Clinical and Radiological Examination of the Shoulder Joint

A Guide for Advanced Practice Physiotherapists

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ISBN 978-3-031-10469-5 ISBN 978-3-031-10470-1 (eBook)
https://doi.org/10.1007/978-3-031-10470-1

This Springer imprint is published by the registered company Springer Nature Switzerland AG
The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland

This book is dedicated to the children of the world with the hope that one day, every child will enjoy the right to education, health care, and pursuing their dreams.

All proceeds of this book's sale will be dedicated to improving the lives of forgotten children who do not get to experience the joy of childhood as they grow up in poverty, illness, war, or injustice.

Acknowledgments

Significance of each scientific discovery, particularly in the field of medicine, is better appreciated when contribution of the former medical scholars is weighed in light of limited diagnostic tools they had access to. By providing an insight into the history of common shoulder conditions, we acknowledge the hardship and contribution of each scholar who originated the voyage we are on today.

Inspiration of our mentors and colleagues, whose love of humanity made them better practitioners in medicine, is acknowledged.

All illustrations are drawn by Madineh Azizy, a talented 16-year-old girl with an autoimmune deficiency illness.

Contents

Chapter 1
Diagnostic Clinical Decision-Making in Shoulder Pathology

The word "diagnostic" is taken from a Greek word, "diagnostikos" meaning able to distinguish (illnesses). Diagnosis refers to identifying an illness or condition based on subjective characteristics of the symptoms and objective clinical signs. Diagnostic clinical decision-making is a vital skill, which incorporates a complex algorithm using cognitive abilities of the clinician to merge new information with the existing knowledge to reach the most accurate identification of the pathology.

Historical Perspective

The term "decision-making" was first described in psychology by Herbert Simon in 1947 [1]. Simon described decision-making as a cognitive process that resulted in selection of a belief or a course of action among several possible alternative options, a process linked with assumptions of values, preferences, and beliefs of the decision-maker [1]. In 1977, he refined the process of decision-making in relation to use of computers, organization structures, work creativity, and employment [2].

In the field of medicine, clinical decision-making has been referred to as a contextual, continuous, and evolving process, where clinical data are gathered, interpreted, and evaluated in order to select an evidence-based choice of action [3]. Historically, clinical decision-making was founded on the expert opinion, based on clinical experience and judgment. In 1969, David Sackett, an American Canadian epidemiologist, changed this when he laid the groundwork for evidence-based practice by applying critical appraisal techniques to the bedside [4, 5]. Sacket suggested the use of epidemiological and biometric methods in diagnostic and therapeutic process to improve the everyday medical practice [6]. In 1991, his student, Gordon Guyatt, used the term "evidence-based medicine (EBM)" when he was designing the core curriculum of the McMaster University Internal Medicine residency program in Canada [7]. Subsequently, the EBM became a millstone that shaped the

H. Razmjou, M. Christakis, *Clinical and Radiological Examination of the Shoulder Joint*, https://doi.org/10.1007/978-3-031-10470-1_1

modern medicine across the world. The initial purpose of Guyatt was educating clinicians in the understanding and use of published literature to optimize clinical care, including the science of systematic reviews. Overtime, however, the literature acknowledged the limitations of the evidence alone, and more stress was put on the need to combine critical appraisal of the evidence with patient's values and preferences through shared decision-making [8]. While Guyatt was the first to use the term EBM, the contribution of many other scientists, epidemiologists, and medical scholars to this movement should not be underestimated or forgotten. The review of rich history of evidence-based medicine is beyond the scope of this chapter, and readers are referred to the relevant literature in this area [4, 9].

As it relates to every day clinical work, the purpose of diagnostic clinical decision-making models is to choose the best action in light of uncertainties arising from clinical information. Unfortunately, generating a valid clinical decision-making algorithm for diagnosis of shoulder pain is not that easy due to an often nonspecific history presented by the patient, the suboptimal accuracy of physical examination tests, and the highly prevalent abnormal imaging findings seen in asymptomatic patients.

The information on accuracy of shoulder clinical tests remains debatable despite the significant number of publications in this area [10, 11]. In summary, pain provocative tests that are solely based on pain (e.g., Neer and Hawkins tests) are sensitive but not very specific, and their positive results cannot pinpoint to a specific pathology [12–15]. In contrast, the strength tests (e.g., Hornblower sign and lift-off test) have high specificity and often a reasonable sensitivity [16–20]. In this chapter, a short summary of the definitions of diagnostic indices is provided to assist with their utility in a clinical setting.

Definition of Diagnostic Indices

Prevalence of a condition is the number of diseased as a proportion of all people and varies depending on the type of clinic or setting. Patients seen at a family physician's office are expected to have a lower prevalence of a specific condition with a milder spectrum of the disease (e.g., shoulder muscle strain). A shoulder specialist, however, is confronted with a more severe form of the condition such as significant rotator cuff tear and advanced arthritis.

Sensitivity is the true-positive rate (number of true positives as a proportion of diseased patients). A highly sensitive test has fewer false-negative results, and as a result, fewer cases of disease are missed. In other words, a negative result of a highly sensitive test rules out a disease with confidence. Considering patients attending primary care clinics often have a disease at an earlier stage, the clinician needs a more sensitive test (less false negatives) to rule out the condition more accurately.

Specificity is the true-negative rate (number of true negatives as a proportion of all non-diseased patients). A highly specific test generates fewer false-positive results, which helps with better diagnosis of a disease. In short, a positive result of a highly specific test rules in a disease with confidence. For most shoulder conditions, clinicians working at tertiary clinics would prefer to have a more specific test with fewer false positives to avoid unnecessary surgery.

Understanding sensitivity and specificity by clinicians who are not familiar with statistical jargon but wish to follow evidence-based practice can be facilitated if we remember that a sensitive test is clinically useful when it is negative. For screening purposes of a high-risk condition, an accurate negative test of a highly sensitive test reduces the risk of missing a serious illness. Similarly, a highly specific test confirms the presence of a condition when it is positive, so it is clinically useful when it is positive (low false-positive rate). It may be reasonable to say that apart from early stages of malignant tumors or metastatic lesions that need a sensitive test to rule them out, for confirmation of a musculoskeletal condition, an accurate positive test of a highly specific test is more useful. In general though, sensitivity and specificity provide limited information in isolation. One important fact that clinicians should be aware of is that neither sensitivity nor specificity alone does determine the power of a clinical or radiological test.

Accuracy of the shoulder diagnostic tests is determined from sensitivity and specificity of the test and the prevalence of the condition. Accuracy is calculated as the sum of the true-positive and true-negative results divided by the sum of all test results. The upper and lower bounds of accuracy are determined by sensitivity and specificity of the clinical test [21–23]. The accuracy varies linearly with the disease prevalence between these bounds [21]. For example, accuracy of a clinical test that has a sensitivity of 50% and specificity of 70% is more than 50% and less than 70%. In cases where the prevalence of disease is low, the accuracy gets closer to specificity and as the prevalence increases, the accuracy of the test gets closer to sensitivity. In situations where no one has the disease (prevalence is 0%), accuracy equals the specificity value, and when everyone has the disease (prevalence is 100%), the accuracy equals sensitivity [21]. Accuracy of the test represents a poor measure of diagnostic test performance and should be provided only as a supplementary information. As noted, accuracy is directly related to the number of true positives and true negatives and prevalence of the condition. If majority of patients do not have the condition (e.g., prevalence is 5%) and the test misses all 5% of the patients, since the other 95% of the patients did not have the disease, the accuracy is calculated as 95%, when in fact the test failed to detect all patients who had the condition.

Likelihood ratios (LRs) are probably the most useful diagnostic indices as they help to revise the probability of a disease at a specific individual patient level. The likelihood ratios are calculated by combining sensitivity and specificity [24, 25]. They revise the probability of having a disease after application of a particular clinical or imaging test, and consequently they help the clinician to move closer to the correct diagnosis through changing the magnitude of the pretest probability [24].

Pretest probability of a pathological condition (chance of having a condition before application of any diagnostic test) is a critical factor in the chain of diagnostic process. Once the sensitivity and specificity and likelihood ratios of a clinical test are established, the estimation of prior probability of the condition is required by the clinician to interpret the test results at an individual patient level. The pretest probability of a shoulder condition is often predicted based on patient's demographics, symptoms, medical history, mechanism of injury, observation of muscle mass or deformities, range of motion, and the knowledge of the examiner about the condition [26]. A simple rule to remember is that when the pretest probability of a condition is either very high or very low, a clinical test does not play an important role in ruling in or out of the condition. In other words, the clinician already knows (based on the pretest probability of the condition that has formed in his/her mind) that the patient either has or does not have the condition. The other important factor that should be considered in the diagnostic process is the risk or the cost associated with the application of a test when the posttest probability is not expected to change significantly from the pretest probability. For example, we know that severe muscle wasting in the supraspinatus and infraspinatus muscles with an inability to actively elevate the arm is highly indicative of a massive cuff tear, and in many cases, a successful repair is not likely. Observing a positive Hornblower clinical sign (observed in major multiple rotator cuff tears) would not change the diagnosis too much as the pre-Hornblower test probability was already very high. Since this test is easy to perform, is highly specific [16], and has no risk or cost, it is wise to use it to confirm the diagnosis. However, using a costly imaging investigation such as magnetic resonance imaging (MRI) for this patient needs to be justified. The MRI will not drastically change the diagnosis or even management as massive tears particularly in older individuals are not amenable to a successful repair. Therefore, ordering costly imaging is inefficient when the results do not change the clinician's practice. In certain cases apart from the cost, an imaging test may even pose a risk to patient's health. For example, ordering an MRI with a gadolinium contrast in a patient with kidney problems who has a labral tear and advanced osteoarthritis of the glenohumeral joint is not wise. While labral pathology is best visualized through the enhanced MRI, the presence of advanced arthritis nullifies the usefulness of the information obtained from imaging as this patient may need joint replacement rather than labral repair. In addition, gadolinium-containing contrast dyes may induce nephropathy and serious systemic complications in patients with diabetes, heart conditions, and chronic kidney disease.

The relationship between prevalence, sensitivity, specificity, likelihood ratios, and pre−/posttest probability is a complex topic and largely beyond the scope of this chapter. Examples on the role of pretest probability on diagnostic and therapeutic decision-making of the shoulder tests is provided elsewhere [27], and the interested readers are referred to the literature that combines the information on accuracy of specific shoulder clinical tests and probability of the shoulder disease [15, 16, 20, 28].

In conclusion, a skilled physiotherapist or a primary family physician should use highly sensitive tests when they are planning to exclude a condition and use highly specific tests when they are aiming to confirm a condition. Another important rule is to reconsider ordering highly costly or harmful imaging tests when the posttest probability is very close to pretest probability and the investigation does not change management. Therefore, the role of pretest probability is more critical when the clinical picture is not very clear (e.g., pretest probability is moderate).

While a number of shoulder diagnostic algorithms have been proposed in the settings of extended role physiotherapists [29, 30], the need for evidence-based algorithms that summarize the contribution of various components of diagnosis and imaging into management remains strong.

Origin of Shoulder Pain

Nociceptive Pain

The pain that comes directly from the shoulder joint, muscles, tendons, or bone is often nociceptive pain. Nociceptive pain results from injury or inflammation of the nonneural tissues of *somatic* or *visceral* structures [31]. The somatic pain caused by skin, joint, bone, tendon, and muscle damage is characterized as a well-localized pain and may be felt as aching, throbbing, or cramping. The shoulder pain of somatic origin is often exacerbated by movement (incident pain) and relieved upon rest. Visceral pain arises from internal organs and blood vessels and is not always localized and may be referred to other parts of body. The pain of visceral origin is more diffuse and poorly defined. Visceral pain is mediated by nociceptors in the cardiovascular, respiratory, gastrointestinal, and genitourinary system [32–34]. Visceral pain is often associated with marked autonomic phenomena, including pallor, profuse sweating, nausea, gastrointestinal disturbances, and changes in body temperature, blood pressure, and heart rate [35, 36].

A referred visceral pain felt in a specific somatic area is due to neuromerical connection with the affected organ. It appears that the overlap of somatic and visceral afferent information into a shared neural pathway may be the cause of misinterpretation at peripheral, spinal, or supraspinal levels. Referred muscle pain from viscera without hyperalgesia is explained on the basis of the convergence of visceral and somatic afferent fibers on the same central neurons. Referred muscle pain from viscera with hyperalgesia is hypothesized to be related to both central (sensitization process) and peripheral (intervention of reflex arcs) mechanisms [37, 38]. The pathophysiology of somatic and visceral pain associations is complex and multifactorial, and clinicians should always consider cardiovascular, intra-abdominal, and lung pathologies as the potential source of referred pain of visceral origin to the shoulder area [39] when the clinical picture is not straight forward.

Neuropathic Pain

Although nociceptive pain is the cause of majority of complaints in orthopedic practice, physiotherapists and primary clinicians need to differentiate this type of pain from the neural tissue origin called neuropathic pain to avoid misdiagnosis and inappropriate treatment [40]. Neuropathic pain is caused by a direct injury to neural tissues and may affect the central or peripheral nervous system. The direct damage to neural tissues may be of external or internal origin such as chemotherapy or diabetes, respectively. The shoulder pain of neuropathic origin has been reported in the impingement syndrome [39, 41], rotator cuff tears, [42, 43], and post-shoulder arthroplasty surgery [44].

Cervical Spine-Related Pain

Pain of spinal origin may be neuropathic or nociceptive. While a true spinal nerve root compression, peripheral nerve entrapment, or dorsal root ganglia compression could cause neuropathic pain, not all referred pains from cervical spine are neuropathic in nature [45]. For example, the mechanical strain to the disc annulus leads to chemical mediator release, causing inflammation. The release of chemical mediators of inflammation in an injured disc produces nociceptive pain when it activates nerve sheath nociceptors. This type of disc pathology is similar to that of a sprained ligament and does not involve a neuropathic process and often presents with complaint of pain referred to more distal joints or limb. According to Bogduk [40, 46], the important features of nerve compression such as numbness, weakness, or paresthesia should be present before the term “neuropathic” is used for referred pain of spinal origin.

A true cervical spine neuropathy is associated with neurological symptoms in specific dermatomes or myotomes. It may occur secondary to disc herniation or bony osteophytes and foraminal stenosis that impinge on the cervical nerve root. High cervical radiculopathies (e.g., C3 or C4 roots) may have a similar pain distribution to suprascapular neuropathy (discussed in this chapter) without a referred pain to the arm. C5 radiculopathy can cause pain and associated symptoms in the upper arms and shoulder blades. In C6 (C5-C6 disc herniation) radiculopathy, symptoms often radiate into the neck, shoulder, and arm, associated with abnormal sensation in the lateral arm, forearm, thumb, and index finger. Often, the biceps and brachioradialis tendon reflexes are depressed or absent. The C7 root (C6-C7 disc herniation) is the most commonly affected level which involves the neck, arm, and middle finger. The symptoms of C8 root (C7-T1 disc herniation) radiculopathy are felt in the neck along the medial arm to the ring and little fingers [47]. Because the clinical management of nociceptive somatic pain arising from a cervical disc bulge or nerve sheets is different than the neuropathic pain arising from a true nerve root compression, differentiating between these two types of pain is critical [40].

Non-spinal Neuropathic Shoulder Pain

In addition to spine-related neuropathies, nerve root entrapment in the scapular area could cause posterior shoulder pain. The two rare neuropathies that have been reported in this area are the suprascapular nerve entrapment and the axillary nerve

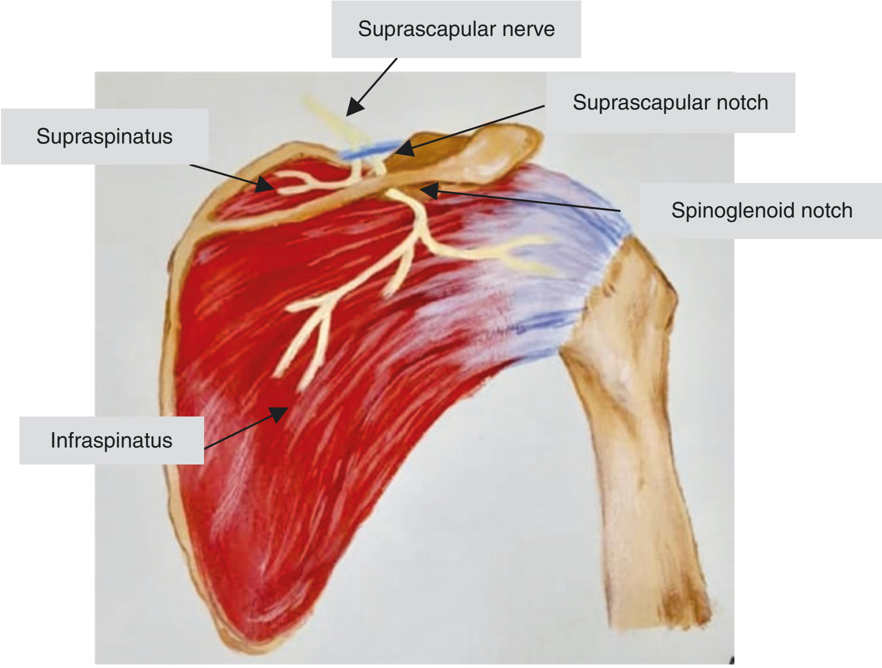

Fig. 1.1 Compression of the suprascapular nerve at the level of the suprascapular notch affects both supraspinatus and infraspinatus. Compression at the spinoglenoid notch will cause isolated atrophy of the infraspinatus muscle

compression seen in quadrilateral space syndrome (QSS). Misdiagnosing these conditions can lead to inappropriate management.

The suprascapular neuropathy may occur at the suprascapular notch or supraglenoid notch and may be associated with a vague pain in the posterior aspect of the shoulder. Compression or traction of the suprascapular nerve may be idiopathic or result from paralabral cysts, tumors, traumatic injuries, or repetitive use [48–51]. The paralabral cyst may develop as a result of traction injuries or repetitive trauma secondary to overhead sports that may cause tearing of the capsulo-labral complex. These cysts often form adjacent to a torn glenoid labrum and may extend into the suprascapular or spinoglenoid notches and compress the suprascapular nerve. The atrophic changes of the infraspinatus are the hallmark of nerve entrapment in the supraglenoid notch. The supraspinatus muscle is often spared as the entrapment often happens beneath the inferior transverse scapular ligament at the spinoglenoid notch when the nerve passes through to innervate the infraspinatus muscle. Figure 1.1 shows the anatomical locations where the suprascapular nerve may be compressed. Figure 1.2 shows the severe atrophy of the infraspinatus muscle at the spinoglenoid notch in a high-level tennis player.

Fig. 1.2 Clinical presentation of suprascapular nerve compression at the spinoglenoid notch in a high-level tennis player with isolated severe atrophy of the infraspinatus muscle and mild changes in supraspinatus. The MRI showed severe fatty infiltration in the infraspinatus muscle

The suprascapular nerve neuropathy has also been reported in association with retracted massive rotator cuff tears [52, 53]. The cause and effect relationship and the optimal management of neuropathy in the presence of retracted cuff tear, however, remain controversial [54, 55]. The MRI can be useful to assess atrophic changes of the rotator cuff muscles and the potential causes of suprascapular nerve compression. The more conclusive tests used to confirm suprascapular neuropathy are electromyography (EMG) and nerve conduction velocity studies that examine the muscle and nerve structures, respectively [56].

The other rare neuropathy of the shoulder joint caused by entrapment is the quadrilateral space syndrome (QSS) which affects the axillary nerve and posterior humeral circumflex artery. The quadrilateral space is bordered by the proximal humerus, the long head of the triceps tendon, teres minor, and teres major [57, 58] (Fig. 1.3). According to Cahill and Palmer who defined this syndrome in 1983 [58], there is a point tenderness in the posterior aspect of the shoulder with symptoms being aggravated with forward flexion and/or abduction and external rotation. At present, there is no gold standard diagnostic imaging for this condition. Diagnosis of axillary nerve entrapment may be confirmed by EMG which can evaluate the presence of neuropathic changes in the deltoid and teres minor muscles. The MRI findings are not very specific and include neurogenic edema and selective fatty atrophy of the teres minor and deltoid muscles [59, 60]. The occlusion of the posterior humeral circumflex artery may be confirmed by arteriogram with the arm in abduction and external rotation [59].

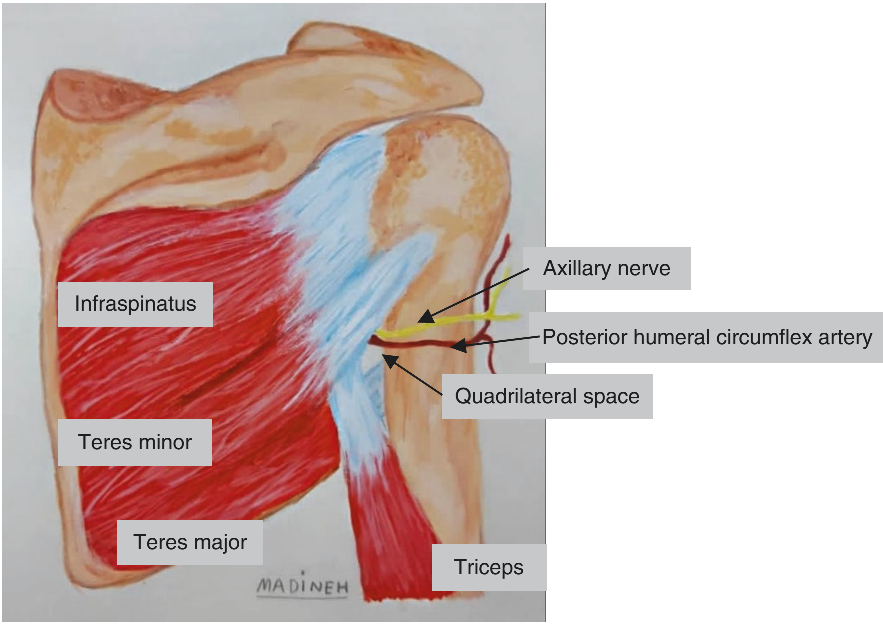

Fig. 1.3 The quadrilateral space is bordered by the proximal humerus, the long head of the triceps tendon, teres minor, and teres major

Major Categories of Common Shoulder Pathology

The specific shoulder pathologies may be divided into six major diagnostic categories: impingement syndrome; biceps tendon pathology including tendinitis, partial tears, and full ruptures of the short and long heads; partial- and full-thickness rotator cuff tears; advanced cuff pathology including cuff tear arthropathy, adhesive capsulitis, arthritis of the glenohumeral joint including primary osteoarthritis, rheumatoid arthritis, secondary arthritis, etc.; superior labral pathologies; and the glenohumeral instability. Chaps. 2–9 provide detailed information on history, etiology, diagnosis, and management of these conditions.

References

1. Simon HA. Administrative behavior: a study of decision-making processes in administrative organization. 1st ed. New York: Macmillan; 1947.
2. Simon HA. The new science of management decision, revised edition ed. Prentice Hall; 1977.
3. Tiffen J, Corbridge SJ, Slimmer L. Enhancing clinical decision making: development of a contiguous definition and conceptual framework. J Prof Nurs. 2014;30(5):399–405.

4. Thoma A, Eaves FF 3rd. A brief history of evidence-based medicine (EBM) and the contributions of Dr David Sackett. Aesthet Surg J. 2015;35(8):NP261-3.
5. Sackett DL. Clinical epidemiology. Am J Epidemiol. 1969;89(2):125–8.
6. Sackett DL. Evidence-based medicine and treatment choices. Lancet. 1997;349(9051):570. author reply 572-3
7. Guyatt GH. Evidence-based medicine. ACP J Club. 1991;114:A-16.
8. Djulbegovic B, Guyatt GH. Progress in evidence-based medicine: a quarter century on. Lancet. 2017;390(10092):415–23.
9. Sur RL, Dahm P. History of evidence-based medicine. Indian J Urol. 2011;27(4):487–9.
10. Thoomes EJ, van Geest S, van der Windt DA, Falla D, Verhagen AP, Koes BW, et al. Value of physical tests in diagnosing cervical radiculopathy: a systematic review. Spine J. 2018;18(1):179–89.
11. Innocenti T, Ristori D, Miele S, Testa M. The management of shoulder impingement and related disorders: a systematic review on diagnostic accuracy of physical tests and manual therapy efficacy. J Bodyw Mov Ther. 2019;23(3):604–18.
12. Leroux JL, Thomas E, Bonnel F, Blotman F. Diagnostic value of clinical tests for shoulder impingement syndrome. Rev Rhum Engl Ed. 1995;62(6):423–8.
13. Hertel R, Ballmer FT, Lombert SM, Gerber C. Lag signs in the diagnosis of rotator cuff rupture. J Shoulder Elb Surg. 1996;5(4):307–13.
14. Itoi E, Minagawa H, Yamamoto N, Seki N, Abe H. Are pain location and physical examinations useful in locating a tear site of the rotator cuff? Am J Sports Med. 2006;34(2):256–64.
15. Razmjou H, Holtby R, Myhr T. Pain provocative shoulder tests: reliability and validity of the impingement tests. Physiother Can. 2004;56(4):229–36.
16. Razmjou H, Christakis M, Dwyer T, van Osnabrugge V, Henry P, Nam D, et al. Diagnostic accuracy of clinical tests in detecting rotator cuff pathology. Orthop Sports Med Open Access J. 2019;2(4):170–9.
17. Hegedus EJ, Goode AP, Cook CE, Michener L, Myer CA, Myer DM, et al. Which physical examination tests provide clinicians with the most value when examining the shoulder? Update of a systematic review with meta-analysis of individual tests. Br J Sports Med. 2012;46(14):964–78.
18. Razmjou H, Christakis M, Dwyer T, van Osnabrugge V, Holtby R. Accuracy of infraspinatus isometric testing in predicting tear size and tendon reparability: comparison with imaging and arthroscopy. J Shoulder Elb Surg. 2017;26(8):1390–8.
19. Razmjou H, Fournier-Gosselin S, Christakis M, Pennings A, ElMaraghy A, Holtby R. Accuracy of magnetic resonance imaging in detecting biceps pathology in patients with rotator cuff disorders: comparison with arthroscopy. J Shoulder Elb Surg. 2015;
20. Holtby R, Razmjou H. Validity of the supraspinatus test as a single clinical test in diagnosing patients with rotator cuff pathology. J Orthop Sports Phys Ther. 2004;34(4):194–200.
21. Guggenmoos-Holzmann I, van Houwelingen HC. The (in)validity of sensitivity and specificity. Stat Med. 2000;19(13):1783–92.
22. Eisenberg MJ. Accuracy and predictive values in clinical decision-making. Cleve Clin J Med. 1995;62(5):311–6.
23. Eisenberg MJ, Pilote L. Pharmacologic stress echocardiography. Ann Intern Med. 1992;117(2):168. author reply 168-9
24. Radack KL, Rouan G, Hedges J. The likelihood ratio. An improved measure for reporting and evaluating diagnostic test results. Arch Pathol Lab Med. 1986;110(8):689–93.
25. Einstein AJ, Bodian CA, Gil J. The relationships among performance measures in the selection of diagnostic tests. Arch Pathol Lab Med. 1997;121(2):110–7.
26. Hamzeh H, Madi M. Using the diagnostic thinking inventory in musculoskeletal physiotherapy: a validity and reliability study. Physiother Res Int. 2021:e1895.
27. Razmjou H, Haines T, Holtby R. Diagnostic and therapeutic decision-making: exploring the role of Pretest probability in patients with rotator cuff pathology. Physiother Can. 2006;58:196–204.

28. Holtby R, Razmjou H. Accuracy of the Speed's and Yergason's tests in detecting biceps pathology and SLAP lesions: comparison with arthroscopic findings. Arthroscopy. 2004;20(3):231–6.
29. Razmjou H, Christakis C, Kennedy D, Robarts S, Holtby R. Impact of radiographic imaging of the shoulder joint on patient management: an advanced practice Physiotherapist's approach. Physiother Can. 2017;69(4):351–60.
30. Raja A, Craig EV, Braman JP. Rotator cuff tendon calcific tendinitis treatment algorithm for primary care musculoskeletal physicians. J Family Med Prim Care. 2019;8(5):1647–52.
31. Loeser JD, Treede RD. The Kyoto protocol of IASP basic pain terminology. Pain. 2008;137(3):473–7.
32. Haasenritter J, Stanze D, Widera G, Wilimzig C, Abu Hani M, Sonnichsen AC, et al. Does the patient with chest pain have a coronary heart disease? Diagnostic value of single symptoms and signs—a meta-analysis. Croat Med J. 2012;53(5):432–41.
33. van Oosterhout REM, de Boer AR, Maas AHEM, Rutten FH, Bots ML, Peters SAE. Sex differences in symptom presentation in acute coronary syndromes: a systematic review and meta-analysis. J Am Heart Assoc. 2020;9(9):e014733.
34. Ray R, Lemire JE. Liver laceration in an intercollegiate football player. J Athl Train. 1995;30(4):324–6.
35. Sanvictores T, Tadi P. Neuroanatomy, autonomic nervous system visceral afferent Fibers and pain. StatPearls Treasure Island (FL): StatPearls Publishing LLC; 2021.
36. Janig W. Systemic and specific autonomic reactions in pain: efferent, afferent and endocrine components. Eur J Anaesthesiol. 1985;2(4):319–46.
37. Affaitati G, Costantini R, Tana C, Cipollone F, Giamberardino MA. Co-occurrence of pain syndromes. J Neural Transm (Vienna). 2020;127(4):625–46.
38. Vecchiet L, Vecchiet J, Giamberardino MA. Referred muscle pain: clinical and pathophysiologic aspects. Curr Rev Pain. 1999;3(6):489–98.
39. Morgan EH. Pain in the shoulder and upper extremity; visceral causes considered by the internist. J Am Med Assoc. 1959;169(8):804–8.
40. Bogduk N. On the definitions and physiology of back pain, referred pain, and radicular pain. Pain. 2009;147(1-3):17–9.
41. Gwilym SE, Oag HC, Tracey I, Carr AJ. Evidence that central sensitisation is present in patients with shoulder impingement syndrome and influences the outcome after surgery. J Bone Joint Surg Br. 2011;93(4):498–502.
42. Karasugi T, Ide J, Kitamura T, Okamoto N, Tokunaga T, Mizuta H. Neuropathic pain in patients with rotator cuff tears. BMC Musculoskelet Disord. 2016;17(1):451-016-1311-5.
43. Ko S, Choi C, Kim S, Chae S, Choi W, Kwon J. Prevalence and risk factors of neuropathic pain in patients with a rotator cuff tear. Pain Physician. 2018;21(2):E173–80.
44. Razmjou H, Woodhouse LJ, Holtby R. Neuropathic pain after shoulder arthroplasty: prevalence, impact on physical and mental function, and demographic determinants. Physiother Can. 2018;70(3):212–20.
45. Seaman DR, Cleveland C 3rd. Spinal pain syndromes: nociceptive, neuropathic, and psychologic mechanisms. J Manip Physiol Ther. 1999;22(7):458–72.
46. Bogduk N. Innervation, pain patterns, and mechanisms of pain production. In: Twomey L, Taylor J, editors. Physical therapy of the low back. 2nd ed. New York: Churchill Livingston; 1994. p. 93–109.
47. Rhee JM, Yoon T, Riew KD. Cervical radiculopathy. J Am Acad Orthop Surg. 2007;15(8):486–94.
48. LiBrizzi CL, Rojas JL, Bontrager NC, Srikumaran U, McFarland EG. Suprascapular nerve entrapment in young kayaker: a case report. Phys Sportsmed. 2020;48(2):236–40.
49. Ohanisian L, Brown N, White SD, Rubay D, Schwartz PM. Persistent shoulder pain due to a suprascapular nerve injury in the setting of trauma. Cureus. 2019;11(3):e4224.
50. Maalouly J, Aouad D, Tawk A, El Rassi G. A spinoglenoid cyst compressing on the suprascapular nerve causing supraspinatus and infraspinatus muscle weakness: a case report. Int J Surg Case Rep. 2020;71:266–9.

51. Memon AB, Dymm B, Ahmad BK, Sripathi N, Schultz L, Chandok A. Suprascapular neuropathy: a review of 87 cases. Muscle Nerve. 2019;60(3):250–3.
52. Kong BY, Kim SH, Kim DH, Joung HY, Jang YH, Oh JH. Suprascapular neuropathy in massive rotator cuff tears with severe fatty degeneration in the infraspinatus muscle. Bone Joint J. 2016;98-B(11):1505–9.
53. Sachinis NP, Boutsiadis A, Papagiannopoulos S, Ditsios K, Christodoulou A, Papadopoulos P. Suprascapular neuropathy in the setting of rotator cuff tears: study protocol for a double-blinded randomized controlled trial. Trials. 2016;17(1):554-016-1672-y.
54. Gerber C, Meyer DC, Wieser K, Sutter R, Schubert M, Kriechling P. Suprascapular nerve decompression in addition to rotator cuff repair: a prospective, randomized observational trial. J Shoulder Elb Surg. 2020;29(8):1633–41.
55. Yang P, Wang C, Zhang D, Zhang Y, Yu T, Qi C. Comparison of clinical outcome of decompression of suprascapular nerve at spinoglenoid notch for patients with posterosuperior massive rotator cuff tears and suprascapular neuropathy. BMC Musculoskelet Disord. 2021;22(1):202-021-04075-1.
56. Freehill MT, Shi LL, Tompson JD, Warner JJ. Suprascapular neuropathy: diagnosis and management. Phys Sportsmed. 2012;40(1):72–83.
57. McClelland D, Paxinos A. The anatomy of the quadrilateral space with reference to quadrilateral space syndrome. J Shoulder Elb Surg. 2008;17(1):162–4.
58. Cahill BR, Palmer RE. Quadrilateral space syndrome. J Hand Surg Am. 1983;8(1):65–9.
59. Chen H, Onishi K, Zhao X, Chang EY. Neuromuscular ultrasound application to the electrodiagnostic evaluation of quadrilateral space syndrome. PM R. 2014;6(9):845–8.
60. Linker CS, Helms CA, Fritz RC. Quadrilateral space syndrome: findings at MR imaging. Radiology. 1993;188(3):675–6.

Chapter 2
Impingement Syndrome

Impingement syndrome is a generic term with a large number of conditions under its umbrella. It refers to a mechanism that compresses the rotator cuff tendons against other structures. The impingement syndrome of the shoulder joint is typically classified as the primary (outlet) or internal impingement [1, 2]. Other types of impingement syndrome discussed in the literature are secondary and functional impingement. Internal and secondary impingement have overlapping characteristics and are commonly reported in overhead athletes with subtle anterior instability [2]. Functional impingement [3] is predominantly caused by altered scapulohumeral mechanics and muscle imbalance. The important note to remember is that most often different types of impingement may coexist and treatment needs to address all aspects of pathology.

Primary or Outlet Impingement

The classic primary impingement syndrome occurs as a result of impingement of the rotator cuff tendons by the ligamentous encroachment or osseous overgrowth in the subacromial area and is more common in older individuals. The primary impingement is referred to as the outlet or subacromial impingement as well [4].

Historical Perspective

Primary impingement syndrome was first described by Neer in 1972 [4]. He felt that the anterior 1/3 of the acromion, the coracoacromial ligament, and the acromioclavicular joint were the causes of impingement of the rotator cuff. In 1983, Neer described the impingement syndrome in three stages. Stage I is identified by edema

H. Razmjou, M. Christakis, *Clinical and Radiological Examination of the Shoulder Joint*, https://doi.org/10.1007/978-3-031-10470-1_2

and hemorrhage of the bursa and is found in younger patients. Stage II impingement is recognized by fibrosis and tendinitis of the rotator cuff tendons, which often are irreversible. Stage III impingement involves more chronic changes and may be associated with partial or complete rotator cuff tears and is often seen in patients between ages of 25–40 years [5]. Causes of primary impingement syndrome have been noted as intrinsic and extrinsic factors.

Intrinsic Factors of Primary Impingement Syndrome

Intrinsic factors appear to play a role in primary impingement syndrome. Intrinsic biologic factors refer to age-related changes, impaired vascular supply to the tendons, chronic inflammation in the bursa or tendons, and chronic tensile or shear overload. The degenerative changes of the supraspinatus and infraspinatus tendons can affect their function as the superior suppressors of the humeral head, leading to narrowing of the subacromial space and ultimately subacromial impingement syndrome. In 1997, Bigliani and Levine summarized the intrinsic factors as overuse, muscle weakness, and degenerative tendinopathy [6]. Overuse and repetitive microtrauma may cause inflammation and thickening of the rotator cuff tendons or the subacromial bursa, leading to tendinitis [7–10]. Tendinitis may progress to partial-thickness and ultimately full-thickness rotator cuff tendon tears.

Calcified (Calcifying) Tendinitis

Rotator cuff calcified tendinitis is a form of tendinitis, characterized by deposits of crystalline calcium hydroxyapatite in the tendons of the rotator cuff [11]. This condition was first described radiologically by Painter in 1907 [12]. The smaller foci of calcium deposits cause symptoms due to an inflammatory reaction which will gradually resolve through vascular development and absorption of macrophages and multinuclear giant cells. The larger deposits may cause mechanical symptoms and impingement. Calcific deposits are found in 3–20% of asymptomatic shoulders and may not be the primary cause of shoulder pain in many cases [13, 14]. Uhthoff and Loehr [15] proposed a cycle to describe pathological and clinical stages of this condition. They proposed three distinct stages of precalcific, calcific, and postcalcific. In the precalcified stage, the patient either is completely asymptomatic or has relatively mild symptoms. During the calcific phase, calcium crystals are deposited in the tendon matrix with the deposits having a chalky consistency with a well-defined border and a dense appearance on plain radiographs. During this phase, the fibrocollagenous tissue gradually forms a border, which indicates that deposition of calcium has terminated. The resorptive phase is the last part of the calcific stage and begins after a variable period of disease inactivity. Presence of vascular tissue at the borders of the deposits signals spontaneous resorption. Macrophages and multinuclear giant cells absorb the deposit during this phase where the calcific deposit may resemble a toothpaste consistency. During this phase, the deposits are less well-defined and appear as cloud-like lesions on plain radiographs. Often, the condition becomes acutely painful when the calcific deposits start to undergo resorption. In addition, occasionally during this phase, the paste may leak into the subacromial bursa, which may result in even more painful symptoms [15]. After the calcific deposit has been resorbed and in the postcalcific stage, fibroblasts reconstitute the collagen pattern of the tendon.

The calcific tendinitis has been reported in individuals between 40 and 60 years of age with women being more affected than men [16]. The most common site of calcium deposit is the supraspinatus tendon, followed by the infraspinatus, teres minor, and subscapularis [13]. The condition is more common in patients with the history of endocrine and metabolic disorders. In an early study in 1989, Mavrikakis reported that 31.8% of the diabetics had shoulder calcification compared with 10.3% of the control group. Hurt and Baker reported that insulin-dependent diabetes had over 30% higher prevalence of tendon calcification. Thyroid disorders are reported be related to the onset of this condition as well [17, 18]. The role of genetic predisposition in calcified tendinitis has been noted by some researchers [19].

In a review by De Carli et al. [16], calcific tendinitis was characterized by a disabling pain which often occurred spontaneously, usually in the morning. Most patients recalled a sudden severe stiffness, resembling a frozen shoulder syndrome which gradually felt better over a period of time. Calcified tendinitis can be easily diagnosed with the imaging studies such as conventional radiography or ultrasound [16, 20, 21]. The lesions may appear well bordered or less well-defined and cloud-like, depending on the stage of the condition.

Management of calcified tendinitis depends on the severity of pain and disability. The initial treatment of calcified tendinitis includes rest, physical therapy, nonsteroidal anti-inflammatory drugs, subacromial corticosteroid injections [22], and fluoroscopically guided needle aspiration or lavage, a minimally invasive technique that is used for larger and more symptomatic calcific tendinitis [23]. The evidence on effectiveness of extracorporeal shockwave therapy in reducing pain and improving range of motion remains controversial [24]. In cases resistant to nonoperative measures, surgical removal of the large calcium deposits may be attempted, although there is no general consensus regarding the type or extent of resection of the deposits [25]. In a systematic review of different surgical approaches for calcified tendinitis, no significant differences were found in functional and clinical outcome between debridement and removal of the calcific deposits plus/minus acromioplasty [26]. In general, removal of the calcium deposits leaves various degrees of cuff defects, and the arthroscopic debridement and excision of calcium deposits should be done with caution to minimize further risks to cuff structures [25].

Extrinsic Factors of Primary Impingement Syndrome

The extrinsic factors for primary impingement syndrome are caused by acromial morphology [6, 27–29], enthesopathic changes of the acromion that cause pressure on the tendons [30, 31], osteophytes secondary to degenerative changes of the acromioclavicular (AC) joint [32], thickening of the coracoacromial ligament [33–35], coracoid process abnormalities [36], and developmental abnormalities of acromion (e.g., os acromiale). Other abnormalities such as osteolysis of the distal clavicle may also cause pain in the subacromial region and have been discussed in this section.

Acromion Morphology

The morphology of the acromion is correlated with clinical signs of impingement syndrome. Four types of acromion morphology have been described: these include type I with a flat undersurface, types II and III with a concave undersurface, and type IV with a convex undersurface near distal end of the acromion. Type II has a gentle undersurface curvature and type III presents with an anterior hook. The flat and concave types were described by Bigliani et al. in 1986 [37], and type IV was later described by Vanarthos et al. in 1995 [38]. In a study of dried scapulae, the distribution of acromial types was as follows: type I, flat, 51 (12.1%); type II, curved, 239 (56.5%); type III, hooked, 122 (28.8%); and type IV, convex, 11 (2.6%) [39]. Types II and III are known to be associated with increased incidence of the clinical syndrome of impingement [28, 38]. Hyvonen et al. [40] reported that the thickness of the anterior part of the acromion at the tendinitis stage of impingement and the acromial angle at the tear stage of impingement were important parameters that differentiated patients with different stages of rotator cuff pathology from the age- and sex-matched control group. While the positive correlation between acromion morphology and nontraumatic cuff pathology has been well established [41], the shape of acromion process does not appear to change with age in individuals without rotator cuff pathology [42].

Subacromial Osseous Impingement

Subacromial osseous impingement is common in patients with advanced rotator cuff impingement. Unfortunately, the terms “osteophyte,” “enthesophyte,” and “traction spur” have been used interchangeably which can cause confusion in understanding the origin of these structures adding to ambiguity in the literature. In short, osteophytes are bony outgrowths originating from the periosteum at the junction of an articular cartilage, where an enthesophyte is a thickened tendon or ligament at its bony attachment with associated calcification (mineralization) and ossification. Traction spurs are often referred to enthesophytes. See Page 20 on coracoacromial

ligament pathology in this chapter and Chap. 11 for more details on the difference between osteophyte and enthesophyte.

Neer [4] reported a rate of 11% spurs or osteophytes on the anteroinferior surface of the acromion in cadavers of unknown age. Nicholson et al. [43] reported that the specimens taken from patients <50 years of age had significantly less osteophytes as compared to the specimens of patients >50 years of age (7% vs. 30%). Mahakkanukrauh and Surin [29] found osteophytes on 28.9% of acromions of cadavers from Thailand (11.2% on the younger age groups and 34.7% on the older age groups). All three groups of investigators reported that most acromial osteophytes were located on the anteroinferior surface of the acromion. Mahakkanukrauh and Surin [29] speculated that the clustering of osteophytes on the anteroinferior surface of the acromion indicates reactive or degenerative process resulted from impingement on the bone.

Different acromial osteophytes/enthesophytes have been described by different investigators. Mahakkanukrauh and Surin [29] used the term osteophyte for all osseous abnormalities and classified them as (1) traction spur (straight) and (2) claw osteophytes (curved or hooked) with traction-type osteophytes being more prevalent (87%). Considering the claw-type osteophytes were more detected in older specimens, the authors felt that the claw-type spurs were indicative of more severe degenerative process and, consequently, greater risk of impingement and tear of the rotator cuff. A more complex morphological classification of acromial spurs was described by Oh et al. [31]. The authors described six types: heel, lateral traction, anterior traction, lateral bird beak, anterior bird beak, and medial spur. The overall rate of acromial spurs was 68%, and their incidence increased with age. In this study, spurs were more frequent in patients with full-thickness cuff tears than in the control group [31]. However, they were not related with the size or retraction of the cuff tears. The heel-type spur was the most common and was observed more frequently in the full-thickness tear group than in the control group.

As noted, apart from osseous impingement caused by osteophytes, the subacromial area can be narrowed by ligamentous degenerative changes referred to as enthesophytes. In a large study of dried scapulae in Germany, all enthesophytes (of 423 cases) were localized at the site of the coracoacromial ligament (CAL) insertion on the acromion. Enthesophytes were significantly more common in type III acromions. In in vivo studies, the location of the enthesophytes at the insertion of the coracoacromial ligament has been confirmed [44, 45].

The conservative management of the osseous impingement is limited to avoiding repetitive overhead activities, strengthening exercises of the superior and posterior cuff muscles, and cortisone injection. In cases where the osseous spurs are large, surgery may be considered to stop further damage to the vulnerable cuff tendons. Surgical management of impingement syndrome of ossific origin involves anterior acromioplasty and acromioclavicular arthroplasty when indicated, to correct impingement by decompressing the subacromial space. Anterior acromioplasty involves debridement of the inflamed subacromial bursa, resection of the anteroinferior aspect of the acromion, resection of overhanging osteophytes from the AC joint, and sometimes resection of the CAL.

Acromioclavicular Joint (AC) Pathologies

Osteoarthritis

The AC joint arthritis is known to contribute to subacromial impingement particularly with advancing age and development of inferior osteophytes. The osteophytes of the AC joint are usually related to predisposing factors such as trauma, rheumatoid arthritis, or osteoarthritis [29]. The nontraumatic osteophytes are part of degenerative process and appear to increase in size and number in consequence to repetitive impingement and increasing age [43]. The fact that majority of people are right-handed and the osteophytes are found more frequently on the right than the left side may support a reactive process theory [29].

In terms of management, most patients with age-related AC joint arthritis have no symptoms. Those who have significant inferior osteophytes will benefit from decompression of the subacromial area. There is controversy about surgical management of those without osteophytes. Kim et al. [46] demonstrated that despite the absence of pain or tenderness in the AC joint and presence of a negative cross-body adduction and negative lidocaine test, better functional outcomes were achieved with distal clavicle resection. Razmjou et al. [47] showed that distal clavicle resection was an independent predictor of outcome at 2 years following surgery and even minor arthritic changes responded well to resection of the distal clavicle. Before any attempt to distal clavicle resection, a proper rehabilitation and corticosteroid injection should be tried as most patients respond well to conservative management.

Os Acromiale

Os acromiale is an unfused epiphysis of the anterior part of the acromion. The incomplete ossification may take as late as 25 years of age and is considered pathologic only after this age [48]. According to Sammarco's review, Gruber has been credited for first describing this entity in 1859 [49]. The frequency of os acromiale is reported at 8% in general population by Sammarco [49] who examined over 1000 human skeletons with bilateral involvement of 33%. Hunt and Bullen [50] reported a similar prevalence of the overall 8% in a large sample of 1594 skeletons. Os acromiale appears to be more frequent in blacks than in whites and more frequent in males than in females [50]. According to a systematic review and meta-analysis of 23 studies in 2014 [51], persons of black ancestry had a significantly higher frequency of os acromiale than persons of white, Native American, and Middle Eastern ancestries. Similarly, individuals of black ancestry had a higher prevalence of bilateral os acromiale. The review did not find significant interactions between presence of os acromiale and sex and involved side. The review supported a genetic basis for this condition rather than the mechanical trauma-induction hypothesis [51].

The os acromiale may not always predispose the supraspinatus and/or infraspinatus tendons to tears. However, individuals with the step-off deformity of an os acromiale are reported to be at a greater risk of rotator cuff impingement and tear than individuals without such deformity [52]. Os acromiale can be easily diagnosed with plain radiographs with at least two views (AP, axillary views). The US can confirm the diagnosis in the equivocal cases.

Treatment of symptomatic os acromiale should initially be nonsurgical. Medication in conjunction with physiotherapy, followed by subacromial and nonunion site corticosteroid injection, also may be used to relieve symptoms. Surgical treatment involves excision of the impinging fragment. The open reduction internal fixation (with or without bone grafting) is indicated when the nonhealing site is unstable and painful. The arthroscopic subacromial decompression with acromioplasty or rotator cuff repair should address the concomitant pathology [53, 54].

Osteolysis of the Distal Clavicle

While osteolysis of the distal clavicle is not a cause of impingement, it has been discussed in this section in relation to structural changes of the subacromial area that could cause anterior shoulder pain. The word “osteolysis” refers to a softening, absorption, and dissolution of the bone or the removal or loss of calcium in the bone. Osteolysis of the distal clavicle is a pathologic process involving resorption of subchondral bone in the distal clavicle [55]. This condition is limited to clavicular component of the AC joint [56] and usually presents as a pain and tenderness localized to the AC joint.

The earliest reference to this condition was published in 1936 by Dupas and colleagues [56–58]. In 1982, Cahill [57] confirmed a link between weight lifting associated with repetitive microtrauma as an etiology of this condition. Cahill [57] noted the presence of microfractures in the subchondral bone in 50% of his cases and proposed that repetitive microtrauma caused subchondral stress fractures and remodeling. In 1986, Brunet et al. [59] reported that most specimens with osteolysis showed disruption of articular cartilage, subchondral cyst formation, and evidence of increased osteoclastic activity. Hypertrophic synovial tissue that migrated across the articular cartilage and invaded subchondral bone has been reported as a potential cause in some cases. Sanders et al. [60] have summarized the potential causes of resorption of the distal clavicle as infection, metabolic, inflammatory, autoimmune, and post-traumatic. More recent studies indicate a higher incidence of osteolysis with repetitive microtrauma (56%) compared with one single trauma (44%) [61].

Clinically, most patients describe a dull ache over the distal end of the clavicle and AC joint. There may be a history of a specific event or repetitive/overuse injuries. Symptoms are aggravated by overhead sports or work activities. Clinical examinations show a full range of motion of the shoulder with normal strength in rotator cuff muscles with a localized tenderness over the AC joint and exacerbation of pain with stressing the AC joint with a positive cross-chest test. As distal clavicle osteolysis is usually a unilateral condition, inflammatory disease should be considered in bilateral cases. This condition can be reversible in some cases with activity modification. Radiological findings are consistent with resorption of the distal clavicle, osteopenia of the distal clavicle, periarticular swelling, and periarticular cyst-like erosions (see Chap. 11 for more details).

In terms of surgical management in persistent cases, arthroscopic resection of the distal clavicle may be considered. Patients with a traumatic etiology are reported to have a slightly worse result compared with patients with a microtraumatic etiology [61].

Coracoacromial Ligament (CAL) Pathology

The coracoacromial ligament (CAL) joins the coracoid process of the scapula to acromion. The CAL is an important static restraint to superior humeral head displacement and an integral component of the coracoacromial arch. According to a review by Rothenberg et al. [62], the CAL plays an important role in shoulder stability via both static restraint and dynamic proprioceptive signals through interactions with other shoulder capsular elements including ligaments, muscles, and osseous structures. The subdeltoid bursa, supraspinatus muscle and tendon, and the long head of the biceps tendon are within the coracoacromial arch and may be affected by the CAL's pathology. It appears that Neer was the first to indicate that CAL could be a contributing extrinsic factor to the pain associated with motion of the rotator cuff against the coracoacromial arch [63].

In a study by Uhthoff et al. [33], the subacromial space under the coracoacromial ligament appeared unusually tight and barely accessible in patients with clinical signs of impingement. Histological examination of the biopsied ligament of these patients showed degenerative changes but no increase in fibrous tissue. The authors concluded that in the absence of bony overgrowth, the initial change was an increased volume of the soft tissues in the subacromial space. In another study by Ogata and Uhthoff in 1990 [44], the radiological and histological changes of the CAL at its insertion into the undersurface of the acromion were studied in 76 autopsy specimens. The authors described two changes, a downward bony projection of the acromion, which might reduce the height of the subacromial compartment, and a thickened layer of fibrocartilage, constituting a potential cause for narrowing of the subacromial space. The authors felt that bony projection could act as a predisposing factor for the impingement syndrome, whereas the thickened ligament could develop in response to pressure from constituents of the subacromial compartment.

The subtle abnormalities of CAL are usually not seen on plain radiographs [33], but a standard anterior-posterior view and lateral scapular view can detect enthesophyte formation within the CAL, which is the sign of pathologic degeneration [64]. The enthesophytes have been shown to form within the substance of the CAL, particularly at its acromial insertion [62]. Reichmister et al. suggested that the presence of enthesophytes within the CAL may be indicative of rotator cuff pathology [65].

The MRI of patients with coracoacromial impingement may show thickening and irregularity in the ligament which indicates pressure on the supraspinatus tendon.

With respect to management of the enthesopathic changes of the CAL, conservative treatment should be exhausted before surgery is considered. There is considerable controversy about CAL's surgical management during acromioplasty [62]. While full resection of the CAL could affect the glenohumeral stability, partial resection may avoid postoperative complications related to its release. There are limited reports that the CAL may regenerate spontaneously at the periosteum of the acromion after partial resection with the same orientation, gross appearance, and viscoelastic properties within a few years of release [66, 67].

Coracoid Impingement Syndrome (CIS)

The coracoid impingement was first described by Goldthwait as a possible cause of rotator cuff impingement in 1909 [68]. Gerber [36, 69] reported three types of coracoid impingement: idiopathic, iatrogenic (e.g., Bristow anterior stabilization or posterior glenoid osteotomy), and traumatic (e.g., displaced fractures or malunion). The idiopathic coracoid impingement is related to a malpositioned coracoid tip.

Symptoms are related to the impingement of the subscapularis tendon between the coracoid and lesser tuberosity of the humerus when the shoulder is in the combined position of flexion, adduction, and internal rotation [68]. Gaskill et al. reported that other structures such as long head of the biceps, anterosuperior cuff (supraspinatus and subscapularis), and rotator interval may be involved as well [70].

An increase in size and a change in the shape of the coracoid process may be difficult to be evaluated by simple radiographic examination, but plain radiographs, in the AP and axillary views, can detect marked anatomical abnormalities of the coracoid process. The MRI can provide more information on all structures of the subcoracoid area, specifically subscapularis tendon.

The initial treatment of the CIS is conservative with a trial of anti-inflammatory medication and physiotherapy to strengthen rotator cuff muscles and scapular stabilizer musculature. Activity modification and avoiding forward flexion, internal rotation, and adduction are recommended. Coracoplasty (excision of the posterolateral border of the coracoid process) is indicated in patients who are refractory to those treatments.

Clinical Findings of Primary Impingement Syndrome

Primary impingement syndrome is a common problem of the shoulder with a similar presentation to partial-thickness and minor rotator cuff tears. History and symptom duration are not typical and cannot help the clinician to accurately differentiate between an impingement syndrome and minor rotator cuff tear. Most patients have a past history of overhead or repetitive activities. Single events may play a part if they involve a certain strenuous arm movement in younger individuals. Night pain can be a major problem in chronic cases of impingement particularly degenerative type and acute calcified tendinitis. Range of motion is well maintained, often associated with a painful arch syndrome, and strength is pain-inhibited but within a normal range.

The shoulder impingement tests that are commonly used in clinical practice are the Neer [4] and Hawkins tests [71]. In an in vivo magnetic resonance imaging (MRI) study, the rotator cuff insertion appeared to be closest to the acromion not at full elevation (Neer sign position) but at 90° of flexion (Hawkins sign position) [72]. In an anatomical study where magnetic resonance imaging was used to examine the subacromial and intra-articular contact of the rotator cuff during the Neer and Hawkins, the authors found that both maneuvers significantly decreased the distance from the supraspinatus insertion to the acromion and posterior glenoid and from the subscapularis insertion to the anterior glenoid [73]. Similarly, Yamamoto et al. [74] found that the contact sites of the coracoacromial arch side were common in both signs; however, those of the humeral head side were anatomically different. Although these findings confirm an anatomical contact during application of these tests, they do not necessarily indicate that these tests help clinicians to rule in or rule out the impingement syndrome. Reliability [75] and validity [76] of these two tests are reported to be poor. Razmjou and Holtby [76] indicated that pain provocation tests that are purely based on production of pain have a limited value in clinical practice. Overall, Neer and Hawkins tests had good sensitivity but low specificity for subacromial impingement syndrome [77].

Internal Impingement Syndrome

The internal impingement syndrome or posterior superior glenoid impingement occurs between the greater tuberosity (supraspinatus and/or infraspinatus) with the posterior-superior glenoid rim in young and active athletes secondary to labral and capsular adaptation during extreme and repetitive external rotation and abduction (e.g., pitching or throwing). The concept of internal impingement was first described by Frank Jobe, an American orthopedic surgeon in 1989, at the fourth Congress of the European Society for Surgery of the Shoulder and Elbow annual meeting. Around the same time, a French orthopedic surgeon, Gil Walch, and his colleagues [1] proposed the same concept under the same terminology. During the arthroscopic assessment of 17 athletes with shoulder pain from 1989 to 1991, Walch noticed impingement of the posterosuperior edge of the glenoid and the articular aspect of rotator cuff when the arm was positioned in the throwing position (90 degrees of abduction and full external rotation). Walch felt that although this impingement was physiological, the repetitive contact of the posterior superior aspect of the glenoid labrum with undersurface fibers of the cuff would lead to mechanical damage and lesions. Jobe described a clinical test, the relocation test for confirming internal impingement [2]. This test is performed with the patient in supine position with the arm in 90/90 position [78]. The examiner applies a passive overpressure in the maximally externally rotated position. The examiner then applies a posteriorly directed pressure to the humeral head. The test is considered positive if pain is reduced or eliminated with the posterior translation of the humeral head. Jobe proposed that a posteriorly directed force would decompress the impinged rotator cuff and the posterior glenoid [78]. As noted, this impingement is common in young athletes as a result of repetitive high-velocity throwing actions, during which certain osseous and soft tissue adaptations may occur. Over time, as a result of adaptive changes and excessive external rotation, glenohumeral internal rotation deficit (GIRD), contracture of the posterior capsule, reduced humeral retroversion, acquired glenohumeral anterior/posterior instability, scapular dyskinesis, and rotator cuff weakness will take place [79–81].

The associated findings of internal impingement are tearing and degeneration of articular fibers of the supraspinatus, tearing and degeneration of articular fibers of the infraspinatus, abnormality of posterosuperior glenoid labrum (SLAP lesions), humeral head impaction or subcortical humeral head cysts, anterior capsule laxity, and posterior capsule thickening [82–84].

Clinical Findings and Management of Internal Impingement Syndrome

Internal impingement is often seen in young athletes involved in overhead sports. There is often no specific single injury and pain appears to have developed over a period of time. The patient is often muscular and fit with no obvious abnormalities on observation with the dominant arm being more affected. Symptoms are characterized by a deep and diffuse posterior shoulder pain felt during extreme external rotation and abduction of the shoulder when the athlete aims to hit a ball with high velocity and accuracy (e.g., baseball, tennis, volleyball). There may be a vague radiating pain to the arm. Presence of numbness or paresthesia is rare in isolated internal impingement syndrome. Range of motion in the dominant arm of an overhead athlete has a specific pattern of restriction and laxity with the affected arm showing an increase in external rotation and decrease in internal rotation at 90 degrees of abduction. The tight posterior capsule however is not considered pathologic as long as the total arc of rotation approximates 180 degrees. Manske and colleagues recommend looking for a sulcus sign and performing anterior/posterior laxity test to rule out subtle inferior and anterior laxity of the glenohumeral joint, respectively [81]. Patients with internal impingement often have increased pain during the anterior and posterior load and shift test performed in supine position with the arm in 90/90 position. This test is positive if the pain is felt/increased with a passive anteriorly directed overpressure of the examiner to the maximally externally rotated arm. The relocation component of the same test involves anteriorly directed pressure that reduces the pain.

In terms of radiological assessment of internal impingement, ultrasound is often inconclusive, and MRI may have difficulty to diagnose subtle labral pathologies. MRI with enhanced gadolinium contrast is the gold standard for imaging of an athlete's shoulder. Common findings are delamination of the rotator cuff tendons, articular-sided partial-thickness tears, and labral tears with paralabral cysts. Although many of these findings may not be related to patient's symptoms, as have been reported in asymptomatic athletes.

Conservative management of pain, stretching of the posterior capsule, and a muscle strengthening program should have failed before surgical treatment is considered. Considering the importance of harmony between dynamic and static stabilizers of the shoulder in these patients, rehabilitation for acquired/adapted instability is focused on improving rotator cuff and scapular strength, endurance, and neuromuscular control. Surgery is indicated only in those with frank instability or major structural pathology. Treatment of articular-sided partial-thickness rotator cuff tears and minor labral lesions remains controversial in athletes.

Secondary and Functional Impingement Syndrome

Secondary and functional impingement syndromes affect the position of the humeral head in the glenoid and are nonstructural and adaptive similar to the early stages of internal impingement syndrome. Similar to internal impingement syndrome, they are common in high-velocity sports activities and may occur in patients with chronic muscle fatigue and imbalance or noncontractile tissue tightness. The term secondary impingement was first suggested by Frank Jobe, an American orthopedic surgeon who discussed the concept of rotator cuff impingement secondary to an occult anterior glenohumeral subluxation in overhead athletes [2]. Frank Jobe was the father of Christopher Jobe who proposed the concept of internal impingement around the same time [78].

Functional impingement is a dynamic phenomenon and is caused by the proximal translation of the humeral head during abduction or flexion [3, 85, 86]. Repeated overhead activities could attenuate the static restraints and fatigue the dynamic restrains such as muscles and cause subtle instability. Subsequently, the humeral head would contact with the coracoacromial arch, ultimately leading to impingement. Tension overload, muscle weakness, and imbalance may contribute to relative loss of subacromial space secondary to altered scapulohumeral mechanics causing functional impingement of the rotator cuff tendons. Weakness of the lower and middle trapezius, serratus anterior, infraspinatus, and deltoid, coupled with tightness of the upper trapezius, pectorals, and levator scapula, affects the normal pattern of scapular movements, thereby reducing the subacromial space [3, 87]. This may explain why some patients with rotator cuff pathology respond well to proper rehabilitation that focuses on strengthening exercises and restoration of normal neuromuscular function.

Muscle imbalances between the deltoid and rotator cuff can also affect the integrity of the force couple and cause compression within the cuff tendons [88, 89]. In the absence of compressive forces of rotator cuff and the upwardly directed shear forces of the deltoid, the humeral head is superiorly migrated at the initiation of abduction, creating a dynamic impingement syndrome. In patients with chronic rotator cuff pathology, the superior migration becomes more permanent (see Chap. 11). Treatment involves rehabilitation of superior and mostly posterior rotator cuff muscles to better improve the position of the humeral head in the glenoid.

Apart from contractile muscle imbalance, the imbalance in the capsule often seen with overstretched ligaments of the glenohumeral joint such as the posterior band of the inferior glenohumeral ligament (IGHL) contributes to altered shoulder kinematics. For example, increased external rotation and internal rotation deficit are common phenomena in overhead athletes with impingement syndrome. Excessive external rotation leads to increased anterior and inferior translation of the humerus, leading to anterior instability. Similarly, loss of internal rotation commonly associated with posterior capsular tightness could cause superior and anterior translation of the humeral head [90–92]. Treatment of the functional impingement includes stretching of posterior capsule and strengthening of the anterosuperior and scapular stabilizers. As noted in the treatment section of internal impingement, surgery is indicated to address structural abnormalities and has little value in functional impingement.

