Part THREE

Treatment Guidelines for Common Diagnoses of the Upper Extremity
Common Shoulder Diagnoses

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KEY TERMS

- Adhesive capsulitis
- Annulus fibrosis
- Autonomic instability
- Axial skeleton
- Bankart lesion
- Bifocal
- Capsular plication
- Closed chain exercises
- Close packed position
- Concentric
- Degrees of freedom
- Directional preference
- Disk herniations
- Eccentric
- Elevated Arm Stress Test
- Extraarticular
- Facet joints
- Force couple
- Gold standard
- Hemiarthroplasty
- Hill-Sachs lesion
- Impingement
- Intervertebral foramen

Intraneural scarring
Mechanoreceptors
Negative intracapsular pressure
Neural mobility
Neurolysis
Nociceptors
Nucleus pulposus
Open chain exercises
Open packed position
Perineural scarring
Plane synovial joint
Plyometric exercises
Premorbid
Roos Test
Rotator interval
Scaption
Scapular kinematics
Sensitivity
Specificity
Spurling's Test
Supraclavicular scalenectomy
Tinel's sign
Trigger point
Uncinate processes
Unifocal
Positioning the hand in space to allow for interaction with the environment is the primary function of the shoulder. Accordingly, dysfunction of the shoulder complex often results in profound impairment of the entire upper extremity (UE). The shoulder will compensate for decreased mobility of the wrist and elbow, which can lead to shoulder dysfunction as the individual tries to perform normal activities of daily living (ADL).

**CLINICAL Pearl**

When treating a client with elbow or wrist dysfunction, the therapist needs to monitor the health of the shoulder. Therefore a thorough understanding of the shoulder is imperative for therapists treating clients with UE dysfunction.

The shoulder has the greatest range of motion (ROM) of any joint in the body. This ROM is the result of the aggregate movement of a series of articulations that make up the shoulder complex. These articulations work in concert to provide a unique balance between mobility and stability, with the emphasis on mobility. A shift in this balance often results in (or can be caused by) the pathologic processes we review in this chapter.

**ANATOMY**

The shoulder complex consists of the following:

- Three bones: the humerus, the clavicle, and the scapula
- Three joints: the glenohumeral, acromioclavicular, and sternoclavicular
- One pseudojoint: the scapulothoracic articulation.

**Glenohumeral Joint**

The glenohumeral joint is a multiaxial, synovial, ball-and-socket joint that moves around three axes of motion: internal/external rotation around a vertical axis, abduction/adduction around a sagittal axis, and flexion/extension around a frontal axis (Fig. 10-1). The humeral head forms roughly half a sphere with the glenoid fossa, forming the socket component of the joint. The glenoid fossa covers only one third to one fourth of the humeral head (Fig. 10-2). The glenoid labrum, a ring of fibrocartilage, surrounds and deepens the glenoid socket by about 50% and increases joint stability by increasing humeral head contact 75% vertically and 56% transversely.²,³

The **open packed position** (joint position in which the capsule and ligaments are most lax and separation of joint surfaces is greatest) of the glenohumeral joint is 55 degrees of abduction and 30 degrees of horizontal adduction. The **close packed position** (joint position in which the capsule and ligaments are under the most tension with maximal contact between joint surfaces) of the joint is full abduction and lateral rotation. At rest, the humerus sits centered in the glenoid cavity; with contraction of the rotator cuff (RC) muscles, the humeral head translates anteriorly, posteriorly, superiorly, inferiorly, or any combination of these movements. These translations are small, but full motion of the glenohumeral joint is impossible without them. The motion of the glenohumeral joint contributes the most to shoulder movement.

**Acromioclavicular Joint**

The acromioclavicular joint is a **plane synovial joint** (joint with a synovium-lined capsule and relatively flat surfaces) that augments the ROM of the glenohumeral
joint, as this is the joint around which the scapula moves. The bones that compose the acromioclavicular joint are the acromion process of the scapula and the distal end of the clavicle. The acromioclavicular joint moves around three axes: pure spin around a longitudinal axis for abduction/adduction of the shoulder, a vertical axis for protraction/retraction of the shoulder, and a horizontal axis for shoulder elevation/depression.

The acromioclavicular and coracoclavicular ligaments support the acromioclavicular joint (Fig. 10-3). The acromioclavicular ligaments contribute the least to joint stability; they function mainly to support the joint.
capsule and check anterior/posterior translation of the clavicle on the acromion. The acromioclavicular ligaments are damaged in grade I shoulder separations. The coracoclavicular ligaments have no attachment to the acromion and consist of the conoid and trapezoid ligaments. They transmit scapular motion to the clavicle and check superior clavicular displacement. Complete rupture of these ligaments represents a grade III separation resulting in a step deformity at the acromioclavicular joint (Fig. 10-4).

The open packed position for the acromioclavicular joint is with the arm by the side. The close packed position is at 90 degrees of shoulder abduction.

**Sternoclavicular Joint**

The sellar-shaped (saddle-shaped) sternoclavicular joint is the only direct articulation between the shoulder complex and the axial skeleton (skeletal components consisting of the skull, rib cage, spine, and pelvis). The articulations of the sternoclavicular joint are between the medial end of the clavicle, the clavicular notch of the sternum, and the cartilage of the first rib. Interposed between the clavicle and the sternum is an articular disk that enhances stability of the joint (Fig. 10-5, A). Movement between the disk and clavicle is greater than movement between the disk and sternum. The joint is stabilized further by the joint capsule and ligaments that primarily check superior and anterior translation. In fact, the sternoclavicular joint is stabilized so well by the disk and ligaments that trauma to the clavicle usually results in fracture instead of dislocation. The three degrees of freedom (direction or type of motion at a joint) at the sternoclavicular joint are elevation/depression, protraction/retraction, and rotation (spin) (Fig. 10-5, B). The open packed position for the sternoclavicular joint is with the arm by the side. The close packed position is full UE elevation.

**Scapulothoracic Articulation**

Because the scapula has no direct bony or ligamentous connections to the thorax, the scapulothoracic articulation cannot be considered an anatomic joint. Scapular movement results in movement of the shoulder girdle. These movements are described as elevation/depression, abduction (protraction)/adduction (retraction), upward rotation, and upward tilt. The bony articulation of the scapula is with the acromioclavicular joint, but the sta-
bility of the scapulothoracic joint comes from muscular attachments to the scapula.

Much like a street performer balancing a ball on the end of a stick, the scapula must change position during shoulder elevation to keep the humeral head balanced in the glenoid fossa. With shoulder elevation, the majority of motion occurs at the glenohumeral joint during the initial (0 to 60 degrees) and final (140 to 180 degrees) phases of motion. During these phases, the scapulothoracic articulation plays a more subtle balancing or stabilizing role. Throughout the middle or critical (60 to 140 degrees) phase of shoulder elevation, the ratio of glenohumeral to scapulothoracic motion shifts, with more emphasis on scapulothoracic movement.6

This movement of the scapula is the result of force couples between groups of muscles that run from the thorax to the scapula (Table 10-1). A force couple is defined as two resultant forces of equal magnitude in opposite directions that produce rotation of a structure. The upward rotation of the scapula that occurs during shoulder elevation primarily results from the concentric (muscle contraction resulting in approximation of the origin and insertion) actions of the upper and lower trapezius and the lower portion of the serratus anterior muscles. Eccentric (muscle contraction to stabilize movement resulting in increased distance between the origin and insertion) actions of the levator scapulae, rhomboids, and pectoralis minor produce smooth motion.

In the normal resting position the scapula sits angled 20 to 30 degrees forward relative to the frontal plane and 20 degrees forward in the sagittal plane, with the medial border angled at 3 degrees top to bottom from the spinous processes. This position, combined with the orientation of the glenoid fossa, results in elevation of the arm in a plane that is 30 to 45 degrees anterior to the frontal plane. This motion is termed scapular plane abduction or scaption.7 The scapula extends from the level of the T2 spinous process to the T7 or T9 spinous process based on size. Because the scapulothoracic articulation is not an anatomic joint, there is no close packed position.

**PROXIMAL (CERVICAL) SCREENING**

Because of the proximity of the cervical spine to the shoulder, the cervical spine must be screened for contribution to the client’s symptoms. A basic understanding of cervical anatomy and of the structures that refer symptoms to the shoulder and UE is essential to the screening process.

**Anatomy**

The cervical structures that refer symptoms to the shoulder and entire UE that are cleared via cervical screening are the following:

- Cervical nerve roots
- Cervical disks
- Cervical facets

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**TABLE 10-1**

<table>
<thead>
<tr>
<th>MOVEMENT</th>
<th>CONCENTRIC FORCE COUPLE</th>
<th>ECCENTRIC STABILIZERS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upward rotation (glenohumeral elevation)</td>
<td>Upper trapezius</td>
<td>Levator scapulae</td>
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<tr>
<td></td>
<td>Lower trapezius</td>
<td>Rhomboid muscles</td>
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<td></td>
<td>Serratus anterior</td>
<td>Pectoralis minor</td>
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<tr>
<td>Retraction</td>
<td>Trapezius</td>
<td>Serratus anterior</td>
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<td></td>
<td>Rhomboid muscles</td>
<td>Pectoralis major</td>
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<td>Pectoralis minor</td>
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<td>Protraction</td>
<td>Serratus anterior</td>
<td>Trapezius</td>
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<td></td>
<td>Pectoralis major</td>
<td>Rhomboid muscles</td>
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<td>Pectoralis minor</td>
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<tr>
<td>Elevation</td>
<td>Upper trapezius</td>
<td>Serratus anterior</td>
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<td></td>
<td>Levator scapulae</td>
<td>Lower trapezius</td>
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<td>Depression</td>
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<td>Lower trapezius</td>
<td>Levator scapulae</td>
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<td>Downward rotation</td>
<td>Levator scapulae</td>
<td>Upper trapezius</td>
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<td>Rhomboid muscles</td>
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<td></td>
<td>Latissimus dorsi</td>
<td>Serratus anterior</td>
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<tr>
<td></td>
<td>Pectoralis minor</td>
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</tr>
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</table>
• Cervical intrinsic soft tissue (muscles, ligaments, joint capsules)
• Cervical extrinsic musculature

Cervical Nerve Roots
The C4 to C7 nerve roots supply structures that overlie or compose the shoulder complex (Fig. 10-6). The C5 and C6 nerve roots innervate most of the glenohumeral joint structures, with the C4 nerve root innervating the acromioclavicular joint.

Because of their location and path of travel, the cervical nerve roots are susceptible to injury. Disk herniations (damage to the annular wall of the disk resulting in disk deformity as the nucleus displaces into the lesion) can entrap the nerve root against the vertebral lamina and encroach upon the dorsal root ganglion. Hypertrophy of the facet joints, spurring of the vertebral end plates, and spurring of the uncinate processes (winglike projections from the superior portion of the cervical vertebrae that articulate with the inferior portion of the vertebrae above) will narrow the intervertebral foramen (bony canal that contains the spinal nerve), resulting in compression of the cervical nerve roots. Degenerative loss of cervical disk height further enhances this process.

