseous fixation. Arthroscopy. 1997;13:720–4.
10. 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;72:89–96.
11. Labbé MR. Arthroscopic technique for patch augmentation of rotator cuff repairs. Arthrosc J Arthrosc Relat Surg. 2006;22:1136.e1–6. https://doi.org/10.1016/j.arthro.2006.03.022.
12. Petri M, Greenspoon JA, Moulton SG, Millett PJ. Patch-augmented rotator cuff repair and superior capsule reconstruction. Open Orthop J. 2016;10:315–23. https://doi.org/10.2174/1874325001610010315.

13. 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;28:8–15. https://doi.org/10.1016/j.arthro.2011.06.038.
14. Ricchetti ET, Aurora A, Iannotti JP, Derwin KA. Scaffold devices for rotator cuff repair. J Shoulder Elb Surg. 2012;21:251–65. https://doi.org/10.1016/j.jse.2011.10.003.
15. DeFranco MJ, Derwin K, Iannotti JP. New therapies in tendon reconstruction. J Am Acad Orthop Surg. 2004;12:298–304.
16. 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 Elb Surg. 2012;21:1064–71. https://doi.org/10.1016/j.jse.2011.05.014.
17. Shea KP, Obopilwe E, Sperling JW, Iannotti JP. A biomechanical analysis of gap formation and failure mechanics of a xenograft-reinforced rotator cuff repair in a cadaveric model. J Shoulder Elb Surg. 2012;21:1072–9. https://doi.org/10.1016/j.jse.2011.07.024.
18. Mikos AG, McIntire LV, Anderson JM, Babensee JE. Host response to tissue engineered devices. Adv Drug Deliv Rev. 1998;33:111–39.
19. Ferguson DP, Lewington MR, Smith TD, Wong IH. Graft utilization in the augmentation of large-to-massive rotator cuff repairs: a systematic review. Am J Sports Med. 2016;44:2984–92. https://doi.org/10.1177/0363546515624463.
20. Le BTN, Wu XL, Lam PH, Murrell GAC. Factors predicting rotator cuff retears. Am J Sports Med. 2014;42:1134–42. https://doi.org/10.1177/0363546514525336.
21. Wu XL, Briggs L, Murrell GAC. Intraoperative determinants of rotator cuff repair integrity. Am J Sports Med. 2012;40:2771–6. https://doi.org/10.1177/0363546512462677.
22. 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.
23. 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.
24. Patte D. Classification of rotator cuff lesions. Clin Orthop Relat Res. 1990;254:81–6.
25. Denard PJ, Koo SS, Murena L, Burkhart SS. Pseudoparalysis: the importance of rotator cable integrity. Orthopedics. 2012;35:e1353–7. https://doi.org/10.3928/01477447-20120822-21.
26. 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.
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. https://doi.org/10.1177/0363546509350081.
28. Gervasi E, Causero A, Parodi PC, Raimondo D, Tancredi G. Arthroscopic Latissimus dorsi transfer. Arthrosc J Arthrosc Relat Surg. 2007;23:1243.e1–4. https://doi.org/10.1016/j.arthro.2006.12.021.
29. Neviaser JS. Ruptures of the rotator cuff of the shoulder. New concepts in the diagnosis and operative treatment of chronic ruptures. Arch Surg. 1971;102:483–5.
30. Nassos JT, Chudik SC. Arthroscopic rotator cuff repair with biceps tendon augmentation. Am J Orthop (Belle Mead NJ). 2009;38:279–81.
31. Derwin KA, Baker AR, Spragg RK, Leigh DR, Farhat W, Iannotti JP. Regional variability, processing methods, and biophysical properties of human fascia lata extracellular matrix. J Biomed Mater Res A. 2008;84A:500–7. https://doi.org/10.1002/jbm.a.31455.
32. 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;88:2665–72. https://doi.org/10.2106/JBJS.E.01307.
33. McCarron JA, Milks RA, Mesiha M, Aurora A, Walker E, Iannotti JP, et al. Reinforced fascia patch limits cyclic gapping of rotator cuff repairs in a human cadaveric model. J Shoulder Elb Surg. 2012;21:1680–6. https://doi.org/10.1016/j.jse.2011.11.039.
34. 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. https://doi.org/10.1016/j.arthro.2013.08.032.
35. Bektaser B. Free coracoacromial ligament graft for augmentation of massive rotator cuff tears treated with mini-open repair. Acta Orthop Traumatol Turc. 2010;44:426–30. https://doi.org/10.3944/AOTT.2010.2423.
36. Mihara S, Ono T, Inoue H, Kisimoto T. A new technique for patch augmentation of rotator cuff repairs. Arthrosc Tech. 2014;3:e367–71. https://doi.org/10.1016/j.eats.2014.02.007.
37. Scheibel M, Brown A, Woertler K, Imhoff AB. Preliminary results after rotator cuff reconstruction augmented with an autologous periosteal flap. Knee Surg Sports Traumatol Arthrosc. 2007;15:305–14. https://doi.org/10.1007/s00167-006-0173-z.
38. Thangarajah T, Pendegrass CJ, Shahbazi S, Lambert S, et al. Augmentation of rotator cuff repair with soft tissue scaffolds. Orthop J Sports Med. 2015;3(6):1–8. https://doi.org/10.1177/2325967115587495.
39. Barber FA, Herbert MA, Ph D, Boothby MH. Ultimate tensile failure loads of a human dermal allograft rotator cuff augmentation. Arthroscopy. 2008;24:20–4. https://doi.org/10.1016/j.arthro.2007.07.013.
40. Mazzocca AD, Trainer G, McCarthy MB, Obopilwe E, Arciero RA. Biologic enhancement of a com-

mon arthroscopic suture. Arthrosc J Arthrosc Relat Surg. 2012;28:390–6. https://doi.org/10.1016/j.arthro.2011.08.306.
41. Wong I, Burns J, Snyder S. Arthroscopic GraftJacket repair of rotator cuff tears. J Shoulder Elb Surg. 2010;19:104–9. https://doi.org/10.1016/j.jse.2009.12.017.
42. Ozaki J, Fujimoto S, Masuhara K, Tamai S, Yoshimoto S. Reconstruction of chronic massive rotator cuff tears with synthetic materials. Clin Orthop Relat Res. 1986;202:173–83.
43. Mckeown ADJ, Beattie RF, Murrell GAC, Lam PH. Biomechanical comparison of expanded polytetrafluoroethylene (ePTFE) and PTFE interpositional patches and direct tendon-to-bone repair for massive rotator cuff tears in an ovine model. Shoulder Elbow. 2016;8:22–31. https://doi.org/10.1177/1758573215601815.
44. 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 Elb Surg. 2010;19:688–96. https://doi.org/10.1016/j.jse.2009.12.008.
45. Proctor CS, Orthopedics A, Barbara S. Long-term successful arthroscopic repair of large and massive rotator cuff tears with a functional and degradable reinforcement device. J Shoulder Elb Surg. 2014;23:1508–13. https://doi.org/10.1016/j.jse.2014.01.010.
46. 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 Elb Surg. 2015;24:915–21. https://doi.org/10.1016/j.jse.2014.09.044.
47. Ciampi P, Scotti C, Nonis A, Vitali M, Di Serio C, Peretti GM, 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;42:1169–75. https://doi.org/10.1177/0363546514525592.
48. Meyer F, Wardale J, Best S, Cameron R, Rushton N, Brooks R. Effects of lactic acid and glycolic acid on human osteoblasts: a way to understand PLGA involvement in PLGA/calcium phosphate composite failure. J Orthop Res. 2012;30:864–71. https://doi.org/10.1002/jor.22019.
49. Fini M, Bondioli E, Castagna A, Torricelli P, Giavaresi G, Rotini R, et al. Decellularized human dermis to treat massive rotator cuff tears: in vitro evaluations. Connect Tissue Res. 2012;53:298–306. https://doi.org/10.3109/03008207.2011.649929.
50. Montgomery SR, Petrigliano FA, Gamradt SC. Biologic augmentation of rotator cuff repair. Curr Rev Musculoskelet Med. 2011;4:221–30. https://doi.org/10.1007/s12178-011-9095-6.
51. 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. https://doi.org/10.2106/JBJS.E.00524.
52. Walton JR, Bowman NK, Khatib Y, Linklater J, Murrell GAC. Restore orthobiologic implant: not recommended for augmentation of rotator cuff repairs. J Bone Joint Surg Am. 2007;89:786–91. https://doi.org/10.2106/JBJS.F.00315.
53. Phipatanakul WP, Petersen SA. Porcine small intestine submucosa xenograft augmentation in repair of massive rotator cuff tears. Am J Orthop (Belle Mead NJ). 2009;38:572–5.
54. Xu H, Wan H, Zuo W, Sun W, Owens RT, Harper JR, et al. A porcine-derived acellular dermal scaffold that supports soft tissue regeneration: removal of terminal galactose-alpha-(1,3)-galactose and retention of matrix structure. Tissue Eng Part A. 2009;15:1807–19. https://doi.org/10.1089/ten.tea.2008.0384.
55. Connor J, McQuillan D, Sandor M, Wan H, Lombardi J, Bachrach N, et al. Retention of structural and biochemical integrity in a biological mesh supports tissue remodeling in a primate abdominal wall model. Regen Med. 2009;4:185–95. https://doi.org/10.2217/17460751.4.2.185.
56. Nicholson GP, Breur GJ, Van Sickle D, Yao JQ, Kim J, Blanchard CR. Evaluation of a cross-linked acellular porcine dermal patch for rotator cuff repair augmentation in an ovine model. J Shoulder Elb Surg. 2007;16:S184–90. https://doi.org/10.1016/j.jse.2007.03.010.
57. Smith RD, Carr A, Dakin SG, Snelling SJ, Yapp C, Hakimi O. The response of tenocytes to commercial scaffolds used for rotator cuff repair. Eur Cell Mater. 2016;31:107–18.
58. 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. https://doi.org/10.1016/j.arthro.2016.01.037.
59. 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 Elb Surg. 2008;17:35S–9S. https://doi.org/10.1016/j.jse.2007.08.005.
60. Giannotti S, Ghilardi M, Dell'osso G, Magistrelli L, Bugelli G, Di Rollo F, 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.
61. Steinhaus ME, Makhni EC, Cole BJ, Romeo AA, Verma NN. Outcomes after patch use in rotator cuff repair. Arthrosc J Arthrosc Relat Surg. 2016;32:1676–90. https://doi.org/10.1016/j.arthro.2016.02.009.
62. Avanzi P. Goal post technique; for arthroscopic biological augmentation of rotator cuff repair; 2017. https://www.vumedi.com/video/goal-post-technique-for-arthroscopic-biological-augmentation-of-rotator-cuff-repair/. Accessed 14 Jan 2017.
63. Beitzel K, McCarthy MB, Cote MP, Russell RP, Apostolakos J, Ramos DM, et al. Properties of biologic scaffolds and their response to mesenchymal stem

