2 Flexor Digitorum Profundus Avulsion ("Jersey Finger")

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BACKGROUND

Avulsion of the flexor digitorum profundus (**"jersey finger"**) can occur in any digit, but it is most common in the ring finger. This injury usually occurs when an athlete grabs an opponent's jersey and feels sudden pain as the distal phalanx of the finger is forcibly extended as it is concomitantly actively flexed (hyperextension stress applied to a flexed finger).

The resultant lack of active flexion of the DIP joint (FDP function loss) must be specifically checked to make the diagnosis (Fig. 2.1). Often the swollen finger assumes a position of extension relative to the other, more flexed fingers. The level of retraction of the FDP tendon back into the palm generally denotes the force of the avulsion.

Leddy and Packer (1977) described three types of FDP avulsions based on where the avulsed tendon retracts: type I, retraction of the FDP to the palm; type II, retraction to the proximal interphalangeal (PIP) joint; and type III, bony fragment distal to the A4 pulley. Smith's (1981) case report described a type III lesion associated with a simultaneous avulsion of the FDP from the fracture fragment. He suggested adding this pattern as a type IV, though he was not the first surgeon to comment on this anomaly. Al-Qattan (2001) reported a case series of type IV fracture with other significant concomitant distal phalanx fractures. He offers an extension of the classification to type V. As the complexity of the bony involvement increases, priorities shift to maintaining articular congruency, pilon fractures, bony mallet, and osseous stability such as shaft fractures, over early tendon excursion. This is logical and then allows extrapolation to treat such anomalies as FDP avulsions through enchondromas (Froimson and Shall 1984).

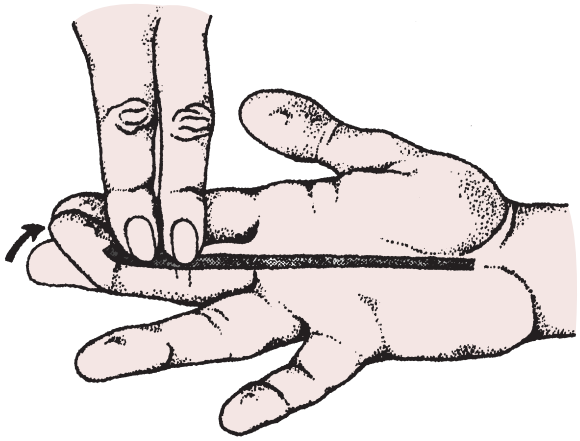

Fig. 2.1 With avulsion of the flexor digitorum profundus, the patient would be unable to flex the distal interphalangeal (DIP) joint, shown here. (From Regional Review Course in Hand Surgery. Rosemont, Illinois, American Society of Surgery of the Hand, 1991, Fig. 7).

TREATMENT

The treatment of FDP avulsion is primarily surgical. The success of the treatment depends on the acuteness of diagnosis, rapidity of surgical intervention, and level of tendon retraction. Tendons with minimal retraction usually have significant attached avulsion bone fragments, which may be reattached bone-to-bone as late as 6 weeks. Tendons with a large amount of retraction often have no bone fragment and have disruption of the vascular supply (vinculum), making surgical repair more than 10 days after injury difficult because of retraction and the longer healing time of the weaker nonbone-to-bone fixation and limited blood supply to the repair. Based on a review of the literature and their clinical experience, Henry et al. (2009) listed four essentials for successful treatment of type IV extensor tendon injuries: (1) a high index of suspicion for this injury, with the use of magnetic resonance imaging (MRI) or ultrasound for confirmation if needed, (2) rigid bony fixation that prevents dorsal subluxation of the distal phalanx, (3) tendon repair that is independent of the bony fixation, and (4) early range of motion therapy (Rehabilitation Protocol 2.1).

Surgical salvage procedures for late presentation include DIP joint arthrodesis, tenodesis, and staged tendon reconstructions. Not all cases of early presentation result in tendon repair. Patient health issues may dictate a nonoperative course as being the most prudent. Patients with preexisting joint disease such as rheumatoid arthritis, osteoarthritis, and gout may be better served by a salvage procedure.

Fixation of the simple bone fragments is best achieved via lag screw fixation with appropriate-sized screws and standard AO technique. Power and Rajaratnam (2006) describe modifying an AO/Synthes modular hand plate by cutting through a hole and bending the resultant prongs to create a hook plate, thereby stabilizing the fracture.

TENDON-TO-BONE REPAIR CONSIDERATIONS

Silva et al. (1998) showed that Bunnell and Kleinert suture techniques had better load characteristics than modified Kessler using 3-0 Prolene (Ethicon, Sommerville, NJ) suture over a button. However, gapping of 8 mm occurred across suture patterns at 20 N, bringing into question the choice of suture material or number of strands. Later work demonstrated improved load to failure with more strands, yet gapping was still a problem. Brustein et al. (2001), in a cadaveric model, showed a 50% improvement in mean load to failure with a four-strand

modified Becker, two micro Mitek (Mitek Pruducts, Norwood, MA) anchor compared to monofilament Bunnell pull-out or single mini Mitek Bunnell. Boyer et al. (2002) compared 3-0 and 4-0 braided suture four-strand through bone modified Kessler and modified Becker in a load to failure model. The 3-0 modified Becker proved a significantly higher load to failure; however, the strain at 20 N load did not differ among the groups. The described models were static load to failure.

Latendresse et al. (2005) performed cyclic load testing of Prolene versus braided polyester, pull-out button extraosseous versus mini Mitek. Gap formation was 2 mm or less for the braided suture, significantly better than the monofilament groups. Load to failure was better in the extraosseous repairs, though all were greater than 20 N. Abboud et al. (2002) colinearly load tested pronged and threaded commercially available anchors in cadaveric carpal bones. They report dramatic failure of the pronged anchors compared to the threaded anchors. There are many potential confounding factors: anchor angle collinear with load, dense cortical and subchondral bone for screw purchase as opposed to cancellous, and size of the implant. This may not prove that threaded anchors will hold similarly in a distal phalanx with thinner cortex and smaller diameter. The Biomet JuggerKnot 1.4-mm suture anchor (Biomet, Warsaw, IN) reports 90 N pull-out force with a 3-0 braided suture and 115 N with a 2-0 braided suture. I have not seen cyclic loading data on this construct; however, its compact structure should be kept in mind as an option for the smaller bones.

McCallister et al. (2006) reported on clinical follow-up on 26 consecutive zone I injuries. Thirteen patients were repaired via extraosseous pull-out button and 2-0 braided polyester modified Kessler suture. The remaining 13 were repaired with 2 micro Mitek 3-0 braided polyester hemi-modified Kessler sutures tied deep to the tendon. The only significant difference between the groups was that the time to return to full-duty work was shorter in the anchor group than the pull-out suture group. Chu et al.'s (2013) cadaveric research failed to show a significant difference between standard anchor, pull-out, and a new technique of tying the suture over the distal phalanx buried proximal to the germinal matrix. This gives another option in the surgeon's arsenal, one that doesn't require further expense.

