

5

Imaging Diagnosis of Rotator Cuff Pathology and Impingement Syndromes

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5.1 Introduction

Rotator cuff disease, which includes tendinopathy and tearing, is incredibly common. A systematic review in 2014 has shown that the prevalence of rotator cuff disease increases with age, from approximately 10% in patients under 20 years of age to approximately 60% in patients greater than 80 years of age [1]. There are a number of controversies that exist when discussing the rotator cuff, including symptomatology and pathoetiology. Although it is clear that cuff disease can be symptomatic and necessitate treatment, the determination of which abnormalities are symptomatic or which are best treated with surgical intervention remains a challenge [2–6].

The etiology of rotator cuff disease is multifactorial with intrinsic and extrinsic contributions [7, 8]. Intrinsic mechanisms are associated with the tendon itself and the *degenerativemicrotrauma model* is likely to be critical to the development of cuff disease in many patients [9]. This model supposes that age-related tendon damage [10, 11] compounded by chronic, repetitive microtrauma results in adverse cellular changes, release of inflammatory mediators, and apoptosis [12, 13]. Extrinsic mechanisms include anatomic variables external to the tendon, such as the various impingement syndromes.

In most patients, it is generally favored that intrinsic mechanisms play a greater role in cuff disease compared with extrinsic factors [14–18]. This is referred to as the intrinsic theory of cuff disease, which states that cuff dysfunction is the causal abnormality, leading to decentering of the humeral lead and resultant formation of enthesophytes and tuberosity lesions [19]. Although biologically engineered scaffolds [20], exogenous growth factors [21], and cellular therapies [22] targeting intrinsic mechanisms are increasing, surgical therapy of cuff disease and treatment of associated extrinsic lesions remain the most widely available nonconservative treatment options. Therefore, it is critical for the radiologist and surgeon to identify the lesions that can be associated with shoulder pain and cases which may be amenable to available treatment.

The diagnosis of impingement syndromes requires all available information, including history, physical examination, and imaging. A practical and commonly used classification scheme of the various shoulder impingement syndromes is to divide based on those where the pathogenesis resides outside the glenohumeral joint capsule (termed external impingement) and those residing inside the glenohumeral joint capsule (termed internal impingement). External impingement syndromes include subacromial impingement and subcoracoid impingement. Internal impingeinclude ment syndromes posterosuperior

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impingement, which belongs in the spectrum of abnormalities leading to the disabled throwing shoulder, and *anterosuperior impingement*. Each impingement syndrome is a distinct entity, predominantly affecting different demographics of patients, but more than one type of impingement syndrome may be seen in an individual.

This chapter reviews (1) the imaging anatomy of the structures related to impingement, including the rotator cuff and biceps pulley; (2) the multi-modality imaging manifestations of rotator cuff disease and the various shoulder impingement syndromes; and (3) the expected and abnormal appearances after surgical therapy.

5.2 Imaging Anatomy

The supraspinatus, infraspinatus, teres minor, and subscapularis contribute to the rotator cuff. The rotator cuff is composed of approximately 75% water and the dry weight composition is approximately 67% type I collagen [23] and 1-5% proteoglycan/glycosaminoglycan [24]. The rotator cuff ultrastructure is complex, with up to five distinct layers that are visible with histological evaluation [25] or MR imaging [26, 27].

An important component of the rotator cuff is the *rotator cable* [28]. Of note, the term *rotator cable* is most commonly utilized in the radiological literature; however the same structure has been described under different names, including the ligamentum semicirculare humeri [29, 30], the transverse band [25], and the circular fiber system [31]. The rotator cable has been described to be an extension of fibrous tissue extending through the rotator interval, which has been termed the coracohumeral ligament (CHL) [25, 32] or the coracoglenohumeral ligament (CGHL) [30] (Fig. 5.1a). The differences in terminology reflect the different perspectives of the dense connective tissue in the rotator interval. While some consider the CHL and superior glenohumeral ligament (SGHL) as separate structures, others have suggested that these structures be considered a single functional unit, called either the CHL (with the SGHL representing a limited portion of this structure) [32] or the CGHL [30].

Burkhart et al. outlined the function of the rotator cable-crescent complex in 1993 [28]. Much like a suspension bridge, the rotator cuff and cable have anterior and posterior supporting limbs, represented by the anterior attachment of the supraspinatus tendon and the posterior attachment of the infraspinatus tendon, respectively. Tears that occur in the thinner, crescentic portion of the cuff between the two intact limbs are felt to be stress-shielded by the cable, explaining why some cuff tears may be less biomechanically significant [28]. In contrast, tears of the rotator cable itself or of the supporting limbs can have dire biomechanical consequences and should be considered for early repair [33–36]. While the rotator cable is consistently identified on anatomic dissections and at surgery [28, 37, 38], it can be seen frequently but not invariably on imaging [37–40]. This may be due to the less conspicuous appearance on imaging (Fig. 5.1b).

The deepest layer of the rotator cuff is the glenohumeral joint capsule [25]. Although previously thought to be only 1-2 mm thick [25], Nimura et al. found a much more substantial contribution of the capsule to the rotator cuff, representing more than half the total tendon width at some locations. According to Nimura et al., the minimum capsular width was 3.5 mm, located near the posterior portion of the supraspinatus footprint, and this was suspected to represent the crescent [41]. The joint capsule was found to be thickest at the anterior margin of the greater tuberosity and posterior margin of the infraspinatus tendon, measuring 5.6 and 9.1 mm on average, respectively [42]. These are believed to represent the greater tuberosity attachment sites of the rotator cable [41].

Our understanding of anatomy pertinent to each rotator cuff muscle and tendon continues to evolve. Classic descriptions in standard anatomical textbooks [43, 44] are now known to be inaccurate or incomplete since significant contributions to the literature have occurred within the last decade. Each cuff component has unique anatomical considerations that are important to biomechanical function. This is particularly relevant to the radiologist for diagnosis and to the orthopedic surgeon for anatomic



Fig. 5.1 Left shoulder of a cadaveric specimen (94-yearold man). (a) Photograph of dissection, viewed from anterosuperior, after reflection of the rotator cuff and capsule shows a distinct rotator cable (thick arrows), which is a continuation of the coracohumeral ligament (black dashed arrow). The superior glenohumeral ligament (black arrow) inserts onto the fovea capitis of the humerus.

Diffuse chondrosis is present over the humeral head. (b) Coronal oblique intermediate-weighted MR image of the same specimen shows the rotator cable as a thickening of the deep surface of the supraspinatus tendon (thick white arrow), which is less apparent compared with the gross image. Dissected specimen is imaged in air, which appears black in the image

restoration. Pertinent soft tissue and osseous anatomy for each component is further described below.

5.2.1 Supraspinatus

The supraspinatus muscle originates from supraspinous fossa as well as the superior surface of the scapular spine and is composed of distinct anterior and posterior muscle bellies. The anterior muscle belly is approximately 3–6 times larger and also demonstrates a larger variation of pennation angles compared with the posterior belly [45, 46]. The greater force generation and different contraction forces present within the anterior belly may explain the higher incidence of anterior tendon tears [45, 47].

The anterior belly gives rise to a longer, cordlike tendon whereas the posterior belly gives rise to a shorter, quadrangular shaped tendon [45] (Fig. 5.2). The humeral attachment of the rotator cuff tendon is frequently referred to as the *footprint*, a term coined by Tierney et al. in 1999 [48]. The footprint of the supraspinatus was first delineated by Minagawa et al. [49], but has subsequently been redefined and refined several times. Our current knowledge of the supraspinatus footprint is that it predominantly occupies the anteromedial portion of the superior facet (or highest impression) of the greater tuberosity and is triangular or trapezoidal in shape [50, 51]. The lateral-most attachment extends over the lip of the greater tuberosity [48]. Anatomic studies have shown that in approximately a quarter of specimens, fibers from the anterior tendon of the supraspinatus cover the bicipital groove and attach to the lesser tuberosity [50, 51]. Moser et al. described an "aponeurotic expansion" of the anterior supraspinatus tendon, coursing anterior and lateral to the long head of the biceps tendon, inserting distally onto the pectoralis major tendon and evident in approximately half of their cadaveric shoulders and clinical cases [52, 53]. According to Moser et al., this same structure has been previously mistermed a fourth head of the pectoralis major [54] and an accessory biceps tendon [55–57]. Precise delineation of the anatomy in the anterosuperior aspect of the shoulder requires reconciliation of the rapidly evolving anatomical, surgical, and imaging literature.

