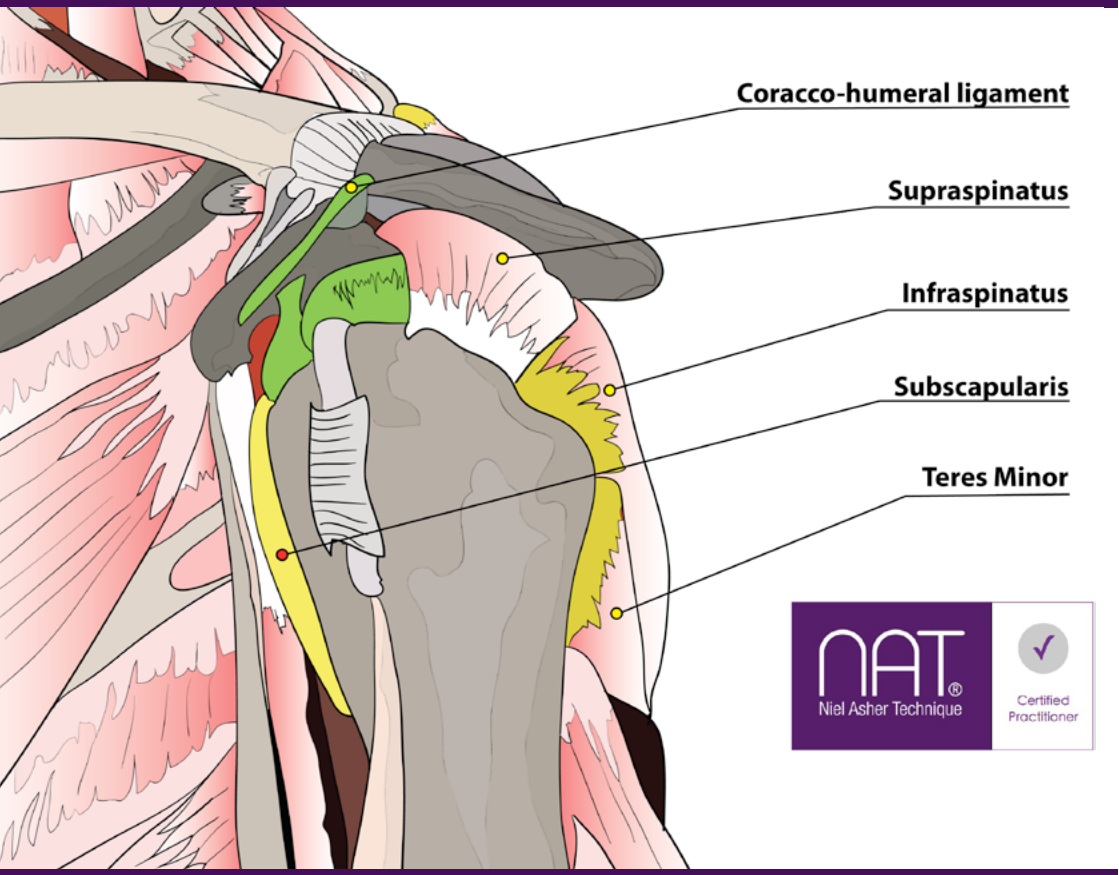
NielAsher.

Advanced Trigger Point Techniques



Trigger Point Therapy

Treating The Frozen Shoulder

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NAT Pro Series:

Treating Frozen Shoulder

Access Course

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Welcome

Welcome to the NAT Access course for treating frozen shoulder syndrome. Thank you for deciding to learn our unique and clinically tested approach to treating the frozen shoulder.

Whilst we hope that you will find this access course clear and simple to follow, there is no substitute for experience. The more you practice with the technique, the more adept you will become. Persevere with the technique and have faith; it really does work, on all types of frozen shoulders. Whilst it is tempting, it is essential you do not add or subtract from the method. Please follow the technique step by step to achieve maximum results.

We have designed this course to give you new information and reinforce what you have previously learnt. It is therefore important that you do not skip sections as each one is essential.