SURGEON'S PREFERENCE

I currently use mini JuggerKnot with a 3-0 braided modified Becker, two anchors side by side if bone size allows. If I feel any concern about the quality of the anchor placement or holding power, I have been adjusting to an extraosseous pull-out technique. With Chu's 2013 publication, I may consider this as my primary repair and certainly my bail-out for anchor difficulties. I only débride the tendon minimally with tenotomy scissors. I am concerned that using a tendon cutter to produce a tidy tendon end has already functionally advanced the tendon. Given Chepla et al.'s (2015) anatomic analysis of the FDP footprint and the length of many suture anchors, we may have been unintentionally advancing the tendon distally to seat the metallic anchors. Using the JuggerKnot can minimize this bias. I personally do not mind attached periosteum or frayed tendon edges because I use a 4-0 absorbable, hug the radial and ulnar distal phalanx edges, and suture the material down. I believe the scarring down of this material can only support the repair.

REHABILITATION PROTOCOL 2.1 Rehabilitation Protocol After Surgical Repair of Jersey Finger With Secure Bony Repair

S. Brent Brotzman, MD

0–10 Days

- DBS the wrist at 30 degrees flexion, the MCP joint 70 degrees flexion, and the PIP and DIP joints in full extension
- Gentle passive DIP and PIP joint flexion to 40 degrees within DBS
- Suture removal at 10 days

10 Days–3 Weeks

- Place into a removable DBS with the wrist at neutral and the MCP joint at 50 degrees of flexion.
- Gentle passive DIP joint flexion to 40 degrees, PIP joint flexion to 90 degrees within DBS
- Active MCP joint flexion to 90 degrees
- Active finger extension of IP joints within DBS, 10 repetitions per hour

3–5 Weeks

- Discontinue DBS (5–6 weeks).
- Active/assisted MCP/PIP/DIP joint ROM exercises
- Begin place-and-hold exercises.

5 Weeks +

- Strengthening/power grasping
- Progress activities
- Begin tendon gliding exercises.
- Continue PROM, scar massage.
- Begin active wrist flexion/extension.
- Composite fist and flex wrist, then extend wrist and fingers

With Purely Tendinous Repair or Poor Bony Repair (Weaker Surgical Construct)

0–10 Days

- DBS the wrist at 30 degrees flexion and the MCP joint at 70 degrees flexion
- Gentle passive DIP and PIP joint flexion to 40 degrees within DBS
- Suture removal at 10 days

10 Days–4 Weeks

- DBS the wrist at 30 degrees flexion and the MCP joint at 70 degrees flexion
- Gentle passive DIP joint flexion to 40 degrees, PIP joint flexion to 90 degrees within DBS, passive MCP joint flexion to 90 degrees
- Active finger extension within DBS
- Remove pull-out wire at 4 weeks.

4–6 Weeks

- DBS the wrist neutral and the MCP joint at 50 degrees of flexion
- Passive DIP joint flexion to 60 degrees, PIP joint to 110 degrees, and MCP joint to 90 degrees
- Gentle place-and-hold composite flexion
- Active finger extension within DBS
- Active wrist ROM out of DBS

6–8 Weeks

- Discontinue daytime splinting; night splinting only
- Active MCP/PIP/DIP joint flexion and full extension

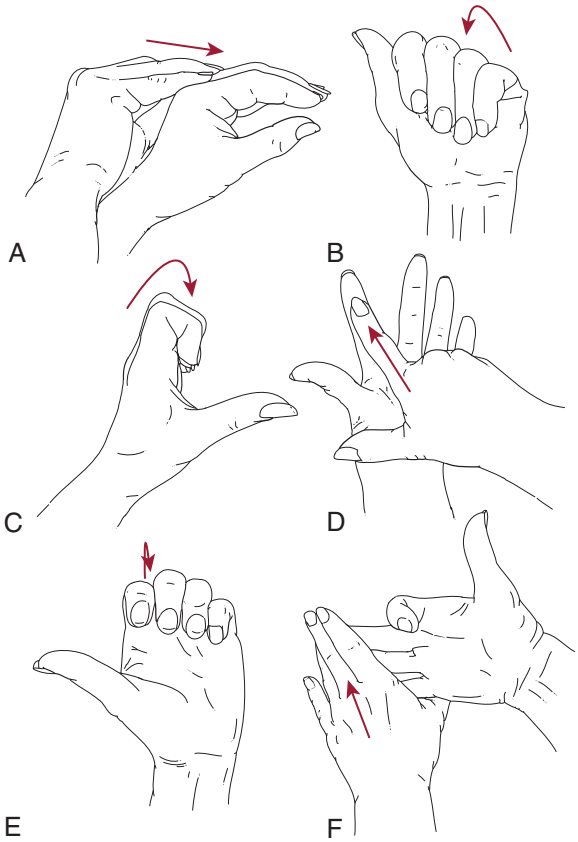

Fig. 4.1 Tendon glide exercises. **A,** Intrinsic plus posture to achieve central slip/lateral bands glide over proximal phalanx (P1). **B,** Sublimis fist posture to promote selective FDS tendon glide. **C,** Claw posture to achieve extensor digitorum communis (EDC) tendon glide over metacarpal bone. **D,** Flexor digitorum profundus (FDP) blocking exercises to glide FDP tendon over P1. **E,** Hook fist posture to promote selective FDP tendon glide. **F,** Flexor digitorum sublimis (FDS) blocking exercise to glide FDS tendon over middle phalanx.

Allowing early PIP and DIP joint motion is essential. Motion prevents adhesions between the tendons and the underlying fracture and controls edema.

PHALANGEAL FRACTURES OF THE HAND

- Phalangeal fractures lack intrinsic muscle support, are more unstable than metacarpal fractures, and are adversely affected by the tension in the long tendons of the fingers.
- Because of the pull of the FDS insertion into the middle phalanx, a proximal fracture of the middle phalanx will angulate with the fracture apex dorsal and a distal fracture will involve angulation with the apex volar (Fig. 5.3). Because of the deforming tendon forces, fractures in these areas that present initially as displaced are unlikely to remain reduced after reduction and typically require operative fixation.
- Phalangeal fractures respond less favorably to immobilization than metacarpal fractures, with a predicted 84% return of motion compared with 96% return of motion in the metacarpals (Shehadi 1991).
- If phalangeal immobilization is continued for longer than 4 weeks, the motion drops to 66%.
- Reasons cited for poor results in the literature typically are comminuted fractures, open fractures, and multiple fractures.

TABLE 4.1 Potential Problems With Phalangeal Fractures and Strategies for Therapeutic Intervention

Maureen A. Hardy PT, MS CHT

Potential Problems	Prevention and Treatment
Loss of MP flexion	Circumferential PIP and DIP extension splint to concentrate flexor power at MP joint; NMES to interossei
Loss of PIP extension	Central slip blocking exercises; during the day MP extension block splint to concentrate extensor power at PIP joint; at night PIP extension gutter splint; NMES to EDC and interossei with dual-channel setup
Loss of PIP flexion	Isolated FDP tendon glide exercises; during the day MP flexion blocking splint to concentrate flexor power at PIP joint; at night flexion glove; NMES to FDS
Loss of DIP extension	Resume night extension splinting; NMES to interossei
Loss of DIP flexion	Isolated FDP tendon glide exercises; PIP flexion blocking splint to concentrate flexor power at DIP joint; stretch ORL tightness; NMES to FDP
Lateral instability, any joint	Buddy strap or finger-hinged splint that prevents lateral stress
Impending boutonnière deformity	Early DIP active flexion to maintain length of lateral bands
Impending swan-neck deformity	FDS tendon glide at PIP joint and terminal extensor tendon glide at the DIP joint
Pseudo claw deformity	Splint to hold MP joint in flexion with PIP joint full extensor glide
Pain	Resume protective splinting until healing is ascertained; address edema, desensitization program

MP, metacarpophalangeal; PIP, proximal interphalangeal; DIP, distal interphalangeal; NMES, neuromuscular electrical stimulation; EDC, extensor digitorum communis; FDP, flexor digitorum profundus; FDS, flexor digitorum superficialis; ORL, oblique retinacular ligament.