cells. Arthrosc J Arthrosc Relat Surg. 2014;30:289–98. https://doi.org/10.1016/j.arthro.2013.11.020.
64. 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.
65. Burkhart SS, Adams CR, Burkhart SS, Schoolfield JD. A biomechanical comparison of 2 techniques of footprint reconstruction for rotator cuff repair: the SwiveLock-FiberChain construct versus standard double-row repair. Arthrosc J Arthrosc Relat Surg. 2009;25:274–81. https://doi.org/10.1016/j.arthro.2008.09.024.
66. Grambart ST. Sports medicine and platelet-rich plasma. Clin Podiatr Med Surg. 2015;32:99–107. https://doi.org/10.1016/j.cpm.2014.09.006.
67. Jo CH, Shin JS, Lee YG, Shin WH, Kim H, Lee SY, et al. Platelet-rich plasma for arthroscopic repair of large to massive rotator cuff tears: a randomized, single-blind, parallel-group trial. Am J Sports Med. 2013;41:2240–8. https://doi.org/10.1177/0363546513497925.
68. Randelli PS, Arrigoni P, Cabitza P, Volpi P, Maffulli N. Autologous platelet rich plasma for arthroscopic rotator cuff repair. A pilot study. Disabil Rehabil. 2008;30:1584–9. https://doi.org/10.1080/09638280801906081.
69. Randelli P, Arrigoni P, Ragone V, Aliprandi A, Cabitza P. Platelet rich plasma in arthroscopic rotator cuff repair: a prospective RCT study, 2-year follow-up. J Shoulder Elb Surg. 2011;20:518–28. https://doi.org/10.1016/j.jse.2011.02.008.
70. Castricini R, Longo UG, De Benedetto M, Panfoli N, Pirani P, Zini R, et al. Platelet-rich plasma augmentation for arthroscopic rotator cuff repair: a randomized controlled trial. Am J Sports Med. 2011;39:258–65. https://doi.org/10.1177/0363546510390780.
71. Barber FA, Hrnack SA, Snyder SJ, Hapa O. Rotator cuff repair healing influenced by platelet-rich plasma construct augmentation. Arthrosc J Arthrosc Relat Surg. 2011;27:1029–35. https://doi.org/10.1016/j.arthro.2011.06.010.
72. Kabuto Y, Morihara T, Sukenari T, Kida Y, Oda R, Arai Y, et al. Stimulation of rotator cuff repair by sustained release of bone morphogenetic Protein-7 using a Gelatin hydrogel sheet. Tissue Eng Part A. 2015;21:2025–33. https://doi.org/10.1089/ten.tea.2014.0541.
73. Lee K, Lee J, Kim Y, Shim Y, Jang J, Lee K. Effective healing of chronic rotator cuff injury using recombinant bone morphogenetic protein-2 coated dermal patch in vivo. J Biomed Mater Res B Appl Biomater. 2016;105:1–7. https://doi.org/10.1002/jbm.b.33716.
74. Omae H, Steinmann SP, Zhao C, Zobitz ME, Wongtriratanachai P, Sperling JW, et al. Biomechanical effect of rotator cuff augmentation with an acellular dermal matrix graft: a cadaver study. Clin Biomech. 2012;27:789–92. https://doi.org/10.1016/j.clinbiomech.2012.05.001.
75. Press D. Biological augmentation of rotator cuff repair using bFGF-loaded electrospun poly (lactide-co-glycolide) fibrous membranes. Int J Nanomedicine. 2014;9:2373–85.
76. Gulotta LV, Kovacevic D, Ehteshami JR, Dagher E, Packer JD, Rodeo SA. Application of bone marrow-derived mesenchymal stem cells in a rotator cuff repair model. Am J Sports Med. 2009;37:2126–33. https://doi.org/10.1177/0363546509339582.
77. Yoon JP, Chung SW, Kim JY, Lee BJ, Kim H-S, Kim JE, et al. Outcomes of combined bone marrow stimulation and patch augmentation for massive rotator cuff tears. Am J Sports Med. 2016;44:963–71. https://doi.org/10.1177/0363546515625044.
78. Guilak F, Estes BT, Diekman BO, Moutos FT, Gimble JM. 2010 Nicolas Andry award: multipotent adult stem cells from adipose tissue for musculoskeletal tissue engineering. Clin Orthop Relat Res. 2010;468:2530–40. https://doi.org/10.1007/s11999-010-1410-9.
79. Gimble JM, Guilak F. Adipose-derived adult stem cells: isolation, characterization, and differentiation potential. Cytotherapy. 2003;5:362–9. https://doi.org/10.1080/14653240310003026.
80. 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 Elb Surg. 2014;23:445–55. https://doi.org/10.1016/j.jse.2013.07.054.
81. Gupta AK, Hug K, Boggess B, Gavigan M, Toth AP. Massive or 2-tendon rotator cuff tears in active patients with minimal glenohumeral arthritis. Am J Sports Med. 2013;41:872–9. https://doi.org/10.1177/0363546512475204.
82. Gupta A, Liberati TA, Verhulst SJ, Main BJ, Roberts MH, Potty AGR, et al. Biocompatibility of single-walled carbon nanotube composites for bone regeneration. Bone Joint Res. 2015;4:70–7. https://doi.org/10.1302/2046-3758.45.2000382.
83. 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. Arthrosc J Arthrosc Relat Surg. 2013;29:459–70. https://doi.org/10.1016/j.arthro.2012.10.022.
84. 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.

How to Manage Failed Rotator Cuff Repair: Latissimus Dorsi Transfer

30

Enrico Gervasi, Enrico Sebastiani, and Enrico Cautero

30.1 Introduction

The "evidence based" medicine and the attention to the analysis of the results demonstrate the ineffectiveness of techniques based on direct repair of the rotator cuff documenting the failures or the recurrences in many massive injuries, even when at the time of surgery the tendons appear firmly repaired to the bone. The role of biology in the healing process is getting to be understood; the functional role of each motor unit throughout the kinetic chain should be analyzed. The goal of the surgery is aimed at restoring the function rather than to repair the injured tissues. The search for effective alternatives to direct repair passes through synthetic tissue as augmentation [1] or subacromial spacer [2] and leads to consider the muscle-tendon transfers. The latter were used in the past to treat neurological injuries, such as obstetrical palsy. The L'Episcopo procedure was directed to children with residual or neglected deformity. The concept supported by L'Episcopo was that, besides release of contracted soft tissues, if the action of one or more of the internal rotators could be reversed, deformity-producing factor could be replaced by a deformity-correcting factor. This operation was performed on the 14th of July 1931. As by the original article, published on July 1934 on *American Journal of Surgery* [3], the transferred tendon was the teres major (TM), not the latissimus dorsi (LD). The idea of rerouting an internal rotator muscle-tendon unit to act as external rotator remains the original contribution of this author.

Gerber gave rise to the modern science of tendon transfers for rotator cuff deficiency. He described the procedure as providing "a large, vascularized tendon…to close a massive cuff defect and that exerts an external rotation (ER) and head depressing moment to allow more effective action of the deltoid muscle" [4]. The pathophysiological concept was to counteract the major consequences of the supraspinatus (SS) and infraspinatus (IS) insufficiency. In this scenario, the weakened fulcrum for deltoid function gets the humeral head to migrate upward instead of rotating, problem magnified by the tear of the long head of biceps (LHB) or the subscapularis (SSC); hence, the active ER is lost. He saw the analogies with the suprascapular nerve palsy: the massive cuff tears causing to adults a similar problem to that of young patients affected by birth palsy. The deltoid function is stated to be crucial. Although the muscle power of the TM is about three times that of the LD, he preferred to transfer the latter one, since the TM can be too bulky to pass and glide between deltoid and the teres minor (Tm) and because its short length is an obstacle to fix it properly at the most anterior aspect of the greater

E. Gervasi · E. Sebastiani (✉) · E. Cautero
Department of Orthopaedics and Traumatology, Ospedale Civile di Latisana, Latisana, UD, Italy