At the end of this course you will find a reflective learning exam. This is not a 'pass or fail' test but a mechanism to see that you have understood the information and can apply it for the good of your patients.

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What is a frozen shoulder?

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What is a frozen shoulder ('FS')?

Frozen shoulder syndrome is a commonly encountered clinical complaint by clinicians (GPs, Rheumatologists, Orthopedic surgeons) and physical therapists (Physiotherapy, Osteopathy, Chiropractic). Defining 'frozen shoulder' is not straightforward. It has been used incorrectly as a general diagnosis for shoulder pain and stiffness. The definition, etiology, pathophysiology and treatment of this condition are subjects of debate. Since being initially labeled as peri-arthritis by Duplay in 1872, it was then coined frozen shoulder by Codman (1934) and finally adhesive capsulitis by Neviaser (1945), who was the first to recognize the 'chronic inflammatory process' that resulted in capsular contraction. The terms frozen shoulder and adhesive capsulitis continue to be used commonly.

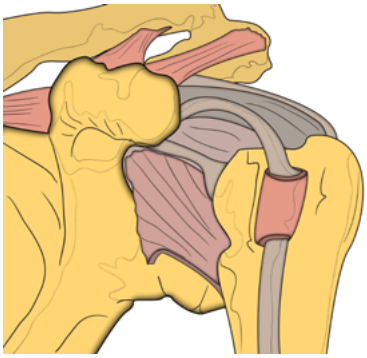
We will define frozen shoulder syndrome after Grubbs as:

"a soft tissue capsular lesion accompanied by painful and restricted active and passive motion at the glenohumeral joint" (Grubbs 1993).

Pathophysiology

Inflammation of: Synovium; LHB; Coracohumeral Ligament; Rotator Cuff Interval; Subacromial Bursae; Subscapularis Muscle

Contracture: Capsular Pattern; Neurologically Mediated; Connections with RSD/CPRS 1



Inflammation

It is believed that inflammation of the synovium and subsynovial tissue is the underlying pathological process in frozen shoulder (Neviaser 1945, Lundberg 1969). It is generally well accepted that synovial inflammation acts as the precursor to the capsular contraction and fibrosis seen in later stages of the disease (Hannafin & Chiaia 2000, Wiley 1991). The reason for this histologically observed inflammation is unclear, but it has been suggested, amongst others, to be a reaction to injury, an infectious agent or an autoimmune reaction.

Inflammation of the long head of the biceps (LHB)

The head of the long head of the biceps tendon has a unique relationship with the capsule. The tendon originates from the supraglenoid tubercle of the scapula just above the socket of the gleno-humeral joint. It runs laterally across the top of the humerus within its sheath until it becomes deviated inferiorly through the bicipital groove of the humerus. After running down the humerus for approximately 5-6 cm it blends with the biceps muscle to form a musculo-tendinous junction. From there it blends with fibers from the short head of the biceps (SHB) to form the main belly of the biceps. It is postulated that a primary inflammation and stenosis in the region of the biceps tendon could spread to involve the capsule itself. This has been borne out through the careful clinical observation of over a thousand cases. Because the LHB is intra-capsular, inflammatory cells, exudates and products of the inflammatory cascade can track along the tendon directly into the capsule. Once inside the capsule, the inflammation seems to spread rapidly.

Inflammation and the capsule

The role of cytokines in the onset of inflammation is well described (Kofler et al. 2004, Cheon et al. 2004). The presence of specific cytokines has been demonstrated in the inflammatory phase of frozen shoulder (Rodeo

et al. 1997). Prolonged production of cytokines has been shown to trigger increased fibroblast activity and fibrosis (Border & Noble 1994, Hannafin & Chiaia 2000).

Within a healthy glenohumeral joint, the potential joint volume amounts to approximately 35-50ml. In frozen shoulder, capsular fibrosis reduces this space to as little as 5-10ml (Manton et al. 2001). It has been suggested that the process is somewhat similar to that of Dupuytren's contracture (Bunker & Anthony 1995), in which fibroblasts lay down collagen as a thick nodular band or fleshy mass.