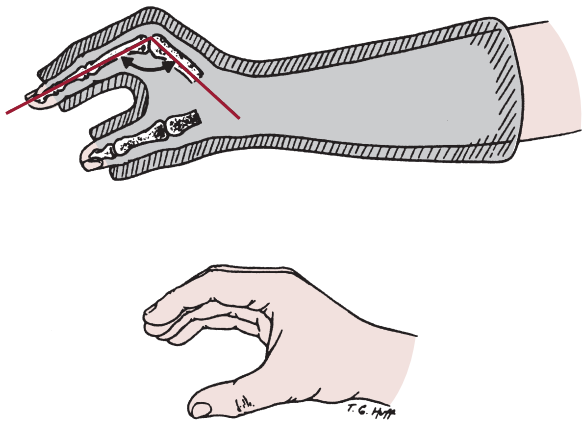

Fig. 4.2 Position of immobilization of the hand involves splinting the wrist in approximately 30 degrees of extension, the metacarpophalangeal (MCP) joints in 60 to 80 degrees of flexion, and the interphalangeal (IP) joints in full extension. (From Delee J, Drez D [eds]: Orthopaedic Sports Medicine. Philadelphia, WB Saunders, 1994.)

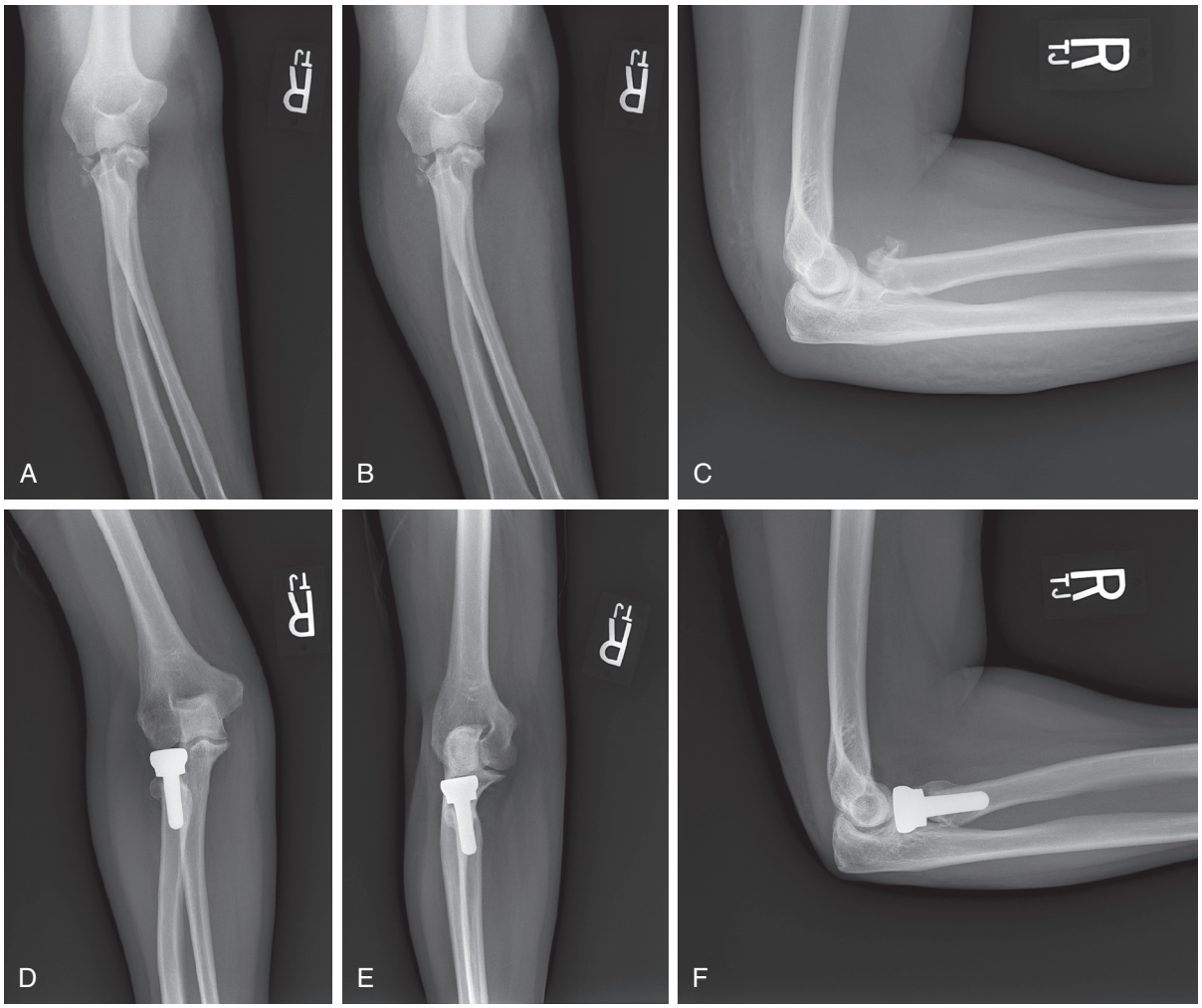

Fig. 12.2 39-year-old male who fell down a flight of stairs and sustained a comminuted radial head fracture treated with radial head arthroplasty. Images **A-C** are preoperative images and images **D-F** demonstrate the radial head component.

healing but generally consists of active and active-assisted ROM exercises. Passive exercises are discouraged. Non-weight bearing occurs for six to eight weeks until bony union is achieved but ultimately initiation of weight bearing is left to the discretion of the surgeon. The posterior interosseous nerve (PIN) may be injured or stretched during surgery and the therapist must inform the surgeon if PIN symptoms are present. The rehab protocol is changed with associated ligamentous disruptions.

SIMPLE OR COMPLEX ELBOW DISLOCATIONS

Elbow dislocations can be broken up into simple and complex patterns. Simple dislocations of the elbow are those in which the injury is only ligamentous without any associated fractures. Complex dislocations are elbow dislocations with associated fractures.

For the majority of simple dislocations, the treatment is nonoperative with initiation of early ROM. Conscious sedation is generally all that is required to reduce the elbow back into place. When the elbow is reduced the treating physician will place the elbow through a ROM with varus and valgus stress. As long as the elbow is stable throughout the arc of motion the patient is generally placed in a splint with the elbow flexed to 90 degrees for a week. Early ROM with a therapist is encouraged. If the elbow is unstable throughout an arc of motion the physician will splint the elbow in a reduced position and may elect for ligamentous repair. The therapy initiated after repair should follow that in the ligamentous repair protocol chapter (Rehabilitation protocols 12.1 and 12.2).