The dimensions of the cuff footprint are clinically relevant since partial-thickness tears should be graded as low, moderate, or high grade based



Fig. 5.2 Anatomy and pathology of the anterior muscle belly of the supraspinatus in a 50-year-old man. (**a** and **b**) Sagittal oblique T1-weighted and T2-weighted fat-suppressed MR images, respectively, show calcium hydroxyapatite deposition in the supraspinatus tendon near the footprint (thick arrow). More medially, cordlike tendon of anterior belly is evident (open arrow). Mild subacro-

on depth [58] and anatomic restoration in the setting of repair requires knowledge of the footprint. Unfortunately, reported cuff footprint dimensions have varied widely in the literature, likely due to a combination of variables, including the precise delineation of the boundaries of the footprint, differences in degrees of capsular dissection from the tendon [42], as well as individual variation such as age, gender, patient size, and race. For instance, Curtis et al. described that the supraspinatus tendon extends over the lateral lip of the greater tuberosity [48]; however it is likely that the authors were measuring a portion of the infraspinatus footprint onto what is now called the *lateral facet* [59] (discussed in further detail below). On cadaveric studies, mean supraspinatus footprint width (medial-lateral dimension) has been reported to vary considerably, ranging from 6.7 to 16 mm [42, 46, 48, 50, 51, 60, 61]. Based on the current concept that the

mial-subdeltoid bursitis was present (not shown). (**c** and **d**) 4 years later, calcium hydroxyapatite had migrated towards the myotendinous junction of the anterior belly with surrounding edema (thin arrow) which separates the tendons of the anterior and posterior muscle bellies of the supraspinatus. (**e**) Concurrent radiograph confirms intra-tendinous migration of crystals (thin arrows)

supraspinatus footprint is not as large as previously described, mean length (anterior-posterior dimension) measures approximately 20.9–32 mm medially and 1.3–6.4 mm laterally [50, 51]. In contrast to gross measurements, there is a paucity of imaging-based tendon measurements, which some may argue would be the most useful for clinical practice. Karthiekeyan et al. [62] performed ultrasound-based measurements in 120 young healthy shoulders and found mean supraspinatus footprint widths of 14.9 mm in men and 13.5 mm in women. In the same study, mean supraspinatus tendon thickness was 5.6 mm in men and 4.9 mm in women.

5.2.2 Infraspinatus

The infraspinatus muscle originates from the infraspinous fossa as well as the inferior surface

of the scapular spine and is composed of two distinct portions. The *oblique* (or inferior) portion is approximately four to five times larger than the *transverse* (or superior) portion [63, 64]. The infraspinatus tendon attaches to the greater tuberosity and, similar to the supraspinatus, the footprint has also been redefined and refined several times in recent years. Kato et al. demonstrated that the footprint is entirely composed of the tendon arising from the oblique portion and that the tendon of the transverse portion is membrane-like and attaches to the posterior surface of the tendinous portion of the oblique part [63, 64].

The greater tuberosity footprint of the oblique portion is larger than what has been historically described. Standard anatomical textbooks recognize three facets (or impressions) of the greater tuberosity: superior (or horizontal), middle (or oblique), and inferior (or vertical) [43, 44]. More recent studies have suggested that the infraspinatus footprint occupies the entire middle facet and approximately half of the superior facet [50, 51]. However, in 2015, Nozaki et al. proposed a fourth facet (or impression) of the greater tuberosity, which they termed the *lateral* facet (Fig. 5.3) [59]. The lateral facet is triangular in shape, variable in size, located posterolateral to the superior facet, and was recognized in all 87 specimens of their study. The authors demonstrated that the anterior extent of the infraspinatus footprint is onto the lateral facet. The orientation of the facets of the humeral tuberosities is related to rotator cuff muscle function and may represent an anatomical factor involved in pathogenesis of rotator cuff tears [65, 66]. Le Corroller et al. demonstrated that a decrease in dorsal orientation of the middle facet in the sagittal plane was associated with higher likelihood of cuff tearing [65].

Similar to the supraspinatus tendon, the reported infraspinatus footprint dimensions have varied widely in the literature. On cadaveric studies, mean infraspinatus footprint width (medial-lateral dimension) has been reported to range from 6.9 to 15.1 mm [42, 48, 50, 51, 60]. Based on the current concept that the footprint of the infraspinatus occupies the entire middle facet and approximately half of the superior facet (or lateral facet), the mean length (anterior-posterior dimension) measures 22.9 mm medially and 25.6–32.7 mm laterally [50, 51]. Of note, Mochizuki et al. found a far anterolateral extent of the infraspinatus footprint, with mean distance between the most anterior edge of the footprint and the bicipital groove measuring 1.3 mm [51]. Lumsdaine et al. found a greater mean distance between the most anterior edge of the infraspinatus footprint and the bicipital groove, measuring 6.4 mm [50]. The differences may be due to ethnic variation since Mochizuki et al. used 128 shoulders from Japanese donors whereas Lumsdaine et al. used 54 shoulders from Australian Caucasoid donors. Using ultrasound on young healthy shoulders, Karthiekeyan et al. [62] found that the mean thickness of the infraspinatus tendon measures 4.9 mm in men and 4.4 mm in women. Michelin et al. measured a mean infraspinatus tendon thickness of 2.2 mm on MRI [67] and 2.4 mm on ultrasound [68].

5.2.3 Teres Minor

The teres minor muscle originates from the middle portion of the lateral edge of the scapula and a variable dense fascia of the infraspinatus muscle [69]. At the myotendinous junction, the teres minor appears as superior and inferior bundles [70]. The superior bundle originates from the lateral edge of the scapula and inserts onto the inferior facet as an oval footprint. The inferior bundle originates from both the lateral edge of the scapula and a dense fascial septum between the infraspinatus and teres minor muscles, and attaches as a band to the surgical neck of the humerus. Saji et al. dissected seven shoulders and found that the dense fascia was aplastic in one case. In the setting of an absent fascia, the teres muscle extends to cover the infraspinatus and the borders between



Fig. 5.3 30-Year-old man with a large lateral facet of the greater tuberosity as described by Nozaki et al. (**a** and **b**) Volume-rendered CT images shows the lateral facet in profile (**a**, thick arrow) and en face (**b**, dashed outline). Superior (arrowhead) and middle (open arrow) facets are marked. (**c** and **d**) Coronal oblique CT and T1-weighted fat-suppressed MR arthrogram images show the large lat-

the infraspinatus and teres minor muscles can be difficult to identify on MR imaging [71] (Fig. 5.4).

Similar to the rest of the cuff, reported mean dimensions of the footprint vary widely in the literature. On cadaveric studies, mean width (medial-lateral dimension) ranges from 11.4 to 21 mm and mean length (superior-inferior dimension) ranges from 20.7 to 29 mm [48, 60].

eral facet (thick arrows), which is located posterolateral to the superior facet and represents the anterior infraspinatus footprint and the bursal side of the cuff at this location. Also evident is moderate-grade partial-thickness articular sided tearing of the supraspinatus tendon (dashed arrow) and posterosuperior labral tearing with adjacent chondral damage (thin arrow)

5.2.4 Subscapularis

The subscapularis muscle originates from the medial two-thirds of the anterior surface of the scapula [72]. The superior two-thirds of the subscapularis muscle transitions to tendon at the level of the glenoid and blends with joint capsule fibers before inserting onto the lesser tuberosity [73, 74]. The inferior one-third is the so-called



Fig. 5.4 Anatomic variations and pathology of the teres minor muscle. (**a**) Sagittal oblique T1-weighted image of a 21-year-old woman with well-delineated infraspinatus (thick arrow) and teres minor (thin arrow) muscles. (**b**) Sagittal oblique T1-weighted image of a 23-year-old woman with an indistinct boundary between the infraspinatus (thick arrow) and teres minor (thin arrow) muscles, indicating a hypoplastic fascial septum. (**c** and **d**) Sagittal

and coronal oblique T1-weighted images in a 53-year-old man with selective atrophy of the superior bundle of the teres minor muscle. An oval-shaped tendon arises from the atrophic superior muscle bundle (open arrow) and attaches onto the inferior facet of the greater tuberosity. The normal inferior bundle attaches onto the posterior aspect of the surgical neck of the humerus (arrowhead)

muscular insertion, attaching onto the surgical neck of the humerus via a thin, membranous structure [66, 73, 74].