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_30

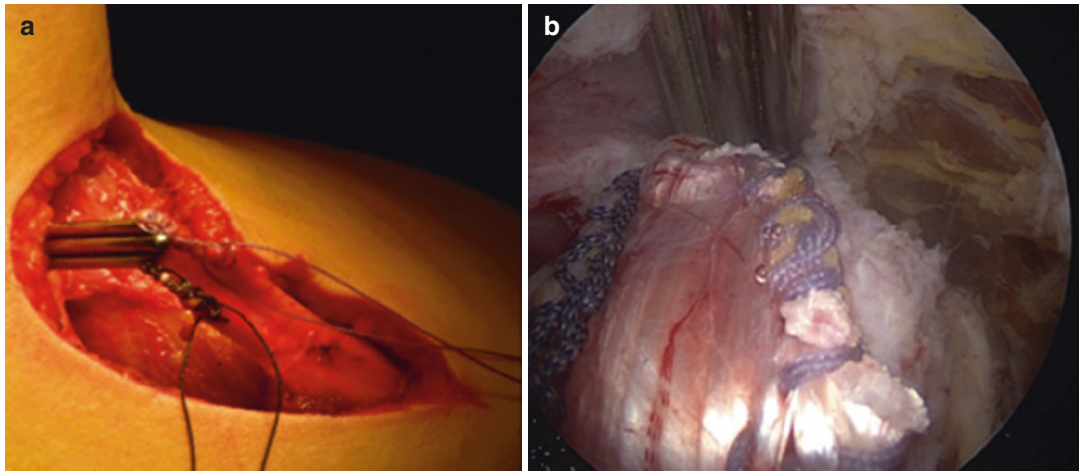

Fig. 30.1 (**a**) Mini-open muscular release and tendon detachment. (**b**) Endoscopic transfer and fixation of latissimus dorsi

tuberosity. The technique described by Gerber has been the gold standard for many years. The downside is the need of detachment of a large part of the deltoid insertion from the acromion to retrieve the graft to the subacromial space.

The next shift to the latissimus dorsi transfer (LDT) is the arthroscopic technique first performed on August 2003 and published in 2007 by Gervasi [5]. The objective of the all-endoscopic technique was to preserve the deltoid integrity. Since the endoscopic graft harvesting was time consuming and not beneficial, the author shifted to a combined technique: mini-open muscular release and tendon detachment and endoscopic transfer and fixation (Fig. 30.1). Further works by the same author proposed a classification of the cuff lesions amenable for this transfer [6, 7]. The deltoid-sparing approach also guarantees a way out in case of failures, when the pathology rises toward a cuff tear arthropathy and the joint replacement with a reverse prosthesis becomes necessary. Other technical variants have been described [8].

30.2 Latissimus Dorsi Transfer

The tendon-muscle transfer is performed to replace a motor unit that is no longer working with another that has similar biomechanical characteristics. The ideal transfer has range of motion (ROM), strength, and line of action similar to the structure whose forces have to be replaced. However, these features cannot be always reproduced in shoulder muscles, because of the complex polyaxial anatomy and biomechanics of the joint and surrounding muscles.

Pectoralis major (PM) and LD play an important role in stabilizing the humeral head in posterior-superior rotator cuff tears [9]. The LDT could lead to imbalance of this compensatory mechanism. Some authors suggest using another muscle, out of this pair of forces, as the lower trapezius, thus leaving the LD intact in order to preserve its centering action on the humeral head [10]. Plastic surgeons first studied if LD removal leads to any abnormality in the shoulder biomechanics. This muscle is used as a free flap for reconstruction, especially for breast reconstruction. In 1985, Laitung and Peck [11] measured that the strength in adduction was not modified by the removal of LD in 19 patients. However, subsequent studies showed reduction of shoulder function after removal of the LD [12–14]. Spear and Hess [15] showed that there is not a real reduction in force, but patients perceived easily fatigued after

prolonged activity in flexion and adduction as swimming, ladder climbing, or painting above the head.

30.3 Operative Technique

The patient is placed in lateral decubitus position. Dorsal tilt is not recommended in order to avoid posterior deltoid collapse and to facilitate the passage of the LD.

Usually we start with arthroscopy. We prepare the subacromial space and the remaining cuff is tensioned as possible. Capsular release is crucial to let the humeral head free to be centered into the glenoid. Coracohumeral ligament (CHL) is detached from its coracoid insertion and the capsule is released all around the glenoid neck. The space between Tm and deltoid is created. We must work inside the fascia in order to avoid axillary nerve injury. The LDT should pass posterior to the axillary nerve or anterior to it (very close to the humerus and Tm). The arm is then abducted and elbow is flexed at 90°. A 6-cm incision is made at the level of the posterior axillary pillar.

The LD muscle and its tendinous insertion on the proximal humeral shaft are identified (Fig. 30.2). The radial nerve cannot be directly visualized because it lies within fat tissue, approximately 2 cm distal to the LD humeral attachment, according to the arm position. The humerus is then placed in maximal internal rotation and LD tendon is carefully detached from the bone. The LD tendon is reinforced by continuous interlocking suture and the muscle is mobilized. We suggest performing an accurate and complete detachment of the muscle belly, to get the LD elastic enough to bring it to the upper portion of the greater tuberosity. Particular attention should be placed in interrupting the connections with the subcutaneous tissue, adhesions with the fascia, the dense fibrous bands that connect the LD tendon to the TM and to the triceps brachii [16], the connections with the serratus anterior, and, if necessary, the insertion with the inferior corner of the scapula. The tendon is shuttled into the subacromial space, and under arthroscopic view two or more anchors are used to fix it to the greater tuberosity.

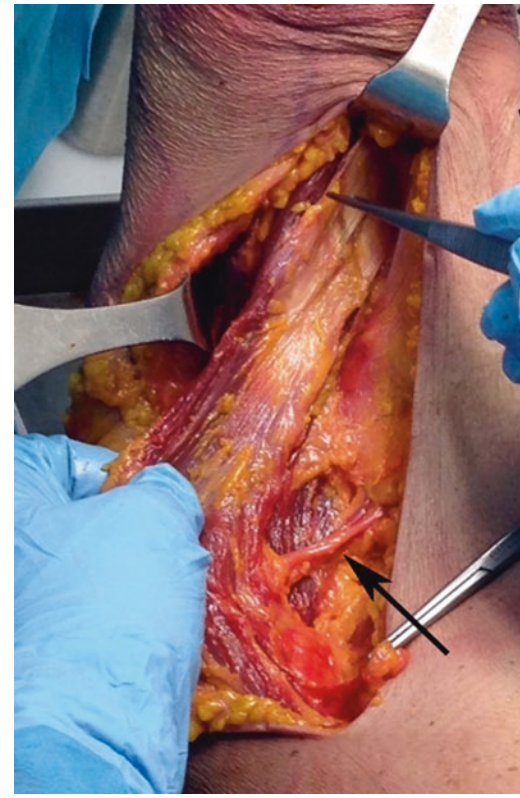

Fig. 30.2 Cadaver dissection of the tendon of latissimus dorsi (forceps) and its neurovascular pedicle (black arrow)

30.4 Literature Review

Many studies have been published about LDT in irreparable cuff tears. Most of these studies are not comparable as they dealt with different patient characteristics, surgical techniques, and outcome measures [17].

Longo et al. [18] reviewed all articles reporting outcomes on shoulders treated with LDT, performed singularly or in addition with other procedures. Eighteen studies (a total of 277 shoulders) reported outcome data of salvage surgeries always made with an open approach, based on the Gerber technique. Some series, not the most recent ones, did not find differences in objective and subjective results between patients operated on in a primary procedure and patients operated on in a revision procedure, most of the

recent published series found that the latter had lower objective and subjective results.

Miniaci and MacLeod [19] reported 82% of patients satisfied in revision surgery, based on UCLA score. For their 18 patients operated for a salvage procedure, Birmingham and Neviaser [20] showed a better active forward elevation, active ER at the side, ASES score, and pain relief at a minimum 12-month follow-up. Pearsall et al. [21] operated seven patients for revision procedure and found modest improvement of function. Debeer and De Smet [22] found no statistical difference between primary and revision surgery, although their postoperative scores were in favor of primary patients. However, they concluded that this lack of difference might be linked to the fact that most of the previous surgeries in revision procedures were arthroscopic with no deltoid damage. Weening and Willems [23], in a series with 16 patients out of whom 9 underwent a revision procedure, found no difference between primary and revision procedures.

All the other series showed lower subjective and objective results in patients operated for revision procedure. Aoki et al. [24] had two failures in their series of 12 patients for the two patients who were operated for a revision procedure. Warner and Parsons [25] had significant lower improvement in Constant score (CS), due mainly to a less significant forward flexion improvement, whereas active ER and strength were not statistically different between revision and primary patients. Gerber et al. [26] found significant lower results for revision patients with postoperative 59% CS weighed score compared with 79% for primary patients; however, they noticed that increase of subjective shoulder value (SSV) was comparable in revision and in primary patients. Nové-Josserand et al. [27] found significantly lower results in revision patients (mean postoperative CS and SSV, 67% and 43%, respectively) than in primary patients (CS and SSV, 75 and SSV 71%, respectively). Valenti et al. [28] found significantly lower results in revision patients than in primary patients (mean postoperative CS: 54 and 61, respectively). Irlenbusch et al. [29] found poorer results in revision patients, albeit they included in their primary cases patients who had been already operated on with an isolated acromioplasty.

As a conclusion, studies that analyze the use of LDT as salvage procedure using an open approach found less favorable results for patients with anterior deltoid damage due to detachment and/or atrophy of the muscle compared to less aggressive previous surgeries, such as arthroscopic debridement, acromioplasty, or LHB tenotomy.