References

1. Walch G, Boileau P, Noel E, Donell ST. Impingement of the deep surface of the supraspinatus tendon on the posterosuperior glenoid rim: An arthroscopic study. J Shoulder Elb Surg. 1992;1(5):238–45.
2. Jobe FW, Kvitne RS, Giangarra CE. Shoulder pain in the overhand or throwing athlete. The relationship of anterior instability and rotator cuff impingement. Orthop Rev. 1989;18(9):963–75.
3. Page P. Shoulder muscle imbalance and subacromial impingement syndrome in overhead athletes. Int J Sports Phys Ther. 2011;6(1):51–8.
4. Neer CS 2nd. Anterior acromioplasty for the chronic impingement syndrome in the shoulder: a preliminary report. J Bone Joint Surg Am. 1972;54(1):41–50.
5. Neer CS 2nd. Impingement lesions. Clin Orthop Relat Res. 1983;173:70–7.
6. Bigliani LU, Levine WN. Subacromial impingement syndrome. J Bone Joint Surg Am. 1997;79(12):1854–68.
7. Soslowsky LJ, Thomopoulos S, Esmail A, Flanagan CL, Iannotti JP, Williamson JD 3rd, et al. Rotator cuff tendinosis in an animal model: role of extrinsic and overuse factors. Ann Biomed Eng. 2002;30(8):1057–63.
8. Cicak N, Klobucar H, Maric D. Overuse injury syndromes in the shoulder area. Arh Hig Rada Toksikol. 2001;52(4):393–402.
9. Cowderoy GA, Lisle DA, O'Connell PT. Overuse and impingement syndromes of the shoulder in the athlete. Magn Reson Imaging Clin N Am. 2009;17(4):577-93. v
10. Ark JW, Flock TJ, Flatow EL, Bigliani LU. Arthroscopic treatment of calcific tendinitis of the shoulder. Arthroscopy. 1992;8(2):183–8.

11. Gartner J, Simons B. Analysis of calcific deposits in calcifying tendinitis. Clin Orthop Relat Res. 1990;254:111–20.
12. Painter CF. Subdeltoid bursitis. Boston Med Surg. 1907;156:345–9.
13. Re LP Jr, Karzel RP. Management of rotator cuff calcifications. Orthop Clin North Am. 1993;24(1):125–32.
14. Riley GP, Harrall RL, Constant CR, Cawston TE, Hazleman BL. Prevalence and possible pathological significance of calcium phosphate salt accumulation in tendon matrix degeneration. Ann Rheum Dis. 1996;55(2):109–15.
15. Uhthoff HK, Loehr JW. Calcific tendinopathy of the rotator cuff: pathogenesis, diagnosis, and management. J Am Acad Orthop Surg. 1997;5(4):183–91.
16. Carli DEA, Pulcinelli F, Rose GD, Pitino D, Ferretti A. Calcific tendinitis of the shoulder. Joints. 2014;2(3):130–6.
17. Mavrikakis ME, Drimis S, Kontoyannis DA, Rasidakis A, Moulopoulou ES, Kontoyannis S. Calcific shoulder periarthritis (tendinitis) in adult onset diabetes mellitus: a controlled study. Ann Rheum Dis. 1989;48(3):211–4.
18. Sengar DP, McKendry RJ, Uhthoff HK. Increased frequency of HLA-A1 in calcifying tendinitis. Tissue Antigens. 1987;29(3):173–4.
19. Hurt G, Baker CL Jr. Calcific tendinitis of the shoulder. Orthop Clin North Am. 2003;34(4):567–75.
20. Darrieutort-Laffite C, Blanchard F, Le Goff B. Calcific tendonitis of the rotator cuff: from formation to resorption. Joint Bone Spine. 2018;85(6):687–92.
21. Faure G, Daculsi G. Calcified tendinitis: a review. Ann Rheum Dis. 1983;42(Suppl 1):49–53.
22. Gosens T, Hofstee DJ. Calcifying tendinitis of the shoulder: advances in imaging and management. Curr Rheumatol Rep. 2009;11(2):129–34.
23. del Cura JL, Torre I, Zabala R, Legorburu A. Sonographically guided percutaneous needle lavage in calcific tendinitis of the shoulder: short- and long-term results. AJR Am J Roentgenol. 2007;189(3):W128–34.
24. Lee SY, Cheng B, Grimmer-Somers K. The midterm effectiveness of extracorporeal shockwave therapy in the management of chronic calcific shoulder tendinitis. J Shoulder Elb Surg. 2011;20(5):845–54.
25. Merolla G, Bhat MG, Paladini P, Porcellini G. Complications of calcific tendinitis of the shoulder: a concise review. J Orthop Traumatol. 2015;16(3):175–83.
26. Verstraelen FU, Fievez E, Janssen L, Morrenhof W. Surgery for calcifying tendinitis of the shoulder: a systematic review. World J Orthop. 2017;8(5):424–30.
27. Bigliani LU, Ticker JB, Flatow EL, Soslowsky LJ, Mow VC. Relationship of acromial architecture and diseases of the rotator cuff. Orthopade. 1991;20(5):302–9.
28. Gill TJ, McIrvin E, Kocher MS, Homa K, Mair SD, Hawkins RJ. The relative importance of acromial morphology and age with respect to rotator cuff pathology. J Shoulder Elb Surg. 2002;11(4):327–30.
29. Mahakkanukrauh P, Surin P. Prevalence of osteophytes associated with the acromion and acromioclavicular joint. Clin Anat. 2003;16(6):506–10.
30. Chambler AF, Pitsillides AA, Emery RJ. Acromial spur formation in patients with rotator cuff tears. J Shoulder Elb Surg. 2003;12(4):314–21.
31. Oh JH, Kim JY, Lee HK, Choi JA. Classification and clinical significance of acromial spur in rotator cuff tear: heel-type spur and rotator cuff tear. Clin Orthop Relat Res. 2010;468(6):1542–50.
32. Petersson CJ, Gentz CF. Ruptures of the supraspinatus tendon. The significance of distally pointing acromioclavicular osteophytes. Clin Orthop Relat Res. 1983;174:143–8.
33. Uhthoff HK, Hammond DI, Sarkar K, Hooper GJ, Papoff WJ. The role of the coracoacromial ligament in the impingement syndrome. A clinical, radiological and histological study. Int Orthop. 1988;12(2):97–104.
34. Soslowsky LJ, An CH, Johnston SP, Carpenter JE. Geometric and mechanical properties of the coracoacromial ligament and their relationship to rotator cuff disease. Clin Orthop Relat Res. 1994;304, 10:–17.

35. Sarkar K, Taine W, Uhthoff HK. The ultrastructure of the coracoacromial ligament in patients with chronic impingement syndrome. Clin Orthop Relat Res. 1990;254:49–54.
36. Gerber C, Terrier F, Ganz R. The role of the coracoid process in the chronic impingement syndrome. J Bone Joint Surg Br. 1985;67(5):703–8.
37. Bigliani L, Morrison DS, April EW. The morphology of the acromion and its relationship to rotator cuff tears. Orthop Trans. 1986;10:228.
38. Vanarthos WJ, Monu JU. Type 4 acromion: a new classification. Contemp Orthop. 1995;30(3):227–9.
39. Natsis K, Tsikaras P, Totlis T, Gigis I, Skandalakis P, Appell HJ, et al. Correlation between the four types of acromion and the existence of enthesophytes: a study on 423 dried scapulas and review of the literature. Clin Anat. 2007;20(3):267–72.
40. Hyvonen P, Paivansalo M, Lehtiniemi H, Leppilahti J, Jalovaara P. Supraspinatus outlet view in the diagnosis of stages II and III impingement syndrome. Acta Radiol. 2001;42(5):441–6.
41. Morelli KM, Martin BR, Charakla FH, Durmisevic A, Warren GL. Acromion morphology and prevalence of rotator cuff tear: a systematic review and meta-analysis. Clin Anat. 2019;32(1):122–30.
42. Vahakari M, Leppilahti J, Hyvonen P, Ristiniemi J, Paivansalo M, Jalovaara P. Acromial shape in asymptomatic subjects: a study of 305 shoulders in different age groups. Acta Radiol. 2010;51(2):202–6.
43. Nicholson GP, Goodman DA, Flatow EL, Bigliani LU. The acromion: morphologic condition and age-related changes. A study of 420 scapulas. J Shoulder Elb Surg. 1996;5(1):1–11.
44. Ogata S, Uhthoff HK. Acromial enthesopathy and rotator cuff tear. A radiologic and histologic postmortem investigation of the coracoacromial arch. Clin Orthop Relat Res. 1990;254:39–48.
45. Ozaki J, Fujimoto S, Nakagawa Y, Masuhara K, Tamai S. Tears of the rotator cuff of the shoulder associated with pathological changes in the acromion. A study in cadavera. J Bone Joint Surg Am. 1988;70(8):1224–30.
46. Kim J, Chung J, Ok H. Asymptomatic acromioclavicular joint arthritis in arthroscopic rotator cuff tendon repair: a prospective randomized comparison study. Arch Orthop Trauma Surg. 2011;131(3):363–9.
47. Razmjou H, ElMaraghy A, Dwyer T, Fournier-Gosselin S, Devereaux M, Holtby R. Outcome of distal clavicle resection in patients with acromioclavicular joint osteoarthritis and full-thickness rotator cuff tear. Knee Surg Sports Traumatol Arthrosc. 2014;
48. McClure JG, Raney RB. Anomalies of the scapula. Clin Orthop Relat Res. 1975;110:22–31. doi(110):22-31
49. Sammarco VJ. Os acromiale: frequency, anatomy, and clinical implications. J Bone Joint Surg Am. 2000;82(3):394–400.
50. Hunt DR, Bullen L. Frequency of Os Acromiale in the Robert. J. Terry Collection. Int J Osteoarchaeol. 2007;17:309–17.
51. Yammine K. The prevalence of Os acromiale: a systematic review and meta-analysis. Clin Anat. 2014;27(4):610–21.
52. Ouellette H, Thomas BJ, Kassarjian A, Fritz B, Tetreault P, Palmer WE, et al. Re-examining the association of os acromiale with supraspinatus and infraspinatus tears. Skelet Radiol. 2007;36(9):835–9.
53. You T, Frostick S, Zhang WT, Yin Q. Os Acromiale: reviews and current perspectives. Orthop Surg. 2019;11(5):738–44.
54. Johnston PS, Paxton ES, Gordon V, Kraeutler MJ, Abboud JA, Williams GR. Os acromiale: a review and an introduction of a new surgical technique for management. Orthop Clin North Am. 2013;44(4):635–44.
55. Schwarzkopf R, Ishak C, Elman M, Gelber J, Strauss DN, Jazrawi LM. Distal clavicular osteolysis: a review of the literature. Bull NYU Hosp Jt Dis. 2008;66(2):94–101.
56. Levine AH, Pais MJ, Schwartz EE. Posttraumatic osteolysis of the distal clavicle with emphasis on early radiologic changes. AJR Am J Roentgenol. 1976;127(5):781–4.
57. Cahill BR. Osteolysis of the distal part of the clavicle in male athletes. J Bone Joint Surg Am. 1982;64(7):1053–8.

58. Stephens M, Wolin PM, Tarbet JA, Alkhayarin M. Osteolysis of the distal clavicle: readily detected and treated shoulder pain. Phys Sportsmed. 2000;28(12):35–44.
59. Brunet ME, Reynolds MC, Cook SD, Brown TW. Atraumatic osteolysis of the distal clavicle: histologic evidence of synovial pathogenesis. A case report Orthopedics. 1986;9(4):557–9.
60. Sanders TG, Jersey SL. Conventional radiography of the shoulder. Semin Roentgenol. 2005;40(3):207–22.
61. Zawadsky M, Marra G, Wiater JM, Levine WN, Pollock RG, Flatow EL, et al. Osteolysis of the distal clavicle: long-term results of arthroscopic resection. Arthroscopy. 2000;16(6):600–5.
62. Rothenberg A, Gasbarro G, Chlebeck J, Lin A. The coracoacromial ligament: anatomy, function, and clinical significance. Orthop J Sports Med. 2017;5(4):2325967117703398.
63. Neer CS 2nd. Anterior acromioplasty for the chronic impingement syndrome in the shoulder. 1972. J Bone Joint Surg Am. 2005;87(6):1399.
64. Kelly BT, Dunn WR, Ramirez L, Warren RF. Traumatic rupture of the coracoacromial ligament. Arthroscopy. 2005;21(6):763.
65. Reichmister JP, Reeder JD, McCarthy E. Ossification of the coracoacromial ligament: association with rotator cuff pathology of the shoulder. Md Med J. 1996;45(10):849–52.
66. Hansen U, Levy O, Even T, Copeland SA. Mechanical properties of regenerated coracoacromial ligament after subacromial decompression. J Shoulder Elb Surg. 2004;13(1):51–6.
67. Levy O, Copeland SA. Regeneration of the coracoacromial ligament after acromioplasty and arthroscopic subacromial decompression. J Shoulder Elb Surg. 2001;10(4):317–20.
68. Anetzberger H, Putz R. Morphometry of the sub-acromial space and its clinical relevance. Unfallchirurg. 1995;98(8):407–14.
69. Gerber C, Terrier F, Zehnder R, Ganz R. The subcoracoid space. An anatomic study. Clin Orthop Relat Res. 1987;215:132–8.
70. Gaskill TR, Braun S, Millett PJ. Multimedia article. The rotator interval: pathology and management. Arthroscopy. 2011;27(4):556–67.
71. Hawkins RJ, Kennedy JC. Impingement syndrome in athletes. Am J Sports Med. 1980;8(3):151–8.
72. Roberts CS, Davila JN, Hushek SG, Tillett ED, Corrigan TM. Magnetic resonance imaging analysis of the subacromial space in the impingement sign positions. J Shoulder Elb Surg. 2002;11(6):595–9.
73. Pappas GP, Blemker SS, Beaulieu CF, McAdams TR, Whalen ST, Gold GE. In vivo anatomy of the Neer and Hawkins sign positions for shoulder impingement. J Shoulder Elb Surg. 2006;15(1):40–9.
74. Yamamoto N, Muraki T, Sperling JW, Steinmann SP, Itoi E, Cofield RH, et al. Impingement mechanisms of the Neer and Hawkins signs. J Shoulder Elb Surg. 2009;18(6):942–7.
75. Norregaard J, Krogsgaard MR, Lorenzen T, Jensen EM. Diagnosing patients with longstanding shoulder joint pain. Ann Rheum Dis. 2002;61(7):646–9.
76. Razmjou H, Holtby R, Myhr T. Pain provocative shoulder tests: reliability and validity of the impingement tests. Physiother Can. 2004;56(4):229–36.
77. Beaudreuil J, Nizard R, Thomas T, Peyre M, Liotard JP, Boileau P, et al. Contribution of clinical tests to the diagnosis of rotator cuff disease: a systematic literature review. Joint Bone Spine. 2009;76(1):15–9.
78. Jobe CM. Superior glenoid impingement. Current concepts. Clin Orthop Relat Res. 1996;330:98–107.
79. Castagna A, Garofalo R, Cesari E, Marcopoulos N, Borroni M, Conti M. Anterior and posterior internal impingement: an evidence-based review. Br J Sports Med. 2010;44(5):382–8.
80. Chambers L, Altchek DW. Microinstability and internal impingement in overhead athletes. Clin Sports Med. 2013;32(4):697–707.
81. Manske RC, Grant-Nierman M, Lucas B. Shoulder posterior internal impingement in the overhead athlete. Int J Sports Phys Ther. 2013;8(2):194–204.
82. Giaroli EL, Major NM, Higgins LD. MRI of internal impingement of the shoulder. AJR Am J Roentgenol. 2005;185(4):925–9.

83. Kaplan LD, McMahon PJ, Towers J, Irrgang JJ, Rodosky MW. Internal impingement: findings on magnetic resonance imaging and arthroscopic evaluation. Arthroscopy. 2004;20(7):701–4.
84. Halbrecht JL, Tirman P, Atkin D. Internal impingement of the shoulder: comparison of findings between the throwing and nonthrowing shoulders of college baseball players. Arthroscopy. 1999;15(3):253–8.
85. Brossmann J, Preidler KW, Pedowitz RA, White LM, Trudell D, Resnick D. Shoulder impingement syndrome: influence of shoulder position on rotator cuff impingement—an anatomic study. AJR Am J Roentgenol. 1996;167(6):1511–5.
86. Deutsch A, Altchek DW, Schwartz E, Otis JC, Warren RF. Radiologic measurement of superior displacement of the humeral head in the impingement syndrome. J Shoulder Elb Surg. 1996;5(3):186–93.
87. Poppen NK, Walker PS. Forces at the glenohumeral joint in abduction. Clin Orthop Relat Res. 1978;135:165–70.
88. Burkhart SS, Esch JC, Jolson RS. The rotator crescent and rotator cable: an anatomic description of the shoulder's "suspension bridge". Arthroscopy. 1993;9(6):611–6.
89. Burkhart SS. Fluoroscopic comparison of kinematic patterns in massive rotator cuff tears. A suspension bridge model. Clin Orthop Relat Res. 1992;284:144–52.
90. Burkhart SS, Morgan CD, 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.
91. Kugler A, Kruger-Franke M, Reininger S, Trouillier HH, Rosemeyer B. Muscular imbalance and shoulder pain in volleyball attackers. Br J Sports Med. 1996;30(3):256–9.
92. Myers JB, Laudner KG, Pasquale MR, Bradley JP, Lephart SM. Glenohumeral range of motion deficits and posterior shoulder tightness in throwers with pathologic internal impingement. Am J Sports Med. 2006;34(3):385–91.

Chapter 3
Biceps Brachii Pathology

The biceps brachii is a Latin word, meaning two-headed [muscle] of the arm. The biceps is consisted of two bundles of muscles with two independent origins, sharing a common point of insertion. Proximally, the biceps inserts to the scapula via its long and short heads. The long head of the biceps tendon originates from the supraglenoid tubercle, while the short head originates from the coracoid process. The bicipital groove is an indentation between the lesser and greater tubercles and the supratubercular ridge is the continuation of the lesser tubercle [1]. Of interest, the proximal short head of the biceps is not a true tendon (cordlike fibers of connective tissue) as previously believed and has a direct muscular attachment to the coracoid process with an aponeurosis (a ribbonlike tendinous expansion) on its anterior surface [2]. Distally, the muscle bellies of short and long heads unite in the middle of the humerus to share a common insertion point at the bicipital tuberosity on the radius [3, 4].

The biceps muscle is the prime supinator of the forearm and assists with elbow flexion. The long head of the biceps is reported to contribute to arm abduction, at about 7–10% of power [5] mostly when the arm is in external rotation [6]. There is controversy about the long head assisting with humeral stabilization with some investigators supporting this role [7–10] and others questioning its significance [11]. The short head assists with humeral adduction when the arm is medially rotated [6].

The distal biceps tendon is composed of a long and a short head, which attaches to the posterior aspect of the radial tuberosity. In a review by Schmidth et al. [12], it is noted that the footprint position of each head is vital in maintaining supination torque throughout the forearm rotation. Therefore, it is not surprising that the distal short and long heads have different mechanical roles. The distal short head generates greater flexion load than the long head because of its distal attachment, and the long head creates a greater supination moment than the short head [13].

The proximal biceps pathology makes up the majority of all biceps tendon problems, of which the long head of the biceps is the most affected one with the short

H. Razmjou, M. Christakis, *Clinical and Radiological Examination of the Shoulder Joint*, https://doi.org/10.1007/978-3-031-10470-1_3

head tears being rarely reported (about 2%) [14]. Distal tears are estimated at about 3% of all biceps tears [15].

Historical Perspective

The original description of proximal biceps pathology goes back to the late 1880s. In a review by Ahrens et al. [16], the first description of biceps pathology was in an 1880s literature and was related to instability of the long head. In 1921, Meyer [17] mentioned the biceps brachii muscle as a possible source of pain and dysfunction in the shoulder. In his review of anatomical specimens, he mentioned degenerative changes in the fibers of the biceps tendon including fraying, shredding, tearing, and partial or complete dislocation of the tendon out of the bicipital groove. Meyer believed that the direct contact of the long head of the biceps tendon with a supratubercular ridge raised cartilaginous margins of the humeral head and bony irregularities of the intertubercular groove increased the vulnerability of the tendon to attrition and subluxation. He felt that using the upper extremity in a position of abduction and external rotation increased these contacts. Since Mayer's observations were based on findings in cadavers with no clinical data, the clinical significance of these alterations was not clear at the time [1, 17]. In 1932, Pasteur was the first to correlate bicipital tenosynovitis with the frozen shoulder [18]. Codman, however, challenged the idea of isolated biceps pathology in his eminent book published in 1934 [19]. He noted that biceps tendon was less apt to be involved in inflammatory conditions of the shoulder than were other soft tissue structures. It was not until the late 1940s and early 1950s when the pathology of the long head of the biceps was more extensively described [6, 20]. In an article by Hitchcock et al. published in 1948 [6], the authors emphasized the role of supratubercular ridge as an extension of lesser tuberosity and the shallow bicipital groove as a contributor to biceps pathology during elevation and medial rotation, when the biceps tendon was pressed against lesser tuberosity.

The earliest description of the proximal short head of the biceps dates back to 1934. In the review of 100 patients with a known rupture of the biceps, Gilcrest et al. [14] found only two cases of isolated short head pathology. In 1941, Tobin et al. [21] reported a single case of proximal short head rupture with damage to the coracobrachialis. In 1977, Postacchini and Ricciardi-Pollini [22] reported a case with subcutaneous rupture of the short tendon of the biceps. In general, there are only a handful of articles on pathology of the short head of the biceps brachii indicating its rarity.

In a review by Quach et al. [15], the first description of the distal biceps injury goes back to 1843. The first known case of surgical description of distal biceps tendon being sutured to the radial tuberosity was reported by Johnson in 1897 [23].

Proximal Long Head of Biceps Brachii Pathology

The long head of the biceps is an intra-articular structure before entering the bicipital groove, at which point it becomes extra-articular. Generally, proximal long head pathologies are rarely seen in isolation and are usually associated with rotator cuff problems with the incidence increasing significantly with age [24, 25]. Pathologies of the long head falls into four major categories: (1) tendinitis/tenosynovitis/tendinosis, (2) subluxation/dislocation, (3) partial tear/complete ruptures, and (4) superior labral anteroposterior (SLAP) lesions. The superior labral pathologies are discussed separately in Chap. 6.

Biceps tendinitis refers to inflammation of the tendon around the long head of the biceps muscle. Tendinosis refers to a chronically damaged tendon with disorganized fibers and a hard, thickened, and scarred appearance. Tenosynovitis of the long head can occur because of the unique anatomy of this tendon being covered by a synovial sheath in the bicipital groove [26–28]. Tenosynovitis and tendinosis are more common and represent progressive degeneration of the biceps tendon potentially due to its unique anatomy and constrained path in the bicipital groove.

Subluxation and dislocation of the long head are commonly reported in patients with associated pathology of rotator cuff tendons. The superior glenohumeral ligament and coracohumeral ligament have a critical role in providing stability of the tendon. Degenerative changes, traction, or hyperextension injuries may damage the restraining structures around the biceps tendon and cause medial (also referred to as anterior) subluxation or dislocation of the tendon. Anterior or medial instability is often associated with a subscapularis tear, whereas a much less frequent type of posterolateral instability is usually associated with fracture of greater tuberosity or supraspinatus tear as the posterior structure providing stability to the long head [29]. A dislocated tendon may be covered in fibrous tissue or become adherent to the subscapularis tendon. Traumatic dislocation of the long head is often associated with a tear of the upper fibers of the subscapularis tendon [16].

Partial tear and complete ruptures of biceps brachii are often seen in association with posterior cuff pathology and occasionally subscapularis pathology. Gill et al. [30] who studied 847 consecutive patients undergoing arthroscopic surgery reported a prevalence rate of 5% for partial tears. A higher rate of 30% has been reported in patients with concomitant full-thickness rotator cuff tears seen at the tertiary care centers [31]. Acute injuries and traumatic tears are related to sports-related acceleration and deceleration and forced flexion of the elbow. Eccentric forces (sudden extension of elbow while carrying a weight) and simultaneous distal traction of the extremity and biceps contraction or forceful biceps contraction in weight lifting, gymnastics, and football are common causes of traumatic tears [32]. Chronic tears of the long head are usually associated with tenosynovitis, tendinosis, and delamination and are secondary to wearing down and fraying of the tendon, age-related or secondary to overuse and repetitive activities that involve rotation and flexion.

Clinical Findings and Management

Patients with proximal biceps pathology usually report a throbbing ache in the anterior shoulder, occasionally referred to the bulk of biceps muscle. Repetitive activities or rotations and elbow supination may aggravate symptoms. Tenderness over bicipital groove with internal/external rotation may be elicited. However, tenderness on palpation of the long head of the biceps tendon has been reported to have low sensitivity and specificity, being insufficient to rule in or out biceps pathology [30]. Local anesthetic injections into the biceps tendon sheath may be used as a diagnostic tool [33]. However, the potential complications such as allergic reaction, flushing, increased blood glucose level, local tissue atrophy, tendon rupture, avascular necrosis, and septic arthritis should be considered before ordering a diagnostic cortisone injection to the biceps tendon sheath. Absolute contraindications to corticosteroid injections of the biceps tendon include local infection, intra-articular fracture, and bacteremia [34]. Considering the above possible side effects, the diagnostic biceps tendon injections are not commonly performed.

Patients with dislocated long head of the biceps may present with a loss of active elevation above 90°, and it is common to find a limitation of active and passive external rotation because the dislocated biceps tendon restrains the inferior part of the subscapularis [16]. Boileau et al. reported that an inflamed and thickened intra-articular segment can block tendon excursion during shoulder motion and block 10° to 20° of passive elevation [35]. An important clinical sign of biceps long head rupture is the classic "Popeye" sign, but not all ruptures produce this deformity [16].

Apart from range of motion and strength testing, a number of specific clinical tests have been proposed for biceps brachii, but they mostly have inconsistent and suboptimal value in clinical settings. Accuracy estimates reported by original authors of small clinical studies are often contaminated by verification bias and lack of proper gold standard, and poor methodologies have made the replication of the results by other authors difficult. The diagnostic accuracy of Speed's and Yergason's, two traditionally used tests for detecting early-stage biceps pathology, is limited as well. Low sensitivity and specificity of these clinical tests make them inadequate for conclusive clinical diagnosis [29, 30, 36, 37]. In general, systematic reviews highlight that diagnosis of tendinitis or partial biceps tears cannot be made reliably with existing physical examination tests [38–40].

Management of early stages of long head pathology is almost always conservative. Physiotherapy and activity modification often relive the symptoms of tendinitis, tenosynovitis, and tendinosis. Local anesthetic injections into the biceps tendon sheath may be tried in more chronic cases [33]. End-stage lesions involving complete rupture usually do not require operative treatment, although it may be considered in younger, active patients with acute tears [41].

Biceps tenotomy and tenodesis are surgical procedures considered for pathology of the proximal long head of the biceps. Tenotomy or tenodesis is recommended for high-grade partial tears >50% of the tendon width, although a smaller number of authors use 25% as the cutoff [42]. Low-grade partial tears <50% of the tendon width are generally treated with debridement of the tear, while preserving the biceps origin at the superior labrum. High-grade partial-thickness tears involving more than 50% of the tendon width require surgical intervention, particularly when associated with rotator cuff or anterior glenohumeral instability. A biceps tenodesis involves releasing the biceps tendon long head from its origin inside the shoulder joint, followed by fixation of the tendon. There is significant debate on fixation method and location of tenodesis. Suturing the long head to the rotator cuff or biceps pulley, suture anchor fixation, interference screw fixation, proximal tenodesis within the bicipital groove, and distally at the exit of the bicipital groove at the pectoralis major insertion or at the coracoid process next to the biceps short head origin have been proposed. A recent systematic review which included 468 patients of 5 randomized controlled studies reported a higher incidence of cosmetic deformity in tenotomy patients (23.3% vs. 6.8%). The tenodesis group had more complaints of biceps cramping at 6 months but not in longer-term follow-ups. No differences were reported in patient-reported outcome measures at an average of approximately 2 years. There were no differences between groups in elbow flexion strength or supination strength in four out of five studies included [43].

Proximal Short Head of Biceps Brachii Pathology

The short head of the biceps brachii arises from the coracoid process with the coracobrachialis muscle. Because of the nature of the proximal insertion being more muscular than tendinous and the location of the coracoid process, the tendon rupture of the short head is extremely rare with just a limited number of closed muscle rupture in the literature [21, 44–48].

In 1978, Heckman and Levine [48] reported a series of injuries in military parachutists. This injury is mainly associated with military parachute jumps where the static line (a fixed cord used to open *parachutes)* is oriented incorrectly around the arm at the onset of jumping causing a straight blunt force on the biceps brachii muscle. In a case report of an isolated complete rupture of the short head of the biceps in a 21-year-old man, the arm was in abduction and external rotation out of a car window, when it hit an object [47]. In summary, isolated pathology of the short head of the biceps is very rare and almost always traumatic due to a direct blow to the muscle.

Clinical Findings and Management

As all reported cases in the literature have been traumatic, signs of blunt trauma such as tenderness, swelling, and ecchymosis are expected in acute cases. In addition, a sizeable bulge in the medial middle third of the injured arm and a hollow in the site normally occupied by the short head of the biceps are visible [22]. Apart from the bulge in the middle third of the arm, a transverse depression in the sagittal plane at the site normally filled by the short head of the biceps muscle belly is observed [47].

In terms of management of closed acute tears of the short head of the biceps brachii muscle, a prompt repair of acute tear has shown successful results [44–46] with untreated ruptures being associated with residual weakness in elbow flexion and supination and deformity. Overall, there are no comprehensive data to support operative vs. nonoperative treatment in more chronic cases of short biceps head pathology.

Distal Biceps Pathologies

Distal biceps tendon rupture is a relatively rare injury as well [49, 50]. Safran and Graham [51] projected an incidence of 1.2 distal biceps tendon ruptures per 100,000 patients per year with an average age of 47 years at the time of injury.

Distally, the biceps brachii tendon inserts onto the bicipital tuberosity on the proximal portion of the radius. Distal biceps tendon ruptures vary from a partial tear to a complete tear. Partial tears may not involve a dramatic event and mostly present as an anterior elbow pain without other specific findings. Complete ruptures are generally associated with a dramatic injury causing weakness with elbow flexion and forearm supination. In cases when the diagnosis of distal biceps tendon rupture is in question, MRI of the elbow is often performed [52]. Chronic irritation on an irregular surface, such as in persistent cubital bursitis or forceful extension of the elbow from a flexed and supinated position, may also cause distal ruptures. Weight lifters, laborers, and athletes are more commonly at risk of having this problem [53].

Clinical Findings and Management

Distal ruptures are usually reported in the dominant extremity of middle-aged men [54]. Patients with an acute distal biceps tendon injury typically experience a tearing sensation and an acute onset of pain after an unexpected or massive extension force has been applied to the flexed elbow. Typically, there is pain and deformity with weakness of supination [55] with bruising and swelling in the antecubital fossa, and patients often describe a painful popping sound or sensation at the time of injury. A "reverse Popeye" deformity with the muscle belly retracted proximally may be seen. In more chronic cases, detection of distal biceps tears is not as straightforward, and a distal tear without retraction may not be present with the usual proximal muscle migration.

A clinical test for a complete rupture of the distal head, the hook test, was described by O'Driscoll et al. in 2007 [56]. This test is performed by inserting the finger under the lateral edge of the biceps tendon between the brachialis and biceps tendons and hooking the finger under the cord-like structure spanning the antecubital fossa with the patient's elbow flexed 90. The authors reported a sensitivity and specificity of 100% and reiterated that a crucial portion of the test is to hook the lateral edge of the biceps tendon, not the medial edge, as the examiner could mistake the lacertus fibrosus for an intact biceps tendon. To date there has been only one retrospective study that has examined this test independently. According to Luokkala et al. [57], the sensitivity for the distal biceps hook test was 78% in all tears and 83% in complete tears with much lower validity for partial tears. A combination of history, mechanism of injury, and multiple tests in association with the hook test has shown to be helpful in diagnosing this pathology [58].

In terms of management, nonoperative treatment of distal biceps rupture consists of temporary immobilization, pain control, and physiotherapy. Nonoperative management may result in a moderate decrease of supination (30–50%) and minimal flexion strength loss (20%) [59]. In 1941, Dobbie [60] described the nonoperative management of distal biceps tendon rupture. He noted good result with conservative treatment. Early surgical reports date back to 1985 by Baker and Bierwagen [59] and Morrey et al. [61] who showed better strength in supination and flexion after operative intervention. The literature in mid-2000 encourages nonoperative treatment for sedentary patients who do not require elbow flexion and supination strength and endurance or for patients who are not medically fit for operative treatment [55, 62]. Surgical reconstruction of distal biceps tendon may get complicated by neurapraxia, superficial wound infection, heterotopic ossification, tendon retear or failed reattachment, and nerve injury which is considered a major complication due to the functional effects associated with a lack of muscle function [63]. A systematic review in 2016 found that the single-incision technique had a greater rate of nerve palsy and rerupture rates compared with the double-incision technique which had a greater rate of heterotopic ossification. These complications need to be disclosed to patients who are trying to make an informed decision on operative repair [63]. A more recent publication in 2021 questions the traditional wisdom that a surgical repair is needed for all distal biceps ruptures [64]. In this prospective study of two middle-aged patients with full rupture of the distal biceps, full improvement of function of elbow flexion and supination was reported at 6 months with structured rehabilitation [64].

References

1. Meyer A. Spontaneous dislocation and destruction of tendon of long head of biceps brachii. Arch Surg. 1928;17:493–506.
2. Crichton JC, Funk L. The anatomy of the short head of biceps - not a tendon. Int J Shoulder Surg. 2009;3(4):75–9.
3. Tagliafico A, Michaud J, Capaccio E, Derchi LE, Martinoli C. Ultrasound demonstration of distal biceps tendon bifurcation: normal and abnormal findings. Eur Radiol. 2010;20(1):202–8.

4. Fogg QA, Hess BR, Rodgers KG, Ashwood N. Distal biceps brachii tendon anatomy revisited from a surgical perspective. Clin Anat. 2009;22(3):346–51.
5. Furlani J. Electromyographic study of the m. biceps brachii in movements at the glenohumeral joint. Acta Anat (Basel). 1976;96(2):270–84.
6. 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., 30-A: 263, 1948. J Bone Joint Surg. 1948;30-A(263):263–73.
7. Neer CS 2nd. Anterior acromioplasty for the chronic impingement syndrome in the shoulder: a preliminary report. J Bone Joint Surg Am. 1972;54(1):41–50.
8. Rodosky MW, Harner CD, Fu FH. The role of the long head of the biceps muscle and superior glenoid labrum in anterior stability of the shoulder. Am J Sports Med. 1994;22(1):121–30.
9. Yamaguchi K, Riew KD, Galatz LM, Syme JA, Neviaser RJ. Biceps activity during shoulder motion: an electromyographic analysis. Clin Orthop Relat Res. 1997;336:122–9.
10. Levy AS, Kelly BT, Lintner SA, Osbahr DC, Speer KP. Function of the long head of the biceps at the shoulder: electromyographic analysis. J Shoulder Elb Surg. 2001;10(3):250–5.
11. Walch G, Edwards TB, Boulahia A, Nove-Josserand L, Neyton L, Szabo I. Arthroscopic tenotomy of the long head of the biceps in the treatment of rotator cuff tears: clinical and radiographic results of 307 cases. J Shoulder Elb Surg. 2005;14(3):238–46.
12. Schmidt CC, Savoie FH 3rd, Steinmann SP, Hausman M, Voloshin I, Morrey BF, et al. Distal biceps tendon history, updates, and controversies: from the closed American shoulder and elbow surgeons meeting-2015. J Shoulder Elb Surg. 2016;25(10):1717–30.
13. Jarrett CD, Weir DM, Stuffmann ES, Jain S, Miller MC, Schmidt CC. Anatomic and biomechanical analysis of the short and long head components of the distal biceps tendon. J Shoulder Elb Surg. 2012;21(7):942–8.
14. Gilcrest EI. The common syndrome of rupture, dislocation and elongation of the long head of the biceps brachii. Analysis of 100 cases. Surg Gynecol Obstet. 1934;58:322–40.
15. Quach T, Jazayeri R, Sherman OH, Rosen JE. Distal biceps tendon injuries—current treatment options. Bull NYU Hosp Jt Dis. 2010;68(2):103–11.
16. Ahrens PM, Boileau P. The long head of biceps and associated tendinopathy. J Bone Joint Surg Br. 2007;89(8):1001–9.
17. Meyer A. Unrecognized occupational destruction of the tendon of the long head of the biceps brachii. Arch Surg. 1921;2:130.
18. Pasteur F. La teno-bursite bicipitale. J deradial, et d'electrol. 1932;16:419.
19. Codman EA. The shoulder, rupture of the supraspinatus tendon and other lesions in or about the subacromial Bursa. Boston: Todd Thomas; 1934.
20. Depalma AF, Callery GE. Bicipital tenosynovitis. Clin Orthop. 1954;3:69–85.
21. Tobin WJ, Cohen LD, Vandover JT. Parachute injuries. JAMA. 1941;117:1318–21.
22. Postacchini F, Ricciardi-Pollini PT. Rupture of the short head tendon of the biceps brachii. Clin Orthop Relat Res. 1977;124:229–32.
23. Johnson AB. Avulsion of biceps tendon from the Radious. NY, Med J. 1897;66:261–2.
24. Chen CH, Hsu KY, Chen WJ, Shih CH. Incidence and severity of biceps long head tendon lesion in patients with complete rotator cuff tears. J Trauma. 2005;58(6):1189–93.
25. Murthi AM, Vosburgh CL, Neviaser TJ. The incidence of pathologic changes of the long head of the biceps tendon. J Shoulder Elb Surg. 2000;9(5):382–5.
26. Post M, Benca P. Primary tendinitis of the long head of the biceps. Clin Orthop Relat Res. 1989;246:117–25.
27. 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.
28. Neviaser TJ. Arthroscopy of the shoulder. Orthop Clin North Am. 1987;18(3):361–72.
29. Lafosse L, Reiland Y, Baier GP, Toussaint B, Jost B. Anterior and posterior instability of the long head of the biceps tendon in rotator cuff tears: a new classification based on arthroscopic observations. Arthroscopy. 2007;23(1):73–80.

30. Gill HS, El Rassi G, Bahk MS, Castillo RC, McFarland EG. Physical examination for partial tears of the biceps tendon. Am J Sports Med. 2007;35(8):1334–40.
31. Razmjou H, Fournier-Gosselin S, Christakis M, Pennings A, ElMaraghy A, Holtby R. Accuracy of magnetic resonance imaging in detecting biceps pathology in patients with rotator cuff disorders: comparison with arthroscopy. J Shoulder Elb Surg. 2015;
32. Grauer JD, Paulos LE, Smutz WP. Biceps tendon and superior labral injuries. Arthroscopy. 1992;8(4):488–97.
33. Churgay CA. Diagnosis and treatment of biceps tendinitis and tendinosis. Am Fam Physician. 2009;80(5):470–6.
34. Schwantes J, Byerly DW. Biceps tendon sheath injection. StatPearls Treasure Island (FL): StatPearls Publishing LLC; 2021.
35. 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.
36. Chen HS, Lin SH, Hsu YH, Chen SC, Kang JH. A comparison of physical examinations with musculoskeletal ultrasound in the diagnosis of biceps long head tendinitis. Ultrasound Med Biol. 2011;37(9):1392–8.
37. Holtby R, Razmjou H. Accuracy of the Speed's and Yergason's tests in detecting biceps pathology and SLAP lesions: comparison with arthroscopic findings. Arthroscopy. 2004;20(3):231–6.
38. Beaudreuil J, Nizard R, Thomas T, Peyre M, Liotard JP, Boileau P, et al. Contribution of clinical tests to the diagnosis of rotator cuff disease: a systematic literature review. Joint Bone Spine. 2009;76(1):15–9.
39. Hegedus EJ, Goode A, Campbell S, Morin A, Tamaddoni M, Moorman CT 3rd, et al. Physical examination tests of the shoulder: a systematic review with meta-analysis of individual tests. Br J Sports Med. 2008;42(2):80–92. discussion 92
40. Munro W, Healy R. The validity and accuracy of clinical tests used to detect labral pathology of the shoulder—a systematic review. Man Ther. 2009;14(2):119–30.
41. Sethi N, Wright R, Yamaguchi K. Disorders of the long head of the biceps tendon. J Shoulder Elb Surg. 1999;8(6):644–54.
42. Checchia SL, Doneux PS, Miyazaki AN, Silva LA, Fregoneze M, Ossada A, et al. Biceps tenodesis associated with arthroscopic repair of rotator cuff tears. J Shoulder Elb Surg. 2005;14(2):138–44.
43. Belk JW, Kraeutler MJ, Houck DA, Chrisman AN, Scillia AJ, McCarty EC. Biceps tenodesis versus tenotomy: a systematic review and meta-analysis of level I randomized controlled trials. J Shoulder Elb Surg. 2021;30(5):951–60.
44. Salmons HI, Warrender WJ, Smith S, Kearns KA, Strohl A. Intrasubstance tear of the short head of biceps with musculocutaneous and median nerve compression. J Am Acad Orthop Surg Glob Res Rev. 2019;3:12. https://doi.org/10.5435/JAAOSGlobal-D-19-00074. eCollection 2019 Dec
45. Kozak TA, Kozak TKW. Isolated rupture of the short head of the biceps at the proximal musculotendinous junction: a report of two cases. JBJS Case Connect. 2019;9(4):e0251.
46. Kalinterakis G, Antonogiannakis E, Rampakakis I, Tsialogiannis E, Syllaios A, Ziogas M. Traumatic closed proximal muscle rupture of the biceps brachii in military paratrooper. Case Rep Orthop. 2019;2019:3472729.
47. Shah AK, Pruzansky ME. Ruptured biceps brachii short head muscle belly: a case report. J Shoulder Elb Surg. 2004;13(5):562–5.
48. Heckman JD, Levine MI. Traumatic closed transection of the biceps brachii in the military parachutist. J Bone Joint Surg Am. 1978;60(3):369–72.
49. De Palma L, Delcogliano A. Subcutaneous rupture of the distal tendon of the biceps brachii. Arch Putti Chir Organi Mov. 1984;34:281–6.
50. Dojcinovic S, Maes R, Hoffmeyer P, Peter R. Surgical treatment for distal rupture of the biceps tendon. Rev Chir Orthop Reparatrice Appar Mot. 2004;90(5):420–5.

51. Safran MR, Graham SM. Distal biceps tendon ruptures: incidence, demographics, and the effect of smoking. Clin Orthop Relat Res. 2002;404:275–83.
52. Festa A, Mulieri PJ, Newman JS, Spitz DJ, Leslie BM. Effectiveness of magnetic resonance imaging in detecting partial and complete distal biceps tendon rupture. J Hand Surg Am. 2010;35(1):77–83.
53. Strauch RJ, Michelson H, Rosenwasser MP. Repair of rupture of the distal tendon of the biceps brachii. Review of the literature and report of three cases treated with a single anterior incision and suture anchors. Am J Orthop (Belle Mead NJ). 1997;26(2):151–6.
54. Carter AN, Erickson SM. Proximal biceps tendon rupture: primarily an injury of middle age. Phys Sportsmed. 1999;27(6):95–101.
55. Miyamoto RG, Elser F, Millett PJ. Distal biceps tendon injuries. J Bone Joint Surg Am. 2010;92(11):2128–38.
56. Odriscoll GLB, Dietz P. The hook test for distal biceps tendon avulsion. Am J Sports Med. 2007;35(11):1865–9.
57. Luokkala T, Siddharthan SK, Karjalainen TV, Watts AC. Distal biceps hook test—sensitivity in acute and chronic tears and ability to predict the need for graft reconstruction. Shoulder Elbow. 2020;12(4):294–8.
58. Devereaux MW, ElMaraghy AW. Improving the rapid and reliable diagnosis of complete distal biceps tendon rupture: a nuanced approach to the clinical examination. Am J Sports Med. 2013;41(9):1998–2004.
59. Baker BE, Bierwagen D. Rupture of the distal tendon of the biceps brachii. Operative versus non-operative treatment. J Bone Joint Surg Am. 1985;67(3):414–7.
60. Dobbie RP. Avulsion of the lower biceps brachii tendon: analysis of fifty-one previously unreported cases. Am J Surg. 1941;51:662–83.
61. Morrey BF, Askew LJ, An KN, Dobyns JH. Rupture of the distal tendon of the biceps brachii. A biomechanical study. J Bone Joint Surg Am. 1985;67(3):418–21.
62. Chavan PR, Duquin TR, Bisson LJ. Repair of the ruptured distal biceps tendon: a systematic review. Am J Sports Med. 2008;36(8):1618–24.
63. Amin NH, Volpi A, Lynch TS, Patel RM, Cerynik DL, Schickendantz MS, et al. Complications of distal biceps tendon repair: a meta-analysis of single-incision versus double-incision surgical technique. Orthop J Sports Med. 2016;4(10):2325967116668137.
64. Parikh P, MacDermid JC, Tuli V, Manley M. Distal biceps tendon rupture: is surgery the best course of treatment? Two case reports. J Hand Ther. 2021;34(3):463–8.

Chapter 4
Tears of Rotator Cuff Tendons

Rotator cuff tears are among the most commonly seen pathologies of the shoulder joint. The variability in symptoms and clinical presentations of these tears and overlap with other pathologies make the clinical diagnosis of this pathology challenging. Similarly, the management of rotator cuff tears is not a straightforward subject as many factors such as age, gender, symptom severity and duration, perceived quality of life, severity of physical disability, physical demands and activity restrictions, and tear size affect the clinical pathway for this pathology.

Historical Perspective

In a review of historical landmarks of rotator cuff pathology, Alexander Monro appears to have provided the first description of a rotator cuff tear in his book *A Description of All the Bursae Mucosae of the Human Body*, published in 1788 [1]. The next publication about rotator cuff tears was by John G. Smith, an English surgeon and anatomist, about 50 years later in 1834. Smith discussed presence of the bursal inflammation and partial- and full-thickness tears related to shoulder dislocation in cadavers [2, 3]. A century later, Ernest Amory Codman, an American orthopedic surgeon, highlighted the importance of the supraspinatus tendon in the clinical picture of subacromial bursitis. Codman is potentially one of the most academically and clinically achieved surgeons in the field of shoulder pathology. He was the first clinician who described the nature of rotator cuff tear in the context of clinical examination and outcome management. He pointed out that the supraspinatus tendon rupture could affect the ability of the arm to abduct [4]. In 1992, the details on the structure of the myotendinous part of rotator cuff was described by John M. Clark. In his detailed anatomical study, he described that the supraspinatus tendon was enveloped by a thick sheet of fibrous tissue derived from the coracohumeral ligament [5].

H. Razmjou, M. Christakis, *Clinical and Radiological Examination of the Shoulder Joint*, https://doi.org/10.1007/978-3-031-10470-1_4

A year later, Burkhart and Jolson [6] coined the terms "rotator crescent" and "rotator cable" to describe a thin, avascular crescent-shaped sheet of rotator cuff comprised of the distal portions of the supraspinatus and infraspinatus insertions and a thick bundle of fibers that span the insertions of supraspinatus and infraspinatus tendons, respectively. With his landmark article in 1993, Stephen S. Burkhart described the sophisticated and complex biomechanical role of the rotator cable as a stress shielder of the rotator cuff tendons and changed our understanding of this critical structure of the shoulder joint.

Characteristics, Classification, and Causes

Rotator cuff tears in older adults who do not recall a traumatic injury are usually caused by attrition and tendon degeneration over time. These tears are fairly common in the aging population and are usually asymptomatic. Acute tears are often seen in younger adults following a traumatic injury [7]. Apart from aging and trauma, other factors such as repetitive activities, acromion morphology, smoking, and inflammatory and metabolic conditions such as diabetes mellitus [8] and low serum level of vitamin D have been proposed as contributing factors to rotator cuff disease [9]. Oh et al. [9] reported that the serum level of vitamin D was an independent predictor of fatty degeneration of the supraspinatus and infraspinatus muscles. Also, genetic factors may aid predisposition to full-thickness cuff tears [10].

The most common rotator cuff tendon tear occurs in the supraspinatus tendon. The involvement of the infraspinatus is indicative of a more significant pathology, most probably of degenerative nature. Degenerative rotator cuff tears most commonly involve a region posterior to the biceps tendon, regarded as either the junction between the supraspinatus and infraspinatus or being purely within the infraspinatus tendon [11]. This may explain why fatty degeneration of the infraspinatus is seen in some patients with a presumed isolated tear of the supraspinatus tendon. The close proximity of the insertion of the supraspinatus and infraspinatus muscle fibers and being innervated by the same nerve [12–15] create an overlap between the clinical findings of the supraspinatus and infraspinatus muscles. Although the supraspinatus is more of a primary abductor and infraspinatus is an external rotator and an important humeral head suppressor, in a study by Razmjou et al. [16], isometric strength in external rotation correlated with pathologic changes in both supraspinatus and infraspinatus muscles. In their study, external rotation weakness was a good indicator of a large and massive cuff tear, involving both tendons.

Fatty degeneration of the rotator cuff muscles is a consequence of full-thickness tears. Fatty degeneration is affected by the tear length and width and consequently the level of tear retraction [17]. Kim et al. have suggested that the disruption of the most anterior part of the supraspinatus tendon is most closely associated with the development of fatty degeneration in the supraspinatus muscle, whereas tear size is most closely associated with the development of fatty degeneration in the infraspinatus [17]. In other words, the tendon tear location (anterior vs. posterior) is more relevant in the supraspinatus, and the tear size is more important in the infraspinatus fatty degeneration (see section on importance of cuff tear location). Involvement of the infraspinatus tendon explains the narrowing of the acromiohumeral distance secondary to superior subluxation of the humeral head, a phenomenon seen more commonly in chronic cuff pathology than in traumatic tears. According to Kim et al., rotator cuff tears that extend into the infraspinatus tendon affect the protective function of rotator cuff cable, resulting in proximal humeral migration, further showing the importance of the infraspinatus tendon in maintaining normal glenohumeral kinematics.

Types of Rotator Cuff Tears (Partial Vs. Full Thickness)

Considering the management of partial cuff tears is different than full-thickness tears, a short summary of these conditions is merited. Generally, cuff tears are categorized as partial-thickness rotator cuff tears (PTRCT) and full-thickness rotator cuff tears (FTRCT). Although a PTRCT does not involve the entire width of the tendon, the size of the tear may involve a large surface area, being quite symptomatic. Codman [4] first described partial tearing of the supraspinatus as “rim rents.” The only other mention of this term in the literature is by Tuite in 1998 and Vinson in 2007 [18, 19]. The more commonly used classification system for partial cuff tears, which is based on location of the tear, was suggested by Ellman in 1990. He described bursal, articular, and intra-substance (intratendinous) [20]. Ellman’s classification system also incorporates the depth of the tear (grade 1, <3 mm; grade 2, 3–6 mm; and grade 3, > 6 mm).

Full-thickness rotator cuff tears affect the entire width of the tendon (Fig. 4.1). The size of a full-thickness rotator cuff tear (largest dimension) is categorized as small <1 cm, moderate (1–3 cm), large (>3–5 cm), and massive (>5 cm) [21]. A small full-thickness tear usually involves the supraspinatus, where a moderate tear may involve the infraspinatus as well. Large and massive tears may affect the teres minor and occasionally subscapularis. Isolated subscapularis tears are mostly related to violent external rotation and abduction, seen in ski accidents. Neer [7] highlighted the similarity of clinical examination and plain imaging of partial- and smaller full-thickness rotator cuff tears (e.g., crepitus, tenderness over the supraspinatus insertion, and pain with active elevation from 70 to 120) which would make these two conditions indistinguishable. Even today, there is a significant reliance on US and MRI for an accurate diagnosis of partial- and small to moderate full-thickness rotator cuff tears. Advanced large to massive full-thickness tears, however, have specific clinical characteristics that help the clinician to have less reliance on costly soft tissue imaging investigations.

In terms of epidemiology, there is a high incidence of bilateral rotator cuff involvement in older individuals, indicative of degenerative nature of cuff tears. In a study of 588 patients with shoulder pain, Yamaguchi et al. [22] reported an overall 57%

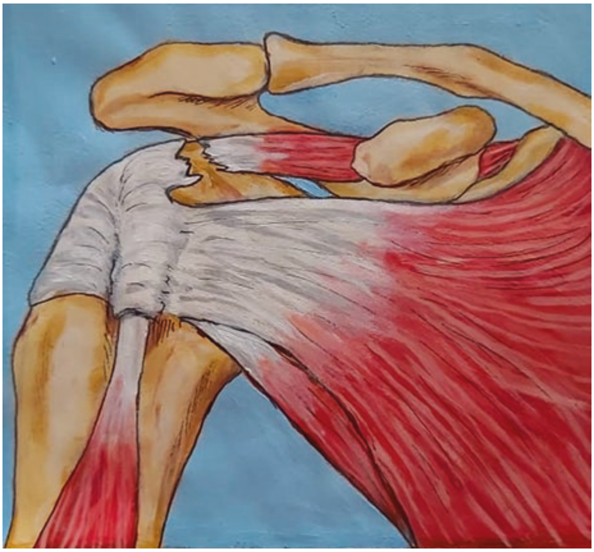

Fig. 4.1 Illustration of a full-thickness supraspinatus tear

prevalence of partial- or full-thickness tear in the opposite shoulder. More specifically, a patient with a full-thickness tear on the painful side had a 35.5% chance of having a full-thickness tear and a 20.8% chance of having a partial-thickness tear on the contralateral asymptomatic side. While cuff tear is common in asymptomatic older individuals, development of pain and decline in shoulder function are usually indicative of tear progression [23]. Tear severity (full thickness vs. partial thickness) and hand dominance are greater risks for symptom progression [24, 25].

Importance of Cuff Tear Location and Involvement of Rotator Cable

Recent studies have proven that anterior tears of the supraspinatus tendon are more clinically relevant than posterior tears of the supraspinatus. In a cadaver study by Roh et al. [26], the cross-sectional areas of the anterior and posterior bellies of the supraspinatus were calculated to be 140 vs. 62 mm^2, whereas their tendon cross-sectional areas were 26 vs. 31 mm^2, respectively. This indicates that a larger anterior muscle pulls through a smaller tendon area, placing a significantly greater stress on the anterior tendon than posterior tendon. In agreement with the above study, Rahu et al. [27] confirmed that the supraspinatus had anterior and posterior muscle bellies, which differed in function and structure, and the supraspinatus tendon consisted of superficial and deep sections with the anterior tendon being thick and more tubular in structure whereas the posterior tendon was flat and wide. Most importantly, the rotator cable was found to be a connecting structure between the supraspinatus, infraspinatus, and teres minor tendons [27] (Fig. 4.2).

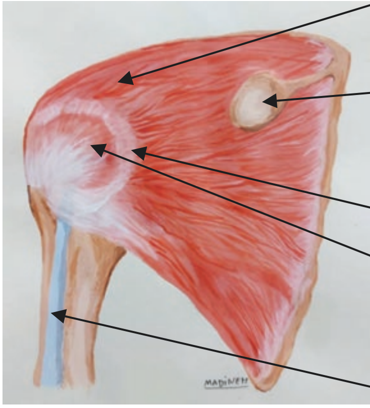

Fig. 4.2 Illustration of rotator cuff cable and rotator crescent

The intact rotator cable is another important factor in supraspinatus tear and ultimate dysfunction of the arm. In his historical description in 1993, Burkhart [6] described the rotator cable as a thick bundle of fibers that span the insertions of supraspinatus and infraspinatus tendons, averaging 2.59 times the thickness of the rotator crescent that it surrounded, a substantial structure that took another couple of decades to be well appreciated. In a cadaver study by Mesiha et al. [28], the rotator cuff cable was located about 8–12 mm anteriorly to the supraspinatus tendon and just posterior to the bicipital groove with an attachment to the superior half of the subscapularis [29]. The rotator cable was shown to be the primary load-bearing structure within the supraspinatus for force transmission to the humerus. This may further support that the anterior tendon stress is significantly greater than posterior tendon and that the supraspinatus muscle force can be normally transmitted to the greater tuberosity if the anterior rotator cuff cable is intact [28]. These findings explain why anterior tears of the supraspinatus are more disabling and have a more significant impact on the shoulder function. In other words, the close connection between the cuff tendons and rotator cable explains why some patients with relatively large rotator cuff tears and an intact rotator cable can maintain a relatively good shoulder function despite loss of integrity of the supraspinatus tendon [27].

One other interesting fact about role of intact rotator cable on reduced disability is highlighted in studies that have examined degenerative tears. In a study by Keener's team [11], the degenerative rotator cuff tears are reported to be most commonly posterior, near the junction of the supraspinatus and infraspinatus. These degenerative tears are thought to initiate in a region about 15 mm (range of 13–17) posterior to the biceps tendon. According to Burkhart [6], who first described the function of rotator cable and rotator crescent, the crescent region undergoes a progressive thinning due to relative avascularity of this region and thus is more vulnerable to tear formation in older individuals. However, as noted earlier, since the rotator cable is intact in these tears, many patients with degenerative posterior supraspinatus tears have minimal symptoms and function relatively well. In agreement with the above findings, in another cadaver study, Duralde et al. [30] showed that partial repairs of massive tears with anterior defects had more inferior results to those with posterior defects and an intact rotator cuff cable. The protection of the supraspinatus tendon by rotator cable has important implications which are highlighted in the section under natural history of rotator cuff disease section.

In summary, clinicians should be mindful of the location of the supraspinatus tear and the status of rotator cuff cable. Considering these findings translate to either maintaining a good elevation or poor function and pseudoparalysis (loss of active elevation with full passive elevation), these are important factors to consider when deciding on suggesting conservative treatment or surgical management. The posterior supraspinatus (posterosuperior) tears that tend to be more age-related and often do not involve the rotator cuff cable are minimally symptomatic, are associated with a reasonable active elevation and external rotation, are less likely to enlarge over time, and are not urgent for surgical repair. On the other hand, acute anterosuperior cuff tears that affect the rotator cable in younger individuals are a candidate for urgent repairs.

Natural History of Rotator Cuff Disease

Rotator cuff tears have been categorized based on the risk of clinically relevant tear progression and need for surgical repair by Keener [31, 32]. The *low-risk group* includes patients with atraumatic full-thickness tears of up to 15 mm in size with an intact anterior supraspinatus tendon and a healthy muscle. Keener noted that many degenerative cuff tears are small to medium and are contained within the rotator crescent and hence are rather protected by the surrounding cable attachment [11]. These patients have the lowest risk for tear progression and need for surgery. Patients >65 years of age with a lower healing rate that may not benefit from surgery fall in this group as well. Once the tear width exceeds 15–20 mm, the infraspinatus tendon becomes disrupted which would affect the normal biomechanics of the joint and subsequent superior migration of the humeral head with respect to the glenoid. Patients with reduced acromiohumeral distance secondary to superior migration of the humeral head associated with infraspinatus muscle atrophy will not benefit from a repair due to poor cuff healing. *Medium-risk group* for tear progression is a group of individuals with good healing capacity (age under 62–65 years). These include atraumatic full-thickness tears 15 mm or larger and tears with disruption of the anterior supraspinatus tendon, as well as previously painful shoulders with recent trauma (acute on chronic tears). These tears do not demonstrate any muscle fatty changes and may represent an opportunity to intervene with surgery. The *high-risk group* includes younger patients with an acute tear, minimal fatty degenerative changes, and thus a better rate of tendon healing. In this group a repair has the greatest potential to interrupt the natural history of an untreated cuff tear and provide successful results [24, 31, 32].

Clinical Findings of Rotator Cuff Tears

Majority of individuals with degenerative rotator cuff disease report an insidious onset of shoulder pain, although some may recall a specific injury prior to the onset of pain. Episodic report of pain followed by remission is common in older individuals. Rest, medication, and proper physical therapy can make the symptoms manageable for many years. However, symptoms become more constant as the disease progresses. Night pain and inability to sleep on the affected shoulder are often mentioned by patients with a progressive rotator cuff tear. With time and progression of the tear size, weakness will gradually develop. When asymptomatic, a rotator cuff tear is associated with a clinically insignificant loss of shoulder function compared with an intact rotator cuff. Therefore, a clinically detectable decline in shoulder function may indicate an "at-risk" asymptomatic tear. The presence of pain is important in cuff-deficient shoulders for creating a measurable loss of shoulder function. Hand dominance appears to be an important risk factor for pain [23].

Observation of rotator cuff muscles in patients with potential tears is extremely important. Clinicians should look for wasting, deformity related to the AC joint or clavicle, soft tissue cysts, and swelling of the glenohumeral joint. Considering scoliosis, kyphosis, and poor posture affect the position of the shoulder girdle, their presence should be commented on during examination. Significant muscle wasting in the supraspinatus is usually associated with infraspinatus atrophic changes which together represent a significant full-thickness rotator cuff tear. It has been reported that fatty degeneration in rotator cuff muscles is nearly exclusive to full-thickness tears [17]. Figure 4.3 shows significant muscle wasting secondary to a massive full-thickness tear involving infraspinatus.

In rare advanced cases, a soft tissue mass that signifies underlying rotator cuff pathology is visible. These cysts are caused by the synovial fluid escaping through the AC joint and are called "geyser" cysts (natural hot spring that intermittently ejects a column of water into the air). This phenomenon was first described by Craig in 1984 [33]. In 2010, Hiller et al. [34] examined the available literature on geyser cysts and found 41 reported

Fig. 4.3 Severe muscle wasting involving infraspinatus (Figure Adapted with permission from University of Toronto Press, Razmjou et al., Diagnostic Value of Acromiohumeral Distance in Rotator Cuff Pathology: Implications for Advanced-Practice Physiotherapists; Physiotherapy Canada, Copyright 2020;72(1):52–62, https://doi.org/10.3138/ptc-2018-0084)

Fig. 4.4 Clinical presentation of a geyser cyst

cases. The authors described two distinct etiologies for the pathogenesis of AC joint cyst formation. Type 1 cyst can form superficially in the presence of an intact rotator cuff and is limited to the AC joint [35]. Type 2 cyst forms in the presence of a massive or traumatic rotator cuff tear. Type 2 cyst is related to the mechanical instability of the humeral head, deterioration of the inferior acromioclavicular capsule, and overproduction of synovial fluid. Overtime, a "geyser" of fluid can form between the glenohumeral and the AC joints. These cysts may be associated with calcium pyrophosphate dihydrate deposition disease [36]. Aspiration and simple cyst excisions are not recommended due to potential for postoperative complications such as recurrence, draining sinus, and infection [37]. Lateral clavicle resection is suggested if there is an irreparable rotator cuff tear [37]. Given treatment of these two cysts is different, the differentiation between them is important. Figure 4.4 shows the clinical appearance of the cyst with skin redness and swelling.

Examination of the shoulder joint starts with the assessment of range of motion (ROM). Inconsistency between active and passive range of motion helps to differentiate between pathology in contractile tissues (muscles with their associated tendons, nerves, and bony insertions) and inert tissues (joint capsule, ligaments, and bursae). James Cyriax, an English orthopedic surgeon widely known as the father of orthopedic medicine, proposed selective tissue tension testing to make this differentiation possible [38]. Cyriax believed that physical therapists were the most suitable professionals to examine and apply soft tissue techniques because of their extensive training and close educational and clinical ties with medicine [39]. In his masterpiece about *selective tissue tension testing*, he discussed the logical, anatomical, and clinical steps to reach the correct diagnosis [38]. Cyriax felt that contractile tissues act differently to tension than the inert structures. He noted that a faulty contractile tissue would show pain by a simple strong isometric contraction. An inert tissue, on the other hand, would not move during a contraction and can be put under tension only by passive stretching.

Range of motion examination is a critical part of the shoulder assessment. Patients with chronic cuff pathology usually suffer from some degree of mild diffuse stiffness and altered movement in the scapulohumeral pattern. Active range of motion examines the quality and integrity of the rotator cuff tendons. Passive range of motion examines the flexibility of the shoulder capsule. Inconsistency between active and passive movements has a diagnostic significance. For example, a limited active elevation with a full passive elevation is a sign of large/massive cuff tears. Inability to actively elevate the arm to 90 degrees in the presence of full passive movement is called "pseudoparalysis." Presence of this phenomenon increases the likelihood of having a major pathology in rotator cuff muscles/tendons.