Complex elbow dislocations are elbow dislocations that include associated fractures. The most common associated fractures include the radial head, coronoid process of the ulna, and the olecranon. The "terrible triad" elbow dislocation commonly mentioned is an elbow dislocation with injuries to the coronoid process, radial head, and lateral collateral ligament. Complex elbow dislocations may be divided further into the particular mechanism: axial, valgus posterolateral rotatory, and varus posteromedial rotatory injuries (Wyrick 2015). The majority of complex fracture dislocations are treated with surgery. The postoperative guidelines generally are dictated by what bony structures are repaired. If any ligamentous structures are concomitantly repaired, then the postoperative protocol will also protect any stress to the repaired collateral ligament.

an overhead throwing sport (the "peel-off" lesion associated with a tight posterior capsule).

SCAPULOTHORACIC DYSKINESIA, CORE STABILITY DEFICITS, AND OTHER FITNESS OR TECHNIQUE-RELATED PROVOCATIONS

Scapulothoracic dyskinesia, core stability deficits, and other fitness issues commonly contribute to shoulder symptoms as a result of secondary irritation of the rotator cuff or other muscle–tendon units resulting from biomechanical overload. There is frequently a history of the atraumatic insidious onset of shoulder pain associated with participation in a new recreational or occupational activity.

ADHESIVE CAPSULITIS ("FROZEN SHOULDER")

The typical "frozen shoulder" is not caused by trauma, although patients will often retrospectively recall some history of minor injury to which they ascribe the symptoms. Characteristically, patients first recognize the problem when they find it difficult to reach behind their back (secondary to an evolving internal rotation deficit). Symptoms are progressive, with "freezing," "frozen," and "thawing" stages having been defined to describe the natural history of the problem. Frequently, secondary rotator cuff pain will account for a substantial portion of the subjective symptoms. Patients may also describe posterior shoulder discomfort with a trapezius or periscapular location because those muscles become fatigued when compensating for poor glenohumeral motion. There is a significant association with diabetes and hypothyroidism, and patients should be questioned about those general health conditions. Adhesive capsulitis occurs bilaterally in this group.

CALCIFIC TENDINITIS

Calcific tendinitis is characterized by the insidious, but rapid, development of extremely severe subacromial or lateral-sided shoulder pain, characteristically in patients of middle age. Narcotics are often necessary to control the discomfort.

BICEPS TENDINOSIS

With advancing age, pathology in the long head of the biceps becomes a frequent source of shoulder pain. Biceps tendinosis is often associated with rotator cuff disease. However, pain originating in the biceps is referred to the anterior arm, as opposed to cuff disease, which is characteristically lateral. It may radiate to the elbow but not typically beyond. Because the biceps is a supinator of the forearm, patients with biceps pathology may complain of symptoms related to forearm rotation (i.e., when turning a doorknob).

ACROMIOCLAVICULAR DEGENERATIVE JOINT DISEASE

The symptom originating from the AC joint most typically is pain over the superior shoulder that increases with horizontal adduction of the arm (because that compresses the AC joint) or use of the affected arm overhead. Injury to the AC joint can occur with a fall onto the lateral shoulder, AC joint arthritis can develop insidiously over a lifetime of use or from prior trauma, and an inflammatory condition known as "osteolysis of the distal clavicle" is associated with weight lifting in young adults. AC joint disease may produce scapulothoracic dyskinesia and secondary rotator cuff discomfort.

GLENOHUMERAL DEGENERATIVE JOINT DISEASE

Glenohumeral arthritis is an uncommon condition producing generalized aching shoulder pain and progressive loss of motion. GH arthritis may be associated with a history of previous surgical procedures (open ligament stabilization, arthroscopic repair of large labral tears, and the use of implantable "pain pumps") and massive rotator cuff tears (cuff tear arthropathy), particularly in elderly women. Symptoms are often maximal at night and more tolerable during daily activities. Systemic rheumatoid arthritis may affect the glenohumeral joint, but particularly in younger individuals, it involves the AC or SC joints.

CERVICAL SPINE PATHOLOGY

Cervical spine disease typically produces pain radiating from the neck toward the posterior or superior shoulder. The pain is usually worse at the end of the day and relieved by support of the head at night. Generally, patients will experience pain and stiffness with neck motion. Especially in the elderly, a coincident association with rotator cuff disease is common. When cervical nerve root compression is present, most commonly C5 and C6 are affected, and radicular symptoms ("sharp," "stabbing," or "burning pain") involving the forearm and hand radiate distal to the elbow in a typical dermatomal distribution.

FRACTURES

Fractures about the shoulder are not uncommon in all age groups. Typically, there is a specific history of trauma, but in the osteoporotic elderly or other special situations, the injury may seem to be of minimal force. The mechanism may be direct (a fall or blow to the shoulder) or indirect (a fall on an outstretched arm). Characteristically, pain is immediate after trauma, localized to the specific point of injury, and severe enough to leave little doubt as to the nature of the problem. Diagnosis is confirmed by radiographs.

GENERAL SHOULDER REHABILITATION GOALS

Range of Motion

Once the intake evaluation is completed, the therapist should be more comfortable anticipating the patient's response to the therapeutic regimen. One of the main keys to recovery is to normalize ROM. Early professions relied on visual estimations or "quick" tests to assess shoulder motion. These tests include combined shoulder movements such as the Apley's scratch test (Fig. 21.2), reaching across the body to the other shoulder (Fig. 21.3), or reaching behind the back to palpate the highest spinous process (Fig. 21.4). These quick tests are great to observe for overall asymmetry, but they cannot give an idea of isolated losses objectively.

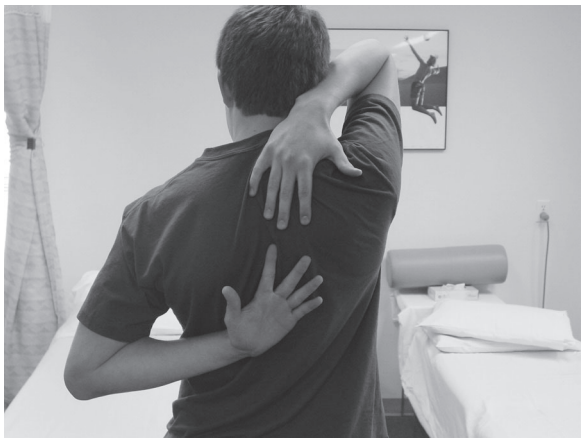

Fig. 21.2 Apley's scratch test.

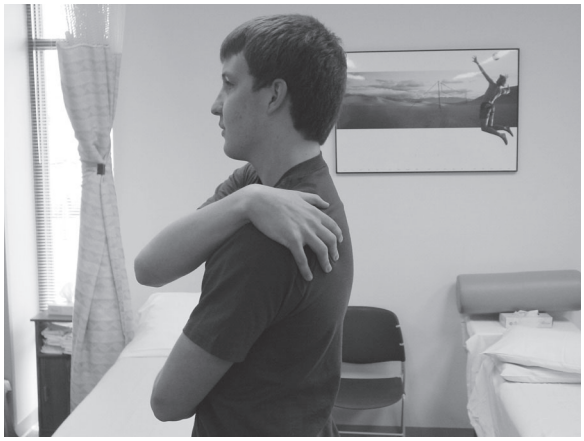

Fig. 21.3 Reaching across the body to the other shoulder to determine range of motion.