Only a few studies reported on arthroscopic LDT. Paribelli et al. [30] compared a group of 20 patients treated with arthroscopic transfer according to the Gervasi's technique with a group of 20 patients treated with a partial rotator cuff repair. The study did not distinguish primary from revision procedures. Grimberg et al. [31] reviewed 57 arthroscopic LDTs, 30 of them being revision procedures. The authors fixed the tubularized tendon of LD into a humeral bone tunnel with knotted sutures over the button to the anterior humeral cortex. Results showed an overall improvement of the CS from 37 to 65.4, but lower postoperative CS results were detected in patients who had previous shoulder surgery. Castricini et al. [32] reported on 86 patients treated with the Gervasi's technique, 14 of whom (16.3%) sustained an irreparable massive rotator cuff tear after a failed arthroscopic rotator cuff repair. At a mean follow-up of 36.4 months, the authors showed that salvage procedure was associated with less strength in forward flexion, lower postoperative CS, and a decrease of internal rotation compared with primary surgery. However, the authors stated that statistically significant difference was probably not clinically relevant, and their findings do not limit the use of LDT to primary surgery. Kanatl et al. [33] reported on 15 patients with pseudoparalysis treated with the Gervasi's technique and routinely reinforcement of the tendon with fascia lata. Four of them were revision surgeries and showed the same results of the ones with primary surgery.

30.5 LDT in Anterosuperior Rotator Cuff Tears

LDT may be utilized for the treatment of anterosuperior rotator cuff tears. Different techniques and muscle transfers are used in case of anterior cuff insufficiency, as in the failed SSC repair. The goal is restoring the strength of the anterior

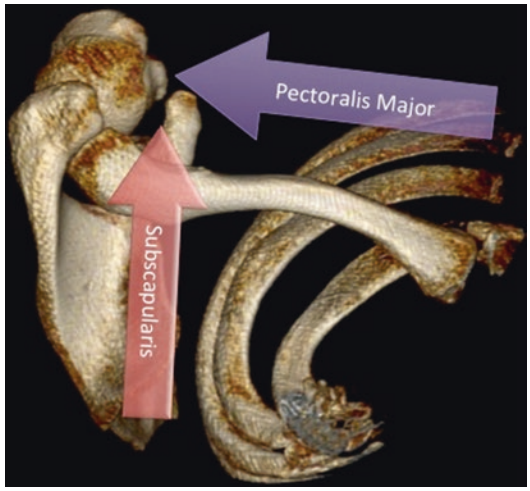

Fig. 30.3 Pectoralis major (blue arrow) comes from anterior wall of rib cage; its direction is perpendicular to the direction of subscapularis (red arrow)

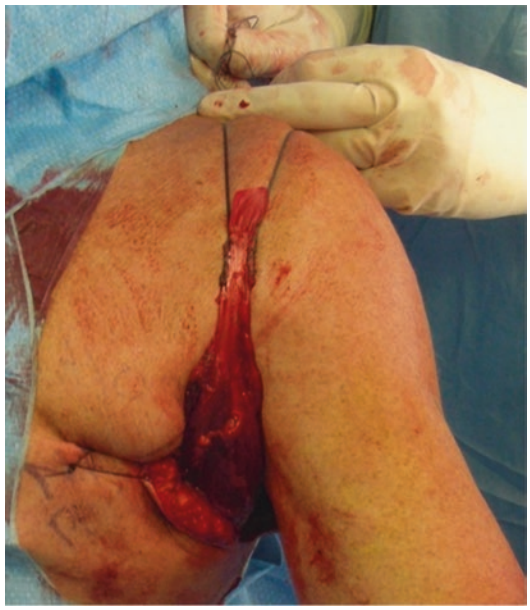

Fig. 30.4 Latissimus dorsi transfer for the treatment of subscapularis tear

compartment, thus obtaining the humeral balancing. The most used technique is the PM transfer [34, 35]; the least used is the transfer of the pectoralis minor (Pm). Recently, the transfer of the upper trapezius has been investigated with poor results [36]. The PM and Pm come from the front wall of the rib cage, while the SSC tendon originates posteriorly. This means that the force vector of the PM forms a wide angle with the SSC. The force vector of the muscle transferred is, therefore, very different from the one of the muscle that must be replaced (Fig. 30.3). To overcome this problem, the transfer of a muscle that originates from the back of the trunk wall (LD) is proposed.

Elhassan et al. [37] described the LDT for irreparable lesions of the SSC (Fig. 30.4). This anatomical study showed that the risk of nerve compression (axillary, radial, and musculocutaneous nerves) is very low. Although this technique is promising, we need additional ex vivo and in vivo studies to prove its effectiveness.

Conclusion

On the basis of the currently available data, we cannot still really understand if LDT has worse clinical results in case of revision surgery. Indeed, all patients who underwent LDT improved in clinical and functional scores compared to preoperative condition. LDT might be a viable option in case of massive posterosuperior lesion of the rotator cuff with muscle atrophy even in revision cases.

References

1. Cooper J, Mirzayan R. Acellular dermal matrix in rotator cuff surgery. Am J Orthop. 2016;45(5):301–5.
2. 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:S19–24.
3. L'Episcopo JB. Tendon transplantation in obstetrical paralysis. Am J Surg. 1934;25(1):122–5.
4. 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.
5. Gervasi E, Causero A, Parodi PC, Raimondo D, Tancredi G. Arthroscopic latissimus dorsi transfer. Arthroscopy. 2007;23(11):1243.e1–4.
6. Gervasi E, Spicuzza A. Arthroscopically assisted latissimus dorsi transfer. Shoulder arthroscopy: principles and practice. London: Springer; 2013. p. 503–14.
7. Castricini R, De Benedetto M, Orlando N, Gervasi E, Castagna A. Irreparable rotator cuff tears: a novel classification system. Musculoskelet Surg. 2014;98(Suppl 1):49–53. https://doi.org/10.1007/s12306-014-0320-5.

8. Jermolajevas V, Kordasiewicz B. Arthroscopically assisted latissimus dorsi tendon transfer in beach-chair position. Arthrosc Tech. 2015;4(4):e359–63.
9. Campbell ST, Acklund 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.
10. 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 Elbow Surg. 2015;24(10):1635–43.
11. Laitung JK, Peck F. Shoulder function following the loss of the latissimus dorsi muscle. Br J Plast Surg. 1985;38:375.
12. Brumback RJ, McBride MS, Ortolani NC. Functional evaluation of the shoulder after transfer of the vascularized latissimus dorsi muscle. J Bone Joint Surg Am. 1992;74:377.
13. Fraulin FOG, Louie G, Zorrilla L, Tilley W. Functional evaluation of the shoulder following latissimus dorsi muscle transfer. Ann Plast Surg. 1995;35:349.
14. Clough KB, Louis-Sylvestre C, Fitussi A, Couturaud B, Nos C. Donor-site sequelae after autologous breast reconstruction with an extended latissimus dorsi flap. Plast Reconstr Surg. 2002;109:1904.
15. Spear SL, Hess CL. A review of the biomechanical and functional changes in the shoulder following transfer of the latissimus dorsi muscle. Plast Reconstr Surg. 2005;115:2070–3.
16. Morelli M, Nagamori J, Gilbart M, Miniaci A. Latissimus dorsi tendon transfer for massive irreparable cuff tears: an anatomic study. J Shoulder Elbow Surg. 2008;17:139–43.
17. 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(1):22–32. https://doi.org/10.1007/s12178-013-9196-5.
18. Longo UG, Franceschetti E, Petrillo S, Maffulli N, Denaro V. Latissimus dorsi tendon transfer for massive irreparable rotator cuff tears. A systematic review. Sports Med Arthrosc Rev. 2011;19:428–37.
19. 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.
20. Birmingham PM, Neviaser RJ. Outcome of latissimus dorsi transfer as a salvage procedure for failed rotator cuff repair with loss of elevation. J Shoulder Elbow Surg. 2008;17:871–4.
21. 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.
22. Debeer P, De Smet L. Outcome of latissimus dorsi transfer for irreparable rotator cuff tears. Acta Orthop Belg. 2010;76:449–55.
23. Weening AA, Willems WJ. Latissimus dorsi transfer for treatment of irreparable rotator cuff tears. Int Orthop. 2010;34:1239–44.
24. Aoki M, Okamura K, Fukushima S, et al. Transfer of latissimus dorsi for irreparable rotator-cuff tears. J Bone Joint Surg Br. 1996;78:761–6.
25. Warner JJ, Parsons IMT. 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.
26. 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;95:1920–6.
27. Nove-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.
28. Valenti P, Kalouche I, Diaz LC, et al. Results of latissimus dorsi tendon transfer in primary or salvage reconstruction of irreparable rotator cuff tears. Orthop Traumatol Surg Res. 2010;96:133–8.
29. Irlenbusch U, Bracht M, Gansen HK, Lorenz U, Thiel J. Latissimus dorsi transfer for irreparable rotator cuff tears: a longitudinal study. J Shoulder Elbow Surg. 2008;17:527–34.
30. Paribelli G, Boschi S, Randelli P, Compagnoni R, Leonardi F, Cassarino AM. Clinical outcome of latissimus dorsi tendon transfer and partial cuff repair in irreparable postero-superior rotator cuff tear. Musculoskelet Surg. 2015;99:127–32.
31. Grimberg J, Kany J, Valenti P, Amaravathi R, Ramalingam AT. Arthroscopic-assisted latissimus dorsi tendon transfer for irreparable posterosuperior cuff tears. Arthroscopy. 2015;31:599–607.
32. Castricini R, Longo UG, De Benedetto M, Loppini M, Zini R, Maffulli N, Denaro V. 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.
33. Kanatl U, Ozer M, Ataoglu MB, Ozturk BY, Hui O, Cetinkaya M, Ayanoglu T. Arthroscopic-assisted latissimus dorsi tendon transfer for massive irreparable rotator cuff tears: technique and short-term follow-up of patients with pseudoparalysis. Arthroscopy. 2016;33(5):929–37. https://doi.org/10.1016/j.arthro.2016.09.023.
34. Resch H, Povacz P, Ritter E, Matschi W. Transfer of pectoralis major muscle for the treatment of irreparable rupture of the subscapularis tendon. J Bone Joint Surg. 2000;82:372–82.
35. Hackl W, Wambacher M, Kralinger F, Smekal V. Pectoralis major in the treatment of chronic subscapularis insufficiency. Oper Orthop Traumatol. 2007;19:433–41.
36. Goutallier D, De Abreu L, Postel JM, Le Giulloux P, Radier C, Zilber S. Is the trapezius transfer a useful treatment option for irreparable tears of the subscapularis? Orthop Traumatol Surg Res. 2011;97:719–25.
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 Elbow Surg. 2014;23:492–9.

