Musculoskeletal Diseases
Diagnostic Imaging and Interventional Techniques
MUSCULOSKELETAL DISEASES

DIAGNOSTIC IMAGING AND INTERVENTIONAL TECHNIQUES

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Preface

The International Diagnostic Course in Davos (IDKD) offers a unique learning experience for imaging specialists in training as well as for experienced radiologists and clinicians wishing to be updated on the current state of the art and the latest developments in the fields of imaging and image-guided interventions.

This annual course is focused on organ systems and diseases rather than on modalities. This year’s program deals with diseases of the musculoskeletal system. During the course, the topics are discussed in group seminars and in plenary sessions with lectures by world-renowned experts and teachers. While the seminars present state-of-the-art summaries, the lectures are oriented towards future developments.

This syllabus represents a condensed version of the contents presented under the 20 topics dealing with imaging and interventional therapies in the musculoskeletal radiology. The topics encompass all the relevant imaging modalities including conventional x-rays, computed tomography, nuclear medicine, ultrasound and magnetic resonance angiography, as well as image-guided interventional techniques.

The volume is designed to be an “aide-mémoire” for the course participants so that they can fully concentrate on the lectures and participate in the discussions without the need of taking notes. Additional information is found on the web page of the IDKD (http://www.idkd.ch).

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SEMINARS
Introduction

This seminar places special emphasis on the MRI manifestations of shoulder pathology. The discussion includes the following topics:
1. Rotator cuff pathology and impingement lesions.
2. Glenohumeral instability and related lesions.
3. Miscellaneous shoulder conditions.

Rotator Cuff Pathology and Impingement Lesions

Impingement syndrome is a clinical entity produced by compression of the supraspinatus tendon under the region of the acromial arch, and it can be related to abnormal morphology of the acromion process, thickening of the coracoacromial ligament, subacromial spurring, or degenerative arthritis of the acromioclavicular joint. Alternatively, it can be related to degeneration, repeated trauma or overuse during overhead exercise, such as swimming. Normal anatomical variants, such as type III undersurface of the acromion with a hooked configuration and os acromiale, have been described associated with rotator cuff impingement and tears.

There are two types of impingement syndrome: primary, associated with abnormalities in the coracoacromial arch; and secondary to rotator cuff dysfunction. The secondary form of rotator cuff impingement may be further subdivided into two types: internal and external. The internal type refers to the articular surface side of the rotator cuff and it is often termed posterosuperior impingement syndrome. The external variety occurs as a result of external compression of the anterior aspect of the cuff in the bursal side and includes the coracoid impingement syndrome. Posterosuperior impingement syndrome occurs in the throwing athlete as a result of continuous strain of the anterior capsular mechanism, which leads to laxity and anterior subluxation of the glenohumeral joint with the arm in abduction and external rotation. This situation produces impingement of the supraspinatus tendon at the level of its insertion in the greater tuberosity of the humerus as well as small impaction fractures and posterosuperior labral lesions. The coracoid impingement syndrome may occur when the distance between the posterior aspect of the coracoid process and the humerus is decreased, producing compression of the rotator cuff, mainly the subscapularis tendon.

Inflammatory changes within the supraspinatus tendon can be seen during the early phases of the disease, along with subacromial bursitis, but this can progress into rotator cuff tear. Three histological stages of impingement syndrome have been described. In stage I, edema and hemorrhage of the subacromial soft tissues are present. In stage II, there is fibrosis and thickening, while in stage III, partial or complete rotator cuff tears are seen.

Full-thickness rotator cuff tears involve most often the supraspinatus tendon, but they can also extend to the infraspinatus and subscapularis tendons. Tear of the teres minor is very rare. Partial-thickness rotator cuff tears may involve the articular or the bursal surfaces, or they may be located within the substance of the tendon. Delaminating tears of the rotator cuff can be partial or full thickness. They extend in the longitudinal direction of the tendon fibers, and there may be different degrees of retraction of the various layers. Delaminating tears may be associated with fluid collections extending from the tear into the muscle (sentinel cyst). Full-thickness tears allow communication between the articular space of the glenohumeral joint and the subacromial-subdeltoid bursa, unless the tear is covered by granulation or scar tissue. On rare occasions, tears may involve the rotator cuff interval, with capsular disruption. Tears of the rotator cuff interval may be associated with lesions of the structures present within this anatomical space, namely, the long head of the biceps tendon, the coracoacromial ligament, the superior glenohumeral ligament and also the superior labrum.

Glenohumeral Instability and Related Lesions

Restraints to anterior translation of the humeral head are provided by the capsule and the glenohumeral ligaments
The labrum is torn as part of the avulsion forces produced by the GHL at the time of the injury. Anteroinferior dislocation is the most frequent cause of anterior glenohumeral instability. A single event originates a constellation of lesions leading to other episodes of dislocation or subluxation. The lesions that may take place during an anteroinferior dislocation include anteroinferior labral tear, tear of the inferior GHL (IGHL) and/or capsular-periosteal stripping, fracture of the anteroinferior glenoid margin and compression fracture of the superior lateral aspect of the humeral head (Hill-Sachs lesion).