Similar to the rest of the cuff, our knowledge of the subscapularis tendon and footprint continues to evolve. The subscapularis tendon is composed of several smaller intramuscular tendons and the superior-most insertion is a thin slip, which attaches to the fovea capitis of the humerus [70, 73] (Fig. 5.5). Many authors have found that the superior glenohumeral ligament also attaches to the fovea capitis [75–77], although some have



Fig. 5.5 Subscapularis anatomy and pathology. (a) Left cadaveric shoulder specimen (same specimen shown in Fig. 5.1, viewed from anterosuperior), after the subscapularis tendon was cut and reflected (thick arrows), shows the articular side of the tendon. Both the superior glenohumeral ligament and superior-most subscapularis tendon insert onto the fovea capitis of the humerus (black arrow). The rotator cable is less apparent than in Fig. 5.1 due to the far reflection of the superior cuff. (b) Volume-rendered CT image of the left shoulder of a 34-year-old man demonstrates the four facets of the subscapularis footprint (F1-F4) as described by Yoo et al. [66] as well as superior-most tendon fibers which

insert onto the fovea capitis as described by Arai et al. [73] (dashed arrow). (c) Coronal oblique T2-weighted fat-suppressed MR image of a 24-year-old man shows an intact subscapularis tendon (thick arrows) inserting onto the top two facets of the lesser tuberosity (LT). Greater tuberosity (GT) is marked. (d) Coronal oblique T2-weighted fat-suppressed MR image of a 46-year-old man shows a tear of subscapularis tendon, which involves the superior-most tendon fibers and first two facet attachments. Tear is of full thickness at the first facet (disrupted from articular side through lateral hood, thin arrows) and partial thickness at the second facet (arrowhead) suggested that the superior glenohumeral ligament attaches to the tendinous slip of the subscapularis instead [78].

Although prior authors have described the footprint of the subscapularis to be shaped as a comma [48, 79] or the state of Nevada [74], a study in 2015 [66] has found that the footprint is best evaluated from a three-dimensional perspective. In a cadaveric and clinical study, Yoo et al. described the three-dimensional footprint anatomy, which consists of four bony facets [66]. The superior-most facet consists of approximately one-third of the entire footprint and the top two facets consist of 60% of the entire footprint. The third and fourth facets represent the so-called *muscular* insertion onto the surgical neck of the humerus (Fig. 5.5b).

Similar to the rest of the cuff, reported mean dimensions of the footprint vary; however, based on cadaveric studies, the mean width (medial-lateral dimension) ranges from 15 to 26 mm and mean length (superior-inferior dimension) ranges from 18 to 24 mm [48, 60, 74, 79]. Yoo et al. [66] found a mean width of 13.5 mm and a combined mean length of 51.5 mm; however their measurements were oblique relative to the standard imaging planes used with imaging, and therefore cannot be directly compared using CT or MRI. Based on

studies that have evaluated the mean widths of both supraspinatus and subscapularis tendon footprints [48, 60, 66], a practical guideline is that the superior aspect of the subscapularis footprint should be approximately 25–40% greater than the supraspinatus footprint. Using ultrasound, mean subscapularis tendon thickness has been described to be 4.4 mm in men and 3.8 mm in women [62].

5.2.5 Biceps Pulley

The biceps pulley (or reflection pulley) [32, 78, 80] is an important part of the rotator interval, serving to maintain the position of the long head of the biceps tendon, and the detailed anatomy is covered in Chap. 13. In brief, the pulley system is a tendoligamentous sling, consisting of the coracohumeral ligament (CHL), superior glenohumeral ligament (SGHL), and fibers of the supraspinatus and subscapularis tendons (Fig. 5.6). As described above, the precise delineation of the CHL and SGHL is debatable and some experts advocate for the consideration of these ligaments as a single ligamentous structure with variable parts rather than separate ligaments [30, 32]. However, many other experts describe each structure individually.



Fig. 5.6 Normal and abnormal biceps pulleys. (**a** and **b**) Reformatted sagittal-oblique MR arthrogram image from a T1-weighted fat-suppressed 3D-FSE acquisition shows a normal biceps pulley, including a normal superior gleno-humeral ligament (white arrows) and coracohumeral ligament (arrowheads). (**c**) Sagittal-oblique T2-weighted

fat-suppressed image shows a thick coracohumeral ligament (open arrow) with partial tearing of the superior glenohumeral ligament (dashed arrow), consistent with chronic injury. Improved visualization of these structures is made possible due to the presence of a joint effusion and synovial proliferation in the subacromial-subdeltoid bursa

5.3 Pathologic Conditions

This section describes the external (subacromial and subcoracoid) and internal (posterosuperior and anterosuperior) impingement syndromes as well as their imaging manifestations. Subacromial, subcoracoid, and anterosuperior impingement syndromes can affect adults of all ages while posterosuperior impingement is more common in young and middle-aged individuals involved in repetitive overhead motions. By far the most common impingement syndrome is subacromial impingement. For this chapter, rotator cuff disease is discussed together with subacromial impingement, although some degree of cuff disease is typically present in all of the impingement syndromes.

5.3.1 Rotator Cuff Disease and Subacromial Impingement

5.3.1.1 Rotator Cuff Disease: Definition and Characterization

Rotator cuff disease is an umbrella term that can include calcific tendinitis, muscle tearing, or disorders involving the glenohumeral joint capsule (adhesive capsulitis) or subacromial-subdeltoid bursa (tendinobursitis). However, in this chapter we use the term *rotator cuff disease* to refer to tendinopathy and tendon tearing. At the histologic level, tendinosis is characterized by microscopic collagen fiber disruption, a decrease in type I collagen, glycosaminoglycan accumulation, and an increase in water content [81–83].

Tendon tears are macroscopically evident, either by gross inspection or by imaging. Partialthickness tears can be classified into articular sided, bursal sided, or intra-substance tears (also referred to as interstitial, intratendinous, or concealed tears). It is generally agreed upon that articular sided tears are at least twice as common as bursal sided tears [58, 84] and both have been associated with shoulder impingement syndromes [8]. Cadaveric studies have shown pure intra-substance tears to be twice as common as articular sided tears [85]; however this has not been confirmed in patients at surgery or on imaging, which may be due to inherent limitations with what is considered the reference standard. Partialthickness tears typically begin 13–15 mm posterior to the biceps tendon, near the junction of the supraspinatus and infraspinatus tendons [86].

The typical site of initiation in the mediallateral dimension may vary depending on the type of partial-thickness tear. In a study of 12 en bloc surgical specimens with bursal sided tears, Fukuda et al.. found all the tears developing within 1 cm of the insertion, with nine beginning slightly farther away from the insertion [87]. In a similar study of 17 specimens with intra-substance tears, Fukuda et al. found 11 (65%) of the specimens with tears that extended into the enthesis (insertion) [88]. To our knowledge, a histological study documenting the frequencies of articular sided tear initiation sites in the medial-lateral dimension has not been performed, but most authors consider these tears to begin at [86] or near [89] the humeral insertion.

The most commonly used classification of partial-thickness tears is the Ellman classification, which characterizes the cuff based on the assumption that an average intact cuff thickness is 10–12 mm [58]. Partial-thickness tears can be classified as low grade (grade 1, <3 mm deep), moderate grade (grade 2, 3-6 mm deep), or high grade (grade 3, >6 mm deep). The natural history of partial-thickness cuff tears is not well understood and some authors have found low rates of tear progression; however there is biomechanical evidence to support repair of tears involving greater than 50% of the tendon [90]. Based on available data, tears that involve less than 50% of the tendon can be debrided with good results [91].

Full-thickness tendon tears allow communication between the glenohumeral joint and subacromial-subdeltoid bursa. A full-thickness tear can be pinhole in size or involve an entire tendon (which is referred to as a full-thickness, full-width tendon tear). Compared with partial-thickness tears, full-thickness tears are associated with more synovial inflammation and tendon degeneration [92]. Full-thickness tears of the supraspinatus and infraspinatus tendons can be classified based on shape at the time of surgery [93] or on preoperative MRI [94], although one study found limited concordance for L-shaped tears [95]. A practical method of reporting is to describe the tendons involved and to measure the anterior-posterior and medial-lateral (retraction) dimensions of the tear [96]. Some authors define massive cuff tears as full-thickness tears that are greater than 5 cm in the largest dimension and involve two or more rotator cuff tendons [97, 98]. Of note, measurement precision can be limited in the setting of markedly degenerated tissue edges, even at surgery [99]. Delamination of the cuff, defined as intratendinous horizontal splitting between the articular and bursal layers, is common and estimated to be approximately 56% on non-contrast MRI exams [100]. The presence of delamination should be detected on imaging since it can be missed during routine arthroscopy and result in lower healing rates if untreated [101].

Changes in muscle volume can be seen in rotator cuff disease, particularly with chronic tendon tears, likely due to a combination of mechanical unloading [102] and denervation [103]. Although part of the same process, fatty infiltration and muscle atrophy have been shown to be independent predictors of functional outcome after repair [104]. Fatty infiltration and muscle atrophy can be readily detected on CT, ultrasound, and MRI. Both of these processes are important [105, 106], but a complete discussion of muscle disease is beyond the scope of this chapter.