By breaking the inflammatory cascade, potentially one can prevent or limit the severity of the ensuing capsular contraction that typifies frozen shoulder. We will be proposing a systematic treatment for breaking this cascade and accelerating its cessation.

Stiffness

Although the initial inflammatory process in FS is intra-capsular, the subsequent immobilization of the arm also results in involvement of the extra-capsular tissues. The cytokine-induced increase in fibroblast activity causes an increase in deposition of collagen fibrils. An increase in the percentage of connective tissue within muscle can lead to a relative ischemia, causing further fibrotic change within the muscle. Fibroblasts, along with myocytes, are mechano-transducers. These ubiquitous cells translate mechanical stress into electrical signals, laying down collagen fibrils along lines of mechanical stress.

In FS there is a lack of both active and passive motion at the gleno-humeral joint. In the absence of mechanical stimuli, collagen fibrils are laid down in a haphazard fashion and connective tissue becomes less organized. Immobilization has been shown (Okita et al. 2004) to result in a reduction of the number of sarcomeres and an increase in cross-bridge

formation between adjacent collagen fibrils within the extracellular matrix of surrounding connective tissue. Muscle length and speed of muscle contraction decreases (D'Antona et al. 2003).

Fibrotic changes appear due to enhanced connective tissue deposition. Muscles therefore become less extensible and investing fascia more extensive and less elastic, further compounding the changes seen in the intra-capsular space. Indeed in some cases, the peri-articular changes may be the primary reason for the restriction of movement, as it has been found that intra-capsular adhesions are not reliably present when the capsule is viewed arthroscopically (Nash & Hazleman 1989). Reduced shoulder movement leads to an alteration in the properties of synovial fluid and reduced production of synovial fluid, further compounding the inability to move the affected limb.

Neurological mediation

Clinical research would suggest that the pain of frozen shoulder is neurologically mediated and due to dysfunction of the sympathetic nervous system in the upper extremities of patients with idiopathic FS. Histological similarities between Complex Regional Pain Syndrome Type one (CRPS1) and FS would suggest that known pain-producing mechanisms in CRPS1 may be responsible for the pain of FS. Such mechanisms include sympathetically-mediated facilitation of joint nociceptors and proprioceptors, local release of substance P, sensitization of interneurons in the dorsal horn of the spinal cord and degeneration of the terminal sensory nerve fibers of the glenohumeral joint capsule.

In addition to the fibrotic changes seen, in the early stages of frozen shoulder there is a rapid muscular wasting, not dissimilar to that seen post-surgically. Such wasting is too rapid to be attributed to

disuse atrophy alone, and is presumed to be a neurological protective mechanism, to prevent movement of the 'injured' part.

Due to the risk of tearing already weakened muscle tissue, the appropriateness of vigorous stretching techniques, to mobilize the frozen shoulder, must therefore be questioned. This risk is further compounded by the failure of the fibrotic tissues to yield, thereby accentuating the forces generated at the musculo-tendinous junction, the site at which most muscle injuries occur.

Epidemiology

Who gets it?

- Frozen shoulder syndrome affects 2-5% of the general population (Grubbs 1993). In diabetics the incidence is between 10-20% (Pal et al. 1986).
- Frozen shoulder affects females slightly more than males (60:40) (Baslund 1990), typically between 40 and 60 years of age (Grubbs 1993); In Japan it is known as 'fifties shoulder'.
- The non-dominant arm is more likely to be involved (Fareed & Gallivan 1989).
- About 12-15% of people are affected bilaterally (Wadsworth 1986), although involvement of the contralateral shoulder frequently occurs in a delayed fashion, after the initially affected shoulder starts to recover.

Etiology

Frozen shoulder can be classified by etiology as either primary or secondary (Lundberg 1969).