Failed Rotator Cuff Repair: Case Example 1

31

Taner Gunes, Umut Akgun, and Recep Kurnaz

31.1 Introduction

Recurrent rotator cuff tears can be frequently seen after primary repair, albeit most of the patients are asymptomatic. However, in active and young populations, symptoms might impair the quality of life. Main causes for repair failure are failure of fixation of the primary repair, biological failure due to poor soft tissue quality (fatty degeneration and atrophy of rotator cuff muscles), or inadequate and/or aggressive postoperative rehabilitation [1–3]. The following case is a rotator cuff repair that failed due to multiple factors.

31.2 Case Presentation

A 52-year-old male, heavy smoker, came to our clinic 6 months after a mini-open rotator cuff repair and acromioplasty procedure that he received in another hospital.

T. Gunes (✉)
Department of Orthopaedics, Acibadem University, Istanbul, Turkey

Acibadem Eskisehir Hospital, Eskisehir, Turkey

U. Akgun
Department of Orthopaedics, Acibadem University, Istanbul, Turkey

R. Kurnaz
Department of Orthopaedics, Acibadem Eskisehir Hospital, Eskisehir, Turkey

The patient was immobilized in a sling for 2 weeks, and then a rehabilitation program consisting of active range of motion (ROM) and resistive exercises was initiated. The patient did not stop smoking. In the 6th postoperative week, patient started complaining about pain and snapping sensation in his shoulder. As the pain increased, ROM started to decline. Although the rehabilitation program was stopped in the 8th week, patient continued to complain about pain during daily activities and at night.

Six months after surgery, the patient complained about mechanical symptoms like catching, snapping, and aggravation of pain, which suggested a clinical suspicion of failed rotator cuff repair. Physical examination revealed an anterolateral surgical scar. Forward flexion, external rotation, and internal rotation were 150, 20°, and L5 vertebrae, respectively. Jobe, Neer, and O'Brien provocative tests were all positive. During active elevation, pain and crepitus started at 90° of abduction. Local signs of infection such as edema and hyperemia were absent; white blood cell (WBC) count, erythrocyte sedimentation rate (ESR), and c-reactive protein (CRP) blood level were normal. Magnetic resonance imaging (MRI) showed one metallic anchor at the level of greater tuberosity and failed repair of the supraspinatus tendon at the level of anchor (Fig. 31.1). Arthroscopic revision surgery was planned. The patient was invited to stop smoking 3 weeks prior to surgery.

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_31

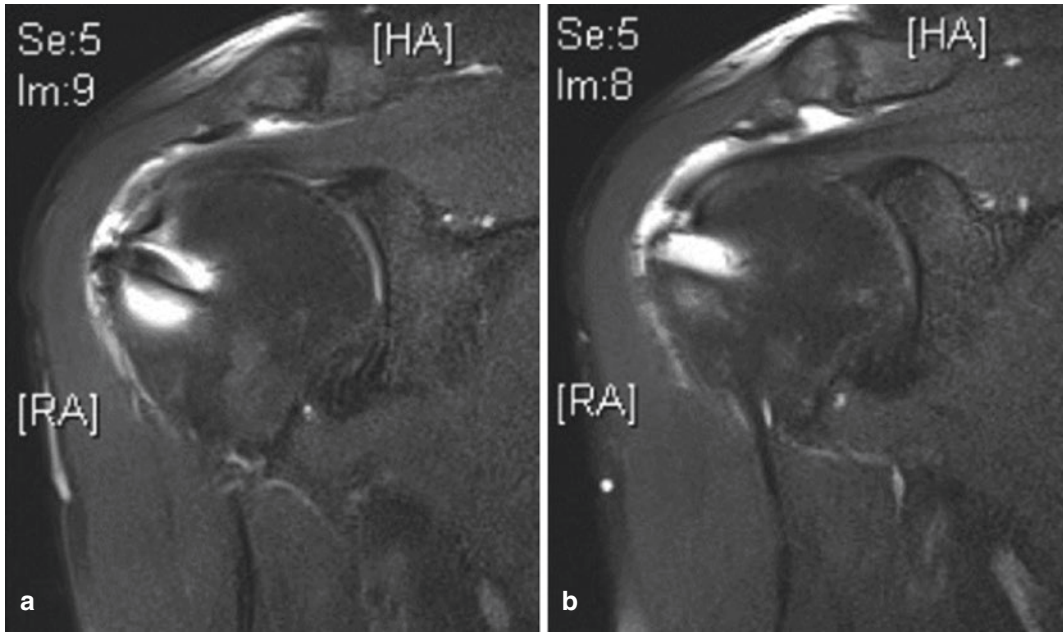

Fig. 31.1 (**a**, **b**) MR images (coronal views) 6 months after primary repair; a clear space between the anchor and the lateral border of the tendon is visible

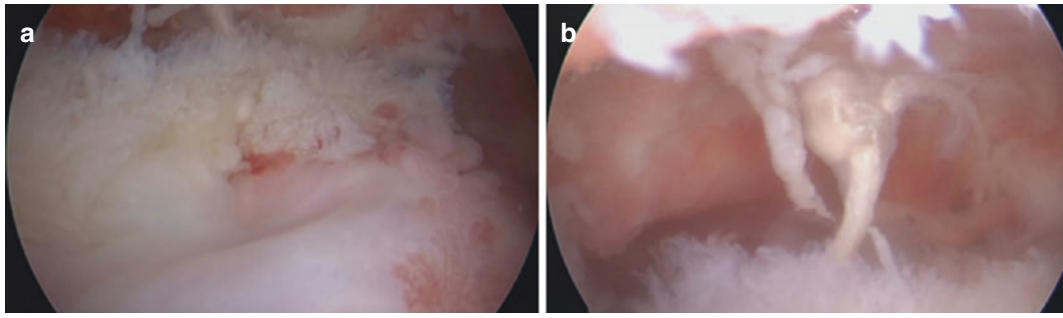

Fig. 31.2 (**a**) Defect in the supraspinatus tendon; (**b**) fraying in the tendon and free suture material

31.2.1 Surgical Treatment

Standard posterior, anterior, and lateral portals were used on the right shoulder, while the patient was in lateral decubitus position. No intra-articular pathology was detected. In the subacromial space, previous knots were failed, and worn suture material was floating in bursa. Full-thickness supraspinatus tendon rupture and fraying was evident (Fig. 31.2). Previous acromioplasty was thought to be inadequate. All suture materials and anchors were taken out, tendon edges were debrided, and additional release of the subacromial space was performed (Fig. 31.3). Bony surface of the greater tuberosity was prepared for tendon refixation (Fig. 31.4). Tendon was repaired with two 5.5-mm metal suture anchors (Corkscrew II; Arthrex, Naples,

FL, USA) by using modified Mason-Allen technique (Fig. 31.5). Acromioplasty was revised after tendon repair. An arm sling with shoulder abduction pillow was used for 4 weeks postoperatively. During this period, patient was allowed to make pendulum exercises and to write or use keyboard. After 4 weeks, sling was taken out, and rehabilitation including passive ROM exercises was started. In the 8th week, active-assistive exercises were started. In the 12th week, resistive exercises were started. Patient was not allowed to smoke for 3 months.

At the 6-month follow-up, patient's shoulder was pain free with full range of motion compared to the other side. In his control MRI, structural integrity of bone-tendon junction with type 1 healing according to Sugaya's classification [4] was seen (Fig. 31.6).