Cervical Disks
The cervical spine contains five disks, with the most superior disk located between C2 and C3, and the most inferior disk located between C7 and T1. The disk con-
sists of three parts: the annulus fibrosis (multilayered ligamentous exterior of the disk), the vertebral end plate (cartilaginous interface between the vertebral disk and the vertebral body), and the nucleus pulposus (pulpy semiliquid center of the disk). The cervical disks are morphologically different from lumbar disks because they essentially lack a posterior annular wall. That role mainly is supplied by the posterior longitudinal ligament. Also, the cervical disk develops horizontal annular clefts or tears in the lateral portion by age 15 that progressively extend across the back of the disk. 8 Likely because of these differences, the cervical disks degenerate more quickly than the lumbar disks. 9

**CLINICAL Pearl**

The onset of neck and arm pain with cervical disk herniation is usually insidious and often starts in the neck and medial scapular border before radiating to the shoulder and arm.

Symptoms can spread as far as the hand, depending on the involved nerve root.

**Cervical Facets**
The facet joints of the cervical spine are paired synovial joints with fibrous capsules. The capsules are heavily innervated by mechanoreceptors (specialized nerve endings that transmit information regarding position and motion) and nociceptors (specialized nerve endings that transmit pain signals) that likely modulate protective muscle reflexes that are important in preventing joint instability and degeneration.10 Studies on normal individuals and clients with neck pain demonstrated pain referral patterns from the cervical facets to the cervical and shoulder regions.11,12 These studies demonstrated a consistent pain referral pattern to the top and lateral parts of the shoulder, extending to the inferior border of the scapula from the C6-C7 facet joints.

**Cervical Intrinsic Soft Tissue**
The intrinsic soft tissue structures of the cervical/thoracic region include the muscles that do not originate or insert on the clavicle or scapula. Of these muscles, the scalene muscles demonstrate trigger point (palpable taut muscle bands that refer pain when compressed) pain referral patterns to the shoulder (see Fig. 10-20 on the CD). Substantial anatomic variations in the attachments of the scalene muscles exist. In general, the proximal portions attach to the transverse processes of the cervical vertebrae. The distal attachments of the anterior and medial scalene muscles are the first rib; the distal attachment of the posterior scalene muscle is the second rib. The trigger points refer pain to the anterior lateral aspect of the shoulder and medial scapular border.13

**Cervical Extrinsic Muscle**
The extrinsic muscles are those that have attachments to the shoulder structures (scapula and clavicle) and cervical spine. Of these, the trapezius and levator scapulae demonstrate trigger point referral patterns to the shoulder.

The trapezius extends down the midline from the occiput to T12, laterally to the acromion, anteriorly to the clavicle, and posteriorly to the scapular spine. Six trigger points with distinctive pain patterns are located in the upper, middle, and lower fibers. The trigger point located in the lower trapezius refers pain to the mastoid area and the posterior acromion11 (see Fig. 10-21 on CD).

The levator scapulae attaches proximally to the transverse processes of the first four cervical vertebrae and distally to the superior angle of the scapula. The trigger point refers pain to the angle of the neck and often projects to the posterior aspect of the shoulder13 (see Fig. 10-22 on CD).

**Diagnosis and Pathology**
The primary goal of the cervical screening examination is to screen efficiently for pathologic cervical conditions that may be contributing to or causing shoulder symptoms. If screening indicates a pathologic cervical condition, the examiner must perform further testing of the cervical spine. Numerous examination procedures are described in the literature that are beyond the scope of this chapter.

**Precaution.** The following screening procedures are not a substitute for a complete examination of the cervical spine.

**Range of Motion Testing: Intrinsic versus Extrinsic Restrictions**
Having the client perform active movements of the cervical spine is an excellent beginning point for your screening examination. By changing the relative position of the shoulder and cervical spine during testing, you can begin to differentiate between intrinsic and extrinsic restrictions to cervical motion.

The client performs the basic motions of the cervical spine (flexion/extension, rotation, lateral flexion) from a corrected neutral seated posture with the arms unsupported. Next, the client performs the same motions in a crossed-arm position (see Fig. 10-23 on the CD). The
client grasps as close to the acromioclavicular joints as possible and then relaxes the arms and shoulders, letting the arms rest against the chest wall.

This position effectively elevates the scapulae, and by having the client grasp the shoulders, the scapular elevators are allowed to relax. An improvement in ROM in this position implicates the extrinsic cervical structures as contributing to motion loss. No change in motion implicates the intrinsic structures. However, the extrinsic structures still may be limiting motion. The test is designed to rule out intrinsic restrictions if a difference exists between the two test positions.

Repeated Motion Testing: The Search for a Directional Preference
Repeated motion testing is the basis of the McKenzie model of examination. By having the client perform the cervical motions of protrusion, retraction, retraction and extension, flexion, lateral flexion, and rotation in groups of 5 to 10 repetitions, the therapist looks for a directional preference.

A directional preference exists if any of these movements centralize or decrease the client’s symptoms. An important note concerning centralization is that the client’s proximal pain levels may intensify.

CLINICAL Pearl
Worsening or peripheralization of the client’s distal symptoms with repeated cervical motion indicates a pathologic cervical condition.14

Intervertebral Foramen Closing/Facet Loading Testing: Spurling’s Test
Although the use of Spurling’s Test as a screening tool to detect radiculopathy has been questioned15 because of a sensitivity (few if any clients with the disease will have negative test results) of 30% compared with electromyogram findings, its specificity (all persons who do not have the disease will have negative test results) was high at 95%. The use of electromyogram as a gold standard (the best available test to diagnose a condition) for detecting radiculopathy is questionable because the American Academy of Electrodiagnostic Medicine16 estimates a sensitivity of 50% to 71%. By accurately applying a test with high specificity, clients without a confirmed pathologic condition should test negative. Therefore in ruling out the cervical spine as a possible source of shoulder pain, the Spurling’s Test is clinically relevant.

Spurling and Scoville17 described the test as positive with provocation of the client’s symptoms when the client’s neck was flexed laterally to the painful side, extended, and with axial loading of the client’s spine added by the examiner after rotation toward and away from the painful side. With the cervical spine in this position the intervertebral foramen diameter closes down, decreasing the available space for an inflamed nerve root. The presence of a space occupying lesion such as disk herniation or osteophytic spur intensifies the test result. Axial loading at end-range of extension and rotation also stresses the facet joints, provoking symptoms if a pathologic condition is present.

THORACIC OUTLET SYNDROME/BRACHIAL Plexopathy
The term thoracic outlet syndrome (TOS) encompasses an assortment of clinical entities involving the shoulder region. The thoracic outlet provides the pathway for the neural and vascular structures to the upper limb; therefore a pathologic condition of this area has profound and often disabling results. Because vascular presentations of TOS are relatively uncommon (3% to 5%), the great majority of clients with TOS have brachial plexopathies.18

Anatomy
Thoracic Outlet
The thoracic outlet can be divided into four regions: the sternocostovertebral space, the scalene triangle, the costoclavicular space, and the pectoralis minor (coracpectoral) space. Each region has distinct boundaries, contents, and potential pathologic conditions that result in neurovascular compression and/or entrapment (Fig. 10-7).

Sternocostovertebral Space
The sternocostovertebral space is bordered anteriorly by the sternum, posteriorly by the spinal column, and laterally by the first rib. The contents are the roots of the plexus, the subclavian artery and vein, jugular vein, and neck lymphatic vessels. Compression of the contents usually is caused by tumors of the lung (Pancoast’s), thymus, parathyroid glands, and lymph nodes.

Scalene Triangle
The scalene triangle is bordered anteriorly by the anterior scalene muscle, posteriorly by the middle scalene muscle, and inferiorly by the first rib. The contents are the roots and trunks of the plexus and subclavian artery. Compression and entrapment of these structures are caused by variations in scalene anatomy and the presence of congenital fibrous bands that may interdigitate with the plexus.19
**Costoclavicular Space**

The costoclavicular space is bordered superiorly by the clavicle and inferiorly by the first rib. The contents are the divisions of the plexus and the subclavian artery and vein. Compression of these structures between the clavicle and first rib occurs as a result of postural deficits resulting in shoulder girdle depression, clavicular and first rib fractures, and the presence of a cervical rib.

**Pectoralis Minor (Coracopectoral) Space**

The coracopectoral space is bordered superiorly by the coracoid process, anteriorly by the pectoralis minor, and posteriorly by the chest wall. The contents are the cords of the plexus and the subclavian artery and vein. Compression of these structures is caused by hypertrophy and contracture of the pectoralis minor and hyperabduction of the arm as they are pulled up against the pectoralis minor tendon.

**Brachial Plexus**

The brachial plexus is netlike, which allows for the individual neurons from the spinal nerves eventually to reach their respective peripheral nerve. The brachial plexus also serves as a force distributor to dissipate traction forces from the peripheral nerve, helping to prevent traction injuries of the lower cervical nerve roots.

Although anatomic variations exist, the brachial plexus is fairly consistent in its organization (Box 10-1). Moving proximal to distal, the plexus is organized into roots (C5 to T1), trunks (upper, middle, lower), divisions (anterior, posterior), and cords (medial, lateral, posterior). Trunks are supraclavicular and cords are infraclavicular, with the divisions occurring under the clavicle.

**Diagnosis and Pathology**

**Vascular Component**

The diagnosis of TOS versus brachial plexopathy is somewhat controversial. TOS, being a syndrome, by definition is a collection of symptoms related to a pathologic condition of an anatomic space (the thoracic outlet). Brachial plexopathy by definition is a pathologic condition of a specific anatomic structure (the brachial plexus). A wide range of criteria are used as to what symptoms make up TOS. To simplify the diagnosis, true TOS should include a component of vascular compression and brachial plexus compression and/or entrapment. The vascular component of TOS ought to be diagnosed via...
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vascular studies because there is often a sympathetic nervous system component to the brachial plexopathy that presents clinically with vascular symptoms.

Unfortunately, the clinical tests advocated in the literature to look for vascular compromise—such as the Adson's and Wright's Tests, which rely on obliteration of the radial pulse while in the test position—have a high incidence (as high as 87%) of positive results in normal persons.\(^{20-23}\) Therefore, drawing conclusions based on the results of these tests in the clinic is questionable.

Neural Component: Brachial Plexopathy

Because of the paucity of diagnostic tests that detect mild to moderate brachial plexopathies, the best way to identify a brachial plexopathy is through careful and thorough evaluation. This should include a detailed history as to the onset of symptoms and mechanism of injury. Onset of symptoms is often traumatic, with trauma involving forced lateral cervical flexion with the shoulder held in a fixed position (as in a seat belt injury), or forced depression of the shoulder combined with forced lateral cervical flexion (as in a sports injury such as “burner” syndrome), or even dislocation of the shoulder. Symptoms may be delayed for months as adhesions form between the neural tissue and surrounding nerve bed. This leads to restricted neural mobility (the ability of the neural structures to adjust to changes in the nerve bed length through a combination of gliding and elongation), ultimately resulting in loss of UE motion and function.