5.3.1.2 External Subacromial Impingement Syndrome: Definition and Associations

The term *impingement syndrome* can be defined as a painful, localized compression of the rotator cuff tendon [107]. The most common subtype of the shoulder impingement syndromes is *external subacromial impingement*, which refers to compression of the rotator cuff by the coracoacromial arch above and the humerus below. There exist so many different uses of the term *subacromial impingement syndrome* in the literature that several authors have proposed abandoning the term altogether and instead using the term *subacromial pain syndrome* [108] or *rotator cuff disease* [109], or simply describing tendinosis or tears of the rotator cuff [110]. However, the term *impingement syndrome* remains commonly used in practice and is recognized as a disease in the tenth revision of the International Statistical Classification of Diseases and Related Health Problems (ICD) published by the World Health Organization.

Much of the controversy behind the term stems from authors who differ in their belief of the relative importance of the factors involved in rotator cuff disease. In 1972, Charles Neer introduced the concept of *impingement syndrome* in his landmark article which included 100 cadaveric scapulae and 46 patients [111]. He suggested that rotator cuff disease resulted from impingement from the anterior one-third of the acromion, coracoacromial ligament, and acromioclavicular joint on the supraspinatus tendon, sometimes extending onto the anterior infraspinatus tendon and long head of the biceps tendon. The belief that extrinsic compression was the primary cause of rotator cuff tendon disease led to the use of the term impingement syndrome to be synonymous with *rotator cuff disease* in general [109]. However, we now know that rotator cuff disease can be asymptomatic and therefore labeling all tendon abnormalities as *impingement syndrome* would be inappropriate. In addition, using dynamic ultrasound and MRI, authors have shown that asymptomatic contact can occur between the intact rotator cuff and the acromion, coracoacromial ligament, and acromioclavicular joint, which is felt to be physiologic [112, 113]. Contact alone cannot be labeled as *impingement* syndrome since, by a common definition, pain must be present.

It is now widely recognized that the pathophysiologic cause of rotator cuff disease is multifactorial, although the relative importance of each component remains debated. Regardless of whether or not contact between the cuff and extrinsic structures is causative (primary) or secondarily involved, after the rotator cuff tendon becomes diseased, nociceptive units in tendon, bursa, and subchondral bone become sensitized [114, 115] and physiologic contact forces can induce pain. This is supported by a study from Gellhorn et al. in 2015, who utilized intense focused ultrasound and were not able to elicit sensation in a control group, but in patients with rotator cuff disease sensations were elicited in the cuff, subacromial bursa, and subchondral bone at intensities less than half of what was used in the control group [116].

An abundance of literature has demonstrated many associations between rotator cuff disease and extrinsic structures, including congenital and developmental variants in bone and soft-tissue shape, acquired and often degenerative bone production, as well as os acromiale. The reader should be aware that statistically significant association is insufficient to establish causality without evidence of direction of influence. However, despite the continued controversy of causation, most practitioners would agree that it is important to be aware of lesions that have been associated with rotator cuff disease.

Neer described *proliferative spurs* which were frequently present in cases of impingement, and when anterolateral acromioplasty and coracoacromial ligament release were performed, patient satisfaction and relief of pain were achieved [111]. Subsequent literature has shown that the proliferative spurs described by Neer represent coracoacromial enthesophytes [88, 117, 118] (Fig. 5.7). Coracoacromial enthesophytes as well as lateral deltoid enthesophytes have been associated with full-thickness cuff tears in symptomatic patients [119].

Bigliani et al. [120] proposed a classification of acromial morphology: type I, flat; type II, curved; and type III, hooked. Classification of acromial morphology is controversial with several investigators showing poor reliability using radiographs [121–127]. This may arise from differences in projection angle or confusion in terminology and misclassification of type I and II acromions with subacromial enthesophytes as type III acromions [128]. These differences may explain the vastly conflicting results of some studies. For instance, Nicholson et al. found that acromial morphology is an age-independent, primary anatomic characteristic [129], whereas others have found it to be an age-dependent acquired characteristic [130, 131]. Many authors have found associations of Bigliani type III acromion morphology with cuff degeneration and tearing [122, 123, 132–137]. In addition some authors have found associations of cuff disease with acromial slope in the sagittal [138, 139] or coronal planes [18, 139, 140], whereas others have not [17]. Previous reports have suggested that scapular dyskinesia was involved in the pathogenesis of impingement syndrome [141]; however a systematic review by Ratcliffe et al. in 2014 demonstrated that there is insufficient evidence to support this [142].

Inferiorly directed osteophytes from the acromioclavicular joint have also been associated with rotator cuff tears [143–145]. Many studies have advocated for arthroscopic distal clavicular resection in the presence of rotator cuff pathologies, although nearly all were level IV evidence [146–153]. Randomized, controlled trials (level I evidence) published in 2014 [154] and 2015 [155] found that arthroscopic distal clavicular resection did not result in better clinical or structural outcomes compared with rotator cuff repair alone. In addition, distal clavicular resection can lead to symptomatic acromioclavicular joint instability [154]. However, arthroscopic distal clavicular resection is still frequently performed and therefore radiologists should make note of large osteophytes when present.

Anatomic studies have also focused on the coracoacromial ligament and its role in impingement. The CAL can have a variety of shapes including a Y-, V-, quadrangular, broad band, and multi-banded configurations [156, 157]. Subacromial enthesophytes preferentially form at the anterolateral aspect of the CAL [158]. Additionally, CAL morphologies that demonstrate more than one band have been associated with rotator cuff degeneration [156]. Although some authors have advocated for coracoacromial ligament release, either alone or in combination with other procedures [159–162], biomechanical studies have suggested that the ligament is an important restraint to superior subluxation of the humeral head [163].



Fig. 5.7 Subacromial enthesophytes associated with rotator cuff disease and external subacromial impingement in a 62-year-old man (**a** and **b**) and a 60-year-old man (**c** and **d**). (**a** and **b**) Supraspinatus outlet radiograph and sagittal-oblique T1-weighted MR image show a large subacromial enthesophyte (thin arrows), which was associated with a full-thickness full-width tear of the supraspi-

natus and partial-thickness tearing of the biceps tendon (not shown). (\mathbf{c} and \mathbf{d}) AP radiograph and coronal oblique T1-weighted MR image show subacromial (thin arrows) and greater tuberosity (open arrows) enthesophytes, which were associated with rotator cuff and biceps tendon disease (not shown). Acromioclavicular joint osteoarthrosis is present (arrowhead)

Os acromiale results from failure of fusion of the anterior acromion during development and has been associated with impingement syndromes and rotator cuff disease [164, 165]. A meta-analysis in 2014 pooled data from 26 articles reported a 7% crude prevalence of os acromiale [166]. The acromial apophysis is composed of four ossification centers: basi-acromion, meta-acromion, meso-acromion, and pre-acromion. The type of os acromiale is defined by the unfused segment immediately anterior to the site of nonunion (Fig. 5.8). The os meso-acromiale subtype is most common (failed fusion between the metaacromial and meso-acromial ossification centers) (Fig. 5.8a, b) [129, 164, 167]. Pain can arise from the nonunion site itself or from dynamic impinge-



Fig. 5.8 Os acromiale variants in three different patients. (**a** and **b**) Coronal oblique and axial T1-weighted fatsuppressed MR images after contrast injection into the glenohumeral joint show communication with the subacromial-subdeltoid bursa through a retracted, full-thickness supraspinatus tendon tear (dashed arrow). In

ment, whereby deltoid contraction during arm elevation narrows the cuff outlet [168].

5.3.1.3 Radiographic and CT Findings

Radiography is the most appropriate initial imaging modality for evaluation of shoulder pain of any etiology [169]. Calcium hydroxyapatite deposition, fractures, acromioclavicular osteoarthrosis, and glenohumeral osteoarthrosis can be readily diagnosed with radiographs. Local radioaddition, there is superior subluxation of the humerus with contrast extending into an unstable os mesoacromiale (open arrow). (c) Sagittal-oblique T1-weighted MR image shows an os pre-acromiale (arrowhead). (d) Axial gradient fat-suppressed MR image shows an os meta-acromiale with degenerative changes (arrow)

graphic protocols vary, but all radiographic shoulder studies should include a frontal radiograph, which can either be an anteroposterior (AP) projection with the humerus in neutral, internal, or external rotation or be a Grashey projection, which is in the plane of the scapula.