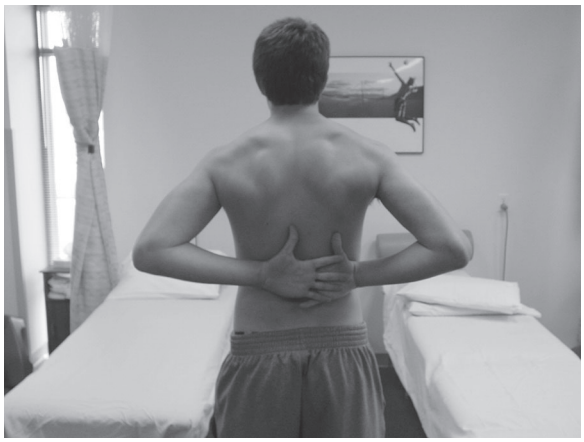

Fig. 21.4 Reaching behind the back to palpate the highest spinous process to determine range of motion.

Even more important is regaining normal arthrokinematic motions at the shoulder. Active shoulder ROM is always gathered before passive motions (Manske and Stovak 2006). Active shoulder ROM measurements are seen in Table 21.1 (Manske and Stovak 2006) and Fig. 21.5. Many times, gross overall shoulder motion may appear to be only slightly limited, whereas arthrokinematic motion is drastically dysfunctional. For example, it is not uncommon for a patient to have full glenohumeral motion yet impinge as a result of altered scapulohumeral motion from a restricted inferior or posterior shoulder capsule creating obligate humeral translations.

Therefore, it is imperative to also ensure evaluation of isolated glenohumeral motions is performed. One of the more common problematic limited motions with a variety of shoulder conditions is that of the posterior or inferior shoulder structures. Debate continues as to whether this is a result of capsular or other soft tissues. Regardless, it becomes an issue whenever elevation of the glenohumeral joint is required because it may increase the risk of impingement. Assessment of the posterior shoulder can be done by measuring isolated glenohumeral internal rotation. To perform this test the humerus is taken into passive internal rotation while the scapula is stabilized by grasping the coracoid process and the spine and monitoring for movement (Fig. 21.6). When passive slack from the posterior shoulder is taken up, the humerus will no longer internally rotate and resistance to movement will allow the scapula to tilt forward. When motion is detected or internal rotation has ceased, the examiner measures isolated glenohumeral internal rotation. Wilk et al. (2009) have shown this to be moderately reliable, whereas Manske et al. using the same technique have proved excellent test–retest reliability (Manske et al. 2015). This motion should be compared bilaterally to assess for a glenohumeral internal rotation deficit (GIRD) between involved and uninvolved shoulders. A difference of greater than 20 degrees of internal rotation is thought to be a precursor to shoulder pathology. Loss of shoulder internal rotation is not always pathologic because some of this motion may be lost as a result of bony changes in the humerus. The concept of total shoulder rotation ROM should also be mentioned. By adding the two numbers of GH internal rotation and external rotation together, a composite of total shoulder motion can be obtained (Fig. 21.7). Ellenbecker et al. (2002), measuring bilateral total rotation ROM in professional baseball and elite junior tennis players, found that although a dominant arm may show increased external rotation and less internal rotation, the total ROM was not significantly different when comparing the two shoulders. However, when Wilk and colleagues (2009) examined professional baseball pitchers, they found that those whose total ROM limitation exceeded more than a 5-degree difference were more prone to injury resulting in loss of playing time. Therefore, one needs to not only address the GIRD but also should ensure that the total ROM is not limited. Using normative data from population specific research can assist the therapist in interpreting normal ROM patterns and identifying when sport-specific adaptations or clinically significant adaptations are present (Ellenbecker 2004). Because there seems to be a threshold to determine what can be considered a clinically significant loss of internal rotation, Manske et al. (2015) have suggested two descriptions of naming GIRD—one which is pathologic and one which is a normal, nonpathologic alternation of shoulder motion in overhead athletes.

Early in rehabilitation following soft tissue shoulder repairs passive motion may predominate. These passive ROMs can be performed using Codman circumduction exercises or by therapist assistance. Passive motions can be gained in all classical directions as long as there are no soft tissue limitations. Other methods of gaining motion are through joint mobilizations.

Passive and active assistive exercises initially should begin with the patient in a supine position with the arm comfortably

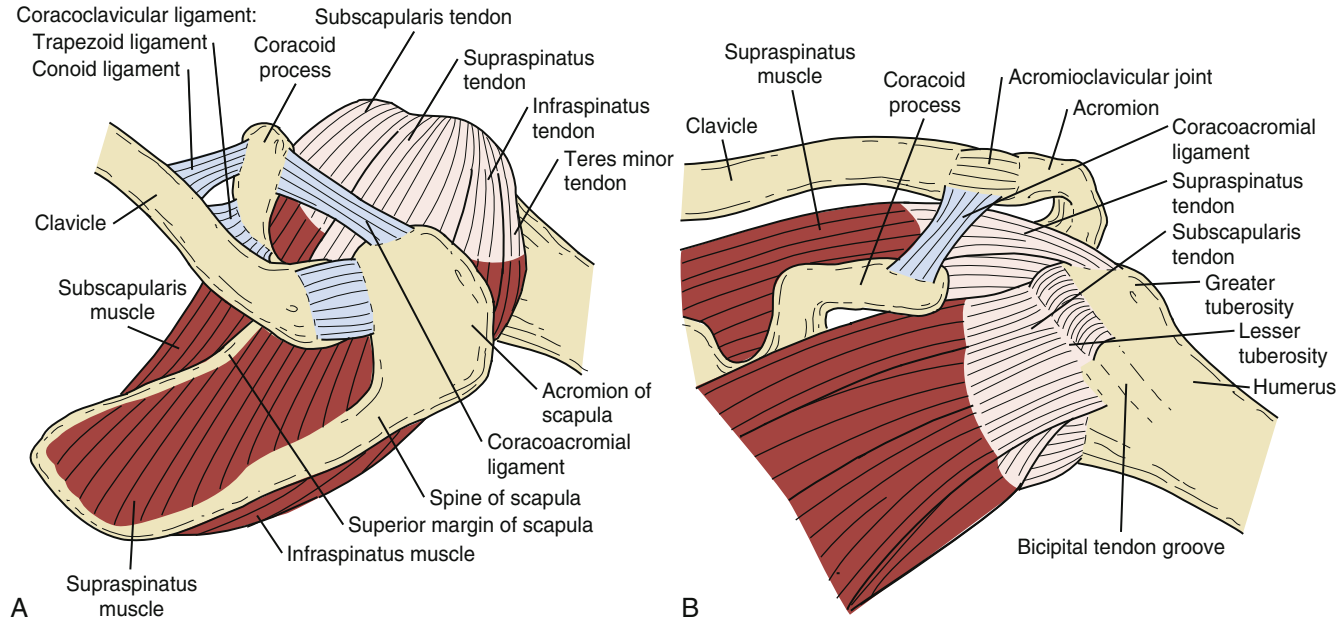

Fig. 23.1 A, superior view of rotator cuff musculature as it courses anteriorly under the coracoacromial arch to insert on the greater tuberosity. B, Anterior view of the shoulder reveals the subscapularis, which is the only anterior rotator cuff muscle that inserts onto the lesser tuberosity. (From Magee DJ, Zachazewski JE, Quillen WS, Manske RC: Pathology and Intervention in Musculoskeletal Rehabilitation. Elsevier Saunders, 2016.