Onset can be insidious, with genetic and morphologic predisposition combined with poor postural and movement habits leading to the development of the condition. During the growth phase into adolescence, the scapulae gradually descend down the posterior thorax, with the descent being greater in women. A strain with resulting weakness of the scapular suspensory muscles that lengthen during this process is associated with the development of brachial plexopathy. This helps to explain the rarity of insidious-onset brachial plexopathy before puberty and the increased incidence of the disease in women.\(^{24}\)

Clients with brachial plexopathy often complain of unilateral headaches in the occipital region, with facial pain from the angle of the jaw to the zygomatic region to the ear. They may complain of shoulder and chest wall pain from the trapezius ridge down the medial border of the scapula, in the supraclavicular/infraclavicular fossa, and from the sternum to the axilla to the epigastric region. These clients often are seen in the emergency room for a suspected heart attack but are misdiagnosed with costal chondritis or gastritis.\(^{25}\)

Arm and hand involvement often includes complaints of pain, paresthesia, and weakness.

BOX 10-1

Organization Highlights of the Brachial Plexus

- Of the five nerve roots that supply the plexus, the top two make up the upper trunk, the bottom two make up the lower trunk, and the middle root makes up the middle trunk
- Upper trunk supplies the scapular musculature and scapular stabilizers
- Lower trunk supplies the hand intrinsics
- Anterior division of the lower trunk supplies the medial cord
- Anterior divisions of the upper and middle trunk supply the lateral cord
- Anterior divisions supply the elbow and wrist flexors with the exception of the brachioradialis, which is supplied by the posterior cord
- Posterior divisions of all trunks supply the posterior cord
- Posterior cord supplies the elbow and wrist extensors

CLINICAL Pearl

A strong clue that the plexus is involved is that these symptoms do not follow dermatomal or peripheral nerve distributions. Other strong indications that the plexus is involved are intolerance to overhead activities, reports of dropping objects, cramping of the hand intrinsics while writing, waking with a “dead arm,” and intolerance of straps across the top of the shoulder.

Timelines and Healing

Full recovery from a brachial plexus injury is rare. However, clients often can achieve enough of a reduction in symptoms to allow for a return to restricted activity. The level of restriction is related to the severity of the original injury and the amount of intraneural scarring (contained within the nerve) and/or perineural scarring (between the nerve and the nerve bed). This is a lifelong injury, and the client must be instructed in management of the condition. Unfortunately, the condition is characterized by periods of high and low neural irritability based on the client’s activity level and the degree of pathology. Reirritation of the injured plexus leads to further scarring and pathology as a result of inflammatory reaction.
Nonoperative Treatment

The most important step to begin healing is to teach the client how not to irritate the injured plexus. Through neural mobility assessment, clients can be taught where the safe boundaries of motion are. If the client is able to follow these movement restrictions and plexus irritation drops to a stable level, the client can attempt to regain plexus mobility through gliding and stretching exercises. As plexus mobility improves, the safe boundaries of motion increase, resulting in improved ADL function.

Clients must be taught how to breathe using the diaphragm, minimizing the use of the scalene muscles; and they must be instructed in safe sleeping positions to avoid stretching or compressing the plexus. Most importantly, the client must be taught to maintain a posture that minimizes stress on the brachial plexus while maximizing the apertures of the thoracic outlet.

Precaution. Clients with brachial plexopathy rarely tolerate weight training at the gym, but guided exercises to strengthen the scapular stabilizers and elevators are essential.

With direct supervision, clients can use resistance bands to strengthen the upper, middle, and lower trapezius, as well as the levator scapulae, rhomboid muscles, and serratus anterior. Doing the exercises in sets of 3 repetitions allows the therapist and client to assess for signs of increased plexus irritation between sets, thereby avoiding overstretching of the thoracic outlet contents.

Clients can regain scapular proprioception through visual feedback exercises. The client stands facing the mirror while performing scapular motions, targeting points of the clock. With 12 o’clock being superior, 9 o’clock anterior, and 3 o’clock posterior, the client symmetrically elevates the shoulders to the 12, 1, and 2 o’clock positions. These exercises are performed in straight lines of motion as smoothly as possible. After each cycle the client assesses the level of irritation and adjusts the exercise accordingly.

The client performs gliding and stretching exercises in front of the mirror as well. The client begins the glide exercise with the arms against the side, elbows flexed to 90 degrees with the palms facing up. Next, the client elevates the shoulders while slowly extending the elbows. To bias the medial and lateral cords of the plexus, the client maintains supination while extending the wrists (see Fig. 10-24 on the CD). To bias the posterior cord of the plexus, the client pronates the forearms and flexes the wrists (see Fig. 10-25 on the CD).

The client begins the stretch exercise with the palm of the hand brought to eye level in front of the face, elbow held close to the body. While maintaining the hand at eye level, the client moves the shoulder into abduction and external rotation, with the wrist held in supination. Again keeping the hand at eye level, the client slowly extends the elbow just until a stretch is felt or a slight increase in symptoms occurs. At this point the client backs off slightly on the elbow extension and flexes and extends the wrist 3 times (Fig. 10-8). The client attempts to straighten the elbow further with each cycle of the exercise. The glide and stretch exercises are performed in sets of 3 as well.
The client performs all exercises from a neutral posture. The client achieves this by lifting the sternum through increasing the lumbar lordosis and elevating the rib cage. This effectively corrects the forward-head-rounded-shoulders posture, relieving stress from the thoracic outlet contents. From this position, the client performs a posture exercise combined with a proximal nerve glide by elevating the shoulders to the 12 o’clock position and then doing the posterior half of a shoulder roll, ending at the starting point. Instruct the client to perform these exercises hourly for 5 to 10 repetitions to reinforce proper posture.

**Precaution.** These clients are not to be instructed to “chin tuck” and retract their shoulders to correct their posture, for this often aggravates their condition.

Clients who demonstrate tight scalene muscles and pectoralis minor muscles must be taught stretching exercises.

**Precaution.** Because these muscles lie against the brachial plexus, the therapist must watch for an increase in the client’s symptoms during stretching.

The rule of threes works in this situation as well: sets of three stretches held for 3 seconds. As your client demonstrates good tolerance to the stretch, the stretch can be held for longer periods, or the number of sets can be increased.

**Precaution.** The scalene muscle stretches described in the literature and in exercise card kits bias stretch the brachial plexus and therefore should be avoided.

The literature usually instructs the client to depress the shoulder while stretching, often causing further irritation to the injured plexus (Fig. 10-9). The scalene muscles, having no attachment to the shoulder and being intrinsic to the cervical and thoracic regions, should be stretched with the shoulder held elevated, thereby relieving tension from the brachial plexus during stretching.

**Operative Treatment**

The primary goal of TOS surgery is decompression of neurovascular contents or neurolysis (the removal of scar tissue from the nerve) of the entrapped brachial plexus. Clients who fared the best were those with confirmed vascular or neurologic compromise in the thoracic outlet via diagnostic testing. Unfortunately, surgical outcomes have been disappointing; therefore surgery is reserved as a last resort. The most common procedures are transaxillary first rib resection and supraclavicular scalenectomy (surgical removal of the anterior scalene muscle) with neurolysis.

**Clinical Pearl**

Maintaining postoperative neural mobility is imperative because the formation of perineural scarring will entrap the plexus, leading to poor surgical outcome.

**Operative Treatment**

Neural mobilization exercises are begun as soon as the client is stable, often within the first 3 postoperative days.

**Questions to Ask the Doctor (for the Postoperative Client)**

- How soon can range of motion exercises begin?
- Are there any restrictions to movement of the neck or shoulder?
What to Say to Clients

About the Condition

“Here is a drawing of your thoracic outlet. You can see that the nerves and blood vessels that supply the arm travel through here. The areas of possible damage are here in your neck (the scalene muscles), between the collar bone and first rib, or under the muscles of your chest wall (pectoralis minor).”

About the Home Exercise Program

“In order for you to move your arm comfortably, your nerves must be able to slide through the thoracic outlet smoothly. You may have developed restrictions that prevent this from happening. Maintaining good posture is critical because you place excessive strain on the nerves with poor posture habits. Your exercises are designed to reinforce proper posture and help the nerves slide through the thoracic outlet, much like sliding a string through a straw.”

“Your postural exercises are to be performed hourly to help reinforce good postural habits. Purchase and wear a cheap digital watch you set to ‘beep’ on the hour to remind you to exercise. Your gliding and stretching exercises are to be performed a minimum of three times a day. Tie these exercises to mealtimes so you will remember to do them.”

Evaluation Tips

• Assess the client’s ability to achieve the corrected posture position described before.
• Check for asymmetry of scapular/shoulder position.
• Look for swelling over the supraclavicular fossa.
• Monitor the client’s UE for evidence of autonomic instability (sympathetic nervous system irritation) during testing; for example, reticular mottling, color changes, or temperature changes.
• Palpate along the course of the brachial plexus and peripheral nerves for tenderness and evidence of a Tinel’s sign (production of tingling or paresthesia with percussion over the nerve)
• Neural mobility testing of the brachial plexus can be graded as follows:
  • 0/5: Shoulder in internal rotation, elbow flexed to 90 degrees with arm across stomach, wrist and fingers in neutral (see Fig. 10-26, A, on the CD)
  • 1/5: Shoulder in neutral, elbow flexed to 90 degrees, wrist and fingers in neutral (see Fig. 10-26, B, on the CD)
  • 2/5: Shoulder in approximately 100 degrees of abduction, neutral rotation, elbow flexed to 90 degrees, wrist and fingers in neutral (see Fig. 10-26, C, on the CD)
  • 3/5: As with the shoulder in approximately 90 degrees of external rotation, forearm in supination, wrist in extension, and fingers in neutral (see Fig. 10-26, D, on the CD)
  • 4/5: As with the elbow flexed to 45 degrees (see Fig. 10-26, E, on the CD)
  • 5/5: As with the elbow to 0 degrees (see Fig. 10-26, F, on the CD)
• Use the plus or minus signs (+/−) for positions between each grade; or for more specific documentation, list the last grade achieved followed by the goniometric measurement of the shoulder or elbow position, such as 1/5 with 45-degree shoulder abduction or 3/5 with 60-degree elbow flexion.
• Record position at provocation of symptoms to grade the test.
• Block shoulder elevation during testing; do not depress the shoulder.
• Use the Elevated Arm Stress Test (EAST; or Roos Test), and record time to provocation of symptoms. The test is described as a 3 minute test28; in my experience, clients with plexopathy will not tolerate more than 1 minute (Fig. 10-10).
• During myotomal screen, focus on scapular elevators for upper trunk lesions and hand intrinsics for lower trunk lesions.
• Using a safety pin flagged with tape as pictured in Fig. 10-11 to check for sensitivity to sharp sensation keeps the level of pressure applied during testing constant. Clients with brachial plexopathy show differential sensation of the middle finger versus sensation of the ring finger for clients with carpal or cubital tunnel syndrome. Decreased sensitivity along the medial half of the ring finger and hand indicates medial cord involvement; decreased sensitivity along the lateral half of the ring finger and hand indicates lateral cord involvement.25
CHAPTER 10 Common Shoulder Diagnoses

Precaution. The inability of your client to achieve stable symptoms at correct posture is one of the best predictors of treatment failure.

Clients often complain that their home exercises are increasing their symptoms. Have them demonstrate their home program regularly and correct any modifications they have made. Adjust the amount of movement during the home exercises to keep symptoms stable.