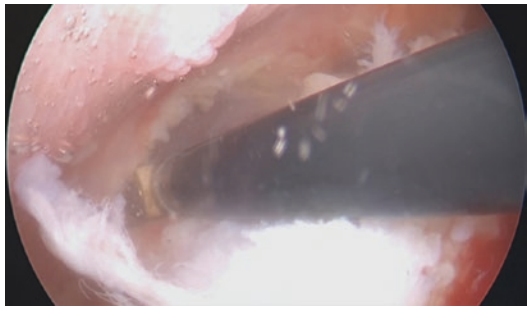

Fig. 31.3 Arthroscopic release of the subacromial space

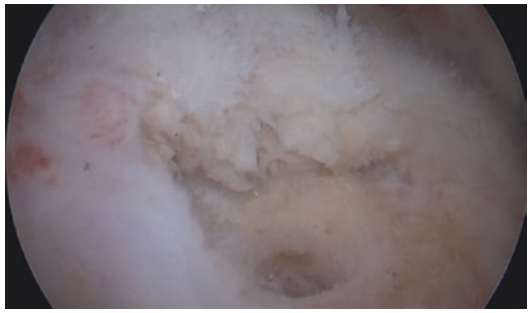

Fig. 31.4 Preparation of the greater tuberosity before fixation

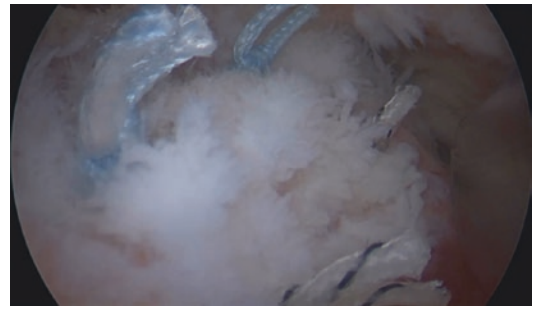

Fig. 31.5 Tendon repair with modified Mason-Allen technique

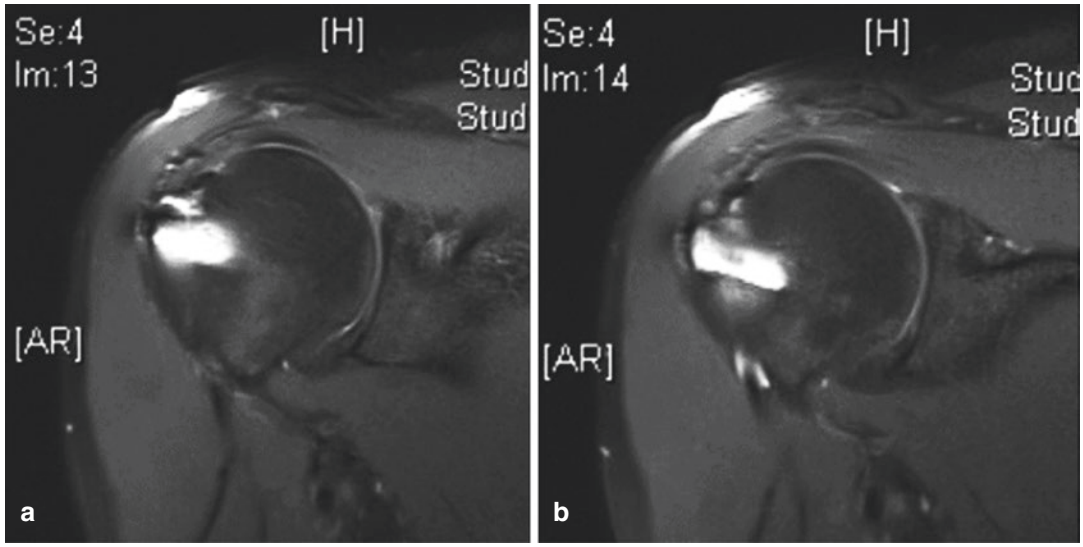

Fig. 31.6 (**a**, **b**) MR images (coronal views) 6 months after revision surgery

31.3 Discussion

This is a simple case for a revision rotator cuff surgery, but a good example for understanding the reason for failure and patient selection. We can classify the reasons for the failure in this relatively small cuff tear case as:

1. Failure of anchor, inadequate anchor number, and/or inadequate fixation (knot security). We can agree that number of anchors used for the primary fixation is not adequate.
2. Tendon biology is one of the important factors. Besides, smoking is the only negative factor for this patient that could affect the tendon biology.
3. Postoperative rehabilitation is another important factor. Early rehabilitation with inadequate fixation can result in early failure.

Indications for revision surgery are more or less the same for primary surgery; but it is not easy for early failures, because it is hard to make a radiological diagnosis during the early postoperative phase. Clinical symptoms like catching and snapping with increase in pain and inappropriate rehabilitation history are main criteria for the diagnosis. On the other side, infection must be ruled out; if there is a doubt, biopsy and cultures must be taken, even in cases with normal WBC count, ESR, and CRP level.

Although it is not clear if acromioplasty in addition to rotator cuff repair has a positive effect on the functional outcomes, it is mandatory to perform acromioplasty in revision cases.

Patients with ongoing symptoms after repair must be carefully assessed to diagnose re-tear and to understand causes of failure. This is crucial to achieve a successful revision procedure. During the revision procedure, each factor leading to failure must be identified, and the procedure must be performed accordingly. Especially in young patients, early diagnosis of re-rupture and immediate revision surgery can lead to biologically good tendon integrity.

Revision surgery should be carefully planned and is important to assess preoperatively if the cuff is repairable or not. Tendon retraction, fatty infiltration, muscle atrophy, and degenerative joint changes are main factors affecting reparability [5]. In addition, other factors that may cause pain should be evaluated.

Technical issues during revision surgery are almost similar to primary surgery. The most important factor is to achieve a stable, tension-free tendon fixation on the footprint area. For this reason, a good subacromial space exposure is needed. Subacromial space, rotator cuff tendons, and biceps tendon should be carefully evaluated. In case of biceps tendon pathology, tenotomy and tenodesis are the main surgical options. In case of subscapularis tendon tear, it should be repaired first [6]. Superior and inferior release of the torn tendons will decrease repair tension. If needed, interval slides can be added before fixation of the tendon to the footprint area. Careful and slow postoperative rehabilitation is another key factor after revision surgery.

References

1. 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.
2. Clement ND, Hallett A, MacDonald D, Howie C, McBirnie J. Does diabetes affect outcome after arthroscopic repair of the rotator cuff? J Bone Joint Surg Br. 2010;92:1112–7.
3. 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.
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;21:1307–16.
5. Kowalsky MS, Keener JD. Revision arthroscopic rotator cuff repair: repair integrity and clinical outcome: surgical technique. J Bone Joint Surg Am. 2011;93(Suppl 1):62–74.
6. Ladermann A, Denard PJ, Burkhart SS. Revision arthroscopic rotator cuff repair: systematic review and authors' preferred surgical technique. Arthroscopy. 2012;28:1160–9.

Failed Rotator Cuff Repair: Case Example 2

32

Maristella F. Saccomanno and Giuseppe Milano

32.1 Introduction

Excellent clinical outcomes have been reported after open or arthroscopic rotator cuff repair [1]. However, recurrent tears remain the most common complication and, therefore, a major clinical problem. Prognostic studies showed that older age and large or massive tears mainly affect the re-tear risk [2]. Surgical factors, such as technique, tension on the repair, fixation, as well as postoperative rehabilitation, could also affect rotator cuff healing [3, 4].

Management of failed rotator cuff repair remains a challenge. Different surgical approaches have been reported in the literature including open or arthroscopic revision surgery with complete or partial repair, various tendon transfers, and patch augmentations by using human or porcine grafts with promising results [5–7]. The best treatment strategy has not been defined yet, since literature data are limited and often of low methodological quality. This is a case report of a patient who underwent two revision surgeries.

32.2 Case Presentation

A 56-year-old woman was referred to the senior author (G.M.) due to persistent pain in her right shoulder. She was a manual worker with right hand dominance. No comorbidities or tobacco use was reported. She has already undergone an open cuff repair and an arthroscopic revision 2 years and 6 months earlier, respectively. Symptoms started after a direct trauma on her right shoulder 6 months before the first surgery due to a fall while skiing. Based on the available documentation, the diagnosis was first made on clinical symptoms and an ultrasound scan. According to the description of the surgical procedure, a full-thickness supraspinatus tear was found and repaired by transosseous suture in an open fashion. After surgery, the arm was immobilized in a sling for 4 weeks. Rehabilitation started after sling removal and lasted for 8 weeks.

After 1 year and half, the patient referred an indirect trauma to the right shoulder. A magnetic resonance imaging (MRI) showed failure of previous surgery, although the supraspinatus tendon was not retracted (Fig. 32.1). Therefore, the patient underwent a revision arthroscopic surgery. According to the surgical report, intraoperative findings confirmed the supraspinatus tendon re-tear retracted to the humeral head. Cuff repair was performed using a biodegradable anchor. A long head of the biceps tenotomy was also performed. After surgery, the patient's arm was

M. F. Saccomanno (✉) · G. Milano
Department of Orthopaedics, Catholic University, Shoulder Service, A. Gemelli University Hospital, Rome, Italy
e-mail: giuseppe.milano@unicatt.it

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9_32

immobilized in a sling for 3 weeks, but passive motion was allowed 1 week after surgery. Rehabilitation protocol lasted for 8 weeks with no complete resolution of shoulder pain.

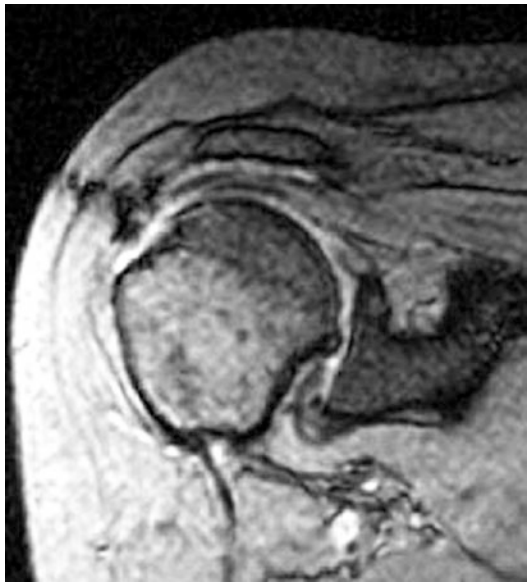

Fig. 32.1 Right shoulder. Postoperative MRI after first surgery (coronal view). The supraspinatus tendon was re-torn, but not retracted

Six months after the second surgery, the patient came to the senior author. She was in good health overall, but complained of persistent pain and weaknesses in her right shoulder. At the clinical examination, she showed full passive and active range of motion (ROM). Specific tests for posterosuperior cuff evaluation (Neer, Hawkins, Yocum, and Jobe tests) were positive for pain with no strength deficit. Clinical evaluation of subscapularis tendon (belly press, lift off, and bear hug tests) was negative.