The classic Bankart lesion is the combination of anterior labral tear and capsuloperiosteal stripping. On arthroscopy, the Bankart lesion is seen as a fragment of labrum attached to the anterior band of the IGHL and to the ruptured scapular periosteum, “floating” in the anterior-inferior aspect of the glenohumeral joint. Extensive bone and soft-tissue damage and persistent instability may lead to multidirectional instability, resulting in episodes of posterior dislocation.

A number of variants of anterior labral tears have been described. The Perthes lesion is similar to the Bankart lesion, but without the tear of the capsule. Anterior labroligamentous periosteal sleeve avulsion (ALPSA) refers to a tear of the anteroinferior labrum, with associated capsuloperiosteal stripping. The torn labrum is rotated medially, and a small cleft or separation can be seen between the glenoid margin and the labrum. In contrast to the Bankart lesion, the ALPSA lesion can heal, leaving a deformed and patulous labrum. The glenoid labral articular disruption (GLAD) represents a tear of the anteroinferior labrum, attached to a fragment of articular cartilage, without associated capsuloperiosteal stripping.

Posterior shoulder dislocation more often occurs as a result of a violent muscle contraction, e.g., by electrical shock or seizures. After the acute episode of dislocation, the arm frequently remains locked in adduction and internal rotation. Posterior instability caused by repeated micro-trauma, without frank dislocation, may cause persistent shoulder pain in young athletes. Abduction, flexion and internal rotation are the mechanism involved in these cases (swimming, throwing, and punching). This may be also associated with posterior capsular laxity. Lesions that may occur during posterior dislocation or in cases of repeated micro-trauma include posterior labral tear, posterior capsular stripping or laxity, fracture, erosion, or sclerosis and ectopic bone formation of the posterior glenoid, and vertical impacted fracture of the anterior aspect of the humeral head (reverse Hill-Sachs, McLaughlin fracture).

Superior labral anterior and superior lesions (SLAP lesions) are not as rare as originally thought. These lesions involve the superior part of the labrum with varying degrees of biceps tendon involvement. Pain, clicking, and occasional instability in a young patient are the typical clinical manifestations. Four types of SLAP lesions were originally described based on arthroscopic findings: Type I is a partial tear of the superior part of the labrum with fibrillation of the LHBT. Type II is an avulsion of the LHBT with tear of the anterior and posterior labrum. Type III is a bucket-handle tear of the labrum and type IV is a bucket-handle tear of the labrum with longitudinal tear to the LHBT. More recently, up to ten types of SLAP lesions have been described, representing a combination of superior labral tears with extension into different areas of the labrum and glenohumeral ligaments.

**Miscellaneous Lesions**

The following lesions are discussed:

- **Biceps tendon**
- **Compressive neuropathies**
- **Inflammatory and other miscellaneous lesions**

**Biceps Tendon**

Tendinosis or tenosynovitis of the LBT may occur in association with shoulder impingement syndrome and rotator cuff tears, where the intracapsular portion of the LBT is compressed between the humeral head, the acromion, and the coracoacromial ligament during abduction and rotation of the arm. Attritional tendinosis is associated with a narrow bicipital groove and hence it affects the extracapsular portion of the tendon. Magnetic resonance imaging (MRI) may demonstrate fluid in the joint extending into the bicipital groove, although this a non-specific sign unless the fluid completely surrounds the tendon, in the absence of a joint effusion. Trauma and degeneration may involve the LBT, producing swelling and increased signal intensity (SI) on T2 and T2* pulse sequences.

Complete rupture of the LBT more often occurs proximally, at the level of the proximal portion of the extracapsular segment, within the groove. MRI demonstrates the absence of the LBT in the groove and its distal displacement. Intracapsular tears of the LBT are seen more often in patients with rotator cuff tears. Attritional tendinosis affecting the intertubercular portion of the LBT can progress to longitudinal splits within the tendon, resulting in thickening of the LBT with increased intrasubstance SI on T2-weighted images. A bifid LBT (normal variant) should not be confused with a partial longitudinal tear.

Biceps tendon dislocation occurs with tears of the subscapularis tendon and coracohumeral ligament. Two types of dislocation of the LBT have been described, depending on whether the tendon is located in front or behind the subscapularis tendon. In the first type, the insertion fibers of the subscapularis tendon are intact. In the second type, the subscapularis tendon is detached and the LBT is medially displaced, becoming entrapped intra-articularly.
Compressive Neuropathies

The suprascapular nerve and its branches can become compressed or entrapped by stretching due to repetitive scapular motion, or they can be damaged by scapular fractures, overhead activities, soft-tissue masses or direct trauma. T2-weighted images can show hyperintensity of the involved muscle. Nerve thickening and muscle atrophy due to denervation may be noted in advanced cases. Ganglion cysts at the scapular incisura typically associated with posterior labral tears can be easily detected by MRI of the shoulder.