The application of heat before exercise helps to calm symptoms. Riding the stationary bike for 10 to 15 minutes using proper posture with the involved arm supported is an excellent warm-up exercise.

Precautions and Concerns

- Avoid overstretching the brachial plexus during treatment.
- Be careful with overhead exercise such as wall pulleys.
- Use of an upper body ergometer is not recommended for these clients.
- Advance the progress of strengthening exercises cautiously.
- Watch exercise positions to avoid overstretching the brachial plexus.

PROXIMAL HUMERUS FRACTURE

Anatomy

Proximal humerus fractures are the most common fracture of the humerus and may involve the articular surface, greater tuberosity, lesser tuberosity, or the surgical neck. These four regions are described as the four major fracture fragments that occur and are the basis of the classification systems for proximal humerus fracture.29

Achieving these basic parameters listed in the Clinical Pearl section above should be the initial goal of treatment. Base the speed and intensity of the rehabilitation program on the client's level of symptom irritability/stability. Inform your clients with chronic restrictions that they may experience increased symptoms for up to 48 hours after treatment.

Precaution. If the client's level of posttreatment irritation persists for more than 48 hours, the intensity of the treatment needs to be decreased.

Tips from the Field

Constantly stress proper posture when your clients are in the clinic. Instruct them to notice the poor posture of others they encounter during their day and to use these observations as a reminder to correct their own posture.

FIGURE 10-11 Example of the use of a flagged safety pin to maintain constant pressure to check for differential of sensation between the medial and lateral aspect of the long finger.

Diagnosis-Specific Information That Affects Clinical Reasoning

CLIENTAL Pearl

Clients who cannot tolerate the corrected posture position have a positive EAST result in less than 30 seconds or who have neural mobility of the plexus lower than 3/5 have a poorer prognosis, for they easily aggravate their condition during ADL.

Diagnosis-Specific Information That Affects Clinical Reasoning

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Diagnosis and Pathology

The majority of proximal humerus fractures occur as a direct result of a fall on the involved shoulder in the elderly population or a direct blow to the humeral region and are stable one-part or A2 fractures involving the surgical neck of the humerus.30

One-part fractures as classified by the Neer system are described as no fracture fragments being displaced more than 1 cm and no more than 45 degrees of angulation. Two-part fractures exceed these position limits and can involve the humeral head and surgical neck or the humeral head and greater tuberosity. Three- and four-part fractures involve the humeral head, greater tuberosity, and lesser tuberosity.29
An alternative classification system is that of AO North America (www.aona.org) and is predicated on disruption of blood supply to the articular segment. Type A fractures are **extraarticular** (outside the joint capsule) and **unifocal** (one fracture line). Type B fractures are extraarticular and **bifocal** (two fracture lines). Type C fractures are articular. Each of these types then is subdivided into three groups. A1 classified fractures have the least severity and best prognosis; C3 classified fractures have the greatest severity and worst prognosis.

**Timelines and Healing**

One-part and A2 fractures are treated by sling immobilization initially for 1 to 3 weeks. Clients can start passive movements when the humeral shaft and head move as a unit, which can be as early as 2 weeks. Type B and C or two- to four-part fractures, being more complex, usually require 4 to 6 weeks of immobilization, except in clients with hemiarthroplasties, who begin passive range of motion (PROM) exercises on postoperative day 1.

**Nonoperative Treatment**

The client begins treatment while still in the immobilizer by performing gripping exercises and active ROM of the elbow and wrist to prevent edema and joint stiffness. Once clinically stable, the client starts PROM exercises in the clinic and pendulum and tabletop PROM exercises at home. The therapist can start more aggressive stretching and the client begins active ROM exercises around 4 to 6 weeks. The focus at this time should be on proper glenohumeral and scapulothoracic movement to prevent the substitution patterns of early scapular elevation and trunk leaning to achieve UE elevation.

**Precaution.** Substitution patterns discourage proper recruitment of the RC muscles, so they need to be avoided at all costs.

The therapist plays a significant role by having the client perform hand-to-hand active-assisted ROM progression to active ROM mirroring exercises in a seated position while the therapist prevents early scapular elevation (see Fig. 10-27, A, on the CD). Once the client understands the movement concept, the client can perform the same exercise through self-scapular stabilization and wall walking (see Fig. 10-27, B, on the CD). These motions are performed in the scapular plane initially to provide the RC muscles the best length-tension relationship to encourage coordinated activity. At 8 to 12 weeks after injury/repair the client can begin resisted strength training. The focus is on the RC muscles and scapular stabilizer/force couple muscles. Therapist-provided manual resistance in diagonal planes of motion are essential at this stage of the program to encourage functional movements and to discourage substitution movements.

A mixture of open and closed chain exercises must be included at this stage because the shoulder functions in both situations. **Open chain exercises** are defined as working against resistance where the extremity is free to move in space resulting in movement of the distal segment. **Closed chain exercises** are defined as working against resistance with the extremity working against a stationary or mobile but motion-constrained object or surface. Closed chain exercises impart a degree of stability during the exercise motion.

Closed chain exercises include wall push-ups, seated press-ups, weight shifting in quadruped, or prone resting on elbows. Variations include ball rolling against the wall and use of a tilt board for weight bearing in quadruped (see Fig. 10-28 on the CD).

If at 12 weeks after injury/repair the client has achieved functional ROM and normal movement patterns, the client can begin **plyometric exercises** (which link strength and speed of movement to produce an explosive-reactive type of muscle response) and sport-specific activities to prepare them for return to full function. Therefore one must understand the client’s goals of rehabilitation and the **premorbid** (before injury) activity level.

**Operative Treatment**

Proximal humeral fractures graded as two- to three-part or type B usually require surgical intervention of open reduction and internal fixation to reduce the displaced fracture fragments. **Hemiarthroplasty** (prosthetic replacement of one joint surface) usually is indicated to replace the avascular humeral head in four-part and type C fractures.

**Questions to Ask the Doctor**

**Operative Clients**

- What structures were repaired? (Ask for a copy of the operative report.)
- How soon can active motion start?
- When can I begin lifting weights?

**Nonoperative Clients**

- When can I begin to take off the sling?
- How much longer do I need to wear the sling to sleep?
• How soon can I begin moving my shoulder?
• When can I begin lifting weights?

What to Say to Clients

About the Injury

If the fracture is classed as one part or A2, say, “Your fracture is considered stable, so you are allowed to begin moving your arm while the healing process continues. In fact, the motion will help the healing process. All motions must be passive, meaning motion provided by the therapist, by gravity, or on a supported surface such as a tabletop. You are not to attempt to raise your arm by itself for the next couple of weeks because this could affect the healing fracture.”

If the fracture has been repaired surgically or if it is a two-part or type B and now is considered stable, say, “Your doctor has determined that the fracture is healed enough to begin motion exercises. Movement will make a big difference in how quickly you recover and is critical to your recovery.”

About Exercise

“You need to move your arm as often and as much as possible. The motion helps to lubricate the joint and keeps it healthy.” If the client is doing tabletop exercises, say, “By using your fingers to pull your arm forward, you will avoid stressing your shoulder during this exercise. Keep your shoulder relaxed while moving; at the end of tolerable motion, rest your hand flat on the table surface while you sit back upright, dragging your arm back to the starting position.” If the client is doing pendulum exercises, say, “Do your exercises next to a table or counter. Using the table for support with your uninjured arm, bend at the waist as far as you can and let your injured arm swing forward as if it were a piece of rope. Now rock your body side to side and in circles to get your arm swinging just as you would do with your hand to swing a rope side to side or in a circle.”

For all ROM exercises, say, “It is not how hard you push your stretches, but how often you stretch and how much cumulative time you spend at the end of motion that counts. Try for a minimum of fifteen minutes of total end-range time by doing fifty stretches a day, each held for twenty seconds.”

Evaluation Tips

Take PROM measurements with the client seated. Make sure motions are slow and gentle because the client will be apprehensive about moving the arm. Often, when you attempt to return the client’s arm to neutral after full elevation, the client will experience sharp pain as the deltoid and humeral head elevators reflexively contract, causing sharp pain. By having the client actively lower the arm against your resistance, this reflex is inhibited and the motion will be considerably more comfortable.

Measure functional internal rotation (IR) by seeing which bony landmark on the pelvis or spinoous process the client can touch with the thumb (e.g., anterior superior iliac spine, iliac crest, posterior superior iliac spine, or L5).

Diagnosis-Specific Information That Affects Clinical Reasoning

CLINICAL Pearl

The type of fracture directly affects how aggressively you may rehabilitate your client.

Obtaining this information from the doctor is crucial. This information is also available from radiology and operative reports.

Alignment of the humeral head to the shaft will affect how much ROM the client ultimately will recover with a minimum of one degree of motion loss in the opposite direction for each degree of deformity. For example, if the shaft is in 45 degrees of extension relative to the humeral head, the client’s expected flexion ROM will be 135 degrees (180 degrees − 45 degrees = 135 degrees). The same holds true for rotational deformities.29

Tips from the Field

Clients presenting to therapy a few weeks after a one-part or type A proximal humeral fracture usually will be apprehensive about moving the arm because of fears about fracture instability. You have two ways to calm their fears. First, tell them that as you rotate their arm, the humeral head will not move with unified motion if the fracture is unstable. Have them place their hand over their injured humeral head while you gently rotate the arm from IR to external rotation (ER); they should feel the humeral head move. The second and rather novel technique involves using a stethoscope. Explain to the client that sound will not travel well across an open fracture. Next, have the client listen through the stethoscope placed on the humeral head of their healthy shoulder while you tap on their lateral epicondyle. Do the same to their injured shoulder, where the intensity of sound should be the same. This often will decrease their apprehension about moving their shoulder and will speed recovery.

Scapular position and posture have a direct affect on shoulder ROM. Clients must be given postural exercises as previously described for brachial plexus clients.
Precautions and Concerns

- RC injuries often are overlooked at the time of injury. Watch for evidence of RC tear during rehabilitation.
- Clients are at a high risk of developing adhesive capsulitis (frozen shoulder). Movement must be the basis of any therapy program.
- Many of these clients have a concurrent axillary nerve or brachial plexus injury. Screen for this during the initial examination by checking sensation over the deltoid (axillary distribution) and by asking if the client is experiencing paresthesias in the hand or arm (possible brachial plexus involvement).

FROZEN SHOULDER/ADHESIVE CAPSULITIS

Anatomy

The fibrous capsule that envelops the glenohumeral joint is lined with synovial tissue. The capsule is attached medially to the glenoid margin and encompasses the glenoid labrum and long head of the biceps. Laterally, the capsule attaches to the anatomic neck of the humeral head near the articular surface. The inferior portion attaches laterally about 1 cm distal to the articular surface on the humeral shaft. The capsule is slack enough that distraction of the glenohumeral joint surfaces up to 3 cm can occur.5

The capsule has three distinct thickened areas known as the glenohumeral ligaments (superior, middle, and inferior) that help stabilize the glenohumeral joint. These ligaments become taut at various portions of glenohumeral motion as their fibers run in radial and circular directions. During abduction and rotation, the capsule becomes shortened, producing a compressive and centering force of the humerus on the glenoid.11 During abduction and rotation, the inferior glenohumeral ligament forms a sling providing anterior, posterior, and inferior stability to the joint. With the arm at rest, this portion of the capsule forms the dependent axillary pouch, which often is obliterated with frozen shoulder (Figs. 10-2 and 10-12).