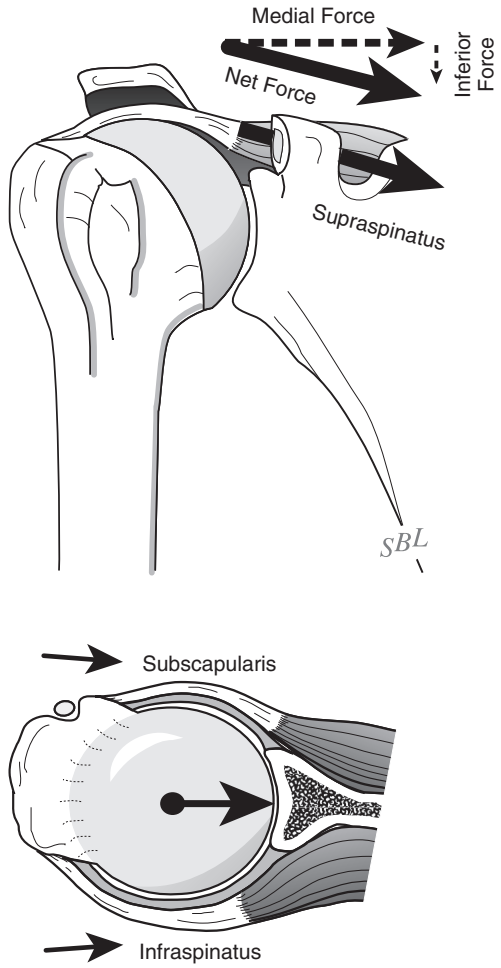

Fig. 23.2 Compressions into the glenoid concavity. The rotator cuff compresses the humeral articular convexity into the glenoid concavity. (Modified from Magee DJ, Zachazewski JE, Quillen WS, et al. Pathology and Intervention in Musculoskeletal Rehabilitation, 2nd edition, 2016, p. 244.)

BOX 23.1 CLASSIFICATION OF TYPES OF ROTATOR CUFF TEARS

Partial-thickness tears
Full-thickness tears
Acute tears
Chronic tears
Traumatic tears
Degenerative tears

TABLE 23.1 Tear Sizes

Name	Centimeters
Small	(0–1 cm^2)
Medium	(1–3 cm^2)
Large	(3–5 cm^2)
Massive	(>5 cm^2)

The all-arthroscopic repair of the rotator cuff actually has a slower rate of early rehabilitation progression owing to the weaker fixation of the repair as compared to that of the open procedures. This technique has to be one of the more demanding ways to operatively repair the rotator cuff. Advantages of the all-arthroscopic technique include preservation of the deltoid attachment, less postoperative pain, decreased surgical morbidity, and an earlier return of function following repair.

Regardless of the surgical approach performed, the underlying biology of healing tendons must be respected for all patients.

TEAR PATTERN

Lo and Burkhart (2003) have described four main types of tear patterns, and these include crescent-shaped tears, U-shaped tears, L-shaped and reverse L-shaped tears, and massive tears. Understanding of and recognition of the tear pattern are the first steps in determining appropriate surgical treatment.

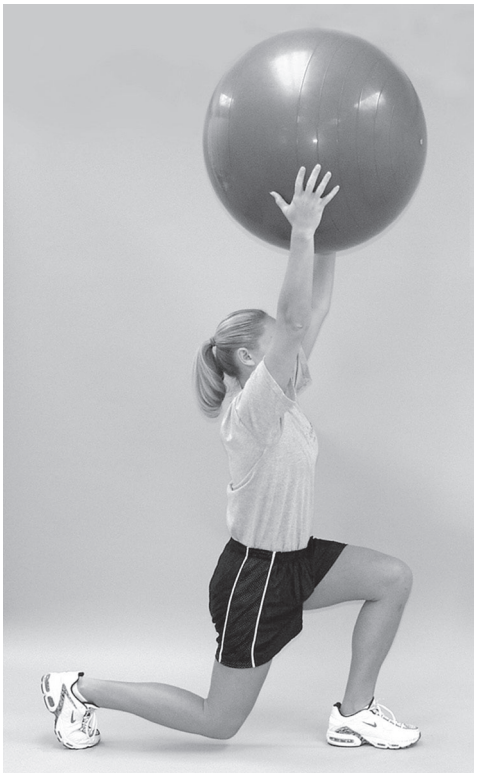

Fig. 50.1 The subject shows excellent body control position in this forward lunge, balancing the ball directly overhead. (Reprinted with permission from Ireland M. The Female Athlete. Philadelphia, Saunders 2002, p. 518, Fig. 43-5.)

Fig. 50.2 In bridging, the left greater trochanter is lifted off the floor while maintaining balance on the ball; support is given by the upper extremity. As advanced control occurs, less hand support is required. (Reprinted with permission from Ireland M. The Female Athlete. Philadelphia, Saunders 2002, p. 518, Fig. 43-8.)

in excessive loading on the uninvolved extremity that lacks sufficient strength and motor control to absorb force when involved in a competitive, athletic situation. Resolution of these final impairments may not only lead to successful reintegration to sports but also may begin to reduce the extraordinarily high incidence of re-injury after return to sports. The program that we developed and described attempted to utilize the best current available evidence and supplemented any deficits in the literature with expert clinical opinion. The final outcome was designed as a template and may stimulate future research attempting to develop more rigorous treatment progressions designed for the end stages of rehabilitation after any lower extremity injury, in addition to designing valid, reliable, and objective means to determine the athlete's readiness to successfully and safely return to sport with minimal risk of re-injury (see Rehabilitation Protocols 50.1 and 50.2).

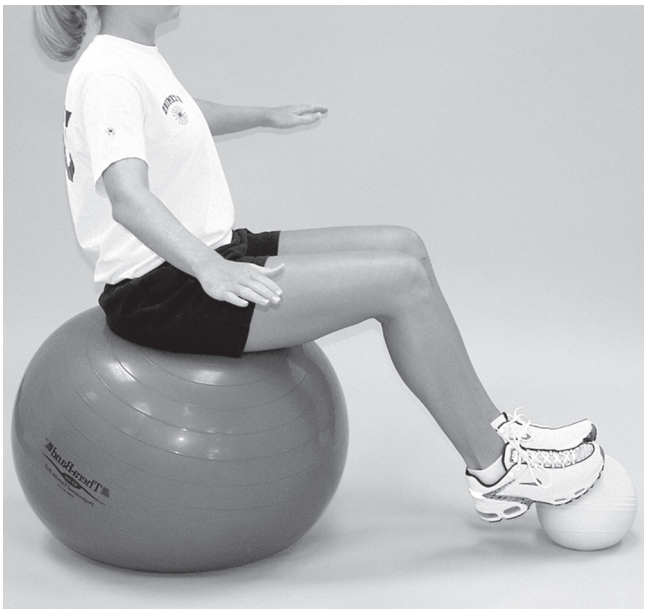

Fig. 50.3 Incorporating balance while seated on an unstable base is shown. Such advanced Swiss ball maneuvers incorporate position awareness and strength. Modifications of these exercises can be made to maintain the interest of the patient. (Reprinted with permission from Ireland M. The Female Athlete. Philadelphia, Saunders 2002, p. 518, Fig. 43-9.)