The quadrilateral space syndrome is caused by compression of the axillary nerve at the quadrilateral space. The teres minor and deltoid muscles and the posterolateral cutaneous region of the shoulder and upper arm are innervated by the axillary nerve. Proximal humeral and scapular fractures, shoulder dislocations, or axillary mass lesions can result in damage or compression of the axillary nerve. Entrapment of this nerve can also be produced by extreme abduction of the arm during sleep, hypertrophy of the teres minor muscle in paraplegic patients or by a fibrous band within the quadrilateral space. Patients may have shoulder pain and paresthesia. In advanced cases, atrophy of the deltoid and teres minor muscles can occur, but more often there is selective atrophy of the teres minor muscle.

Parsonage-Turner syndrome, also referred to as acute brachial neuritis, is clinically characterized by sudden onset of severe atraumatic pain in the shoulder girdle. The pain typically decreases spontaneously in 1-3 weeks, and is followed by weakness of at least one of the muscles about the shoulder. The exact etiology has not been established but viral and immunological causes have been considered. MRI findings in the acute stage include diffuse increased SI on T2-weighted images consistent with interstitial muscle edema associated with denervation. The most commonly affected muscles are those innervated by the suprascapular nerve, including the supra- and infraspinatus. The deltoid muscle can also be compromised in cases of axillary nerve involvement. Later in the course of the disease, there may be muscle atrophy, manifested by decreased muscle bulk.

Inflammatory and Other Miscellaneous Lesions

The manifestations of idiopathic synovial osteochondromatosis on MRI depend on the degree of calcification or ossification of the cartilaginous bodies. If no calcification is present, it may simulate a joint effusion, with low SI on T1-weighted images and high SI on T2-weighted images. However, high-resolution MRI may be able to demonstrate a signal that is more inhomogeneous than fluid. If calcifications are present, these will manifest themselves as multiple small foci of decreased SI on both T1- and T2-weighted pulse sequences, surrounded by high SI haloes on T2-weighted images, which represent the cartilaginous coverage. The presence of low-SI material mixed with hyperintense cartilage may mimic pigmented villonodular synovitis, especially if bone erosions are present. Other differential diagnostic considerations include entities that can produce multiple intra-articular bodies, such as osteocartilaginous loose bodies related to osteoarthritis or osteochondral trauma, and “rice bodies”, such as those seen in rheumatoid arthritis and tuberculosis (see below).

The appearance of PVNS on MRI is quite distinct due to the paramagnetic effect of the hemosiderin deposits, which produces characteristic foci of low SI on T1- and T2-weighted sequences. An heterogeneous pattern is also frequently observed, due to the presence of areas of low hemosiderin deposition and associated joint effusion. The paramagnetic effect of hemosiderin is enhanced on gradient-echo pulse sequences. Associated ancillary findings, such as bone erosions and capsular distension, are often seen in the diffuse form of PVNS. The differential diagnosis of hypointense intra-articular material includes urate crystals of gout, synovial osteochondromatosis, and amyloid deposition.

MRI of rheumatoid arthritis shows joint effusion, subacromial-subdeltoid bursitis, rotator cuff tendinosis and tears secondary to the effect of the inflamed synovium on the undersurface of the tendons, and “rice bodies”. Chronic articular inflammation evolves into proliferation of elongated synovial villi that become fibrotic and eventually detach, producing grains similar to polished rice. On MRI, these “rice bodies” manifest themselves as numerous rounded nodules of intermediate SI occupying the joint space and/or the subacromial bursa. Similar findings can be seen in tuberculous arthritis and even synovial chondromatosis.

Suggested Readings

Shanley DJ, Mulligan ME (1990) Osteochondrosis dissecans of the glenoid. Skeletal Radiol 19:419-421
Magnetic Resonance Imaging of the Elbow

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Elbow injuries are common, especially in the athlete, and can be basically classified into acute or chronic injuries. The following discussion of magnetic resonance imaging (MRI) of the elbow will address variations in normal anatomy that represent pitfalls in imaging diagnosis, and commonly encountered osseous and soft-tissue pathology.

Osseous Anatomic Considerations and Pathology

The lateral articulating surface of the humerus is formed by the capitellum, a smooth, rounded prominence that arises from its anterior and inferior surfaces. As it does so, its width decreases from anterior to posterior. This morphology of the capitellum (smooth surface), in conjunction with the knowledge that the adjacent lateral epicondyle (rough surface) is a posteriorly oriented osseous projection of the distal humerus, explains the pseudodefect of the capitellum which must be distinguished from a post-traumatic osteochondral lesion [1].

The articular surface of the proximal ulna is formed by the combination of the posterior olecranon and the anterior coronoid processes, with the articular surfaces taking the configuration of a figure of eight. At the waist of the eight, or junction between anterior and posterior aspects of the ulna, the articular surface is traversed by a cartilage-free bony ridge. This trochlear ridge is 2 to 3 mm wide and is at the same height as the adjacent cartilaginous surface. It should not be mistaken for a central osteophyte. The waist of the figure of eight is formed by the tapered central surfaces of the coronoid and olecranon processes both medially and laterally, forming small cortical notches devoid of cartilage. On sagittal MRI, these focal regions devoid of cartilage could be mistaken for a focal chondral lesion [2].