Primary

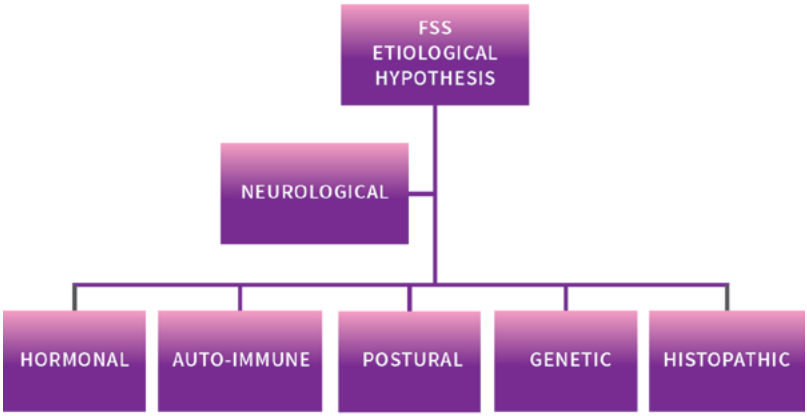
Primary or idiopathic frozen shoulder appears for no apparent reason, although patients often try to attribute the onset to some minor strain such as from playing tennis or reaching for the back seat of the car. The inability to identify a cause can sometimes add to the distress of the condition, as patients are unable to put a rationale or cause to their symptom picture. Idiopathic frozen shoulder is associated with many medical conditions including: diabetes mellitus, chronic obstructive pulmonary disease, ischemic heart disease, cervical disc disease, thyroid disorders and various neurological conditions, including Parkinson's and hemiplegia (Rizk 1982). Other factors such as depression, immunologic factors, posture and occupation have been implicated in the etiology (Murnaghan 1990).

Secondary

Secondary frozen shoulder is associated with a pre-existing painful condition, either intrinsic or extrinsic. Intrinsic causes of frozen shoulder syndrome include rotator cuff tears or tendonitis, whereas extrinsic causes of secondary frozen shoulder syndrome are of the post-traumatic variety such as fracture, surgery or a fall on an outstretched arm. It is interesting to note that frozen shoulder syndrome may result after shoulder surgery, especially if this involves immobilization/splinting. It may also follow from mastectomy and breast reconstruction.

There are several competing theories as to the causes:

- Hormonal
- Auto-immune
- Genetic
- Postural



Capsular Pattern

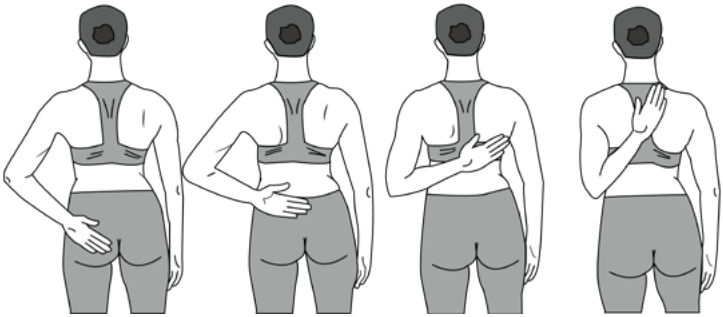
Although the majority of patients exhibit the typical capsular pattern of restriction as described by Cyriax (1983), researchers have found that there is no universal capsular pattern (Rundquist et al. 2003). This is consistent with our findings in the clinical setting where we have classified frozen shoulder sufferers into 3 types designated as anterior, posterior, and lateral, according to the particular pattern of restriction presented.

Treating trigger points initiates a cascade of neuro-vascular responses at the local tissue level, the spinal cord and in the cortex. Mitchell and Schmidt (1977) demonstrated that stimulating myofascial mechanoreceptors produced a response in the local autonomic loop,

altering the blood pressure in local arterioles and capillaries. Additionally, stimulation of Ruffini endings appears to have a similar effect in terms of a lowering of sympathetic activity (Van den Berg and Cabri 1999).