The coracohumeral ligament extends from the base of the coracoid process as two bands that blend with the capsule running to the greater and lesser tuberosities. Parts of the ligament form the tunnel for the biceps tendon and reinforce the rotator interval (the region between the superior edge of the subscapularis and anterior edge of the supraspinatus tendons).

The tendons of the subscapularis, supraspinatus, infraspinatus, and teres minor fuse with the lateral part of the joint capsule forming the RC. With contraction of the RC muscles, the lax capsule is pulled away from the movement path of the humeral head, preventing capsular impingement.1

![FIGURE 10-12 Interior view of the right shoulder joint, looking into the glenoid fossa and joint capsule. (From Canale ST: Campbell's operative orthopaedics, ed 10, St Louis, 2003, Mosby.)](image)
CHAPTER 10  Common Shoulder Diagnoses

Diagnosis and Pathology

The terms adhesive capsulitis and frozen shoulder are used interchangeably in the literature.

**CLINICAL Pearl**

Frozen shoulder is characterized by a progressive loss of glenohumeral ROM usually in the capsular pattern of ER being most limited followed by abduction and IR.

Magnetic resonance imaging (MRI) studies demonstrate capsular thickening with loss of the axillary recess. The disease appears to be periarticular, with most authors finding little to no capsular adhesions during arthroscopic examination.

The condition is found in 2% to 3% of the U.S. population, most commonly in the fourth through sixth decades of life. Frozen shoulder is more common among women, with one study reporting women making up 70% of the frozen shoulder clients. Frozen shoulder usually is classified as primary or secondary adhesive capsulitis, with the course of the disease following three phases: the freezing phase, the frozen phase, and the thawing phase.

**Primary Adhesive Capsulitis**

Primary frozen shoulder is idiopathic. Considerable debate has occurred over the pathogenesis of frozen shoulder, with possible causes being inflammatory, immunologic, endocrinal alterations, or biochemical. This form of the disease is overrepresented in clients with diabetes, with rates 3 times that of the normal population.

Histologic findings in a group of clients with primary frozen shoulder revealed active fibroblastic proliferation of the coracohumeral ligament and rotator interval with the absence of inflammation or synovial involvement, much like that of Dupuytren's disease (disease process resulting in thickening and contracture of the palmar fascia). In examining the hands of 58 clients with frozen shoulder, Smith et al. found 30 clients had a pit, nodule, or band of Dupuytren's contracture.

**Secondary Adhesive Capsulitis**

Secondary frozen shoulder is characterized by a precipitating event such as surgery, trauma to the shoulder, or specific pathologic condition of the shoulder such as bursitis, impingement syndrome, or tendonitis. Although the same pattern of motion loss occurs, these clients may not go through all the stages of freezing, frozen, and thawing.

**Freezing Phase**

The freezing phase is characterized by shoulder pain interrupting sleep, pain with ADL such as brushing one's hair or tucking in one's shirt, and often pain at rest. Distinguishing frozen shoulder from pathologic conditions such as RC tendonitis, shoulder bursitis, or impingement syndrome is difficult.

Examination of the client during this phase reveals ROM to be close to full with pain occurring often before the end of motion. Palpation reveals nonspecific tenderness at the anterior, lateral, and posterior aspects of the shoulder. Strength is often normal or slightly decreased with pain on resisted testing.

Clients tend to limit the use of the affected extremity because all movements are painful, leading to further loss of motion. Over the next 2 to 9 months, the pain subsides and the client is left with the typical frozen shoulder with pain occurring at the end of motion.

**Frozen Phase**

The frozen phase may last up to a year. This phase is characterized by distinct pathologic movement patterns as the client attempts to substitute scapulothoracic motion to compensate for the lack of glenohumeral mobility. In this phase, pain occurs with stretching of the joint capsule at the end of motion.

**Thawing Phase**

The thawing phase is characterized by a gradual return of motion and lasts on average up to 26 months. The idea that full motion will return is a misconception. Thirty percent of clients with primary frozen shoulder exhibited some degree of motion loss compared with the uninvolved shoulder at an average follow-up of 7 years after surgery.

**Timelines and Healing**

As noted before, there are average timelines for each phase of the disease. The majority of clients complete the thawing phase within 18 months to 3 years from onset.

**Nonoperative Treatment**

No evidence suggests that therapeutic modalities such as ultrasound or interferential electric stimulation affect the outcome of the disease. Treatment should be directed at the process occurring during each phase of the disease.

Precaution. Overstretching the capsule during the freezing phase may enhance the inflammatory process, stimulating further capsular fibrosis.

When the joint has achieved the frozen and thawing phases, the stretching exercises can be more aggressive.
However, avoid pushing to the point that reinitiates the inflammatory process.

The role of the occupational therapist in assisting clients with bilateral frozen shoulder in ADL modifications or adaptive equipment for grooming, bathing, and dressing cannot be overestimated. Clients with unilateral frozen shoulder may benefit from workstation modifications to help them remain productive during the protracted course of the disease.

The use of corticosteroids intraarticularly in the freezing phase of the disease may be helpful in stabilizing the synovial tissue, allowing for better tolerance to stretching exercises.33

Operative Treatment

For frozen shoulder cases that fail to progress after protracted conservative treatment, manipulation under anesthesia and arthroscopic release are two of the most common surgical interventions. Of the two, arthroscopic release of the anterior glenohumeral ligament and coracohumeral ligament currently has the most promising outcome.40

Evaluation Tips

- Take careful baseline and follow-up ROM measurements with the client supine to stabilize the trunk and scapula. This will allow for careful tracking of the progress of the condition.
- If all passive and resisted motions are painful throughout the ROM, the client is still in the freezing stage.
- If resisted motion is pain free and pain occurs only at end ROM, the client is in the frozen or thawing phase.

Diagnosis-Specific Information That Affects Clinical Reasoning

The intensity of the therapy program is directly proportional to the phase of the condition. The primary treatment goal of the freezing phase is to prevent motion loss. The primary treatment goal of the frozen and thawing phase is to restore functional ROM.

Tips from the Field

CLINICAL Pearl

Proper posture and normal scapular kinematics (scapular movement in sequence and proportion to humeral movement) must be stressed during exercise at all times.

Clients with frozen shoulder quickly develop the pathologic motion of early scapular elevation to raise their arm. This movement pattern can lead to secondary cervical problems, further complicating the recovery process.

Precautions and Concerns

- Do not push ROM during the freezing phase to the point of pain that lasts more than a few minutes. This will only enhance the inflammatory and fibrosing process.
- These clients must avoid self-imposed immobilization.

Glenohumeral Instability

Anatomy

Glenohumeral instability could be considered the antithesis of adhesive capsulitis. However, laxity of the glenohumeral joint is a quality that allows full ROM; laxity is not synonymous with instability. When laxity leads to pain with loss of power and shoulder function, then glenohumeral instability exists.
The concepts and structures that contribute to glenohumeral stability can be categorized as static and dynamic. The static stabilizers have a larger role when the shoulder is at rest, whereas the dynamic stabilizers play a larger role when the shoulder is in motion.

The static restraints include negative intracapsular pressure (air pressure inside the joint capsule being lower than pressure outside the capsule), the suction effect of the glenoid labrum acting on the humeral head like a plunger, and cohesion-adhesion between the wet smooth surfaces of the humeral head and glenoid fossa. The orientation of the humeral head and glenoid fossa contribute to the static stability of the glenohumeral joint as well. With proper postural positioning of the scapula, the dynamic stabilizers need minimal effort to maintain glenohumeral congruency.41

The dynamic restraints include the RC that provides a compressive and positioning force and to a certain degree the long head of the biceps tendon. Although the glenohumeral ligaments are passive structures, they are under relatively little tension with the shoulder at rest. These ligaments serve as a restrictive leash to check force and limit ROM at various positions of the glenohumeral joint during movement. Of these ligaments, the inferior glenohumeral ligament is the most crucial to dynamic glenohumeral stability. The role and functions of this ligament are described in the anatomy section of the discussion of frozen shoulder.

**Diagnosis and Pathology**

Two major categories are useful in understanding shoulder instability. They are known by the acronyms of AMBRII and TUBS. The major points of each are summarized in Table 10-2. The pathology and treatment of AMBRII and TUBS shoulders are different.

AMBRII shoulders have no history of dislocation or subluxation. The client's major complaint is pain with activity, usually in overhand-throwing motions. This pain often results from impingement (compression of soft tissue between bony structures) that is related to the client's inability to adequately stabilize the scapulothoracic and/or glenohumeral joint because of a pathologic condition of the RC, capsular laxity, and altered proprioception (awareness of joint position). Budoff et al.42 described this condition as primary instability leading to secondary impingement.

Primary instability is often a combination of global capsular laxity and pathologic imbalances of the RC and shoulder muscles. Weak and/or proprioceptively compromised RC muscles cannot effectively oppose the upward pull of the deltoid muscle during UE elevation. The result is superior migration of the humeral head and impingement of the greater tuberosity and RC against the underside of the acromion and coracoclavicular ligament (Fig. 10-13).

**TABLE 10-2**

<table>
<thead>
<tr>
<th>TUBS or “Torn Loose”</th>
<th>AMBRII or “Born Loose”</th>
</tr>
</thead>
<tbody>
<tr>
<td>Traumatic etiology</td>
<td>Atraumatic or microtrauma with no specific episode</td>
</tr>
<tr>
<td>Unidirectional instability</td>
<td>Multidirectional instability may be present</td>
</tr>
<tr>
<td>Bankart lesion is the pathology</td>
<td>Bilateral: asymptomatic shoulder is also loose</td>
</tr>
<tr>
<td>Surgery is required</td>
<td>Rehabilitation is the treatment of choice</td>
</tr>
<tr>
<td></td>
<td>Inferior capsular shift and</td>
</tr>
<tr>
<td></td>
<td>Interval between the supraspinatus and subscapularis closed surgically if conservative measures fail</td>
</tr>
</tbody>
</table>

**FIGURE 10-13** Force couple between the rotator cuff and deltoid muscle resulting in inferior glide of the humeral head during elevation of the arm. (From Greenfield BH et al: Impingement syndrome and impingement-related instability, in Donatelli RA, editor: Physical therapy of the shoulder, ed 4, St Louis, 2004, Churchill Livingstone.)
TUBS shoulders have a history of dislocation, usually in the anterior direction. The mechanism of injury is a fall or blow to the arm while in the position of abduction and ER. Recurrent subluxation or dislocation results when the client places the arm in the position of injury, leading to apprehension and dysfunction. These clients often have a resulting Bankart lesion, which consists of damage to the anterior glenohumeral capsule and labrum, and a Hill-Sachs lesion, which consists of an osseous defect of the posterolateral portion of the humeral head caused during traumatic anterior dislocation. Both conditions usually require surgery to restore stability to the glenohumeral joint.