Osteochondral Lesions

In the case of acute medial elbow injury, the involvement of a valgus force is usually described as one of the most common mechanisms of injury [3]. Subchondral bone and cartilage injuries that occur in this setting result from impact and shearing forces applied to the articular surfaces. The overall configuration of the humeroradial articulation, in this case, can be likened to a mortar and pestle, with the capitellar articular surface impacting that of the radius to result in a chondral or osteochondral lesion of the capitellar surface. These acute post-traumatic lesions are manifested on MRI as irregularity of the chondral surface, disruption or irregularity of the subchondral bone plate, and or the presence of a fracture line. The acuity of the lesion and a post-traumatic etiology are implied by the presence of marrow edema and joint effusion. Close inspection of the location of the lesion on coronal and sagittal MRI is of the utmost importance in order to distinguish a true osteochondral lesion from the pseudodefect of the capitellum. Correlation with presenting clinical history is also helpful in determining the etiology of imaging findings.

The entity of osteochondritis dissecans remains controversial, primarily due to debate over its etiology. The precise relationship of osteochondritis dissecans and an osteochondral fracture is unclear, but many investigators regard the former as a post-traumatic abnormality that may lead to osteonecrosis. Osteochondritis dissecans is thought to occur in immature athletes between 11 and 15 years of age, rarely in adults [4]. Osteochondritis dissecans of the elbow involves primarily the capitellum, but reports have described this process in the radius and trochlea [5].

Regardless of the etiology of the osteochondral injury, the role of imaging is to provide information regarding the integrity of the overlying articular cartilage, the viability of the separated fragment, and the presence of associated intra-articular bodies. Both computed tomography (CT) and MRI with and without arthrography can provide this information to varying degrees, although no scientific investigation has been performed to date that establishes specific indications for each study. MRI, with its excellent soft-tissue contrast, allows direct visualization of the articular cartilage, as well as of the character of the interface of the osteochondral lesion with native bone (Fig. 1). The presence of joint fluid or granulation tissue at this interface, manifested as increased signal intensity on fluid-sensitive MRI, generally indicates an unstable lesion. The in-
Introduction of contrast into the articulation in conjunction with MRI can be helpful in two ways: (1) to facilitate the identification of intra-articular bodies, and (2) to establish communication of the bone-fragment interface with the articulation by following the route of contrast, providing even stronger evidence for an unstable fragment [6, 7].

Ligament Pathology

Valgus Instability

The principle function of the ulnar collateral ligament complex is to maintain medial joint stability to valgus stress. The anterior bundle is the most important component of the ligamentous complex to this end, as it serves as the primary medial stabilizer of the elbow from 30 to 120 degrees of flexion. The most common mechanisms of ulnar collateral ligament insufficiency are chronic attenuation, as seen in overhead or throwing athletes, and post-traumatic, usually after a fall on the outstretched arm. In the case of the latter, an acute tear of the ulnar collateral ligament may be encountered.

With throwing sports, high valgus stresses are placed on the medial aspect of the elbow. The maximum stress on the ulnar collateral ligament occurs during the late cocking and acceleration phases of throwing [8]. Repetitive insults to the ligament allow microscopic tears that progress to significant attenuation or frank tearing within its substance (Fig. 2). While MRI facilitates direct

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Fig. 1. A Conventional radiograph demonstrates a lytic osteochondral lesion in the capitellum (arrow). B This lesion is low signal intensity on a T1-weighted image and has a high signal intensity rim on a T2-weighted axial image. C suggesting instability (arrow)

Fig. 2. Coronal FSE T2-weighted image with fat suppression shows a full-thickness tear of the anterior band of the ulnar collateral ligament at the attachment to the sublime tubercle (arrow)
visualization of the ligament complex, in chronic cases, the development of heterotopic calcification along the course of the ligament has been described [9].

**Varus Instability**

Lateral elbow instability related to isolated abnormalities of the lateral collateral ligament complex is not as well described as that on the medial side of the elbow. If it were to occur, the mechanism would be a stress or force applied to the medial side of the articulation, resulting in compression on that side, with opening of the lateral articulation and subsequent insufficiency of the radial collateral ligament. As the radial collateral ligament attaches on and is intimately associated with the annular ligament, an abnormality discovered in one of the structures obligates careful inspection of the other.

Varus stress applied to the elbow may occur as an acute injury, but rarely as a repetitive stress, as encountered on the medial side. While lateral collateral ligament injuries rarely occur as the result of an isolated varus stress, other causes can commonly lead to this injury, including dislocation, subluxation and overly aggressive surgery (release of the common extensor tendon or radial head resection).