Although radiographs cannot directly visualize the rotator cuff, the acromiohumeral distance has been used to indirectly evaluate the tendon. A systematic review in 2015 has questioned the reliability of this measurement on radiographs, particularly using non-standardized techniques [170]. Despite this, the acromiohumeral distance continues to be used in general practice. A measurement of less than 6–7 mm has been reported to be a specific sign of a full-thickness cuff tear [171] and the amount of reduced distance is correlated with the size of the tear [172, 173]. More recently, Goutallier et al. suggested that an AHD of less than 6 mm almost systematically involves a full-thickness, full-width, or near-full-width tear of the infraspinatus tendon with advanced fatty degeneration, and is not amenable to surgical repair [174].

Osseous changes near the tuberosities of the humerus have also been reported to be associated with cuff disease. Most but not all [175] studies have found an association between intraosseous cystic changes near the superior facet of the greater tuberosity and cuff disease [176–180]. Similarly, most but not all [178] studies have found the same association for cysts near the lesser tuberosity [181–183]. However, more posteriorly located cysts near the bare area generally have not shown an association with cuff disease [176, 177, 184] and have been considered a normal variant by some authors [185].

The association of enthesophytes, cortical thickening, and subcortical sclerosis at the tuberosities and cuff disease is less well established. There are conflicting results in the literature with some authors finding an association between enthesophyte formation/subcortical sclerosis at the greater tuberosity, and rotator cuff disease (Fig. 5.7c, d) [175, 186] whereas others have found no association [187]. Koh et al. reported that the Grashey view is more sensitive than conventional AP view for the detection of greater tuberosity enthesophytes, cysts, and sclerosis [186].

A subacromial enthesophyte is a highly specific but late radiographic finding of external subacromial impingement (Fig. 5.7) [188–190]. To improve detection of a subacromial enthesophyte, the AP projection can be modified with 30-degree caudal angulation of the beam [189, 191]. The supraspinatus outlet view (also known as the modified trans-scapular lateral or Y- views) is obtained at 5–10° of caudal angulation and can also demonstrate acromial morphology and subacromial enthesophytes (Fig. 5.7a) [191]. Fluoroscopy has a limited role in the evaluation of patients with cuff disease, but may help identify subacromial enthesophytes [192] and may be useful for directing injections.

An os acromiale can be detected on radiographs, with a higher sensitivity using the axillary radiograph compared with the AP view or the supraspinatus outlet view (73.5%)[193]. Familiarity with the appearance of the overlapping shadows of the os acromiale and remaining acromion on the AP and supraspinatus outlet views can facilitate detection [193]. Despite this, a metaanalysis in 2014 demonstrated that crude radiological prevalence (4.2%) was less than half of the true anatomical prevalence (9.6%), confirming the suboptimal sensitivity of radiographs [166].

Computed tomography (CT) arthrography has also been used for evaluation of the rotator cuff, particularly when MR imaging is contraindicated. For evaluation of full-thickness tears of the supraspinatus and infraspinatus tendons, sensitivity and specificity of CT arthrography have been reported to be similar to those of MR arthrography [194, 195]. However, sensitivity for subscapularis tendon tear detection has been shown to be lower compared with the other cuff tendons when evaluated with CT arthrography [194, 196, 197]. In addition, CT arthrography with intra-articular contrast is less sensitive than MRI for partial-thickness tears, especially bursal sided tears [194, 195].

5.3.1.4 Ultrasound Findings

Ultrasound technique and findings of the normal and abnormal rotator cuff are covered in *the Sonographic Evaluation of the Shoulder* chapter. A meta-analysis by Roy et al. in 2015 has found that ultrasound demonstrates comparable diagnostic accuracy to MRI and MR arthrography for the characterization of full-thickness cuff tears with overall sensitivity and specificity estimates greater than 90% [198]. As for the diagnosis of partial tears and tendinopathy on ultrasound, estimates for specificity were high (94%), but sensitivity was lower (68% for partial tears and 79% for tendinopathy). When considering accuracy, cost, and safety, the authors concluded that ultrasound was the best option [198]. When greater tuberosity irregularities are detected on ultrasound, the operator should have a high index of suspicion for rotator cuff tearing since this finding has been shown to be a reliable indicator [199].

Dynamic assessment of the rotator cuff and surrounding structures can also be performed with ultrasound. Dynamic imaging signs that have been associated with subacromial impingement include increased thickness (also referred as gathering or bunching) of the subacromialsubdeltoid bursa [200, 201] or supraspinatus tendon [201] lateral to the coracoacromial arch during arm abduction. Less commonly, upward migration of the humeral head during active elevation of the arm prevents passage of the greater tuberosity and cuff beneath the acromion [201]. Other authors have found that thickening of the bursa during abduction is a less useful sign for impingement since it may be seen to a similar degree in healthy volunteers [202] and may be negative in approximately 20% of patients with impingement [203]. Patient pain during dynamic maneuvers should be noted since diagnostic accuracy for impingement is increased when both objective ultrasound signs and subjective pain are simultaneously present [204, 205].

5.3.1.5 MR Findings

There are limited studies evaluating the accuracy of diagnosing tendinosis on MRI. This is largely due to the complex structure as well as the orientation of the rotator cuff. On MRI, tendinosis of cylindrical tendons such as the Achilles is diagnosed by the presence of increased signal intensity [206]. However, unlike the Achilles tendon which demonstrates parallel orientation to the main magnetic field (B_0) through its course, the superior rotator cuff tendon makes a near-90degree turn as it originates from the muscle and inserts onto the greater tuberosity. It is well known that as collagen fiber orientation approaches 54.7° relative to the main magnetic field, frequency changes from dipolar interactions are minimized and signal intensity is maximum [207]. This is known as the *magic angle effect* [208] and up to a sixfold change in signal intensity has been shown in histologically normal regions of the rotator cuff tendon at 3 T depending on orientation [27]. Furthermore, the rotator cuff is composed of distinct tendons that course in different orientations. For instance, at the superior facet of the greater tuberosity, the predominant orientation of the supraspinatus is medial to lateral whereas the predominant orientation fibers is anterior to posterior. This can result in different signal intensities of the individual contributions to the cuff [26, 27, 67].

However, not all increases in intratendinous signal are artifactual and MRI-histology correlation studies have shown that signal increases and increased thickness of the cuff tendon can correlate with histologically determined tendinosis [209, 210]. A practical approach for the diagnosis of tendinosis is to rely on the combined findings of increased signal intensity within the cuff without extension to the articular or bursal surfaces as well as swelling, or increased thickness of the tendon [211]. The signal intensity abnormality should be less than that of fluid. Additionally, in the setting of increased signal without tendon caliber change, recognizing the usual location of the magic angle effect in the adducted shoulder (downsloping region) can prevent false-positive diagnoses [211]. Sein et al. found excellent intraobserver reliability for the grading of MRIdetermined supraspinatus tendinosis at 1.5 T (intraclass correlation coefficient, ICC, 0.85), but only fair to good inter-observer reliability (ICC, 0.55). At 3 T, Bauer et al. found excellent intraobserver reliability (kappa, 0.84-0.93) and moderate-to-good inter-observer reliability (kappa, 0.55–0.74) [212].

Partial-thickness tears of the rotator cuff can be diagnosed when there is signal abnormality extending to a surface of the cuff, approaching the intensity of fluid. Increased linear fluid-signal intensity that extends along the long axis of the tendon can represent a partial-thickness intrasubstance tear [34] or delamination when there is communication with the bursal or articular surfaces. The accuracy of MRI for partial-thickness cuff tears is lower than that for full-thickness cuff tears, and meta-analyses have found standard MRI to demonstrate 64–67% sensitivity and 92–94% specificity and direct MR arthrography to demonstrate 83–86% sensitivity and 93–96% specificity [198, 213]. Pitfalls for the diagnosis of a partial-thickness tear include volume averaging for small tears due to a low ratio between tear size and voxel size as well as fibrovascular tissue residing in the tear, both of which will cause signal intensity to be less than that of fluid. A unique partial-thickness bursal sided tear involves the transverse head of the infraspinatus tendon, which can be avulsed and retracted from the oblique portion [26, 63].

Full-thickness tears of the rotator cuff typically demonstrate a fluid signal intensity defect [214]. MRI is very accurate for full-thickness cuff tears with meta-analyses showing 90–92% sensitivity and 93% specificity for standard MRI and 90–95% sensitivity and 95–99% specificity with direct MR arthrography [198, 213]. For the diagnosis of partial- or full-thickness tendon tears using indirect MR arthrography, studies have shown comparable sensitivity, specificity, and accuracy with direct MR arthrography [215, 216]. In addition, a study in 2014 has suggested that a single 3D T1-weighted FSE sequence is comparable to conventional 2D sequences [217].