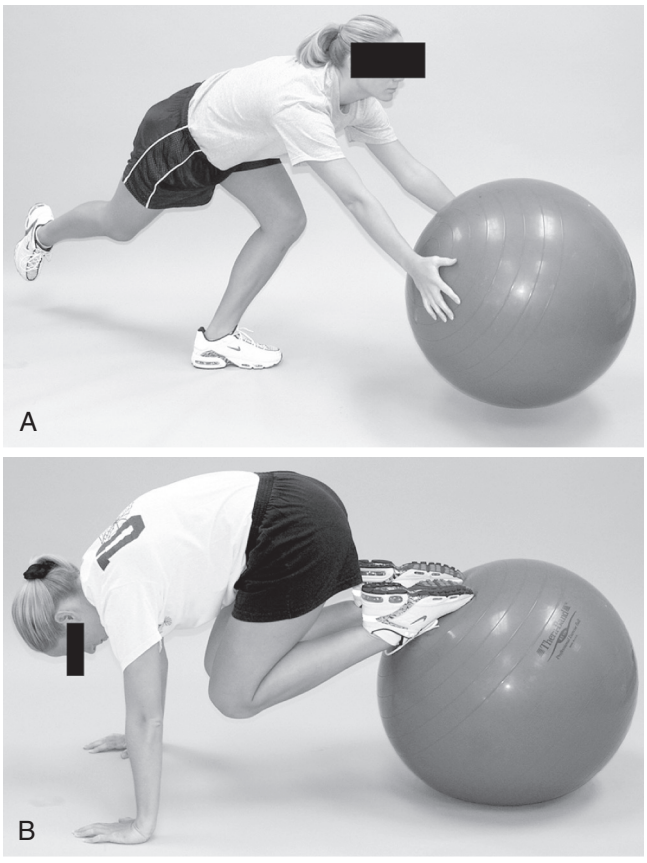

Fig. 50.4 A, The model is in the "around the clock" position, touching the ball to the floor and extending the right leg. B, In the prone balance position the subject maintains control; going from hip flexion and knee flexion into extension combines for core stabilization, balance, and neuromuscular control. (Reprinted with permission from Ireland M. The Female Athlete. Philadelphia, Saunders 2002, p. 518, Figs. 43-6 and 43-7.)

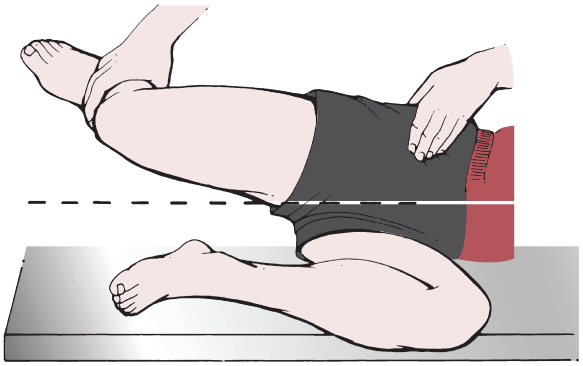

Fig. 56.11 Ober test assesses iliotibial band tightness. The unaffected hip and the knee are flexed. The involved knee is flexed 90 degrees, and the ipsilateral hip is abducted and hyperextended. A tight iliotibial band will prevent the extremity from dropping below the horizontal. (Reprinted with permission from DeLee J: Delee & Dreez's Orthopaedic Sports Medicine, ed 2. Philadelphia, Saunders, 2002, Fig. 28E10.4.)

J-Sign

The J-sign refers to the inverted J path the patella takes in early knee flexion (or terminal knee extension) as the patella begins its path from a laterally subluxated starting position and then suddenly shifts medially as it engages the bony femoral trochlear groove (or the reverse in terminal extension). It is indicative of possible patellar maltracking and/or patellar instability (Fig. 56.12).

Examination for knee instability should include a full evaluation of the cruciate and collateral ligaments to assess for any rotatory component and to examine the patellar restraints. Patients with posterolateral corner knee instability may develop secondary patellar instability owing to a dynamic increase in the Q-angle. Similarly, patients with chronic MCL laxity may also develop secondary patellar instability. Apprehension on medial or lateral displacement testing of the patella should raise the suspicion of underlying instability in the patellar restraints. Superior and inferior patellar mobility should also be assessed; they may be decreased in situations of global contracture.

Patellar Glide Test

The patellar glide test is useful to assess the medial and lateral patellar restraints. In full extension, the patella lies above the trochlear groove and should be freely mobile both medially and laterally. As the knee is flexed to 20 degrees, the patella should center in the trochlear groove, providing both bony and soft tissue stability.

Lateral Glide Test

The lateral glide test evaluates the integrity of the medial restraints. Lateral translation is measured as a percentage of patellar width (Fig. 56.13). Translations of 25% of patellar width are considered normal; translations greater than 50% indicate laxity within the medial restraints. The medial patellofemoral ligament (MPFL) has been noted to provide 53% of the stabilizing force to resist lateral subluxation and normally presents with a solid endpoint when the lateral glide test is performed. Reproduction of the patient's symptoms with passive lateral translation of the patella pulling on the medial structures is referred to as a positive lateral apprehension sign. This signals lateral patellar instability.

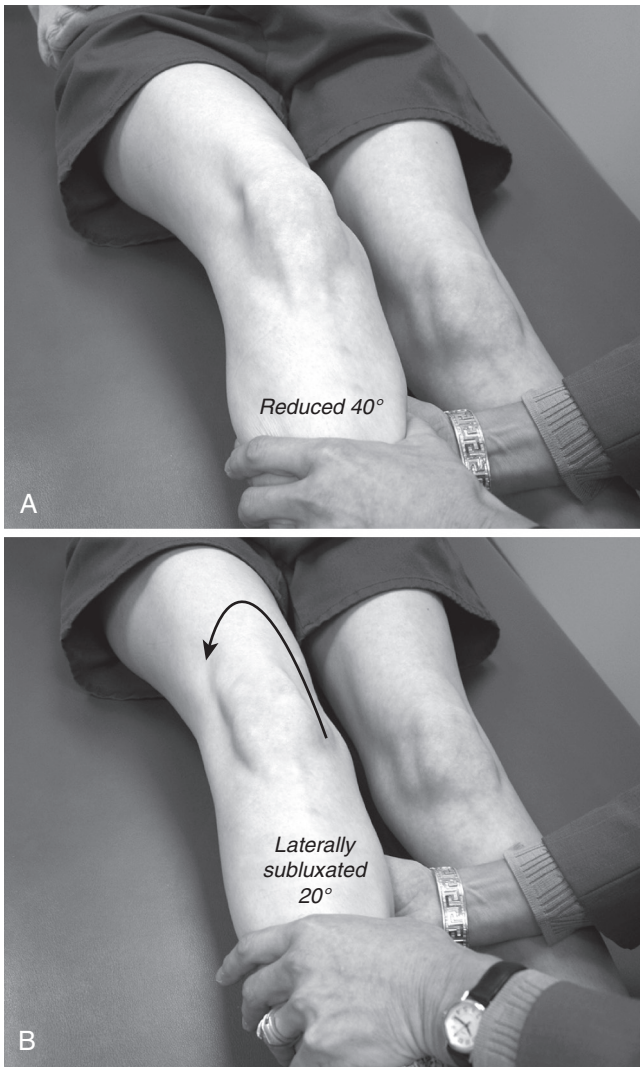

Fig. 56.12 A and B, A positive "J" sign is demonstrated as the patient's patella is at 40 degrees of flexion and subluxes laterally at 20 degrees of flexion. Asking the patient to straighten the leg against examiner's resistance can demonstrate this sign of lateral patellar instability. (Copyright 2002, ML Ireland.)