Varus instability is also tested with the elbow in full extension and 30 degrees of flexion to unlock the olecranon. A varus stress is applied to the elbow while palpating the lateral joint line.

**Posterolateral Rotary Instability and Elbow Dislocation**

The subject of elbow instability is complex and has been a challenge due to the difficulty in establishing the mechanism of injury and reliable clinical tests for diagnosis. With the realization that elbow instability is more common than previously thought, marked advances in the understanding of this entity are occurring.

For recurrent instability, posterolateral rotary instability is the most common pattern. This type of instability represents a spectrum of pathology consisting of three stages, according to the degree of soft-tissue disruption. In stage 1, there is posterolateral subluxation of the ulna on the humerus that results in insufficiency of the lateral ulnar collateral ligament (Fig. 3) [10, 11, 12]. In stage 2, the elbow dislocates incompletely so that the coronoid is perched under the trochlea. In this stage, the radial collateral ligament, and anterior and posterior portions of the capsule are disrupted, in addition to the lateral ulnar collateral ligament. Finally, in stage 3, the elbow dislocates fully so that the coronoid rests behind the humerus. Stage 3 is subclassified into three further categories. In stage 3A, the anterior band of the medial collateral ligament is intact and the elbow is stable to valgus stress after reduction. In stage 3B, the anterior band of the medial collateral ligament is disrupted so that the elbow is unstable with valgus stress. In stage 3C, the entire distal humerus is stripped of soft tissues, rendering the elbow grossly unstable even when a splint or cast is applied with the elbow in a semi-flexed position. This classification system is helpful, as each stage has specific clinical, radiographic and pathologic features that are predictable and have implications for treatment [10].

Subluxation or dislocation of the elbow can be associated with fractures. Fracture-dislocations most commonly involve the coronoid and radial head, a constellation of findings referred to as the “terrible triad” of the elbow, as the injury complex is difficult to treat and prone to unsatisfactory results [10]. Radial-head fractures do not cause clinically significant instability unless the medial collateral ligament is disrupted. An important feature of elbow injuries to recognize is that the small flake fracture of the coronoid, commonly seen in elbow dislocations, is not an avulsion fracture. Nothing attaches to the very tip of the coronoid; rather, the capsule attaches on the downward slope of the coronoid, the brachialis even more distally. This fracture is a shear fracture and is likely pathognomonic of an episode of elbow subluxation or dislocation. A second consideration with respect to elbow dislocation is that, as the ring of soft tissues is disrupted from posterolateral to medial, the capsule is torn and insufficient. In the absence of an intact capsule, joint fluid dissected as the soft-tissue planes of the forearm, negating an indirect radiographic sign of trauma in the elbow, that of joint effusion.

**Tendon Pathology**

The many muscles about the elbow can be divided into four groups: posterior, anterior, medial and lateral. The
Tenderness to palpation over the anterior aspect of the med-ondas are involved most frequently, resulting in pain and bowlers. The pronator teres and flexor carpi radialis ten-

throwers, racquetball and squash players, swimmers and sport of golfing. It has also been reported with javelin

mon flexor tendon and is associated primarily with the
tensity changes.

made by consideration of both morphology and signal in-

tion of “epicondylitis” for the purpose of clinical
diagnosis, inflammatory osseous changes rarely occur.

been termed “epicondylitis” for the purpose of clinical

disease process. Histologically, necrosis, round-cell infiltration, focal calcification and scar formation have been shown

[16]. In addition, invasion of blood vessels, fibroblastic

proliferation, and lymphatic infiltration, the combination

of which are referred to as angiofibroblastic hyperplasia, occur and ultimately lead to mucoid degeneration as the

process continues [17, 18]. The absence of a significant

inflammatory response has been emphasized repeatedly, and may explain the inadequacy of the healing process.

The imaging findings in this process are exactly those

encountered in the clinical entity of medial epicondylitis (Fig. 4). As on the medial side, when pathology is en-
countered in the tendon, close scrutiny of the underlying

ligamentous complex is necessary to exclude concomi-
tant injury. In particular, thickening and tears of the lat-

eral ulnar collateral ligament have been encountered with lateral epicondylitis [13].

Fig. 4. Coronal T1-weighted (left) and fat-suppressed FSE T2-

weighted images show thickening and intermediate signal intensi-
ty in the common extensor tendon (arrows), consistent with tendi-
nosis (lateral epicondylitis)
**Biceps Tendon**

Rupture of the tendon of the biceps brachii muscle at the elbow is rare and constitutes less than 5% of all biceps tendon injuries [19]. It usually occurs in the dominant arm of males. Injuries to the musculotendinous junction have been reported, but the most common injury is complete avulsion of the tendon from the radial tuberosity. Although the injury often occurs acutely after a single traumatic event, the failure is thought to be due to pre-existing changes in the distal biceps tendon, due to intrinsic tendon degeneration, enthesopathy at the radial tuberosity, or cubital bursal changes. The typical mechanism of injury relates to forceful hyperextension applied to a flexed and supinated forearm. Athletes involved in strength sports, such as competitive weightlifting, football and rugby, often sustain this injury.