A MRI revealed the failure of previous repair with osteolysis around the anchor, posterosuperior cuff tendon retraction, some muscle atrophy, but no signs of fatty infiltration according to the Goutallier's classification [8] (Fig. 32.2).

The patient was actually referred to the senior author for a latissimus dorsi transfer. However, based on patient's age, clinical evaluation and functional demand, an arthroscopic re-revision was attempted.

Under general anesthesia and in beach chair position, standard arthroscopic portals were performed (posterior, lateral, and anterosuperior). The diagnostic evaluation revealed an intact subscapularis tendon, a full-thickness V-shaped tear

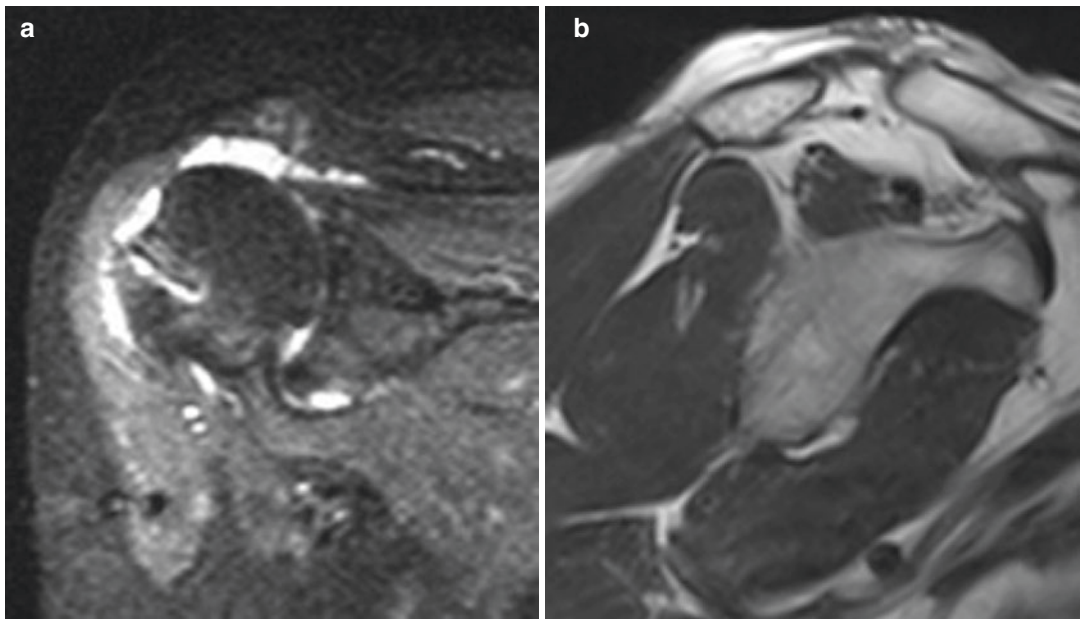

Fig. 32.2 Right shoulder. Postoperative MRI after second surgery. (**a**) Coronal view: the supraspinatus tendon is re-torn and retracted to the glenoid. Osteolysis around the anchor is visible. (**b**) Sagittal view: no signs of fatty infiltration

of the supraspinatus and infraspinatus tendons with atrophic tendon margins (Fig. 32.3). A fibrotic bursitis was also found. Moreover, the osteolysis around the anchor was confirmed (Fig. 32.4), and it was clear that the re-tear was basically due to the anchor failure since the sutures were still into the cuff (Fig. 32.5). Therefore, after a careful bursectomy by using radiofrequency, the anchor was removed and the cuff was completely repaired by performing three side-to-side sutures with three nonabsorbable sutures and two metal anchors preloaded with two nonabsorbable sutures, respecting the direction of the force vectors. Two additional superior lateral portals were performed for allowing anchors placement and cannulas were used for sutures management. Nanofractures of greater tuberosity were performed to enhance bone-tendon healing (Fig. 32.6). Moreover, taking into consideration that it was a second revision surgery and that tendon quality was not excellent, a cuff augmentation was also performed with a porcine dermis patch graft. The graft was fixed medially with two nonabsorbable sutures of different colors and laterally by using two knotless polyetheretherketone

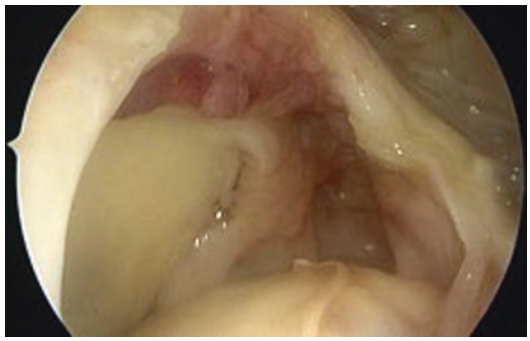

Fig. 32.3 Right shoulder. Arthroscopic re-revision. View from the lateral portal shows a V-shaped rotator cuff re-tear. The tendons are retracted to the glenoid

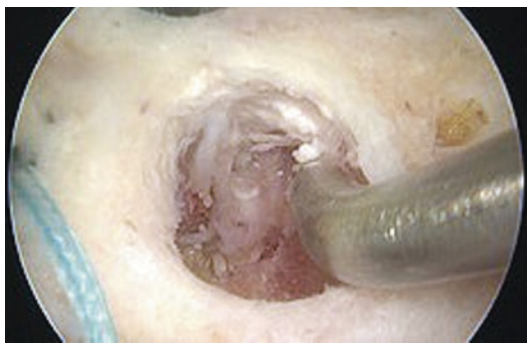

Fig. 32.4 Right shoulder. Arthroscopic re-revision. View from the lateral portal shows osteolysis from previous biodegradable anchor

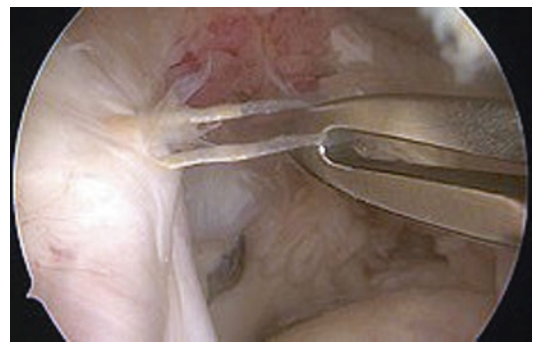

Fig. 32.5 Right shoulder. Arthroscopic re-revision. View from the lateral portal shows intact previous sutures into the cuff

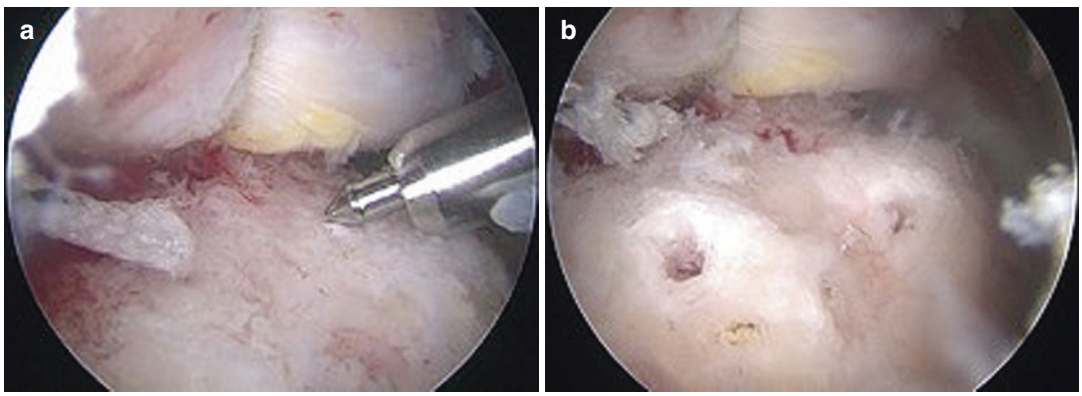

Fig. 32.6 Right shoulder. Arthroscopic re-revision. View from the lateral portal. (**a**) Arthroscopic awl for nanofractures. (**b**) Nanofractures of the greater tuberosity

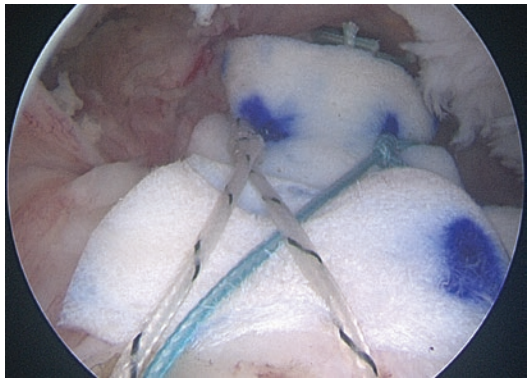

Fig. 32.7 Right shoulder. Arthroscopic re-revision. View from the lateral portal: a porcine dermal extracellular matrix was used as an augmentation. The matrix was fixed over the cuff by using two nonabsorbable sutures of different colors medially and two knotless polyetheretherketone anchors laterally in a suture-bridge configuration

(PEEK) anchors in a suture-bridge configuration (Fig. 32.7). The patient was discharged from the hospital the day after the surgery.

The arm was immobilized in a sling for 6 weeks. Three weekly subacromial injections of autologous conditioned plasma (ACP) were performed, starting from 10 days after surgery. Recovery of passive ROM was allowed after sling removal. Strengthening exercises started after recovery of full passive ROM, around 10 weeks after surgery. Sports activities and manual work were not allowed up to 6 months after surgery. No intra- or postoperative complications were reported.

At a 6-month follow-up, the patient showed full passive and active ROM with no pain. Moreover, the MRI revealed a complete cuff healing, graded as type 2 according to Sugaya's classification [9] (Fig. 32.8).