There is a large number of clinical examination tests in the field of orthopedics used without sufficient research supporting their validity or reliability. Pain provocative tests that rely solely on production of pain do not appear to be highly reliable or valid [40, 41]. In clinical practice, tests that are based on pain production are not truly diagnostic due to their low specificity and lack of ability to identify the type of cuff pathology. However, lack of pain with pain provocative maneuvers helps to rule out rotator cuff pathology [42]. One of the pain provocative clinical tests that has shown better validity is the scarf test or cross-body adduction test for AC joint arthritis and inflammation which is discussed in this chapter as it often affects rotator cuff pathology. This clinical test involves passive horizontal adduction of the flexed arm across the patient's body, bringing the elbow toward the contralateral shoulder (Fig. 4.5). The examiner's force results in compression of the acromioclavicular joint. This test has shown moderate validity [43].

On the other hand, having weakness is a good clinical predictor of significant cuff tear [44–49]. Commonly used clinical tests that are based on strength are the supraspinatus test (Jobe test) [50] (Fig. 4.6), infraspinatus test (external rotation in neutral), dropping sign [51], teres minor test (Hornblower's sign) [52] (Fig. 4.7),

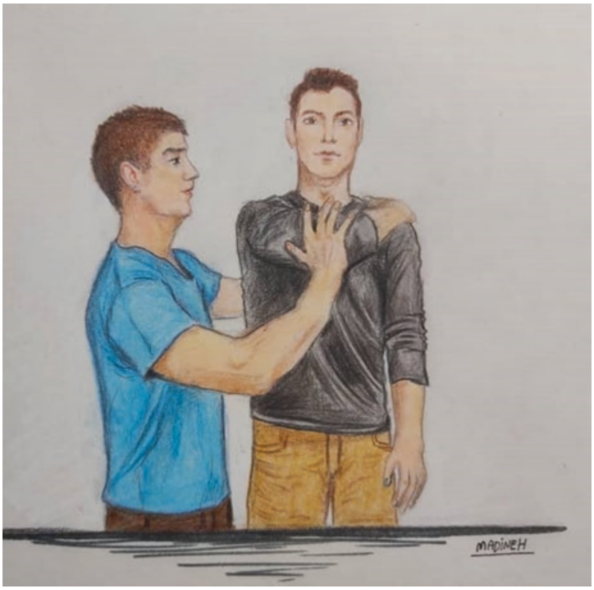

Fig. 4.5 Scarf test (cross-body adduction)

Fig. 4.6 Jobe's test to examine weakness in supraspinatus muscle

Fig. 4.7 Hornblower sign showing excessive abduction of arm during external rotation seen with large and massive tears

Fig. 4.8 Positive Napoleon sign on the right side showing inability to internally rotate the arm while pressing on the abdominal area

and two subscapularis tests both described by Gerber et al. including lift-off test [53] and Napoleon test (internal rotation from front of body) [54] (Fig. 4.8). The supraspinatus and infraspinatus tests are based on activation of contractile tissue testing. The Hornblower sign is based on observing a faulty posture. The lift-off test is based on inability to initiate a movement (lifting hand away from lumbar spine). The dropping sign is based on inability to maintain a position. A summary of the strength tests and their measurement properties is provided by Razmjou et al. [42].

Differential Diagnosis

Many shoulder and cervical spine pathologies have similar symptoms as those of rotator cuff tears. These include cervical radiculopathy, impingement syndrome, calcified tendonitis, biceps tendonitis, glenohumeral osteoarthritis, inflammatory arthritis, and so on. Clinically, active range of motion may be diminished in rotator cuff pathology. Passive range of motion remains unaffected in less significant tears if other pathologies such as frozen shoulder or arthritis are not present. Partial-thickness and small full-thickness rotator cuff tears are specifically hard to differentiate from the above conditions as they are more associated with pain than weakness. Clearly, the severity of weakness is correlated with the tear size and location. Major full-thickness tears affect mostly external rotation and to a lesser degree elevation of the arm.

Management of Rotator Cuff Tears

Management of rotator cuff tears depends on their cause (degenerative vs. traumatic), symptom severity, functional disability, and tear size. A summary of best practices is provided for each condition.

Partial-Thickness Rotator Cuff Tears

In a review by Finnan et al., the authors highlighted the lack of high-quality data on management of symptomatic partial-thickness rotator cuff tears compared with those available on full-thickness tears [55]. Neer proposed flattening of the underneath of the acromion process to reduce impingent of the supraspinatus tendon, which could lead to chronic rotator cuff pathology [7]. Generally, most surgeons do not repair partial-thickness rotator cuff tears, but if the tear is associated with an osseous impingement (osteophytes or enthesophyte), rotator cuff decompression is attempted. Surgical treatment of articular sided partial tear in athletes remains controversial.

Full-Thickness Rotator Cuff Tears

Although there are no strict guidelines for management of full-thickness rotator cuff tears, oral medication, injections, and physiotherapy are initially recommended, particularly for management of degenerative tears [56]. Surgical cuff decompression and repair are largely dependent on severity of symptoms, acuteness of the trauma, tendon quality, fatty infiltration, tear size, and functional limitations. Keener and colleagues have suggested useful directions with respect to risk of tear progression [24, 31, 32] that assist clinicians with surgical decision-making (see section on natural history of cuff pathology). Surgery may be considered for the purpose of functional recovery for painful, weak, and disabling tears refractory to medical treatment [56]. The tear size obviously affects the decision on how and when to repair the defect, although other factors influence the urgency of repairing a full-thickness tear. Zingg et al. [57] were able to demonstrate that patients with a moderately symptomatic massive rotator cuff tear could maintain satisfactory shoulder function for at least 4 years. However, significant progression of degenerative structural joint changes, and increasing risk of a reparable tear progressing to an irreparable tear is a chance that both patients and clinicians should be willing to take.

Historical Landmarks and Surgical Decision-Making

In the review by Adams et al., [1] the first cuff-related surgery is credited to Karl Hüter and George Clemens Von Perthes led to further development of cuff surgery in 1906. He carried out three rotator cuff repairs using suture anchors, with cat gut sutures, an innovation that reshaped the modern surgical methods of tendon to bone reattachment. According to a review by Tokish and Hawkins [58], the history of arthroscopic rotator cuff surgery goes back to 1931. According to this review, Burman performed a series of cadaveric diagnostic arthroscopies in the shoulder and correlated his findings after opening the specimens. Codman as one of the most influential figures in the field of shoulder surgery described the first modern surgical technique to repair supraspinatus tears in 1911 [59].

The open rotator cuff decompression was proposed in1972 by Charles Neer, an American orthopedic surgeon [7]. Neer proposed flattening of the underneath of the acromion process to reduce impingement of the supraspinatus tendon, which could lead to chronic rotator cuff pathology [7]. In 1985, almost 50 years after Burman's diagnostic arthroscopies on cadavers, James R. Andrews, an American orthopedic surgeon, adopted arthroscopic surgery for clinical use [60].

The literature on recovery following repair of full-thickness cuff tear indicates that although tear size affects recovery, it may not be a significant contributing factor in the overall recovery of disability. Burkhart et al. [61] found no differences in four categories of tear size (small, medium, large, massive) when evaluated pre- and postoperative UCLA scores. Razmjou et al. [62] found that although the rate of improvement was similar between patients with small/moderate and those with large/massive tears, the larger tears were associated with slightly more disability at 2 years following surgery. Apart from the tear size, patient's age and existence of fatty infiltration should be considered when contemplating surgical repairs. Elderly patients with large tears and a reasonable range of motion and mild symptoms should exhaust a comprehensive rehabilitation program before considering any repair as the success rate of repair of chronic degenerative tears with muscle fatty infiltration is not high. A systematic review of nine retrospective and two prospective studies, all of fair quality with a variety of tear sizes, has shown that despite a high potential for retear or persistent defects on imaging, repair of cuff tear after age 70 is associated with good outcomes and pain relief [63]. It is however important to bear in mind that since the tendon quality is compromised in elderly, surgical skills of the surgeon and avoiding too many simultaneous procedures play a critical role in the success of the cuff repair. Use of conjunct biceps tenodesis in elderly patients with mild tenosynovitis or degenerative partial tears will sacrifice the intra-articular segment of the biceps tendon and may produce instability and dysfunction and accelerate progression to cuff tear arthropathy [64]. The proper biceps loading leads to a significant decrease in both anterosuperior and superior glenohumeral translation [29]. In cases where biomechanics of the shoulder joint has already been disturbed due to an incompetent rotator cuff muscle that leads to significant superior subluxation of humeral head, the role of biceps becomes even more important. In addition, the risks pertaining to biceps tenodesis include increased operative time, infection, persistent bicipital groove pain, and failure of fixation and recurrence of Popeye deformity and should be justified considering a degenerative biceps pathology is often a normal finding in older patients. In summary, extensive alteration in biomechanics of the glenohumeral joint may lead to an accelerated superior subluxation of the humeral head and cuff tear arthropathy.

Surgical management of associated pathologies related to cuff tear such as geyser cysts is considered in symptomatic cysts. As noted, the type 1 cyst forms superficially in the presence of an intact rotator cuff and is limited to the AC joint. The type 2 cyst forms between the glenohumeral and the AC joints [35]. Repair of the rotator cuff tear is usually not possible for type 2 geyser cysts due to an extent of cuff tear. In the case of an irreparable defect, good results can be achieved through arthroscopic incision of the AC joint and resection of the cyst base. Aspiration and simple cyst excision are not recommended, because of the potential for recurrence, development of a draining sinus, and infection. In cases where the tendon cannot be repaired, lateral clavicle resection should be performed [37]. Hemiarthroplasty in patients with a combined cuff arthropathy and geyser cysts may be effective in indirectly decompressing these cysts [65].

References

1. Adam JR, Nanjayan SKT, Monga P. Management of rotator cuff tears - key historical landmarks. J Clin Orthop Trauma. 2021;18:6–12.
2. Smith JG. The classic: pathological appearances of seven cases of injury of the shoulder-joint: with remarks. 1834. Clin Orthop Relat Res. 2010;468(6):1471–5.
3. Smith JG. Occurrence of tendon rupture after shoulder injury. London Medical Gazette; 1834.
4. Codman EA. The shoulder, rupture of the supraspinatus tendon and other lesions in or about the subacromial Bursa. Boston: Todd Thomas; 1934.
5. Clark JM, Harryman DT 2nd. Tendons, ligaments, and capsule of the rotator cuff. Gross and microscopic anatomy. J Bone Joint Surg Am. 1992;74(5):713–25.
6. Burkhart SS, Esch JC, Jolson RS. The rotator crescent and rotator cable: an anatomic description of the shoulder's "suspension bridge". Arthroscopy. 1993;9(6):611–6.
7. Neer CS 2nd. Anterior acromioplasty for the chronic impingement syndrome in the shoulder: a preliminary report. J Bone Joint Surg Am. 1972;54(1):41–50.
8. Nho SJ, Yadav H, Shindle MK, Macgillivray JD. Rotator cuff degeneration: etiology and pathogenesis. Am J Sports Med. 2008;36(5):987–93.
9. Oh JH, Kim SH, Kim JH, Shin YH, Yoon JP, Oh CH. The level of vitamin D in the serum correlates with fatty degeneration of the muscles of the rotator cuff. J Bone Joint Surg Br. 2009;91(12):1587–93.
10. Gwilym SE, Watkins B, Cooper CD, Harvie P, Auplish S, Pollard TC, et al. Genetic influences in the progression of tears of the rotator cuff. J Bone Joint Surg Br. 2009;91(7):915–7.
11. Kim HM, Dahiya N, Teefey SA, Middleton WD, Stobbs G, Steger-May K, et al. Location and initiation of degenerative rotator cuff tears: an analysis of three hundred and sixty shoulders. J Bone Joint Surg Am. 2010;92(5):1088–96.

12. Minagawa H, Itoi E, Konno N, Kido T, Sano A, Urayama M, et al. Humeral attachment of the supraspinatus and infraspinatus tendons: an anatomic study. Arthroscopy. 1998;14(3):302–6.
13. Andarawis-Puri N, Ricchetti ET, Soslowsky LJ. Interaction between the supraspinatus and infraspinatus tendons: effect of anterior supraspinatus tendon full-thickness tears on infraspinatus tendon strain. Am J Sports Med. 2009;37(9):1831–9.
14. Adams CR, DeMartino AM, Rego G, Denard PJ, Burkhart SS. The rotator cuff and the superior capsule: why we need both. Arthroscopy. 2016;32(12):2628–37.
15. Vosloo M, Keough N, De Beer MA. The clinical anatomy of the insertion of the rotator cuff tendons. Eur J Orthop Surg Traumatol. 2017;
16. Razmjou H, Christakis M, Dwyer T, van Osnabrugge V, Holtby R. Accuracy of infraspinatus isometric testing in predicting tear size and tendon reparability: comparison with imaging and arthroscopy. J Shoulder Elb Surg. 2017;26(8):1390–8.
17. Kim HM, Dahiya N, Teefey SA, Keener JD, Galatz LM, Yamaguchi K. Relationship of tear size and location to fatty degeneration of the rotator cuff. J Bone Joint Surg Am. 2010;92(4):829–39.
18. Tuite MJ, Turnbull JR, Orwin JF. Anterior versus posterior, and rim-rent rotator cuff tears: prevalence and MR sensitivity. Skelet Radiol. 1998;27(5):237–43.
19. Vinson EN, Helms CA, Higgins LD. Rim-rent tear of the rotator cuff: a common and easily overlooked partial tear. AJR Am J Roentgenol. 2007;189(4):943–6.
20. Ellman H. Diagnosis and treatment of incomplete rotator cuff tears. Clin Orthop Relat Res. 1990;254:64–74.
21. DeOrio JK, Cofield RH. Results of a second attempt at surgical repair of a failed initial rotator-cuff repair. J Bone Joint Surg Am. 1984;66(4):563–7.
22. Yamaguchi K, Ditsios K, Middleton WD, Hildebolt CF, Galatz LM, Teefey SA. The demographic and morphological features of rotator cuff disease. A comparison of asymptomatic and symptomatic shoulders. J Bone Joint Surg Am. 2006;88(8):1699–704.
23. Keener JD, Steger-May K, Stobbs G, Yamaguchi K. Asymptomatic rotator cuff tears: patient demographics and baseline shoulder function. J Shoulder Elb Surg. 2010;19(8):1191–8.
24. Hsu J, Keener JD. Natural history of rotator cuff disease and implications on management. Oper Tech Orthop. 2015;25(1):2–9.
25. Razmjou H, Dwyer T, Holtby R. Impact of symptom bilaterality and hand dominance on patient-reported disability outcomes. SAGE Open Med. 2018;6:2050312118797566.
26. Roh MS, Wang VM, April EW, Pollock RG, Bigliani LU, Flatow EL. Anterior and posterior musculotendinous anatomy of the supraspinatus. J Shoulder Elb Surg. 2000;9(5):436–40.
27. Rahu M, Kolts I, Poldoja E, Kask K. Rotator cuff tendon connections with the rotator cable. Knee Surg Sports Traumatol Arthrosc. 2017;25(7):2047–50.
28. Mesiha MM, Derwin KA, Sibole SC, Erdemir A, McCarron JA. The biomechanical relevance of anterior rotator cuff cable tears in a cadaveric shoulder model. J Bone Joint Surg Am. 2013;95(20):1817–24.
29. Su WR, Budoff JE, Luo ZP. The effect of anterosuperior rotator cuff tears on glenohumeral translation. Arthroscopy. 2009;25(3):282–9.
30. Duralde XA. How important is the anterior rotator cuff cable?: commentary on an article by Mena M. Mesiha, MD, et al.: "the biomechanical relevance of anterior rotator cuff cable tears in a cadaveric shoulder model". J Bone Joint Surg Am. 2013;95(20):e156.
31. Keener JD. Editorial commentary: progression of degenerative full-thickness rotator cuff tears: are we finally using natural history data to define at-risk tears? Arthroscopy. 2019;35(1):235–6.
32. Keener JD, Patterson BM, Orvets N, Chamberlain AM. Degenerative rotator cuff tears: refining surgical indications based on natural history data. J Am Acad Orthop Surg. 2019;27(5):156–65.
33. Craig EV. The geyser sign and torn rotator cuff: clinical significance and pathomechanics. Clin Orthop Relat Res. 1984;191:213–5.
34. Hiller AD, Miller JD, Zeller JL. Acromioclavicular joint cyst formation. Clin Anat. 2010;23(2):145–52.
35. Negi RS, Gogoi B, Randhawa AS. MR geyser sign in chronic rotator cuff tears. Med J Armed Forces India. 2018;74(3):304–6.

36. Tshering Vogel DW, Steinbach LS, Hertel R, Bernhard J, Stauffer E, Anderson SE. Acromioclavicular joint cyst: nine cases of a pseudotumor of the shoulder. Skelet Radiol. 2005;34(5):260–5.
37. Cho CH. Complicated acromioclavicular joint cyst with massive rotator cuff tear. Am J Orthop (Belle Mead NJ). 2014;43(2):70–3.
38. Cyriax J. Textbook of orthopaedic medicine: diagnosis of soft tissue lesions. 8th ed. London, England: Bailiere-Tindall; 1982.
39. Pettman E. A history of manipulative therapy. J Man Manip Ther. 2007;15(3):165–74.
40. Razmjou H, Holtby R, Myhr T. Pain provocative shoulder tests: reliability and validity of the impingement tests. Physiother Can. 2004;56(4):229–36.
41. Park HB, Yokota A, Gill HS, El Rassi G, McFarland EG. Diagnostic accuracy of clinical tests for the different degrees of subacromial impingement syndrome. J Bone Joint Surg Am. 2005;87(7):1446–55.
42. Razmjou H, Christakis M, Dwyer T, van Osnabrugge V, Henry P, Nam D, et al. Diagnostic accuracy of clinical tests in detecting rotator cuff pathology. Orthop Sports Med Open Access J. 2019;2(4):170–9.
43. Powell JW, Huijbregts PA. Concurrent criterion-related validity of acromioclavicular joint physical examination tests: a systematic review. J Manual Manipulative. 2006;14(2):19–29.
44. Hermans J, Luime JJ, Meuffels DE, Reijman M, Simel DL, Bierma-Zeinstra SM. Does this patient with shoulder pain have rotator cuff disease?: the rational clinical examination systematic review. JAMA. 2013;310(8):837–47.
45. Hegedus EJ, Goode A, Campbell S, Morin A, Tamaddoni M, Moorman CT 3rd, et al. Physical examination tests of the shoulder: a systematic review with meta-analysis of individual tests. Br J Sports Med. 2008;42(2):80–92. discussion 92
46. Beaudreuil J, Nizard R, Thomas T, Peyre M, Liotard JP, Boileau P, et al. Contribution of clinical tests to the diagnosis of rotator cuff disease: a systematic literature review. Joint Bone Spine. 2009;76(1):15–9.
47. Hegedus EJ, Goode AP, Cook CE, Michener L, Myer CA, Myer DM, et al. Which physical examination tests provide clinicians with the most value when examining the shoulder? Update of a systematic review with meta-analysis of individual tests. Br J Sports Med. 2012;46(14):964–78.
48. Itoi E, Minagawa H, Yamamoto N, Seki N, Abe H. Are pain location and physical examinations useful in locating a tear site of the rotator cuff? Am J Sports Med. 2006;34(2):256–64.
49. Yuen CK, Mok KL, Kan PG. The validity of 9 physical tests for full-thickness rotator cuff tears after primary anterior shoulder dislocation in ED patients. Am J Emerg Med. 2012;30(8):1522–9.
50. Jobe FW, Moynes DR. Delineation of diagnostic criteria and a rehabilitation program for rotator cuff injuries. Am J Sports Med. 1982;10(6):336–9.
51. Neer C. Shoulder reconstruction. Philadelphia, etc. In: Neer CS, editor. Anatomy of shoulder reconstruction. W. B. Saunders Company; 1990. p. 1–39.
52. Arthuis M. Obstetrical paralysis of the brachial plexus. I. Diagnosis. Clinical study of the initial period. Rev Chir Orthop Reparatrice Appar Mot. 1972;58(Suppl 1):124–6.
53. Gerber C, Krushell RJ. Isolated rupture of the tendon of the subscapularis muscle. Clinical features in 16 cases. J Bone Joint Surg Br. 1991;73(3):389–94.
54. Gerber C, Hersche O, Farron A. Isolated rupture of the subscapularis tendon. J Bone Joint Surg Am. 1996;78(7):1015–23.
55. Finnan RP, Crosby LA. Partial-thickness rotator cuff tears. J Shoulder Elb Surg. 2010;19(4):609–16.
56. Beaudreuil J, Dhenain M, Coudane H, Mlika-Cabanne N. Clinical practice guidelines for the surgical management of rotator cuff tears in adults. Orthop Traumatol Surg Res. 2010;96(2):175–9.
57. Zingg PO, Jost B, Sukthankar A, Buhler M, Pfirrmann CW, Gerber C. Clinical and structural outcomes of nonoperative management of massive rotator cuff tears. J Bone Joint Surg Am. 2007;89(9):1928–34.

58. Tokish JM, Hawkins RJ. Current concepts in the evolution of arthroscopic rotator cuff repair. JSES Rev Rep Tech. 2021;1(2):75–83.
59. Codman EA. Complete rupture of the supraspinatus tendon. Operative treatment with report of two successful cases. 1911. J Shoulder Elb Surg. 2011;20(3):347–9.
60. 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.
61. Burkhart SS, Danaceau SM, Pearce CE Jr. Arthroscopic rotator cuff repair: analysis of results by tear size and by repair technique-margin convergence versus direct tendon-to-bone repair. Arthroscopy. 2001;17(9):905–12.
62. Razmjou H, Holtby R, Wessel J, Alexander P, Moola F. Size of tear as a prognostic indicator for outcome following rotator cuff full-thickness tear repair. J Bone Joint Surg Br Proc. 2008;90-B:139.
63. Altintas B, Anderson NL, Pitta R, Buckley PS, Bhatia S, Provencher MT, et al. Repair of rotator cuff tears in the elderly: does it make sense? A systematic review. Am J Sports Med. 2020;48(3):744–53.
64. Kumar VP, Satku K, Balasubramaniam P. The role of the long head of biceps brachii in the stabilization of the head of the humerus. Clin Orthop Relat Res. 1989;244:172–5.
65. Cooper HJ, Milillo R, Klein DA, DiFelice GS. The MRI geyser sign: acromioclavicular joint cysts in the setting of a chronic rotator cuff tear. Am J Orthop (Belle Mead NJ). 2011;40(6):E118-21.

Chapter 5
Cuff Tear Arthropathy

Cuff tear arthropathy (CTA) is a progressive and often devastating cuff disease that is characterized by extensive rotator cuff deficiency and superior instability of the glenohumeral joint and may be associated with a destructive inflammatory process. The abnormal glenohumeral biomechanics associated with superior excursion of the humeral head lead to gradual destruction of the cartilage and joint space narrowing both superiorly and medially. The simultaneous destruction of soft tissues, bony structures, and joint surfaces make the CTA a unique and challenging condition to manage. To date, the role of calcium phosphate crystals in the development of CTA remains controversial, and this adds to the complexity of its clinical management. Considering that the richly illustrated literature on this condition goes back to the nineteenth century, a detailed history of its discovery is warranted.

Historical Perspective

The first description of cuff related arthropathy was in the nineteenth century. One of the most unique features of CTA is the superior migration of the humeral head which was described in full detail by a Dutch physician and anatomist Eduard Sandifort and his son Gerard Sandifort in 1793. The description was based on the oldest Dutch collection of human specimens kept in the University of Leiden in the Netherlands. The Sandiforts felt that superior subluxation was secondary to traumatic injuries [1].

Despite lack of sophisticated imaging (the first X-ray technology was discovered by Röntgen in 1895), an accurate description of the CTA was provided by a number of physicians and anatomists in the mid-1880s. In 1845, in an autopsy report by Alfred Smee, an English senior surgeon and chemist [2], presence of a massive rotator cuff tear associated with the long head of biceps rupture, an upward migration of

H. Razmjou, M. Christakis, *Clinical and Radiological Examination of the Shoulder Joint*, https://doi.org/10.1007/978-3-031-10470-1_5

the humeral head, and abnormal articulation between the humeral head and the inferior part of the acromion were described as the hallmarks of this condition.

The most detailed information on this disease is credited to two Irish surgeons, Robert William Smith [3, 4] and Robert Adams [5]. These authors described a chronic massive rotator cuff tear with destructive glenohumeral changes, a form of chronic arthritis that resembled rheumatoid arthritis. In 1853, Robert William Smith described the biomechanics of the long head of the biceps and underlined the importance of its absence in the development of abnormal elevation of the humeral head to which the name “partial luxation upward” was given. He felt that the upward elevation of the humeral head was a leading factor to humeral head destruction caused by an external trauma or chronic rheumatic arthritis. Around the same time, Robert Adams reported the presence of this condition in his autopsy drawings [5]. He similarly described a condition that was associated with chronic rheumatic arthritis with upward partial luxation of the humeral head. In his drawings, the long tendon of the biceps was ruptured and subluxed or reinserted in the groove without connection with the scapula but adherent to the highest part of the bicipital groove in the humerus. Adams noted that osseous changes and superior migration of the humeral head could occur within the frame of chronic rheumatic arthritis without any history of trauma. In his report and anatomical drawings, Adams described the development of a new articular surface on the upper part of the glenoid cavity and on the root of the coracoid process. He also described thickened and hypertrophied capsular ligaments with the overdistension of the capsule with a large amount of synovial fluid. Adams observed the deposits of pieces of “foreign bodies” in the capsular ligaments with femoralization of the humeral head in more advanced cases and noted an overall joint swelling, bony prominence, and elevation of the humeral head in his report [5].

About 80 years later in 1934, a pioneer American surgeon, Ernest Amory Codman, described a relationship between rotator cuff pathology and glenohumeral arthritis [6]. He presented a case of a woman in her 50s, with recurrent swelling of the shoulder, massive tear of the rotator cuff, cartilaginous bodies attached to the synovial tissue, and severe destructive glenohumeral arthritis.

Charles Neer, an American orthopedic surgeon, coined the term “cuff tear arthropathy” in 1977 [7]. In 1983, further description of CTA as a clinical entity was published by Neer, Craig, and Fukuda [8]. They stated that “We could find no description of this lesion in the literature prior to brief reports by the senior one of us (C.S.N.,II) who introduced the term cuff-tear arthropathy” (p. 1232) [8]. The authors described a typical swelling in the shoulder joint from the presence of synovial fluid that communicated between the glenohumeral joint and the subacromial bursa. They noted presence of a massive rotator cuff tear, rupture or dislocation of the long head of biceps, atrophy of the glenohumeral articular cartilage, and osteoporosis of the subchondral humeral bone as the pathoanatomical changes of the CTA. The upward displacement of the humeral head leading to superior instability of the humeral head, erosion of the anterior part of the acromion, and the humeral head collapse were noted to occur in final stages of this progressive disease [8].

A few years later in 1981, a reference to an inflammatory condition that affected the shoulder joint was made by Daniel McCarty, an orthopedic surgeon from Milwaukee, Wisconsin. He described a form of arthropathy associated with recurrent shoulder effusions, massive tear of the rotator cuff, and advanced radiographic destructive changes of the glenohumeral joints [9–11]. He and his colleagues designated the term “Milwaukee shoulder” to this constellation of findings that represented an inflammatory disease.

In summary, the detailed descriptions of the pathology of the CTA are found in illustrations and anatomical notes of Sandifort [1], Smith [3, 4], Canton [12], Adams [5], and Codman [6]. In a comprehensive review of the historical clinical notes, autopsy reports, and illustrations from the nineteenth-century written sources, Brorson [13] reports that the pathoanatomical and biomechanical changes later termed “CTA” were well understood and described in the medical literature at the time. The rich collection of details and drawings on the unique hallmarks of this condition, including altered humeral head anatomy, degenerative changes of the long head of the biceps, superior migration of the humeral head, and remodeling of the anterior part of the acromion and acromioclavicular joint, are all indicative of the knowledge of the former scientists in this area [13]. Even today’s terminologies used in analogy with the hip joint, such as humeral head femoralization or acetabularization of the acromion that referred to osseous changes in advanced cases, were used and described in illustrations by Canton in 1855 [12] and Adams in 1857 [5]. Therefore, while the term CTA was initially used by Neer, his description of the condition was very similar to what had been described by Canton [12], Smith [3, 4], Adams [5], and others more than a century before him. Considering the lack of access to sophisticated imaging technology and histological or hematological testing in the mid-1880s, the acknowledgment of the contribution of these pioneer clinicians and anatomists should not be understated and deserve a full credit by the present-day clinicians.

Characteristics, Classification, and Causes

The exact etiology of cuff-related arthropathy is not well understood. As noted, an early description of cuff arthropathy points out to an inflammatory disease. In 1981, long after documenting the association between this condition and a rheumatic arthritis, McCarthy and collogues proposed an inflammatory-mediated theory based on the elevated levels of calcium phosphate crystals within the affected joints [9–11]. Calcium phosphate crystals in synovial fluid and tissue can induce a low-grade inflammatory response which would initiate cellular and fibroblast proliferation. These basic calcium phosphate crystals induce the synthesis of proteolytic enzymes that are responsible for the degradation of cartilage matrix components. Human fibroblasts may then secrete enzymes that cause rapid degradation of the cartilage matrix components [14]. Predisposing factors include deposition of calcium pyrophosphate dihydrate crystals, direct trauma or chronic joint overuse, chronic renal failure, and denervation [9].

The biomechanical model that pointed to the vital role of muscle force couples in the development of superior subluxation of the humeral head was first mentioned in 1944, when Verne T. Inman and two of his more senior colleagues, Saunders and Abbott, proposed the concept of force couple theory [15]. The muscle force couples exist in the "coronal" and "transverse" planes and represent the contribution of rotator cuff to dynamic joint stability during active arm elevation. Inman et al. described the *coronal force couple* as a balanced force created between the deltoid and the inferior cuff muscles (infraspinatus, subscapularis, and teres minor). The coronal force couple provides the superior-inferior stability by depressing the humeral head against the upward force of the deltoid during active abduction. In Chap. 4, it was noted that the supraspinatus has two distinct anterior and posterior fibers [16] and there is a distinct functional difference between the anatomic regions of the supraspinatus. Tears of the anterior supraspinatus are more disabling than the posterior region, because the anterior fibers bear significantly higher levels of stress and their tears often affect the rotator cable, a structure that spans from the biceps tendon to the inferior border of the infraspinatus. In addition, the anterior supraspinatus is involved in both internal and external rotation in the scapular plane depending on the initial position of the humerus, where the posterior supraspinatus appears to act as an external rotator only [17, 18]. We also learned that the degenerative rotator cuff tears initiate posteriorly from the junction between the supraspinatus and infraspinatus or within the infraspinatus tendon and are often asymptomatic or minimally symptomatic [19] because they do not affect the rotator cable, the primary load-bearing structure within the supraspinatus. In the presence of an intact rotator cuff cable, the rotator cuff crescent insertion is rather stress-shielded and plays a significantly lesser role in supraspinatus force transmission [20]. Larger tears located in the anterior supraspinatus tendon that interrupt the rotator cable, on the other hand, are most at risk for tear propagation [21] and higher levels of disability.

The *transverse force* couple was first described by Amulya Kumar Saha, an Indian born surgeon who completed his research about "theories of shoulder mechanism" in Liverpool in 1971 [22]. Saha extensively studied the functional anatomy, morphology, and imaging features of the shoulder and performed electrophysiological studies on role of different muscles in the shoulder joint. He described the transverse couple force between the subscapularis anteriorly and the infraspinatus and teres minor posteriorly. The transverse force couple provides anteroposterior glenohumeral stability throughout active abduction. Disruption of coronal plane force couple results in superior head migration, while disruption of the transverse force couple results in loss of concavity compression, decreased active abduction, and the subluxation of the humeral head posteriorly (in the presence of infraspinatus tear) and anteriorly (in the presence of subscapularis tear) [22]. In 1991, Burkhart et al. [23] suggested that the biomechanical integrity of the rotator cuff was more important than its anatomical soundness and, as long as coronal and transverse force couple forces were intact, patients could elevate the arm and function reasonably well. This would explain why a partial repair of a massive cuff tear that restored the couple forces could improve function. More recent cadaver or electromyographic studies have confirmed the vital role of the infraspinatus and especially subscapularis on transverse plane motions. Su et al. confirmed that infraspinatus tears predisposed the shoulder to posterosuperior glenohumeral translation [24] and tears of lower component of the subscapularis predisposed the shoulder to anterosuperior translation [25]. In both studies [24, 25], the supraspinatus tear had minimal impact on the transverse plane movements as long as the anterior rotator cable remained intact. In summary, the biomechanical integrity of each rotator cuff structure is quite unique and has a significant impact on the overall presentation of symptoms and disability. As it relates to biomechanical factors, the tear location in the supraspinatus (anterior vs. posterior) and infraspinatus is important in maintaining the coronal plane force couple, and the tear location in the subscapularis (lower vs. upper fibers) is more important in the transverse force couple, which its absence is a prerequisite for development of the CTA.

In 1983, Neer [8] suggested that both mechanical and nutritional reasons were responsible for development of the CTA. He hypothesized that the mechanical factors associated with massive rotator cuff tears lead to unbalanced muscle forces and superior migration of the humerus, which would impose destructive forces. The nutritional factors associated with massive full-thickness tears were hypothesized to be related to loss of motion and periarticular damage. Neer felt that the torn cuff facilitates loss of nourishing factors and a reduction in the joint fluid pressures required for nutrients to disperse through the articular cartilage. The loss of fluid pressure and the accompanying reduction in the chemical content of synovial fluid lead to cartilage and bone atrophy [8]. A few years later, Collins and colleagues synthesized all proposed theories and suggested that in CTA the humeral head escapes anterosuperiorly through the massively torn cuff. As a result of mechanical impact of the humeral head and cartilage fragmentation and particulate debris, an enzymatic response is initiated, setting off the previously described crystal-mediated inflammatory cascade. The loss of joint space and overall inactivity contribute to articular cartilage atrophy and disuse osteoporosis of the subchondral bone, and over time further deterioration of the articular surface will occur [26].

Prevalence of CTA

There is a significant number of articles on prevalence or incidence of full-thickness rotator cuff tears both before and after surgical interventions, but the information on frequency of the CTA is not clearly stated in the literature. The age of patients suffering from CTA usually ranges from the mid-60s to late-80s [11, 27–30]. Although the exact prevalence of CTA is not stratified by patient sex, this condition is more prevalent in elderly women, and the highest rate of having reverse shoulder arthroplasty is reported in women after 75 years of age [31]. Although both shoulders are often involved in cuff tears, the vulnerability of the dominant side to cuff tears in general has been reported [32]. Certain sports that are associated with high-energy activity may also accelerate the cuff tear progression [33]. Neer estimated that only 4% of patients with a rotator cuff tear progress to cuff arthropathy [8]. Therefore, although a massive tear is a requirement for CTA, not all patients with a massive tear will develop CTA. This fact may give more merit to the inflammatory-mediated theory. Patients with massive rotator cuff tears who continue to maintain a well-compensated forward elevation of above 90° and a reasonable overall function often do not develop CTA, although at present, there is no clear information on which tears will progress to CTA.

While the exact prevalence of CTA is not very clear postoperatively, there is evidence that CTA could develop following an unsuccessful rotator cuff repair. The surgery-induced CTA is often devastating because the postoperative pain and dysfunction are more significant than prior to surgery. One important consideration by the advanced practice physiotherapists and family physicians is to make sure that in failed rotator cuff repairs that have progressed to CTA, infection is ruled out. In a study by Misir et al. [34], the rate of development of CTA was 11.5% (36 of 312 patients). Factors that increased the rate of CTA development after a cuff repair were poor integrity of the supraspinatus tendon after repair, tear size, higher fatty infiltration, and early postoperative pseudoparalysis. In this study, early postoperative pseudoparalysis was found to be an independent risk factor in the development of CTA [34].

Clinical Findings of Cuff Tear Arthropathy

Symptoms of CTA involve pain, swelling, significant weakness, night pain, and increased discomfort with activity. Neer has mentioned an average pain duration of 10 years, although most patients recall a recent traumatic event that has set off their disability.

Clinical observation of patients with CTA often shows a visible soft swelling and an anterosuperior subluxation of the humeral head (Fig. 5.1). Atrophic changes of the supraspinatus and infraspinatus muscles and diffuse wasting of other muscles such as the deltoid are quite common.

Clinical examination is consistent with involvement of active and passive range of motion due to a dysfunctional rotator cuff and fixed glenohumeral subluxation and destruction of glenohumeral joint in more severe cases. Crepitus that is a hallmark of primary osteoarthritis is not always present in patients with CTA.

Range of Motion Assessment

Range of motion restriction in CTA is variable depending on the patient's age, sex, tear size, and activity level. Active external rotation is almost always severely affected, although passively it may be only moderately limited or not affected at all.

Fig. 5.1 Anterosuperior scape of the humeral head and moderate swelling of the glenohumeral joint in an 81-year-old man suffering from CTA. The swelling is caused by the leakage of synovial fluid through the ruptured rotator cuff into the subacromial area

Despite the fact that the supraspinatus and infraspinatus are often absent in this condition, the compressive effect of the subscapularis, teres minor, and scapular-stabilizing muscles may allow a functional active elevation of the shoulder.

In general, the isolated tears of the supraspinatus or combined tears of the supraspinatus and superior infraspinatus have minimal impact on the shoulder function, because the rotator cuff cable (see Chap. 4) remains intact in such cases. The location of tear in the subscapularis also makes a difference in how much of the normal biomechanics is altered. As noted earlier, evidence shows that tears involving supraspinatus and superior subscapularis interrupt the rotator cable and alter the normal kinematics of the glenohumeral joint, but it is the tears of the inferior half of the subscapularis that play an important role in maintaining the anterior force couple and have a significant impact on elevation and function [23, 25]. Therefore, inability to elevate the arm is often indicative of involvement of the subscapularis (more specifically lower fibers) and/or teres minor [25]. Clinically, physically active patients who are involved is sports in older ages and do not recall a single traumatic event often keep a well-maintained elevation regardless of the cuff tear size and severity of the CTA. This may be explained by the compensating teres minor and an efficient use of scapular-stabilizing muscles.

Pseudoparalysis

As a part of range of motion examination, presence of pseudoparalysis should be ruled out or confirmed as it affects prognosis and response to surgery. While the exact definition of pseudoparalysis is not agreed upon, this phenomenon is defined as active forward elevation of less than 90° with full preservation of passive range of motion. In a study by Denard et al. [35], presence of pseudoparalysis predicted a disruption of rotator cable attachment with a high specificity. In a study by Rhee et al. [36], the loss of volume in inferior subscapularis muscle was associated with presence of pseudoparalysis. Holtby and Razmjou [37] found the higher percentage of pseudoparalysis in massive vs. large tears and in U-shaped vs. crescent-shaped tears. The U-shaped tears have a larger mediolateral component which may affect the rotator cable. In a more recent study [38], patients with a healthy rotator cuff (control group) were compared with patients with active scapular plane abduction of less than 45° (pseudoparalysis group) and patients with active abduction of more than 45° and less than 90° (pseudoparesis group). The chronic pseudoparalysis group always had more than 50% of the subscapularis involvement with stage 3 fatty infiltration. The superior subscapularis showed smaller muscle volume in the pseudoparesis compared with the control group. More importantly, the pseudoparalysis group had a smaller inferior subscapularis than both the control group and the group pseudoparesis. Overall, all these studies highlight the importance of inferior subscapularis muscle in maintaining the transverse couple force in the shoulder joint.

Strength-Related Clinical Tests

Isometric Testing in Neutral The weakness of a muscle can be easily detected by isometric testing in neutral position [39]. James Cyriax, a British orthopedic surgeon known as the father of orthopedic medicine, worked closely with skilled physiotherapists and introduced the concept of selective muscle testing in the early 1980s. He noted that the isometric external rotation in neutral position did not affect the joint capsule, ligaments, or bursae and could provide a more accurate examination of the damaged muscle/tendon in patients with a frozen shoulder or osteoarthritis who have limited active and passive external rotation [39]. Although it is believed the supraspinatus muscle acts as the abductor, it does participate in rotational motions of the humeral head as well [17]. Moreover, there is evidence that isolated tears of the supraspinatus could be associated with fatty atrophy of the infraspinatus [40]. Therefore external rotation in neutral is not only assessing the infraspinatus muscle but also can provide a clear picture of integrity of both muscles.

The Dropping Sign This clinical sign can be observed in upright (standing/sitting) position at 0° of abduction and 45° of external rotation when pushing against an examiner's hand. In a positive test, the forearm is dropped back to neutral position. Research has shown that patients with more severe cuff pathology will show more weakness in 45° of external rotation than in neutral position and the test is not specific to the infraspinatus [41]. This is related to the length dependency of isometric force generation of a healthy muscle during daily activities [42] which is greatest when the muscle has the same length that it has in the body [43]. As a result, strength is normally decreased at the shorter muscle lengths [44] which theoretically makes the dropping sign more sensitive in patients with CTA.

The Hornblower Sign A positive Hornblower sign [45] is probably the most commonly seen clinical sign in patients with CTA (see Chap. 4). To elicit this clinical sign, patient is asked to bring the affected or both hands to the mouth, pretending the act of eating. In cases where the patient is not able to reach the mouth without abducting the affected arm, the sign is considered positive.

The Lift-off Test The other clinical test that may be positive in patients with an advanced CTA is the lift-off test [46]. This lift-off test examines the integrity of the subscapularis involved in lifting the hand of the affected side away from the lumbar spine. Razmjou et al. [41, 47], who have examined the accuracy of the shoulder clinical tests against arthroscopy, reported that the dropping sign and Hornblower sign were highly specific in predicting a major tear, advanced fatty infiltration, and associated infraspinatus tear, which are the common features of the CTA. The lift-off test was reported to have high specificity for the overall tear size detection and reparability and subscapularis full-thickness tear [47, 48]. The strength-related clinical tests are reported to be helpful in clinical decision-making in relation to tear size prediction, ability of the surgeon to achieve a full repair, and presence of fatty infiltration only when they are positive, whereas a negative dropping or Hornblower sign or lift-off test does not exclude the possibility of the above conditions [47].

Imaging Investigations

Apart from the above clinical findings, presence of CTA needs to be confirmed with specific radiographic criteria, including the anterosuperior subluxation of the humeral head, superior migration of the humeral head in relation to the glenoid (acromiohumeral interval <6mm), collapse of the subchondral bone in humeral head, degenerative changes in the glenohumeral joint, and presence of femoralization and acetabularization bony abnormalities (see Chap. 11 for more details). One important fact to remember is that if the patient presents with severe cuff atrophy, marked weakness of external rotation, positive pseudoparalysis, and positive Hornblower sign, there is no need for ordering MRI as the findings will not change patient's management. In such cases, patients whose pain and disability have affected their health-related quality of life may consider reverse shoulder arthroplasty. The imaging investigation conducted before shoulder arthroplasty is the computed tomography (CT) scan to examine the integrity of bony structures and planning for surgical purposes.

Management of Cuff Tear Arthropathy

Although a reparable tear may progress to an irreparable tear, there is evidence that patients with a nonoperatively managed, moderately symptomatic massive rotator cuff tear can maintain satisfactory shoulder function for at least 4 years despite significant progression of degenerative structural joint changes [49]. Considering the destructive and complex nature of CTA, a higher incidence of medical comorbidities in the older population, and challenging surgical treatments, most surgeons do exhaust conservative treatment before attempting surgery. Conservative treatment includes nonsteroidal anti-inflammatory medications, intra-articular corticosteroid injections conducted under sonography and rehabilitation. Recurrent glenohumeral effusions are commonly reported with blood-tinged effusions seen in a smaller percentage of patients [8, 50]. Aspiration of fluid may be beneficial in patients with an inflammatory component and larger amounts of effusion.

Physical therapy in CTA should emphasize maintaining passive range of motion and compensating for the deficient rotator cuff by improving the compressive effect of the teres minor, scapular-stabilizing muscles, and subscapularis if intact. Strengthening exercises of the isolated external rotators of the shoulder are often not very successful due to complete rupture of the supraspinatus, infraspinatus, and in some cases subscapularis. Considering that the teres minor is the only muscle apart from the infraspinatus that has an appreciable moment arm for external rotation torque generation, its rehabilitation is highly encouraged [51, 52]. In a clinical commentary by Razmjou et al. [53], the authors suggested to strengthen the teres minor and posterior deltoid by using two resistance bands. In this exercise, the patient resists against abduction and external rotation simultaneously. Strengthening of the scapular stabilizers (trapezius, serratus anterior, rhomboids, and levator scapula muscles) play an important role in improving a cuff-deficient shoulder. The greatest activation for the trapezius, serratus anterior, and anterior and middle deltoid is reported to occur with external rotation at 90 of abduction [54].

According to Saha [22], the action of the muscles of the shoulder joint complex is affected by the anatomical position of the arm. One of the most fascinating muscles, whose function changes with different levels of elevation, is the subscapularis. This muscle is an internal rotator when the arm is by the side but becomes an external rotator toward the end of elevation. Reed et al. confirmed that the subscapularis activation is affected by the level of abduction of the shoulder joint [55]. The upper fibers of subscapularis act as an agonist for internal rotation and are strengthened with internal rotation [56]. The lower subscapularis fibers act as the humeral head depressor and anterior stabilizer [57] and are active in external rotation in the abducted position. The clinical implication of the higher-level activation of lower subscapularis fibers during abduction, flexion, and external rotation movements in both concentric and eccentric phases is to rehabilitate this muscle in elevation rather than neutral position. To improve internal rotation in patients with CTA, strengthening of the latissimus dorsi and pectoralis major which act as internal rotators throughout abduction and flexion is also encouraged [58].

With respect to surgical options, rotator cuff decompression and debridement may be considered in low-demand patients who do not wish to undergo a major surgery due to medical reasons or comorbidity but have an osteophyte or enthesophyte pressing on the compromised cuff tendons. Arthroscopic surgery is obviously less invasive as it does not violate the insertion of the deltoid. Decompression should not include a release of the coracoacromial ligament (CAL) as this will increase the risk of anterosuperior escape as the disease progresses [59]. Removing the osteophytes and loose bodies and minor flattening and shaping of the acromion may help with symptoms in younger patients. Because of anterosuperior instability of the glenohumeral joint, anatomical total joint replacement is not indicated in patients with CTA. A deficient cuff and abnormal contact forces on the superior aspect of the glenoid component of the prosthesis increase the tendency to develop glenoid loosening.

The other surgical option is humeral hemiarthroplasty which is slowly being replaced by reverse arthroplasty. A number of studies [50, 60–64] have examined the results of humeral hemiarthroplasty with or without rotator cuff repair for CTA. Using Neer's limited goals of achieving only 90° active elevation, 30° active external rotation, and significant pain relief, the results of the studies published in the late 1990s and early to mid-2000 show a moderate satisfaction rate of 67–89%. For the same reason noted above, the CAL should be preserved to enhance the stability of the glenohumeral joint after hemiarthroplasty. One of the complications of hemiarthroplasty is bone loss in glenoid and acromion [62], but humeral hemiarthroplasty remains the treatment of choice for younger active patients with an intact CA arch, minimal superior humeral migration, and a potentially repairable rotator cuff who have, at least, active forward elevation of 90°.

Reverse total shoulder arthroplasty (RTSA) is among the commonly used surgical options that is gaining momentum among orthopedic surgeons. RTSA was first introduced in early 1970s [65]. The use of early prostheses was discontinued because of multiple complications that led to loosening of the implant due to absence of the rotator cuff and the deltoid muscle subluxing effect. In late 1970s, the modern version of the RTSA was introduced by Paul Grammont, a French orthopedic surgeon [65–67].

In reverse arthroplasty, the position of the ball and socket is reversed to increase the deltoid lever arm and to assist with elevation of the arm in the presence of a deficient rotator cuff. The medialized center of rotation in reverse arthroplasty helps the recruitment of the anterior and posterior deltoid to act as abductors. Moreover, lowering the humerus in relation to the acromion increases the tension of the deltoid fibers. These biomechanical changes allow the deltoid to efficiently compensate for the absent or deficient rotator cuff muscles [65].

The RTSA was approved in the United States in 2003. Today, the indications of RTSA have increased to include massive irreparable rotator cuff tears in the absence of osteoarthritis, proximal humerus fractures, glenohumeral osteoarthritis with excessive posterior glenoid erosion, and revisions for failed anatomical arthroplasty [68–74]. Even though the traditional role of arthroplasty procedure was to address arthritis-related changes, this role continues to be evolving, particularly in patients without any evidence of associated glenohumeral arthritis. More recently, RTSA is being increasingly advocated to help with pain relief and improving function in patients with massive rotator cuff tear without degenerative changes in the glenohumeral joint. Caution however is warranted when considering RTSA in patients with massive rotator cuff tears with well-maintained active forward elevation (greater than 90°) or those without pseudoparalysis (inability to actively elevate arm with a full passive elevation) as the results may not be as impressive as the patient may expect. More importantly, deltoid weakness and cervical spine pathology have to be ruled out in candidates for RTSA [75]. Preoperative impairment in deltoid muscle may not be an absolute contraindication for RSTA [76], but due to heavy reliance of the reverse arthroplasty biomechanics on the function of the deltoid, its integrity should be considered to avoid dislocation.

Complications noted in association with reverse arthroplasty are scapular notching [77] and glenoid and humeral radiolucencies and polyethylene wear [78]. A large study of 527 reverse shoulder arthroplasties reported progressive radiographic changes after 5 years with increasing frequency of large notches [79]. They expressed concern regarding the longevity of the reverse shoulder arthroplasty, especially in younger patients [79].

The postoperative physiotherapy program requires a sophisticated rehabilitation program as the role of muscles changes in RTSA as compared to the native shoulder. In a review of the literature and based on the principles of pertinent muscle loading, Razmjou et al. have proposed a comprehensive postoperative rehabilitation program for cuff-deficient shoulders [53]. Considering the stabilizing impact of the deltoid is more evident when the arm is in the elevated position [80], most exercises are conducted in higher levels of scapular elevation.

References

1. Sandifort E, Sandifort G. Museum anatomicum Academiae Lugduno-Batavae. Samuel Luchtmans, Leiden. Nederlands: Lugduni Batavorum:Samuel et Johannes Luchtmans; 1793.
2. Smee A. Dissection of a partial dislocation of the shoulder upwards. Lancet. 1845;1:323–4.
3. Smith RW. Observations upon chronic rheumatic arthritis of the shoulder [part II]. Dublin Quart J Med Sci. 1853;15:343–58.
4. Smith RW. Observations upon chronic rheumatic arthritis of the shoulder [part I]. Dublin Quart J Med Sci. 1853;15:1–16.
5. Adams R. Illustrations of the effects of rheumatic gout or chronic rheumatic arthritis on all the articulations. With descriptive and explanatory statements. London: John Churchill and Sons; 1857. p. 1–31.
6. Codman EA. The shoulder. The Shoulder. New York: Miller & Co. Medical Publishers Inc.; 1934. p. 216–24.
7. Neer CS, Cruess RL, Sledge CB, Wilde AH. Total glenohumeral replacement. A preliminary report. Orthop Trans. 1977;1:244–5.
8. Neer CS 2nd, Craig EV, Fukuda H. Cuff-tear arthropathy. J Bone Joint Surg Am. 1983;65(9):1232–44.
9. McCarty DJ, Halverson PB, Carrera GF, Brewer BJ, Kozin F. "Milwaukee shoulder"—association of microspheroids containing hydroxyapatite crystals, active collagenase, and neutral protease with rotator cuff defects. I. Clinical aspects. Arthritis Rheum. 1981;24(3):464–73.
10. Garancis JC, Cheung HS, Halverson PB, McCarty DJ. "Milwaukee shoulder"—association of microspheroids containing hydroxyapatite crystals, active collagenase, ad neutral protease with rotator cuff defects. III. Morphologic and biochemical studies of an excised synovium showing chondromatosis. Arthritis Rheum. 1981;24(3):484–91.
11. Halverson PB, Cheung HS, McCarty DJ, Garancis J, Mandel N. "Milwaukee shoulder"—association of microspheroids containing hydroxyapatite crystals, active collagenase,

and neutral protease with rotator cuff defects. II. Synovial fluid studies. Arthritis Rheum. 1981;24(3):474–83.
12. Canton E. Surgical and pathological observations. London: Samuel Highley; 1855.
13. Brorson S. Cuff tear arthropathy in the nineteenth century: 'chronic rheumatic arthritis' with 'partial luxation upwards' of the humeral head. Int Orthop. 2019;43(10):2415–23.
14. McCarthy GM, Cheung HS, Abel SM, Ryan LM. Basic calcium phosphate crystal-induced collagenase production: role of intracellular crystal dissolution. Osteoarthr Cartil. 1998;6(3):205–13.
15. Inman VT, Saunders JB, Abbott LC. Observations of the function of the shoulder joint. 1944. Clin Orthop Relat Res. 1996;330:3–12. doi(330):3-12
16. Rahu M, Kolts I, Poldoja E, Kask K. Rotator cuff tendon connections with the rotator cable. Knee Surg Sports Traumatol Arthrosc. 2017;25(7):2047–50.
17. Gates JJ, Gilliland J, McGarry MH, Park MC, Acevedo D, Fitzpatrick MJ, et al. Influence of distinct anatomic subregions of the supraspinatus on humeral rotation. J Orthop Res. 2010;28(1):12–7.
18. Ihashi K, Matsushita N, Yagi R, Handa Y. Rotational action of the supraspinatus muscle on the shoulder joint. J Electromyogr Kinesiol. 1998;8(5):337–46.
19. Kim HM, Dahiya N, Teefey SA, Middleton WD, Stobbs G, Steger-May K, et al. Location and initiation of degenerative rotator cuff tears: an analysis of three hundred and sixty shoulders. J Bone Joint Surg Am. 2010;92(5):1088–96.
20. Mesiha MM, Derwin KA, Sibole SC, Erdemir A, McCarron JA. The biomechanical relevance of anterior rotator cuff cable tears in a cadaveric shoulder model. J Bone Joint Surg Am. 2013;95(20):1817–24.
21. Miller RM, Thunes J, Musahl V, Maiti S, Debski RE. Effects of tear size and location on predictions of supraspinatus tear propagation. J Biomech. 2018;68:51–7.
22. Saha AK. Dynamic stability of the glenohumeral joint. Acta Orthop Scand. 1971;42(6):491–505.
23. Burkhart SS. Arthroscopic treatment of massive rotator cuff tears. Clinical results and biomechanical rationale. Clin Orthop Relat Res. 1991;267:45–56.
24. Su WR, Budoff JE, Luo ZP. Posterosuperior displacement due to rotator cuff tears. Arthroscopy. 2011;27(11):1472–7.
25. Su WR, Budoff JE, Luo ZP. The effect of anterosuperior rotator cuff tears on glenohumeral translation. Arthroscopy. 2009;25(3):282–9.
26. Collins DN, Harryman DT 2nd. Arthroplasty for arthritis and rotator cuff deficiency. Orthop Clin North Am. 1997;28(2):225–39.
27. Halverson PB, Carrera GF, McCarty DJ. Milwaukee shoulder syndrome. Fifteen additional cases and a description of contributing factors. Arch Intern Med. 1990;150(3):677–82.
28. Campion GV, McCrae F, Alwan W, Watt I, Bradfield J, Dieppe PA. Idiopathic destructive arthritis of the shoulder. Semin Arthritis Rheum. 1988;17(4):232–45.
29. Ecklund KJ, Lee TQ, Tibone J, Gupta R. Rotator cuff tear arthropathy. J Am Acad Orthop Surg. 2007;15(6):340–9.
30. Zeman CA, Arcand MA, Cantrell JS, Skedros JG, Burkhead WZ Jr. The rotator cuff-deficient arthritic shoulder: diagnosis and surgical management. J Am Acad Orthop Surg. 1998;6(6):337–48.
31. Jain NB, Yamaguchi K. The contribution of reverse shoulder arthroplasty to utilization of primary shoulder arthroplasty. J Shoulder Elb Surg. 2014;23(12):1905–12.
32. Razmjou H, Dwyer T, Holtby R. Impact of symptom bilaterality and hand dominance on patient-reported disability outcomes. SAGE Open Med. 2018;6:2050312118797566.
33. Brasseur JL, Lucidarme O, Tardieu M, Tordeur M, Montalvan B, Parier J, et al. Ultrasonographic rotator-cuff changes in veteran tennis players: the effect of hand dominance and comparison with clinical findings. Eur Radiol. 2004;14(5):857–64.
34. Misir A, Uzun E, Kizkapan TB, Ozcamdalli M, Sekban H, Guney A. Factors associated with the development of early- to mid-term cuff-tear arthropathy following arthroscopic rotator cuff repair. J Shoulder Elb Surg. 2021;30(7):1572–80.

35. Denard PJ, Koo SS, Murena L, Burkhart SS. Pseudoparalysis: the importance of rotator cable integrity. Orthopedics. 2012;35(9):e1353–7.
36. Rhee YG, Cho NS, Song JH, Park JG, Kim TY. Volumetric evaluation of the rotator cuff musculature in massive rotator cuff tears with pseudoparalysis. J Shoulder Elb Surg. 2017;26(9):1520–6.
37. Holtby R, Razmjou H. Relationship between clinical and surgical findings and reparability of large and massive rotator cuff tears: a longitudinal study. BMC Musculoskelet Disord. 2014;15:180-2474-15-180.
38. Ernstbrunner L, El Nashar R, Favre P, Bouaicha S, Wieser K, Gerber C. Chronic pseudoparalysis needs to be distinguished from pseudoparesis: a structural and biomechanical analysis. Am J Sports Med. 2021;49(2):291–7.
39. Cyriax J. Textbook of orthopaedic medicine: diagnosis of soft tissue lesions. 8th ed. London: Bailiere-Tindall; 1982.
40. 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.
41. Razmjou H, Christakis M, Dwyer T, van Osnabrugge V, Holtby R. Accuracy of infraspinatus isometric testing in predicting tear size and tendon reparability: comparison with imaging and arthroscopy. J Shoulder Elb Surg. 2017;26(8):1390–8.
42. Gordon AM, Huxley AF, Julian FJ. The variation in isometric tension with sarcomere length in vertebrate muscle fibres. J Physiol. 1966;184(1):170–92.
43. Abbott BC, Wilkie DR. The relation between velocity of shortening and the tension-length curve of skeletal muscle. J Physiol. 1953;120(1-2):214–23.
44. Desbrosses K, Babault N, Scaglioni G, Meyer JP, Pousson M. Neural activation after maximal isometric contractions at different muscle lengths. Med Sci Sports Exerc. 2006;38(5):937–44.
45. Arthuis M. Obstetrical paralysis of the brachial plexus. I. Diagnosis. Clinical study of the initial period. Rev Chir Orthop Reparatrice Appar Mot. 1972;58(Suppl 1):124–6.
46. Gerber C, Krushell RJ. Isolated rupture of the tendon of the subscapularis muscle. Clinical features in 16 cases. J Bone Joint Surg Br. 1991;73(3):389–94.
47. Razmjou H, Christakis M, Dwyer T, van Osnabrugge V, Henry P, Nam D, et al. Diagnostic accuracy of clinical tests in detecting rotator cuff pathology. Orthop Sports Med Open Access J. 2019;2(4):170–9.
48. França FO, Godinho GG, Freitas JMA, Lang AS, Ammar CD, Martinelli F. Correlation of physical examination with arthroscopic findings in the treatment of rotator cuff tear. Rev Bras Ortop (Sao Paulo). 2022;57(3):467–71.
49. Zingg PO, Jost B, Sukthankar A, Buhler M, Pfirrmann CW, Gerber C. Clinical and structural outcomes of nonoperative management of massive rotator cuff tears. J Bone Joint Surg Am. 2007;89(9):1928–34.
50. Williams GR Jr, Rockwood CA Jr. Hemiarthroplasty in rotator cuff-deficient shoulders. J Shoulder Elb Surg. 1996;5(5):362–7.
51. Werner CM, Steinmann PA, Gilbart M, Gerber C. Treatment of painful pseudoparesis due to irreparable rotator cuff dysfunction with the Delta III reverse-ball-and-socket total shoulder prosthesis. J Bone Joint Surg Am. 2005;87(7):1476–86.
52. 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(20):1886–95.
53. Razmjou H, van Osnabrugge V, Anunciacion M, Nunn A, Drosdowech D, Roszkowski A, et al. Maximizing muscle function in cuff-deficient shoulders: a rehabilitation proposal for reverse arthroplasty. J Shoulder Elbow Arthroplasty. 2021;5:1–14.
54. Alizadehkhaiyat O, Hawkes DH, Kemp GJ, Frostick SP. Electromyographic analysis of the shoulder girdle musculature during external rotation exercises. Orthop J Sports Med. 2015;3(11):2325967115613988.
55. Reed D, Cathers I, Halaki M, Ginn KA. Does load influence shoulder muscle recruitment patterns during scapular plane abduction? J Sci Med Sport. 2016 Sep;19(9):755–60.

56. Rathi S, Taylor NF, Green RA. The upper and lower segments of subscapularis muscle have different roles in glenohumeral joint functioning. J Biomech. 2017;63:92–7.
57. Wickham J, Pizzari T, Balster S, Ganderton C, Watson L. The variable roles of the upper and lower subscapularis during shoulder motion. Clin Biomech (Bristol, Avon). 2014;29(8):885–91.
58. Ackland DC, Pandy MG. Moment arms of the shoulder muscles during axial rotation. J Orthop Res. 2011;29(5):658–67.
59. Hockman DE, Lucas GL, Roth CA. Role of the coracoacromial ligament as restraint after shoulder hemiarthroplasty. Clin Orthop Relat Res. 2004;419:80–2.
60. Field LD, Dines DM, Zabinski SJ, Warren RF. Hemiarthroplasty of the shoulder for rotator cuff arthropathy. J Shoulder Elb Surg. 1997;6(1):18–23.
61. Goldberg SS, Bell JE, Kim HJ, Bak SF, Levine WN, Bigliani LU. Hemiarthroplasty for the rotator cuff-deficient shoulder. J Bone Joint Surg Am. 2008;90(3):554–9.
62. Sanchez-Sotelo J, Cofield RH, Rowland CM. Shoulder hemiarthroplasty for glenohumeral arthritis associated with severe rotator cuff deficiency. J Bone Joint Surg Am. 2001;83-A(12):1814–22.
63. Visotsky JL, Basamania C, Seebauer L, Rockwood CA, Jensen KL. Cuff tear arthropathy: pathogenesis, classification, and algorithm for treatment. J Bone Joint Surg Am. 2004;86-A(Suppl 2):35–40.
64. Zuckerman JD, Scott AJ, Gallagher MA. Hemiarthroplasty for cuff tear arthropathy. J Shoulder Elb Surg. 2000;9(3):169–72.
65. 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.
66. Grammont P, Laffay J, Deries X. Concept study and realization of a new total shoulder prosthesis [French]. Rhumatologie. 1987;39:407–18.
67. Grammont P, Bourgon J, Pelzer P. Study of a Mechanical Model for a Shoulder Total Prosthesis: Realization of a Prototype [in French]. Thèse de Sciences de l'Ingénieur. Dijon, France: Université Dijon; Lyon, France: ECAM de Lyon; 1981. 1981.
68. Ernstbrunner L, Andronic O, Grubhofer F, Camenzind RS, Wieser K, Gerber C. Long-term results of reverse total shoulder arthroplasty for rotator cuff dysfunction: a systematic review of longitudinal outcomes. J Shoulder Elb Surg. 2019;28(4):774–81.
69. Ho JC, Kane L, Stone MA, Romeo AA, Abboud JA, Namdari S. Arthroscopic debridement of irreparable rotator cuff tears: predictors of failure and success. J Shoulder Elb Surg. 2019;
70. Sevivas N, Ferreira N, Andrade R, Moreira P, Portugal R, Alves D, et al. Reverse shoulder arthroplasty for irreparable massive rotator cuff tears: a systematic review with meta-analysis and meta-regression. J Shoulder Elb Surg. 2017;26(9):e265–77.
71. 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(5):527–40.
72. Frankle MA, Teramoto A, Luo ZP, Levy JC, Pupello D. Glenoid morphology in reverse shoulder arthroplasty: classification and surgical implications. J Shoulder Elb Surg. 2009;18(6):874–85.
73. Mizuno N, Denard PJ, Raiss P, Walch G. Reverse total shoulder arthroplasty for primary glenohumeral osteoarthritis in patients with a biconcave glenoid. J Bone Joint Surg Am. 2013;95(14):1297–304.
74. Bois AJ, Knight P, Alhojailan K, Bohsali KI. Clinical outcomes and complications of reverse shoulder arthroplasty used for failed prior shoulder surgery: a systematic review and meta-analysis. JSES Int. 2020;4(1):156–68.
75. Harreld KL, Puskas BL, Frankle M. Massive rotator cuff tears without arthropathy: when to consider reverse shoulder arthroplasty. J Bone Joint Surg Am. 2011;93(10):973–84.
76. Ladermann A, Walch G, Denard PJ, Collin P, Sirveaux F, Favard L, et al. Reverse shoulder arthroplasty in patients with pre-operative impairment of the deltoid muscle. Bone Joint J. 2013;95-B(8):1106–13.