Clinically the patient describes a history of feeling a “pop” or sudden sharp pain in the antecubital fossa. The classic presentation of a complete distal biceps rupture is that of a mass in the antecubital fossa due to proximal migration of the biceps muscle belly. Accurate diagnosis is more difficult in cases of the rare partial tear of the tendon, or more common complete tear of the tendon without retraction. The latter can occur with an intact bicipital aponeurosis, which serves to tether the ruptured tendon to the pronator flexor muscle group.

MRI diagnosis of biceps tendon pathology becomes important in patients who do not present with the classic history or mass in the antecubital fossa, or for evaluation of the integrity of the lacertus fibrosus. MRI diagnosis of tendon pathology, as previously mentioned, is largely dependent on morphology, signal intensity and the identification of areas of tendon discontinuity (Fig. 5). In the case of the biceps tendon, an important indirect sign of tendon pathology is the presence of cubital bursitis.

**Triceps Tendon**

Rupture of the triceps tendon is quite rare. The mechanism of injury has been reported to result from a direct blow to the triceps insertion, or a deceleration force applied to the extended arm with contraction of the triceps, as in a fall. Similar to the pathology encountered in the distal biceps tendon, most ruptures occur at the insertion site, although musculotendinous junction and muscle belly injuries have been reported. Complete ruptures are more common than partial tears. Associated findings may include olecranon bursitis, subluxation of the ulnar nerve, or fracture of the radial head. Accurate clinical diagnosis relies on the presence of local pain, swelling, ecchymosis, a palpable defect, and partial or complete loss of the ability to extend the elbow. With more than 2 cm of retraction between the origin and the insertion, a 40% loss of extension strength can result [19].

For MRI diagnosis of triceps tendon pathology, it is imperative to be aware that the triceps tendon appearance is largely dependent on arm position. The tendon will appear lax and redundant when imaged in full extension, whereas it is taut in flexion. The MRI features of a tear are similar to those associated with any other tendon.

**Entrapment Neuropathy**

The ulnar, median and radial nerves may become compressed at the elbow, leading to symptoms of entrapment neuropathy. Abnormal nerves may have increased signal intensity on T2-weighted images, focal changes in girth, and deviation that may result from subluxation or displacement by an adjacent mass.

Ulnar nerve entrapment most commonly occurs in the cubital tunnel. Nerve compression may be caused by a medial trochlear osteophyte or incongruity between the trochlea and olecranon process [20]. Anatomic variations also contribute. The absence of the triangular reticulum, the anatomic roof of the cubital tunnel, occurs in about 10% of cases, permitting subluxation of the nerve with flexion. It is necessary, therefore, to include axial images of the flexed elbow in patients suspected of this disorder.

The presence of the anomalous anconeous epitrochlearis muscle over the cubital tunnel causes static compression of the nerve. In addition, there are many other causes of ulnar neuritis, including thickening of the overlying ulnar collateral ligament, medial epicondylitis, adhesions, muscle hypertrophy, direct trauma, and callus from a fracture of the medial epicondyle. MRI can be used to identify these abnormalities and to assess the ulnar nerve itself. When compressed, the nerve may become enlarged and edematous. If conservative treatment fails, the nerve can be transposed anteriorly, deep to the flexor muscle group, or more superficially, in the subcutaneous tissue. One can follow these patients with MRI.

**Fig. 5.** Axial-fat-suppressed T2-weighted image shows complete disruption of the distal biceps at the radial tuberosity (arrow)
postoperatively if they become symptomatic to determine whether symptoms are secondary to scarring or infection around the area of nerve transposition.

Compression of the median nerve may be seen with osseous or muscular variants and anomalies, soft-tissue masses and dynamic forces. In the pronator syndrome, compression occurs as the median nerve passes between the two heads of the pronator teres and under the fibrous arch of the flexor digitorum profundus.

The radial nerve can become entrapped following direct trauma, mechanical compression by a cast or overlying space-occupying mass, or a dynamic compression as a result of repeated pronation, forearm extension, and wrist flexion, as is seen in violinists and swimmers. Motor neuropathy of the hand extensors is a dominant feature when the posterior interosseous nerve is entrapped [21].

References
Radiology of Hand and Wrist Injuries

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Introduction

Musculoskeletal trauma is common and the distal upper extremity is one of the most frequent sites of injury. Imaging of hand and wrist injuries should always begin with conventional radiographs. While computed tomography (CT) and magnetic resonance imaging (MRI) are very helpful in some cases, their overall impact on trauma imaging in the hand and wrist is small. Radiographs remain the primary diagnostic modality. It is therefore essential for radiologists who work in a trauma and emergency setting to be familiar not only with the normal radiographic anatomy of the hand and wrist but also with the range of injuries that can occur. Our learned colleague, Lee F. Rogers, put it all quite simply in a few statements that can be called “Rogers’ Rules”: Rule #1, make the diagnosis; Rule #2, avoid embarrassment; Rule #3, stay out of court. In order to meet these objectives, we must get adequate radiographs and we must interpret them correctly. Thus, not only should we know where to look when there is nothing obvious at first glance but we must also know where else to look when there are obvious findings.