5.3.2 External Subcoracoid Impingement

5.3.2.1 Definition

External subcoracoid impingement (also known as coracoid impingement) is an uncommon cause of anterior shoulder pain, resulting from impingement of the subscapularis or biceps tendon between the coracoid process and lesser tuberosity [218–221]. Unfortunately a literature review by Martetschlager et al. [222] in 2011 found that our knowledge of subcoracoid impingement is not supported by rigorous scientific studies, especially with regard to diagnosis, physical examination, imaging, treatment options, and expected outcomes. In fact, there have been no prospective randomized trials or comparative studies published to date. However, the concept of subcoracoid impingement has been recognized for over a century [223].

External subcoracoid impingement may be due to idiopathic, iatrogenic, or traumatic causes. Idiopathic causes include anatomic variations, such as a long coracoid process, protuberant lesser tuberosity, or space-occupying lesions including ganglion cysts and heterotopic ossification [220, 222, 224–228]. Iatrogenic causes include surgical procedures such as coracoid transfer, posterior glenoid osteotomy, or acromionectomy [220]. Posttraumatic causes can be due to fractures of the scapula, including the coracoid process, glenoid or neck, or proximal humerus [220]. Furthermore, anterior glenohumeral instability can also cause narrowing of the coracohumeral distance [229, 230].

The diagnosis of subcoracoid impingement is challenging. Symptoms are described as dull, anterior shoulder pain aggravated by forward flexion and internal rotation [220]. The most common findings reported on imaging include subscapularis tendon disease (either bursal sided or articular sided [8]) and/or narrowing of the coracohumeral interval, which is the space between the coracoid process and anterior humerus.

5.3.2.2 Radiographic and CT Findings

Radiographs may demonstrate a far laterally projecting or a chevron-shaped coracoid process on the AP or supraspinatus outlet views, respectively [231, 232]. Axillary views have not been reported to be helpful for diagnosis [233]. Cystic changes near the lesser tuberosity may be present [233]. The coracoid index was first described on CT, defined as the lateral projection of the coracoid process beyond the glenoid joint line [233]. Dines et al. reported a mean value of 8.2 mm (range-2.5 to 25 mm) in healthy shoulders and an index of 23.5 mm in one of their patients [233]. The coracohumeral interval has also been measured on CT. In healthy shoulders, Gerber et al. reported a mean value of 8.7 mm for an adducted arm and 6.8 mm for the arm in flexion and internal rotation, concluding that subcoracoid impingement was more likely during forward flexion of a shoulder



Fig. 5.9 67-year-old man with left shoulder pain. (**a** and **b**) Axial intermediate-weighted MR images show a high-grade partial-thickness tear of the subscapularis tendon involving the articular side and lateral hood (arrow). The biceps tendon is also partially torn and medially subluxed

(arrowhead). There is a narrowed coracohumeral interval, measuring 5 mm, with cystic changes within the lesser tuberosity. Subcoracoid and subacromial-subdeltoid bursitis is present. Subcoracoid impingement was raised which was clinically confirmed

with a far laterally projecting coracoid tip close to the scapular neck [234]. Masala et al. also found that CT was useful for the measurement of the coracohumeral interval and was sensitive to even slight bone changes [235]. Abnormal coracohumeral interval values have been described on MRI and subsequently adopted to CT, although to date there are no studies correlating measurements made between the two modalities.

5.3.2.3 Ultrasound Findings

Tracy et al. performed sonography on asymptomatic volunteers and patients with the clinical diagnosis of subcoracoid impingement. Using a linear array transducer with the arm adducted across the chest, mean coracohumeral distance was 12.2 mm (range 7.8–17.5 mm) for the volunteers and 7.9 mm (range 5.9–9.6 mm) for the patients. In addition, in patients with subcoracoid impingement, bursal thickening in the subcoracoid region can be seen which can cause an anterior snapping sensation visible on dynamic sonography [236, 237]. As described above, ultrasound is also useful for the diagnosis of subscapularis tendon disease, including tendinosis.

5.3.2.4 MR Findings

Several investigators have reported on coracohumeral intervals as measured on MRI [8, 238–245] (Fig. 5.9). Although previous authors have found statistically significant differences in mean values between individuals with and without subcoracoid impingement, no ideal cutoff value exists with high sensitivity and specificity [241]. However, in patients clinically suspected to have subcoracoid impingement, a value of 6 mm or less has been used to be consistent with the disease [8, 241–244].

Associated subscapularis tendon disease can be diagnosed on MRI. Partial-thickness tendon tears can be articular sided, bursal sided (involving the anterior surface), or intra-substance (also called *interstitial delamination* [246] or a *concealed lesion* [66]). Full-thickness tears demonstrate a focus of complete tendon discontinuity [247], which can either extend from the articular side to the bursal side or extend from the articular side to the lateral edge of the tendon (also termed the *lateral hood* or *lateral end* [66]) when involving the footprint (Fig. 5.5d). Several classifications of subscapularis tendon tears exist, including the LaFosse [248], Fox and Romeo [249], and the Yoo classifications [66]. However, similar to the superior cuff, a practical method is to describe partial- or full-thickness involvement and the location of the tear, and provide measurements in the superior-inferior and medial-lateral (retraction) directions. Involvement of the inferior, extraarticular portion of the tendon (so-called muscular attachment) or tears of the myotendinous portions should be described since these may influence the decision for an open rather than arthroscopic approach for repair [250].

Combined full-thickness tears that involve the subscapularis and supraspinatus tendons are referred to as anterosuperior rotator cuff tears [251], and have been associated with combined subcoracoid and subacromial impingement [242]. The retracted edges of the two tendons can be connected by a bridge of connective tissue which has been described to represent the coracohumeral ligament [25, 32, 252]. This tissue has been called the "comma sign" [253] at surgery or the "bridging sign" on MRI [254] and may be thickened to various degrees. Recognition of this sign is useful to avoid misdiagnosing an intact subscapularis tendon [254] or a medially dislocated long head of the biceps tendon.

5.3.3 Internal Posterosuperior Impingement

5.3.3.1 Definition

The term *posterosuperior impingement* is typically, but not always, used in association with the throwing shoulder [255, 256]. Similar to external subacromial impingement, the term and concept of *posterosuperior impingement* are controversial. It is generally accepted that there is physiologic contact of the undersurface of the cuff against the edge of the glenoid in the abducted, externally rotated position [257, 258]. Furthermore, it is generally accepted that posterosuperior impingement can cause articular sided tears of the superior rotator cuff in throwing athletes. However, there are two different views of posterosuperior impingement in the literature with regard to throwing athletes: those who believe that it

explains the mechanism of most articular sided tears of the superior rotator cuff and those who believe that it explains only a minority of them.

In 1992, Walch et al. proposed that repetitive, forceful contact leads to cuff tearing in throwing athletes [255, 259]. Subsequent authors supported this view for several years, although there was disagreement about the anterior capsuloligamentous structures in the disabled throwing shoulder [256, 257]. Some believed that the presence of anterior instability worsened internal impingement [256, 260, 261], whereas others believed that instability was not a typical part of the pathology in the throwing shoulder [257]. Burkhart et al., in a series of articles published in 2003, summarized the literature and proposed a pathologic cascade in the throwing shoulder, beginning with acquired posteroinferior capsular contracture [262]. This results in a posterosuperior shift of the glenohumeral contact point during the late cocking phase, allowing hyper-external rotation of the humerus due to reduced camming effect, but causing peel-back forces which could lead to a SLAP lesion. Burkhart theorized that cuff failure in throwing athletes was typically due to repetitive tensile and torsional loading rather than impingement, although cuff tearing due to internal impingement could be seen in pitchers who hyper-externally rotate their arms in excess of 130° during the late cocking phase [262, 263].

Unfortunately, there is no consensus of the causative pathophysiologic process of the disabled throwing shoulder. In the literature, there are several imaging findings that have been associated with *posterosuperior impingement* and the disabled throwing shoulder. However, it should be emphasized that the use of the term *posterosuperior impingement* differs between individual physicians and practices. The radiologist is urged to reconcile their nomenclature with their referring physicians.

5.3.3.2 Radiographic and CT Findings

In patients diagnosed with posterosuperior internal impingement, cystic changes of the greater tuberosity may be seen on radiographs in approximately half [264], although similar findings have also been reported in 39% of asymptomatic professional baseball pitchers [265]. Remodeling of the posterior glenoid rim can also be seen radiographically, although cross-sectional imaging would optimally evaluate this region [264].