77. Sadoghi P, Leithner A, Vavken P, Holzer A, Hochreiter J, Weber G, et al. Infraglenoidal scapular notching in reverse total shoulder replacement: a prospective series of 60 cases and systematic review of the literature. BMC Musculoskelet Disord. 2011;12:101.
78. Nam D, Kepler CK, Neviaser AS, Jones KJ, Wright TM, Craig EV, et al. Reverse total shoulder arthroplasty: current concepts, results, and component wear analysis. J Bone Joint Surg Am. 2010;92(Suppl 2):23–35.
79. Favard L, Levigne C, Nerot C, Gerber C, De Wilde L, Mole D. Reverse prostheses in arthropathies with cuff tear: are survivorship and function maintained over time? Clin Orthop Relat Res. 2011;
80. Collin PG, Gain S, Nguyen Huu F, Ladermann A. Is rehabilitation effective in massive rotator cuff tears? Orthop Traumatol Surg Res. 2015;101(4 Suppl):S203–5.

Chapter 6
Frozen Shoulder

Frozen shoulder is a general term that refers to loss of active and passive range of motion. It does not always occur in the capsular pattern and is often secondary to trauma, surgery, immobility, injection, vaccination, cuff pathologies, and other reasons. Adhesive capsulitis or primary frozen shoulder is the idiopathic type of frozen shoulder, a painful inflammatory condition of the glenohumeral joint, and is characterized by restriction of active and passive range of motion in a capsular pattern. While a frozen shoulder is often a painful stiffness related to shoulder or global conditions, adhesive capsulitis presents with distinct stages or phases of recovery.

Primary frozen shoulder is a clinical diagnosis; however, subtle changes may be observed on plain radiographs, MRI, or MR arthrogram. Although the etiology is considered benign in most cases, the severity of symptoms and inability to perform the most basic functional activities make this condition quite debilitating. The natural history of adhesive capsulitis remains a controversial subject due to a wide range of predisposing factors such as endocrinological, rheumatological, and autoimmune conditions that play an important role in the duration of symptoms and extent of disability.

Historical Perspective

The entity of painful stiff shoulder was first described in the literature by a French surgeon, Simon Emmanuel Duplay in 1872 [1]. Duplay provided a comprehensive description of a condition known today as "scapulohumeral periarthritis," sometimes referred to as "Duplay's periarthritis." The term periarthritis defines a painful shoulder condition where the pathology lies in the extra-articular tissues with radiographic preservation of the joint surfaces and bony structures. Duplay believed that an inflammatory process of the subacromial bursa was the causative agent producing this syndrome [1, 2].

H. Razmjou, M. Christakis, *Clinical and Radiological Examination of the Shoulder Joint*, https://doi.org/10.1007/978-3-031-10470-1_6

The updated clinical description of the condition was provided approximately 60 years later in 1934 by a pioneer American orthopedic surgeon, Ernest Amory Codman, who used the term "frozen shoulder" [3]. Codman was not specific about the etiology of frozen shoulder or the pathological basis of the condition but felt that the commonly associated features of this condition were tendinitis and involvement of the subacromial bursa. He wrote that the condition was "difficult to define, difficult to treat, and difficult to explain...." He described the symptoms as the pain in the deltoid area, inability to sleep on the affected side, and painful and restricted elevation and external rotation with normal radiological findings.

The term "adhesive capsulitis" was later coined by Julius Neviaser, an American orthopedic surgeon in 1945 [4], describing the inflammatory nature of the condition. Neviaser described fibrosis, inflammation, and capsular contracture as the features of the idiopathic adhesive capsulitis [4]. He felt that the adherence of the axillary fold to itself and the anatomic neck of the humerus was the cause of stiffness [4]. Better understanding of adhesive capsulitis etiology through arthroscopic visualization of the shoulder joint, histological studies, and more sophisticated imaging has disputed some of Neviaser's description, particularly with respect to adhesions. While primary adhesive capsulitis has been related to abnormality of axillary pouch, synovium, anterior and inferior capsule, rotator interval, and ligaments [5–11], adhesions are not proven to be a part of this condition. Increased collagen production and cytokine concentrations, proliferation of fibroblasts, fibroplasia, and neovascularity are histological features of this condition that are interlinked with reduced joint volume, coracohumeral ligament fibrosis, and thickening and contracture of the joint capsule [12], but presence of adhesions to the humerus is not proven by the recent evidence. Therefore, many investigators have suggested that the term adhesive is confusing and should be revised [6, 13–15].

In 2011, the American Shoulder and Elbow Surgeons society defined adhesive capsulitis as a condition of uncertain etiology characterized by significant restriction of both active and passive shoulder motion that occurs in the absence of a known intrinsic shoulder disorder with unremarkable radiographs of the glenohumeral joint, except for the possible presence of osteopenia or calcific tendinitis [16]. To date and approximately 150 years from its original description and despite advances in understanding the nature of this condition, there is still significant debate and disagreement on the exact etiology, terminology, number of phases, causes, prognosis, and treatment of this condition.

Characteristics, Classification, and Causes

There are different types of frozen shoulder. The primary or idiopathic adhesive capsulitis, which most of this chapter is based on, does not appear to be caused by trauma or certain comorbidities or conditions. The secondary type of frozen shoulder is associated with a predisposing factor such as a recent surgery, immobilization, or a traumatic injury. Secondary frozen shoulder might coexist with metabolic (diabetes, hyperlipidemia, thyroid disease) [17], heart conditions (coronary artery disease) [18, 19], neurological diseases (Parkinson's disease and stroke) [20–22], and connective tissue disorders (e.g., Dupuytren's contracture) [12].

In terms of stages of the primary adhesive capsulitis, there are various alternative models referring to two-, three-, and four-phase stages, with majority of the systematic reviews perpetuating the three-phase model [14]. Of interest, there is not much agreement on the self-limiting (i.e., complete recovery regardless of type of treatment) nature of the condition either. In a systematic review of 508 citations published in 2017, the authors showed that the theory of recovery phases leading to complete resolution without treatment for frozen shoulder was unfounded, and although short-term improvement is a feature of the natural history of this condition, there is a high chance of ongoing low-level restriction and pain for a subset of patients [23]. Therefore, it might be wise to avoid referring to concrete stages of recovery or labeling this condition as self-limiting [14, 23].

Incidence and Risk Factors

In 1984, Binder et al. [24] reported an incidence of 2–5% for adhesive capsulitis, involving mostly females with an opposite shoulder involvement in about 20% to 30% of the patients. Contralateral involvement has been acknowledged by other investigators [25, 26]. More recent articles report that the nondominant side is more frequently involved in adhesive capsulitis [27, 28]. Age is variable in primary frozen shoulder, ranging from 40 to 60 years [29, 30] being rare in patients over 70 with the exception of secondary frozen shoulder due to trauma [31]. The incidence among women is reported to be 1.6- [28] to four-fold higher than in men [32].

Genetic predisposition has been suggested for primary frozen shoulder [33]. The Dupuytren's disease, a form of a fibrosing condition with a heritable component and a persistent low-grade inflammation, has been reported to occur more often in patients with adhesive capsulitis of the shoulder [12, 34]. A systematic review of the literature in 2016 indicated the presence of a genetic predisposition to development of frozen shoulder. This was proven by observing a higher incidence of frozen shoulder in the first-degree relatives, a higher heritability rate after adjusting for age in twins, a racial predilection favoring the white race, and higher leukocyte antigen (HLA)-B27 in patients with a primary frozen shoulder [35].

There is evidence that people with certain comorbidities have a higher risk of developing adhesive capsulitis. Diabetes mellitus increases the chance of adhesive capsulitis [36–41] with an incidence of 10.8% as compared to 2.3% in nondiabetics [40]. This incidence further increases to 36% in insulin-dependent diabetics. Type II diabetes, however, is reported to double the chance of adhesive capsulitis as compared with type I (22.4% vs. 10.3%, respectively) [38]. The incidence of bilateral shoulder involvement is noted to be higher in patients with parathyroid dysfunction

[42, 43]. Thyroid disease, especially hypothyroidism [44], heart conditions leading to myocardial infarction [45, 46], and Parkinson's disease [22] also increase the incidence of adhesive capsulitis.

Clinical Findings and Role of Capsular Pattern of Restriction

Primary or idiopathic frozen shoulder or the so-called adhesive capsulitis is characterized by loss of both active and passive range of motion in a capsular pattern, affecting external rotation more than abduction, more than internal rotation. A capsular pattern is a joint-specific pattern of restriction of passive movements. It indicates the existence of a contraction of the joint capsule. The concept of selective tissue tension scheme that separates contractile tissues from capsular (inert) structures was conceptualized by James Cyriax, a British orthopedic surgeon whose description of differentiating between muscles and noncontractile tissues changed the field of orthopedic medicine and specifically clinical examination [47]. Cyriax proposed a capsular pattern of restriction for the shoulder joint based mostly on his own clinical observations. Accordingly, in capsular pattern of restriction, external rotation is the most limited movement. While there is not much concrete research on the exact ratio or validity of the capsular pattern of restriction in the shoulder joint, any condition that affects the mobility of the capsule (mostly primary frozen shoulder and to some degree osteoarthritis) leads to a specific pattern of constriction.

The very limited literature in this area indicates questionable reliability on resisted isometric testing proposed by Cyriax by some investigators [48]. Others report high inter-tester reliability of the overall Cyriax model or validity of the isometric testing in neutral position [49–51]. The only study that has examined the capsular pattern of restriction is by Rundquist and colleagues [52]. The investigators used a small sample of 25 patients with stiffness and found the pattern of restriction proposed by Cyriax in 72% of the symptomatic subjects (18 of 25). However, the IR < ABD < ER pattern was also the most frequent pattern demonstrated in the noninvolved shoulders, and the investigators suggested that the capsular pattern of restriction as proposed by Cyriax was not valid [52]. In this study, the sample included patients with active and passive motion loss of at least 25% in at least two of shoulder directions. Clinically, 25% loss is not considered a frozen shoulder, and including patients with such mild restrictions is expected to have affected the study results. In light of lack of concrete normative data on shoulder movements in healthy nonathletes, a small sample size, and choosing a random 25% loss as the definition of frozen shoulder or the random 5% as the difference between affected and nonaffected sides, their conclusion should be viewed with caution.

In 1982, Cyriax [47] proposed a useful system that could differentiate between contractile and inert structures, and it would take properly conducted studies to declare that it is an invalid system. Validity studies are complicated and should use trained and skilled clinicians, patients whose presentation is a true reflection of the disease, properly justified sample sizes, and standardized maneuvers as described by the original authors. While the exact capsular pattern of the glenohumeral joint is not well described and the role of ligamentous length, glenohumeral joint conformity, and muscles should all be considered as potential constraints of the passive motions, clinically, patients with established moderate to severe primary frozen shoulder have minimal or no external rotation in neutral position. Abduction is often the second limited movement which correlates closely with scapular plane elevation.

In non-capsular pattern of restriction, external rotation is often well-preserved or minimally restricted. Clinically, patients with traumatic secondary frozen shoulder or rotator cuff disease show more limitation in elevation in scapular plane and abduction than external rotation.

In summary, diagnosis of primary adhesive capsulitis is based on clinical examination. The clinical finding depends on the stage of the condition [53, 54]. Natural history of adhesive capsulitis indicates a few stages [25, 55] that is not always agreed upon. Julius Neviaser described four phases with variable timeframes [4]. The initial phase is the painful phase where patients have a preserved range of motion but suffer from diffuse, severe, and debilitating shoulder pain with a typical night discomfort. The next phase of the adhesive capsulitis or the "freezing phase" is characterized by increasing stiffness. In the third or the "frozen" phase, a global and progressive loss of motion sets in while pain gradually gets less intense. The fourth or the "thawing" phase is described as a recovery phase with a gradual return of range of motion to normal. A full recovery may take anywhere from 1 to 3.5 years [25] and up to 7 years [26]. The contralateral shoulder might become affected between 6 months to 7 years after the initial onset of symptoms of the first shoulder [53].

The imaging findings of primary frozen shoulder may be osteoporosis and occasional calcification foci on plain radiographs [16] and reduction of glenohumeral joint volume, thickening of the joint capsule and ligaments, and synovitis that are recognized on MRI, more specifically, MR arthrogram [11, 12].

Differential Diagnosis for Frozen Shoulder

Painful shoulder conditions that have similar presentation as a frozen shoulder include glenohumeral osteoarthritis, neoplasm, septic arthritis, severe cervical disc protrusion/herniation, autoimmune diseases (e.g., rheumatoid arthritis), and locked posterior dislocation. Malignancy in the shoulder girdle is not common but often present with symptoms similar to that of a frozen shoulder. Luckily, clinical examination of patients with more serious conditions is usually more alarming, and patients with a serious or masquerading pathology often have a relevant clinical history suggestive of a need for plain radiographs, and not everyone needs to undergo routine imaging [56]. Clinically, presence of significant muscle wasting in the absence of trauma is a potential sign of a serious condition.

Considering the consequences of missing malignant tumors and strict contraindication to corticosteroid injection or arthrographic distension of the shoulder, plain radiographs should be performed to rule out serious pathologies in anyone with an alarming clinical history or clinical findings. In cases where plain radiographs are equivocal for malignancy, a CT scan investigation should be ordered. The immunocompromised patients with a history of insidious onset of moderate to severe pain who have failed conservative treatment should also be subjected to either ultrasound or MRI to avoid a delay in diagnosis of septic arthritis [57].

Conservative and Surgical Management

Management of adhesive capsulitis remains controversial, and due to the small study samples, lack of placebo-controlled trials, and suboptimal methodology used in most studies, the evidence to guide treatment is inconclusive.

Codman [3] noted that most frozen shoulders would have a complete recovery within a maximum of 2 years from the onset of symptoms. However, longer-term follow-ups have shown that 50% of patients continue to have pain or stiffness at a mean of 7 years from the onset of the condition [26]. A systematic review published in 2017 indicates that some patients will suffer from a low-level restriction and pain [23], and since early improvement is a key to resolution of the symptoms, exploring different modes of treatment should be encouraged. At present, conservative treatment includes nonsteroidal anti-inflammatory drugs, physical therapy, and corticosteroids injections. Surgical options include release of adhesions and capsule with or without manipulation under anesthesia and are intended when all conservative treatments have failed.

Nonsteroidal Anti-Inflammatory Drugs (NSAIDS) and Oral Corticosteroid

The NSAIDs may help with temporary relief of pain with insignificant improvement of function and range of motion [58]. Oral corticosteroids have been shown to improve pain, especially night pain, and range of motion in the short term [59]. A Cochrane review that examined three small trials (two placebo-controlled trials and one no-treatment controlled trial) found a "silver"-level evidence that oral steroids provide significant short-term benefits in pain, range of movement of the shoulder, and function in adhesive capsulitis, but the effect did not maintain beyond 6 weeks [60]. This small short-term benefit may not justify the use of oral steroid therapy considering the significant side effects and risks of this type of medication on other systems.

Rehabilitation and Manual Therapy

Physical therapy is probably the most commonly prescribed intervention used to improve motion and pain in different types of frozen shoulder. Active exercises and passive mobilization such as capsular stretching are expected to avoid further stiffness and improve range of motion. However, evidence that physiotherapy alone would improve frozen shoulder has not been found [61, 62]. The limited literature on physical therapy appears to support exercising within pain-free range of motion. Griggs et al. [63] who included patients with phase 2 idiopathic adhesive capsulitis treated with a specific pain-free stretching program found a satisfactory outcome in 90% of the patients. Patients with more severe pain and functional limitations before treatment had relatively worse outcomes. Diercks et al. [64] compared pendulum exercises and active exercises within a pain threshold to an intensive physical therapy composed of strenuous active and passive exercise and stretching beyond pain threshold. The investigators found that the majority of those in the less intense therapy group (90%) had near normal shoulder function at 2 years as compared with 63% in the more intense therapy group.

Favejee et al. [65] found moderate evidence in favor of mobilization techniques in the short and long term as an addition to active physiotherapy in the short term. In a randomized controlled study that compared a low-grade exercise program within a pain-free range to a high-grade mobilization, statistically significant greater change scores were found in the high-intensity mobilization group for abduction and external rotation. However, the overall differences between the two interventions were considered small [66]. The proprioceptive neuromuscular facilitation and mirror therapy are more recent techniques for rehabilitation. Interventions such as pain neuroscience education, high-intensity interval training, and lifestyle changes have been suggested without much research to examine their effectiveness [67].

Corticosteroid Injections

Corticosteroid injection into the glenohumeral or subacromial space appears to be the most favorable treatment option [68–71]. In a systematic review by Blanchard et al. [72], the effectiveness of corticosteroid injections was compared with physiotherapeutic interventions for the treatment of adhesive capsulitis/frozen shoulder. A medium effect for corticosteroid injections was reported at 6 weeks compared with physiotherapeutic interventions for the outcomes of pain, passive external rotation, and shoulder disability. A systematic review published in 2016 revealed that corticosteroid injection was superior to placebo and physiotherapy in the short term (up to 12 weeks). The authors suggested that corticosteroid injection to treat adhesive capsulitis, especially in the early painful stage, is a safe and effective treatment [73].

Similarly, a systematic review published in 2020 [74] suggested that the early use of intra-articular corticosteroid in patients with frozen shoulder of less than 1-year duration was associated with better outcomes. The authors encouraged the use of an adjunct home exercise program to maximize the chance of recovery. The use of ultrasound or fluoroscopy to guide injections has been proposed to improve the accuracy of the corticosteroid injections [68, 70, 75, 76].

Distension Arthrography

Distension arthrography first described in 1965 consists of injection of a solution (saline solution alone or combined with corticosteroids) with the aim of breaking apart the fibrotic tissue by hydrostatic pressure [77]. In a couple of randomized placebo-controlled trials, Buchbinder and colleagues reported significant short-term benefit from an intra-articular corticosteroid injection combined with arthrographic distension of the glenohumeral joint with saline solution [78, 79]. Role of physiotherapy following this type of injection appears to be somewhat beneficial. Good results have been reported with two repeated arthrographic distensions with steroid injection and an intensive rehabilitation [80]. Buchbinder et al. reported that physiotherapy following joint distension provided no additional benefits in terms of pain, function, or quality of life but resulted in sustained greater active range of shoulder movement improvement up to 6 months [81]. Elleuch et al. reported that capsular distension and subsequent intensive rehabilitation had long-term results for up to 12 months [82].

In a recent systematic review of 92 trials and 5946 patients published in 2021 [83], capsular distension was a highly recommended choice for treatment of frozen shoulder; steroid injection was also an effective intervention. The authors acknowledged the importance of individualized interventions, given that treatment effect is moderated by the disease stage, time of assessment, adjunctive therapies, female sex, and diabetes.

Closed Manipulation under Anesthesia

More invasive procedures such as manipulation under anesthesia (MUA) have shown successful results in the refractory cases. Closed manipulation under anesthesia may be considered in patients with unsuccessful response to conventional treatment. In this intervention, the shoulder is passively stretched in forward flexion, abduction, and adduction, while the scapula is being stabilized. With the elbow at a right angle, the upper arm is gently rotated through extremes of internal and external rotation by use of a short lever arm. Tearing of the contracted capsule may be palpated and even audibly confirmed by the surgeon [53].

Like other treatments suggested for adhesive capsulitis, there is controversial literature on utility of manipulation under anesthesia. While some authors [84–86] report significant increase in global range of motions of the shoulder joint, others [87, 88] do not report any superior results with this intervention. In a blinded, randomized, controlled trial, Kivimaki et al. [87] compared patients who underwent manipulation under anesthesia with a control group at baseline and at 6 weeks and 3, 6, and 12 months after randomization. Both groups were instructed on specific therapeutic exercises by physiotherapists. The authors reported that manipulation under anesthesia did not have superior results as compared with an exercise program.

It is notable that excessive forceful manipulation can cause humeral fractures, glenohumeral dislocation, rotator cuff or labral tears, brachial plexus injuries, tears of the middle glenohumeral ligament, hemorrhagic effusions, and hematomas, which can be detrimental to articular cartilage [53, 89]. Use of closed manipulation in post-traumatic or postsurgical frozen shoulder should be limited because of increased risk of fracture [53]. A recent systematic review of studies that examined the effectiveness of three treatments, physiotherapy and steroid injection, MUA and steroid injection, and arthroscopic capsular release, found that neither treatment was superior [90]. This may indicate that more invasive procedures should be considered only for refractory cases who meet strict criteria.

Surgical Release of Capsule

Arthroscopic capsular release is the procedure that is left for refractory adhesive capsulitis. Arthroscopic capsulotomy has key advantages of confirming the diagnosis allowing for direct visualization of the tightened ligaments, thickened rotator interval, and contracted capsule. The standard arthroscopic capsulotomy is antero-inferior capsular release [91].

In a small case control study, 30 patients with a frozen shoulder who underwent MUA were compared with 30 patients who underwent a capsular release. Mean external rotation and internal rotation were significantly greater in the MUA group than in the capsular release group at 3, 6, and 12 months after procedure. Elevation and pain score and ASES score were significantly better in the MUA group than in the capsular release group at 3 months after procedure [92].

A systematic review published in 2013 included 22 studies (21 studies provided level IV evidence) including 989 patients resulting in a comparison of 9 MUA and 17 capsular release groups. The authors concluded that the quality of evidence available was low and the data demonstrated little benefit for a capsular release instead of, or in addition to, an MUA [93]. As noted, a systematic review published in 2021 has also failed to show the superiority of arthroscopic capsular release over MUA with steroid injection or physiotherapy with a steroid injection [90]. In summary, the quality of the evidence supporting capsular release procedure is low at present, and further research is required to better evaluate the clinical benefit of this surgical intervention [15, 93].

In conclusion, primary frozen shoulder is a condition diagnosed clinically and is often managed solely by the primary care physicians or extended care physiotherapists or practitioners with a very small number of patients needing surgical consultation. Imaging investigations are expected to be normal and are important to rule out other serious conditions. Osteoporosis and soft tissue calcification may be present on plain radiographs with an MRI or MR arthrogram being often unnecessary as they don't change management. A CT scan is needed if plain radiographs show any abnormality pointing to malignancy. This condition is responsive to a combination of oral NSAIDs, physiotherapy, and steroid injections, but given that treatment effect is moderated by the associated risk factors and the phase of the condition, individualized approach is encouraged.

References

1. Duplay ES. De la periarthritis scapulohumerale et des radieurs de l'epaule quien son la consequence. Arch Gen Med. 1872;20:513–42.
2. Wright V, Haq AM. Periarthritis of the shoulder. I. Aetiological considerations with particular reference to personality factors. Ann Rheum Dis. 1976;35(3):213–9.
3. Codman EA. The shoulder, rupture of the supraspinatus tendon and other lesions in or about the subacromial Bursa. Boston: Todd Thomas; 1934.
4. Neviaser JS. Adhesive capsulitis of the shoulder. J Bone Joint Surg Am. 1945;27(2):211–22.
5. Lee JC, Sykes C, Saifuddin A, Connell D. Adhesive capsulitis: sonographic changes in the rotator cuff interval with arthroscopic correlation. Skelet Radiol. 2005;34(9):522–7.
6. Wiley AM. Arthroscopic appearance of frozen shoulder. Arthroscopy. 1991;7(2):138–43.
7. Connell D, Padmanabhan R, Buchbinder R. Adhesive capsulitis: role of MR imaging in differential diagnosis. Eur Radiol. 2002;12(8):2100–6.
8. Sofka CM, Ciavarra GA, Hannafin JA, Cordasco FA, Potter HG. Magnetic resonance imaging of adhesive capsulitis: correlation with clinical staging. HSS J. 2008;4(2):164–9.
9. Neer CS 2nd, Satterlee CC, Dalsey RM, Flatow EL. The anatomy and potential effects of contracture of the coracohumeral ligament. Clin Orthop Relat Res. 1992;280:182–5.
10. Kim KC, Rhee KJ, Shin HD. Adhesive capsulitis of the shoulder: dimensions of the rotator interval measured with magnetic resonance arthrography. J Shoulder Elb Surg. 2009;18(3):437–42.
11. Ozaki J, Nakagawa Y, Sakurai G, Tamai S. Recalcitrant chronic adhesive capsulitis of the shoulder. Role of contracture of the coracohumeral ligament and rotator interval in pathogenesis and treatment. J Bone Joint Surg Am. 1989;71(10):1511–5.
12. Bunker TD, Anthony PP. The pathology of frozen shoulder. A Dupuytren-like disease. J Bone Joint Surg Br. 1995;77(5):677–83.
13. Bunker TD. Time for a new name for 'frozen shoulder'. Br Med J (Clin Res Ed). 1985;290(6477):1233–4.
14. Abrassart S, Kolo F, Piotton S, Chih-Hao Chiu J, Stirling P, Hoffmeyer P, et al. 'Frozen shoulder' is ill-defined. How can it be described better? EFORT Open Rev. 2020;5(5):273–9.
15. Lewis J. Frozen shoulder contracture syndrome—aetiology, diagnosis and management. Man Ther. 2015;20(1):2–9.
16. Zuckerman JD, Rokito A. Frozen shoulder: a consensus definition. J Shoulder Elb Surg. 2011;20(2):322–5.
17. Jump CM, Duke K, Malik RA, Charalambous CP. Frozen shoulder: a systematic review of cellular, molecular, and metabolic findings. JBJS Rev. 2021;9(1):e19.00153.
18. Matei M, Brie S, Dumitru F. Left scapulohumeral periarthritis associated with coronary disease. Viata Med Rev Inf Prof Stiint Cadrelor Medii Sanit. 1987;35(12):275–8.
19. Welfling J. Periarthritis of the shoulder and cardiovascular diseases. Coeur Med Interne. 1963;58:431–6.

20. Cucchi D, Marmotti A, De Giorgi S, Costa A, D'Apolito R, Conca M, et al. Risk factors for shoulder stiffness: current concepts. Joints. 2017;5(4):217–23.
21. Rizk TE, Christopher RP, Pinals RS, Salazar JE, Higgins C. Arthrographic studies in painful hemiplegic shoulders. Arch Phys Med Rehabil. 1984;65(5):254–6.
22. Riley D, Lang AE, Blair RD, Birnbaum A, Reid B. Frozen shoulder and other shoulder disturbances in Parkinson's disease. J Neurol Neurosurg Psychiatry. 1989;52(1):63–6.
23. Wong CK, Levine WN, Deo K, Kesting RS, Mercer EA, Schram GA, et al. Natural history of frozen shoulder: fact or fiction? A systematic review. Physiotherapy. 2017;103(1):40–7.
24. Binder AI, Bulgen DY, Hazleman BL, Roberts S. Frozen shoulder: a long-term prospective study. Ann Rheum Dis. 1984;43(3):361–4.
25. Reeves B. The natural history of the frozen shoulder syndrome. Scand J Rheumatol. 1975;4(4):193–6.
26. Shaffer B, Tibone JE, Kerlan RK. Frozen shoulder. A long-term follow-up. J Bone Joint Surg Am. 1992;74(5):738–46.
27. Levine WN, Kashyap CP, Bak SF, Ahmad CS, Blaine TA, Bigliani LU. Nonoperative management of idiopathic adhesive capsulitis. J Shoulder Elb Surg. 2007;16(5):569–73.
28. Hand C, Clipsham K, Rees JL, Carr AJ. Long-term outcome of frozen shoulder. J Shoulder Elb Surg. 2008;17(2):231–6.
29. Kingston K, Curry EJ, Galvin JW, Li X. Shoulder adhesive capsulitis: epidemiology and predictors of surgery. J Shoulder Elb Surg. 2018;27(8):1437–43.
30. Lloyd-Roberts GC, French PR. Periarthritis of the shoulder: a study of the disease and its treatment. Br Med J. 1959;1(5137):1569–71.
31. Robinson CM, Seah KT, Chee YH, Hindle P, Murray IR. Frozen shoulder. J Bone Joint Surg Br. 2012;94(1):1–9.
32. Mezian K, Coffey R, Chang KV. Frozen Shoulder. StatPearls Treasure Island (FL): StatPearls Publishing LLC; 2021.
33. Hakim AJ, Cherkas LF, Spector TD, MacGregor AJ. Genetic associations between frozen shoulder and tennis elbow: a female twin study. Rheumatology (Oxford). 2003;42(6):739–42.
34. Smith SP, Devaraj VS, Bunker TD. The association between frozen shoulder and Dupuytren's disease. J Shoulder Elb Surg. 2001;10(2):149–51.
35. Prodromidis AD, Charalambous CP. Is there a genetic predisposition to frozen shoulder?: a systematic review and meta-analysis. JBJS Rev. 2016;4:2. https://doi.org/10.2106/JBJS.RVW.O.00007.
36. Thomas SJ, McDougall C, Brown ID, Jaberoo MC, Stearns A, Ashraf R, et al. Prevalence of symptoms and signs of shoulder problems in people with diabetes mellitus. J Shoulder Elb Surg. 2007;16(6):748–51.
37. Fisher L, Kurtz A, Shipley M. Association between cheiroarthropathy and frozen shoulder in patients with insulin-dependent diabetes mellitus. Br J Rheumatol. 1986;25(2):141–6.
38. Arkkila PE, Kantola IM, Viikari JS, Ronnemaa T. Shoulder capsulitis in type I and II diabetic patients: association with diabetic complications and related diseases. Ann Rheum Dis. 1996;55(12):907–14.
39. Balci N, Balci MK, Tuzuner S. Shoulder adhesive capsulitis and shoulder range of motion in type II diabetes mellitus: association with diabetic complications. J Diabetes Complicat. 1999;13(3):135–40.
40. Bridgman JF. Periarthritis of the shoulder and diabetes mellitus. Ann Rheum Dis. 1972;31(1):69–71.
41. Garcilazo C, Cavallasca JA, Musuruana JL. Shoulder manifestations of diabetes mellitus. Curr Diabetes Rev. 2010;6(5):334–40.
42. Wohlgethan JR. Frozen shoulder in hyperthyroidism. Arthritis Rheum. 1987;30(8):936–9.
43. Bowman CA, Jeffcoate WJ, Pattrick M, Doherty M. Bilateral adhesive capsulitis, oligoarthritis and proximal myopathy as presentation of hypothyroidism. Br J Rheumatol. 1988;27(1):62–4.
44. Cakir M, Samanci N, Balci N, Balci MK. Musculoskeletal manifestations in patients with thyroid disease. Clin Endocrinol. 2003;59(2):162–7.

45. Boyle-Walker KL, Gabard DL, Bietsch E, Masek-VanArsdale DM, Robinson BL. A profile of patients with adhesive capsulitis. J Hand Ther. 1997;10(3):222–8.
46. Lo SF, Chen SY, Lin HC, Jim YF, Meng NH, Kao MJ. Arthrographic and clinical findings in patients with hemiplegic shoulder pain. Arch Phys Med Rehabil. 2003;84(12):1786–91.
47. Cyriax J. Textbook of orthopaedic medicine: diagnosis of soft tissue lesions. 8th ed. London, England: Bailiere-Tindall; 1982.
48. Hayes KW, Petersen CM. Reliability of classifications derived from Cyriax's resisted testing in subjects with painful shoulders and knees. J Orthop Sports Phys Ther. 2003;33(5):235–46.
49. Razmjou H, Christakis M, Dwyer T, van Osnabrugge V, Holtby R. Accuracy of infraspinatus isometric testing in predicting tear size and tendon reparability: comparison with imaging and arthroscopy. J Shoulder Elb Surg. 2017;26(8):1390–8.
50. Pellecchia GL, Paolino J, Connell J. Intertester reliability of the cyriax evaluation in assessing patients with shoulder pain. J Orthop Sports Phys Ther. 1996;23(1):34–8.
51. Schmidt M, Enger M, Pripp AH, Nordsletten L, Moosmayer S, Melhuus K, et al. Interrater reliability of physical examination tests in the acute phase of shoulder injuries. BMC Musculoskelet Disord. 2021;22:1. 770-021-04659-x
52. Rundquist PJ, Ludewig PM. Patterns of motion loss in subjects with idiopathic loss of shoulder range of motion. Clin Biomech (Bristol, Avon). 2004;19(8):810–8.
53. Hsu JE, Anakwenze OA, Warrender WJ, Abboud JA. Current review of adhesive capsulitis. J Shoulder Elb Surg. 2011;20(3):502–14.
54. Neviaser AS, Hannafin JA. Adhesive capsulitis: a review of current treatment. Am J Sports Med. 2010;38(11):2346–56.
55. Neviaser RJ, Neviaser TJ. The frozen shoulder. Diagnosis and management. Clin Orthop Relat Res. 1987;223:59–64.
56. Roberts S, Dearne R, Keen S, Littlewood C, Taylor S, Deacon P. Routine X-rays for suspected frozen shoulder offer little over diagnosis based on history and clinical examination alone. Musculoskeletal Care. 2019;17(2):288–92.
57. Sambandam SN, Atturu M. A case of septic arthritis of shoulder presenting as stiffness of the shoulder. J Orthop Case Rep. 2016;6(2):31–3.
58. Rhind V, Downie WW, Bird HA, Wright V, Engler C. Naproxen and indomethacin in periarthritis of the shoulder. Rheumatol Rehabil. 1982;21(1):51–3.
59. Lorbach O, Anagnostakos K, Scherf C, Seil R, Kohn D, Pape D. Nonoperative management of adhesive capsulitis of the shoulder: oral cortisone application versus intra-articular cortisone injections. J Shoulder Elb Surg. 2010;19(2):172–9.
60. Buchbinder R, Green S, Youd JM, Johnston RV. Oral steroids for adhesive capsulitis. Cochrane Database Syst Rev. 2006;4:CD006189.
61. Green S, Buchbinder R, Hetrick S. Physiotherapy interventions for shoulder pain. Cochrane Database Syst Rev. 2003;2(2):CD004258.
62. Cleland JA, Durall CJ. Physical therapy for adhesive capsulitis: systematic review. Physiotherapy. 2002;88(8):450–7.
63. Griggs SM, Ahn A, Green A. Idiopathic adhesive capsulitis. A prospective functional outcome study of nonoperative treatment. J Bone Joint Surg Am. 2000;82-A(10):1398–407.
64. Diercks RL, Stevens M. Gentle thawing of the frozen shoulder: a prospective study of supervised neglect versus intensive physical therapy in seventy-seven patients with frozen shoulder syndrome followed up for two years. J Shoulder Elb Surg. 2004;13(5):499–502.
65. Favejee MM, Huisstede BM, Koes BW. Frozen shoulder: the effectiveness of conservative and surgical interventions—systematic review. Br J Sports Med. 2011;45(1):49–56.
66. Vermeulen HM, Rozing PM, Obermann WR, le Cessie S, Vliet Vlieland TP. Comparison of high-grade and low-grade mobilization techniques in the management of adhesive capsulitis of the shoulder: randomized controlled trial. Phys Ther. 2006;86(3):355–68.
67. Mertens MG, Meeus M, Verborgt O, Vermeulen EHM, Schuitemaker R, Hekman KMC, et al. An overview of effective and potential new conservative interventions in patients with frozen shoulder. Rheumatol Int. 2021;

68. Naredo E, Cabero F, Beneyto P, Cruz A, Mondejar B, Uson J, et al. A randomized comparative study of short term response to blind injection versus sonographic-guided injection of local corticosteroids in patients with painful shoulder. J Rheumatol. 2004;31(2):308–14.
69. Carette S, Moffet H, Tardif J, Bessette L, Morin F, Fremont P, et al. Intraarticular corticosteroids, supervised physiotherapy, or a combination of the two in the treatment of adhesive capsulitis of the shoulder: a placebo-controlled trial. Arthritis Rheum. 2003;48(3):829–38.
70. Lee HJ, Lim KB, Kim DY, Lee KT. Randomized controlled trial for efficacy of intra-articular injection for adhesive capsulitis: ultrasonography-guided versus blind technique. Arch Phys Med Rehabil. 2009;90(12):1997–2002.
71. Shah N, Lewis M. Shoulder adhesive capsulitis: systematic review of randomised trials using multiple corticosteroid injections. Br J Gen Pract. 2007;57(541):662–7.
72. Blanchard V, Barr S, Cerisola FL. The effectiveness of corticosteroid injections compared with physiotherapeutic interventions for adhesive capsulitis: a systematic review. Physiotherapy. 2010;96(2):95–107.
73. Koh KH. Corticosteroid injection for adhesive capsulitis in primary care: a systematic review of randomised clinical trials. Singap Med J. 2016;57(12):646–57.
74. Challoumas D, Biddle M, McLean M, Millar NL. Comparison of treatments for frozen shoulder: a systematic review and meta-analysis. JAMA Netw Open. 2020;3(12):e2029581.
75. Yamakado K. The targeting accuracy of subacromial injection to the shoulder: an arthrographic evaluation. Arthroscopy. 2002;18(8):887–91.
76. Lorbach O, Kieb M, Scherf C, Seil R, Kohn D, Pape D. Good results after fluoroscopic-guided intra-articular injections in the treatment of adhesive capsulitis of the shoulder. Knee Surg Sports Traumatol Arthrosc. 2010;18(10):1435–41.
77. Andren L, Lundberg BJ. Treatment of rigid shoulders by joint distension during arthrography. Acta Orthop Scand. 1965;36:45–53.
78. Buchbinder R, Green S, Youd JM, Johnston RV, Cumpston M. Arthrographic distension for adhesive capsulitis (frozen shoulder). Cochrane Database Syst Rev. 2008;1(1):CD007005.
79. Buchbinder R, Green S, Forbes A, Hall S, Lawler G. Arthrographic joint distension with saline and steroid improves function and reduces pain in patients with painful stiff shoulder: results of a randomised, double blind, placebo controlled trial. Ann Rheum Dis. 2004;63(3):302–9.
80. Fouquet B, Griffoul I, Borie MJ, Roger R, Bonnin B, Metivier JC, et al. Adhesive capsulitis: evaluation of a treatment coupling capsular distension and intensive rehabilitation. Ann Readapt Med Phys. 2006;49(2):68–74.
81. Buchbinder R, Youd JM, Green S, Stein A, Forbes A, Harris A, et al. Efficacy and cost-effectiveness of physiotherapy following glenohumeral joint distension for adhesive capsulitis: a randomized trial. Arthritis Rheum. 2007;57(6):1027–37.
82. Elleuch MH, Yahia A, Ghroubi S, Kharrat O, Mezghanni M, Ayedi K. The contribution of capsular distension to the treatment of primary adhesive capsulitis of the shoulder: a comparative study versus rehabilitation. Ann Readapt Med Phys. 2008;51(9):722–8.
83. Zhang J, Zhong S, Tan T, Li J, Liu S, Cheng R, et al. Comparative efficacy and patient-specific moderating factors of nonsurgical treatment strategies for frozen shoulder: an updated systematic review and network meta-analysis. Am J Sports Med. 2021;49(6):1669–79.
84. Farrell CM, Sperling JW, Cofield RH. Manipulation for frozen shoulder: long-term results. J Shoulder Elb Surg. 2005;14(5):480–4.
85. Dodenhoff RM, Levy O, Wilson A, Copeland SA. Manipulation under anesthesia for primary frozen shoulder: effect on early recovery and return to activity. J Shoulder Elb Surg. 2000;9(1):23–6.
86. Amir-Us-Saqlain H, Zubairi A, Taufiq I. Functional outcome of frozen shoulder after manipulation under anaesthesia. J Pak Med Assoc. 2007;57(4):181–5.
87. Kivimaki J, Pohjolainen T, Malmivaara A, Kannisto M, Guillaume J, Seitsalo S, et al. Manipulation under anesthesia with home exercises versus home exercises alone in the treatment of frozen shoulder: a randomized, controlled trial with 125 patients. J Shoulder Elb Surg. 2007;16(6):722–6.

88. Quraishi NA, Johnston P, Bayer J, Crowe M, Chakrabarti AJ. Thawing the frozen shoulder. A randomised trial comparing manipulation under anaesthesia with hydrodilatation. J Bone Joint Surg Br. 2007;89(9):1197–200.
89. Loew M, Heichel TO, Lehner B. Intraarticular lesions in primary frozen shoulder after manipulation under general anesthesia. J Shoulder Elb Surg. 2005;14(1):16–21.
90. Rex SS, Kottam L, McDaid C, Brealey S, Dias J, Hewitt CE, et al. Effectiveness of interventions for the management of primary frozen shoulder : a systematic review of randomized trials. Bone Jt Open. 2021;2(9):773–84.
91. Le HV, Lee SJ, Nazarian A, Rodriguez EK. Adhesive capsulitis of the shoulder: review of pathophysiology and current clinical treatments. Shoulder Elbow. 2017;9(2):75–84.
92. Kim DH, Song KS, Min BW, Bae KC, Lim YJ, Cho CH. Early clinical outcomes of manipulation under anesthesia for refractory adhesive capsulitis: comparison with arthroscopic capsular release. Clin Orthop Surg. 2020;12(2):217–23.
93. Grant JA, Schroeder N, Miller BS, Carpenter JE. Comparison of manipulation and arthroscopic capsular release for adhesive capsulitis: a systematic review. J Shoulder Elb Surg. 2013;22(8):1135–45.

Chapter 7
Arthritis of the Glenohumeral Joint

There are two major types of arthritis of the glenohumeral joint. The primary osteoarthritis refers to degeneration of the glenohumeral joint without any identifiable causes, such as systemic diseases or traumatic injuries (idiopathic etiology).

One important radiological feature of *primary glenohumeral osteoarthritis* is the posterior bone erosion of the glenoid. This very unique pathology is known to be related to increased glenoid retroversion which appears to be affected by independent and complex genetic and biomechanical factors [1]. This may explain why some people are more prone to primary osteoarthritis of the glenohumeral joint. The glenoid version is defined as an angle between the glenoid face and the scapular body. A normal glenoid version is estimated as a mean of 2 ± 5 degrees of glenoid anteversion. This version is significantly different in an arthritic joint, with the glenoid face being tilted backward at about11 ± 8 degrees of retroversion [2]. Association between posterior glenoid wear and posterior humeral head subluxation has been a subject of discussions for a few decades. In 1990, Neer felt that glenoid erosion preceded subluxation, and Walch et al. were able to show that subluxation (dynamic and statistic) proceeded erosion. Walch et al. were able to exclude all potential causes of the subluxation except one thing, and that was increased retroversion of the glenoid [3].

While over the last 20 years our knowledge of glenohumeral arthritis has improved significantly, the exact contribution of osseous morphology vs. contractile and noncontractile soft tissue balance to the development of posterior glenoid subluxation remains unclear. Although the direction of cause and effect is still not clear, the association between the glenoid inclination, posterior humeral head subluxation, and muscle imbalance in the transverse force couple (infraspinatus and teres minor vs. subscapularis) has been reported in multiple independent studies. Aleem et al. [4] reported that the asymmetric posterior glenoid wear and posterior humeral head subluxation in osteoarthritis were associated with asymmetric atrophy within the rotator cuff transverse plane. Hartwell et al. [5] found that the amount of fatty infiltration in the infraspinatus muscle was strongly correlated with the glenoid version.

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Similarly, Mitterer et al. suggested that increased glenoid retroversion and chronic muscle volume imbalance could increase posterior force during contraction pushing the humeral head posteriorly and contribute to the permanent posterior displacement of the humeral head [6]. To date, however, the exact causative or associative nature of this relationship is not proven. Further discussion of imaging findings of the arthritic shoulder joint has been provided in Chap. 12.

Secondary glenohumeral arthritis is a term used to reflect all other types of arthritis with known predisposing or risk factors, impacting the normal biomechanics, joint structure integrity, joint blood supply, or nutrition. Trauma (e.g., injury related, chronic anterior dislocation, unrecognized posterior dislocation, malunion of proximal humerus fracture, etc.) is a common cause of biomechanical alteration of the glenohumeral joint surfaces. Postsurgical arthropathy (e.g., capsulorrhaphy arthropathy) may cause secondary arthritis by altering the biomechanics of the joint as well. Cuff tear arthropathy as discussed in Chap. 5 may be related to massive tears or more commonly chronic inflammation. This condition initiates with gradual superior migration of the humeral head position in relation to the glenoid due to cuff deficiency and altered joint forces, leading to degenerative changes at multiple sites of the glenohumeral joint.

Other forms of secondary arthritis are related to blood supply disorders (e.g., avascular necrosis), metabolic conditions (e.g., abnormally elevated levels of uric acid seen in gout, erosive arthropathy seen in the chronic renal disease), infection (e.g., septic arthritis), and neuropathic arthritis (e.g., syringomyelia). Systemic autoimmune disorders and inflammatory conditions (e.g., rheumatoid arthritis, lupus, Sjogren syndrome) are linked to malfunctioning autoimmune system and often involve multiple joints and organs.

Congenital, hereditary, and genetic causes of secondary arthritis are less common. One of the rare causes of secondary arthritis is the congenital anomaly of the shoulder girdle and muscles (e.g., Sprengel deformity characterized as malposition and dysplasia of the scapula) [7, 8]. The inherited genetic disorders could also cause arthropathy (e.g., Gaucher disease, a rare condition caused by a lipid storage disorder and genetic enzyme deficiency, associated with enlargement of the viscera and progressive deformity of the skeleton and subsequent osteonecrosis) [9]. Among important blood-related genetic disorders, sickle cell disease can be named. The sickle cell disease is an autosomal recessive genetic disease that is associated with abnormal hemoglobin levels which lead to chronic hemolysis and consequent anemia and vaso-occlusive events that overtime cause osteonecrosis and secondary osteoarthritis in the glenohumeral joint [10].

Historical Perspective

It is not clear when shoulder joint arthritis was first described in the medical literature. Gout appears to have been studied and understood more than other forms of secondary joint disease throughout centuries despite lack of laboratory and other diagnostic tests at the time. There is some documentation about gout as an inflammatory form of arthritis, dating back to 2600 BC by the Egyptians. In the ninth century, Abu Bakr Muhammad ibn Zakariyya al-Razi known by the Latin name Rhazes, a Persian physician, chemist, and philosopher (865 – 925 AD), provided details about the nature, prevalence, causes, and treatment of the gout. He highlighted that the disease involved swelling, edema, and pain, often commenced from the big toe with a higher prevalence in men, appeared in women at a later age, and was more seen in people who belonged to higher socioeconomic classes, evidently due to higher consumption of purine-rich food such as fish, alcoholic beverages, organs such as liver, and some meats [11–13]. In the nineteenth-century literature, there seems to have been confusion between acute attacks of gout and other inflammatory conditions. In 1848, Alfred Baring Garrod, an English physician, made a significant contribution to our knowledge of the causation of gout [14]. He reported increased uric acid in the blood of patients with gout. In 1859, he demonstrated smaller quantities of uric acid in the normal serum and deposits of urate in the articular cartilage of gout [14].

The details on symptoms of systemic inflammatory type of arthritis that was associated with severe deformity of the joints are reported about a thousand years ago by an advisor of Constantine IX, the Byzantine emperor who suffered from a chronic inflammatory illness in the eleventh century. While signs and symptoms were described in detail, the cause of this type of arthritis was not known at the time. The credit for early medical description of the inflammatory arthritis is given to Landre-Beauvais, a French medical student over 700 years after the death of Constantine IX [15]. In 1809, Augustin-Jacob Landre-Beauvais presented the symptoms of a series of diseases he named “Goutte Asthénique Primitive” as a dissertation. Based on his meticulous observations, the condition was associated with capsular swelling, stiffness, and bony ankylosis of multiple joints and affected mostly the poor and the women. In a review by Tsoucalas and Sgantzos [16], while the classification of rheumatoid arthritis by Landre-Beauvais as a relative of gout was inaccurate, his dissertation encouraged other researchers in the field of bone and joint disorders to further study this disease.

The credit of using the term “rheumatoid arthritis” that has remained in the medical literature to the present time is given to Alfred Baring Garrod in 1876 [17], half a century after Landre-Beauvais’ description. Garrod differentiated between gout, caused by an increase in uric acid in the blood, and the acute rheumatism, as an inflammatory form of arthritis. It was not until 1970, that the role of human leukocyte (HLA) was first implicated as a pathogenesis of rheumatoid arthritis [18]. Rheumatoid arthritis remains one of the most challenging and debilitating diseases with an impact on human history. Pierre-Auguste Renoir, a renowned impressionist painter who lived from 1841 to 1919, suffered from rheumatoid arthritis with severe multiple joint deformity. He is quoted as saying “The pain passes, but the beauty remains” [19, 20], reminding us that living with chronic destructive arthritis should not take away our perception of the beauty and happiness around us.

Characteristics, Classification, and Causes

Primary osteoarthritis is an age-related progressive degenerative disease of the glenohumeral without any known or identifiable causes, predisposing factors, or systemic illnesses. Considering our knowledge of risk factors for development of arthritis such as genetics, heredity, harmful enzymes, detrimental environmental factors, poor diet, and nutritional factors is improving with time, the category of primary osteoarthritis has been getting smaller over time as we are identifying more risk factors to explain the presence of joint damage.

Most traditional text books have described primary osteoarthritis as a condition caused by simple wear and tear. It may be argued that heavy manual work and strenuous overhead sports activities may contribute or accelerate wear and tear in the glenohumeral joint, but many patients with advanced primary arthritis of the glenohumeral joint cannot recall any significant injury. In addition, an appropriate amount of loading can maintain or even improve the biomechanical properties of healthy hyaline cartilage and subchondral bone [21].

As the population ages, the number of individuals with arthritis is projected to increase significantly. There are no accurate figures on incidence or prevalence of primary arthritis of the glenohumeral joint in different regions in the world. Most often, the information on primary and secondary arthritis has been merged together. In a study of cadaveric specimens and radiographs [22], alterations in the articular cartilage and rotator cuff were associated with characteristic osseous changes in the glenohumeral joint. In the review of 50 symptomatic patients, idiopathic glenohumeral osteoarthritis was discovered in 10 (20%) of 50 symptomatic patients age 60 years and over. In agreement with cadaver studies, the prevalence rates in the middle-aged and elderly Korean and Japanese populations have been estimated as 17%–19% [23–25]. In 2011, the prevalence of arthritis in Canada was approximately 18%, being significantly higher in women (25 vs 15%) [26]. The number of people living with arthritis in Canada is expected to increase by about three million to a total of nine million people by the year 2040 (20% of the Canadian population), highlighting the importance and impact of arthritis on the healthcare system [27].

Symptomatology is variable in mild and moderate arthritis. Individuals with mild to moderate glenohumeral joint osteoarthritis present with episodic mild symptoms for a number of years. Often, the relatively short symptomatic timeframe does not match the length of time it takes for severe radiological changes to occur. This may indicate that most patients with mild and even moderate osteoarthritis have minimal episodic symptoms that are often manageable by rest and occasional painkillers. As the arthritis progresses, disability increases, and the advanced radiological changes take place. The typical symptom of severe arthritis is a deep ache at rest, aggravated with activity and inability to sleep on the affected side in more advanced cases. Mechanism of injury is usually insidious in primary arthritis, but patients may recall a traumatic event concurring at the same time of the initial symptoms.

Typically, a patient with advanced primary osteoarthritis of the glenohumeral joint is over 60 years of age with limited movement without significant weakness in rotator cuff muscles in neutral position. In patients with severe osteoarthritis, range of motion is quite restricted in all directions, mostly in external and internal rotation which affects performing simple activities of daily living. Marked crepitus, associated with grinding and occasionally clunking, is common. Weakness in rotator cuff muscles is usually not an issue, unless other risk factors are involved.

The final diagnosis of glenohumeral osteoarthritis is based on simple plain radiographs. Typically, the imaging of primary osteoarthritis shows increased glenoid retroversion, posterior humeral head subluxation, and posterior glenoid wear. The posterior glenoid wear is best viewed on axial plain radiographs or axial computed tomography (CT) scan images. Inflammatory arthritis is often associated with concentric and central glenoid erosion. The arthritis caused by cuff deficiency is associated with superior subluxation of the humeral head and superior glenoid wear (see Chap. 11 for details). More costly imaging such as CT scans is not indicated for diagnosis or treatment of glenohumeral osteoarthritis in primary care but is used for surgical planning or to identify potential causes of arthritis.

In younger individuals with similar clinical findings (e.g., restricted range of motion without crepitus), the diagnosis of adhesive capsulitis needs to be ruled out. Other differential diagnoses for primary osteoarthritis are malignant tumor, infection (septic arthritis), unreduced dislocation, or inflammatory arthritis. The laboratory findings of patients with mono joint involvement are within normal limits.

Conservative treatment such as activity modification, rest, medication, and physical therapy helps with temporary relief of symptoms in patients with mild to moderate primary osteoarthritis. Patients with advanced osteoarthritis would benefit from an intra-articular injection of corticosteroid. Due to variable accuracy of injections administered in the office and limitations for repeated corticosteroid injections, fluoroscopy or ultrasound is recommended, which adds to accuracy and effectiveness of the injections [28, 29].

Minor surgical treatments include debridement, removal of loose bodies, and capsular release. Rotator cuff decompression or distal clavicle resection can ease the symptoms in patients with associated subacromial osseous impingement and advanced associated arthritis of the acromioclavicular joint, respectively. Research shows that minor surgical procedures such as subacromial debridement are helpful in patients with clinical and radiological signs of osseous impingement [30]. More major surgeries include hemiarthroplasty and anatomical total shoulder arthroplasty. The literature suggests that total shoulder arthroplasty is more superior to hemiarthroplasty in terms of relieving symptoms, improving range of motion, and incidence of revision [31–33]. However, the indications for each surgery are different. Age of the patient, physical demands, extent of rotator cuff deficiency, glenoid bone loss, and quality of glenoid bone stock are important factors to consider when choosing between different types of arthroplasty. Young and active patients have a higher incidence of glenoid component loosening because of their physical demands and will benefit from hemiarthroplasty without glenoid prosthetic resurfacing.

Severe glenoid deficiency (abnormal glenoid version and/or posterior wear) leads to lack of bone stock which would limit ability of the surgeon to perform total shoulder arthroplasty.

As it relates to *secondary arthritis*, clinical history is often helpful in making the connection between joint damage and trauma or other medical risk factors. Patients with systemic inflammatory diseases often have multiple joint complaints. History of prior stabilization surgery and restricted motion usually indicate a potential capsulorrhaphy arthropathy. Prior history of corticosteroid use, liver disease, alcoholism, or presence of other risk factors in younger patients should prompt the clinicians on ordering imaging investigations to rule out destructive conditions such as avascular necrosis.

As noted, clinical history is often very helpful in making the diagnosis. Patients with systemic inflammatory diseases are often under care of a rheumatologist for multiple joint complaints. Traumatic remote dislocation and surgery in middle aged patients is a warning sign of altered glenohumeral biomechanics. Previous treatments with corticosteroid medications and other chronic conditions that affect kidney, liver, or the immune system should be taken into consideration as the cause of secondary arthritis. The movement restriction and radiological findings of each condition are unique to the cause of arthritis.

Rheumatoid Arthritis of Glenohumeral Joint

Rheumatoid arthritis is a systemic autoimmune disease that involves multiple joints and is characterized by inflammation of peri- and intra-articular soft tissue structures and organs. The incidence of rheumatoid arthritis varies by age and population. Rheumatoid arthritis has been reported worldwide with a three- to fivefold higher prevalence in females than males.

The symptoms of rheumatoid arthritis are often of insidious onset with progressive and debilitating progression over time. It has been shown that the inflammatory process takes place in the synovium and synovial fluid [34]. Overtime, persistent inflammation leads to cartilage degradation and bone erosion. The exact pathogenesis of rheumatoid arthritis is still unknown. However, the disease is multifactorial and is caused by genetic and environmental exposure to chemicals, air pollution [35–37], climatic changes, infectious diseases, and food [38, 39]. Poor socioeconomic conditions and living in rural areas have been reported to be linked to increased risk of developing rheumatoid arthritis [40].

Clinical examination depends on severity of the disease. Weakness can be observed in those with associated rotator cuff pathology. Progressive cuff pathology such as large or massive full-thickness cuff tears leads to cuff tear arthropathy discussed in Chap. 5. In terms of laboratory findings, presence or absence of rheumatoid factor (RF) and anticyclic citrullinated protein antibodies (ACPA) can divide rheumatoid arthritis into two types (seropositive and seronegative) [41]. While presence of ACPA suggests a genetic risk factor for rheumatoid arthritis, it's not necessary for either antibody (RF factor or ACPA) to be present in the blood for a diagnosis of seronegative rheumatoid arthritis. A higher use of healthcare services and medications (methotrexate (73.2% versus 30.3%) and biologic agents (7.9% versus 2.9%) has been reported by the patients with a seropositive as compared with the seronegative rheumatoid arthritis [42].

Imaging of the inflammatory arthritis shows glenoid erosion in a concentric and central pattern, as opposed to posterior glenoid wear commonly seen in primary osteoarthritis. The severity of the medial glenoid erosion can affect the type of surgery (anatomical shoulder replacement vs. reverse arthroplasty). Patients with rheumatoid arthritis usually present with extensive soft tissue involvement of synovium, rotator cuff, and biceps tendons [43]. Chap. 12 provides more details on the imaging differences between primary and inflammatory arthritis.

Surgical treatment of the secondary arthritis due to inflammatory conditions depends on patient's age, severity of pathology, lifestyle, and physical demands. Reverse shoulder arthroplasty is often more feasible as cuff deficiency may affect the durability and success of the prosthesis.

Total shoulder arthroplasty is not recommended in patients with rotator cuff deficiency [44]. The eccentric loading of the glenoid secondary to superior migration of the humeral head is the major cause of implant loosening in these patients. It is noteworthy that indications for reverse shoulder arthroplasty have been expanding significantly over the past decade and cuff tear arthropathy that was initially the primary indication for this type of surgery is now only one of the many indications for this surgery [45–51].

Capsulorrhaphy Arthropathy

Capsulorrhaphy arthropathy occurs after stabilization surgeries and appears to be related to intra-articular hardware, excessive tightening of the soft tissues, changes of glenoid shape, neurovascular scarring from surgery, and bone deficiencies. Factors that are associated with post-stabilization arthritis are older age at the time of initial dislocation or surgery, higher number of dislocations, deficiency in external rotation range of motion, involvement in high-energy sports, and alcohol abuse [52, 53].

Prevalence of radiological degenerative changes following dislocations and stabilization surgical procedures is reported to be as high as 56–68%, but symptoms are typically infrequent or mild [54–56]. According to Hovelius et al. who won the Neer Award in 2008, incidence of moderate/severe arthropathy depends on the recurrence, being 18% in shoulders without a recurrence, increasing to 39% in shoulders with recurrent dislocation. In their study, patients with surgical stabilization had a slightly less prevalence of moderate/severe arthritis at about 26% [56]. A positive correlation between secondary osteoarthritis and recurrent dislocation has been reported by other investigators. Marx et al. reported that glenohumeral arthritis was 20 times more likely after recurrent dislocations [53].

In capsulorrhaphy arthropathy, the plain radiographs have a more complicated presentation. The intra-articular metallic internal fixation devices or bone grafts often change the normal biomechanics of the joint. In addition, capsular tightening may affect the glenohumeral contact and change the direction of pressure and joint surface wear. Excessive tightening of the anterior capsule produces posterior translation causing posterior glenoid wear. The concomitant humeral articular cartilage wear and flattening are common in these cases.

Avascular Necrosis or Osteonecrosis

Avascular necrosis (AVN), also known as osteonecrosis, ischemic necrosis, or aseptic necrosis, is one of the causes of secondary arthritis. AVN may be traumatic, caused by disruption of the blood supply, or atraumatic caused by alcohol abuse, corticosteroids, liver disease, radiation, and cytotoxic drugs used to treat systemic conditions or cancer or other systemic disorders such as systemic lupus erythematosus [57–63]. In this condition, the normal healing process does not occur after a traumatic injury, and the bone tissues break down faster than the body can repair them.

A long-term and excessive use of alcohol produces intracellular lipid deposits resulting in the death of osteocytes, leading to development of osteonecrosis [64–66]. Alcoholic patients often are susceptible to develop diffuse intravascular coagulation and AVN in the humeral head [66]. In patients with nonalcoholic liver disease (e.g., viral hepatitis), chronic inflammation seems to be the causal effect of AVN. Prolonged use of corticosteroids creates marked alterations in lipid metabolism. Intra-arterial infusion of fat produces embolic vascular obstruction, focal marrow necrosis, and osteocytic death leading to AVN [67].

In patients with a fracture caused by osteonecrosis, the presence of liver disease, sickle cell disease, and corticosteroid usage should be investigated, as the early detection affects prognosis [68]. There is increasing evidence that arthroscopic rotator cuff surgery may be another possible cause of osteonecrosis, due to damage to the arterial blood supply to the humeral head during surgery [69].

Advanced practice physiotherapists or practitioners may encounter patients with shoulder arthritis who have used medications such as prednisone in the past or have other comorbidities such as liver disease, have received radiation therapy for cancer, or have a suppressed immune system diseases. Before ordering subacromial or intra-articular corticosteroid injections, they should rule out AVN in high-risk patients. A single shot corticosteroid injection may cause rapid joint destruction due to further reducing the blood flow to the humeral head. This has been reported in the femoral head [70, 71]. Therefore, before steroid administration in high-risk patients, plain radiographs should be taken to rule out an existing AVN and to avoid significant complications.

Hemiarthroplasty or humeral head replacement is preferred in patients with osteonecrosis isolated to the humeral head as this procedure preserves the native and congruent glenoid. Otherwise anatomical or reverse shoulder arthroplasty may be incited based on the status of the rotator cuff and glenoid bone stock.

Septic Arthritis

Important clinical features of acute sepsis are progressive symptoms in a single joint, erythema (redness of the skin caused by dilatation and congestion of the capillaries) overlying the joint, and joint effusions [72]. Severe stiffness should always be considered a potential presentation of septic arthritis [73]. Clinical comorbidities associated with septic shoulder are alcohol-related liver disease, arteriosclerotic heart disease, and leukemia [74]. Laboratory features of septic arthritis includes elevated C-reactive protein, which can also happen during inflammatory conditions such as rheumatoid arthritis and some cardiovascular diseases [75].

Neuropathic Arthropathy

Neuropathic arthropathy of the shoulder is a rare disorder characterized by joint degeneration and is associated with loss of sensory innervation. The presenting symptoms of neuropathy are pain, swelling, and loss of range of motion. Neurological symptoms such as hypoesthesia and loss of temperature sense are commonly reported. The genetic neuropathies should be distinguished from acquired (nongenetic) neuropathies caused by venereal disease or diabetes referred to as Charcot neuropathy.