Normal Anatomy

Before considering injury patterns and mechanisms, it essential to have a working knowledge of the normal radiographic anatomy. The standard trauma series for the hand includes three views, which should cover the anatomy from the radiocarpal joint to the finger tips. These views are a pronated frontal view (PA), a pronated oblique view and a lateral view. For wrist injuries, these same three projections are used but are centered and collimated to cover the wrist area, from the metadiaphyses of the distal radius and ulna to the proximal metacarpal diaphyses. A fourth view, the so-called scaphoid view, should always be included in the wrist trauma series. This is a PA view, more tightly collimated than the other three, that is centered on the scaphoid, with the wrist in maximum ulnar deviation. This view rotates the scaphoid about its short axis, presenting the waist of the bone in profile.

When evaluating radiographs of the wrist, several anatomic points are important to observe. First, look at the soft tissues. On the lateral view, convexity of the dorsal soft-tissue margin represents soft-tissue swelling around the carpus and distal radius. It is often a sign of subtle underlying bone or joint injury. Also on the lateral view is the pronator fat pad, which lies parallel to the palmar cortex of the distal radius in most normal individuals. When the distal radius is fractured, the pronator fat pad will be deformed and displaced, becoming convex in a palmar direction. A second but less frequently present fat pad is the scaphoid fat pad. When present, it should be relatively straight and lateral and parallel to the scaphoid bone. If the scaphoid fat pad is convex laterally, a scaphoid fracture should be suspected.

There are several lines and angles that can be drawn in and around the carpus that are helpful in detecting injuries which may otherwise be overlooked. On the PA view, the three carpal arcs (of Gilula) are smooth curves that will be disrupted in injuries to the intercarpal joints. Arc I is drawn across the proximal surfaces of the proximal carpal row. Arc II is drawn across the distal surfaces of the proximal carpal row. Arc III is drawn across the proximal surfaces of the distal carpal row (Fig. 1). The long axis of the capitate, drawn on the PA view, should bisect the third metacarpal shaft regardless of the degree of ulnar or radial deviation (Fig. 1).

The second through fifth carpometacarpal joints should be seen in profile on a good-quality PA view, forming a “lazy M” shape on the radiograph (Fig. 1). While it may not always be possible to see the entire lazy M, most of it should be visible if the wrist is positioned correctly. The key to the carpometacarpal joints is to look at those joint surfaces that have been profiled by the X-ray beam. If one side of a joint (carpal or metacarpal) is seen in profile, the other side of that same joint should be seen in profile and parallel to its mate. When only one side is profiled or the articular surfaces are overlapping or not parallel, the joint is either subluxed or dislocated.

On the lateral view, the distal radial articular surface and proximal lunate articular surface should form parallel curves. Similarly, the distal lunate and proximal capi-
should form parallel curves (Fig. 2). If one or more of these articulations are not parallel, the carpus has been dislocated or subluxed. By determining the long axes of the scaphoid, lunate and capitate on the lateral view and measuring the angles between them, the presence of various carpal instabilities and/or ligament injuries can be predicted. The normal scapholunate angle lies between 30 and 60°. The normal capitolunate angle is ±30° (Fig. 3).

An increase in the scapholunate angle indicates a dorsal intercalated segment instability (DISI). A decrease in the scapholunate angle indicates a palmar intercalated segment instability (PISI). In both DISI and PISI, the capitolunate angle will usually be increased.

The articular cartilage has approximately the same thickness throughout the carpus. If the apparent space between any two carpal bones appears wider than the apparent space between the others, a ligament disruption has probably occurred. The joints most commonly affected by ligament injuries are the scapholunate and lunotriquetral joints. Therefore, the apparent space between the lunate and scaphoid and the lunate and triquetrum should always be carefully evaluated.

**Injury Patterns and Mechanisms**

The majority of upper-extremity injuries are the result of a fall onto the out-stretched hand (FOOSH). Many of these FOOSH injuries are concentrated around the wrist and some involve the hand. Those around the wrist are somewhat age-dependent. In very small children, whose bones are relatively soft, buckle or torus fractures of the distal radius are the most common injuries. While most of these are obvious, the findings may be limited to very subtle angulation of the cortex, seen only on the lateral view. These injuries are often associated with similar fractures of the distal ulna.

As adolescents enter the growth spurt associated with puberty, their physes become weaker and subject to fracture. The commonest FOOSH injuries in this age group are physial fractures of the distal radius, which may or may not be associated with ulnar fractures, particularly of the styloid process. These physial fractures are described in the Salter-Harris classification as follows: type 1, physial shear injury; type 2, physial shear with marginal metaphyseal fracture; type 3, physial shear with epiphyseal fracture; type 4, epiphyseal, physial and metaphyseal fractures; type 5, physial crush injury. In general, these injuries are displaced and easy to recognize, with exception of type 5 injuries. However, in some patients, partial auto-reduction may make a type 1 or 2 fracture difficult to find on the radiographs. Secondary signs, such as displacement the pronator fat pad, may be helpful.