Bennett lesions, which are described as mineralization near the posteroinferior glenoid rim, have been defined exclusively in baseball pitchers, although they are seen in approximately 22% of asymptomatic major league baseball pitchers [265–267]. Bennett lesions are theorized to be caused by traction on the posterior band of the inferior glenohumeral ligament and may also be identified on CT [268, 269].

5.3.3.3 Ultrasound Findings

As described above, ultrasound is sensitive for partial-thickness articular sided tears of the rotator cuff. In patients diagnosed with posterosuperior internal impingement, ultrasound may demonstrate cortical irregularity of the posterolateral humeral head region [270]. In addition, posterosuperior labral detachment or tears may be seen, characterized as an anechoic or hypoechoic cleft between the labrum and glenoid or within the labral substance, respectively. This may be emphasized with dynamic ultrasound and may be associated with paralabral ganglion cysts [270]. Posterior capsular thickening may be associated with the diagnosis of internal impingement and can be measured with ultrasound [271].

5.3.3.4 MR Findings

Direct MR arthrography is most useful for evaluation of the constellation of imaging findings associated with posterosuperior internal impingement, which includes cystic changes near the posterolateral humeral head, partialthickness articular sided tears of the infraspinatus and posterior supraspinatus tendons, and posterosuperior labral lesions [272 - 274](Fig. 5.10). For partial-thickness cuff tears, meta-analyses have found that direct MR arthrography is slightly superior to standard MRI with a higher range of sensitivity (83-86% vs. 64-67%, respectively), but comparable specificity (93-96% vs. 92-94%, respectively) [198, 213]. For labral tears, meta-analyses have found that direct MR arthrography appears marginally superior to standard MRI with higher sensitivity (83 vs. 79%, respectively) and specificity (93 vs. 87%) [275, 276]. In pitchers with glenohumeral internal rotation deficits, posterior capsular fibrosis may be evident on MR arthrography [277]. Tuite et al. found a tendency for a thicker posteroinferior labrum and shallower capsular recess in overhead throwing athletes with internal impingement and internal rotation deficit compared with controls using the standard, adducted MRI position [278].

The abducted and externally rotated (ABER) position may be helpful to detect delamination of the rotator cuff tendon (Fig. 5.10d) and for increased accuracy for diagnosis of labral lesions [279–281], although it adds an extra 5–10 min to the examination due to necessary patient repositioning and coil changes. As described above, physiologic contact between the undersurface of the rotator cuff and postero-superior glenoid in the ABER position is considered physiologic [257].

5.3.4 Internal Anterosuperior Impingement

5.3.4.1 Definition

Anterosuperior impingement is less well defined compared with the previously discussed entities. This entity was first described in 2000 by Gerber and Sebesta in 16 patients, nearly all of whom were involved with regular overhead activity, most during their profession as manual laborers [282]. The authors postulated that repetitive contact of the superior subscapularis tendon and biceps pulley against the anterosuperior glenoid rim caused damage to these structures since pain could be reproduced when the arm was horizontally adducted, internally rotated, and positioned with various degrees of anterior elevation [282].

In 2002, Struhl reported on ten nonathletic patients who demonstrated partial-thickness articular sided tears of the supraspinatus tendon which appeared to be compressed between the humeral head and the anterosuperior labrum [283]. Struhl



Fig. 5.10 MR arthrogram images of a 30-year-old major league baseball pitcher with shoulder pain. (a) Coronal intermediate-weighted fat-suppressed image shows a high-grade, partial-thickness, articular sided tear at the footprint of the supraspinatus-infraspinatus tendon junction (arrow). A posterosuperior labral tear is present (arrowhead). (b) Axial T1-weighted fat-suppressed image confirms labral tear (arrowhead) and shows

marked irregularity at the greater tuberosity (thick arrow). (c) Sagittal intermediate-weighted fat-suppressed image confirms cystic changes near the posterosuperior aspect of the humeral head with adjacent articular sided tearing of the infraspinatus tendon. (d) ABER view improves delineation of the extent of medial delamination (arrow). The same posterosuperior humeral head cyst is seen (dashed arrow)

stated that contact of the cuff and superior labrum was normal in the intact shoulder, but abnormal in the setting of cuff tears. Nearly all his patients had identical clinical presentations to subacromial impingement, but the arthroscopic findings were consistent with the entity of *anterior internal impingement*. Subsequent authors have interpreted his study to refer to anterosuperior internal impingement [284, 285]. Notably, Struhl did not diagnose biceps pulley lesions in any of his

patients and in only two of the ten cases was a subscapularis tendon tear present [283].

In 2004, Habermeyer defined anterosuperior *impingement* as the presence of an anterosuperior labral lesion and positive impingement of the subscapularis tendon between the lesser tuberosity and anterosuperior glenoid rim in the flexed, horizontally adducted, and internally rotated position during arthroscopy [284]. He included 89 patients, none of whom performed regular overhead activity, but all with surgically confirmed pulley lesions. Notably he excluded patients with complete tears of the supraspinatus or subscapularis tendons. He found that the presence of anterosuperior impingement increased when a partialthickness articular sided tear of the subscapularis tendon was present. Habermeyer proposed a classification scheme and outlined the pathologic cascade, which begins with a degenerative or traumatic tear of the biceps pulley [284]. During the anterosuperior impingement position, the long head of the biceps tendon medially subluxates and causes a tear of the subscapularis tendon. Due to a lack of dynamic soft-tissue restraints, the humeral head migrates anterosuperiorly, impinging against the glenoid rim and causing the entity of anterosuperior impingement [284].

The diagnosis of anterosuperior impingement is very challenging and there are only a handful of scientific articles from which to draw conclusions. First, there is no patient population that is typically affected. Anterosuperior impingement has been diagnosed in young and elderly patients [282, 284]. Additionally, patients may be regularly engaged in overhead activities [282] or not [284], or may even be wheelchair bound [272, 286]. Second, clinical tests have not been reported to be sensitive or specific for this entity [283, 285]. Third, the existing literature does not support a mandatory lesion. The pulley system was surgically intact in 3 of the 16 patients in Gerber and Sebesta's study [282] and in presumably most of the patients in Struhl's study [283]. Furthermore, anterosuperior impingement has been diagnosed in many patients without subscapularis tendon lesions [283, 284]. Fourth, although used as a criterion in Habermeyer's study [284], subsequent cadaveric and in vivo MRI studies have shown that contact between the subscapularis tendon and glenoid rim typically occurs during the Hawkins position (90-degree forward elevation and maximal internal rotation) [287, 288]. Finally, authors have noted that anter-osuperior impingement tests may be negative in patients with pulley lesions, suggesting that anterosuperior impingement is not the only pathomechanism for pulley lesions [289].

5.3.4.2 Imaging Findings

As described above, there are no pathognomonic lesions for the diagnosis of anterosuperior impingement. However, several articles have focused on the biceps pulley, and in particular the superior glenohumeral ligament [284, 290]. Habermeyer [284] described a surgical classification scheme for intra-articular lesions associated with anterosuperior impingement which has been adopted to MR arthrography: group 1 lesions involve the superior glenohumeral ligament (SGHL), group 2 lesions involve the SGHL with partial-thickness articular sided supraspinatus tendon tears, group 3 lesions involve the SGHL with partial-thickness articular sided subscapularis tendon tears, and group 4 lesions involve the SGHL with both partial-thickness articular sided supraspinatus and subscapularis tendon tears.

Diagnosis of SGHL abnormalities can be readily made with MR arthrography [77, 291], or in the presence of a joint effusion (Figs. 5.6c and 5.11a). The biceps tendon may be subluxed, dislocated, and torn to various degrees [284]. Subscapularis tendon tears are usually visible to some degree in all three standard imaging planes, including coronal oblique (Fig. 5.5), sagittal oblique [290], and axial (Fig. 5.9) [292] planes and all three should be used for complete evaluation. According to Habermeyer's theory, an unstable biceps tendon causes the subscapularis tendon to tear, and these would invariably involve the superior-most fibers (Fig. 5.11). However, it should be reinforced that full-thickness tears of the subscapularis tendon are excluded in Habermeyer's classification Scheme [284], although they may be seen in later stages of the disease.