Normal and Functional Range of Motion

Full Movement Patters	Functional Motion
Flexion (FW elevation) 180°	120 – 150°
Abduction 180°	120 – 150°
External Rotn. @ side 90°	65 – 90°
Horizontal External Rotn. 90° or more	65 – 90°
Horizontal Internal Rotn. 75°	60 – 75°
Reach behind back	Bottom of shoulder blade Top of hip



What is the capsular pattern?

The shoulder's capsular pattern is as follows:

1. Limitation in active and passive external (lateral) rotation,
2. Limitation in active and passive (scapulohumeral) abduction,
3. Least limitation in passive internal (medial) rotation in front of the body.

Natural History

Frozen shoulder passes through the three phases of freezing, frozen and thawing (painful, stiff and resolving) lasting an average of 30 months (Reeves 1975, Rizk 1982). These stages are not discretely defined entities, but represent more a continuum of the disease process.

The freezing (painful) phase lasts between 2-8 months. This is followed by the frozen (stiff) phase, which lasts between 4-12 months.

Spontaneous recovery of mobility (thawing) follows over the next four to twelve months. On recovery, shoulder movement may return fully, but an objective restriction of mobility often persists (Reeves 1975). In one study involving 62 cases, half still had symptoms at 2-11 years (Shaffer 1992).

Pre Phase I

It is worth noting that some authorities talk of a 'pre-adhesive' stage (Neviaser 1987) before the freezing phase. Here patients present with signs and symptoms of 'impingement syndrome', where range of movement is unaffected but there is a catching in certain positions. At this stage, confirmation of a diagnosis of FS would depend on arthroscopic evidence of synovial inflammation and an increase in capsular thickness. Ultrasound at this point may also be of value in demonstrating a loss of the inferior capsular fold/recess.

Clinical Findings

The diagnosis of frozen shoulder is usually based on clinical findings, but in some patients may require further investigations to exclude alternative pathologies. Determining from the history which stage a patient is in is vital to determine the appropriate treatment approach.

Phase I - Freezing

The freezing (painful) phase: At rest the affected arm is held in adduction and internal rotation, and the scapula of the affected side is usually elevated, laterally rotated and abducted (Reeves 1975). This self-immobilization only serves to exacerbate the stiffness. Depending on the longevity of symptoms, the body may develop a compensatory mechanical adaptation.

Patients commonly complain of sharp pain reaching for the back pocket or a high shelf, combing the hair, or doing up the bra (Kesler 1983). Severe night pain is a common feature of this phase. People often complain that they are unable to sleep on the affected side. The pain itself can be quite horrendous. Patients usually describe 3 types of pain:

- A constant 'internal' dull burning in the gleno-humeral joint.
- Pain down the lateral aspect of the upper arm (possibly in the C5 & C6 dermatome).
- Severe sharp catching pain on certain innocuous movements lasting up to two minutes (in the region of the LHB bicipital groove).

Along with this there can often be rapid stiffening of the whole shoulder.

Phase II - Frozen

The frozen (stiff) phase: There may still be night pain but this usually diminishes as shoulder mobility decreases. Here patients are usually able to sleep but find it increasingly difficult to perform daily chores, especially in those affected bilaterally. Menial tasks become titanic achievements. Pain can often radiate into the forearm or hand, and in some cases the hand can become swollen and painful. This may be the result of reflex sympathetic dystrophy (RSD). Pain may also start at the back of the shoulder in the region of the triceps muscle, due to a 'triceps tendonitis'.

Common Associated Phenomena* include:

• Adhesions between the layers of the sub acromial bursa (adhesive bursitis)

• Extra-articular adhesions

• Adherence of the anterior and inferior capsular folds to each other and the adjacent glenoid

• Contracture of subscapularis and the biceps tendon.
Shortening of the coracohumeral ligament

• Contracture of the girdle muscles.

• Stenosis of the biceps tendon

** (Henry 1995)*

Phase III - Thawing

The thawing (resolving) phase: Spontaneous recovery of mobility follows over the next four to twelve months, although full recovery is commonly protracted. Occasionally people may awake after 18 months to find they are fully better, although in my experience this is rare. Without treatment, even after the thawing phase an objective restriction of mobility may often persist for several years (Shaffer et al. 1992).