In young adults, the bones are at their strongest. This puts the ligaments at increased risk. The center of most frequent injury moves to the carpus, where fractures and dislocations are most likely to occur in the so-called zone of vulnerability (Fig. 4). This zone runs in a curved manner across the radial styloid, scaphoid, capitate, triquetrum and ulnar styloid. The commonest injury within the zone of vulnerability is a scaphoid fracture. The second commonest is an avulsion fracture of the dorsal triquetrum. Next in frequency are various dislocations and fracture dislocations, involving predominantly the midcarpal joint. Scaphoid fractures are important to consider in all injured wrists for two reasons. First, they have a high incidence of nonunion and ischemic necrosis. Second, they tend to be truly nondisplaced and may be difficult to see on radi-
ographs taken on the day of injury. Follow up radiographs, after 2 weeks, will often show these occult fractures. If prompt diagnosis is needed, MRI is much more sensitive in revealing nondisplaced fractures than radiography.

In older adults, as osteoporosis sets in and the bones become weaker, the distal radius once again becomes the commonest site for FOOSH injuries. The most common variety of distal radial fracture is one in which the distal fracture fragment is displaced and angulated in a dorsal direction. This fracture was first described by Abraham Colles, in 1814, and now bears his name. Since Colles described this fracture 81 years before the discovery of X-rays, he did not know the detail or radiographic manifestations of this injury. His real contribution was to point out that these are fractures, not dislocations. He showed that they could be reduced and splinted and could heal with excellent results. When the deformity is in the opposite direction (palmar) we refer to the injury as a Smith’s fracture. When there is no deformity, the injury should be described simply as a nondisplaced, distal, radial fracture. Fractures of the ulnar styloid commonly occur in association with distal radial fractures but are not always present. Their presence does not change the designation as a Colles’, Smith’s or nondisplaced fracture. One of the most important findings to observe in these fractures is extension into the distal radial articular surface. Intra-articular fractures often require surgical repair and should be further evaluated with CT.

When fractures of the distal radius are associated with radiocarpal dislocations, they are referred to as “Barton’s fractures”. If the dorsal lip is fractured, the carpus will be displaced dorsally. This is referred to as a “dorsal Barton’s fracture”. Conversely, if the palmar lip of the radius is fractured, the carpus will be displaced palmarly. This is referred to as a “palmar Barton’s fracture”. While pure dislocations of the radiocarpal joint can occur without radial lip fractures, they are much less frequent than Barton’s fracture-dislocations.

Carpal dislocations

Most carpal dislocations involve the midcarpal joint, which is between the proximal and distal carpal rows. On the lateral view, these injuries show disruption of the normal relationship between lunate and capitate, usually with dorsal displacement of the capitate. The distal articular surface of the lunate is “empty”. On the PA projection, the lunate takes on a triangular shape as it rotates about its horizontal axis. Arcs I and II are disrupted, while arc III is normally intact. These dislocations usually occur around the lunate and are therefore called “perilunate” dislocations. The majority of perilunate dislocations are associated with fractures through the scaphoid waist but any fracture within the zone of vulnerability is possible. Perilunate dislocation without an associated fracture is not uncommon. The description of the injury includes the fractures and the words “perilunate dislocation”. For example: a trans-radial, trans-scaphoid, trans-capitate, perilunate dislocation would be one of these dislocations with fractures through the radial styloid, scaphoid waist and capitate neck. Ulnar styloid fractures are frequently present but are usually not included in the descriptive classification. When the lunate is displaced from the radial articular surface in a midcarpal joint disruption, it is called a “lunate dislocation”. “Midcarpal dislocation” is the term used to describe the intermediate position, when the capitate is dislocated from the lunate and the lunate is subluxed from the radius. This term is confusing, since all of these patterns are dislocations of the midcarpal joint.

Other, less-common, carpal dislocations include the longitudinal variety. These are the result of high-energy trauma and separate the carpus into medial and lateral portions. They are usually obvious radiographically and frequently require surgical repair.

Carpometacarpal dislocations

Perhaps the most commonly missed serious injury to the hand and wrist is dislocation along the carpometacarpal joint. These injuries can be surprisingly subtle on initial radiographs. In spite of this, they are serious injuries that usually require surgical repair. There are two keys to finding them: (1) they are frequently associated with avulsion fractures of the distal carpals or proximal metacarpals; (2) on at least one of the standard views, the affected carpometacarpal joints will show loss of parallelism. On the lateral radiograph, dorsal displacement of the metacarpal bases may be apparent. So, the important point to remember is: any time a fracture at the carpometacarpal junction is seen, a dislocation must be assumed, until proven otherwise.

CT or fluoroscopy may be required to resolve this issue.