32.3 Discussion

One of the most substantial difficulties in the management of rotator cuff pathology is determining whether a rotator cuff tear will heal successfully. Failure of rotator cuff repair healing, which most commonly occurs at the tendon-bone interface, has been reported up to 94% of

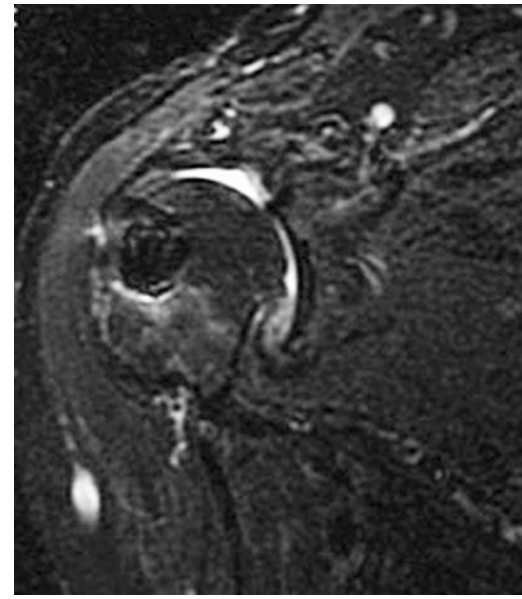

Fig. 32.8 Right shoulder. Postoperative MRI (coronal view) shows healed rotator cuff (type 2 according to Sugaya's classification)

patients [10]. Although not always symptomatic, rotator cuff re-tear can be a debilitating and painful condition, especially in young and active patients. When it happens, a systematic approach to the problem may lead to a successful outcome. It is not always easy to understand why the cuff repair failed, but it is worth to try before attempting a new surgical procedure. First, patient's history including not only information about symptoms onset but also comorbidities that can influence bone and tendon quality, as well as at-risk activities, must be investigated. Patient's functional request is also very important. A symptomatic cuff re-tear in low-demanding patients can be managed with conservative strategies or palliative surgeries such as biodegradable spacer. On the opposite, a symptomatic re-tear in young active patients needs a surgical revision. Second, clinical examination is of utmost importance. Concomitant pathologies such as cervical spine issues or neurological deficits, related or not to the previous surgery, must be ruled out. Third, surgical reports of previous surgeries are helpful to acquire information about initial tear size and morphology, reducibil-

ity, and repair technique. Nevertheless, it is important to know what devices were used, e.g., sutures only or suture anchors, permanent or biodegradable materials, etc. Here, it has been presented a case where a biodegradable anchor was used. Biodegradable anchors were designed to provide secure fixation while allowing for later resorption and replacement by host tissue. Unfortunately, literature data showed that implants degraded relatively rapidly causing foreign-body reactions, synovitis, fragmentation, and osteolysis. It is not known if the primary cause of the osteolysis is biological (precipitated by breakdown products of the polymer) or mechanical (caused by initial loss of implant stability) [11–13]. Recent clinical studies reported that osteolysis around biodegradable anchors do not appear to adversely affect the healing and clinical outcome of rotator cuff repair [14, 15]. However, as it has already been shown in a previous case report [11], in the present case, the use of a biodegradable anchor could actually represent a possible explanation to the failure. Fourth, imaging findings verify clinical hypothesis and clarify re-tear characteristics and the degree of fatty infiltration. Although the role of fatty infiltration in assessing the re-tear risk is still controversial [2], it should be surely taken into account when planning a revision surgery. Although numerous techniques are available for revision rotator cuff repair, understanding the biology of the rotator cuff is of utmost importance if anatomic techniques are considered. In the present case, since it was a second failure with no traumatic onset, a new simple arthroscopic repair did not seem a reasonable option. At the same time, a latissimus dorsi transfer was also not considered because it is a salvage procedure in irreparable posterosuperior cuff tear with unpredictable functional results [16]. In the present case, the patient was an active young woman with high functional demand, and according to imaging findings the remaining cuff was still repairable with no sign of fatty infiltration. Therefore, it was decided for an arthroscopic re-revision by performing a biologic augmentation.

The primary goal of biologic augmentation is to improve tendon-to-bone healing by providing a structural support. Patch grafts of several materials have been proposed in the last 20 years. Besides human dermal allograft, porcine dermal extracellular matrices have been proved to have the most suitable characteristics [17]. Particularly, they promote cell proliferation and expression of collagen types I and III, with no inflammatory reactions [18, 19].

The additional administration of three subacromial injections of ACP aimed to enhance the patient's natural healing response by delivering growth factors required for tendon healing. No clear algorithms have been reported for the use of growth factors in association with patch augmentation. However, a recent study on a rabbit model of chronic cuff tear showed enhanced tendon-to-bone healing after patch augmentation and local administration of autologous platelet-rich plasma [20].

In conclusion, understanding the cause of failure and, if still possible, enhancing the cuff biology are probably the key of success in cuff revision surgery.

References

1. Gurnani N, van Deurzen DFP, van den Bekerom MPJ. Shoulder-specific outcomes 1 year after nontraumatic full-thickness rotator cuff repair: a systematic literature review and meta-analysis. Shoulder Elbow. 2017;9:247–57. https://doi.org/10.1177/1758573217711903.
2. Saccomanno MF, Sircana G, Cazzato G, et al. Prognostic factors influencing the outcome of rotator cuff repair: a systematic review. Knee Surg Sports Traumatol Arthrosc. 2016;24:3809–19. https://doi.org/10.1007/s00167-015-3700-y.
3. Elhassan BT, Cox RM, Shukla DR, et al. Management of failed rotator cuff repair in young patients. J Am Acad Orthop Surg. 2017;25:e261–71. https://doi.org/10.5435/JAAOS-D-17-00086.
4. Saltzman BM, Zuke WA, Go B, et al. Does early motion lead to a higher failure rate or better outcomes after arthroscopic rotator cuff repair? A systematic review of overlapping meta-analyses. J Shoulder Elbow Surg. 2017;26:1681–91. https://doi.org/10.1016/j.jse.2017.04.004.
5. 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;24:823–8. https://doi.org/10.5435/JAAOS-D-15-00229.
6. Steinhaus ME, Makhni EC, Cole BJ, et al. Outcomes after patch use in rotator cuff repair. Arthroscopy. 2016;32:1676–90. https://doi.org/10.1016/j.arthro.2016.02.009.
7. Flury M. Patch augmentation of the rotator cuff. A reasonable choice or a waste of money? Orthopade. 2016;45:136–42. https://doi.org/10.1007/s00132-015-3211-z.
8. Goutallier D, Postel JM, Bernageau J, et al. Fatty muscle degeneration in cuff ruptures. Pre- and postoperative evaluation by CT scan. Clin Orthop. 1994;304:78–83.
9. 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. https://doi.org/10.1016/j.arthro.2005.08.011.
10. Galatz LM, Ball CM, Teefey SA, et al. The outcome and repair integrity of completely arthroscopically repaired large and massive rotator cuff tears. J Bone Joint Surg Am. 2004;86A:219–24.
11. Park AY, Hatch JD. Proximal humerus osteolysis after revision rotator cuff repair with bioabsorbable suture anchors. Am J Orthop (Belle Mead NJ). 2011;40:139–41.
12. Kim SH, Kim DY, Kwon JE, et al. Perianchor cyst formation around biocomposite biodegradable suture anchors after rotator cuff repair. Am J Sports Med. 2015;43:2907–12. https://doi.org/10.1177/0363546515608484.
13. Cobaleda Aristizabal AF, Sanders EJ, Barber FA. Adverse events associated with biodegradable lactide-containing suture anchors. Arthroscopy. 2014;30:555–60.https://doi.org/10.1016/j.arthro.2014.02.011.
14. Park J-Y, Jang S-H, Oh K-S, Li YJ. Radiolucent rings around bioabsorbable anchors after rotator cuff repair are not associated with clinical outcomes. Arch Orthop Trauma Surg. 2017;137:1539–46. https://doi.org/10.1007/s00402-017-2772-1.
15. Pilge H, Spang J, Rose T, et al. Osteolysis after rotator cuff repair with bioabsorbable anchors. Arch Orthop Trauma Surg. 2012;132:305–10. https://doi.org/10.1007/s00402-011-1369-3.
16. Anastasopoulos PP, Alexiadis G, Spyridonos S, Fandridis E. Latissimus Dorsi transfer in posterior irreparable rotator cuff tears. Open Orthop J. 2017;11:77–94. https://doi.org/10.2174/1874325001711010077.
17. Xu H, Sandor M, Qi S, et al. Implantation of a porcine acellular dermal graft in a primate model of rotator cuff repair. \J Shoulder Elbow Surg. 2012;21:580–8. https://doi.org/10.1016/j.jse.2011.03.014.
18. Smith RDJ, Zargar N, Brown CP, et al. Characterizing the macro and micro mechanical properties of scaffolds for rotator cuff repair. J Shoulder Elbow Surg. 2017;26:2038–46. https://doi.org/10.1016/j.jse.2017.06.035.
19. Smith RD, Carr A, Dakin SG, et al. The response of tenocytes to commercial scaffolds used for rotator cuff repair. Eur Cell Mater. 2016;31:107–18.
20. Chung SW, Song BW, Kim YH, et al. Effect of platelet-rich plasma and porcine dermal collagen graft augmentation for rotator cuff healing in a rabbit model. Am J Sports Med. 2013;41:2909–18. https://doi.org/10.1177/0363546513503810.

Index

© ESSKA 2018
G. Milano et al. (eds.), *Management of Failed Shoulder Surgery*,
https://doi.org/10.1007/978-3-662-56504-9