Between these categories of shoulder instability are a group of shoulder pathologies related to asymmetric capsular tightness. This occurs from the excessive distraction force on the glenohumeral joint during the deceleration phase of throwing, leading to thickening and contracture of the posterior-inferior portion of the capsule. The sequela of this asymmetric capsular tightening is a loss of glenohumeral IR resulting in a cascade of events that may lead to pathologic conditions of the biceps tendon and labrum.

The long head of the biceps tendon helps stabilize the glenohumeral joint during the overhead throwing motion. The unstable and asymmetrically tight shoulder places extra stress on the biceps tendon, leading to bicipital tendinitis and superior labrum anterior to posterior (SLAP) lesions. The SLAP lesion is hypothesized to result from increased torsional force from the biceps tendon that “peels back” the biceps and posterior labrum from the glenoid rim. The SLAP lesion then enhances the dynamic and static instability of the already unstable shoulder.

### Timelines and Healing

For the client with an unstable shoulder not surgically corrected, 4 to 8 weeks of rehabilitation is common. The length of rehabilitation depends on the client’s ability to gain control of the instability. Once stable, the client is released to a sustained home exercise program to continue indefinitely. Surgically corrected unstable shoulders require more time for rehabilitation. After a period of immobilization lasting 2 to 4 weeks, 3 to 6 months of rehabilitation is common. These clients also require a sustained home exercise program. Most postoperative clients report that it takes 6 months to a year before their shoulder feels “normal.”

### Nonoperative Treatment

Treatment focuses on strengthening the RC and scapular stabilizers. Strengthening starts with shoulder isometric exercises in the safe position of the arm at the side. The motions resisted include shoulder flexion/extension, IR/ER, and abduction/adduction, as well as elbow flexion and extension. While performing isometric exercises, clients must “set” their scapula against their rib cage in a corrected posture position to engage the scapular stabilizers.

The next step in the rehabilitation program, once the client demonstrates fair to good control of the instability, is progression to isotonic exercises in a subimpingement range using light resistance and a high number of repetitions. For clients with anterior instability the focus is on strengthening the internal rotators, adductors, and biceps. For clients with global instability, the focus is on the RC, scapular stabilizers, deltoid, biceps, and triceps.

An essential at this stage is incorporation of open and closed chain exercises as previously described for proximal humeral fractures. The intensity of the closed chain exercises can be increased by using weighted medicine balls against the wall; by moving the body into a more horizontal position, or even working off the exercise ball while in prone UE weight-bearing.

Therapist-applied manual resistance can be used throughout each phase of the rehabilitation process. Starting with isometric exercises and active-assisted ROM and then progressing to concentric and eccentric exercises, the therapist controls the speed of movement and amount of force. The major benefit of manual resistance is the immediate feedback the therapist receives from the client during rehabilitation.

For the clients with posteroinferior capsule restrictions, stretching exercises to restore IR are critical. Use of towel behind back stretches and the sleeper stretch (Fig. 10-14) work well.

### Operative Treatment

Surgical correction of the multidirectionally unstable shoulder should be considered only after a minimum of 3 months of conservative therapy has failed. Two surgical procedures most often recommended in the literature are the open inferior capsular shift (surgical detachment and superior advancement of the inferior glenohumeral ligament) and thermal capsulorraphy (selective heating of portions of the joint capsule, resulting in capsular shrinkage). Each of these procedures has advantages and disadvantages.

Greater surgical morbidity occurs with the open capsular shift, and the repair has good and predictable outcomes. This procedure is often augmented by surgical closure of the rotator interval.

Thermal capsulorraphy can trace its origins to Hippocrates’ time. He describes inserting a hot iron into the axilla to cauterize the unstable joint. Treatment...
has improved much since then, and this is now an arthroscopic procedure. However, there is a paucity of long-term studies about surgical outcomes. Complications include return of the instability, axillary nerve damage, and adhesive capsulitis.50 Because of these complications and less than promising long-term outcomes, this surgery as a stand-alone procedure is becoming less popular because it is used increasingly in combination with, or is being replaced by, arthroscopic capsular plication (suturing folds into the joint capsule).

Precaution. Great care must be taken in the rehabilitation of these clients to avoid attenuating (weakening, stretching) the healing capsule.

The client can initiate ROM exercises earlier because the RC structures are left intact, but the therapist needs to supervise carefully the return of motion. For clients with traumatic anterior dislocation, the need for surgical repair of the Bankart lesion varies based on the client’s age range and the physical demands on the shoulder. Rates of recurrent dislocation were highest in clients younger than 30 years old,51 with rates ranging from 79% to 100%. Conservative therapy had little effect on reducing rates of recurrence.52 Therefore, clients younger than 30 and those over 30 who perform UE labor-intensive jobs should consider surgical repair.

Questions to Ask the Doctor

Nonoperative Clients

• What is the nature and direction of the instability?
• Are there any secondary pathologies that need to be addressed (such as rotator cuff tear or SLAP lesion)?
• Is this client a surgical candidate?

Operative Clients

• What type of repair was performed? Ask to see an operative report.
• What are the range of motion restrictions?
• How soon can the client begin strengthening exercises?
• Do you have a specific postoperative protocol for rehabilitation?

What to Say to Clients

About the Injury

“Your shoulder is a ball-and-socket joint with the ball much larger than the socket. This design allows for a lot of motion, but your shoulder must rely on the muscles and ligaments to keep the joint stable.”

For the AMBRII client, say, “Because the ligaments that support your joint are so loose, your rotator cuff muscles need to work much harder to keep the joint stable. When they fatigue, the ball is able to slide up and pinch the tendons of the rotator cuff against the bony roof of your shoulder during throwing and overhead activities.”

For the TUBS client, say, “When you fell with your arm out to the side, the ball was forced from the socket, which likely caused damage to the rim of the socket and the supportive ligaments in the front of the shoulder. As a result, your shoulder is unstable, and the ball can slip out of the socket easily if you raise your arm out to the side as if you are going to throw a ball. While your shoulder heals, you must avoid this position or the problem will keep occurring. There is a chance that even if you avoid this position during your recovery, the damage is great enough that your shoulder will remain unstable.”

For the client with the tight posterior capsule, say, “Because of the way your shoulder has adapted to the stress of throwing, you have developed tightness in a portion of the ligaments that support the shoulder joint. As a result, your joint has a decreased ability to rotate internally. This restriction causes abnormal motion of the ball in the socket when your shoulder is under the stress of throwing, resulting in your pain and loss of function.”

About Exercises

For the AMBRII client, say, “Your exercises are designed to compensate for your unstable shoulder by increasing the strength, coordination, and endurance of your rotator cuff and scapular stabilizing muscles. This exercise program is a lifelong commitment, because if the weakness returns, your shoulder problems will return as well.”

For the TUBS client, say, “Your exercises strengthen the muscles in the front of your shoulder to support the...
damaged part of the joint. You must follow the motion restrictions carefully during exercise to avoid disrupting the repair process.”

For the client with the tight posterior capsule, say, “You need to regain the internal rotation motion of your shoulder to restore the normal function. The stretches work best if performed frequently. Three sets a day is not enough. You should try for a minimum total of thirty minutes of stretch time each day. Remember, it is not how hard you push the stretch, but how much time you spend at the end of motion that counts.”

**Evaluation Tips**

- For the unstable shoulder, the goals are to find the direction(s) of instability and to reproduce the client’s symptoms. Their feedback during examination is critical.
- The client must be as relaxed as possible during examination because muscle guarding will hide the degree of instability.
- Various tests are described in the literature to test for shoulder instability. Basic tests to check for the direction of instability are as follows:
  - **Anterior and Posterior Drawer Test:** With the client seated, the therapist grasps the humeral head with one hand while stabilizing the scapula and clavicle with the other. Next, the therapist applies an anterior and then a posterior translation pressure tangential to the glenoid surface while assessing the amount of humeral head movement. The therapist then compares the amount and quality of movement to the opposite side.
  - **Sulcus Sign Test:** With the client seated and the client’s arm supported in 20 to 50 degrees of abduction, the therapist pulls inferiorly on the client’s arm. A depression of more than a finger width resulting between the acromion and humeral head indicates a positive test. This test indicates multidirectional instability.48
  - **Apprehension Test:** With the client supine the therapist moves the client’s shoulder into 90 degrees of abduction and end-range ER. If with the application of overpressure the client experiences apprehension but not pain, the test is considered positive for anterior instability.51
  - **Relocation Test:** With the client positioned as at the end of the Apprehension Test, the therapist applies posteriorly directed pressure from the anterior aspect of the humeral head. The test is positive if the client’s apprehension disappears. This test helps to confirm anterior instability.54

**Diagnosis-Specific Information That Affects Clinical Reasoning**

The direction of the instability dictates the course of treatment as described before. Consequently, you must have a clear understanding of the client’s instability pattern.

**Precaution.** Applying an incorrect exercise and stretching program may aggravate the client’s condition.

Many of these clients have impingement and/or SLAP lesions as well. If a SLAP lesion is present, your client may need to avoid rotary exercises with the arm above shoulder height. In these cases, overhead throwing exercises are contraindicated.

**Tips from the Field**

- Proprioception exercises (activities to enhance position and movement sense/control of the scapula and shoulder complex) need to be stressed with these clients.
- Manual resisted exercises in diagonal patterns at various speeds using concentric and eccentric force are a valuable component of the rehabilitation program.
- Have your client perform UE exercises to strengthen the RC while concurrently working on balance while in quadruped, sitting, kneeling, and standing positions.
- Stress proper posture as described before.

**Precautions and Concerns**

- Do not perform joint mobilization or stretches on the client with multidirectional instability.
- Clients with anterior instability require that their posterior capsule be mobilized. Avoid stretching the anterior capsule.
- Pay close attention to ROM restrictions for postoperative clients.

**ROTATOR CUFF DISEASE**

**Anatomy**

After neck and back pain, shoulder pain is the third most common musculoskeletal disorder. Up to 70% of shoulder disorders are related to RC disease.55

The structures of the shoulder that are involved in RC disease include the muscles of the RC, the long head of the biceps tendon, the subdeltoid-subacromial bursa, and coracoacromial arch.

The supraspinatus, infraspinatus, and teres minor make up the greater tuberosity attachments of the RC.
The subscapularis attaches to the lesser tuberosity. All the RC muscles work together to stabilize the head of the humerus in the glenoid fossa during shoulder motion while their tendons form a cuff that surrounds the humeral head.

Along with their primary role of stabilizing the glenohumeral joint, each muscle of the RC imparts specific motion to the humeral head. The supraspinatus is an abductor, the infraspinatus is an external rotator, the teres minor is an external rotator and weak adductor of the humerus, and the subscapularis is an internal rotator (see Figs. 10-12 and 10-15).

The stabilizing role of the long head of the biceps tendon is reviewed in the section on glenohumeral instability. As the arm elevates overhead, the head of the humerus glides along the biceps tendon as it sits in the bicipital groove between the greater and lesser tuberosities. The long head of the biceps plays a role in shoulder flexion and in forearm flexion and supination.

The subdeltoid-subacromial bursa is a smooth serosal sac that sits between RC tendons and the coracoacromial arch. Above, the bursa is adherent to the underside of the deltoid, coracoacromial ligament, and the acromion. Beneath, the bursa is adherent to the RC and greater tuberosity. This structure provides a cushioning and low-friction interface between the convex humeral head and RC as they rotate below the concave coracoacromial arch during arm elevation.