Fig. 5.11 53-year-old woman with shoulder pain during elevation and internal rotation of the arm. (a) Sagittal-oblique intermediate-weighted fat-suppressed image shows a tear of the superior glenohumeral ligament (arrow) and partial tearing of the long head of the biceps tendon (arrow-head). Subacromial-subdeltoid and subcoracoid bursitis is present. (b and c) Axial intermediate-weighted fat-sup-

5.4 Postoperative Imaging

Surgical therapy for the impingement syndromes is primarily directed at the rotator cuff, which includes debridement or repair. Although many surgeons routinely perform partial acromioplasty and coracoacromial ligament release, existing level I and level II studies do not support their routine use [293]. Incidence of rotator cuff repairs is increasing, particularly with arthroscopic techniques [294], which have become favored over open or mini-open techniques. In general, arthroscopic repair of fullthickness rotator cuff tears leads to good clinical outcomes [295, 296]. Structural failure after cuff repair is common, although counterintuitively a number of studies with high levels of evidence have shown a lack of correlation between recurrent tear and clinical or functional outcomes [295, 297]. This was confirmed in a systemic review and meta-analysis published in 2015 covering over 30 years of studies [296]. The reasons behind this are unclear and this remains an area of intense study.

5.4.1 Techniques

For appropriate interpretation of postoperative images, familiarity with the common techniques used for repair is necessary. High-grade partial-

pressed images including at the level of the superior edge of the subscapularis tendon (**b**) show tendon tearing involving the superior-most fibers of the subscapularis at the lateral hood with delamination (thick arrow). The partially torn long head of the biceps tendon is medially subluxed (arrowhead). Anterosuperior impingement was suggested based on imaging, and confirmed by the orthopedic surgeon

thickness tendon tears can be repaired through an arthroscopic trans-tendon repair technique where a single row of suture anchors are placed at the medial margin of the rotator cuff footprint [298, 299] or surgical completion of the tear and subsequent full-thickness cuff repair [300]. A metaanalysis published in 2015 found that the existing evidence supports the trans-tendon technique rather than tear conversion followed by repair for partial-thickness articular sided tears involving more than 50% of the thickness [301]. Fullthickness tendon tears can be repaired in a number of different ways, which depend on surgeon preference and many patient variables. The goal of surgical treatment of full-thickness tendon tears is to recreate the native anatomy. However, full-thickness tears that have a large medial-lateral component with poor mobility of the retracted tendon edge result in fewer choices for the orthopedic surgeon. Side-to-side suturing of the tendon edges can be performed to close the defect, either without (Fig. 5.12) [302] or with fixation of the converged tendon margin to bone [303].

Torn rotator cuff tendons that can be reduced to the greater tuberosity without undue tension are transfixed with sutures that pass through bone tunnels or through a suture anchor. These anchors can be made of metal alloy or biocomposite material, which may be partially or entirely bioresorbable [304]. Traditionally, arthroscopic rotator cuff repair used a single row of suture



Fig. 5.12 54-year-old man with previous cuff repair 1 year prior, now with worsening shoulder pain and U-shaped tear. (**a** and **b**) Coronal oblique intermediateweighted fat-suppressed MR images show a full-thickness retear of the supraspinatus tendon with differential retraction to the glenoid margin. Superior migration of the humeral head is evident. (**c**) Arthroscopic image during

anchors placed in the greater tuberosity in a linear anterior-to-posterior configuration, which could either be medial or lateral. However, this has been shown to only restore approximately 67% of the original cuff footprint [305], and the double-row repair was devised in an attempt to create more surface contact between the healing tendon and bone. The double-row repair was initially described with a medial row of anchors with sutures in a mattress configuration and a lateral row of anchors with sutures in a simple configuration, but subsequent studies showed limited contact pressures between tendon and bone com-

revision surgery with scope in subacromial-subdeltoid bursa through posterior portal shows side-to-side tendon repair. Sutures extend across U-shaped tear. (d) Coronal oblique intermediate-weighted fat-suppressed MR image 3 years after revision surgery shows an attenuated but intact repair (dashed arrows). Subacromial-subdeltoid bursitis was present, but no full-thickness tear was visualized

pared with newer double-row techniques [306, 307]. One double-row technique that has gained popularity is the transosseous equivalent, otherwise known as the suture bridge technique. This was developed in 2006 by Park et al. [308] to optimize footprint contact area, pressure, and pullout strength. The transosseous equivalent technique uses a medial row of suture anchors and a lateral row of knotless anchors. The double-row techniques, including the transosseous equivalent technique, are significantly stronger than single-row repairs in time-zero cadaveric studies and several studies have suggested higher

rates of healing [295, 297, 309]. True arthroscopic transosseous (anchorless) fixation has also been described [310], although biomechanical studies have shown superior results with transosseous equivalent techniques [311].

5.4.2 Imaging

In patients with persistent or new shoulder pain after surgical therapy, imaging may be indicated. First-line imaging modalities of the postoperative cuff include ultrasound, MRI, or MR arthrography. Prickett et al. used ultrasound to evaluate postoperative rotator cuff integrity and reported the sensitivity, specificity, and accuracy to be 91, 86, and 89%, respectively [312]. However, Lee et al. found that accuracy of ultrasound for the postoperative cuff was 78% when compared to MR arthrography [313]. They found that ultrasound accuracy increased to 93% with the use of intra-articular contrast (arthrosonography) [313]. MRI without or with intra-articular contrast can be used to evaluate the status of the repaired rotator cuff [300, 314–316] (Fig. 5.13). The appearance of the repaired rotator cuff on

Fig. 5.13 52-year-old man with repair of full-thickness supraspinatus tendon tear. (a) Coronal oblique intermediate-weighted MR image shows a focal full-thickness tear of the supraspinatus tendon at the footprint (thick arrow) with delamination. (b) Arthroscopic image in glenohumeral joint through posterior portal confirms articular sided supraspinatus tendon tear (black arrows). Humeral head (HH) is marked. (c) Arthroscopic image in

subacromial-subdeltoid bursa through posterior portal after purple marking suture was placed through articular side. Probe easily extended through bursal surface, confirming the focal full-thickness tear (black arrowhead). (d) Coronal oblique T1-weighted fat-suppressed image 2 years after repair shows well-healed footprint after single-row repair (dashed arrow)





Fig. 5.14 65-Year-old woman status post-rotator cuff repair 4 months prior with worsening shoulder pain and characteristic failure location after double-row repair. (a) Coronal oblique intermediate-weighted fat-suppressed MR image shows a full-thickness retear of the distal supraspinatus tendon with retraction (thick arrow). Small

MRI varies depending on the time of imaging. Within the first 3 months, there can be increased signal within the repaired cuff and the appearance of poor footprint coverage, which can improve by the first postoperative year [317]. In a group of 15 asymptomatic patients 1.5–5 years after rotator cuff repair, Spielmann et al. found that only 10% of tendons demonstrated normal low signal intensity [318].

If there is unequivocal full-thickness fluid signal traversing the entire repaired tendon at any time point, a retear can be diagnosed [317, 319]. Structural failure, as determined with imaging, is common after both single-row and double-row repair techniques. Multiple studies with high levels of evidence show conflicting results regarding retear rates after each technique, suggesting that there may not be a true difference between these techniques [320, 321]. However, studies have suggested characteristic tear patterns which are dependent on technique. Cho et al. found that in a single-row repair group, 74% of retearing occurred at the insertion site of the cuff, whereas in a transosseous equivalent group, 74% of retearing occurred in the tendon near the medial row [322].

amount of tendon remains visible at the footprint (thin arrow). (b) Arthroscopic image during revision surgery with scope in subacromial-subdeltoid bursa through posterior portal confirms full-thickness retear (thick arrows). Tear is centered medial to the medial row (arrowhead marks medial row suture from initial repair)

Similar to the transosseous equivalent technique, the failure pattern in the double-row suture anchor method tends to involve the tendon near the medial row rather than at the insertion [323] (Fig. 5.14).

In 2015, Saccomanno et al. performed a systematic review of MRI criteria for the assessment of rotator cuff repair and identified 26 different criteria that have been previously used [324]. This included structural integrity, footprint coverage, tendon thickness, signal intensity, partial retearing, and muscle atrophy and fatty infiltration. The principal finding of the study was that, with the data available, only structural integrity showed good intra- and inter-observer reliability [324]. Specifically, reliability was highest when a binary classification scheme was used (dichotomization of cuffs into intact versus retear groups).

5.5 Conclusion

In summary, rotator cuff disease is common and the diagnosis of impingement syndromes requires all available information, including history, physical examination, and imaging. Our knowledge of the anatomy involving the rotator cuff is rapidly evolving, and this has many clinical implications. The etiology of rotator cuff disease is multifactorial with intrinsic and extrinsic contributions and knowledge of both mechanisms is required for targeted therapy. Evaluation of the rotator cuff after surgery is challenging, but imaging plays an important role and familiarity with the different repair techniques as well as expected and abnormal postoperative appearances will aid the radiologist in making an accurate diagnosis.

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