Charcot-Marie-Tooth (CMT) is a hereditary neuropathy and refers to a group of disorders characterized by a chronic motor and sensory polyneuropathy. The CMS is named after three physicians who independently described the signs and symptoms of this disease in 1886, Jean-Martin Charcot and Pierre Marie [76] from France and Howard Henry Tooth [77] from England. This condition typically affects distal muscles (weakness and atrophy) and is often associated with mild to moderate sensory loss [78–82]. The neuropathic joint arthropathy secondary to venereal disease was first described by William Musgrave in 1703 [83]. In 1868, Jean-Martin Charcot [76] provided a detailed description of neuropathic arthropathy as a complication of syphilis which remained the most common cause of neuropathic arthropathy until the mid-1990s. In 1936, William Riely Jordan associated this condition to neuropathy secondary to diabetes [84]. Diabetes is considered to be the most common etiology of neuropathic arthropathy in the Western world today, but syringomyelia may also contribute to its development. Diabetic patients are most commonly afflicted with foot and ankle disease, while syphilis affects the knee and syringomyelia affects the shoulder and elbow joints [85–87]. Arthropathy secondary to acquired neuropathy is believed to be related to loss of normal proprioception and sensation, leading to recurrent trauma, resulting in joint destruction and neurovascular complications leading to neurally initiated reflex hyperemia and very active bone resorption by osteoclasts [88].

In terms of surgical management, shoulder arthroplasty is reported to be a reasonable treatment for neuropathic arthropathy [89]. The clinical indications of hemiarthroplasty, total shoulder arthroplasty, and reverse shoulder arthroplasty are not clear at this point. Use of reverse arthroplasty may be the most promising treatment for recovering function [90], although studies with properly powered sample sizes are not available due to rarity of the condition.

Rare Genetic and Hereditary-Induced Arthritis

The genetic form of arthritis is often considered a secondary arthritis, as the contributing internal markers could explain its presence in certain individuals. Existence of predisposing genes [91–93] might partially explain occurrence of osteoarthritis in the absence of trauma. Family and sibling studies and large epidemiological surveys suggest genetically determined factors for the presence of secondary arthritis. Presence of family clustering of certain features of arthritis such Heberden's nodes of the fingers and increased incidence of knee joint damage are all indicative of a hereditary nature to some forms of secondary arthritis. Spector and MacGregor [94] point out that genes act through a complex web of mechanisms, such as vulnerability to injury, body weight, muscle mass, and bone and cartilage structure and turnover. Treatment for these cases depends on the severity of the osseous and soft tissue structures.

References

1. Landau JP, Hoenecke HR. Genetic and biomechanical determinants of glenoid version: implications for glenoid implant placement in shoulder arthroplasty. J Shoulder Elb Surg. 2009;18(4):661–7.
2. Friedman RJ, Hawthorne KB, Genez BM. The use of computerized tomography in the measurement of glenoid version. J Bone Joint Surg Am. 1992;74(7):1032–7.
3. Domos P, Checchia CS, Walch G. Walch B0 glenoid: pre-osteoarthritic posterior subluxation of the humeral head. J Shoulder Elb Surg. 2018;27(1):181–8.
4. Aleem AW, Chalmers PN, Bechtold D, Khan AZ, Tashjian RZ, Keener JD. Association between rotator cuff muscle size and glenoid deformity in primary glenohumeral osteoarthritis. J Bone Joint Surg Am. 2019;101(21):1912–20.
5. Hartwell MJ, Harold RE, Sweeney PT, Seitz AL, Marra G, Saltzman MD. Imbalance in axial-plane rotator cuff fatty infiltration in posteriorly worn Glenoids in primary glenohumeral osteoarthritis: an MRI-based study. Clin Orthop Relat Res. 2021;479(11):2471–9.

6. Mitterer M, Matis N, Steiner G, Vasvary I, Ortmaier R. Muscle volume imbalance may be associated with static posterior humeral head subluxation. BMC Musculoskelet Disord. 2021;22(1):279-021-04146-3.
7. Dilli A, Ayaz UY, Damar C, Ersan O, Hekimoglu B. Sprengel deformity: magnetic resonance imaging findings in two pediatric cases. J Clin Imaging Sci. 2011;1:13-7514.76691. Epub 2011 Feb 11
8. Harvey EJ, Bernstein M, Desy NM, Saran N, Ouellet JA. Sprengel deformity: pathogenesis and management. J Am Acad Orthop Surg. 2012;20(3):177–86.
9. Mikosch P. Miscellaneous non-inflammatory musculoskeletal conditions. Gaucher disease and bone. Best Pract Res Clin Rheumatol. 2011;25(5):665–81.
10. Ferreira TFA, Dos Santos APT, Leal AS, de Araujo PG, Silva SS, Moraes-Souza H. Chronic osteo-articular changes in patients with sickle cell disease. Adv Rheumatol. 2021;61(1):11-021-00169-5.
11. Choi HK, Liu S, Curhan G. Intake of purine-rich foods, protein, and dairy products and relationship to serum levels of uric acid: the third National Health and nutrition examination survey. Arthritis Rheum. 2005;52(1):283–9.
12. Tabatabaei SM, Tabatabaei SM, Zamani MM, Sabetkish N, Roshani F. Rhazes viewpoints about causes, diagnosis, treatment and prognosis of gout. J Med Ethics Hist Med. 2012;5:3.
13. Ghafouri RR, Araj-Khodaei M, Targhi ST, Varshochi M, Parsian Z, Yarani R, et al. First report of a disease by Rhazes 10 centuries ago. Int J Prev Med. 2019;10:6.
14. Storey GD. Alfred Baring Garrod (1819-1907). Rheumatology (Oxford). 2001;40(10):1189–90.
15. Caughey DE. The arthritis of Constantine IX. Ann Rheum Dis. 1974;33(1):77–80. https://doi.org/10.1136/ard.33.1.77.
16. Tsoucalas G, Sgantzos M. Primary asthenic gout by Augustin-Jacob Landre-Beauvais in 1800: is this the first description of rheumatoid arthritis? Mediterr J Rheumatol. 2017;28(4):223–6.
17. Beck EM. The classic: a treatise on gout. Alfred Baring Garrod. Clin Orthop Relat Res. 1970;71:3–13.
18. McMichael AJ, Sasazuki T, McDevitt HO, Payne RO. Increased frequency of HLA-Cw3 and HLA-Dw4 in rheumatoid arthritis. Arthritis Rheum. 1977;20(5):1037–42.
19. da Mota LM, Neubarth F, Diniz LR, de Carvalho JF, dos Santos Neto LL. Pierre-auguste Renoir (1841-1919) and rheumatoid arthritis. J Med Biogr. 2012;20(2):91–2.
20. Zeidler H. Great artists with rheumatoid arthritis: what did their disease and coping teach? Part I. Pierre-Auguste Renoir and Alexej von Jawlensky. J Clin Rheumatol. 2012;18(7):376–81.
21. Sanchez-Adams J, Leddy HA, McNulty AL, O'Conor CJ, Guilak F. The mechanobiology of articular cartilage: bearing the burden of osteoarthritis. Curr Rheumatol Rep. 2014;16(10):451-014-0451-6.
22. Kerr R, Resnick D, Pineda C, Haghighi P. Osteoarthritis of the glenohumeral joint: a radiologic-pathologic study. AJR Am J Roentgenol. 1985;144(5):967–72.
23. Kobayashi T, Takagishi K, Shitara H, Ichinose T, Shimoyama D, Yamamoto A, et al. Prevalence of and risk factors for shoulder osteoarthritis in Japanese middle-aged and elderly populations. J Shoulder Elb Surg. 2014;23(5):613–9.
24. Ibounig T, Simons T, Launonen A, Paavola M. Glenohumeral osteoarthritis: an overview of etiology and diagnostics. Scand J Surg. 2020:1457496920935018.
25. Oh JH, Chung SW, Oh CH, Kim SH, Park SJ, Kim KW, et al. The prevalence of shoulder osteoarthritis in the elderly Korean population: association with risk factors and function. J Shoulder Elb Surg. 2011;20(5):756–63.
26. Public Health Agency of Canada. Arthritis in Canada: an ongoing Challenge.
27. Badley EM, Wilfong JM, Zahid S, Perruccio AV. The status of arthritis in Canada: National Report. Arthritis Community Research and Evaluation Unit (ACREU) 2019.: https://arthritis.ca/getmedia/13aff08f-f206-4c6e-a709-beb80b97bd51/ACREU_Arthritis-Society_National-Report-2019_final.pdf.
28. Hall S, Buchbinder R. Do imaging methods that guide needle placement improve outcome? Ann Rheum Dis. 2004;63(9):1007–8.

29. Naredo E, Cabero F, Beneyto P, Cruz A, Mondejar B, Uson J, et al. A randomized comparative study of short term response to blind injection versus sonographic-guided injection of local corticosteroids in patients with painful shoulder. J Rheumatol. 2004;31(2):308–14.
30. Henry P, Razmjou H, Dwyer T, Slade Shantz JA, Holtby R. Relationship between probability of future shoulder arthroplasty and outcomes of arthroscopic debridement in patients with advanced osteoarthritis of glenohumeral joint. BMC Musculoskelet Disord. 2015;16:280-015-0741-9.
31. Baumgarten KM, Lashgari CJ, Yamaguchi K. Glenoid resurfacing in shoulder arthroplasty: indications and contraindications. Instr Course Lect. 2004;53:3–11.
32. Gartsman GM, Roddey TS, Hammerman SM. Shoulder arthroplasty with or without resurfacing of the glenoid in patients who have osteoarthritis. J Bone Joint Surg Am. 2000;82(1):26–34.
33. Bishop JY, Flatow EL. Humeral head replacement versus total shoulder arthroplasty: clinical outcomes—a review. J Shoulder Elb Surg. 2005;14(1 Suppl S):141S–6S.
34. Croia C, Bursi R, Sutera D, Petrelli F, Alunno A, Puxeddu I. One year in review 2019: pathogenesis of rheumatoid arthritis. Clin Exp Rheumatol. 2019;37(3):347–57.
35. Alsaber A, Pan J, Al-Herz A, Alkandary DS, Al-Hurban A, Setiya P, et al. Influence of ambient air pollution on rheumatoid arthritis disease activity score index. Int J Environ Res Public Health. 2020;17:2. https://doi.org/10.3390/ijerph17020416.
36. Zhou S, Huang G. Some important inhibitors and mechanisms of rheumatoid arthritis. Chem Biol Drug Des. 2021;
37. Radu AF, Bungau SG. Management of rheumatoid arthritis: an overview. Cell. 2021;10:11. https://doi.org/10.3390/cells10112857.
38. Taylor-Gjevre R, Nair B, Jin S, Quail J. Geographic variation in incidence and prevalence rates for rheumatoid arthritis in Saskatchewan, Canada 2001-2014. Can J Public Health. 2018;109(3):427–35.
39. Costenbader KH, Chang SC, Laden F, Puett R, Karlson EW. Geographic variation in rheumatoid arthritis incidence among women in the United States. Arch Intern Med. 2008;168(15):1664–70.
40. Parks CG, D'Aloisio AA, DeRoo LA, Huiber K, Rider LG, Miller FW, et al. Childhood socioeconomic factors and perinatal characteristics influence development of rheumatoid arthritis in adulthood. Ann Rheum Dis. 2013;72(3):350–6.
41. Zhang X, Li W, Zhang X, Zhang X, Jiang L, Guo Y, et al. Association between polymorphism in TRAF1/C5 gene and risk of rheumatoid arthritis: a meta-analysis. Mol Biol Rep. 2014;41(1):317–24.
42. Kim H, Cho SK, Choi S, Im SG, Jung SY, Jang EJ, et al. Comparison of healthcare resource utilization and medical costs between patients with seropositive and seronegative rheumatoid arthritis. Ther Adv Musculoskelet Dis. 2021;13:1759720X211024830.
43. Levine RB, Sullivan KL. Rheumatoid arthritis: skeletal manifestations observed on portable chest roentgenograms. Skelet Radiol. 1985;13(4):295–303.
44. AAOS. The treatment of glenohumeral joint osteoarthritis guideline and evidence report: American Academy of Orthopaedic Surgeons, Board of Directors. December 2009.
45. Ernstbrunner L, Andronic O, Grubhofer F, Camenzind RS, Wieser K, Gerber C. Long-term results of reverse total shoulder arthroplasty for rotator cuff dysfunction: a systematic review of longitudinal outcomes. J Shoulder Elb Surg. 2019;28(4):774–81.
46. Ho JC, Kane L, Stone MA, Romeo AA, Abboud JA, Namdari S. Arthroscopic debridement of irreparable rotator cuff tears: predictors of failure and success. J Shoulder Elb Surg. 2019;
47. Sevivas N, Ferreira N, Andrade R, Moreira P, Portugal R, Alves D, et al. Reverse shoulder arthroplasty for irreparable massive rotator cuff tears: a systematic review with meta-analysis and meta-regression. J Shoulder Elb Surg. 2017;26(9):e265–77.
48. 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(5):527–40.

49. Frankle MA, Teramoto A, Luo ZP, Levy JC, Pupello D. Glenoid morphology in reverse shoulder arthroplasty: classification and surgical implications. J Shoulder Elb Surg. 2009;18(6):874–85.
50. Mizuno N, Denard PJ, Raiss P, Walch G. Reverse total shoulder arthroplasty for primary glenohumeral osteoarthritis in patients with a biconcave glenoid. J Bone Joint Surg Am. 2013;95(14):1297–304.
51. Bois AJ, Knight P, Alhojailan K, Bohsali KI. Clinical outcomes and complications of reverse shoulder arthroplasty used for failed prior shoulder surgery: a systematic review and meta-analysis. JSES Int. 2020;4(1):156–68.
52. Hovelius L, Olofsson A, Sandstrom B, Augustini BG, Krantz L, Fredin H, et al. Nonoperative treatment of primary anterior shoulder dislocation in patients forty years of age and younger. A prospective twenty-five-year follow-up. J Bone Joint Surg Am. 2008;90(5):945–52.
53. Marx RG, McCarty EC, Montemurno TD, Altchek DW, Craig EV, Warren RF. Development of arthrosis following dislocation of the shoulder: a case-control study. J Shoulder Elb Surg. 2002;11(1):1–5.
54. Kavaja L, Pajarinen J, Sinisaari I, Savolainen V, Bjorkenheim JM, Haapamaki V, et al. Arthrosis of glenohumeral joint after arthroscopic Bankart repair: a long-term follow-up of 13 years. J Shoulder Elb Surg. 2012;21(3):350–5.
55. Hovelius L, Rahme H. Primary anterior dislocation of the shoulder: long-term prognosis at the age of 40 years or younger. Knee Surg Sports Traumatol Arthrosc. 2016;24(2):330–42.
56. Hovelius L, Saeboe M. Neer Award 2008: arthropathy after primary anterior shoulder dislocation—223 shoulders prospectively followed up for twenty-five years. J Shoulder Elb Surg. 2009;18(3):339–47.
57. Rossleigh MA, Smith J, Straus DJ, Engel IA. Osteonecrosis in patients with malignant lymphoma. A review of 31 cases. Cancer. 1986;58(5):1112–6.
58. Cruess RL. Corticosteroid-induced osteonecrosis of the humeral head. Orthop Clin North Am. 1985;16(4):789–96.
59. Cruess RL. Osteonecrosis of bone. Current concepts as to etiology and pathogenesis. Clin Orthop Relat Res. 1986;208:30–9.
60. Gruson KI, Kwon YW. Atraumatic osteonecrosis of the humeral head. Bull NYU Hosp Jt Dis. 2009;67(1):6–14.
61. McKee MD. Atraumatic osteonecrosis of the humeral head. J Rheumatol. 2000;27(7):1582–4.
62. Mont MA, Payman RK, Laporte DM, Petri M, Jones LC, Hungerford DS. Atraumatic osteonecrosis of the humeral head. J Rheumatol. 2000;27(7):1766–73.
63. Lau MW, Blinder MA, Williams K, Galatz LM. Shoulder arthroplasty in sickle cell patients with humeral head avascular necrosis. J Shoulder Elb Surg. 2007;16(2):129–34.
64. Wang Y, Li Y, Mao K, Li J, Cui Q, Wang GJ. Alcohol-induced adipogenesis in bone and marrow: a possible mechanism for osteonecrosis. Clin Orthop Relat Res. 2003;410:213–24. doi(410):213-224
65. Painter CF. Subdeltoid bursitis. Boston Med Surg. 1907;156:345–9.
66. Jissendi Tchofo P, Brasseur P. Aseptic osteonecrosis of alcoholic origin: a case report. Rev Med Brux. 2001;22(3):170–3.
67. Fisher DE. The role of fat embolism in the etiology of corticosteroid-induced avascular necrosis: clinical and experimental results. Clin Orthop Relat Res. 1978;130:68–80.
68. Hernigou P, Hernigou J, Scarlat M. Shoulder osteonecrosis: pathogenesis, causes, clinical evaluation, imaging, and classification. Orthop Surg. 2020;12(5):1340–9.
69. Keough N, Lorke DE. The humeral head: a review of the blood supply and possible link to osteonecrosis following rotator cuff repair. J Anat. 2021;239(5):973–82.
70. Thompson AR, Ensrud ER. Rapid onset of femoral head osteonecrosis after a single intra-articular hip joint injection of corticosteroid. Am J Phys Med Rehabil. 2020;99(4):e54–5.
71. Yamamoto T, Schneider R, Iwamoto Y, Bullough PG. Rapid destruction of the femoral head after a single intraarticular injection of corticosteroid into the hip joint. J Rheumatol. 2006;33(8):1701–4.

72. Courtney P, Doherty M. Joint aspiration and injection and synovial fluid analysis. Best Pract Res Clin Rheumatol. 2009;23(2):161–92.
73. Sambandam SN, Atturu M. A case of septic arthritis of shoulder presenting as stiffness of the shoulder. J Orthop Case Rep. 2016;6(2):31–3.
74. Master R, Weisman MH, Armbuster TG, Slivka J, Resnick D, Goergen TG. Septic arthritis of the glenohumeral joint. Unique clinical and radiographic features and a favorable outcome. Arthritis Rheum. 1977;20(8):1500–6.
75. Du Clos TW, Mold C. C-reactive protein: an activator of innate immunity and a modulator of adaptive immunity. Immunol Res. 2004;30(3):261–77.
76. Charcot JM, Marie P. Sur une forme particulaiere d'atrophie musculaire progressive souventfamiliale debutant par les pied et les jambes et atteignant plus tard les mains. Rev Med. 1886;6:97–138.
77. Tooth H. The peroneal type of progressive muscular atrophy. London, England: Lewis, HK; 1886.
78. Bird TD. Charcot-Marie-Tooth hereditary neuropathy overview. In: Pagon RA, Bird TD, Dolan CR, Stephens K, Adam MP, editors. . GeneReviews Seattle (WA): University of Washington, Seattle; 1993.
79. Gupta R. A short history of neuropathic arthropathy. Clin Orthop Relat Res. 1993;296:43–9.
80. Rajbhandari SM, Jenkins RC, Davies C, Tesfaye S. Charcot neuroarthropathy in diabetes mellitus. Diabetologia. 2002;45(8):1085–96.
81. Barisic N, Claeys KG, Sirotkovic-Skerlev M, Lofgren A, Nelis E, De Jonghe P, et al. Charcot-Marie-Tooth disease: a clinico-genetic confrontation. Ann Hum Genet. 2008;72(Pt 3):416–41.
82. Jani-Acsadi A, Krajewski K, Shy ME. Charcot-Marie-Tooth neuropathies: diagnosis and management. Semin Neurol. 2008;28(2):185–94.
83. Kelly M. De Arthritide Symptomatica of William Musgrave (1657-1721): his description of neuropathic arthritis. Bull Hist Med. 1963;37:372–7.
84. Ramanujam CL, Zgonis T. The diabetic charcot foot from 1936 to 2016: eighty years later and still growing. Clin Podiatr Med Surg. 2017;34(1):1–8.
85. Brain R, Wilkinson M. Cervical arthropathy in syringomyelia, tabes dorsalis and diabetes. Brain. 1958;81(3):275–89.
86. Rickert MM, Cannon JG, Kirkpatrick JS. Neuropathic arthropathy of the shoulder: a systematic review of classifications and treatments. JBJS Rev. 2019;7(10):e1.
87. Storey G. Charcot joints. Br J Vener Dis. 1964;40:109–17.
88. Brower AC, Allman RM. The neuropathic joint: a neurovascular bone disorder. Radiol Clin N Am. 1981;19(4):571–80.
89. Schoch B, Werthel JD, Sperling JW, Cofield RH, Sanchez-Sotelo J. Is shoulder arthroplasty an option for charcot arthropathy? Int Orthop. 2016;40(12):2589–95.
90. Wawrzyniak A, Lubiatowski P, Kordasiewicz B, Brzoska R, Laprus H. Shoulder arthropathy secondary to syringomyelia: case series of 10 patients. Eur J Orthop Surg Traumatol. 2021;
91. Loughlin J. The genetic epidemiology of human primary osteoarthritis: current status. Expert Rev Mol Med. 2005;7(9):1–12.
92. Loughlin J. Genetic epidemiology of primary osteoarthritis. Curr Opin Rheumatol. 2001;13(2):111–6.
93. Valdes AM, Spector TD. The contribution of genes to osteoarthritis. Rheum Dis Clin N Am. 2008;34(3):581–603.
94. Spector TD, MacGregor AJ. Risk factors for osteoarthritis: genetics. Osteoarthr Cartil. 2004;12(Suppl A):S39-44.

Chapter 8
Superior Labral Anterior and Posterior Lesions

The glenoid labrum is a fibrocartilaginous structure and assists with shoulder stability by deepening the glenoid cavity and by restricting anterior and posterior movement of the humerus. The morphology of the labrum is distinctly different in its inferior and superior regions. The inferior portion of the labrum is firmly attached to the glenoid rim and is a fibrous and an immobile extension of the articular cartilage, while the superior part of the labrum is more loosely attached to glenoid and is more susceptible to injury. The long head of the biceps tendon is anchored to the superior labrum and the superior glenoid tubercle, near the 12-o'clock position. The superior portion of the labrum is less vascular than other portions, which does affect the management of tears involving the superior portion of the labrum [1, 2].

Historical Perspective

The superior labral lesions were first observed in baseball pitchers during shoulder arthroscopy by Andrews et al. in 1985 [3]. The authors described an injury of the superior labrum that extended to the anterior and posterior of the labrum, including the biceps tendon [3, 4]. It 1990, the term SLAP (superior labral anterior and posterior) was coined by Snyder and colleagues [5] who provided more details on the location and nature of this pathology. The original classification of the SLAP tears, described by Snyder, included four types of lesions of isolated or concurrent pathology in the superior labrum [5]. The categories were then expanded by Maffet et al. 5 years later and included types V–VII [6]. Further addition to SLAP tear types (VIII–X) was proposed by Nord and Ryu in 2004 (reprinted in 2012) [7], which included more extensive tears, extending to other parts of the labrum. While the original classification by Snyder is reported to be reliable in the hands of experienced surgeons [8], the reliability of Maffet's expansion, often used in practice today, has been reported to be fair with an overall kappa value of 0.26 [9]. The

H. Razmjou, M. Christakis, *Clinical and Radiological Examination of the Shoulder Joint*, https://doi.org/10.1007/978-3-031-10470-1_8

significance and diagnostic reliability of more advanced SLAP tears remain unclear at present.

The clinical interest in SLAP lesions and the publication fever on the subject appears to have raised between years 2000 and 2009, being almost twice as publication rate between 1990 and 1999 [10]. Of interest, the dramatic increase in the number of SLAP repair surgeries in early years of its recognition declined after 2009, and the percentage of SLAP repairs performed in comparison to the total number of shoulder arthroscopic surgeries has significantly decreased over the more recent years [11]. This may somewhat be related to the suboptimal surgical results in certain age groups or those with concomitant pathologies. These tears are reported to account for up to 90% of labral pathology in the stable shoulders and are often seen with other shoulder pathologies [12]. Based on a recently published article in 2021, the level of evidence of majority of the early articles on SLAP lesions was of level IV indicating that more vigorous research is needed to better understand the diagnosis, clinical relevance, and the best care for this pathology [10].

Characteristics, Classification, and Causes

According to Snyder's classification, type I SLAP lesions are commonly associated with age-related degenerative changes [13] and involve fraying of the superior glenoid labrum with an intact biceps tendon [5, 14, 15]. Type II SLAP lesion is usually associated with repetitive microtrauma or a significant traction injury and accounts for majority of SLAP lesions. In type II lesions, the superior labrum along with the biceps anchor from the superior glenoid rim is detached, which may lead to significant instability at the anterior-posterior and superior-inferior directions [5, 15, 16]. Type III SLAP lesion is a bucket-handle tear of the superior labrum with no involvement of the biceps tendon. Type IV SLAP lesions involve a bucket-handle tear of the superior labrum with extension into the biceps tendons. Associated pathologies include posterosuperior impingement leading to articular-sided rotator cuff tears and cystic changes of the humeral head. In cases of anterior subluxation or dislocation, a Bankart lesion or fracture may be seen in the anteroinferior aspect of the labrum or glenoid, respectively. Types V–VII involve other structures [6]. Type V SLAP lesion is a Bankart lesion that continues superiorly and includes the biceps tendon (combined SLAP lesion type II and Bankart lesion). Type VI is a combined SLAP type II lesion and an unstable labral flap, and type VII involves SLAP type II with an extension of the tear to the middle glenohumeral ligament.

In terms of etiology of the SLAP tears, they may be caused by an acute traumatic injury or chronic or repetitive microtrauma. Traction injuries may involve an isolated significant injury seen in gymnasts or during a fall when the arm remains attached to an immobile object, like a banister. Microtrauma may be secondary to overhead arm movements (deceleration phase) seen in baseball, tennis, swimming, or repetitive throwing as a part of certain occupations such as garbage collection. The direct axial compression load during the fall on an outstretched arm may damage the labrum superiorly, due to impaction of the humeral head against the glenoid. Bracing oneself during a rear-ended motor vehicle accident initiates an eccentric contraction of the long head of the biceps at the time of impact and can cause a type II SLAP lesion. Overall, falls on an outstretched arm causing compression loading and traction injuries are the most common causes of acute traumatic SLAP lesions [5, 15, 17, 18].

Clinical Findings of SLAP Lesions

The symptoms of SLAP lesions are nonspecific and inconsistent and include a dull pain, catching sensation, numbness, or tingling. In more advanced cases, instability may be reported [19]. The symptoms are often produced or aggravated by excessive abduction and external rotation during a hard throw in athletes or similar positions in overhead occupations. This phenomenon is referred to as the "dead arm syndrome" [20] when a transient stretch to the brachial plexus causes numbness, tingling, and sudden paralysis of the arm, affecting the velocity of the throwing. Presence of posterior shoulder pain, indicating humeral head mal-tracking, which allows internal impingement of the infraspinatus on the posterior superior glenoid, has been noted as a critical component of SLAP lesion symptomatology [21]. Gymnasts have higher complaints of positive apprehension in addition to pain due to increased glenohumeral translation [22].

Physical examination may reveal shoulder laxity, increased external rotation in abduction, and a tight posterior capsule, associated with decreased internal rotation. Common associated lesions such as rotator cuff tears and anteroinferior labral pathology make the clinical examination difficult, and therefore, imaging plays an important role in diagnosis of SLAP lesions [23]. Clinically, a large number of specific tests have been proposed to detect SLAP tears. However, the original studies do not often meet the rigor of a proper validity study, and to date independent studies and systematic reviews [24–29] have not found a single physical test or sign that would accurately rule in or out SLAP lesions. Dessaur and Magarey highlight that most often the high validity and reliability claimed by the original developers are not supported by subsequent independent authors [24]. Proper anatomical studies are usually needed to examine the biomechanical basis of new clinical tests. In an attempt to examine the anatomical basis for O'Brian test, Green et al. [30] measured active and passive tension in the long head of biceps in the two positions of the O'Brien test in cadavers. The authors found that active and passive tensions were higher in the negative position, thus refuting the proposed anatomic basis of the test. They emphasized that failure to support the proposed anatomic basis may partly explain the variable likelihood ratios obtained in clinical accuracy studies of the O'Brien test. Lack of accuracy has been reported for other clinical tests such as the crank test [26, 27, 31] and Speed's test [18]. Overall, no single test has shown sufficient sensitivity and specificity for the consistent clinical diagnosis of SLAP lesions [32, 33]. In a systematic review published in 2017 and based on limited number of studies, the SLAP lesion clinical tests appear to be more specific than sensitive, indicating that a negative clinical test does not accurately rule out the lesion [34].

The gold standard for detection of SLAP lesions at this point is the MR arthrography as it provides a more reliable and accurate mode of imaging compared with other imaging modes [35]. MR arthrography may be conducted directly or indirectly. In direct arthrography, an intra-articular contrast material is injected into the joint, where the indirect MR arthrography is performed with an intravenous contrast material injection. The direct MR arthrography provides good inspection of anatomic findings due to joint distension, which allows passage of contrast material into the labral substance in the setting of unstable tears or labral detachment. With indirect MR arthrography, intravenously administered gadolinium-based contrast agent is used to enhance the joint space [36]. A systematic review of the literature by Symanski indicates that the direct MR arthrography is more accurate compared with MRI and indirect MR arthrography [37].

One important note of caution is that the imaging-detected SLAP lesions are very common and seen in up to 72% in middle-aged asymptomatic patients [38]. In older individuals without history of instability, use of MR arthrogram may lead to overdiagnosing of clinically irrelevant SLAP lesions and unnecessary surgical procedures. Therefore, reassuring the patient of the incidental or clinically irrelevant nature of the MRI-detected lesions is important as many do not need surgery, especially in the absence of an acute injury. Moreover, the widespread variations of labral anatomy identified on the MRI of asymptomatic overhead throwers may indicate that the labrum is not always the source of the patient's pain [39]. For example, a Buford complex, defined as the absence of the anterior superior labrum in conjunction with a thickened cord-like MGHL, is found in 1.5% of individuals [40]. This normal variation can be confused with a sublabral hole or pathologic labral detachment. If mistakenly surgically reattached to the neck of the glenoid cartilage, severe painful restriction of humeral rotation and elevation can occur [23].

Management

Since the conception of SLAP lesions in 1985, the clinician's understanding of the nature of this pathology has improved, but due to the inconsistent clinical presentation, inability to reliably diagnose different types of SLAP lesions, and variability in proposed surgical techniques, the best approach to management of these lesions remains debatable and controversial [9].

The literature has shown that over time the number of SLAP repairs has decreased and the number of biceps tenodesis procedures has increased. In addition, role of age and clinical presentation (e.g., instability) is being more acknowledged in the choice of conservative vs. surgical management and different types of surgery. Conservative treatment includes activity modification (refraining from provocative sports or occupation-related activities), strengthening of the rotator cuff muscles, proprioceptive neuromuscular rehabilitation exercises, and core strengthening and scapular exercises in addition to restoration of shoulder range of motion, especially internal rotation. Nevertheless, an acute superior labral tear in younger individuals presenting with shoulder instability may not respond successfully to conservative treatment.

Surgery should be considered only when nonoperative treatment modalities have been exhausted. The current surgical approach for type II lesions, which involves separation of the biceps tendon and superior labrum from the supraglenoid cartilage rim, is affected by the patient's age with labral repair being more recommended in younger patients, whereas biceps tenodesis is more preferable in older patients. The literature indicates that patients >40 years of age, who suffer from biceps pathology but do not have a history of a traumatic injury, do not often benefit from the SLAP repair and may just need biceps tenotomy/tenodesis [21, 41, 42]. Younger patients with a history of a significant and easily recalled trauma; complaints of shifting, clicking, or popping with certain movements; and posterior shoulder pain and clinical signs of instability are more suitable for SLAP repair [21].

In summary, the nonoperative management including rigorous physiotherapy should be exhausted first. Surgical management includes biceps tenotomy or tenodesis for older patients without instability symptoms and SLAP repair with or without biceps tenotomy/tenodesis for younger patients with symptomatic instability [9, 11, 43]. Treatment of overhead throwing athletes should be first focused on aggressive rehabilitation with minimal surgical interventions to improve their chances of return to sport and pre-injury activity level [21].

References

1. Cooper DE, Arnoczky SP, O'Brien SJ, Warren RF, DiCarlo E, Allen AA. Anatomy, histology, and vascularity of the glenoid labrum. An anatomical study. J Bone Joint Surg Am. 1992;74(1):46–52.
2. Howell SM, Galinat BJ. The glenoid-labral socket. A constrained articular surface. Clin Orthop Relat Res. 1989;243:122–5.
3. 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.
4. Andrews JR, Broussard TS, Carson WG. Arthroscopy of the shoulder in the management of partial tears of the rotator cuff: a preliminary report. Arthroscopy. 1985;1(2):117–22.
5. Snyder SJ, Karzel RP, Del Pizzo W, Ferkel RD, Friedman MJ. SLAP lesions of the shoulder. Arthroscopy. 1990;6(4):274–9.
6. Maffet MW, Gartsman GM, Moseley B. Superior labrum-biceps tendon complex lesions of the shoulder. Am J Sports Med. 1995;23(1):93–8.
7. Powell SE, Nord KD, Ryu RKN. The diagnosis, classification, and treatment of SLAP lesions. Oper Tech Sports Med. 2012;20(1):46–56.
8. Jia X, Yokota A, McCarty EC, Nicholson GP, Weber SC, McMahon PJ, et al. Reproducibility and reliability of the Snyder classification of superior labral anterior posterior lesions among shoulder surgeons. Am J Sports Med. 2011;
9. Wang KK, Yalizis M, Hoy GA, Ek ET. Current trends in the evaluation and treatment of SLAP lesions: analysis of a survey of specialist shoulder surgeons. JSES Open Access. 2018;2(1):48–53.

10. Allegra PR, Greif DN, Desai SS, Yakkanti RR, Munoz J, Kaplan LD, et al. The fifty most-cited articles regarding SLAP lesions. Arthrosc Sports Med Rehabil. 2021;3(1):e135–47.
11. Erickson BJ, Jain A, Abrams GD, Nicholson GP, Cole BJ, Romeo AA, et al. SLAP lesions: trends in treatment. Arthroscopy. 2016;32(6):976–81.
12. Ilahi OA, Labbe MR, Cosculluela P. Variants of the anterosuperior glenoid labrum and associated pathology. Arthroscopy. 2002;18(8):882–6.
13. Mohana-Borges AV, Chung CB, Resnick D. Superior labral anteroposterior tear: classification and diagnosis on MRI and MR arthrography. AJR Am J Roentgenol. 2003;181(6):1449–62.
14. Handelberg F, Willems S, Shahabpour M, Huskin JP, Kuta J. SLAP lesions: a retrospective multicenter study. Arthroscopy. 1998;14(8):856–62.
15. 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.
16. Panossian VR, Mihata T, Tibone JE, Fitzpatrick MJ, McGarry MH, Lee TQ. Biomechanical analysis of isolated type II SLAP lesions and repair. J Shoulder Elb Surg. 2005;14(5):529–34.
17. Bey MJ, Elders GJ, Huston LJ, Kuhn JE, Blasier RB, Soslowsky LJ. The mechanism of creation of superior labrum, anterior, and posterior lesions in a dynamic biomechanical model of the shoulder: the role of inferior subluxation. J Shoulder Elb Surg. 1998;7(4):397–401.
18. Holtby R, Razmjou H. Accuracy of the Speed's and Yergason's tests in detecting biceps pathology and SLAP lesions: comparison with arthroscopic findings. Arthroscopy. 2004;20(3):231–6.
19. Park D. Clinical characteristics of patients with posterosuperior labral tear: a comparison with patients with other shoulder disorders. J Pain Res. 2018;11:1795–802.
20. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology part I: pathoanatomy and biomechanics. Arthroscopy. 2003;19(4):404–20.
21. Hester WA, O'Brien MJ, Heard WMR, Savoie FH. Current concepts in the evaluation and management of type II superior labral lesions of the shoulder. Open Orthop J. 2018;12:331–41.
22. Takeuchi Y, Sugaya H, Takahashi N, Matsuki K, Tokai M, Morioka T, et al. Superior labral injuries in elite gymnasts: symptoms, pathology, and outcomes after surgical repair. Orthop J Sports Med. 2020;8(7):2325967120935001.
23. Chang D, Mohana-Borges A, Borso M, Chung CB. SLAP lesions: anatomy, clinical presentation, MR imaging diagnosis and characterization. Eur J Radiol. 2008;68(1):72–87.
24. Dessaur WA, Magarey ME. Diagnostic accuracy of clinical tests for superior labral anterior posterior lesions: a systematic review. J Orthop Sports Phys Ther. 2008;38(6):341–52.
25. Calvert E, Chambers GK, Regan W, Hawkins RH, Leith JM. Special physical examination tests for superior labrum anterior posterior shoulder tears are clinically limited and invalid: a diagnostic systematic review. J Clin Epidemiol. 2009;62(5):558–63.
26. Karlsson J. Physical examination tests are not valid for diagnosing SLAP tears: a review. Clin J Sport Med. 2010;20(2):134–5.
27. Hegedus EJ, Goode A, Campbell S, Morin A, Tamaddoni M, Moorman CT 3rd, et al. Physical examination tests of the shoulder: a systematic review with meta-analysis of individual tests. Br J Sports Med. 2008;42(2):80–92. discussion 92
28. Jones GL, Galluch DB. Clinical assessment of superior glenoid labral lesions: a systematic review. Clin Orthop Relat Res. 2007;455:45–51.
29. Munro W, Healy R. The validity and accuracy of clinical tests used to detect labral pathology of the shoulder—a systematic review. Man Ther. 2009;14(2):119–30.
30. Green RA, Taylor NF, Mirkovic M, Perrott M. An evaluation of the anatomic basis of the O'Brien active compression test for superior labral anterior and posterior (SLAP) lesions. J Shoulder Elb Surg. 2008;17(1):165–71.
31. Stetson WB, Templin K. The crank test, the O'Brien test, and routine magnetic resonance imaging scans in the diagnosis of labral tears. Am J Sports Med. 2002;30(6):806–9.
32. Hegedus EJ, Goode AP, Cook CE, Michener L, Myer CA, Myer DM, et al. Which physical examination tests provide clinicians with the most value when examining the shoulder? Update of a systematic review with meta-analysis of individual tests. Br J Sports Med. 2012;46(14):964–78.

33. Parentis MA, Glousman RE, Mohr KS, Yocum LA. An evaluation of the provocative tests for superior labral anterior posterior lesions. Am J Sports Med. 2006;34(2):265–8.
34. Gismervik SO, Drogset JO, Granviken F, Ro M, Leivseth G. Physical examination tests of the shoulder: a systematic review and meta-analysis of diagnostic test performance. BMC Musculoskelet Disord. 2017;18(1):41. -017-1400-0
35. 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(1):127–32.
36. De Coninck T, Ngai SS, Tafur M, Chung CB. Imaging the glenoid Labrum and labral tears. Radiographics. 2016;36:1628–47.
37. Symanski JS, Subhas N, Babb J, Nicholson J, Gyftopoulos S. Diagnosis of superior Labrum anterior-to-posterior tears by using MR imaging and MR arthrography: a systematic review and meta-analysis. Radiology. 2017;285(1):101–13.
38. Schwartzberg R, Reuss BL, Burkhart BG, Butterfield M, Wu JY, McLean KW. High prevalence of superior labral tears diagnosed by MRI in middle-aged patients with asymptomatic shoulders. Orthop J Sports Med. 2016;4(1):1–7.
39. Mathew CJ, Lintner DM. Superior labral anterior to posterior tear management in athletes. Open Orthop J. 2018;12:303–13.
40. Stoller DW. MR arthrography of the glenohumeral joint. Radiol Clin N Am. 1997;35(1):97–116.
41. 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(4):880–6.
42. 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(5):929–36.
43. LeVasseur MR, Mancini MR, Hawthorne BC, Romeo AA, Calvo E, Mazzocca AD. SLAP tears and return to sport and work: current concepts. J ISAKOS. 2021;6(4):204–11.

Chapter 9
Glenohumeral Joint Instability

The integrity of the glenohumeral joint depends on the congruent articulating surfaces and surrounding soft tissues for static and dynamic stability. The ligaments are the most important passive stabilizers. The glenoid labrum, rotator interval, and rotator cable are other important static stabilizers. Rotator cuff muscles and the biceps tendon are the dynamic stabilizers of the glenohumeral joint.

The middle glenohumeral ligament (MGHL) acts as a secondary restraint to anterior translation [1]. The superior glenohumeral ligament (SGHL) is the primary restraint to inferior translation of the adducted shoulder [2]. Other inferior stabilizers are the coracohumeral ligament [3] and the inferior glenohumeral ligament (IGHL). The IGHL adheres to the peripheral aspect of the labrum, reinforces the joint capsule inferiorly, and inserts into the anatomical neck of the humerus [4, 5]. The IGHL is usually affected in congenital or acquired instability secondary to overhead sports activities [6]. The posterior inferior glenohumeral ligament (PIGHL) and posterior labrum and capsule are the primary static stabilizers against posterior translation [7]. The PIGHL is a major stabilizing structure in flexion and internal rotation, a vulnerable position for posterior dislocation [8].

Glenoid labrum is an important static stabilizer of the glenohumeral joint. Presence of the labrum increases the depth of the glenoid fossa from 2.5 to 5 mm which assists with passive stability of the joint [9]. The intra-articular negative pressure and the viscous articular liquid are other static stabilizing structures of the glenohumeral joint as they help to increase the cohesion and adhesion, while preserving mobility of the glenohumeral joint [10].

The role of rotator interval and rotator cable as important static stabilizers of the glenohumeral joint has been more recently acknowledged [11]. The rotator interval is a triangular and multilayer area that contains multiple stabilizers of the glenohumeral joint such as the long head of the biceps tendon and several ligaments (SGHL, MGHL, and coracohumeral). Rotator interval is surrounded by the coracoid process medially, the subscapularis anteroinferiorly, and the supraspinatus posterosuperiorly [12–17] and plays an important role in anterior and inferior stability of the

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shoulder. The deepest layer of the rotator interval consists of a capsule which in adhesive capsulitis can be significantly affected by synovitis [18].

The rotator cable is a strong fibrous tissue that acts as a stress-shielding structure for the rotator crescent, an avascular crescent-shaped area at the insertion of the supraspinatus and infraspinatus tendons. Rotator cable originates from the deep layer of the coracohumeral ligament and moves along the supraspinatus and infraspinatus tendon fibers [19]. It has been reported that the rotator cable becomes thicker with aging to compensate for the thinner and more vulnerable rotator crescent [20]. This may have significant clinical implication and explain why some elderly with rotator cuff tear remain fully functional as long as the rotator cable is intact. In short, as the person ages, the rotator cuff tendon integrity becomes more dependent on the rotator cable, and this explains asymptomatic cuff tears in older patients [20] (see Chap. 4 for details).

Dynamic stabilization of the glenohumeral joint is provided by the rotator cuff tendons [21], particularly in the mid- and end range of motion [22]. The rotator cuff provides a concavity-compression effect of the humeral head against the glenoid cavity [23]. The infraspinatus has a critical role in providing superior instability, and the subscapularis is the primary dynamic restraint to posterior translation [24].

The long head of the biceps tendon and deltoid muscle are important dynamic stabilizers of the glenohumeral joint as well. The importance of the long head of the biceps in glenohumeral stability was well recognized in the nineteenth-century literature [25–29]. Biceps brachii is a complex muscle with its long head acting as a restraint to superior subluxation [30–32]. More recent research [33] has shown that loading of the long head of the biceps tendon resists posterior translation of the humeral head during forward flexion. Accordingly, sacrificing of the intra-articular segment of this tendon will have a negative impact on superior stability of the glenohumeral joint [31], which may explain an accelerated cuff tear arthropathy after a failed cuff repair and biceps tenodesis in patients with a normal preoperative biomechanics and well-maintained acromiohumeral distance. In addition, it has been suggested that unloading the long head of the biceps tendon that occurs in biceps tenotomy or tenodesis may eventually lead to posterior labral pathology or to the posterior glenoid wear commonly seen with osteoarthritis [33].

Historical Perspective

The earliest documentation of shoulder instability goes back to 3000 BC in the Egyptian tomb wall paintings. The ancient Egyptian medical papyrus bought by Edwin Smith, an American Egyptologist in 1862, describes several cases of traumatic injuries and shoulder dislocations [34]. Hippocrates, an ancient Greek physician, is credited for early detailed description of anatomy, type of dislocation, and techniques used for reduction of anterior instability around 400 BC. He described a procedure that involved using a red-hot iron to burn the soft tissues of the shoulder joint by scar formation [35]. His methods are still being used today with cauterization of the shoulder capsule for chronic instability.

The more formal literature on anterior dislocation was published in 1890 by two French general surgeons, Broca and Hartman in relation to war injuries [36]. In 1923, a British orthopedic surgeon, Arthur Sidney Blundell Bankart [37, 38], provided a brief description of the labral complex avulsion from the scapular periosteum and proposed a reconstruction surgery of the labrum and capsule. In 1938, he provided a detailed description of the pathology and surgical approach for what was named after him as the Bankart repair [38].

In contrast with anterior instability, posterior shoulder instability was not well reported in the literature until 1741, when a chronic posterior dislocation case was first discussed by White [39]. The first seizure-related posterior dislocation was reported by Cooper in 1839 [40]. In this report, the symptoms and clinical signs of posterior dislocation were noted as posterior fullness of the affected side, inability to externally rotate, and severe limitation of abduction. In 1952, McLaughlin described a range of posterior instability-related conditions, including the locked posterior dislocation [41]. To honor his contribution to this field, the reverse Hill-Sachs lesion was named after him as the McLaughlin lesion.

Generalized hypermobility was first described by Hippocrates in warriors of Central Asia [42]. The first mention of hypermobility in modern scientific literature was in 1965 when Kirk described the clinical presentation of this condition [43]. In 1980, the term multidirectional instability (MDI) was coined by Neer and Foster for symptomatic instability of the shoulder in two or more directions secondary to ligamentous and capsular redundancy and increased capsular volume [44].

Characteristics, Classification, and Causes

The most common dislocation of the glenohumeral join is anterior, with rare cases of posterior and inferior dislocations. In anterior dislocation, the injury affects the anteroinferior aspect of the labrum with or without an impaction fracture of the posterolateral of the humeral head (Hill-Sachs lesion). Posterior dislocation leads to damage of the posteroinferior capsulolabral shoulder complex and the anteromedial humeral head (reverse Hill-Sachs lesion). Multidirectional instability is a symptomatic instability in more than one direction, which is a different clinical entity than asymptomatic benign hypermobility. Redundant inferior capsular structures are the hallmark of symptomatic multidirectional instability. The improper alignment of the scapula also can inhibit subscapularis, serratus anterior, and lower trapezius which affects the overall dynamic stability provided by the muscles [45].

An overall incidence rate of 23.9 dislocation per 100,000 person-years has been reported in the United States, most frequently related to a fall at home or at sports or recreation sites, with 71.8% of the dislocations occurring in males. The maximum incidence rate of shoulder dislocation is reported to occur between the ages of 20 and 29 years [46].

Anterior Glenohumeral Instability

Anterior instability accounts for 95% of acute traumatic dislocations [10]. Approximately, more than 50% of patients with primary traumatic anterior dislocations treated conservatively are reported to have an additional instance of instability within 2 years of the initial traumatic dislocation with a slight increase within 5 years [47]. Chance of dislocation remains a possibility even after surgery. Flinkkila et al. reported a recurrence dislocation rate of 19% (of 186 shoulders) in patients who had undergone a Bankart repair [48]. Younger age seemed to be the most important factor for recurrent instability [47, 48].

Age at the time of initial dislocation is an important contributing factor for occurrence of other associated pathologies. For instance, older age has been associated with a concurrent rotator cuff tear [49] which presents with an inability to abduct the shoulder in some patients post-reduction [50]. A prospective controlled study [49] of 167 patients with primary traumatic anterior shoulder dislocation found a prevalence of 32% (53/167) full-thickness cuff tears being more prevalent in older patients (>60 years) and in women. The authors reported a high suspicion of rotator cuff tear (76.7%) in those who at 2 weeks post-dislocation/reduction could not elevate more than 90 degrees.

Apart from rotator cuff injury, anterior dislocation may be associated with nerve injury. The cause of injury is believed to be related to the nerve being crushed between the humeral head and the axillary border of the scapula or simply being stretched across the humeral head in the abducted and externally rotated position of the arm [51, 52]. A less commonly seen cause of nerve injury is connected with damage to vascular structures at the time of dislocation or the abrupt movement during reduction [53]. More complex neurological deficits are often reported with a low-energy fall in an elderly female. Single nerve injury is more likely to occur as a result of high-energy trauma in a younger patient [54, 55].

The imaging characteristics of anterior dislocation may include the Hill-Sachs lesion (posterolateral osteochondral compression fracture of the humeral head), Bankart lesion (soft tissue avulsion of the labral complex from the scapular periosteum), and Bankart fracture (anteroinferior bony glenoid fractures) [10]. Patients with larger Hill-Sachs lesions are more prone to recurrent dislocations [56–58]. Hill-Sachs defects are estimated to occur in 18% to 90% of anterior shoulder dislocations [59–65]. The incidence has been reported to be higher at 96% in patients with rotator cuff full-thickness tears [66] or recurrent dislocations [60]. Apart from age and number of previous dislocations, mechanism of dislocation could affect the severity of this pathology. The most significant mechanisms of injury are reported to be a fall greater than one flight of stairs, a fight/assault episode, and a motor vehicle crash [67].

Chronic anterior dislocation is a rare entity, potentially because of severity of the presenting deformity that cannot be missed clinically. Generally, any dislocation that has gone unreduced for at least 3 weeks is considered a fixed or chronic dislocation. There is a limited number of publications on chronic anterior dislocation

[68–71]. Elderly alcoholics who may not receive appropriate and immediate care or people with a history of seizure are at risk of having a missed anterior dislocation [72, 73]. Most cases of missed anterior dislocation cannot be successfully treated with closed reduction and require either open reduction and capsulolabral complex repair or joint replacement [69, 70]. Reduction of chronic unreduced shoulder dislocation using arthroscopy has not been widely recommended [71]. The severe soft tissue contracture, muscle imbalance, and bone deficiency make the surgical treatment challenging. Anatomical arthroplasty may also be associated with a high failure rate in these cases secondary to altered anatomy. Reverse arthroplasty is reported to have an acceptable success rate [74]. Currently, the suggested surgical treatment for neglected anterior shoulder dislocation is open reduction combined with Latarjet procedure [75].

Posterior Glenohumeral Instability

Earlier articles have noted the incidence of posterior dislocation at approximately 5% of all dislocations [10]. More recent literature reports a similar incidence of 4.64 per 100,000 person-years in the general population [76]. Posterior dislocation is more prevalent in men between ages of 20 and 49 and in the elderly patients over 70 years old [77]. Seizures have been noted as the cause of dislocation in 34% of cases [78]. This rate increases significantly for bilateral posterior shoulder dislocations. Almost all bilateral post-dislocations are reported to be due to a seizure or electrocution injury due to unbalanced muscle contraction with the internal rotators of the shoulder contracting with greater force than the external rotators, causing posterior dislocation due to superior and posterior movement of the humeral head [79].

Locked or undetected posterior dislocation is a rare but a disabling condition with a poor prognosis. McLaughlin referred to the locked posterior dislocation as a “diagnostic trap” for its uncommon occurrence that can be a problem for an unexperienced surgeon. There are a number of reasons for misdiagnosis of posterior dislocation. First, the position of the locked joint in adduction and internal rotation appears like a protective position patients may adopt after an injury. In posterior dislocation, patients can still elevate the arm up to the head level, which somehow contradicts the clinical impression of a dislocation. The chronic locked posterior dislocation is associated with significant loss of abduction and complete lack of external rotation which mimics a frozen shoulder, an important differential diagnosis that McLaughlin referred to in 1963 [80, 81]. He explained the importance of the history and mechanism of injury that is very specific to this type of dislocation (convulsive disorder, electric shock, injury to adducted internally rotated arm). In association with a typical mechanism of injury, a sudden loss of abduction and inability to externally rotate after injury are important red flags for possibility of a locked posterior dislocation. Since, misinterpretation of the AP view radiograph is quite common in the emergency setting, including an axillary view is a “must” for anyone with an injury that may cause posterior dislocation of the humeral head.

Inferior Glenohumeral Instability

Inferior dislocation or luxatio erecta is the least common type of dislocation of the glenohumeral joint, accounting for only 0.5% of all shoulder dislocations. The mechanism of injury is through direct axial loading forces on the fully abducted shoulder which may occur during a fall, motor vehicle accidents, or swimming [82, 83]. The presentation of this dislocation is very classic with the arm being in hyperabduction, elbow in flexion, and arm locked above the head. The upper arm appears shorter than the opposite side. Luxatio erecta is often associated with neurovascular injuries (neuropraxia of brachial plexus, radial, and ulnar nerve) and rotator cuff injury [82, 83]. The inferior dislocation is often managed in the ER due to its severity.

Multidirectional Hypermobility (Laxity)

The spectrum of the glenohumeral joint instability extends from hyperlaxity to complete dislocation. Two forms of instability have been described by Thomas and Matsen in 1989, AMBRI and TUBS [84]. AMBRI stands for atraumatic, multidirectional, bilateral, rehabilitation, and inferior capsular shift and refers to patients with atraumatic onset, multidirectional and often bilateral instability who often respond to a rehabilitation program but may need procedures such as capsular shift if they failed conservative treatment [84]. It is generally agreed upon that hyperlaxity is an asymptomatic excessive translation of glenohumeral joint. Instability is a more proper term when hyperlaxity is associated with symptoms. Hyperlaxity may be congenital or acquired. The congenital type is most often multidirectional. In a review by Corner and Emery [85], role of other static stabilizers of the shoulder, such as glenoid retroversion [16, 17], abnormal muscle recruitment, cortical spinal control [86], and scapular positioning [87, 88], has been highlighted. Excessive retroversion of the glenoid has been associated with presence of multidirectional instability and specifically posterior instability. Brewer and colleagues [89] reported that excessive glenoid retroversion caused by developmental deformity was a major contributor to nontraumatic posterior instability of the shoulder. Two other independent studies that have used computed tomography to measure glenoid version found that the glenoid version was statistically significantly different in posteriorly unstable shoulders as compared with stable shoulders [16, 17].

Rare Hereditary Disorders

One of the conditions that is associated with extensive joint hypermobility is the Ehlers-Danlos syndrome (EDS), a rare inherited connective tissue disorder that is associated with skin hyperextensibility, generalized joint hypermobility, and systematic organ involvement. The initial diagnosis of the classic hypermobile EDS is based on family history and clinical examination. While more than 13 different types of the EDS have been identified, the classic EDS is associated with a skin that can be stretched up to 3 cm in certain parts of body (e.g., neck and elbow). Extensive atrophic scars at pressure points are common, and patients often have history of recurrent subluxations or frank joint instability. Diagnosis of specific subtype level of other EDS disorders requires genetic testing [90].

Clinical Findings of Glenohumeral Joint Instability

A detailed history with emphasis on mechanism of injury and a proper clinical examination would identify the type, direction, and etiology of the instability [91]. A number of tests have been described for instability. The anterior and posterior drawer tests were described initially by Gerber and Ganz in 1984 [92]. The anterior and posterior drawers must be conducted with the patient supine, as the reproducibility is fundamentally reliant upon relaxation of the musculature. The load and shift test attempts to determine the amount of translation of the head of the humerus on the glenoid. This test was first described by Hawkins and colleagues, in 1988 [93].

The validity of the laxity tests has not been well established, and biomechanical testing or cadaveric studies have demonstrated large variations in shoulder laxity both anteriorly and posteriorly. In 1999, Levy et al. [94] reported poor inter- and intra-examiner reliability of the laxity tests. Therefore, diagnosis of shoulder instability should not solely be based on translation tests. Sulcus sign is a basic clinical sign of multidirectional or inferior instability. This sign was first described by Neer and Foster [95] as a hallmark of multidirectional instability. Neer and Foster suggested that a positive sulcus indicates inferior capsular redundancy.

An advanced practice physiotherapist or primary care clinician should pay extra attention to older patients who have severe pain and inability to elevate or abduct the arm post-dislocation/reduction. Both cuff tear and axillary nerve injury are common in those aged over 60 years and should be ruled out if the patient cannot abduct the arm at 1-week post-injury [96]. In patients with axillary nerve injuries from dislocation, a significant percentage (41%) are reported to have concurrent rotator cuff tears [97]. As a result of overlapping pathologies, it is imperative that the cause of impaired range of movement be properly investigated as the finding of one injury may detract from investigation of the other. Generally, the deltoid paralysis following an axillary nerve injury will often have an insignificant impact on abduction of the arm, and in acute dislocations, inability to abduct may be caused by a significant rotator cuff tear or associated suprascapular nerve injury [98, 99]. The investigation of patients who manifest muscle weakness or altered sensation after shoulder dislocation should be more comprehensive with a more systematic approach [100]. In cases with a potential nerve injury, electromyogram (EMG) or nerve conduction velocity (NCV) should be ordered. The EMG looks for abnormal findings of electrical signals within the muscles where NCV examines the speed of electrical signals within the nerves. Since fibrillation potentials that indicate denervation take time, the first EMG examination should be performed with a delay of at least 3 weeks [99, 100].

Management

Prior to proceeding with surgery, dynamic factors should be addressed in patients with MDI by a comprehensive rehabilitation program. With exercises that improve proprioception, dynamic stability, neuromuscular control, and scapular muscle strengthening, symptoms can be more manageable. However, despite the major role of rehabilitation in the treatment of multidirectional instability, the symptoms may often reoccur or remain partially resolved in this population [101]. It is noteworthy that a number of patients with MDI and voluntary dislocations may need emotional or psychological support [102, 103]. Role of rehabilitation as the sole conservative treatment in traumatic instability with associated structural abnormalities is less significant.

The earliest arthroscopic repair of Bankart lesion in which staples were used was described by Johnson, in 1980 [104]. At present, majority of stabilization procedures are successfully performed arthroscopically [105–107]. Improvement and use of new techniques in arthroscopy have led to less surgical failure and better patient outcomes. To ensure a more successful outcome, however, the assessment of glenoid bone loss and proper patient selection remain critical. For example, arthroscopic repair alone is contraindicated when a large, engaging Hill-Sachs lesion exists or the glenoid bone loss is more than 25% [108, 109].

References

1. 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.
2. Warner JJ, Deng XH, Warren RF, Torzilli PA. Static capsuloligamentous restraints to superior-inferior translation of the glenohumeral joint. Am J Sports Med. 1992;20(6):675–85.
3. Burkart AC, Debski RE. Anatomy and function of the glenohumeral ligaments in anterior shoulder instability. Clin Orthop Relat Res. 2002;400:32–9.
4. O'Brien SJ, Neves MC, Arnoczky SP, Rozbruck SR, Dicarlo EF, Warren RF, et al. The anatomy and histology of the inferior glenohumeral ligament complex of the shoulder. Am J Sports Med. 1990;18(5):449–56.
5. McMahon PJ, Dettling J, Sandusky MD, Tibone JE, Lee TQ. The anterior band of the inferior glenohumeral ligament. Assessment of its permanent deformation and the anatomy of its glenoid attachment. J Bone Joint Surg Br. 1999;81(3):406–13.
6. Taljanovic MS, Nisbet JK, Hunter TB, Cohen RP, Rogers LF. Humeral avulsion of the inferior glenohumeral ligament in college female volleyball players caused by repetitive microtrauma. Am J Sports Med. 2011;39(5):1067–76.
7. O'Brien SJ, Schwartz RS, Warren RF, Torzilli PA. Capsular restraints to anterior-posterior motion of the abducted shoulder: a biomechanical study. J Shoulder Elb Surg. 1995;4(4):298–308.
8. Provencher MT, LeClere LE, King S, McDonald LS, Frank RM, Mologne TS, et al. Posterior instability of the shoulder: diagnosis and management. Am J Sports Med. 2011;39(4):874–86.
9. Moseley HF, Overgaard B. The anterior capsular mechanism in recurrent anterior dislocation of the shoulder. Morphological and clinical studies with special references to the glenoid labrum and the Gleno-humeral ligaments. J Bone Joint Surg Br. 1962;44B(4):913–27.

10. Matsen FAI, Thomas SC, Rockwood CA, editors. The shoulder, glenohumeral instability. 2nd ed. Philadelphia: W. B. Saunders; 1990.
11. Huri G, Kaymakoglu M, Garbis N. Rotator cable and rotator interval: anatomy, biomechanics and clinical importance. EFORT Open Rev. 2019;4(2):56–62.
12. Matsen FA 3rd, Chebli CM, Lippitt SB. Principles for the evaluation and management of shoulder instability. Instr Course Lect. 2007;56:23–34.
13. Lee TQ, Black AD, Tibone JE, McMahon PJ. Release of the coracoacromial ligament can lead to glenohumeral laxity: a biomechanical study. J Shoulder Elb Surg. 2001; 10(1):68–72.
14. Shaffer B, Evans B, Ferrero G. Release and reattachment of the coracoacromial ligament: a cadaveric study. J Shoulder Elb Surg. 1997;6(3):297–305.
15. Chahal J, Leiter J, McKee MD, Whelan DB. Generalized ligamentous laxity as a predisposing factor for primary traumatic anterior shoulder dislocation. J Shoulder Elb Surg. 2010;19(8):1238–42.
16. Inui H, Sugamoto K, Miyamoto T, Yoshikawa H, Machida A, Hashimoto J, et al. Three-dimensional relationship of the glenohumeral joint in the elevated position in shoulders with multidirectional instability. J Shoulder Elb Surg. 2002;11(5):510–5.
17. 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(4):396–400.
18. Jost B, Koch PP, Gerber C. Anatomy and functional aspects of the rotator interval. J Shoulder Elb Surg. 2000;9(4):336–41.
19. Clark JM, Harryman DT 2nd. Tendons, ligaments, and capsule of the rotator cuff. Gross and microscopic anatomy. J Bone Joint Surg Am. 1992;74(5):713–25.
20. Burkhart SS, Esch JC, Jolson RS. The rotator crescent and rotator cable: an anatomic description of the shoulder's "suspension bridge". Arthroscopy. 1993;9(6):611–6.
21. Lippitt S, Matsen F. Mechanisms of glenohumeral joint stability. Clin Orthop Relat Res. 1993;291:20–8.
22. Lee SB, Kim KJ, O'Driscoll SW, Morrey BF, An KN. Dynamic glenohumeral stability provided by the rotator cuff muscles in the mid-range and end-range of motion. A study in cadavera. J Bone Joint Surg Am. 2000;82(6):849–57.
23. Pagnani MJ, Warren RF. Stabilizers of the glenohumeral joint. J Shoulder Elb Surg. 1994;3(3):173–90.
24. Blasier RB, Soslowsky LJ, Malicky DM, Palmer ML. Posterior glenohumeral subluxation: active and passive stabilization in a biomechanical model. J Bone Joint Surg Am. 1997;79(3):433–40.
25. Smee A. Dissection of a partial dislocation of the shoulder upwards. Lancet. 1845;1:323–4.
26. Smith RW. Observations upon chronic rheumatic arthritis of the shoulder [part II]. Dublin Quart J Med Sci. 1853;15:343–58.
27. Smith RW. Observations upon chronic rheumatic arthritis of the shoulder [part I]. Dublin Quart J Med Sci. 1853;15:1–16.
28. Brorson S. Cuff tear arthropathy in the nineteenth century: 'chronic rheumatic arthritis' with 'partial luxation upwards' of the humeral head. Int Orthop. 2019;43(10):2415–23.
29. Adams R. Illustrations of the effects of rheumatic gout or chronic rheumatic arthritis on all the articulations. With descriptive and explanatory statements. London: John Churchill and Sons; 1857. p. 1–31.
30. Warner JJ, McMahon PJ. The role of the long head of the biceps brachii in superior stability of the glenohumeral joint. J Bone Joint Surg Am. 1995;77(3):366–72.
31. Kumar VP, Satku K, Balasubramaniam P. The role of the long head of biceps brachii in the stabilization of the head of the humerus. Clin Orthop Relat Res. 1989;244:172–5.
32. Kido T, Itoi E, Konno N, Sano A, Urayama M, Sato K. The depressor function of biceps on the head of the humerus in shoulders with tears of the rotator cuff. J Bone Joint Surg Br. 2000;82(3):416–9.