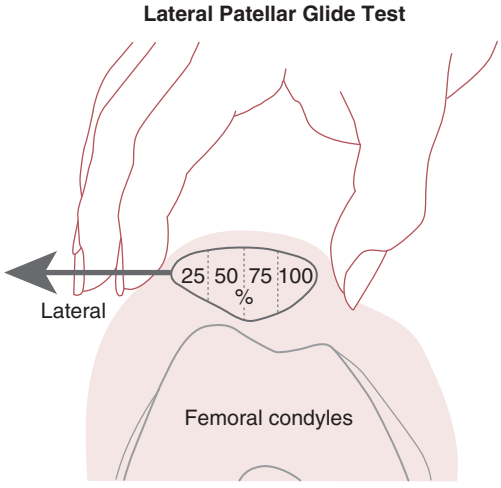

Fig. 56.13 Lateral patellar glide test.

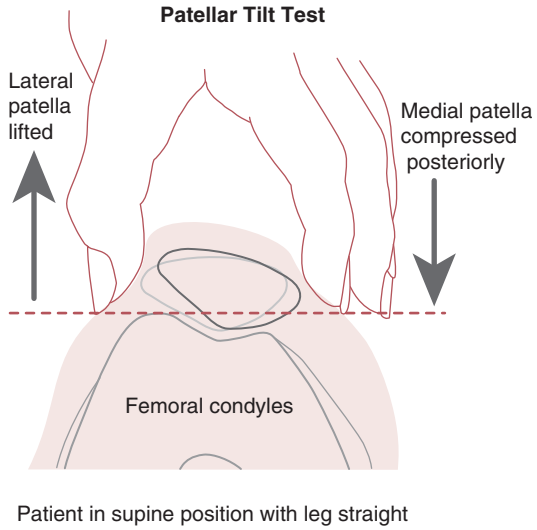

Fig. 56.14 Patellar tilt test.

Medial Glide Test

The medial glide test is performed with the knee in full extension. The patella is centered on the trochlear groove, and medial translation from this "zero" point is measured in millimeters. Greater than 10 mm of translation is abnormal. The lateral retinacular laxity may result from a hypermobile patella or, less commonly, medial instability. Medial patellar instability is rare and usually presents as an iatrogenic complication following patellar realignment surgery, typically from an overaggressive lateral release. Six to 10 mm of translation is considered normal. Translation less than 6 mm medially indicates a tight lateral restraint and may be associated with ELPS. See Rehabilitation Protocol 56.2 for procedures following distal and/or proximal patellar realignment procedures.

Patellar Tilt

A tight lateral restraint may contribute to patellar tilt. Patellar tilt is evaluated as the knee is brought to full extension and an attempt is made by the examiner to elevate the lateral border of the patella (Fig. 56.14). Normally, the lateral border should be able to be elevated 0 to 20 degrees above the medial border. Less than 0 degrees indicates tethering by a tight lateral retinaculum, vastus lateralis, or IT band. Presence of clinical and radiographic lateral patellar tilt is indicative of tight lateral structures. This may be responsible for ELPS. If extensive rehabilitation fails, the presence of a lateral patellar tilt correlates with a successful outcome after lateral release.

Patellar tilt is evaluated by the patellofemoral angle. This angle is formed by the lines drawn along the articular surfaces of the lateral patella facet and the lateral wall of the trochlear groove. The lines should be roughly parallel. Divergence is measured as a positive angle and is considered normal, whereas convergence of the lines is measured as a negative angle and indicates the presence of abnormal patellar tilt.

Bassett Sign

Tenderness over the medial epicondyle of the femur may represent an injury to the medial patellofemoral ligament in the patient with an acute or recurrent patellar dislocation.

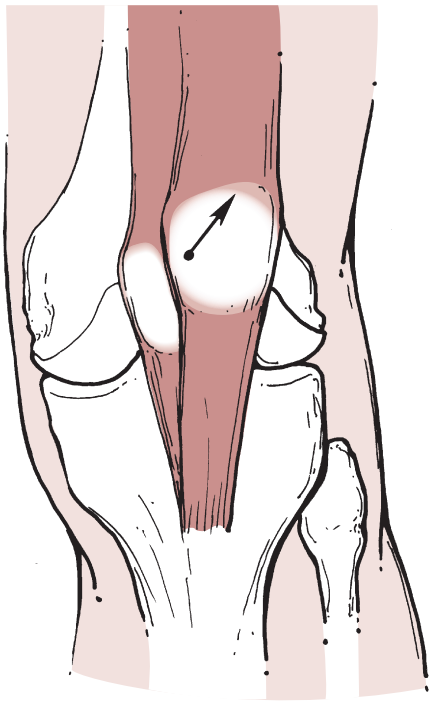

Fig. 56.15 Lateral pull sign. In this left knee, when the quadriceps is contracted, the patella moves in an exaggerated lateral and proximal direction. This also indicates predominance of lateral forces. (Reprinted with permission from DeLee J: Delee & Dreez's Orthopaedic Sports Medicine, ed 2. Philadelphia, Saunders, 2002, Fig. 28E2.21.)

Lateral Pull Test/Sign

The lateral pull test is performed by contraction of the quadriceps with the knee in full extension. Test results are positive (abnormal) if lateral displacement of the patella is observed. This test demonstrates excessive dynamic lateral forces (Fig. 56.15).

Radiographic Evaluation

Three views of the patella—an AP, a lateral in 30 degrees of knee flexion, and an axial image—should be obtained. The AP view can assess for the presence of any fractures, which should be distinguished from a bipartate patella, a normal variant. The overall size, shape, and gross alignment of the patella can also be ascertained. The lateral view is used to evaluate the patellofemoral joint space and to look for patella alta or baja. In addition, the presence of fragmentation of the tibial tubercle or inferior patellar pole can be seen. Both the AP and the lateral views can also be used to confirm the presence and location of any loose bodies or osteochondral defects that may exist. An axial image, typically a Merchant (knee flexed 45 degrees and x-ray beam angled 30 degrees to axis of the femur) or skyline view, may be the most important. It is used to assess patellar tilt and patellar subluxation. The anatomy of the trochlear groove is also well visualized, and the depth and presence of any condylar dysplasia can be determined. One important point deserves mention. The radiographs visualize only the subchondral bone of the patella and trochlea and do not show the articular cartilage. The articular surfaces are not necessarily of uniform thickness in these regions. Therefore, any measurements made from plain radiographs are only an indirect indication of the actual anatomic structure.