The coracoacromial arch consists of the anteroinferior aspect of the acromion process, the inferior surface of the acromioclavicular joint, and the coracoacromial ligament. This structure forms a roof over the RC and humeral head.65 The coracoacromial arch not only serves as an attachment site for the deltoid and subdeltoid-subacromial bursa but also provides superior stability and protection to the glenohumeral joint.

**Diagnosis and Pathology**

Two major hypotheses have been proposed about the cause of RC disease. One is based on extrinsic causes and the other is based on intrinsic causes. Current evidence demonstrates that both contribute to the disease process and are affected by age, postural habits, movement quality, and activity level.

Extrinsically caused lesions result from the repeated impingement of the RC tendon against different structures of the glenohumeral joint. Neer56 describes impingement between the long head of the biceps and supraspinatus tendons and the coracoacromial arch during UE elevation, resulting in lesions on the bursal side of the RC. His three-stage classification of impingement syndrome is still used today (Table 10-3). Bigliani et al.57 described three types of acromion morphology (Fig. 10-16) with cadaver studies demonstrating a 70% incidence of RC tears in subjects with a type III acromial shape and a 3% incidence in subjects with a type I acromial shape. Walch et al.58 described a type of impingement between the supraspinatus and infraspinatus tendons in the late cocking phase of throwing on the glenoid rim, resulting in lesions on the articular side of the RC.

Intrinsically caused lesions result from age-related degeneration of the RC tendon. These lesions are related to the vascularization of the RC cuff and are on the articular side of the tendon.66,67 Lindblom61 was the first to describe an area of hypovascularity of the supraspinat
tus tendon where it attaches to the greater tuberosity. Codman referred to the same area as the “critical zone” because it appeared to be at greater risk of developing a tear.

Because most RC tears are partial-thickness tears, the condition is often progressive and can lead to a full-thickness lesion. As tendon fibers fail, they retract because the RC is under constant tension. This process leads to at least four adverse effects:

- Increased load on intact neighboring fibers, leading to their potential failure
- Loss of muscle fibers attached to bone, leading to decreased strength and function of the RC
- Blood supply of intact tendon placed at risk by distorted anatomy from fiber failure, leading to progressive ischemia and tendon degeneration
- Loss of tendon repair potential as the tendon is exposed to joint fluid containing lytic enzymes, which inhibit hematoma formation that would facilitate healing

Usually beginning in the supraspinatus tendon, the tear may progress to involve the infraspinatus tendon. Once this occurs, the ability of the RC to stabilize the humeral head in the glenoid fossa is compromised severely, leading to superior migration under the unopposed pull of the deltoid. Humeral head superior migration loads the long head of the biceps tendon, leading to tendonopathy and potential failure. Traction spurs develop at the coracoacromial ligament attachment on the acromion through repeated loading from the upward displacement of the humeral head, leading to further RC damage. This damage allows the RC tendon to slide down below the axis of joint rotation. Much like a boutonniere deformity of the finger, the buttonholed RC becomes a humeral head elevator instead of a depressor. The RC is then ineffective as a humeral head stabilizer, and the client is unable to elevate the arm above a horizontal position.

**Timelines and Healing**

Recovery from pathologic conditions of the RC varies greatly because of multiple presentations of the disease. If the case is uncomplicated, such as a tendonitis, the condition can stabilize in 2 to 6 weeks. If secondary pathologic conditions are present—such as frozen shoulder, impingement, instability, and RC tear—recovery time lengthens considerably. Complex cases may take up to a year to resolve with or without surgical intervention.

**Nonoperative Treatment**

Initial treatment focuses on rest and antiinflammatory modalities to stabilize the disease process. Early ROM exercises such as pendulum and wand-assisted elevation in the scapular plane to avoid impingement help to moderate pain through the analgesic effect of mechanoreceptor stimulation.

**CLINICAL Pearl**

Maintaining full, pain-free IR and ER is critical to preventing frozen shoulder.

Strengthening of the healthy portion of the RC and scapular stabilizer muscles, usually in the motions of shoulder IR, adduction, and extension, is safe and encourages the stabilizing function of the RC. The use of isometric exercises and resistance band exercises are effective during this portion of the program. As pain levels decrease and RC function improves, the next step is to strengthen the UE elevators and external rotators.
Throughout this process, you must stress muscle balance and proper scapular kinematics. Incorporating open and closed chain and manual exercises, as described for proximal humerus fracture and for glenohumeral instability, complete the exercise program.

You should focus on strengthening the scapular force couples once your client demonstrates good control of the RC muscles. The addition of sport/activity-specific exercise is usually the final phase of your client’s rehabilitation program.

**Operative Treatment**

Indications for RC surgery are the presence of a full or partial tear that has not responded to a course of conservative care and that interferes with the client’s ADL. RC surgery is evolving rapidly as more and more physicians perform complex repairs through the arthroscope.

Arthroscopic debridement for freshening of the frayed, partially torn RC tendon stimulates healing. For full-thickness tears, the surgeon debrides the tear edges and then closes the defect to provide a foundation to regain RC strength and shoulder function. The surgeon often performs an acromioplasty at the time of RC repair to decompress the subacromial space and to prevent impingement of the repaired structures.

Most postoperative therapy programs include a 2- to 4-week period of immobilization while the tissue stabilizes. The client then starts therapy to regain ROM. For the next 2 to 3 weeks, ROM progresses from passive to active motion exercise. At approximately 8 weeks postoperative, the client begins strengthening exercises and follows the program for the nonoperative client listed before.

**Questions to Ask the Doctor**

**Nonoperative Clients**

- Are there any concurrent pathologies (such as instability or impingement)?
- Is surgery a possibility?

**Operative Clients**

- What structures were repaired? Try to obtain an operative report.
- Do you have a specific rehabilitation protocol?
- Are there any range of motion restrictions or precautions?
- How soon can strengthening begin?

**What to Say to Clients**

**About the Injury**

“Your shoulder relies on the rotator cuff muscles to stabilize the head of the humerus in the socket and to prevent the humeral head from being pinched against the roof of your shoulder when you raise your arm. Your rotator cuff is damaged, so this protective function has been interrupted, placing your shoulder at risk. If the problem progresses, you may lose the ability to raise your arm.”

**About Exercise**

“By strengthening the healthy portions of your injured rotator cuff, there is a good chance you can regain the ability to use your arm for your daily needs. The exercises are specific and need to be performed regularly.”

“When you perform the range of motion exercises, you should not experience sharp sudden pain when raising your arm up. To prevent this from occurring, you must lead the motion with the thumb side of your hand while keeping the point of your elbow facing the ground throughout the motion. Before raising your arm, you must correct your posture [as described in the section on thoracic outlet syndrome and brachial plexopathy]. By following these movement precautions, you will minimize the chance of pinching the rotator cuff under the bony roof of your shoulder.”

**Evaluation Tips**

A multitude of tests are used to detect RC disease. The following tests are included because they are easy to perform and research indicates they have reasonable sensitivity and specificity:

- A quick screening test to detect an RC tear involving the supraspinatus and infraspinatus is to resist ER with the client’s shoulders in neutral and the elbows flexed to 90 degrees. In the presence of an RC tear, the unopposed deltoid will abduct the arm while the hand dips into IR (Fig. 10-17).
- Client positioning to palpate the RC insertions are as follows:
  - Supraspinatus: With the client’s dorsum of the hand resting on the posterior iliac crest, palpate just inferior to the anterior aspect of the acromion (see Fig. 10-29, A, on the CD).
  - Infraspinatus: With the client’s shoulder in ER and the elbow brought to the navel, palpate just inferior to the posterior aspect of the acromion (see Fig. 10-29, B, on the CD).
  - The long head of the biceps tendon: With the client’s arm held in IR and the forearm resting on a pillow in the client’s lap, the tendon should lie in the deltopectoral interval (the sulcus formed by the medial border of the deltoid and the lateral edge of the pectoral muscle belly) (see Fig. 10-29, C, on the CD).
  - Subscapularis: With the client positioned as previously, bring the shoulder to neutral rotation. Palpate the lesser tuberosity and tendon in the deltopectoral interval (see Fig. 10-29, D, on the CD).
Shoulder impingement is a sign of RC disease. The Hawkins-Kennedy Test and Neer Impingement Test are useful in detecting this pathologic condition.65

- **Hawkins-Kennedy Test:** With the client’s shoulder flexed to 90 degrees, bring the shoulder into full IR. This drives the greater tuberosity under the coracoacromial arch and will elicit pain if impingement is present (Fig. 10-18, A).

- **Neer Impingement Test:** Passively flex the client’s shoulder to end ROM. Positioning the shoulder in IR at the start of the test enhances the impingement of the RC on the underside of the anterior third of the acromion and coracoacromial ligament (Fig. 10-18, B).

- **Isometric testing of the specific muscles of the RC and the long head of the biceps aids in detecting tendonopathy.** These tests include the Jobe or “Empty Can” Test, the Patte Test, the Gerber Liftoff Test, and the Speed’s Test.

- **The Jobe or “Empty Can” Test:** Bring the client’s arms to horizontal in the scapular plane with the shoulder in IR. Next, apply downward pressure while the client provides isometric resistance. Weakness or the inability to hold this position implicates the supraspinatus66 (Fig. 10-19, A).

- **The Patte Test:** The client’s arm is positioned in 90 degrees of abduction with neutral rotation in rotator cuff disease. A, End test position of the Hawkins-Kennedy Impingement Test. B, End test position of the Neer Impingement Test.
FIGURE 10-19  Isometric testing of muscles of the rotator cuff. A, Test position for isometric testing of the supraspinatus or the "empty can" test. B, Test position for the Patte Test for isometric strength of the infraspinatus and teres minor. C, Test position for the Gerber Liftoff Test for isometric strength of the subscapularis. D, Speed's Test for pathologic conditions of the long head of the biceps tendon and superior glenoid labrum.

the scapular plane. Apply pressure at the client’s wrist to resist shoulder ER while stabilizing the arm at the client’s elbow. Weakness and/or pain implicates the infraspinatus66 (Fig. 10-19, B).

- The Gerber Liftoff Test: Position the client’s arm so the dorsum of the hand rests against the posterior iliac crest. Have the client actively raise the hand 2 to 5 inches from the back while you apply resistance (Fig. 10-19, C). The client’s inability to apply pressure or hold the hand in this position implicates the subscapularis.67

- The Speed’s Test: Position the client’s shoulder in 90 degrees of flexion with the elbow in extension and the forearm in supination. Resist shoulder flexion while palpating the long head of the biceps tendon in the bicipital groove. A painful response implicates the long head of the biceps68 (Fig. 10-19, D).

- No resistance test is specific to isolating the teres minor.

- While performing these tests, watch for patterns that support specific locations of RC lesions.
## Diagnosis-Specific Information That Affects Clinical Reasoning

The existence of an RC tear does not always lead to a surgical repair. Clinical experience shows that many clients function well with RC deficits confirmed via MRI. Clients who are unable to regain pain-free shoulder function are the best candidates for surgical repair.69

### Tips from the Field

Good palpation skills aid substantially in differentiating subacromial-subdeltoid bursitis from RC tendonitis. The bursa lies beneath the therapist’s finger as well when palpating the supraspinatus and infraspinatus insertions as described earlier. Since the bursa is a relatively fixed structure, its position changes little during arm movement. Conversely, the palpation locations for the RC tendons change with arm movement. A pain response with palpation implicates both structures.