33. Rauck RC, Jahandar A, Kontaxis A, Dines DM, Warren RF, Taylor SA, et al. The role of the long head of the biceps tendon in posterior shoulder stabilization during forward flexion. J Shoulder Elb Surg. 2022;
34. Breasted JH. The Edwin Smith surgical papyrus, published in facsimile and hieroglyphic transliteration with translation and commentary in two volumes (PDF). The University of Chicago, Oriental Institute Publications 1930;III (https://oi-uchicago-edu.myaccess.library.utoronto.ca/sites/oi.uchicago.edu/files/uploads/shared/docs/oip3.pdf).
35. Mattick A, Wyatt JP. From Hippocrates to the Eskimo—a history of techniques used to reduce anterior dislocation of the shoulder. J R Coll Surg Edinb. 2000;45(5):312–6.
36. Broca A, Hartmann H. Contribution à l'étude des luxations de l'épaule (luxationsanciennesetluxationsrécidivantes). Bull Soc Anat. 1890;4:416–23.
37. Bankart AS, Cantab MC. Recurrent or habitual dislocation of the shoulder-joint. 1923. Clin Orthop Relat Res. 1993;291:3–6.
38. Bankart A. The pathology and treatment of recurrent dislocation of the shoulder joint. Br J Surg. 1938;26:23–9.
39. Loebenberg MI, Cuomo F. The treatment of chronic anterior and posterior dislocations of the glenohumeral joint and associated articular surface defects. Orthop Clin North Am. 2000;31(1):23–34.
40. Cooper A. On the dislocations of the os humeri upon the dorsum scapulae, and upon fractures near the shoulder joint. Guys Hosp Rep. 1839;4:265–84.
41. McLaughlin HL. Posterior dislocation of the shoulder. J Bone Joint Surg Am. 1952;24 A(3):584–90.
42. Navlet MG, Asenjo-Gismero CV. Multidirectional instability: natural history and evaluation. Open Orthop J. 2017;11:861–74.
43. Lawrence A. Benign hypermobility syndrome. J Indian Rheumatol Assoc. 2005;13:150–5.
44. Neer CS 2nd, Foster CR. Inferior capsular shift for involuntary inferior and multidirectional instability of the shoulder. A preliminary report. J Bone Joint Surg Am. 1980;62(6):897–908.
45. Kibler WB, Sciascia A. The role of the scapula in preventing and treating shoulder instability. Knee Surg Sports Traumatol Arthrosc. 2016;24(2):390–7.
46. Zacchilli MA, Owens BD. Epidemiology of shoulder dislocations presenting to emergency departments in the United States. J Bone Joint Surg Am. 2010;92(3):542–9.
47. Robinson CM, Howes J, Murdoch H, Will E, Graham C. Functional outcome and risk of recurrent instability after primary traumatic anterior shoulder dislocation in young patients. J Bone Joint Surg Am. 2006;88(11):2326–36.
48. Flinkkila T, Hyvonen P, Ohtonen P, Leppilahti J. Arthroscopic Bankart repair: results and risk factors of recurrence of instability. Knee Surg Sports Traumatol Arthrosc. 2010;18(12):1752–8.
49. Berbig R, Weishaupt D, Prim J, Shahin O. Primary anterior shoulder dislocation and rotator cuff tears. J Shoulder Elb Surg. 1999;8(3):220–5.
50. Neviaser RJ, Neviaser TJ, Neviaser JS. Concurrent rupture of the rotator cuff and anterior dislocation of the shoulder in the older patient. J Bone Joint Surg Am. 1988;70(9):1308–11.
51. McGregor AL, du Plessis DJ. A synopsis of surgical anatomy. 10th ed. Bristol: John Wright & Sons Ltd.; 1969.
52. Stevens JH. Brachial plexus paralysis. By J. H. Stevens, M,D., 1934. Clin Orthop Relat Res. 1988;237:4–8.
53. Murata K, Maeda M, Yoshida A, Yajima H, Okuchi K. Axillary artery injury combined with delayed brachial plexus palsy due to compressive hematoma in a young patient: a case report. J Brachial Plex Peripher Nerve Inj. 2008;3:9-7221-3-9.
54. Robinson CM, Shur N, Sharpe T, Ray A, Murray IR. Injuries associated with traumatic anterior glenohumeral dislocations. J Bone Joint Surg Am. 2012;94(1):18–26.
55. Gutkowska O, Martynkiewicz J, Stepniewski M, Gosk J. Analysis of patient-dependent and trauma-dependent risk factors for persistent brachial plexus injury after shoulder dislocation. Biomed Res Int. 2018;2018:4512137.

56. Kaar SG, Fening SD, Jones MH, Colbrunn RW, Miniaci A. Effect of humeral head defect size on glenohumeral stability: a cadaveric study of simulated Hill-Sachs defects. Am J Sports Med. 2010;38(3):594–9.
57. Cho SH, Cho NS, Rhee YG. Preoperative analysis of the Hill-Sachs lesion in anterior shoulder instability: how to predict engagement of the lesion. Am J Sports Med. 2011;39(11):2389–95.
58. Burkhart SS, De Beer JF. 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.
59. Edwards TB, Boulahia A, Walch G. Radiographic analysis of bone defects in chronic anterior shoulder instability. Arthroscopy. 2003;19(7):732–9.
60. Spatschil A, Landsiedl F, Anderl W, Imhoff A, Seiler H, Vassilev I, et al. Posttraumatic anterior-inferior instability of the shoulder: arthroscopic findings and clinical correlations. Arch Orthop Trauma Surg. 2006;126(4):217–22.
61. Perron AD, Ingerski MS, Brady WJ, Erling BF, Ullman EA. Acute complications associated with shoulder dislocation at an academic emergency department. J Emerg Med. 2003;24(2):141–5.
62. Suder PA, Frich LH, Hougaard K, Lundorf E, Wulff JB. Magnetic resonance imaging evaluation of capsulolabral tears after traumatic primary anterior shoulder dislocation. A prospective comparison with arthroscopy of 25 cases. J Shoulder Elb Surg. 1995;4(6):419–28.
63. 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(2):159–68.
64. Yiannakopoulos CK, Mataragas E, Antonogiannakis E. A comparison of the spectrum of intra-articular lesions in acute and chronic anterior shoulder instability. Arthroscopy. 2007;23(9):985–90.
65. Antonio GE, Griffith JF, Yu AB, Yung PS, Chan KM, Ahuja AT. First-time shoulder dislocation: high prevalence of labral injury and age-related differences revealed by MR arthrography. J Magn Reson Imaging. 2007;26(4):983–91.
66. Jouve F, Graveleau N, Nove-Josserand L, Walch G. Recurrent anterior instability of the shoulder associated with full thickness rotator cuff tear: results of surgical treatment. Rev Chir Orthop Reparatrice Appar Mot. 2008;94(7):659–69.
67. Emond M, Le Sage N, Lavoie A, Rochette L. Clinical factors predicting fractures associated with an anterior shoulder dislocation. Acad Emerg Med. 2004;11(8):853–8.
68. Rouhani A, Navali A. Treatment of chronic anterior shoulder dislocation by open reduction and simultaneous Bankart lesion repair. Sports Med Arthrosc Rehabil Ther Technol. 2010;2:15.
69. Mansat P, Guity MR, Mansat M, Bellumore Y, Rongieres M, Bonnevialle P. Chronic anterior shoulder dislocation treated by open reduction sparing the humeral head. Rev Chir Orthop Reparatrice Appar Mot. 2003;89(1):19–26.
70. Rowe CR, Zarins B. Chronic unreduced dislocations of the shoulder. J Bone Joint Surg Am. 1982;64(4):494–505.
71. Yanmis I, Komurcu M, Oguz E, Basbozkurt M, Gur E. The role of arthroscopy in chronic anterior shoulder dislocation: technique and early results. Arthroscopy. 2003;19(10):1129–32.
72. Datt R, Jain G, Nag HL. Management of neglected locked anterior dislocation of shoulder: a report of two cases. Cureus. 2021;13(3):e13843.
73. Sanel S, Sencan S, Ocguder A, Solakoglu C. Bilateral, locked, recurrent anterior shoulder dislocation: case report. Eklem Hastalik Cerrahisi. 2015;26(1):52–5.
74. Cerciello S, Corona K, Morris BJ, Paladini P, Porcellini G, Merolla G. Shoulder arthroplasty to address the sequelae of anterior instability arthropathy and stabilization procedures: systematic review and meta-analysis. Arch Orthop Trauma Surg. 2020;140(12):1891–900.
75. Lubis AMT, Primaputra MRA, Dilogo IH. A case report of ten-month-neglected anterior shoulder dislocation managed by open reduction combined with Latarjet procedure. Int J Surg Case Rep. 2019;57:93–6.
76. Woodmass JM, Lee J, Wu IT, Desai VS, Camp CL, Dahm DL, et al. Incidence of posterior shoulder instability and trends in surgical reconstruction: a 22-year population-based study. J Shoulder Elb Surg. 2019;28(4):611–6.

77. 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.
78. Rouleau DM, Hebert-Davies J. Incidence of associated injury in posterior shoulder dislocation: systematic review of the literature. J Orthop Trauma. 2011;15
79. Buhler M, Gerber C. Shoulder instability related to epileptic seizures. J Shoulder Elb Surg. 2002;11(4):339–44.
80. Hill NA, McLaughlin HL. Locked posterior dislocation simulating a 'frozen shoulder'. J Trauma. 1963;3:225–34.
81. McLaughlin HL. Locked posterior subluxation of the shoulder: diagnosis and treatment. Surg Clin North Am. 1963;43:1621–2.
82. Imerci A, Golcuk Y, Ugur SG, Ursavas HT, Savran A, Surer L. Inferior glenohumeral dislocation (luxatio erecta humeri): report of six cases and review of the literature. Ulus Travma Acil Cerrahi Derg. 2013;19(1):41–4.
83. Gokkus K, Sagtas E, Saylik M, Aydin AT, Atmaca H. Luxatio erecta humeri: report of a swimming injury with analysis of the mechanism of the injury and associated injuries in literature. J Emerg Trauma Shock. 2015;8(1):43–8.
84. Thomas SC, Matsen FA 3rd. An approach to the repair of avulsion of the glenohumeral ligaments in the management of traumatic anterior glenohumeral instability. J Bone Joint Surg Am. 1989;71(4):506–13.
85. Corner T, Emery RJH. Multidirectional instability of the shoulder: does it exist? Shoulder Elbow. 2010;2:71–6.
86. Alexander CM. Altered control of the trapezius muscle in subjects with non-traumatic shoulder instability. Clin Neurophysiol. 2007;118(12):2664–71.
87. Struyf F, Nijs J, Baeyens JP, Mottram S, Meeusen R. Scapular positioning and movement in unimpaired shoulders, shoulder impingement syndrome, and glenohumeral instability. Scand J Med Sci Sports. 2011;21(3):352–8.
88. von Eisenhart-Rothe R, Matsen FA 3rd, Eckstein F, Vogl T, Graichen H. Pathomechanics in atraumatic shoulder instability: scapular positioning correlates with humeral head centering. Clin Orthop Relat Res. 2005;433:82–9.
89. Brewer BJ, Wubben RC, Carrera GF. Excessive retroversion of the glenoid cavity. A cause of non-traumatic posterior instability of the shoulder. J Bone Joint Surg Am. 1986;68(5):724–31.
90. Caliogna L, Guerrieri V, Annunziata S, Bina V, Brancato AM, Castelli A, et al. Biomarkers for Ehlers-Danlos syndromes: there is a role? Int J Mol Sci. 2021;22:18. https://doi.org/10.3390/ijms221810149.
91. Silliman JF, Hawkins RJ. Classification and physical diagnosis of instability of the shoulder. Clin Orthop Relat Res. 1993;291:7–19.
92. Gerber C, Ganz R. Clinical assessment of instability of the shoulder. With special reference to anterior and posterior drawer tests. J Bone Joint Surg Br. 1984;66(4):551–6.
93. Hawkins RJ, Schutte JP, Janda DH, Huckell GH. Translation of the glenohumeral joint with the patient under anesthesia. J Shoulder Elb Surg. 1996;5(4):286–92.
94. Levy AS, Lintner S, Kenter K, Speer KP. Intra- and interobserver reproducibility of the shoulder laxity examination. Am J Sports Med. 1999;27(4):460–3.
95. Neer CS 2nd. Anterior acromioplasty for the chronic impingement syndrome in the shoulder: a preliminary report. J Bone Joint Surg Am. 1972;54(1):41–50.
96. Visser CP, Coene LN, Brand R, Tavy DL. The incidence of nerve injury in anterior dislocation of the shoulder and its influence on functional recovery. A prospective clinical and EMG study. J Bone Joint Surg Br. 1999;81(4):679–85.
97. Perlmutter GS. Axillary nerve injury. Clin Orthop Relat Res. 1999;368:28–36.
98. Hems TE, Mahmood F. Injuries of the terminal branches of the infraclavicular brachial plexus: patterns of injury, management and outcome. J Bone Joint Surg Br. 2012;94(6):799–804.
99. Zoltan JD. Injury to the suprascapular nerve associated with anterior dislocation of the shoulder: case report and review of the literature. J Trauma. 1979;19(3):203–6.

100. Gutkowska O, Martynkiewicz J, Urban M, Gosk J. Brachial plexus injury after shoulder dislocation: a literature review. Neurosurg Rev. 2020;43(2):407–23.
101. Kiss J, Damrel D, Mackie A, Neumann L, Wallace WA. Non-operative treatment of multidirectional shoulder instability. Int Orthop. 2001;24(6):354–7.
102. Johnson SM, Robinson CM. Shoulder instability in patients with joint hyperlaxity. J Bone Joint Surg Am. 2010;92(6):1545–57.
103. Rowe CR, Pierce DS, Clark 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(3):445–60.
104. Johnson LL. Arthroscopy of the shoulder. Orthop Clin North Am. 1980;11(2):197–204.
105. Tjoumakaris FP, Bradley JP. The rationale for an arthroscopic approach to shoulder stabilization. Arthroscopy. 2011;27(10):1422–33.
106. Fabbriciani C, Milano G, Demontis A, Fadda S, Ziranu F, Mulas PD. Arthroscopic versus open treatment of Bankart lesion of the shoulder: a prospective randomized study. Arthroscopy. 2004;20(5):456–62.
107. Bottoni CR, Smith EL, Berkowitz MJ, Towle RB, Moore JH. Arthroscopic versus open shoulder stabilization for recurrent anterior instability: a prospective randomized clinical trial. Am J Sports Med. 2006;34(11):1730–7.
108. Di Giacomo G, De Vita A, Costantini A, de Gasperis N, Scarso P. Management of humeral head deficiencies and glenoid track. Curr Rev Musculoskelet Med. 2014;7(1):6–11.
109. DeFroda S, Bokshan S, Stern E, Sullivan K, Owens BD. Arthroscopic Bankart repair for the Management of Anterior Shoulder Instability: indications and outcomes. Curr Rev Musculoskelet Med. 2017;10(4):442–51.

Chapter 10
Principles of Radiological Examination

In this chapter, we review the historical perspective and principles of radiological assessment by providing basic information on conventional plain radiographs and other modes of imaging within the scope of advanced practice physiotherapists, extended role healthcare providers, and primary physicians. The indications for different views of plain radiographs are specifically emphasized as primary clinicians are expected to understand the contribution of each view to management of patients in a non-trauma setting. Important radiographic terminology used in relation to soft tissue pathology is reviewed.

In 1895, Wilhelm Conrad Roentgen, a son of a German merchant from the Rhine province, changed the world of medicine when he discovered a specific radiation he referred to as the "X," to indicate that it was an unknown type of radiation. In fact, the science of radiology was born when the shadows of bones and a ring, his wife was wearing, showed up in a picture he took of her hand in that year. Roentgen was a private man with admirable qualities. Just 1 year before his extraordinary discovery, in his inaugural address quoted in "Wilhelm Conrad Roentgen," Dictionary of Scientific Biography (New York: Scribner's, 1975), p. 531, he cautioned that "pride in one's profession is demanded, but not professional conceit, snobbery, or academic arrogance, all of which grow from false egoism." Roentgen was awarded the first Nobel Prize in physics for his extraordinary discovery in 1901. He donated its entire cash prize to his university to promote scientific studies. Despite many prizes, medals, and honorary doctorates he received after his discovery, he retained the characteristic of a modest man, and all published literature on his personality and private life is in agreement that he was indeed one of the noblest and greatest men of his generation [1–4].

H. Razmjou, M. Christakis, *Clinical and Radiological Examination of the Shoulder Joint*, https://doi.org/10.1007/978-3-031-10470-1_10

Conventional Radiography

The conventional or plain radiograph often referred to as X-rays may not appear as comprehensive or specific as other modes of imaging, but it remains the first-line of imaging modality that is used in patients with shoulder pain and provides the foundation of diagnosis for many clinical conditions. Plain radiographs help to identify the cause of stiffness in patients who have overlapping clinical features of adhesive capsulitis vs. advanced osteoarthritis of the glenohumeral joint. Similarly, assessment of subacromial spurs which contribute to subacromial impingement can be well achieved by simple plain radiographs. Foci of periarticular calcification may suggest calcific tendinitis as a case of shoulder pain. In rotator cuff pathology, simple views play a critical role in assessing the severity of a tear by showing the acromiohumeral distance (AHD) which is commonly reduced in major rotator cuff tears. In many cases, proper views of the plain radiographs will guide the primary care clinicians in perusing the proper management, such as ordering more costly investigations or referral for surgical consultation.

Standard Anteroposterior (AP) View

The standard AP view of the shoulder joint provides a basic visualization of the glenohumeral joint, AC joint, distal clavicle, and acromion process and may be taken in neutral, internal, or external rotation of the arm. The standard AP projection results in overlap of the glenoid and humeral head because of the anterior anatomical tilt of the glenohumeral joint. However, despite this shortcoming, this view can help with evaluation of fracture or anterior dislocations in acute trauma settings [5, 6]. Figure 10.1 shows normal anatomy of the left shoulder in the standard AP view.

In many institutes and especially trauma settings, the AP neutral and/or AP internal rotation and AP external rotation views are taken as a part of standard practice. One commonly seen osseous pathology, associated with anterior glenohumeral dislocation, is the impression fracture in the humeral head, referred to as the Hill-Sachs lesion. In 1940, Harold Arthur Hill and Maurice David Sachs, two American radiologists who worked together, described a defect located on the posterolateral aspect of the articulating surface of the humeral head, based on their review of 119 shoulder dislocation cases [7]. They determined that the defect was related to impaction of the humeral head against the inferior glenoid at the time of dislocation. Before their description, this bony defect was referred to as a “typical defect” of anterior dislocation by other clinicians.

In AP internal rotation view, the lesser tubercle is visualized in full profile, which is helpful in detecting suspected posterolateral impaction fractures (Hill-Sachs lesions) or lesser tuberosity avulsions. In AP external rotation view, the greater tubercle is in full profile. This view is a preferable view for detecting calcified tendinitis of the supraspinatus and infraspinatus tendons. In a reliability study by Charousset et al. [8], the AP internal rotation view, which was obtained by simply

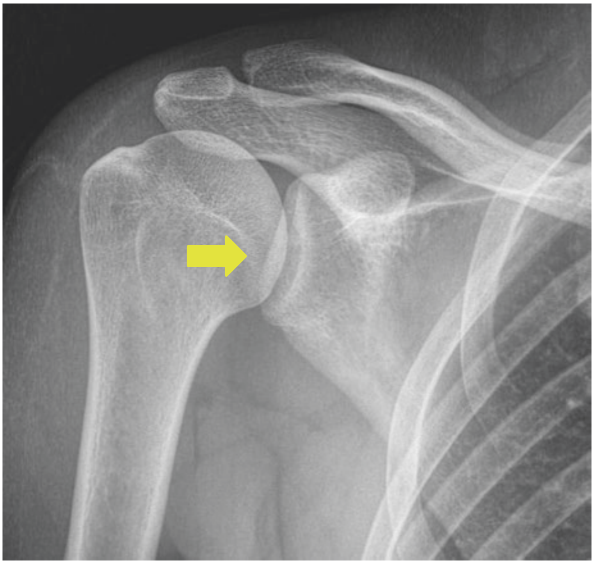

Fig. 10.1 Normal anatomy of the shoulder in standard AP view where there is an overlap of the humeral head and glenoid fossa

placing one's hand on the stomach, showed a better intra- and inter-tester agreement on detection of osseous lesions of anterior instability such as Hill-Sachs lesion. The agreement on presence of pathology on the AP external rotation view was less optimal because of variability of maximal rotation depending on the degrees of hyperlaxity, with the notch becoming totally masked by the contour of the anterior and superior humeral head in extreme external rotation [8]. According to an earlier study by Balg and Boileau in 2007 [9], the visibility of a Hill-Sachs lesion on the AP external rotation view was a sign of a more serious defect. In other words, if the defect remained visible on the AP image with maximum external rotation, there was a higher chance of recurrent instability after an arthroscopic Bankart procedure.

True Anteroposterior (Grashey) View

The true AP or Grashey view is probably the most useful view in the non-acute, non-trauma primary or tertiary centers. In this view, to eliminate the overlap of the glenoid rim and the humeral head, the cassette parallels the plane of the scapula [5, 6]. This projection is often used for assessment of the glenohumeral arthritis (joint space narrowing, congruity of the glenohumeral bones, or inferior humeral osteophytes) or biomechanical impact of rotator cuff deficiency on the humeral head (superior migration of the humeral head with respect to glenoid). It has been suggested that the true AP view is significantly more sensitive than the standard AP view in detecting the pathognomonic findings of rotator cuff tear, especially the medium-sized full tears that may go undetected in the standard AP view [10]. Koh et al. felt that the main reason for the difference between views was a better

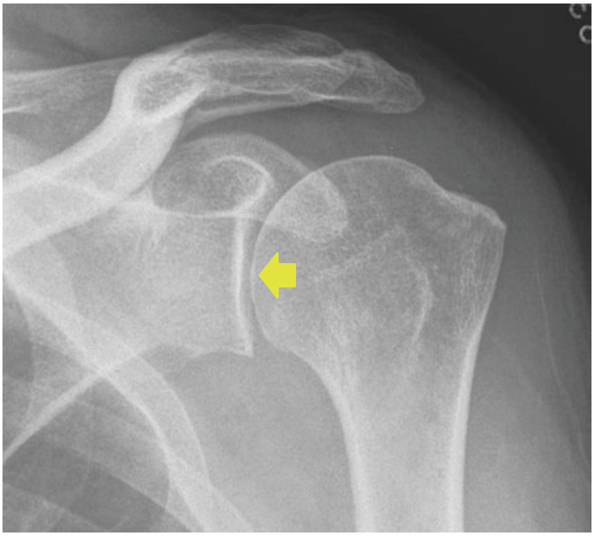

Fig. 10.2 Normal anatomy of the shoulder in true AP (Grashey) view. The glenohumeral joint is seen in profile without overlap of the humerus and glenoid

exposure of the greater tuberosity in the true AP view. The reduced acromiohumeral distance (AHD) of <6 mm measured on the true AP view is another important imaging sign that has been noted to be strongly associated with the presence of major rotator cuff tears [11].

Despite the overall popularity of the true AP (Grashey) view, this view may be suboptimal for instability cases due to higher soft tissue density caused by the obliquity and overlapping of the acromion, acromioclavicular joint, and distal clavicle [12]. Another disadvantage of the Grashey view in instability cases is that subtle abnormalities, such as nondisplaced Bankart fractures, can be missed due to anterior and posterior glenoid rim overlap. Overall, the AP view can be misleading as it often looks near normal in posterior dislocation. Figure 10.2 shows normal anatomy of the shoulder in true AP view.

Supraspinatus Outlet View

The supraspinatus outlet view was first introduced by Neer and Poppen [13]. For this view, patient stands with the arm at the side and the affected shoulder being rotated anteriorly at 45°. The beam is projected in the PA direction with the cassette being perpendicular to the body of the scapula and parallel to the glenoid fossa. The beam is angled 10–15° caudally and centered on the glenohumeral joint [5, 6]. The supraspinatus outlet view helps to evaluate the subacromial space, contour of the undersurface of the acromion process, and the overall

Fig. 10.3 Normal anatomy of the shoulder joint in the supraspinatus outlet view

acromion morphology. Subacromial spurs which often are associated with rotator cuff pathology are best seen in this view. Although this view provides useful information on the subacromial area, patient's faulty posture or suboptimal beam projection could affect the accuracy of the measurement of the subacromial narrowing or the AHD in this view [14]. The supraspinatus outlet and true AP view have shown similar measurement properties in terms of reliability and validity of the AHD measurement which helps with clinical decision-making such as a need for ordering more costly investigations or a referral for surgical consideration [11]. Figure 10.3 shows normal anatomy of the left shoulder in the supraspinatus outlet view.

Scapular Outlet "Y" View

In 1991, Walch and his colleagues [15] proposed a normalized lateral view of the scapula (Y view) carried out under fluoroscopic control with further evaluation of reproducibility in 1998 [16]. The scapular Y view gets its name from the "Y" configuration (Mercedes Benz sign) that is formed between the axis of the body of the scapula, the coracoid process, and the acromion process. The center of the glenoid is located at the intersection of the Y. This view is obtained with the patient upright or prone with the anterior aspect of the affected side rotated 30° to 45° toward the cassette with no caudal angulation as opposed to the supraspinatus outlet view which has a 15° of caudal tube angulation [5, 6]. The scapular outlet Y view is helpful to evaluate for anterior or posterior dislocation in acute settings. However, it is not sensitive for detecting subtle subluxation.

Fig. 10.4 Normal anatomy of the shoulder joint in the scapular outlet Y view

Most often, fractures of the glenoid rim are difficult to examine with this view [12]. This view is also useful for fractures of the coracoid process, scapula, acromion process, and proximal humeral shaft [5, 6]. Figure 10.4 shows normal anatomy of the left shoulder in the scapular Y view.

Acromioclavicular (Zanca) View

The specific AC joint view is referred to as Zanca view in the honor of Peter Zanca, an American radiologist. In 1971, Zanca reviewed 1000 plain radiographs of individuals with shoulder pain and noted that a standard AP view failed to show the AC joint accurately due to superimposition of the acromion process and suggested an AP radiograph with 10 to 15° of cephalic tilt which better facilitates the evaluation of inferiorly protruding osteophytes [17]. Sclerosis of the greater tuberosity, enthesopathic changes of the anterior edge of the acromion, subchondral cysts, separation, calcific tendinitis, and widening of the AC joint commonly seen in lateral clavicle osteolysis are easily identified in this view. Figure 10.5 shows normal anatomy of the left shoulder in the Zanca view.

Fig. 10.5 Normal anatomy of the shoulder joint in the acromioclavicular (Zanca) view

Axillary Lateral View

The axillary view is taken with the arm in abduction preferably to 90°. The cassette is placed on the superior aspect of the shoulder. The beam is centered over the mid-glenohumeral joint and is directed in a distal-to-proximal direction while tilted approximately 15°–30° toward the spine.

The axillary view provides the best image for glenohumeral joint space and is very important in patients with osteoarthritis of the shoulder and in suspected dislocations. In typical osteoarthritis, the joint space narrowing and posterior subluxation caused by posterior erosion and retroversion of the glenoid are evident in this view. Os acromiale is a developmental abnormality caused by an unfused accessory center of ossification of the acromion. This abnormality is often associated with impingement and rotator cuff pathology and is easily detected on an axillary view but may go undetected in other views [5, 6, 18].

In acute trauma setting, the axillary view allows for assessment of fractures of the humeral head, glenoid, and lesser tuberosity of the humerus [5, 6, 18]. This view provides an excellent method for assessment of anterior and especially posterior glenohumeral subluxation or dislocation including osseous Bankart or reverse Bankart fractures involving the anterior or posterior glenoid rim, respectively. Axillary view can be used to provide an estimate of the size of the humeral head defects when CT scan is not available.

In patients with mild traumatic injuries, obtaining 70–90° of abduction is usually feasible; however, for patients with glenohumeral joint dislocation, obtaining the axial image is extremely difficult due to pain and joint incongruity. In cases where obtaining an axillary view is difficult, modified views may be taken with the patient's

Fig. 10.6 Normal anatomy of the shoulder joint in the axillary lateral view

arm held passively in at least 20° of abduction. In acute post-op or post-reduction cases where the patient is wearing a sling, the Velpeau view is used [19]. For this view, the patient leans backward about 30° toward the table. Figure 10.6 shows normal anatomy of the left shoulder in the normal axillary view.

As noted in Chap. 9, in posterior glenohumeral dislocation, the arm is locked in internal rotation which severely limits external rotation of the shoulder. The "acute" mechanical block to external rotation is a classic sign of posterior dislocation. The other two conditions that affect external rotation are severe osteoarthritis and advanced adhesive capsulitis, both being of chronic nature. The missed locked posterior dislocation may have a longer timeframe depending on the onset of the dislocation. Therefore, any patient who reports a history of an acute or subacute injury with immediate loss of external rotation should be investigated by an axillary view.

Stryker Notch View

The Stryker notch view is another important view taken in suspected glenohumeral dislocations. This view was first described by three American surgeons, Hall, Isaac, and Booth in 1959 [20]. They suggested that conventional AP views could miss small posterolateral lesions that would potentially be visible on the AP internal rotation view. To obtain a non-distorted view of the posterolateral notch, they suggested the Stryker view, in which the patient lies in the supine position with the arm externally rotated and abducted and the X-ray beam angled cephalad about 10° and centered on the coracoid process. The patient's hand is placed on the back of the head with the elbow pointed toward the ceiling. The Hill-Sachs impaction fracture are clearly visible on this view [6]. Figure 10.7 shows normal anatomy of the left shoulder on the Stryker notch view.

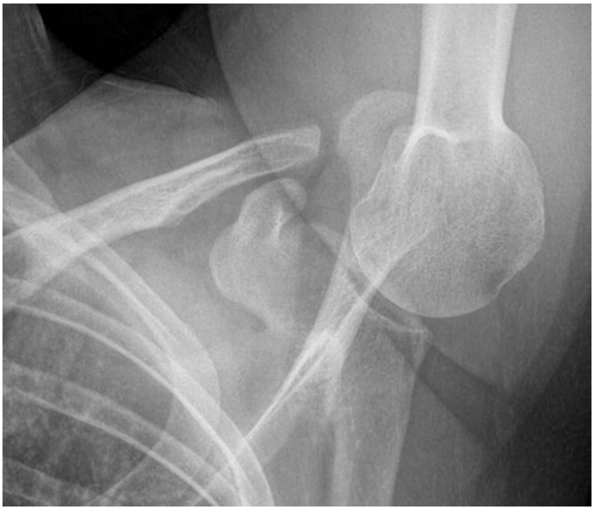

Fig. 10.7 Normal anatomy of the shoulder joint in the Stryker notch view

Fig. 10.8 Normal anatomy of the shoulder joint in the west point view

West Point View

The west point view is a variation of the lateral axillary view that was developed to improve detection of a Bankart fracture of the anterior glenoid rim. This view is obtained by placing the patient in the prone position with the arm abducted to 90° from the long axis of the body with the elbow and forearm hanging off the side of the table. The beam is directed 15° to 25° in an inferior-to-superior direction and tilted 25° toward the spine. Although this projection improves detection of an osseous Bankart lesion, it can be difficult if not impossible to obtain in the acute setting [21]. Figure 10.8 shows normal anatomy of the left shoulder in the west point view.

Other Diagnostic Imaging Modalities of the Shoulder Joint

Apart from plain radiography, there are other modes of imaging such as ultrasonography (US), magnetic resonance imaging (MRI), and computed tomography (CT) scan that are used to diagnose shoulder pathologies. Each imaging has certain indications, strengths, and limitations, and their diagnostic value varies depending on the type of pathology they assess. Before reviewing each diagnostic test, a short summary of commonly used radiological terms is provided. Two common terminologies used in radiological reports are muscle atrophy and fatty infiltration or degeneration.

Muscle atrophy refers to reduced muscle bulk and may occur following muscle disuse, tendon tear, or denervation. Using MRI, muscle atrophy of the supraspinatus muscle is examined in a number of ways: (1) through the presence of a radiological sign called tangent sign and (2) through assessing the ratio between the supraspinatus muscle bulk and the area of the supraspinatus fossa. Tangent sign is examined by drawing a line from the superior aspect of the scapula to the superior portion of the scapular spine. A normal supraspinatus muscle lies above this line [22]. The cross-sectional area of the supraspinatus muscle is measured as an occupation ratio of area of the supraspinatus muscle divided by the overall area under the tangent in the supraspinatus fossa [23]. Muscle atrophy of neurological nature may occur without rotator cuff tear or retraction. The more common neurological muscle atrophies are related to suprascapular and axillary nerve compression (see Chap. 1). The suprascapular nerve compression often occurs in the suprascapular notch under the transverse scapular ligament secondary to cysts, lipoma, or humeral/scapular fractures and may be associated with atrophy of the supraspinatus and infraspinatus muscles [24, 25] with weakness in external rotation and abduction. When entrapment occurs at or distal to the suprascapular notch, the supraspinatus is spared, and atrophic changes occur in the infraspinatus only.

The axillary nerve may also suffer from denervation injury due to excessive compression of the structures that pass through an anatomical tunnel called quadrilateral space syndrome (QSS) or direct trauma (e.g., inappropriate injection) to the nerve (see Chap. 1). The QSS is located between the teres major, teres minor, long head of triceps, and humeral neck [26, 27]. Apart from the deltoid, the teres minor is innervated by the posterior branch of the axillary nerve and may show atrophic changes in this syndrome which would affect abduction and external rotation of the shoulder. Pain and paresthesia in a non-dermatomal distribution are other clinical features of QSS. This syndrome is often related to repetitive overhead activities, lipomas, hematomas, labral cysts, and acute glenohumeral subluxation or dislocation.

Fatty infiltration or degeneration refers to fatty accumulation within the muscles. Accumulation of intramuscular fat occurs as a result of aging, disuse, or muscle injury. The significance of fatty infiltration was first described by Goutallier et al. in 1994. Presence of advanced fatty infiltration is a negative prognostic factor in the success of rotator cuff repair. Radiologically, the amount of fatty degeneration is graded according to the Goutallier grading scale [28]. This classification system is based on the percentage of fatty changes in the involved muscle with grade 0 corresponding to no fatty deposits, grade 1 showing some fatty streaks, grade 2 indicating less fat than muscle, grade 3 representing as much fat as muscle, and grade 4 showing more fat than muscle. It is important to note that fatty infiltration refers to the muscle substance and not the size or the space it occupies within its normal compartment. Therefore, decreased muscle size should not be confused with fatty infiltration. For example an atrophic supraspinatus muscle without fatty infiltration corresponds to a Goutallier stage 0 or 1 (normal or mild degenerative changes). It has been shown [29] that measurements of skeletal muscle mass (atrophy) and quality (fatty infiltration) are interchangeable between MRI and CT scan with high agreement between two imaging modalities.

Ultrasonography (US)

Advantages of ultrasound include portability, low cost, tolerance for metallic implants, and lack of contraindications. These advantages and the high accuracy of the US conducted in academic center by musculoskeletal trained radiologists make the US as the first choice of modality in the evaluation of rotator cuff pathology. Ultrasound can detect impingement by active abduction of the shoulder that causes pressure on the rotator cuff tissue or buckling of the coracoacromial ligament, a flexibility that other modes of imaging do not have [30, 31]. Apart from being a diagnostic tool, US can be used for abscess drainage or aspiration and lavage of calcium deposits in the rotator cuff tendons. Initially, calcium deposits are identified by the US, and a needle can then be placed precisely into the tendon to flush or wash out the calcium with fluid.

Of note, variation in examination techniques and operator performances create difficulty in direct interpretation of the images by the primary clinicians, and the quality of US reports varies among community clinics and academic institutes.

Magnetic Resonance Imaging (MRI) without Contrast

The conventional or non-contrast MRI is used for examination of the soft tissues of the shoulder joint, especially partial- or full-thickness tears of the rotator cuff and muscle fatty infiltration and atrophy. MRI is superior to US as more information on potential associated pathologies can be obtained. It has been noted that the MRI may not be necessary in posterior dislocation of the shoulder because soft tissue injury is rare in posterior instability [32]. For optimal diagnosis of soft tissue injuries following anterior subluxation or dislocation, MR arthrography is considered the gold standard.

Magnetic Resonance Arthrography (MRA)

MRA uses gadolinium-based diluted contrast material to enhance imaging and is a standard modality for assessment for labral tears such as Bankart lesions and superior labral anteroposterior (SLAP) lesions. In enhanced MRI, the contrast agent (e.g., gadolinium) is injected into the joint (intra-articular MRA) or into the vein (intravenous MRA). MRA is not indicated for diagnosis of the cuff pathology. In MRA, the bursal and intrasubstance lesions will not be visualized on the majority of the images as they do not communicate with the joint space. They can however be seen on T_2-weighted images, and for this purpose in certain institutes, a T2-weighted image in one plane is often added to an MRA study. Consequently, it is imperative for the primary clinicians to be clear about the purpose of ordering MRA. Should a primary clinician expect cuff pathology in association with labral pathology, this needs to be clearly worded in the imaging requisition, so at least one T_2-weighted sequence is included in the MR arthrography protocol.

Computed Tomography (CT) Scan

The standard computed tomography scan is the method of choice for studying humeral head defects and glenoid bone loss. CT scanning can accurately evaluate the size of the instability-related defects and the intra-articular structures such as the labrum. The CT scan has a limited role in examining rotator cuff pathology but can evaluate rotator cuff muscle atrophy and fatty degeneration. CT arthrography may be used to evaluate rotator cuff tears that communicate with the articular surface only when the MRI is contraindicated. CT scan is an important modality in surgical cases with metal anchors as their associated artifacts often compromise the diagnostic quality of MRI images [31, 33, 34]. The CT scan is the optimal choice in diagnosing malignant primary or metastatic tumors.

Disadvantages, Precautions, and Contraindications

Conventional Radiography

In terms of disadvantages, plain radiographs do not have the ability to rule in minor rotator cuff pathologies (tendinitis, partial-thickness or small full-thickness tears), although chronic pathologies are usually associated with irregularities of the greater tuberosity or narrowing of the subacromial area. Plain radiographs are contraindicated in the management of pregnant women in non-trauma settings. With respect to precautions, plain radiographs produce ionizing radiation, considered carcinogenic. While a prolonged and cumulative radiation dosage may increase the risk of cancer [35, 36], the benefits of plain radiographs far outweigh the potential negative consequences of using them.

Ultrasonography (US)

The disadvantage of the US is its limited ability for assessment of the capsule, labrum, cartilage, and the intraosseous abnormalities. Certain patient-related factors such as obesity impede the interpretation of the US images due to low penetration rate of the US waves into deep tissues. The standard US imaging does not have any contraindications [31, 33]. Using US for abscess drainage or aspiration and lavage of calcium deposits is considered mildly invasive and may be associated with complications such as infection.

Magnetic Resonance Imaging (MRI) without Contrast

The disadvantages of standard or unenhanced MRI relate to its limitations with respect to assessment of the structures that look dark on pulse sequences and are hard to differentiate. Pathologies such as articular and superior labral tears, cartilage defects, partial tears of the long head of the biceps tendon, partial-thickness rotator cuff tears, and adhesive capsulitis which is associated with thickening of the joint capsule of the axillary pouch are among pathologies that may require an enhanced MRI if the assessment affects management.

The absolute contraindications for non-contrast MRI apply to certain metallic implants and electronic devices [37]. Patients who have a metallic foreign body in the eye and gastric reflux devices cannot have an MRI scan because the magnetic field may dislodge the metal, affect the functionality of the device, or cause thermal injuries. Primary clinicians should inform the radiology department of cochlear implants, insulin pumps, and brain aneurysm clips as they may or may not be exposed to MRI depending on the institute. More recently, MRI has been suggested as a safe mode of imaging in patients with implanted cardiac pacemakers in some institutes. However, the patient's electrocardiogram (ECG) should be continuously monitored during the procedure by a physician. In addition, the device should be fully examined and reprogrammed to MRI-safe settings before entering the MRI room and after the procedure to ensure that no damage to the device has occurred and the patient's original device settings are restored [38]. Considering US taken in academic centers is nearly as valid as the MRI for most shoulder pathologies, there is no need to use the MRI investigation in patients with cardiac pacemakers, although this may change in the near future.

Potential metal foreign bodies in the eyes should be checked and ruled out in patients who have worked with sheet metal as metal fragments may have entered their orbits. The radiologist must clear the orbital foreign body with plain radiographs or a screening CT scan prior to the MRI. In addition, use of MRI for patients who have drug infusion pumps, epidural catheters, intracranial vascular clips, prosthetic heart valves, some foley catheters, and breast or penile implants is unadvisable and needs to be discussed with the radiologist. While exposure to MRI during the second and third trimester of pregnancy is not reported to be associated with increased risk of harm to the fetus, ordering an MRI for the pregnant patients should be done after consultation with the radiologist especially in the first trimester of pregnancy [39].

Those with metallic devices placed in the shoulder (e.g., plates, screws) could have an MRI scan, but the resolution of the scan is often significantly affected by the metal implants, and the soft tissues are not well visualized. Patients with symptoms of claustrophobia may require the administration of a mild sedative.

Magnetic Resonance Arthrography (MRA)

All contraindications of the standard MRI apply to the enhanced MRI or MRA. In addition to the above-noted contraindications, the enhanced MRI is more invasive than the MRI due to the contrast agent (e.g., gadolinium) injected into the joint (intra-articular MRA) or into the vein (intravenous MRA) to improve visualization of the soft tissues. The intravenous contrasts are mostly eliminated from the body through normal kidneys, but use of enhanced MRI is not recommended in patients with preexisting kidney problems as the gadolinium retention may lead to a rare condition called nephrogenic systemic fibrosis. Gadolinium-based intravenous MRI is contraindicated at any time during pregnancy and is reported to be associated with increased risk of a broad set of rheumatological, inflammatory, or infiltrative skin conditions and for stillbirth or neonatal death and should be avoided [39]. The intra-articular MR arthrogram may be associated with potential allergic hemorrhagic or infectious complications, and ordering clinicians should ensure that patients are not on anticoagulation or antiplatelet aggregation medication and verify the absence of allergies to the contrast before ordering MR arthrogram [40].

Computed Tomography (CT) Scan

In terms of contraindications, the use of non-contrast or enhanced CT scan is not indicated in pregnant women in non-trauma settings [41, 42]. The use of CT scan in trauma settings or patients with potential malignancy or metastasis where CT scan can provide valuable information should justify the contraindications and thus should be discussed with the radiologist.

References

1. Dunn PM. Wilhelm Conrad roentgen (1845-1923), the discovery of x rays and perinatal diagnosis. Arch Dis Child Fetal Neonatal Ed. 2001;84(2):F138-9.
2. Kraft E, Finby N. Wilhelm Conrad Roentgen (1845-1923). Discoverer of x-ray. N Y State J Med. 1974;74(11):2066–70.
3. Eisenberg R, Amorosa JK. The discoverer of x-rays: Wilhelm Conrad roentgen. N J Med. 1995;92(11):723–4.
4. Nusslin F. Wilhelm Conrad Rontgen: the scientist and his discovery. Phys Med. 2020;79:65–8.
5. Goud A, Segal D, Hedayati P, Pan JJ, Weissman BN. Radiographic evaluation of the shoulder. Eur J Radiol. 2008;68(1):2–15.
6. Sanders TG, Jersey SL. Conventional radiography of the shoulder. Semin Roentgenol. 2005;40(3):207–22.
7. Hill HA, Sachs MD. The grooved defect of the humeral head a frequently unrecognized complication of dislocations of the shoulder joint. Radiology. 1940;35(6):690–700.
8. Charousset C, Beauthier V, Bellaiche L, Guillin R, Brassart N, Thomazeau H, et al. Can we improve radiological analysis of osseous lesions in chronic anterior shoulder instability? Orthop Traumatol Surg Res. 2010;96(8 Suppl):S88-93.
9. 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(11):1470–7.
10. Koh KH, Han KY, Yoon YC, Lee SW, Yoo JC. True anteroposterior (Grashey) view as a screening radiograph for further imaging study in rotator cuff tear. J Shoulder Elb Surg. 2013;22(7):901–7.
11. Razmjou H, Palinkas V, Christakis C, Kennedy D, Robarts S. Diagnostic value of acromiohumeral distance in predicting rotator cuff pathology: implications for advanced practice physiotherapists. Physiother Can. 2020;72(1):52–62. https://doi.org/10.3138/ptc-2018-0084.
12. Sanders TG, Zlatkin M, Montgomery J. Imaging of glenohumeral instability. Semin Roentgenol. 2010;45(3):160–79.
13. Neer CS 2nd, Poppen NK. Supraspinatus outlet. Orthop Trans. 1987;11:234.
14. Duralde XA, Gauntt SJ. Troubleshooting the supraspinatus outlet view. J Shoulder Elb Surg. 1999;8(4):314–9.
15. Liotard JP, Cochard P, Walch G. 2 roentgen projections for the subacromial space before and following acromioplasty. Results of a study series of 40 patients. Orthopade. 1991;20(5):310–4.
16. Liotard JP, Cochard P, Walch G. Critical analysis of the supraspinatus outlet view: rationale for a standard scapular Y-view. J Shoulder Elb Surg. 1998;7(2):134–9.
17. Zanca P. Shoulder pain: involvement of the acromioclavicular joint. (analysis of 1,000 cases). Am J Roentgenol Radium Therapy, Nucl Med. 1971;112(3):493–506.
18. Rokous JR, Feagin JA, Abbott HG. Modified axillary roentgenogram. A useful adjunct in the diagnosis of recurrent instability of the shoulder. Clin Orthop Relat Res. 1972;82:84–6.
19. Bloom MH, Obata WG. Diagnosis of posterior dislocation of the shoulder with use of Velpeau axillary and angle-up roentgenographic views. J Bone Joint Surg Am. 1967;49(5):943–9.
20. Hall RH, Isaac F, Booth CR. Dislocations of the shoulder with special reference to accompanying small fractures. J Bone Joint Surg Am. 1959;41-A(3):489–94.
21. Rowe CR, Patel D, Southmayd WW. The Bankart procedure: a long-term end-result study. J Bone Joint Surg Am. 1978;60(1):1–16.
22. Zanetti M, Gerber C, Hodler J. Quantitative assessment of the muscles of the rotator cuff with magnetic resonance imaging. Investig Radiol. 1998;33(3):163–70.
23. Dwyer T, Razmjou H, Henry P, Gosselin-Fournier S, Holtby R. Association between preoperative magnetic resonance imaging and reparability of large and massive rotator cuff tears. Knee Surg Sports Traumatol Arthrosc. 2015;23(2):415–22.

24. Carroll KW, Helms CA, Otte MT, Moellken SM, Fritz R. Enlarged spinoglenoid notch veins causing suprascapular nerve compression. Skelet Radiol. 2003;32(2):72–7.
25. Lajtai G, Wieser K, Ofner M, Raimann G, Aitzetmuller G, Jost B. Electromyography and nerve conduction velocity for the evaluation of the infraspinatus muscle and the suprascapular nerve in professional beach volleyball players. Am J Sports Med. 2012;40(10):2303–8.
26. Dalagiannis N, Tranovich M, Ebraheim N. Teres minor and quadrilateral space syndrome: a review. J Orthop. 2020;20:144–6.
27. Molina Granados JF, Dominguez FE. Quadrilateral space syndrome with complete denervation of the deltoid and teres minor muscles. Radiologia. 2010;52(4):373–4.
28. 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.
29. Faron A, Sprinkart AM, Kuetting DLR, Feisst A, Isaak A, Endler C, et al. Body composition analysis using CT and MRI: intra-individual intermodal comparison of muscle mass and myosteatosis. Sci Rep. 2020;10(1):11765. -020-68797-3
30. Singh JP. Shoulder ultrasound: what you need to know. Indian J Radiol Imaging. 2012;22(4):284–92.
31. Nazarian LN, Jacobson JA, Benson CB, Bancroft LW, Bedi A, McShane JM, et al. Imaging algorithms for evaluating suspected rotator cuff disease: Society of Radiologists in Ultrasound consensus conference statement. Radiology. 2013;267(2):589–95.
32. Cicak N. Posterior dislocation of the shoulder. J Bone Joint Surg Br. 2004;86(3):324–32.
33. Draghi F, Robotti G, Jacob D, Bianchi S. Interventional musculoskeletal ultrasonography: precautions and contraindications. J Ultrasound. 2010;13(3):126–33.
34. Lecouvet FE, Simoni P, Koutaissoff S, Vande Berg BC, Malghem J, Dubuc JE. Multidetector spiral CT arthrography of the shoulder. Clinical applications and limits, with MR arthrography and arthroscopic correlations. Eur J Radiol. 2008;68(1):120–36.
35. Akinmade A, Ikem I, Ayoola O, Orimolade E, Adeyeye A. Comparing ultrasonography with plain radiography in the diagnosis of paediatric long-bone fractures. Int Orthop. 2019;43(5):1143–53.
36. Thaker A, Navadeh S, Gonzales H, Malekinejad M. Effectiveness of policies on reducing exposure to ionizing radiation from medical imaging: a systematic review. J Am Coll Radiol. 2015;12(12 Pt B):1434–45.
37. Ghadimi M, Sapra A. Magnetic resonance imaging (MRI), contraindications. StatPearls Treasure Island (FL): StatPearls Publishing LLC; 2020.
38. Schukro C, Puchner SB. Safety and efficiency of low-field magnetic resonance imaging in patients with cardiac rhythm management devices. Eur J Radiol. 2019;118:96–100.
39. Ray JG, Vermeulen MJ, Bharatha A, Montanera WJ, Park AL. Association between MRI exposure during pregnancy and Fetal and childhood outcomes. JAMA. 2016;316(9):952–61.
40. Aubry S, Belanger D, Giguere C, Lavigne M. Magnetic resonance arthrography of the hip: technique and spectrum of findings in younger patients. Insights Imaging. 2010;1(2):72–82.
41. Yoon I, Slesinger TL. Radiation exposure in pregnancy. StatPearls Treasure Island (FL): StatPearls Publishing LLC; 2020.
42. Tirada N, Dreizin D, Khati NJ, Akin EA, Zeman RK. Imaging pregnant and lactating patients. Radiographics. 2015;35(6):1751–65.

Chapter 11
Radiographic Features of Rotator Cuff and Biceps Tendon Pathologies

Presence of impingement syndrome and rotator cuff or biceps tendon tears could be suggested by plain radiographs, despite the origin of pathology being in soft tissues and supposedly invisible on X-rays. Chronic encroachment upon the supraspinatus outlet will create visible changes that affect the greater tuberosity and may lead to subchondral cysts, sclerosis, and spur formation on the undersurface of the acromioclavicular joint. Most importantly, significant cuff deficiency affects the overall biomechanics of the glenohumeral joint in more advanced cases of cuff tear arthropathy that can provide critical information about the reparability of the cuff tear and type of required investigations and management (reverse shoulder arthroplasty vs. partial repair).

Subacromial Impingement Syndrome

Radiographic finding of subacromial impingement on plain radiographs includes acromial and clavicular sclerotic changes, subacromial enthesophytes and osteophytes, subchondral cysts, calcified tendinitis, irregularities of the greater tuberosity, and osteoarthritis of the acromioclavicular (AC) joint. Of note, all of these findings are common in asymptomatic older individuals [1].

Inferior Cortical Acromial Sclerosis (Sourcil Sign)

This radiological sign refers to a hyperdense white line seen undersurface of the acromion that resembles an eyebrow above the humeral head (sourcil is a French word for eyebrow) [2]. Sclerotic changes of the inferior acromion are commonly seen in combination with other abnormal radiological signs such as acromial spurs,

H. Razmjou, M. Christakis, *Clinical and Radiological Examination of the Shoulder Joint*, https://doi.org/10.1007/978-3-031-10470-1_11

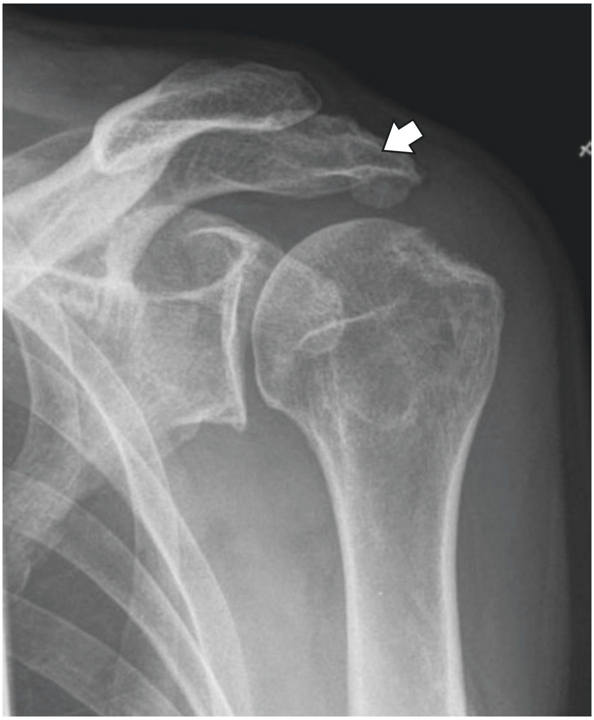

Fig. 11.1 Sourcil sign, sclerotic changes undersurface of the acromion that resembles an eyebrow above the humeral head

humeral cysts, and superior migration of the humeral head. They, however, can be age-related [3], and to date the cause and effect relationship between acromion sclerotic changes and impingement syndrome remains controversial [2]. Both standard and true AP views are used to examine inferior acromion changes. However, as noted in Chap. 10, the true AP view provides a better visualization of the pathognomonic findings of greater tuberosity [4]. Fig. 11.1 shows acromial sclerosis of the acromion on true AP view.

Subacromial Spurs

The word “spur” is a lay term used to refer to osseous overgrowth or bony projections. The appropriate medical term for the spur is “enthesophyte” or “osteophyte,” often used interchangeably but refer to completely different entities.

Subacromial Enthesophytes

Enthesophytes are bony projections developed at an enthesis, which is the attachment site of a ligament or tendon onto the bone. The inferior acromial enthesophytes were first reported in the literature in 1922, by Dr. William Washington

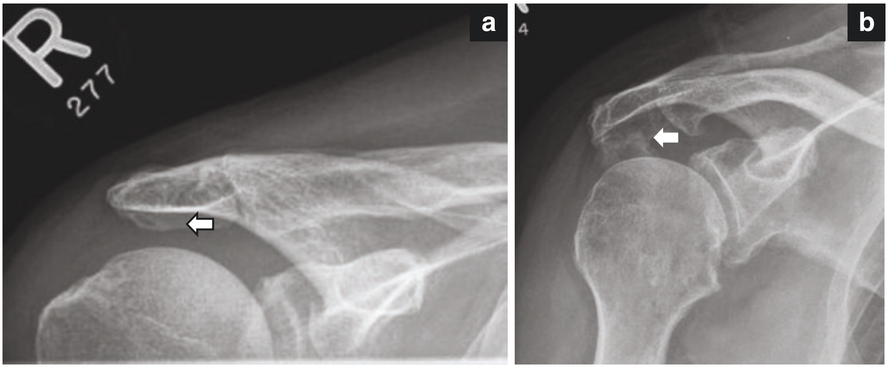

Fig. 11.2 (**a**) A round subacromial enthesophyte in standard AP view. (**b**): Large lateral enthesophyte in standard AP view

Grave who examined human cadavers of variable ages [5]. Grave referred to them as bony plaques and noted that they were strongly suggestive of ossification of the acromial portion of the coracoacromial ligament [5, 6]. The relationship between subacromial enthesophytes and impingement was first described in relation to subacromial impingement syndrome by Neer in 1972 [7, 8].

Enthesophytes are caused by the inflammation of the enthesis or repetitive strain [9] and are formed in the direction of the natural pull of the ligament or tendon involved.

During shoulder flexion and internal rotation, the coracoacromial ligament functions as an anterosuperior restraint. The enthesophytes appear to be the consequence of the load on the insertion of the ligament during these activities [7, 10–13]. Natsis et al. reported that enthesophytes were most common in hooked-type acromion (type III) and rarely reported with type I and II acromions [14]. They are evident on plain radiographs at the anteroinferior surface [11] or lateral aspect of acromion [3, 15]. The true AP view is the best view to see lateral acromial enthesophytes. The supraspinatus outlet view is the best view to see subacromial enthesophytes. Figures 11.2a, b demonstrate different-sized lateral enthesophytes.

Subacromial Osteophytes

The osteophytes are degenerative bony projections that form along margins of the synovial joints like the acromioclavicular joint. The mechanism of the osteophyte formation is not fully understood but it appears that osteophyte growth requires mediation by cells in the synovial lining which is related to cartilage damage of the joint [16].

Dissimilar to subacromial enthesophytes which are caused by physical or mechanical stressor inflammation of enthesis, osteophytes are mainly age-related and are associated with osteoarthritis of the AC joint, although the mechanical stress

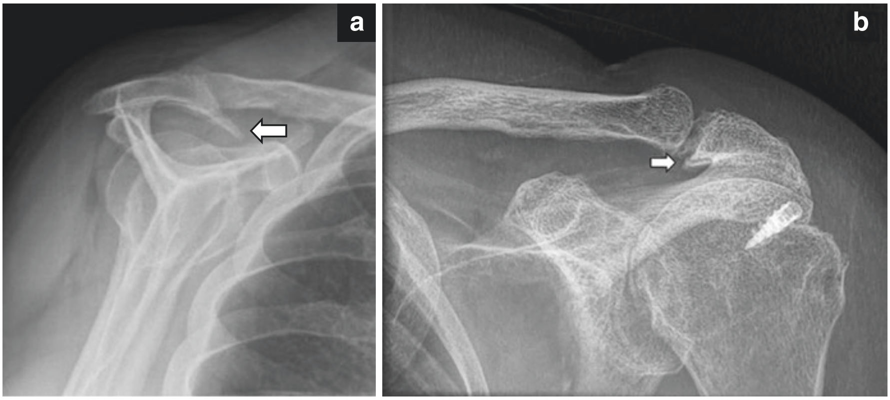

Fig. 11.3 (**a**): A large sharp subacromial enthesophyte in supraspinatus view. (**b**) A subacromial osteophyte in Zanca view

in an arthritic joint with altered biomechanics should not be overlooked [9, 17]. The osteophytes related to the AC joint are better visualized in the Zanca and supraspinatus views [18]. Figures 11.3a, b show a subacromial enthesophye and an AC joint related osteophye.

Greater Tuberosity Cortical Irregularity

Degenerative changes of the greater tuberosity are commonly seen in chronic rotator cuff disease with or without a tear and have been established to be independent of aging [19]. These changes appear to be related to decreased bone mineral density and inflammation of the enthesis where the supraspinatus and infraspinatus tendons connect to the bone [9, 20]. Both the AP and axillary views can show bony irregularities of the greater tuberosity. Figure 11.4 shows cortical irregularity of the greater tuberosity on standard AP view.

Calcified Tendinitis

This type of tendinitis occurs when a portion of the tendon undergoes fibrocartilaginous transformation, and calcification occurs in the transformed tissue. The smaller foci of calcium deposits cause symptoms due to an inflammatory reaction which will gradually resolve through vascular development and absorption of macrophages and multinuclear giant cells. The larger deposits may cause additional

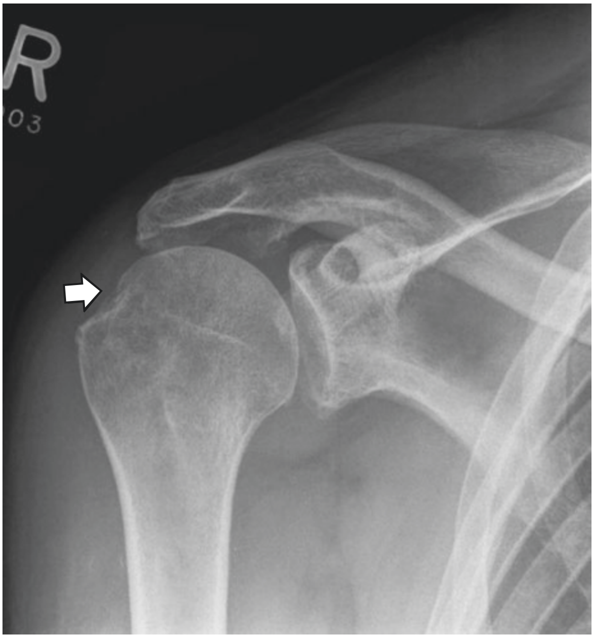

Fig. 11.4 Cortical irregularity of the greater tuberosity in standard AP view

mechanical symptoms and impingement. The calcified paste occasionally leaks into the subacromial bursa, which may cause painful symptoms [21]. Conventional radiography is the most appropriate imaging approach for this condition, and the use of ultrasonography (US) or magnetic resonance imaging (MRI) is usually not necessary for diagnostic purposes. The calcific deposits resemble chalklike spots on plain radiographs. Most cases resolve spontaneously, and the size reduction over time can be visualized and monitored on plain radiographs.

Calcified tendinitis of the supraspinatus is detectable on standard (preferably external rotation) and true AP views at the tendon insertion to the anteromedial part of greater tuberosity. The infraspinatus calcified tendinitis is very rare and occurs at the anterolateral part of greater tuberosity. The subscapularis calcifications occur at lesser tuberosity and can be missed on the AP view and are better identified on scapular Y view and even more clearly on the axillary view [22]. According to Ailent et al., the visibility of the subscapularis tendon calcification depends on the rotation of the arm in AP view, for example, it overlaps with lesser tuberosity on AP neutral rotation; it is lateralized on AP external rotation and is medialized on AP internal rotation. The calcification is visible under the coracoid process on the Y view [22]. Overall, the axillary view is recommended for lesser tuberosity pathologies including calcified tendinitis and small and minimally displaced fractures [23]. Figs. 11.5a–c show different-sized calcified tendinitis of the rotator cuff tendons.

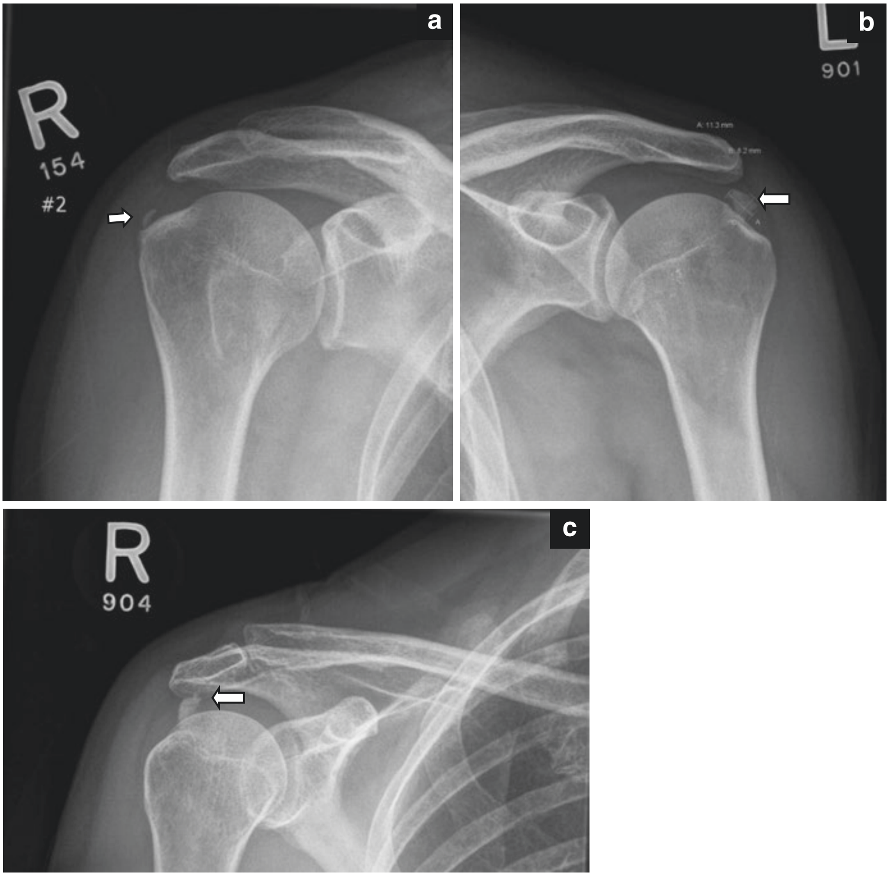

Fig. 11.5 (**a**) A small calcified tendinitis of the supraspinatus tendon in standard AP view. (**b**) A large calcification in the region of the distal supraspinatus tendon in standard AP view. (**c**). A large posterior globular focus of calcification adjacent to the greater tuberosity in keeping with calcific tendinosis of the infraspinatus tendon

Acromion Morphology

The first classification of acromion morphology was based on variations in the architecture of the coracoacromial arch and was described by Bigliani in 1986 [24]. The classification includes a flat undersurface (type I), a gentle undersurface curvature (type II), and an anterior hook (type III) [24]. The authors highlighted the importance of distinguishing between acquired spurs with variations in the native architectural type of the acromion [24]. However, there is controversy about the hooked acromion (type III) being a normal morphology with some authors suggesting that hooked acromions have acquired characteristic and are a result of degenerative changes that occur with the aging process in persons with cuff pathology [14, 25]. Consistent with this suggestion, it has been shown that the shape of acromion does not change with age in individuals without rotator cuff pathology [26].

Types II and III acromions are associated with an increased incidence of impingement syndrome [27, 28], and cuff tear sizes are significantly larger in type III acromion [29]. Hyvonen et al. [30] reported that the acromial angle of patients with rotator cuff tear was significantly greater than the acromial angle of the age-matched healthy controls. The thickness of the anterior part of the acromion at the tendinitis stage (stage I) and the acromial angle at the tear stage of impingement syndrome (stage III) are reliable parameters for rotator cuff pathology [30]. The acromion morphology is best evaluated on the supraspinatus outlet view and to a lesser extent on the scapular Y view. Both views are useful for evaluating the contour of the undersurface of the acromion process where impingement syndrome is suspected. Figures 11.6a–c show different types of acromion morphology.

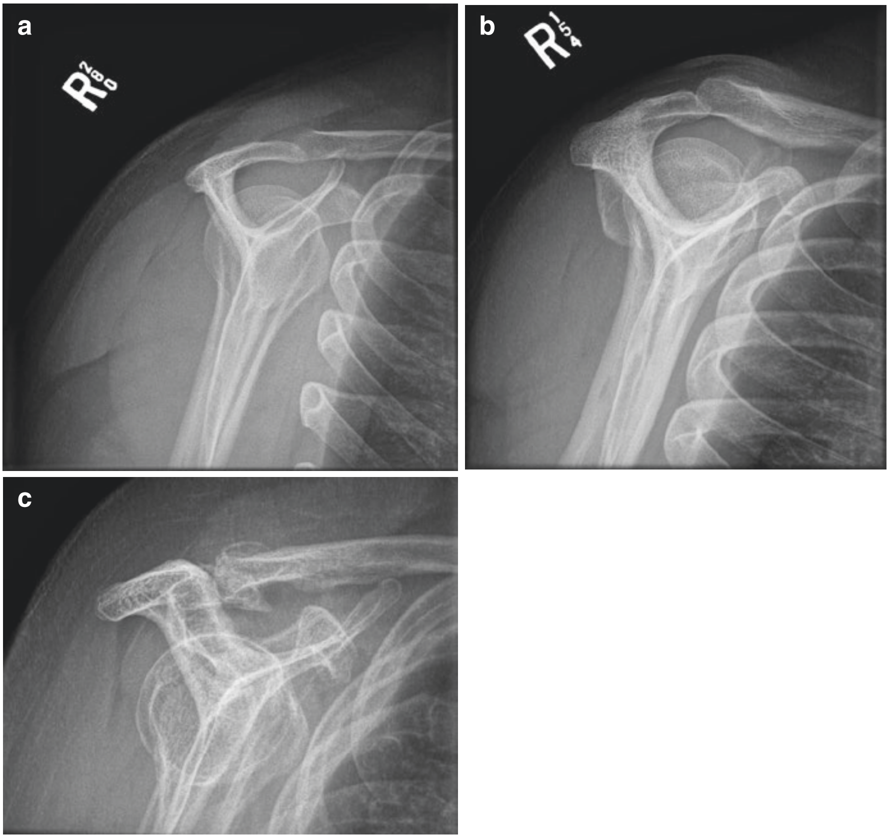

Fig. 11.6 (**a**) Acromion morphology (type I Bigliani acromion). (**b**) Acromion morphology (type II Bigliani acromion). (**c**) Acromion morphology (type III Bigliani acromion)

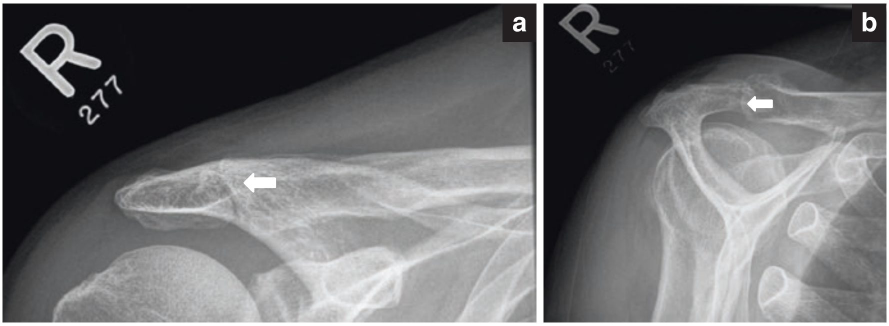

Fig. 11.7 (**a**) Osteoarthritis and joint space narrowing of the AC joint in the Zanca view. (**b**) AC joint profile in supraspinatus outlet view. The supraspinatus outlet view reveals acromial slope and inferior osteophytes at the AC joint but is not a good view for assessment of the AC joint

Acromioclavicular Joint Osteoarthritis

The evidence of mild osteoarthritis of the AC joint represents normal and age-related degeneration commonly seen in asymptomatic individuals as early as the second decade of life [10, 31, 32]. The more advanced arthritic changes include joint space narrowing, incongruency of the articular surfaces, subchondral sclerosis and cysts, and inferior osteophytes rising from margins of the joint. Larger inferior osteophytes may cause significant impingement on the rotator cuff tendons. Optimal visualization of the AC joint is obtained with the Zanca view, where the caudal tilt makes the enthesopathic changes more visible. This projection can be used to demonstrate AC joint pathology including fracture, separation, and arthritis [33]. Figures 11.7a, b demonstrate the AC joint profile on Zanca and supraspinatus outlet views, respectively.

Chondrocalcinosis

The term AC chondrocalcinosis (CC) refers to the radiographic evidence of calcifications in the AC joint cartilage (linear calcifications within the articular discs or punctuate calcifications with cysts and pseudotumor formation seen adjacent to and cephalad above the joint space). Chondrocalcinosis is a common presentation of an inflammatory arthritic disorder, the calcium pyrophosphate dihydrate (CPPD) crystal deposition disease seen in elderly. The AC joint CC is reported to be linked to pseudogout or metabolic conditions such as hyperparathyroidism [34, 35] and is best visualized on Zanca view. Figures 11.8a, b show AC joint CC on Zanca and AP views, respectively.

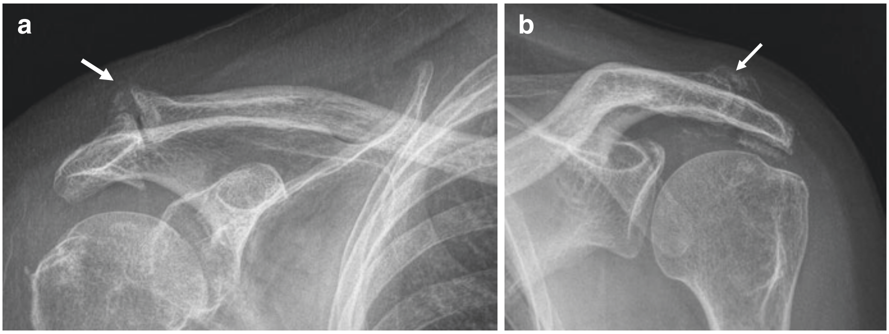

Fig. 11.8 (**a**, **b**) Chondrocalcinosis of the AC joint

Geyser Cyst

Theses cysts are associated with massive rotator cuff tears and may progress to a large "geyser" with an appearance of a tumor due to communication of the cyst with the degenerated AC joint space [36]. These cysts may be associated with the crystal deposition disease and have a very distinct clinical presentation (see Chap. 4). Figure 11.9 shows a geyser cyst on true AP view.

Osteolysis of the Distal Clavicle

Osteolysis results from an imbalance in which bone resorption by osteoclasts is favored over bone formation by osteoblasts. Osteolysis of the AC joint leads to widening of the joint space and may occur as a result of an acute traumatic injury to the shoulder [37] or secondary to repeated microtrauma, described in weight lifters [38]. The lateral clavicle osteolysis is usually unilateral and may not be detectable on plain radiographs for weeks or months after the injury.

Another form of rare resorption of the undersurface of the distal clavicle has been reported in rheumatic arthritis [39, 40]. According to Zanca, the normal AC joint width is between 1 and 3 mm [31]. Progression of the osteolysis and resorption and erosion of the distal clavicle will increase the gap in the AC joint. It is reported that bone loss usually does not exceed 3 cm [41]. Osteolysis of lateral clavicle is best visualized on the Zanca view. Figures 11.10a, b show a range of different severities of osteolysis of the AC joint.

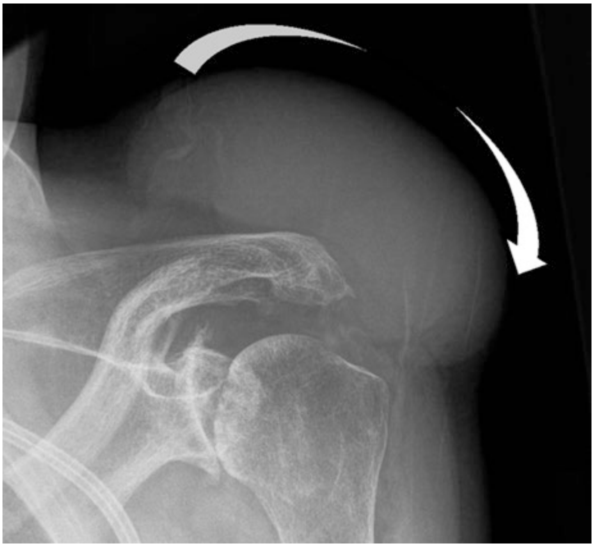

Fig. 11.9 A large geyser cyst on standard AP view. Note the shadow over the superior aspect of the shoulder

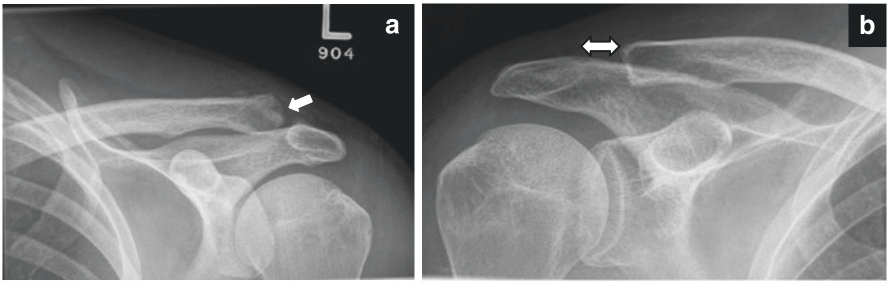

Fig. 11.10 (**a**) Mild osteolysis of the distal end of the clavicle. (**b**) Severe osteolysis of the distal end of the clavicle of traumatic cause

Acromioclavicular Joint Separation

The AC joint separation is a common shoulder injury usually occurring as a result of a fall on an adducted shoulder. The grade I injury involves stretching of the AC ligaments without disruption of the joint capsule and presents with normal radiographs. The radiological features of grade II are slight elevation of distal clavicle relative to acromion with some contact remaining (AC joint capsule and ligaments are disrupted but coracoclavicular ligament is intact) with grade III showing

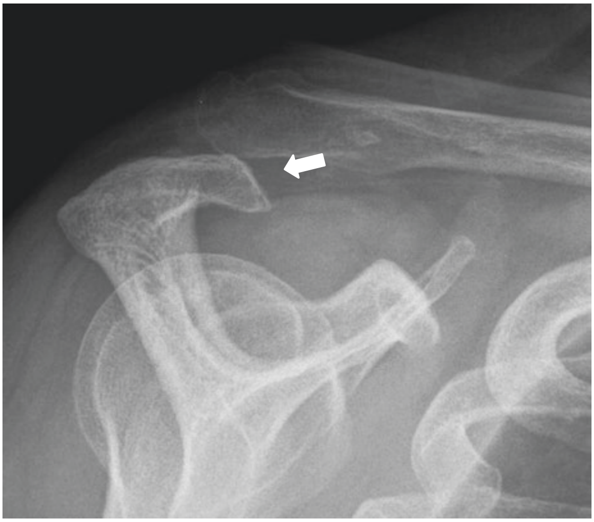

Fig. 11.11 Superior displacement of the clavicle with respect to the acromion by one shaft width compatible with type III AC joint separation

significant elevation of clavicle due to complete disruption of the AC ligaments, capsule, and the coracoclavicular ligament. The Zanca view with and without weight is used to more accurately measure the AC joint traumatic separation. For this purpose, the arms are kept at the sides and sandbags of equal weight are held in each hand. The addition of weights will accentuate AC joint separation by demonstrating elevation of the distal clavicle on the injured side. Figure 11.11 shows a grade III AC separation in Zanca view.

Os Acromiale

The unfused epiphysis of the anterior part of the acromion leads to a deformity called os acromiale, first described by John Gregory Smith, a British anatomist in 1834 [42]. The frequency of os acromiale is reported at 8% in general population [43]. An os acromiale may not always predispose the rotator cuff tendons to tears. However, individuals with step-off deformity of an os acromiale are at a greater risk of rotator cuff tears than are similar persons without such deformity [44]. While a double-density sign on the standard AP view and a cortical irregularity on the supraspinatus outlet view are suggestive of an os acromiale, this condition is best visualized on the axillary view [45, 46]. Figures 11.12a, b show an os acromiale abnormality on axillary view. Figure 11.12c shows a double-density sign on AP view.

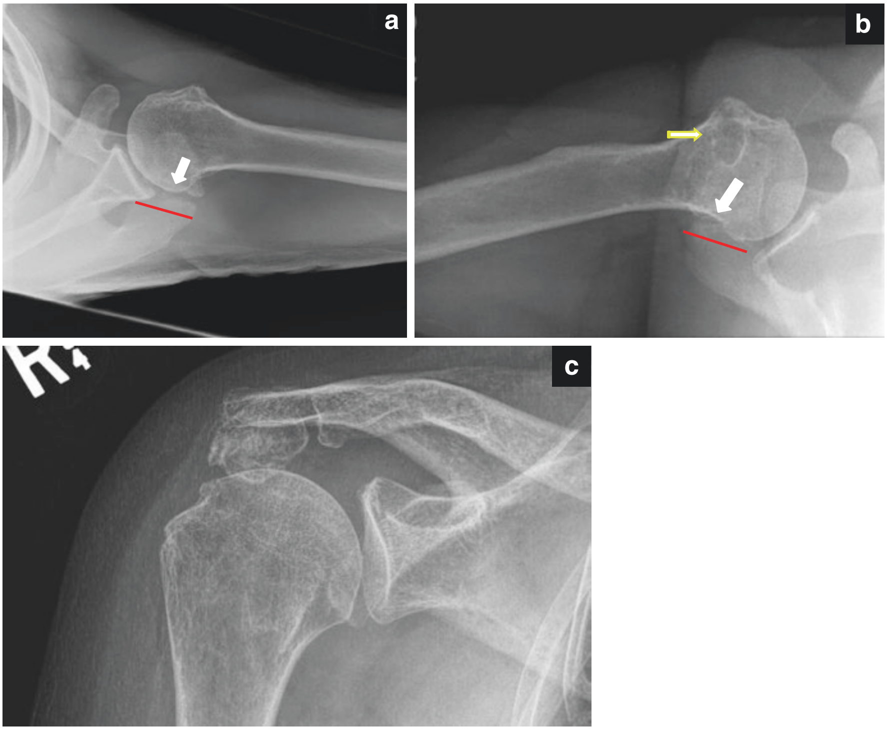

Fig. 11.12 (**a**) Os acromiale in axillary view. Red line shows direction of the defect in relation to humeral shaft. (**b**) Os acromiale in axillary view. Notice a cyst adjacent to greater tuberosity (yellow marker). (**c**) A double-density sign on the standard AP view with downsloping of the acromion and cortical irregularity

Biceps Pathology

Proximal Biceps Tendon Pathology

Plain radiographs are of limited value in diagnosing proximal long head of the biceps (LHB) pathology. Presence of calcification in the bicipital groove or at the insertion of the biceps and cystic changes in the lesser tuberosity may seldom appear in chronic cases. Bicipital calcified tendinitis forms adjacent to proximal humeral shaft and is visualized on the AP and axillary views. Calcific tendinitis of the long head of the biceps brachii tendon origin is uncommon and is seen adjacent to the superior glenoid [47]. Figures 11.13a, b show biceps calcified tendinitis on AP views in two separate cases. Figure 11.13c shows calcification adjacent to the tip of the coracoid in keeping with calcific tendinosis of the short head of the biceps.

Considering other forms of biceps tendon pathology such as tears are not visualized on plain radiographs, other types of imaging investigations are required to examine this tendon more accurately. US has been used to evaluate

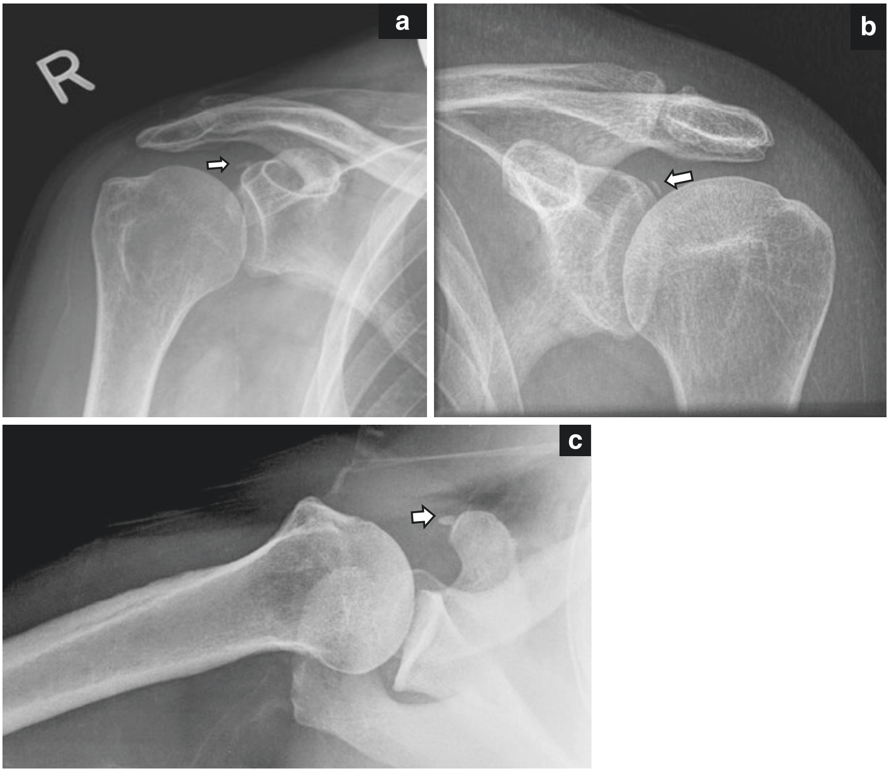

Fig. 11.13 (**a**, **b**). Small foci of calcification deposits adjacent to the superior glenoid margin in keeping with calcified tendinosis of the long head of the biceps tendon. (**c**) Focus of calcification adjacent to the tip of the coracoid process in keeping with calcific tendinosis of the short head of the biceps

the integrity and hypertrophic changes of the LHB by visually examining the gray scale image texture of the tendon [48]. Healthy tendons have a uniform hypoechoic pattern of collagen along the long axis of the tendon. Conversely, tendons with pathology have a more disorganized, diffuse, or hypoechoic appearance [49]. The variability of the US protocols and interpretation of the results may affect its reliability and validity in diagnosing biceps tendon pathologies. The interobserver reliability of biceps pathology has been reported to be fair among experts in musculoskeletal ultrasonography [50]. The principal differences appeared to be related to dynamic examination, definition of tendon lesions, and pathological versus physiological fluid within joints, tendon sheaths, and bursae [50]. Overall, US is noted to be valid for diagnosis of complete LHB rupture and subluxation or dislocation with a questionable reliability for detecting intra-articular partial-thickness tears [51].

Standard MRI and specifically MR arthrogram are suggested for visualizing the intra-articular biceps tendon pathology. Of interest, similar to US, the standard MRI has shown to be valid for detection of full-thickness tears of the biceps tendon and unstable long head of the biceps with limited accuracy for diagnosis of partial tears of the biceps tendon [52, 53].

Distal Biceps Tendon Pathology

The distal biceps tendon originates from bicipital tuberosity of the radius. While majority of proximal biceps tendon pathology is related to aging and associated cuff pathology and treated conservatively, distal biceps pathology is often caused by an acute injury to distal biceps attachment and may need surgical intervention. Plain radiographs are of limited value in diagnosing distal biceps pathology. It has been reported that US can accurately diagnose complete distal biceps tears [54, 55] with less certainty for low-grade partial tears at the attachment site [54]. For distal biceps pathology, MRI has been postulated to be the gold standard for identifying different types of pathology [56]. In a retrospective MRI review of 46 patients with distal biceps tendon pathology and 10 asymptomatic patients by 2 blinded radiologists, Festa et al. [56] reported a sensitivity of 100% and specificity of 82.8% for complete tears of the distal biceps. The sensitivity and specificity of MRI for partial tears were 59.1% and 100%, respectively. Accordingly, although MRI is quite helpful in diagnosing complete tears, it is substantially less sensitive in ruling out partial tears. The difficulty differentiating tendinosis from low-grade partial tears and high-grade partial tears from non-retracted complete tears was noted by these authors. The accuracy of MRI in the study conducted by O'Driscoll et al. was 85% in detecting complete tears and 92% in detecting partial tears [57].

Partial- and Full-Thickness Rotator Cuff Tears

The most accurate assessment of the extent of rotator cuff tear is through the standard MRI. Nevertheless, an indirect evidence of rotator cuff pathology can be seen on plain radiographs via impact on the humeral head, subacromial space, or acromion process. While different aspects of rotator cuff tear can be visualized on standard AP view, supraspinatus outlet view, and axillary view, the most useful view for detecting full-thickness tears is the true AP view [4]. An inadequate overlap between the anterior and posterior glenoid rims affects the accuracy of certain measurements and especially critical shoulder angle (see below) [58]. Different studies have shown that regardless of type of bony abnormality associated with cuff tear, the true AP is more sensitive and reliable than the standard AP view [4, 59–61].

In traumatic tears, particularly in younger individuals, plain radiographs fail to show significant bony abnormality following an injury. In individuals with chronic degenerative tears, enthesophytes, and osteophytes, irregularity of the greater tuberosity, cystic changes in the humeral head, and superior subluxation of humeral head may be evident depending on the chronicity and severity of the rotator cuff tear. The presence of inferior cortical acromial sclerosis, lateral acromial spur, and cysts in the greater tubercle and superior migration of the humeral head are reported to be independent predictors of rotator cuff tears [3]. The morphology of the acromion is another important factor that primary care clinicians should consider when assessing the possibility of rotator cuff tear.

Inferior Cortical Acromial Sclerosis (Sourcil Sign)

Sclerotic changes of inferior acromion are common in chronic rotator cuff pathology as a component of abnormal stress on the acromion process. The best view to visualize the cortical changes under the acromion process and the sourcil sign is true AP view (Fig. 11.14, short arrow).

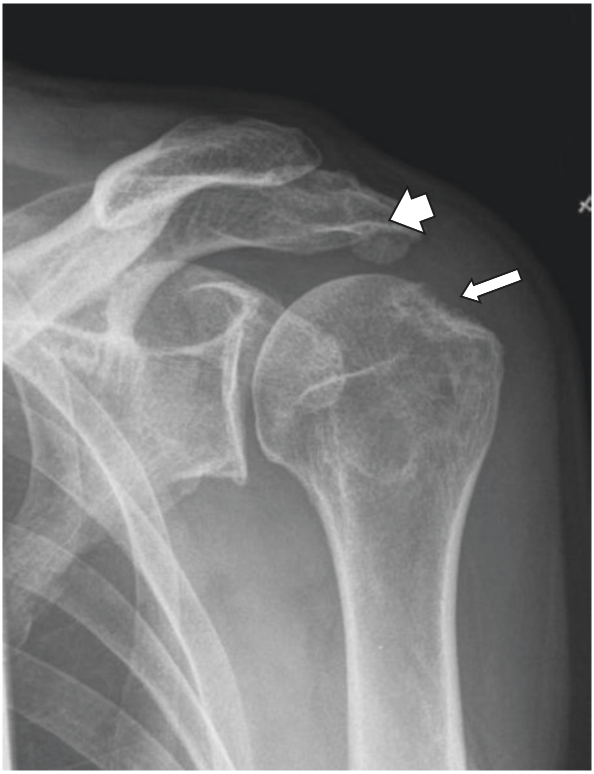

Fig. 11.14 Bony irregularity in greater tuberosity consistent with rotator cuff pathology (long arrow). Note sourcil sign (short arrow)

Greater Tuberosity Cortical Irregularity

The association between irregularity of the greater tuberosity and rotator cuff tears has been established to be independent of aging [19]. In a study by Chung et al. [62], greater tuberosity sclerosis seen on plain radiographs was associated with supraspinatus and infraspinatus muscle atrophy, as well as supraspinatus fatty infiltration. In a study by Ghandour et al., greater tuberosity sclerosis was a valid sign of full-thickness rotator cuff tear and was more commonly seen with large-sized rotator cuff tears [63]. In a study that compared the sensitivity of standard and true AP plain radiographs, the investigators found that the true AP was superior over the standard AP view in detecting medium-sized full tears. Both views appeared to be similar in detecting small or large tears [4]. Fig. 11.14 (long arrow) shows greater tuberosity enthesopathic changes.

Humeral Head Cysts

In 1964, Cotton and Rideout [64] hypothesized that microavulsive cuff tears might incite an inflammatory reaction, resulting in synovial fluid entering the cysts sometimes seen on plain radiographs of the humeral head. Other reasons such as elevated intra-articular pressure and impact between opposing osseous surfaces have been proposed [65]. Cysts located at or near footprint of the cuff tendon have shown fluid or soft tissue signal intensities [66].

The osseous cystic changes of the greater tuberosity may be anterior (supraspinatus insertion site) or posterior (infraspinatus insertion site). Cysts have been reported to be seven times more frequent in the posterior aspect of the greater tuberosity than anteriorly with most cysts having communication with the joint [67]. Similarly, Fritz et al, [68] who examined the location of humeral cysts in 238 consecutive patients, reported that posterior cysts occurred in about 57% of shoulders and showed no statistical correlation to age or cuff diagnosis. Anterior cysts occurred in about 23% of shoulders and showed strong association with cuff disease. Of interest, after controlling for cuff disorders, the age was not a significant contributor to anterior cysts either. In summary, anterior cysts were closely associated with cuff disorders and posterior cysts showed nearly random distribution among patients, regardless of age and cuff diagnosis [68]. The cystic changes within lesser tuberosity are less common. Wissman et al. who reviewed 1000 consecutive MRI of the shoulder reported that lesser tuberosity cysts were rare (less than 3.2%) but were indicative of subscapularis and supraspinatus tendon abnormalities [66]. The association of anterior greater tuberosity cysts with rotator cuff tears has been highlighted by other investigators [69]. The subchondral cysts may reduce biological healing capacity and the strength of the repair fixation [70].

To summarize the above findings, the posterior greater tuberosity cysts are common in asymptomatic shoulder and do not have a strong correlation with aging or cuff disease. On the other hand, cysts within the anterior aspect of greater tuberosity

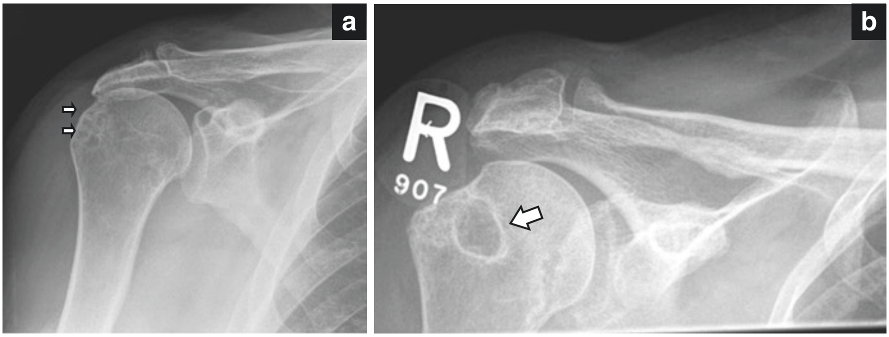

Fig. 11.15 (**a**, **b**) Humeral head cysts of different sizes in standard AP view

and lesser tuberosity cysts are rare but are strongly associated with supraspinatus and subscapularis tendon tears. Figs. 11.15a, b show humeral head cysts.

Subacromial Enthesophytes and Osteophytes

Enthesophytes are reported to be more present in patients with full-thickness cuff tears than in those without a tear. The heel-type spur has been reported to be the most common feature in full-thickness tears [15]. The true AP view is the best view to see lateral enthesophytes. The enthesophytes and osteophytes related to the AC joint are better visualized on Zanca and supraspinatus outlet views (Figs. 11.2 and 11.3).

Superior Migration of the Humeral Head in Relation to the Glenoid

It has been suggested that superior glenohumeral migration during the arm elevation [71, 72] is influenced both by rotator cuff pathology and the state of fatigue of the rotator cuff musculature. In a study by Chen and colleagues [71], two series of radiographs were performed before and immediately after performing a series of deltoid and rotator cuff fatiguing exercises. After fatigue, excursion of the humeral head increased to an average of 2.5 mm and with the initiation of abduction, the humeral head demonstrated significant superior migration or translation in all positions tested. Similarly, Royer et al. [72] who examined the dynamic glenohumeral arthrokinematics provided evidence that simple muscle fatigue could cause a superior migration of the humeral head in relation to glenoid during arm elevation subjecting the rotator cuff to outlet impingement.

Ogata and Uhthoff [12] have suggested that partial tears of the rotator cuff may contribute to proximal migration of the humeral head. This finding is common in elderly with multiple degenerative partial cuff tears. Large and massive full-thickness rotator cuff tears are commonly associated with a high-riding humeral head which leads to decreased acromiohumeral distance (AHD) [73–75]. In two studies we conducted on the value of AHD in relation to extent of rotator cuff disease, we found that reduced AHD <6 mm was positively correlated with muscle wasting and weakness on clinical examination and with fatty infiltration of the supraspinatus and infraspinatus muscles on the MR imaging [18, 76]. Figures 11.16a–d show progression of superior subluxation of the humeral head in relation to glenoid process overtime.

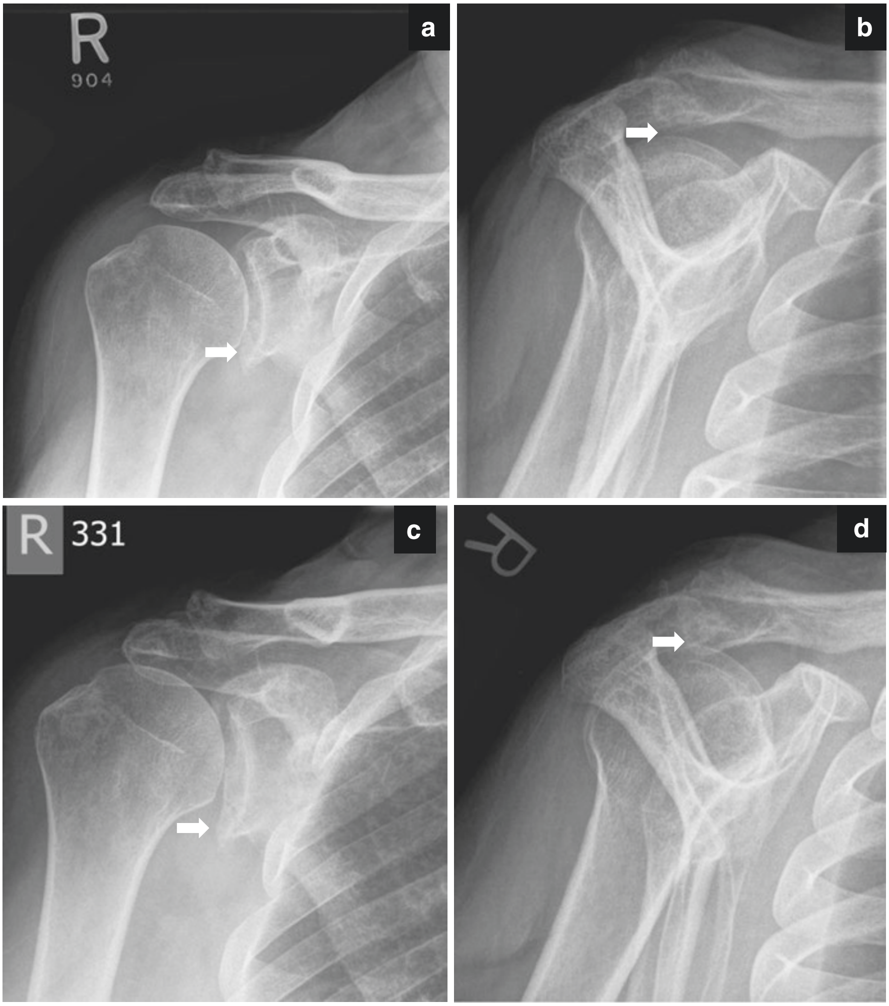

Fig. 11.16 (**a**–**c**). Progression of superior subluxation of the humeral head as a result of a large rotator cuff tear in 8 months. Figure **a** (AP view) and **b** (supraspinatus outlet view) taken in April and Figs. **c** and **d** show the same views in December of the same year

Critical Shoulder Angle

In 2013, Moor et al. described the critical shoulder angle (CSA), an imaging feature of scapular morphology which incorporates the glenoid inclination and the lateral offset of the acromion [77]. The literature in this area has shown that an increased CSA in the true AP view is associated with full-thickness cuff tears and may be a risk factor for re-tears following an arthroscopic repair [78–81]. In a comparative study we conducted, both radiographic features (AHD < 6 mm and CSA > 35°) were able to confirm the presence of a significant cuff pathology. However, the AHD < 6 mm, which occurs as a result of failure of cuff muscles or tendons, had better measurement properties [82]. It is important to highlight that the standard AP view is not a proper view to measure the CSA on and a true AP view with a full overlap between the anterior and posterior glenoid rims is necessary for an accurate measurement of this angle [58].

Other Imaging Modes of Rotator Cuff Tears

Ultrasound has proven to be an effective imaging modality in the evaluation of rotator cuff disorders. Cortical irregularity of the greater tuberosity and joint and subacromial-subdeltoid bursal fluid are often correlated with the primary signs of impingement and cuff pathology. Tendon degeneration, tendinosis, and intrasubstance tear are demonstrated as internal heterogeneity [83]. With respect to calcific tendinitis, US is sensitive at demonstrating focal calcium hydroxyapatite deposition within the cuff [84, 85]. This deposit accumulation most commonly occurs within the supraspinatus tendon near the greater tuberosity insertion but may be seen in other cuff tendons. With regard to evaluation of the rotator cuff by US, higher variability has been reported in the diagnosis of partial-thickness tears as compared with full-thickness tears [86–88]. The US signs for supraspinatus tendon tears are tendon nonvisualization for complete full-thickness tears [83]. In relation to detecting fatty infiltration in rotator cuff muscles, specificity has been reported to be high, but sensitivity values are low [89]. Tests that are highly specific and poorly sensitive are not optimal for screening purposes. This means that a negative US for fatty infiltration is not necessarily indicative of a lack of fatty infiltration. So, if the clinician identifies muscle wasting on examination and US indicates lack of fatty changes in the muscles, an MRI is recommended. In summary, US is considered an accurate modality for the initial investigation of the rotator cuff, especially supraspinatus tears [90]. Overall, recent publications indicate US imaging can be considered almost equally effective in detecting partial tears of the rotator cuff compared to MRI, particularly located in the area of the supraspinatus tendon. It is recommended that MRI be reserved for doubtful or complex cases, in which delineation of adjacent structures is mandatory prior to surgical intervention [91].

The MRI findings of the rotator cuff tear depend on the severity of the tear. A partial-thickness tear is defined as an increase in the signal noted on the T1-weighted images with brighter signal on the T2-weighted images, as well as an identification of a focal defect on either the bursal or the articular surface of the involved tendon [92]. Articular surface tears are more common and can be easily visualized in association with joint effusion, as a focal region of fiber discontinuity that is filled with fluid signal. These tears may partially heal with granulation tissue or scarring, making them difficult to identify. Bursal surface tears can be visualized as a focal extra-articular fluid-filled gap in the superior (bursal) surface of the tendon. Intrasubstance tears are characterized by abnormal intra-tendinous fluid signal on T_2-weighted images without extension to the bursal or the articular surface [90].

The most specific sign of a full-thickness tear on MRI is discontinuity of the cuff fibers. Tendon retraction is a sign of larger and often more chronic tears. The myotendinous junction of the supraspinatus muscle is normally located directly above the humeral head on coronal oblique images with the arm held in neutral position. The extent of tendon retraction can be determined by measuring the distance between the greater tuberosity and the location of the retracted myotendinous junction on the coronal oblique images [93–95]. Atrophic changes (reduced muscle mass) of the rotator cuff muscles are often associated with fatty degeneration of the muscle (replacement of muscle fibers by fat tissue) and occur as a result of chronic rotator cuff tears or less commonly denervation as a result of traction injury or compression. Fatty degeneration can be best seen on T_1-weighted images as strands of high signal within the substance of the muscle [96]. The subscapularis may become torn after massive rotator cuff tears of the supraspinatus and infraspinatus. Less commonly, the subscapularis may be torn in isolation in chronic subcoracoid impingement or with acute traumatic anterior shoulder dislocation or forced external rotation with or without hyperextension [96, 97]. In general, conventional MRI has a high degree of sensitivity and specificity in detecting cuff pathologies [98–100].

References

1. Bonsell S, Pearsall AW 4th, Heitman RJ, Helms CA, Major NM, Speer KP. The relationship of age, gender, and degenerative changes observed on radiographs of the shoulder in asymptomatic individuals. J Bone Joint Surg Br. 2000;82(8):1135–9.
2. Smith C, Dattani R, Dean V, Drew S. The sourcil sign: a useful finding on plain X-ray? Shoulder Elbow. 2010;2(1):9–12.
3. van der Reijden JJ, Nienhuis SL, Somford MP, van den Bekerom MPJ, Doornberg JN, van't Riet E, et al. The value of radiographic markers in the diagnostic work-up of rotator cuff tears, an arthroscopic correlated study. Skelet Radiol. 2020;49(1):55–64.
4. Koh KH, Han KY, Yoon YC, Lee SW, Yoo JC. True anteroposterior (Grashey) view as a screening radiograph for further imaging study in rotator cuff tear. J Shoulder Elb Surg. 2013;22(7):901–7.
5. Grave W. Observation on ages changes in the scapula. Am J Phys Anthrop. 1922;5:21–34.
6. Chambler AF, Emery RJ. Acromial morphology: the enigma of terminology. Knee Surg Sports Traumatol Arthrosc. 1997;5(4):268–72.

7. Neer CS 2nd. Anterior acromioplasty for the chronic impingement syndrome in the shoulder. 1972. J Bone Joint Surg Am. 2005;87(6):1399.
8. Neer CS 2nd. Anterior acromioplasty for the chronic impingement syndrome in the shoulder: a preliminary report. J Bone Joint Surg Am. 1972;54(1):41–50.
9. Rogers J, Shepstone L, Dieppe P. Bone formers: osteophyte and enthesophyte formation are positively associated. Ann Rheum Dis. 1997;56(2):85–90.
10. Nicholson GP, Goodman DA, Flatow EL, Bigliani LU. The acromion: morphologic condition and age-related changes. A study of 420 scapulas. J Shoulder Elb Surg. 1996;5(1):1–11.
11. Mahakkanukrauh P, Surin P. Prevalence of osteophytes associated with the acromion and acromioclavicular joint. Clin Anat. 2003;16(6):506–10.
12. Ogata S, Uhthoff HK. Acromial enthesopathy and rotator cuff tear. A radiologic and histologic postmortem investigation of the coracoacromial arch. Clin Orthop Relat Res. 1990;254:39–48.
13. Ozaki J, Fujimoto S, Nakagawa Y, Masuhara K, Tamai S. Tears of the rotator cuff of the shoulder associated with pathological changes in the acromion. A study in cadavera. J Bone Joint Surg Am. 1988;70(8):1224–30.
14. Natsis K, Tsikaras P, Totlis T, Gigis I, Skandalakis P, Appell HJ, et al. Correlation between the four types of acromion and the existence of enthesophytes: a study on 423 dried scapulas and review of the literature. Clin Anat. 2007;20(3):267–72.
15. Oh JH, Kim JY, Lee HK, Choi JA. Classification and clinical significance of acromial spur in rotator cuff tear: heel-type spur and rotator cuff tear. Clin Orthop Relat Res. 2010;468(6):1542–50.
16. Markhardt BK, Li G, Kijowski R. The clinical significance of osteophytes in compartments of the knee joint with normal articular cartilage. AJR Am J Roentgenol. 2018;210(4):W164–71.
17. Resnick D, Niwayama G. Entheses and enthesopathy. Anatomical, pathological, and radiological correlation. Radiology. 1983;146(1):1–9.
18. Razmjou H, Palinkas V, Christakis C, Kennedy D, Robarts S. Diagnostic value of acromiohumeral distance in predicting rotator cuff pathology: implications for advanced practice physiotherapists. Physiother Can. 2020;72(1):52–62. https://doi.org/10.3138/ptc-2018-0084.
19. Wohlwend JR, van Holsbeeck M, Craig J, Shirazi K, Habra G, Jacobsen G, et al. The association between irregular greater tuberosities and rotator cuff tears: a sonographic study. AJR Am J Roentgenol. 1998;171(1):229–33.
20. Waldorff EI, Lindner J, Kijek TG, Downie BK, Hughes RE, Carpenter JE, et al. Bone density of the greater tuberosity is decreased in rotator cuff disease with and without full-thickness tears. J Shoulder Elb Surg. 2011;20(6):904–8.
21. Uhthoff HK, Loehr JW. Calcific tendinopathy of the rotator cuff: pathogenesis, diagnosis, and management. J Am Acad Orthop Surg. 1997;5(4):183–91.
22. Alilet M, Behr J, Nueffer JP, Barbier-Brion B, Aubry S. Multi-modal imaging of the subscapularis muscle. Insights Imaging. 2016;7(6):779–91.
23. Ross GJ, Love MB. Isolated avulsion fracture of the lesser tuberosity of the humerus: report of two cases. Radiology. 1989;172(3):833–4.
24. Bigliani L, Morrison DS, April EW. The morphology of the acromion and its relationship to rotator cuff tears. Orthop Trans. 1986;10:228.
25. Edelson JG. The 'hooked' acromion revisited. J Bone Joint Surg Br. 1995;77(2):284–7.
26. Vahakari M, Leppilahti J, Hyvonen P, Ristiniemi J, Paivansalo M, Jalovaara P. Acromial shape in asymptomatic subjects: a study of 305 shoulders in different age groups. Acta Radiol. 2010;51(2):202–6.
27. Vanarthos WJ, Monu JU. Type 4 acromion: a new classification. Contemp Orthop. 1995;30(3):227–9.
28. Gill TJ, McIrvin E, Kocher MS, Homa K, Mair SD, Hawkins RJ. The relative importance of acromial morphology and age with respect to rotator cuff pathology. J Shoulder Elb Surg. 2002;11(4):327–30.
29. Hirano M, Ide J, Takagi K. Acromial shapes and extension of rotator cuff tears: magnetic resonance imaging evaluation. J Shoulder Elb Surg. 2002;11(6):576–8.

30. Hyvonen P, Paivansalo M, Lehtiniemi H, Leppilahti J, Jalovaara P. Supraspinatus outlet view in the diagnosis of stages II and III impingement syndrome. Acta Radiol. 2001;42(5):441–6.
31. Zanca P. Shoulder pain: involvement of the acromioclavicular joint. (analysis of 1,000 cases). Am J Roentgenol Radium Therapy, Nucl Med. 1971;112(3):493–506.
32. Worcester JN Jr, Green DP. Osteoarthritis of the acromioclavicular joint. Clin Orthop Relat Res. 1968;58:69–73.
33. Davies AM, Hodler J, Bianchi S, Prato N, Martinoli C, Derchi L. Shoulder radiography, imaging of the shoulder. Med Radiol. 2006:3–13.
34. Parperis K, Carrera G, Baynes K, Mautz A, Dubois M, Cerniglia R, et al. The prevalence of chondrocalcinosis (CC) of the acromioclavicular (AC) joint on chest radiographs and correlation with calcium pyrophosphate dihydrate (CPPD) crystal deposition disease. Clin Rheumatol. 2013;32(9):1383–6.
35. Huang GS, Bachmann D, Taylor JA, Marcelis S, Haghighi P, Resnick D. Calcium pyrophosphate dihydrate crystal deposition disease and pseudogout of the acromioclavicular joint: radiographic and pathologic features. J Rheumatol. 1993;20(12):2077–82.
36. Tshering Vogel DW, Steinbach LS, Hertel R, Bernhard J, Stauffer E, Anderson SE. Acromioclavicular joint cyst: nine cases of a pseudotumor of the shoulder. Skelet Radiol. 2005;34(5):260–5.
37. Madsen B. Osteolysis of the acromial end of the clavicle following trauma. Br J Radiol. 1963;36:822–8.
38. Cahill BR. Osteolysis of the distal part of the clavicle in male athletes. J Bone Joint Surg Am. 1982;64(7):1053–8.
39. Lehtinen JT, Kaarela K, Belt EA, Kauppi MJ, Lehto MU. Coracoclavicular involvement--an atypical manifestation in rheumatoid arthritis. Scand J Rheumatol. 1999;28(4):252–3.
40. Resnick D, Niwayama G. Resorption of the undersurface of the distal clavicle in rheumatoid arthritis. Radiology. 1976;120(1):75–7.
41. Levine AH, Pais MJ, Schwartz EE. Posttraumatic osteolysis of the distal clavicle with emphasis on early radiologic changes. AJR Am J Roentgenol. 1976;127(5):781–4.
42. Smith JG. The classic: pathological appearances of seven cases of injury of the shoulder-joint: with remarks. 1834. Clin Orthop Relat Res. 2010;468(6):1471–5.
43. Sammarco VJ. Os acromiale: frequency, anatomy, and clinical implications. J Bone Joint Surg Am. 2000;82(3):394–400.
44. Ouellette H, Thomas BJ, Kassarjian A, Fritz B, Tetreault P, Palmer WE, et al. Re-examining the association of os acromiale with supraspinatus and infraspinatus tears. Skelet Radiol. 2007;36(9):835–9.
45. Lee DH, Lee KH, Lopez-Ben R, Bradley EL. The double-density sign: a radiographic finding suggestive of an os acromiale. J Bone Joint Surg Am. 2004;86(12):2666–70.
46. Burbank KM, Lemos MJ, Bell G, Lemos DW. Incidence of os acromiale in patients with shoulder pain. Am J Orthop (Belle Mead NJ). 2007;36(3):153–5.
47. Goldman AB. Calcific tendinitis of the long head of the biceps brachii distal to the glenohumeral joint: plain film radiographic findings. AJR Am J Roentgenol. 1989;153(5):1011–6.
48. Teefey SA, Middleton WD, Yamaguchi K. Shoulder sonography. State of the art. Radiol Clin N Am. 1999;37(4):767–85. ix
49. Collinger JL, Gagnon D, Jacobson J, Impink BG, Boninger ML. Reliability of quantitative ultrasound measures of the biceps and supraspinatus tendons. Acad Radiol. 2009;16(11):1424–32.
50. Naredo E, Moller I, Moragues C, de Agustin JJ, Scheel AK, Grassi W, et al. Interobserver reliability in musculoskeletal ultrasonography: results from a "teach the teachers" rheumatologist course. Ann Rheum Dis. 2006;65(1):14–9.
51. Armstrong A, Teefey SA, Wu T, Clark AM, Middleton WD, Yamaguchi K, et al. The efficacy of ultrasound in the diagnosis of long head of the biceps tendon pathology. J Shoulder Elb Surg. 2006;15(1):7–11.
52. Razmjou H, Fournier-Gosselin S, Christakis M, Pennings A, ElMaraghy A, Holtby R. Accuracy of magnetic resonance imaging in detecting biceps pathology in patients with rotator cuff disorders: comparison with arthroscopy. J Shoulder Elb Surg. 2015;

53. Mohtadi NG, Vellet AD, Clark ML, Hollinshead RM, Sasyniuk TM, Fick GH, et al. A prospective, double-blind comparison of magnetic resonance imaging and arthroscopy in the evaluation of patients presenting with shoulder pain. J Shoulder Elb Surg. 2004;13(3):258–65.
54. de la Fuente J, Blasi M, Martinez S, Barcelo P, Cachan C, Miguel M, et al. Ultrasound classification of traumatic distal biceps brachii tendon injuries. Skelet Radiol. 2018;47(4):519–32.
55. Garcia Rodriguez C, Garcia-Polin Lopez C, Del Olmo HT, Moros Marco S, Jacobo Edo O, Avila Lafuente JL. Distal biceps tendon rupture: diagnostic strength of ultrasonography and magnetic resonance. Rev Esp Cir Ortop Traumatol. 2020;64(2):77–82.
56. Festa A, Mulieri PJ, Newman JS, Spitz DJ, Leslie BM. Effectiveness of magnetic resonance imaging in detecting partial and complete distal biceps tendon rupture. J Hand Surg Am. 2010;35(1):77–83.
57. O'Driscoll, Goncalves LB, Dietz P. The hook test for distal biceps tendon avulsion. Am J Sports Med. 2007;35(11):1865–9.
58. Suter T, Gerber Popp A, Zhang Y, Zhang C, Tashjian RZ, Henninger HB. The influence of radiographic viewing perspective and demographics on the critical shoulder angle. J Shoulder Elb Surg. 2015;24(6):e149-58.
59. Navarro-Ledesma S, Luque-Suarez A. Comparison of acromiohumeral distance in symptomatic and asymptomatic patient shoulders and those of healthy controls. Clin Biomech (Bristol, Avon). 2018;53:101–6.
60. Mura N, O'Driscoll SW, Zobitz ME, Heers G, Jenkyn TR, Chou SM, et al. The effect of infraspinatus disruption on glenohumeral torque and superior migration of the humeral head: a biomechanical study. J Shoulder Elb Surg. 2003;12(2):179–84.
61. van de Sande MA, Rozing PM. Proximal migration can be measured accurately on standardized anteroposterior shoulder radiographs. Clin Orthop Relat Res. 2006;443:260–5.
62. Chuang HC, Hong CK, Hsu KL, Kuan FC, Lin CL, Su WR. The radiographic morphology of the greater tuberosity is associated with muscle degeneration in patients with symptomatic rotator cuff tears. J Shoulder Elb Surg. 2019;28(10):1964–70.
63. Ghandour TM, Elghazaly SA, Ghandour AM. Greater tuberosity sclerosis: a radiographic sign of rotator cuff tear? Acta Orthop Belg. 2017;83(3):416–20.
64. Cotton RE, Rideout DF. Tears of the humeral rotator cuff; a radiological and pathological necropsy survey. J Bone Joint Surg Br. 1964;46:314–28.
65. Resnick D. Common disorders of synovium-lined joints: pathogenesis, imaging abnormalities, and complications. AJR Am J Roentgenol. 1988;151(6):1079–93.
66. Wissman RD, Kapur S, Akers J, Crimmins J, Ying J, Laor T. Cysts within and adjacent to the lesser tuberosity and their association with rotator cuff abnormalities. AJR Am J Roentgenol. 2009;193(6):1603–6.
67. Williams M, Lambert RG, Jhangri GS, Grace M, Zelazo J, Wong B, et al. Humeral head cysts and rotator cuff tears: an MR arthrographic study. Skelet Radiol. 2006;35(12):909–14.
68. Fritz LB, Ouellette HA, O'Hanley TA, Kassarjian A, Palmer WE. Cystic changes at supraspinatus and infraspinatus tendon insertion sites: association with age and rotator cuff disorders in 238 patients. Radiology. 2007;244(1):239–48.
69. Suluova F, Kanatli U, Ozturk BY, Esen E, Bolukbasi S. Humeral head cysts: association with rotator cuff tears and age. Eur J Orthop Surg Traumatol. 2014;24(5):733–9.
70. Chillemi C, Paglialunga C, Guerrisi M, Mantovani M, Osimani M. Arthroscopic transosseous repair of rotator cuff tear and greater tuberosity cysts. Arthrosc Sports Med Rehabil. 2020;2(3):e241–50.
71. Chen SK, Simonian PT, Wickiewicz TL, Otis JC, Warren RF. Radiographic evaluation of glenohumeral kinematics: a muscle fatigue model. J Shoulder Elb Surg. 1999;8(1):49–52.
72. Royer PJ, Kane EJ, Parks KE, Morrow JC, Moravec RR, Christie DS, et al. Fluoroscopic assessment of rotator cuff fatigue on glenohumeral arthrokinematics in shoulder impingement syndrome. J Shoulder Elb Surg. 2009;18(6):968–75.
73. Gruber G, Bernhardt GA, Clar H, Zacherl M, Glehr M, Wurnig C. Measurement of the acromiohumeral interval on standardized anteroposterior radiographs: a prospective study of observer variability. J Shoulder Elb Surg. 2010;19(1):10–3.

74. Werner CM, Conrad SJ, Meyer DC, Keller A, Hodler J, Gerber C. Intermethod agreement and interobserver correlation of radiologic acromiohumeral distance measurements. J Shoulder Elb Surg. 2008;17(2):237–40.
75. Goutallier D, Le Guilloux P, Postel JM, Radier C, Bernageau J, Zilber S. Acromio humeral distance less than six millimeter: its meaning in full-thickness rotator cuff tear. Orthop Traumatol Surg Res. 2011;97(3):246–51.
76. Razmjou H, Christakis C, Kennedy D, Robarts S, Holtby R. Impact of radiographic imaging of the shoulder joint on patient management: an advanced practice physiotherapist's approach. Physiother Can Fall. 2017;69(4):351–60.
77. Moor BK, Bouaicha S, Rothenfluh DA, Sukthankar A, Gerber C. Is there an association between the individual anatomy of the scapula and the development of rotator cuff tears or osteoarthritis of the glenohumeral joint? A radiological study of the critical shoulder angle. Bone Joint J. 2013;95-B(7):935–41.
78. Li H, Chen Y, Chen J, Hua Y, Chen S. Large critical shoulder angle has higher risk of tendon retear after arthroscopic rotator cuff repair. Am J Sports Med. 2018;46(8):1892–900.
79. Li X, Olszewski N, Abdul-Rassoul H, Curry EJ, Galvin JW, Eichinger JK. Relationship between the critical shoulder angle and shoulder disease. JBJS Rev. 2018;6(8):e1.
80. Song JG, Yun SJ, Song YW, Lee SH. High performance of critical shoulder angle for diagnosing rotator cuff tears on radiographs. Knee Surg Sports Traumatol Arthrosc. 2018;
81. Spiegl UJ, Horan MP, Smith SW, Ho CP, Millett PJ. The critical shoulder angle is associated with rotator cuff tears and shoulder osteoarthritis and is better assessed with radiographs over MRI. Knee Surg Sports Traumatol Arthrosc. 2016;24(7):2244–51.
82. Razmjou H, Palinkas V, Christakis M, Robarts S, Kennedy D. Reduced acromiohumeral distance and increased critical shoulder angle: implications for primary care clinicians. Phys Sportsmed. 2020:1–8.
83. Papatheodorou A, Ellinas P, Takis F, Tsanis A, Maris I, Batakis N. US of the shoulder: rotator cuff and non-rotator cuff disorders. Radiographics. 2006;26(1):e23.
84. Kayser R, Hampf S, Seeber E, Heyde CE. Value of preoperative ultrasound marking of calcium deposits in patients who require surgical treatment of calcific tendinitis of the shoulder. Arthroscopy. 2007;23(1):43–50.
85. Ambacher T, Kirschniak A, Holz U. Intraoperative localization of calcification in the supraspinatus via a percutaneous marking suture after preoperative ultrasound. J Shoulder Elb Surg. 2007;16(2):146–9.
86. Le Corroller T, Cohen M, Aswad R, Pauly V, Champsaur P. Sonography of the painful shoulder: role of the operator's experience. Skelet Radiol. 2008;37(11):979–86.
87. Middleton WD, Payne WT, Teefey SA, Hildebolt CF, Rubin DA, Yamaguchi K. Sonography and MRI of the shoulder: comparison of patient satisfaction. AJR Am J Roentgenol. 2004;183(5):1449–52.
88. Hedtmann A, Heers G. Imaging in evaluating rotator cuff tears. Orthopade. 2007;36(9):796–809.
89. Park BK, Hong SH, Jeong WK. Effectiveness of ultrasound in evaluation of fatty infiltration in rotator cuff muscles. Clin Orthop Surg. 2020;12(1):76–85.
90. Fotiadou AN, Vlychou M, Papadopoulos P, Karataglis DS, Palladas P, Fezoulidis IV. Ultrasonography of symptomatic rotator cuff tears compared with MR imaging and surgery. Eur J Radiol. 2008;68(1):174–9.
91. Vlychou M, Dailiana Z, Fotiadou A, Papanagiotou M, Fezoulidis IV, Malizos K. Symptomatic partial rotator cuff tears: diagnostic performance of ultrasound and magnetic resonance imaging with surgical correlation. Acta Radiol. 2009;50(1):101–5.
92. Yamakawa S, Hashizume H, Ichikawa N, Itadera E, Inoue H. Comparative studies of MRI and operative findings in rotator cuff tear. Acta Med Okayama. 2001;55(5):261–8.
93. 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.

94. Duc SR, Mengiardi B, Pfirrmann CW, Jost B, Hodler J, Zanetti M. Diagnostic performance of MR arthrography after rotator cuff repair. AJR Am J Roentgenol. 2006;186(1):237–41.
95. Koivikko MP, Mustonen AO. Shoulder magnetic resonance arthrography: a prospective randomized study of anterior and posterior ultrasonography-guided contrast injections. Acta Radiol. 2008;49(8):912–7.
96. Opsha O, Malik A, Baltazar R, Primakov D, Beltran S, Miller TT, et al. MRI of the rotator cuff and internal derangement. Eur J Radiol. 2008;68(1):36–56.
97. Patten RM. Tears of the anterior portion of the rotator cuff (the subscapularis tendon): MR imaging findings. AJR Am J Roentgenol. 1994;162(2):351–4.
98. Rutten MJ, Spaargaren GJ, van Loon T, de Waal Malefijt MC, Kiemeney LA, Jager GJ. Detection of rotator cuff tears: the value of MRI following ultrasound. Eur Radiol. 2010;20(2):450–7.
99. Liu F, Cheng X, Dong J, Zhou D, Han S, Yang Y. Comparison of MRI and MRA for the diagnosis of rotator cuff tears: a meta-analysis. Medicine (Baltimore). 2020;99(12):e19579.
100. Roy JS, Braen C, Leblond J, Desmeules F, Dionne CE, MacDermid JC, et al. Diagnostic accuracy of ultrasonography, MRI and MR arthrography in the characterisation of rotator cuff disorders: a meta-analysis. Br J Sports Med. 2015;

Chapter 12
Radiographic Features of Glenohumeral Arthritis

The imaging findings of the glenohumeral joint depend on the cause of arthritis. Primary osteoarthritis is caused by normal degenerative changes and occurs in the process of aging. The inflammatory arthritis is caused by the autoimmune systemic inflammatory disorders. The secondary arthritis is often related to a known cause such as trauma, infection, neuropathy, etc. In this section, we review the imaging findings of common forms of arthritis seen by advanced practice physiotherapists and primary care clinicians. Details on history, etiology, clinical findings, and management of different types of glenohumeral arthritis are discussed in Chap. 7.

Primary Glenohumeral Osteoarthritis

The primary osteoarthritis or idiopathic arthritis occurs without any inciting factors. As it relates to imaging of an arthritic shoulder joint, it is important to remember that patient's report of pain or loss of motion does not always have a linear relationship with the severity of radiographic findings, particularly joint space narrowing [1]. The size of the inferior osteophytes, however, appears to have a negative correlation with active and passive range of motion of the shoulder joint, but not necessarily with symptoms [2]. In other words, majority of radiological parameters appear to play a relatively insignificant role in clinical decision-making process for shoulder arthroplasty candidates.

Typical radiological signs of glenohumeral arthritis are joint space narrowing, asymmetrical glenoid wear, subchondral sclerosis, joint incongruity, and osteophyte formation at the inferior margin of the humeral head or glenoid [3]. Of interest, site of glenoid wear depends on the type of arthritis; posterior glenoid wear which is associated with posterior subluxation of the humeral head is common in primary arthritis, and central glenoid wear with medialization of the humeral head is the hallmark of rheumatic arthritis. Contracture of the anterior capsule and anterior

H. Razmjou, M. Christakis, *Clinical and Radiological Examination of the Shoulder Joint*, https://doi.org/10.1007/978-3-031-10470-1_12

muscles (subscapularis in particular) contributes to posterior humeral head subluxation and a posterior load concentration on the glenoid accelerating the glenoid wear [4]. Subchondral sclerosis is caused by regrowing and remodeling of the bone and is detected in the later stages of osteoarthritis.

In terms of grading of severity of glenohumeral arthritis on imaging, Samilson and Prieto [5] proposed a classification system in 1983. This system was originally developed to describe the degree of arthritis in patients with instability-related arthropathy and later expanded its application to arthritis of other etiologies. Accordingly, three criteria were considered when grading the degree of arthritis: presence and height of inferior osteophytes on the humerus and/or glenoid, joint space narrowing, and presence of sclerosis. Mild arthrosis is defined as the presence of an inferior osteophyte measuring less than 3 mm in height. Moderate arthrosis is indicated when the spur measures between 3 and 7 mm with slight glenohumeral joint irregularity, and severe arthrosis is indicated by the presence of inferior osteophyte measuring more than 7 mm in height, greater narrowing of the glenohumeral joint space, and presence of sclerosis. It is noteworthy that the measurement of the osteophyte height is affected by the rotation of the shoulder joint at the time of radiological examination which can have a negative impact on the accuracy of this classification system [6].

In 1999, another classification of glenoid morphology was proposed by Walch et al., which was based on the amount of central and posterior glenoid wear patterns in patients with primary and noninflammatory arthritis. According to Walch and colleagues [7], the glenoid could have an A shape (central erosion, symmetrical erosion of the glenoid), B shape (asymmetrical posterior erosion), and C and D shapes that mostly refer to dysplasia and anteversion, respectively. Asymmetric bone loss rarely develops in type A glenoid, whereas initial posterior translation of the humeral head in type B glenoid is associated with subsequent development and progression of posterior glenoid bone loss over time. The type B glenoid is suggested to begin with posterior humeral head subluxation (B1), progressing to posteroinferior glenoid wear (B2) which may further erode into a mono-concave and severely retroverted glenoid (B3) [8]. The Walch B-zero (B0) was suggested as a new category by Walch's team as the pre-osteoarthritic posterior subluxation of the humeral head some 20 years after introduction of their initial classification system. Certain developmental conditions such as glenoid dysplasia or repetitive trauma especially in throwing athletes may affect glenoid version over time (initially dynamic, eventually evolving into a static condition) [9].