### CASE Studies

### CASE 10-1

J.S. is a 48-year-old right dominant stock broker, non-smoker, who received a diagnosis of mild brachial plexus stretch injury. He reported symptoms of headache in the C2 distribution, anterior chest wall pain on the left, and pain in the left anterolateral neck extending to the angle of his jaw and to the anterior aspect of his shoulder. He noted occasional swelling with flushing of the left side of his face, extending to his ear. This symptom became more prevalent when his left upper quarter pain was high. He was experiencing slight numbness of the thumb, index, and long finger, and arm pain on the inside of the proximal humerus of the left UE while sitting at his desk and with overhead activities. He reported difficulty combing his hair because of arm pain.

Symptoms began 2 months ago when he decided to return to surfing after a 15-year hiatus. He stopped surfing about 1 month ago because his face, chest wall, and arm symptoms would increase dramatically during and for days after time spent on his board.

He had a thorough cardiac workup and was cleared. X-ray films and MRI of the cervical spine were unremarkable. He is in excellent physical shape, with an unremarkable medical history.

On exam, he demonstrated a drooped left shoulder with a forward-head–rounded-shoulders posture. Cervical ROM was limited 25% only for rotation and lateral flexion to the right. Crossed-arm cervical ROM testing showed no improvement in motion. Shoulder ROM was limited in flexion to horizontal by arm pain, with IR and ER being full. RC strength was at 5/5 bilaterally and pain free on testing. Muscle spasm occurred in the suboccipital region, across the trapezius ridge and along the medial scapular border.

Palpation at the left supraclavicular fossa was tender with spread of pain into the side of the face to the ear. Palpation at the left infraclavicular fossa and neurovascular bundle was painful with an increase of left UE symptoms. Tinel’s sign was positive in the same areas.

Decreased sensitivity to pin prick of the thumb, index, and radial side of the long finger on the left was noted. The Roos (EAST) Test was positive immediately. Neural mobility of the brachial plexus was at 2+=5 on the left with increase of left upper quarter symptoms and 5/5 on the right.

### Impressions

Based on clinical presentation, J.S. demonstrated evidence of an upper trunk–lateral cord brachial plexopathy with decreased plexus mobility restricting UE elevation. Cervical motion loss implicated scalene muscle tightness.
Facial symptoms with headache combined with shoulder droop implicate the upper trunk. Decreased sensitivity to pin prick on the radial side of the long finger continuing to the index finger and thumb implicate the lateral cord as well. No change in cervical motion with the crossed-arm position combined with the pattern of cervical motion loss implicates the scalene muscles.

Because J.S. has full shoulder IR and ER, his limited UE elevation likely was not due to a capsular restriction. A pathologic condition of the RC is unlikely as well because RC strength was 5/5 and he was pain free on testing.

**Treatment**

Treatment on the first visit consisted of instruction in posture correction exercises, modification of computer placement to encourage proper posture while working, and to limit all overhead activities. J.S. was instructed in nerve gliding exercises to be performed 3 times a day in sets of 3, with his posture correction exercises to be performed hourly in sets of 10.

J.S.’s work schedule only allowed for weekly visits, so his treatment program was designed accordingly. On his second visit, J.S. demonstrated excellent technique with his home program. He had avoided all overhead activity and modified his work station to encourage proper posture. He reported a 50% improvement in symptoms, with no headache for the past week.

Brief exam revealed neural mobility at 3+/5, good postural awareness and less left shoulder drooping.

Treatment consisted of pectoral minor and scalene muscle stretching with nerve glides. Neural mobility improved to 4+/5 by the end of treatment. J.S.’s home program was modified to include scalene muscle stretching and pectoralis minor doorway stretches. He was allowed to perform nerve glide and stretching exercises as long as his symptoms remained stable in sets of 3 for 3 times a day. Scapular clock exercises were added to work on proprioception on an hourly basis.

On J.S.’s visit 1 week later, he again demonstrated excellent compliance with his home program. He had full neural mobility and his cervical ROM was full. Shoulder position was equal bilaterally. The EAST was positive at 45 seconds of testing.

Treatment consisted of home program modification with the addition of scapular strengthening with focus on the upper, middle, lower trapezius, and serratus anterior using resistance bands. All exercises were performed daily in sets of 3 once through each exercise before starting the next set. J.S. was instructed to stop exercising if symptoms returned.

**Results**

On the fourth visit 2 weeks later, J.S. had been symptom free for 1 week. Exam was normal except for a positive EAST Test at 1 minute of testing, leading to paresthesia into the thumb and index finger. Because his condition was stable and he had excellent understanding of his home program, J.S. was discharged from therapy with instruction to call as needed for program progression or if his status changed. He was instructed to avoid surfing.

Because J.S. was in excellent shape before his injury and he was compliant and motivated, he quickly stabilized and returned to a functional baseline. Many clients have difficulty modifying their lifestyle to avoid further injury to their brachial plexus, resulting in a protracted recovery period.

**CASE 10-2**

F.B. is a 75-year-old right dominant female who tripped over her cat and sustained an A2 fracture of the proximal humerus. Three weeks after injury, she came to therapy in a simple sling for immobilization. Therapy orders were for ROM exercises with no strengthening for the next 3 weeks. F.B. was apprehensive about moving her arm because she felt that the break was still unstable since not enough time had passed from her injury date.

Before moving F.B.’s right shoulder, I explained that sound will not travel across an open fracture and used a stethoscope at the humeral head while tapping on the lateral epicondyle to demonstrate to her there was no difference in the sound level. Next, I showed her that the head of her humerus was moving as I rolled her arm from IR to ER, again indicating that she was ready to begin ROM exercise.

Exam revealed severe ecchymosis in the axilla and around the elbow. I explained that when she fractured her arm, there was a lot of bleeding from the bone and that blood had run down her arm along the interior tissue planes to collect around her elbow and distal axilla.

Shoulder elevation was 50 degrees, abduction was 40 degrees, IR was arm across the abdomen and thumb to the iliac crest while attempting arm behind back position. ER was neutral. ROM of the elbow was 20 to 110 degrees with wrist and hand ROM normal. Strength testing was deferred at that time.

**Treatment**

I instructed F.B. in pendulum and tabletop ROM exercises that she was to perform hourly in sets of 3 to 10, up to 3 sets each exercise session. She started ROM exercises for her elbow that she was to perform whenever sitting.

F.B.’s treatment over the next 2 weeks focused on scapulothoracic stabilization via manual resistance applied to the scapula along with scapular mobilization and pro-
prioception scapular clock exercises. PROM of the shoulder also was stressed, focusing on avoidance of substitution patterns of excessive scapular elevation and protraction.

Reevaluation 2 weeks later revealed ROM of elevation at 110 degrees, abduction at 90 degrees, and ER to 30 at 75 degrees of abduction and 40 degrees at neutral shoulder abduction. IR was at thumb to L5 reaching behind the back.

Treatment continued to focus on ROM, stressing active motion in normal movement patterns as F.B., mirrored my UE elevation while I stabilized her shoulder to prevent early scapular elevation. F.B. then added wall walking to her program incorporating self-stabilization of the scapula with her left hand.

At 6 weeks after injury, F.B.’s shoulder elevation was 150 degrees, abduction was 135 degrees, ER at 90 degrees of abduction was 50 degrees, ER at shoulder neutral was 60 degrees, and IR behind the back had progressed little at thumb to L3. Elbow, hand, and wrist ROM were full.

Because F.B. then was allowed to begin strengthening, isometric exercises for all planes of shoulder movement and resistance band exercises of the elbow flexors and extensors were added. Behind the back stretches with good scapular position and upright posture were stressed as well. At this point the therapy program included joint mobilization and more aggressive stretching to focus on gaining end ROM.

**Results**

F.B. attended therapy for 6 weeks. She had 160 degrees of elevation, 150 degrees of abduction, and 70 degrees of ER at 90 degrees of abduction and with the shoulder at neutral. IR behind the back remained restricted with the thumb reaching the L2 spinous process. F.B. was fully independent in self-care with her right arm, and she was pain free. Shoulder strength was 4/5 except for abduction and ER, which were 4–5/5. She never regained full IR because there may have been a rotary component to her fracture as she healed, limiting the potential for normal IR ROM.

**CASE 10-3**

T.B. is an 18-year-old high school football player who sustained a traumatic anterior dislocation of his right (dominant) shoulder. MRI demonstrated a Bankart lesion with anterior glenoid labrum and capsule tear. Because T.B. was a heavily recruited athlete, he opted to have arthroscopic shoulder surgery 4 weeks after injury.

T.B. presented to therapy 2 weeks after surgery with his arm in a sling immobilizer.

Active motion was contraindicated to avoid disrupting the surgical repair. PROM of the right shoulder was 90 degrees of flexion, 40 degrees of abduction, and 10 degrees of ER. IR was arm across stomach. Strength testing was deferred.

**Treatment**

Treatment initially focused on protecting the surgical repair by following ER ROM restrictions to 40 degrees. Scapular proprioception exercises were issued for home program to get a head start on preventing poor scapular kinematics. These consisted of half shoulder rolls in the posterior direction only and clock shoulder elevation to the 12, 1, and 2 o’clock positions performed in front of a mirror for feedback.

Over the next 4 weeks treatment consisted of submaximal isometric exercises of the internal and external rotators with ER limited to 40 degrees to avoid disrupting the repaired anterior structures. Scapular stabilization exercises continued during this period. PROM manual therapy was the primary component of treatment.

At 8 weeks, T.B. had 145 degrees of flexion with active flexion at 135 degrees and ER at 40 degrees. ER safe zone was now to 60 degrees. The protocol allowed for resistance band exercises of the external and internal rotators. Active shoulder elevation by T.B. while mirroring the therapist with the therapist preventing early scapular elevation was started.

During the next 4 weeks, the resistance band exercises progressed to include shoulder elevation and abduction as long as scapular substitution patterns were avoided. Strengthening of the triceps and scapular depressors/stabilizers by doing seated press-ups were added. Manual resistance exercises in diagonal planes supine and seated with focus on normal kinematics also were started. Throughout all phases, ROM exercises were stressed.

By 12 weeks, flexion was 170 degrees, abduction was 165 degrees, and ER was 60 degrees with IR at 70 degrees. Strength of the shoulder was 5/5 for extension, with flexion and IR at 4+/5; ER and abduction was at 4/5. By then, all motion restrictions were removed. The focus of the next 4 to 6 weeks was to gain full ROM and full strength and to start sport-specific training. Plyometric drills with two-hand throw-catch activities using weighted balls were started.

**Results**

By 18 weeks, T.B. had full ROM, and scapular kinematics/shoulder mechanics were normal. He had returned to work out at the gym using a custom program designed to strengthen the anterior shoulder structures and globally strengthen the RC, deltoid, serratus anterior, pectoral, and back muscles. Because of his successful rehabilitation, T.B. went on to play collegiate football.
References