Differences in fatty infiltration of the posterior aspect of the rotator cuff were seen between A-type and B-type glenoid [10]. Higher fatty infiltration of the infraspinatus and teres minor is associated with increasing glenoid retroversion, with the B3 glenoid being associated with the highest muscle fatty infiltration [11]. The clinical implication is that observing a high-grade glenoid retroversion on plain radiographs is a negative predictor of strength and poorer future surgical outcome. True AP, axillary, and scapular Y views are appropriate for examining the severity of glenohumeral joint arthritis. Figures 12.1 and 12.2 show common features of primary glenohumeral osteoarthritis.

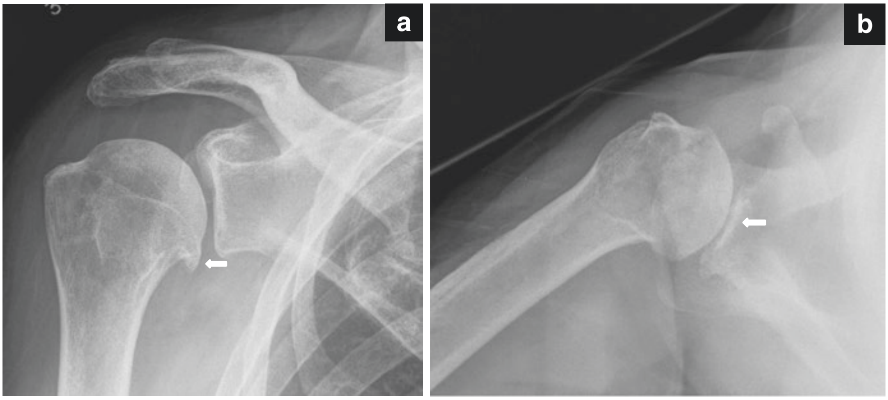

Fig. 12.1 True AP (**a**) and axillary (**b**) views showing mild primary osteoarthrosis of the glenohumeral joint. Note small inferior osteophyte in the AP view and narrowing of the glenohumeral joint space

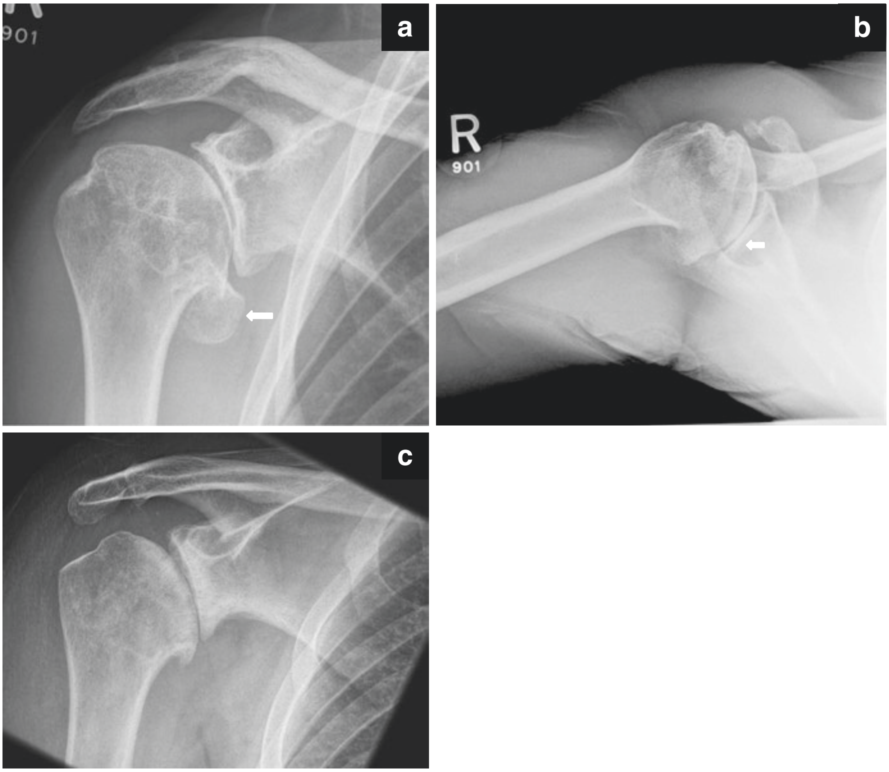

Fig. 12.2 Severe primary osteoarthritis of the glenohumeral joint: (**a**) A large inferior osteophyte (white arrow) and significant joint space narrowing. Figures **b** and **c** show flattening of the humeral head and posterior wear of the glenoid (white arrows), common features of primary osteoarthritis

Autoimmune and Inflammatory Arthritis

There are a number of autoimmune-related conditions that could cause arthritis. Among those, rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), ankylosing spondylitis (AS), and Sjogren's syndrome can be named. Rheumatoid arthritis is the most common autoimmune chronic inflammatory rheumatic disease with progressive joint destruction and will be discussed in this chapter.

The early imaging feature of RA is periarticular osteopenia. Articular bone erosion is the central element of the diagnosis of RA and represent localized cortical bone loss and can be detected in early stages of the disease. Bone erosions are visible on plain radiographs as breaks in the cortical bone surface and are often accompanied by loss of the adjacent trabecular bone. In RA, marginal erosions are the result of mechanical action of the hypertrophied synovium and granulation tissue and typically emerge at the site at which the synovium comes into direct contact with the bone [12].

Progression of joint damage is seen as a gradual superior subluxation and medial displacement of the humeral head due to destruction of the cartilage. The systemic inflammatory nature of RA and synovial swelling cause subcortical cysts seen as translucent lesions surrounded by a sclerotic rim due to reparative processes. Intra-articular loose bodies made of osseous and cartilaginous fragments are frequently observed in late stages.

The Lévigne classification system is used to assess the severity of RA changes [13]. This classification identifies three stages of glenoid wear using an AP radiograph; stage 1 wear is defined by intact or minimally deformed subchondral bone. Stage 2 wear is present when wearing reaches the foot of the coracoid, and stage 3 wear is marked by wearing extending medial to the foot of the coracoid.

The widening of the acromioclavicular joint space (osteolysis of lateral clavicle articular bone) is another unique feature of the systemic inflammation in patients with RA which occurs as a result of localized bone loss. Lehtinin et al. [14] reported that an AC joint space of >7 mm in men and > 5 mm in women is a sign of destructive changes of the acromioclavicular joint in RA.

In summary, there are important differences between imaging of the primary glenohumeral joint arthritis and RA. In primary osteoarthritis, the glenoid is worn posteriorly, and proliferation of the bone occurs on the joint edges. In RA, the cartilage is destroyed evenly across the joint surfaces, and bone destruction occurs at the entire joint surfaces. In primary osteoarthritis, the joint space narrowing occurs early in the process of the disease, but in RA joint, space narrowing is a late phenomenon, and existence of rheumatoid involvement in the glenohumeral joint is based on erosions, not on joint space narrowing [15].

Another important differentiating feature of the RA is the simultaneous involvement of the rotator cuff structures. While primary osteoarthritis is often associated with an intact rotator cuff and normal acromiohumeral distance, chronic cuff

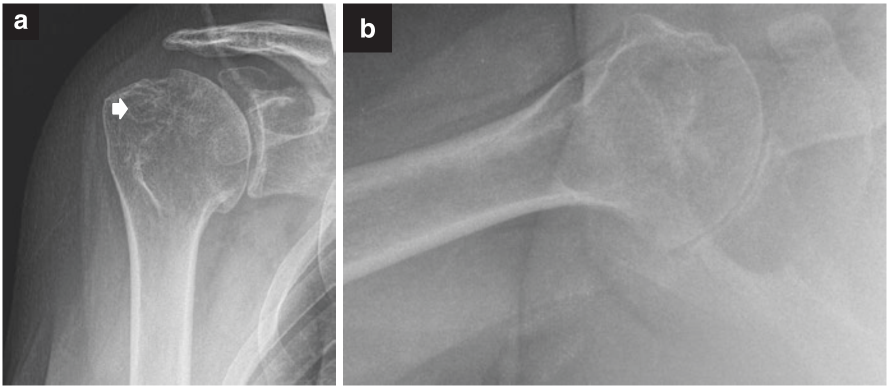

Fig. 12.3 True AP (**a**) and axillary (**b**) views of the glenohumeral joint in a 30-year-old female with the diagnosis of juvenile rheumatoid arthritis. Typical osteopenia and osseous erosion at the superior head junction with the greater tuberosity are visible (white arrow). There are moderate glenohumeral arthritic changes with joint space loss and marginal osteophytes reflecting secondary osteoarthrosis on a background of inflammatory arthropathy

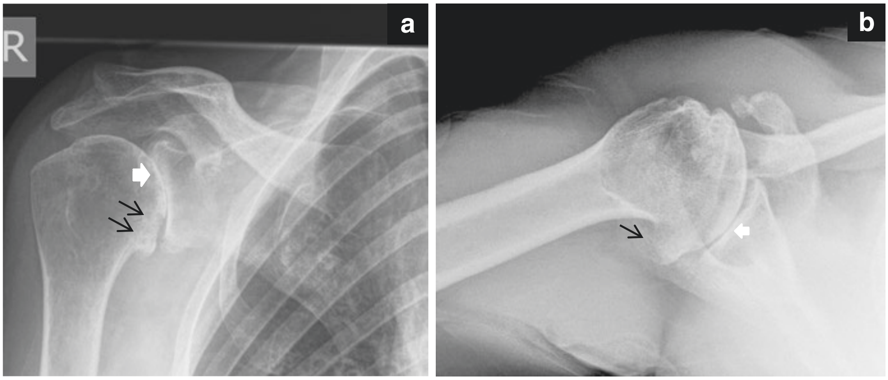

Fig. 12.4 True AP (**a**) and axillary (**b**) views of RA as an example of inflammatory arthropathy with secondary degenerative changes. There is osteopenia with marked narrowing of the glenohumeral joint space (white arrow) and erosive changes at the inferior margin (black arrows) of the humeral head with marginal osteophyte formation

deficiency and superior subluxation of the humeral head are common in RA [16]. The other difference is that the osteolysis of the AC joint is usually considered irreversible in RA, which contrasts with the substantial periosteal bone formation response noted in conjunction with bone erosions in patients with noninflammatory osteoarthritis and traumatic AC joint osteolysis [17]. Figures 12.3 and 12.4 show typical radiographic manifestation of RA in juvenile and adult RA.

Secondary Glenohumeral Arthritis

Secondary glenohumeral arthritis is caused by a known predisposing factor (e.g., massive rotator cuff tear, failed surgery, trauma, recurrent dislocation, old fracture, infection, avascular necrosis, and metabolic conditions) which has adversely altered the joint structures. A sequel deformity such as malunion, Hill-Sachs, or Bankart fracture affects the bone integrity in direct traumatic injuries, where bone necrosis secondary to infection or avascular necrosis is characterized by the death of cellular components of the bone secondary to spread of organisms and/or interruption of the subchondral blood supply.

Cuff Tear Arthropathy

Cuff tear arthropathy (CTA) is the end of the spectrum of a progressive degenerative disease that starts with rotator cuff pathology involving chronic tendon tears and advanced muscle atrophic changes and fatty infiltration and ends with severe joint surface and bone destruction. CTA is one of the most disabling shoulder conditions, and although there is some understanding about its pathomechanics, many questions about this pathology remain unanswered.

Chapter 5 provides a detailed review of this pathology including the clinical presentation and management. In this chapter, we examine radiological features of this pathology with its unique progressive destruction of the coracoacromial arch and glenohumeral joint. In CTA, the humeral head subluxes superiorly due to chronic absence of rotator cuff dynamic stabilizing effect [18]. While all patients with CTA have massive cuff tears, not all massive cuff tears progress to CTA [18].

The early radiological findings of this disease are erosive changes of the insertion of the rotator cuff at the greater tuberosity, acromion process, lateral end of the clavicle, and superior aspect of the glenoid. The subluxation of the humeral head with respect to the glenoid is often seen in early stages of CTA and gradually leads to narrowing of the subacromial space. With recurring contact between bony and articular surfaces, the greater tuberosity becomes worn down, and the acromion gets thinner, rounded, and eroded, a process that finally leads to a concave deformity of the acromial undersurface or acetabularization of the acromion [19]. As the disease progresses, the proximal aspect of humeral articular surface collapses, and degenerative changes affect the glenohumeral joint more medially. Neer considered the collapse of the proximal aspect of humeral articular surface as an important requirement for the diagnosis of CTA syndrome [18].

In contrast with Neer's theory, Hamada et al. [20] suggested that all massive cuff tears progress to CTA through several pathomechanic steps with corresponding roentgenographic changes. Therefore, Hamada's classification system is primarily based on the acromiohumeral interval (AHI) [20]. Accordingly, grade I involves a normal AHI $\geq$6 mm and normal glenohumeral joint. In grade II, the AHI is $\leq$5 mm with normal glenohumeral joint, grade III has an AHI of $\leq$5 mm plus acromial acetabularization, grade IV has an AHI of ≤5 mm plus narrowing of the glenohumeral joint, and finally grade 5 requires the AHI of ≤5 mm plus collapse of the head of the humerus. Hamada's classification system has some flaws that limit its applicability. While massive tears of the rotator cuff lead to abnormal joint mechanics from upward migration of the humeral head, collapse of the proximal aspect of the humeral articular surface does not occur in all patients [21], a theory that had already been suggested by Neer [18]. In addition, Hamada's classification is limited in defining the glenohumeral morphologic changes and anterosuperior escape of the humeral head that occurs in advanced cases [22]. In 2004, Visotsky and Seebauer proposed another imaging classification system that incorporated not only the degree of superior migration of the humeral head from the center of rotation but also the amount of instability of the center of rotation [23]. According to Seebauer's classification, in type IA, the humeral head is centered with intact anterior restraints (coracoacromial ligament, acromion process, and rotator cuff) with minimal superior subluxation. In type IB, the humeral head is still centered but medialized due to glenoid erosion with higher amount of superior subluxation. Types IIA and IIB show instability and decentered humeral head with superior translation and an anterosuperior escape, respectively [23].

In an updated article by Hamada and colleagues in 2011 [24], the authors examined patient-related factors that affected the disease progression. The authors noted that while presence of fatty atrophy in the supraspinatus and particularly infraspinatus muscles is directly related to poorer outcome, the integrity of the subscapularis played an important role in progression to higher grades of glenohumeral joint destruction.

For diagnosis of CTA, plain X-rays are usually sufficient. Computed tomography (CT) scans are often required for preoperative arthroplasty planning to assess the extent of bone loss in the glenoid. Magnetic resonance imaging would not add important information in advanced cases as majority of these cases have a massive and an irreparable rotator cuff tear but may be of benefit in younger patients with early signs of CTA who may benefit from tendon transfer. Figure 12.5 shows an advanced CTA with all features of complete failure of the rotator cuff leading to acetabularization of the acromion and superior dislocation of the humeral head.

Capsulorrhaphy Arthropathy

Capsulorrhaphy arthropathy is a common type of secondary arthritis. Direction of initial instability has a significant association with the severity of the arthritic progression. Patients with posterior glenohumeral instability have a more severe arthritis than those with anterior instability [5]. One of the causes of capsulorrhaphy arthropathy is the overtightening of the anterior capsule which is associated with reduced external rotation and shifting the humeral head posteriorly. This change in normal biomechanics of the shoulder forces the humeral head out of its normal

Fig. 12.5 Standard AP (**a**), supraspinatus outlet (**b**), and axillary (**c**) views of the right shoulder showing superior dislocation of humeral head with respect to glenoid secondary to a massive rotator cuff tear. Note remodeling of undersurface of the acromion, coracoid, and clavicle

concentric relationship with the glenoid fossa causing posterior glenoid wear [25]. Fig. 12.6 shows severe asymmetrical joint space loss, subchondral cysts, and sclerosis on the AP and axillary views.

Avascular Necrosis (AVN) or Osteonecrosis

Avascular necrosis or aseptic osteonecrosis is caused by interruption of the blood supply to the humeral head which leads to death of the cells and is a sequel of trauma, corticosteroid use, excessive alcohol consumption, or severe postsurgical

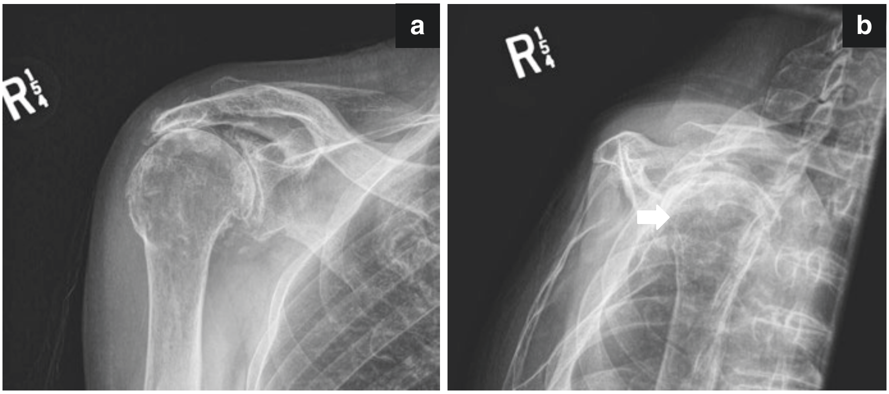

Fig. 12.6 Standard AP view (**a**) and transcapular view (**b**) of the right shoulder with severe secondary glenohumeral osteoarthritis with joint space loss, marginal osteophytes, and articular remodeling secondary to an old anterior dislocation. Superior and anterior subluxation of the humeral head with respect to the glenoid with bony remodeling of the joint surfaces. Note displacement of the humeral head anteriorly in 30B (white arrow)

altered biomechanics. In septic arthritis, the AVN can occur as a result of septic emboli that can cause articular occlusion. See Chap. 5 for more details on etiology and management of the AVN.

The AVN is not detectable on plain radiographs in the early stages of development. An ill-defined mottling of the trabecular pattern with mild lucency and sclerosis is the earliest evidence of osteonecrosis on plain radiographs. The gradual progression of bone resorption and remodeling will cause subchondral bone discontinuity, humeral head collapse, and arthritic changes in advanced cases. AVN has been classified according to the degree of involvement of the glenohumeral joint by Cruess et al. [26]. Stage 1 is identified only with magnetic resonance imaging. In stage 2, sclerotic changes of the humeral head are visualized on plain radiographs. In stage 3, progression occurs to subchondral fracture with presence of a crescent sign. Presence of crescent sign in stage 3 is a hallmark of advanced AVN and usually indicates further collapse of the humeral head [27–29]. Stage 4 involves loss of humeral head normal spherical shape and further collapse of the bone. Finally in stage 5, glenoid arthrosis sets in when degenerative changes are present on both the humeral head and glenoid.

Since clinical examination in early stages of AVN is nonspecific and plain radiographs are often within normal limits, an MRI should be requested if the clinician expects an AVN [30–32]. MRI has an ability to show subchondral bone marrow edema with or without microfracture and is strongly recommended in individuals with a history of trauma or other comorbidities or risk factors (liver damage, history of corticosteroids use, alcohol abuse) or infection (postinjection or post-surgery). A rapid development of AVN with other destructive features seen on plain radiographs following shoulder surgery is often indicative of septic arthritis.

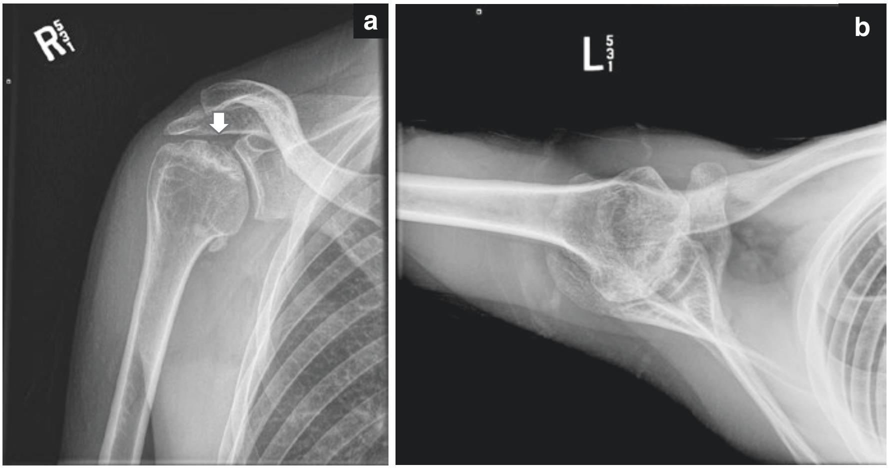

Fig. 12.7 True AP (**a**) and axillary (**b**) views showing evidence of avascular necrosis in the humeral head (white arrow)

Plain radiographs (neutral rotation AP is the most conclusive view) would reveal osteolytic lesions, subchondral sclerosis, subchondral collapse, depression of articular surface, humeral head deformities, severe joint space narrowing, and degenerative joint changes in its end stages. Figures 12.7a, b show collapse of the superior aspect of the humeral head on AP and axillary views.

Septic Arthritis

Organisms may reach the bone and articular cartilage directly from a contiguous focus of infection (e.g., surgical wound), as a result of a direct skin penetration or by hematogenous spread (seen in gonorrhea or following joint arthroplasties). The incidence of septic arthritis is increased in patients who are medically compromised by diabetes mellitus or vascular insufficiency. The early signs of septic arthritis may not be evident on plain radiographs. The MRI findings in septic joints are reported to be fairly similar with non-septic joints, although presence of bone erosions with marrow edema is highly suggestive for a septic articulation; the additional coexistence of synovial thickening, synovial edema, and soft tissue edema increases the above level of confidence [33]. Of note, synovial thickening, periarticular myositis/cellulitis, and bone marrow edema can persist after resolution of the infection [34]. The late sequelae of septic shoulder arthritis are easily detectable on plain radiographs and include rapid destruction of the joint surfaces, cuff tear arthropathy, septic necrosis, and osteomyelitis [35]. In rare cases, an unsuccessful repair of the rotator cuff may alter the biomechanics of the shoulder so drastically that a rapid destruction of the joint may mimic septic arthritis. Figure 12.8a–d shows a postoperative AVN secondary to altered glenohumeral joint as infection was ruled out.

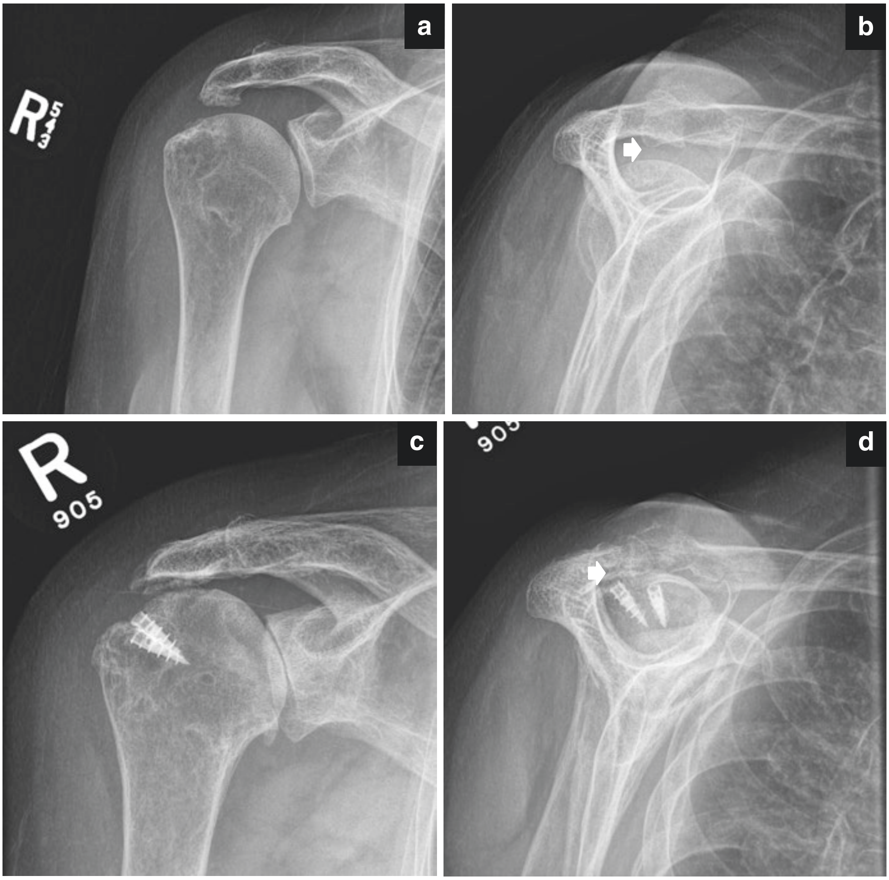

Fig. 12.8 (**a**, **b**) Preoperative standard AP (**a**) and supraspinatus outlet views (**b**) showing mild glenohumeral osteoarthrosis with a small lateral enthesophyte and nonspecific bony irregularity of the greater tuberosity. Note that subacromial space is preserved (white arrow). (**c**, **d**) Postsurgical secondary arthritis due to altered biomechanics and rotator cuff repair failure. Standard AP (**c**) and axillary (**d**) views of the same shoulder 6 months after rotator cuff repair with two suture anchors in place. New flattening and sclerosis of the humeral head reflect avascular necrosis. Note severe joint space narrowing of the glenohumeral joint with osteophyte proliferation (white arrow)

Figures 12.9a, b show destructive changes following a confirmed septic arthritis that resembles an advanced CTA.

Neuropathic Arthropathy

The radiographic appearance of neuropathic arthropathy may mimic septic arthritis. Radiographic diagnosis is predominantly based on typical appearance of humeral head destruction that resembles amputation with or without

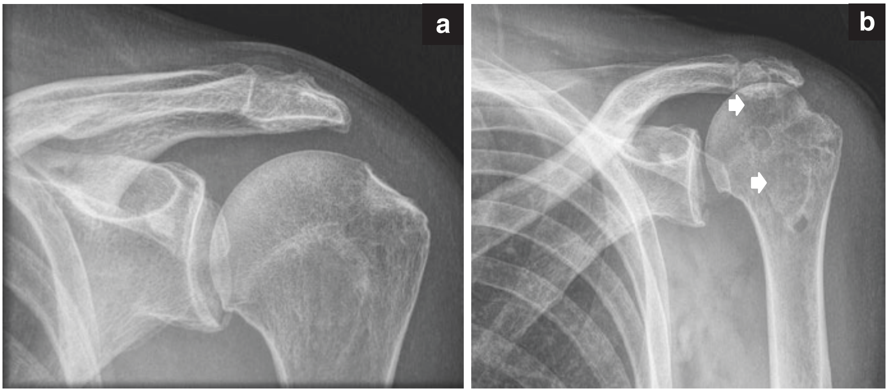

Fig. 12.9 Pre- (**a**) and postoperative (**b**) AP views of the glenohumeral joint of a failed rotator cuff repair complicated with septic arthritis. The postoperative lucencies of the greater tuberosity and humeral neck are related to infection (white arrows)

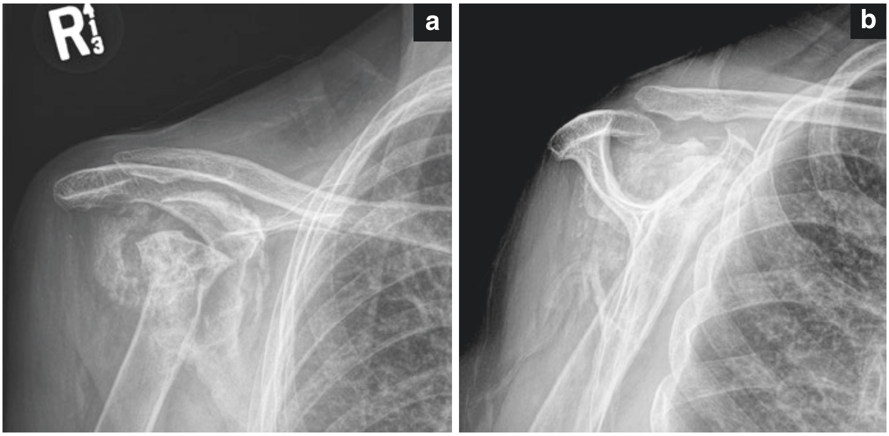

Fig. 12.10 Standard AP (**a**) and transcapular (**b**) views of the right shoulder with neuropathic arthropathy. Collapse and fragmentation of the humeral head with evidence of extensive intra-articular bony debris and disorganization of the glenohumeral articulation

subluxation or dislocation of the glenohumeral joint. The MRI may show peri-articular fluid collection, synovial hypertrophy, and rotator cuff pathology, resembling chronic septic arthritis or sarcoma. Biopsy is the only definitive way of clarifying the diagnosis [36]. Figures 12.10a, b show features of advanced neuropathy on AP and transcapular views.

Metabolic Conditions and Glenohumeral Joint Arthritis

Obesity is a complex metabolic condition that has been linked to arthritis. Dyslipidemia is a part of metabolic syndrome as well and includes hypertriglyceridemia (defined as serum triglycerides ≥150 mg/dL) and a low high-density lipoprotein (HDL) cholesterol concentration [37]. The imaging features of arthritic changes that are linked to common metabolic disorders are not greatly distinguishable from primary arthritis. There are a number of metabolic disorders that may present with recurrent episodes of acute arthritis. Gout is one of the metabolic conditions that affects the big toe and may rarely affect the shoulder joint. Plain radiographic findings depend on the stage of chronicity, but erosive changes and joint space narrowing and posterior subluxation have been reported. The chronic deposition of monosodium urate crystals in joints can gradually lead to severe joint damage [38].

Radiographic Features of Glenohumeral Instability

Anterior Glenohumeral Instability

Anterior instability accounts for 95% of acute traumatic dislocations with the posterior dislocation being way less frequent [39]. The characteristics of anterior dislocation on plain radiographs are the Hill-Sachs lesions and Bankart fractures. Chronic or permanent anterior dislocation is a rare entity with a limited number of publications on the subject [40–43]. Generally any dislocation that has gone unreduced for at least 3 weeks is considered a chronic dislocation. Most cases cannot be successfully treated with closed reduction and require either open reduction and capsulolabral complex repair or joint replacement [41, 42]. Reduction of chronic unreduced shoulder dislocations using arthroscopy has not been widely recommended [43].

Hill-Sachs Lesions

Hill-Sachs lesions are osteochondral compression fracture of the humeral head, caused by impaction of the humeral head into the glenoid during the anterior dislocation. They occur as a result of the soft bone in this region being impacted against the harder glenoid rim and are the most common traumatic chondral lesions of the glenohumeral joint with an incidence ranging from 30% to 71% following the initial anterior dislocation [44–47] increasing to 100% with recurrent dislocations [48]. Hill-Sachs lesions are found on the posterolateral aspect of the humeral head, with

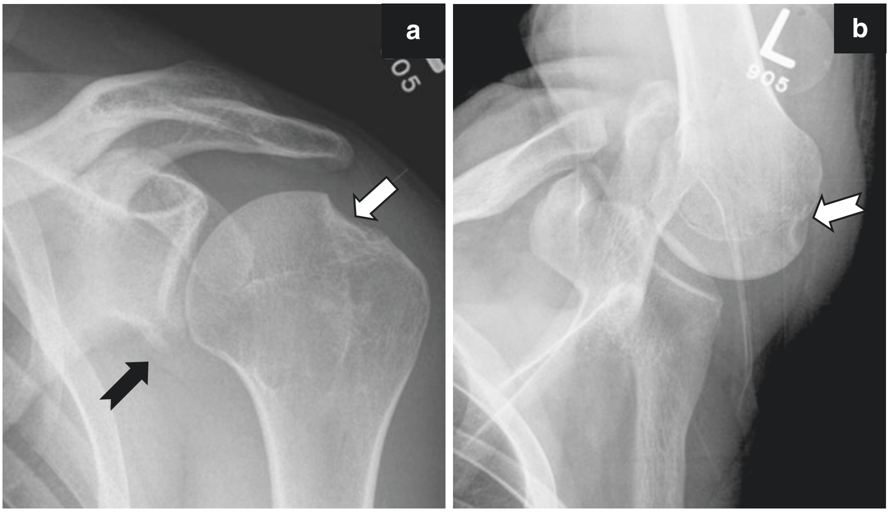

Fig. 12.11 Instability series, standard AP (**a**) showing Hill-Sachs defect (white arrow) associated with a Bankart fracture at the anterior-inferior margin of the bony glenoid (black arrow). Stryker view (**b**), deep Hill-Sachs deformity of the posterolateral humeral head

the specific location varying depending on the amount of abduction and external rotation at the time of dislocation. A positive correlation between the extent and depth of these lesions and the number and frequency of recurrent dislocations has been established [48] with larger lesions being more prone to recurrent dislocations [49–51]. Hill-Sachs lesions are visible on standard or true AP view (Fig. 12.11a). However, the Stryker view is the best view to see the impression fractures caused by anterior dislocation (Fig. 12.11b). A combination of an internally rotated AP view and the Stryker notch view is optimal for evaluation of a Hill-Sachs deformity.

Bankart Cartilaginous and Bony Lesions

The classic Bankart lesion is defined as a soft tissue avulsion of the labral complex from the scapular periosteum, where Bankart fractures involve a bony pathology of the anteroinferior glenoid rim [39]. Bankart cartilaginous or bony lesions are the second common sequel of traumatic anterior shoulder instability. The co-occurrence of cartilaginous or bony Bankart lesions depends on the size of a Hill-Sachs lesion with bigger Hill-Sachs lesions co-occurring with bony rather than cartilaginous Bankart lesions [52].

While an AP view can identify significant Bankart fractures, the west point view is the most specific radiographic projection to assess the anteroinferior glenoid rim and bony Bankart lesions (Figs. 12.12a, b). As noted in Chap. 10, in acute trauma

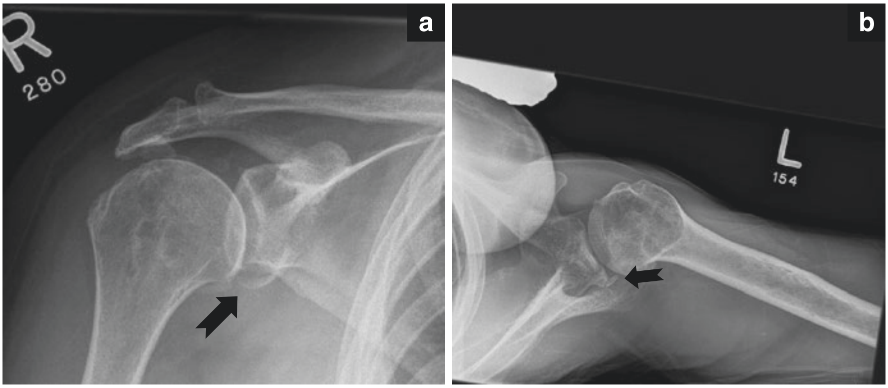

Fig. 12.12 Standard AP view (**a**) and west point axillary view (**b**) showing bony density adjacent to the inferior glenoid in keeping with an osseous Bankart fracture

Fig. 12.13 Traumatic fracture of the anterior inferior glenoid with mild distraction on Velpeau view

settings, where obtaining standard instability views is difficult due to pain and wearing a sling, patient leans backward about 30° toward the table (Velpeau view). Figure 12.13 shows a traumatic fracture of the anteroinferior glenoid fracture.

Figure 12.14a–c shows chronic anterior dislocation of the humeral head with respect to glenoid process on AP, transcapular, and axillary views.

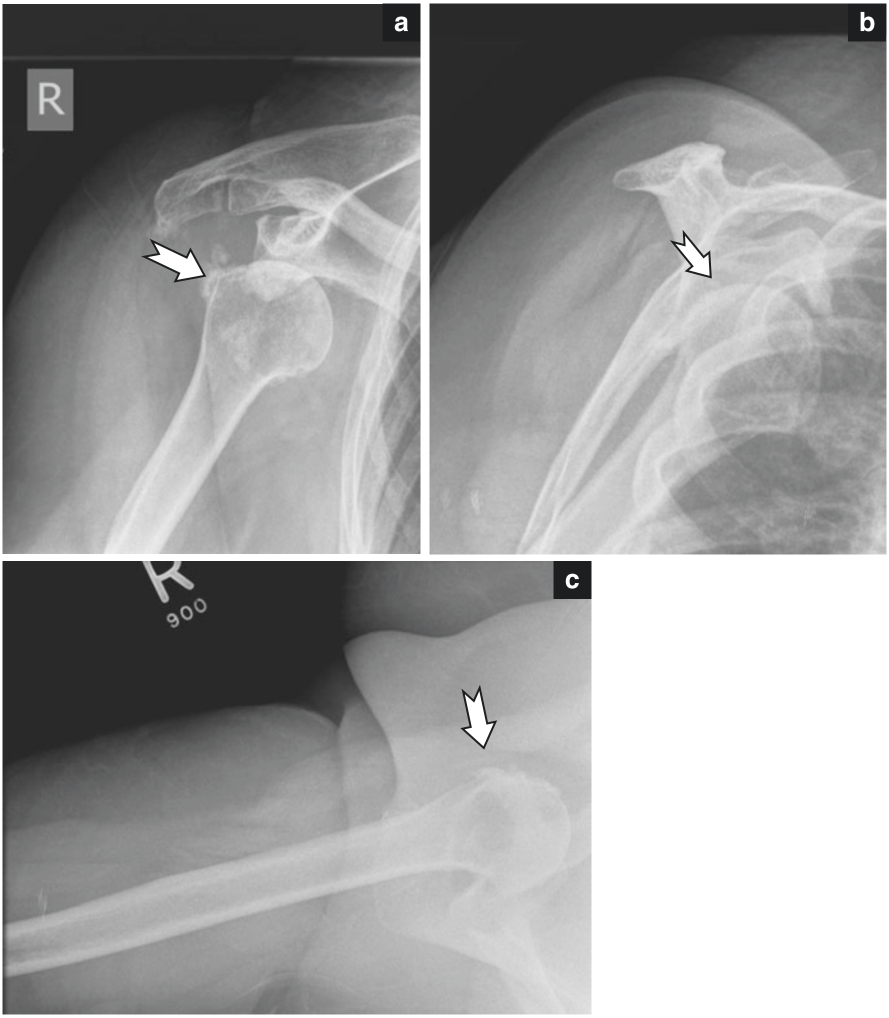

Fig. 12.14 Chronic anterior dislocation of the glenohumeral joint on standard AP view (**a**), transcapular (**b**) and axillary (**c**) views showing the humeral head (white arrow) is anteriorly dislocated with respect to the glenoid

Posterior Glenohumeral Instability

The incidence of posterior dislocation is approximately 5% of all dislocations [39]. Seizures are an important cause of posterior dislocation, and almost all bilateral posterior shoulder dislocations are due to a seizure or electrocution injury [53]. Posterior dislocation may be associated with reverse Bankart lesion, defined as the detachment of the posteroinferior labrum with avulsion of posterior capsular periosteum. Posterior dislocation may occasionally be undetected and become chronic.

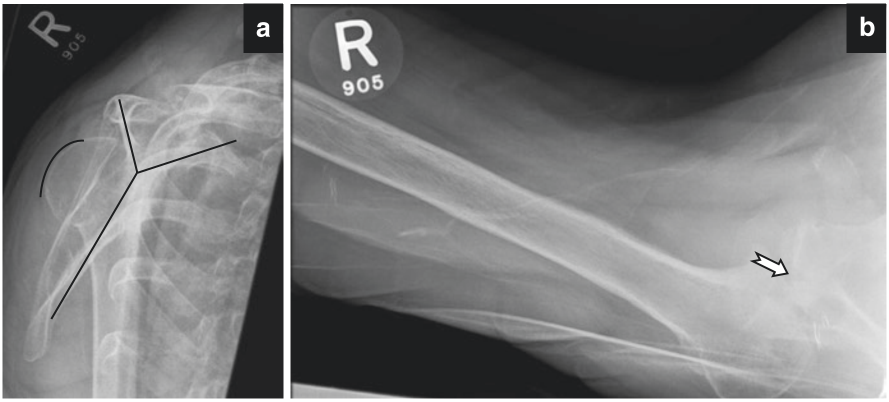

Fig. 12.15 Chronic posterior dislocation of the glenohumeral joint on transcapular (**a**) and axillary view (**b**) showing posterior Bankart fracture plus a deep reverse Hill-Sachs defect (anteromedial defect) in the proximal humerus

Acute or chronic posterior dislocation of the humeral head is best detected on the transcapular and axillary views. Figures 12.15a, b show chronic posterior dislocation with a posterior Bankart fracture and a reverse Hill-Sachs defect.

Imaging Features of Capsular, Labral, and Rare Pathologies

Adhesive Capsulitis

In patients with clinical signs of frozen shoulder regardless of the etiology, plain radiographs are indicated to rule out potential hidden pathologies such as septic arthritis, malignancy, or metastasis. Adhesive capsulitis and early to moderate osteoarthritis have similar clinical findings in younger patients but can be easily differentiated by plain radiographs. Patients with isolated adhesive capsulitis often have normal plain radiographs. Patients with chronic adhesive capsulitis may show radiological signs of osteopenia of the proximal humerus and slight superior migration of the humeral head secondary to dysfunction of the rotator cuff muscles [54, 55].

Adhesive capsulitis is primarily a clinical diagnosis, and US and MRI have only a supportive role in confirming the suspected diagnosis in clinically equivocal cases [56]. Apart from plain radiographs, dynamic US may be used to confirm refractory adhesive capsulitis. The thickened and rigid coracohumeral ligament (CHL) [57–59] and thickened rotator interval capsule [60, 61] are imaging hallmarks of adhesive capsulitis.

Magnetic resonance (MR) imaging is usually not the first choice for identifying adhesive capsulitis due to its cost and lack of availability. The primary signs of adhesive capsulitis on the MRI include thickening of the ligaments and joint capsule in the axillary recess and presence of scarring, synovitis, and inflammation in the rotator interval. MR arthrogram appears to be a more reliable mode of examination for adhesive capsulitis [61–63]. Characteristics of MR arthrographic findings are similar to non-contrast MRI [64], but decreased joint volume detected by painful and backflow of injected liquid and resistance to amount of injected fluid are highly suggestive of adhesive capsulitis [65–67]. It is important to note that when adhesive capsulitis is associated with a full-thickness rotator cuff tear, the injection volume may appear to be normal [68] as the injected contrast may escape from the glenohumeral joint via the torn cuff tendons [69].

Patients with immunocompromised conditions presenting with insidious onset of moderate to severe pain who have failed conservative treatment should be subjected to either ultrasound or MRI to avoid a delay in diagnosis of septic arthritis [70]. As an important note to advanced practice physiotherapists and primary care physicians, frozen shoulder secondary to serious conditions is often associated with a more alarming clinical history or clinical examination findings (e.g., history of previous malignancy, severe muscle wasting), and these clinical signs should be taken seriously. Missing malignancy as the cause of stiffness and management with cortisone injection or arthrographic distension of the shoulder have grave consequences. Such procedures may change the surgical management from being a limb-preserving resection to a forequarter amputation [71]. Therefore, importance of careful review of the plain radiographs in refractory frozen shoulder should not be underestimated. Plain AP and axillary radiographs of the shoulder should be performed as a routine to rule out the presence of any lytic lesions before corticosteroid injection.

Superior Labral Pathologies

Plain radiographs are not useful in diagnosing superior labral anteroposterior (SLAP) lesions and in isolated SLAP tears. Plain radiographs are usually normal and most often are taken to rule out other pathologies.

Ultrasound does not have a diagnostic value for lesions of the superior labrum with or without biceps tendon involvement [72]. Initially, standard MRI was the most utilized mode of imaging used to detect SLAP tears. In 1991, Legan et al. [73] reported that although standard MRI had a high sensitivity for detecting labral lesions, its sensitivity in identifying SLAP lesions was much lower. In 1996, Gusmer et al. [74] reported a sensitivity of 86% and specificity of 100% for detecting superior labral lesions.

Today, MR arthrography is considered the imaging of choice with arthroscopy being the gold standard for diagnosis of the SLAP lesions. In a systematic review of the literature in 2019, the pooled sensitivity and specificity of MR arthrogram were 0.92 and 0.98, respectively [75]. MR arthrography can be performed directly with an intra-articular injection or indirectly with intravenous injection [76]. A controlled distention of the joint assists with outlining of intra-articular and synovial surfaces.

The leakage of contrast through labral pathologies helps in the differentiation of SLAP lesions from the anatomic variants, such as sublabral recess and sublabral foramen [76]. An important secondary sign of labral tear is a paralabral cyst. A paralabral cyst may be the first indication of a labral tear and almost always means a labral tear is present. Other intra-articular diseases associated with labral pathology include rotator cuff tears, Bankart lesions, and glenohumeral chondromalacia [76] and need to be ruled out. In summary, the importance of differentiating normal age-related deterioration from truly unstable labral lesions is important [77]. In light of extreme anatomic variability of the superior labrum, primary care clinicians should consider patient's age, mechanism of injury, and nature of symptoms before ordering costly imaging investigations such as MR arthrogram.

Benign Bone Tumors

A significant number of benign tumors may grow in long bones, including the humerus. The most distinguished radiologic feature associated with these tumors is bone expansion with an intact rim of cortex and lack of periosteal reaction. Enchondroma is probably the most common benign tumor seen in the humerus which presents with no gross bone destruction, periosteal reaction, or soft tissue mass (Fig. 12.16).

Malignancy

Malignant tumors or metastasis to shoulder girdle is an uncommon cause of shoulder pain and stiffness but often presents with symptoms and a clinical presentation of primary adhesive capsulitis. As noted earlier, misdiagnosing

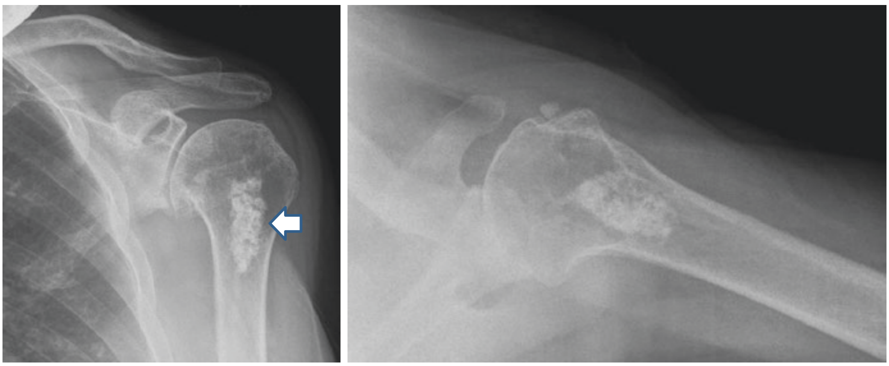

Fig. 12.16 True AP and axillary views of the glenohumeral joint, showing intramedullary calcification in the neck of the proximal humerus compatible with an enchondroma (white arrow). Osteoarthritic changes with joint space loss are noted in the glenohumeral articulation

Fig. 12.17 Standard AP view of a 59-year-old man showing a lytic lesion in the humeral head extending into the proximal humeral shaft with poorly defined distal margins. The cortex in the humeral head and medial aspect of the humeral neck is markedly thinned. The subsequent CT scan showed the metastasis of a tumor from the kidney (metastatic renal cell carcinoma) to the shoulder, lung, and brain which had gone undiagnosed up to the time of the plain radiographs of the shoulder

malignancy as the cause of stiffness and management with cortisone injection or arthrographic distension of the shoulder have grave consequences with changing surgery from a limb-preserving resection to a forequarter amputation [71]. Clinically, there is a complaint of deep bone pain, occasionally accompanied by a soft tissue mass or swelling with loss of motion and severe muscle wasting. In some cases, pathologic fracture may be the first radiological sign of malignancy. The radiologic signs of primary malignant tumors or metastatic lesions from other organs include medullary and cortical bone destruction, moth-eaten appearance in bone, described as multiple lucent lesions often with poorly defined margins and aggressive periosteal reaction. Fig. 12.17 shows a lytic lesion related to a metastatic renal cell carcinoma in a 59-year-old man who had 6 months of unsuccessful physiotherapy. This man had significant muscle wasting and marked stiffness at the time of visit with the clinician.

References

1. Kohan EM, Hill JR, Lamplot JD, Aleem AW, Keener JD, Chamberlain AM. Severity of glenohumeral osteoarthritis does not correlate with patient-reported outcomes. J Shoulder Elb Arthroplast. 2020;
2. Kircher J, Morhard M, Magosch P, Ebinger N, Lichtenberg S, Habermeyer P. How much are radiological parameters related to clinical symptoms and function in osteoarthritis of the shoulder? Int Orthop. 2010;34(5):677–81.
3. Tauber M, Martetschlager F. Shoulder Osteoarthritis-pathogenesis, classification, diagnostics and treatment. Orthopade. 2019;48(9):795–808.
4. Roche CP, Diep P, Grey SG, Flurin PH. Biomechanical impact of posterior glenoid wear on anatomic total shoulder arthroplasty. Bull Hosp Jt Dis. 2013;2013(71 Suppl 2):S5-11.
5. Samilson RL, Prieto V. Dislocation arthropathy of the shoulder. J Bone Joint Surg Am. 1983;65(4):456–60.
6. Ilg A, Bankes MJ, Emery RJ. The intra- and inter-observer reliability of the Samilson and Prieto grading system of glenohumeral arthropathy. Knee Surg Sports Traumatol Arthrosc. 2001;9(3):187–90.
7. Walch G, Badet R, Boulahia A, Khoury A. Morphologic study of the glenoid in primary glenohumeral osteoarthritis. J Arthroplast. 1999;14(6):756–60.
8. Chan K, Knowles NK, Chaoui J, Gauci MO, Ferreira LM, Walch G, et al. Characterization of the Walch B3 glenoid in primary osteoarthritis. J Shoulder Elb Surg. 2017;26(5):909–14.
9. Domos P, Checchia CS, Walch G. Walch B0 glenoid: pre-osteoarthritic posterior subluxation of the humeral head. J Shoulder Elb Surg. 2018;27(1):181–8.
10. Walker KE, Simcock XC, Jun BJ, Iannotti JP, Ricchetti ET. Progression of glenoid morphology in glenohumeral osteoarthritis. J Bone Joint Surg Am. 2018;100(1):49–56.
11. Donohue KW, Ricchetti ET, Ho JC, Iannotti JP. The association between rotator cuff muscle fatty infiltration and glenoid morphology in glenohumeral osteoarthritis. J Bone Joint Surg Am. 2018;100(5):381–7.
12. Martel W, Hayes JT, Duff IF. The pattern of bone erosion in the hand and wrist in rheumatoid arthritis. Radiology. 1965;84:204–14.
13. Lévigne C, Franceschi JP. Rheumatoid arthritis of the shoulder: radiological presentation and results of arthroplasty. In: Walch G, Boileau P, editors. Shoulder arthroplasty. Berlin, Germany: Springer; 1999. p. 221–30.
14. Lehtinen JT, Lehto MU, Kaarela K, Kautiainen HJ, Belt EA, Kauppi MJ. Radiographic joint space in rheumatoid acromioclavicular joints: a 15 year prospective follow-up study in 74 patients. Rheumatology (Oxford). 1999;38(11):1104–7.
15. Lehtinen JT, Lehto MU, Kaarela K, Kautiainen HJ, Belt EA, Kauppi MJ. Radiographic joint space in rheumatoid glenohumeral joints. A 15-year prospective follow-up study in 74 patients. Rheumatology (Oxford). 2000;39(3):288–92.
16. Lehtinen JT, Belt EA, Lyback CO, Kauppi MJ, Kaarela K, Kautiainen HJ, et al. Subacromial space in the rheumatoid shoulder: a radiographic 15-year follow-up study of 148 shoulders. J Shoulder Elb Surg. 2000;9(3):183–7.
17. Lukas C, van der Heijde D, Fatenajad S, Landewe R. Repair of erosions occurs almost exclusively in damaged joints without swelling. Ann Rheum Dis. 2010;69(5):851–5.
18. Neer CS 2nd, Craig EV, Fukuda H. Cuff-tear arthropathy. J Bone Joint Surg Am. 1983;65(9):1232–44.
19. McCarty DJ, Halverson PB, Carrera GF, Brewer BJ, Kozin F. "Milwaukee shoulder"—association of microspheroids containing hydroxyapatite crystals, active collagenase, and neutral protease with rotator cuff defects. I. Clinical aspects. Arthritis Rheum. 1981;24(3):464–73.
20. 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.

21. Nove-Josserand L, Walch G, Adeleine P, Courpron P. Effect of age on the natural history of the shoulder: a clinical and radiological study in the elderly. Rev Chir Orthop Reparatrice Appar Mot. 2005;91(6):508–14.
22. Brolin TJ, Updegrove GF. Department of Orthopaedic Surgery, The Rothman Institute, Thomas Jefferson University Hospital, Philadelphia, PA, U.S.A., Horneff JG. Classifications in brief: Hamada classification of massive rotator cuff tears. Clin Orthop Relat Res 2017; 475(11):2819–2823.
23. Visotsky JL, Basamania C, Seebauer L, Rockwood CA, Jensen KL. Cuff tear arthropathy: pathogenesis, classification, and algorithm for treatment. J Bone Joint Surg Am. 2004;86-A(Suppl 2):35–40.
24. Hamada K, Yamanaka K, Uchiyama Y, Mikasa T, Mikasa M. A radiographic classification of massive rotator cuff tear arthritis. Clin Orthop Relat Res. 2011;469(9):2452–60.
25. Werner CM, Nyffeler RW, Jacob HA, Gerber C. The effect of capsular tightening on humeral head translations. J Orthop Res. 2004;22(1):194–201.
26. Cruess RL. The current status of avascular necrosis of the femoral head. Clin Orthop Relat Res. 1978;131:309–11.
27. Brown TD, Baker KJ, Brand RA. Structural consequences of subchondral bone involvement in segmental osteonecrosis of the femoral head. J Orthop Res. 1992;10(1):79–87.
28. Pappas JN. The musculoskeletal crescent sign. Radiology. 2000;217(1):213–4.
29. Zhang N, Qi S, Chai J. Mechanism of "crescent sign" formation in avascular necrosis of femoral head. Zhongguo Xiu Fu Chong Jian Wai Ke Za Zhi. 2008;22(3):303–6.
30. Gillespy T 3rd, Genant HK, Helms CA. Magnetic resonance imaging of osteonecrosis. Radiol Clin N Am. 1986;24(2):193–208.
31. Beltran J, Herman LJ, Burk JM, Zuelzer WA, Clark RN, Lucas JG, et al. Femoral head avascular necrosis: MR imaging with clinical-pathologic and radionuclide correlation. Radiology. 1988;166(1 Pt 1):215–20.
32. Mitchell MD, Kundel HL, Steinberg ME, Kressel HY, Alavi A, Axel L. Avascular necrosis of the hip: comparison of MR, CT, and scintigraphy. AJR Am J Roentgenol. 1986;147(1):67–71.
33. Graif M, Schweitzer ME, Deely D, Matteucci T. The septic versus nonseptic inflamed joint: MRI characteristics. Skelet Radiol. 1999;28(11):616–20.
34. Bierry G, Huang AJ, Chang CY, Torriani M, Bredella MA. MRI findings of treated bacterial septic arthritis. Skelet Radiol. 2012;
35. Duncan SF, Sperling JW. Treatment of primary isolated shoulder sepsis in the adult patient. Clin Orthop Relat Res. 2008;466(6):1392–6.
36. Atalar AC, Sungur M, Demirhan M, Ozger H. Neuropathic arthropathy of the shoulder associated with syringomyelia: a report of six cases. Acta Orthop Traumatol Turc. 2010;44(4):328–36.
37. Li H, George DM, Jaarsma RL, Mao X. Metabolic syndrome and components exacerbate osteoarthritis symptoms of pain, depression and reduced knee function. Ann Transl Med. 2016;4(7):133.
38. Bao H, Qi Y, Wei B, Ma B, Wang Y, Xu Y. Severe erosive lesion of the glenoid in gouty shoulder arthritis: a case report and review of the literature. BMC Musculoskelet Disord. 2021;22(1):343.
39. Matsen FAI, Thomas SC, Rockwood CA, editors. The shoulder, glenohumeral instability. 2nd ed. Philadelphia: W. B. Saunders; 1990.
40. Rouhani A, Navali A. Treatment of chronic anterior shoulder dislocation by open reduction and simultaneous Bankart lesion repair. Sports Med Arthrosc Rehabil Ther Technol. 2010;2:15.
41. Mansat P, Guity MR, Mansat M, Bellumore Y, Rongieres M, Bonnevialle P. Chronic anterior shoulder dislocation treated by open reduction sparing the humeral head. Rev Chir Orthop Reparatrice Appar Mot. 2003;89(1):19–26.
42. Rowe CR, Zarins B. Chronic unreduced dislocations of the shoulder. J Bone Joint Surg Am. 1982;64(4):494–505.
43. Yanmis I, Komurcu M, Oguz E, Basbozkurt M, Gur E. The role of arthroscopy in chronic anterior shoulder dislocation: technique and early results. Arthroscopy. 2003;19(10):1129–32.

44. Calandra JJ, Baker CL, Uribe J. The incidence of Hill-Sachs lesions in initial anterior shoulder dislocations. Arthroscopy. 1989;5(4):254–7.
45. Taylor DC, Arciero RA. Pathologic changes associated with shoulder dislocations. Arthroscopic and physical examination findings in first-time, traumatic anterior dislocations. Am J Sports Med. 1997;25(3):306–11.
46. Antonio GE, Griffith JF, Yu AB, Yung PS, Chan KM, Ahuja AT. First-time shoulder dislocation: high prevalence of labral injury and age-related differences revealed by MR arthrography. J Magn Reson Imaging. 2007;26(4):983–91.
47. Hintermann B, Gachter A. Arthroscopic findings after shoulder dislocation. Am J Sports Med. 1995;23(5):545–51.
48. Cetik O, Uslu M, Ozsar BK. The relationship between Hill-Sachs lesion and recurrent anterior shoulder dislocation. Acta Orthop Belg. 2007;73(2):175–8.
49. Kaar SG, Fening SD, Jones MH, Colbrunn RW, Miniaci A. Effect of humeral head defect size on glenohumeral stability: a cadaveric study of simulated Hill-Sachs defects. Am J Sports Med. 2010;38(3):594–9.
50. Cho SH, Cho NS, Rhee YG. Preoperative analysis of the Hill-Sachs lesion in anterior shoulder instability: how to predict engagement of the lesion. Am J Sports Med. 2011;39(11):2389–95.
51. Burkhart SS, De Beer JF. 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.
52. Horst K, Von Harten R, Weber C, Andruszkow H, Pfeifer R, Dienstknecht T, et al. Assessment of coincidence and defect sizes in Bankart and Hill-Sachs lesions after anterior shoulder dislocation: a radiological study. Br J Radiol. 2014;87(1034):20130673.
53. Rouleau DM, Hebert-Davies J. Incidence of associated injury in posterior shoulder dislocation: systematic review of the literature. J Orthop Trauma. 2011;
54. Okamura K, Ozaki J. Bone mineral density of the shoulder joint in frozen shoulder. Arch Orthop Trauma Surg. 1999;119(7–8):363–7.
55. Lundberg BJ, Nilsson BE. Osteopenia in the frozen shoulder. Clin Orthop Relat Res. 1968;60:187–91.
56. Petchprapa CN, Beltran LS, Jazrawi LM, Kwon YW, Babb JS, Recht MP. The rotator interval: a review of anatomy, function, and normal and abnormal MRI appearance. AJR Am J Roentgenol. 2010;195(3):567–76.
57. Fitzpatrick MJ, Powell SE, Tibone JE, Warren RF. The anatomy, pathology, and definitive treatment of rotator interval lesions: current concepts. Arthroscopy. 2003;19(Suppl 1):70–9.
58. Neer CS 2nd, Satterlee CC, Dalsey RM, Flatow EL. The anatomy and potential effects of contracture of the coracohumeral ligament. Clin Orthop Relat Res. 1992;280:182–5.
59. Shaffer B, Tibone JE, Kerlan RK. Frozen shoulder. A long-term follow-up. J Bone Joint Surg Am. 1992;74(5):738–46.
60. Homsi C, Bordalo-Rodrigues M, da Silva JJ, Stump XM. Ultrasound in adhesive capsulitis of the shoulder: is assessment of the coracohumeral ligament a valuable diagnostic tool? Skelet Radiol. 2006;35(9):673–8.
61. Mengiardi B, Pfirrmann CW, Gerber C, Hodler J, Zanetti M. Frozen shoulder: MR arthrographic findings. Radiology. 2004;233(2):486–92.
62. Lefevre-Colau MM, Drape JL, Fayad F, Rannou F, Diche T, Minvielle F, et al. Magnetic resonance imaging of shoulders with idiopathic adhesive capsulitis: reliability of measures. Eur Radiol. 2005;15(12):2415–22.
63. Connell D, Padmanabhan R, Buchbinder R. Adhesive capsulitis: role of MR imaging in differential diagnosis. Eur Radiol. 2002;12(8):2100–6.
64. Gokalp G, Algin O, Yildirim N, Yazici Z. Adhesive capsulitis: contrast-enhanced shoulder MRI findings. J Med Imaging Radiat Oncol. 2011;55(2):119–25.
65. Yilmaz MH, Kantarci F, Adaletli I, Ulus S, Gulsen F, Ozer H, et al. Pain & resistance in patients with adhesive capsulitis during contrast material injection phase of MR arthrography. Indian J Med Res. 2007;125(4):572–6.

66. Hall FM. Frozen shoulder. Radiology. 2005;235(2):713–4. author reply 714
67. Lee MH, Ahn JM, Muhle C, Kim SH, Park JS, Kim SH, et al. Adhesive capsulitis of the shoulder: diagnosis using magnetic resonance arthrography, with arthroscopic findings as the standard. J Comput Assist Tomogr. 2003;27(6):901–6.
68. Hall FM. MR arthrography of the posterior labrocapsular complex. AJR Am J Roentgenol. 2003;181(2):595. author reply 595-6
69. Mengiardi B. Frozen shoulder: MR arthrographic findings. Author reply. Radiology. 2005;235:713–4.
70. Sambandam SN, Atturu M. A case of septic arthritis of shoulder presenting as stiffness of the shoulder. J Orthop Case Rep. 2016;6(2):31–3.
71. Quan GM, Carr D, Schlicht S, Powell G, Choong PF. Lessons learnt from the painful shoulder; a case series of malignant shoulder girdle tumours misdiagnosed as frozen shoulder. Int Semin Surg Oncol. 2005;2(1):2-7800-2-2.
72. Zanetti M, Pfirrmann CW. Biceps tendon disorders: ultrasound, MR imaging and MR arthrography. Radiologe. 2004;44(6):591–6.
73. Legan JM, Burkhard TK, Goff WB 2nd, Balsara ZN, Martinez AJ, Burks DD, et al. Tears of the glenoid labrum: MR imaging of 88 arthroscopically confirmed cases. Radiology. 1991;179(1):241–6.
74. Gusmer PB, Potter HG, Schatz JA, Wickiewicz TL, Altchek DW, O'Brien SJ, et al. Labral injuries: accuracy of detection with unenhanced MR imaging of the shoulder. Radiology. 1996;200(2):519–24.
75. Liu F, Cheng X, Dong J, Zhou D, Sun Q, Bai X, et al. Imaging modality for measuring the presence and extent of the labral lesions of the shoulder: a systematic review and meta-analysis. BMC Musculoskelet Disord. 2019;20(1):487-019-2876-6.
76. Chang D, Mohana-Borges A, Borso M, Chung CB. SLAP lesions: anatomy, clinical presentation, MR imaging diagnosis and characterization. Eur J Radiol. 2008;68(1):72–87.
77. Hester WA, O'Brien MJ, Heard WMR, Savoie FH. Current concepts in the evaluation and management of Type II superior labral lesions of the shoulder. Open Orthop J. 2018;12:331–41.

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H. Razmjou, M. Christakis, *Clinical and Radiological Examination of the Shoulder Joint*, https://doi.org/10.1007/978-3-031-10470-1