Without Treatment	**NAT**
Pre-Freezing (0-4 weeks)	1-5 Sessions
Freezing (1-8 months)	7-13 Sessions
Frozen (9-16 months)	5-8 Sessions
Thawing (12-40 months)	4-7 Sessions

Risk Factors

Aging - In Japan frozen shoulder syndrome is called 'Fifties Shoulder'.

Posture - especially round-shouldered Shoulder-intensive sports

Occupation - Shoulder intensive or repetitive manual work

Diabetes - Types I and II Trauma

Immobilization / splinting

Fracture of the collar bone or humerus (arm bone)

Surgery (especially after shoulder surgery, or mastectomy with breast reconstruction)

Part II

Anatomy

Bursae

Capsule and ligaments

Muscles of the upper arm and shoulder

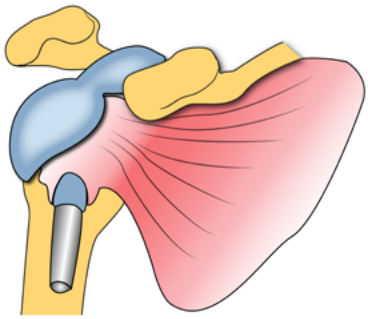
Anatomy

Key anatomical landmarks

Bursae

Many bursae adjoin the shoulder joint. Those consistently present are: The sub acromial bursa between deltoid and the capsule, extending under the acromion and coracoacromial ligament, and between them and supraspinatus: Birnbaum & Lierse (1992) found it to be attached, together with the sub deltoid fascia, to the acromion between the tendon of subscapularis and the articular capsule, communicating with the joint through the foramen of Weitbrecht, lying between the superior and middle glenohumeral ligaments; Between teres major and the long head of triceps; anterior and posterior to the tendon of latissimus dorsi; on the superior aspect of the acromion; Between the coracoid process and capsule (not always present).

Glenohumeral joint



The glenohumeral joint is a multiaxial, spheroidal joint, which links the free limb through the head of the humerus to the pectoral girdle. The glenohumeral joint depends for its support on the integrity of the rotator cuff muscles, rather than its bony architecture or ligaments, having sacrificed stability for mobility. The glenoid cavity is deepened by the presence of a fibrocartilaginous rim, the glenoid labrum, which blends above with two fasciculi from the long tendon of biceps.

Fibrous capsule

The fibrous capsule envelops the joint, attaching medially to the margin of the glenoid labrum, and encroaching on the coracoid process to include the attachment of the long head of biceps. Laterally, it is attached to the humeral anatomical neck, except inferomedially where it descends more than 1cm on the humeral shaft. It is so lax that in the abducted position the bones can be distracted 2 or 3cm, allowing for a very wide range of movement. With the arm hanging loosely at the side, there is a loose recess inferiorly, sometimes called the axillary fold, to allow space for the head of the humerus during full abduction. The fibrous capsule is supported by the tendons of supraspinatus (above), infraspinatus and teres minor (behind), subscapularis (in front) and by the long head of triceps (below). The rotator cuff muscles reinforce the capsule and actively support it. Inferiorly the capsule is least supported and also subjected to the greatest strain, being stretched tightly across the humeral head when the arm is in full abduction. Extensions from the tendons of pectoralis major and teres major serve to further strengthen the capsule anteriorly.

Joint Space

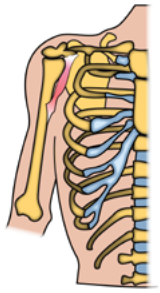
Although authors commonly speak of the glenohumeral joint space, the space is a potential space, which can only be demonstrated after distension of the capsule with air, saline or contrast medium. There is minimal free synovial fluid within the confines of the articular surfaces and the joint capsule of the normal glenohumeral joint. Authorities have suggested a normal shoulder has between 60-70ml of free synovial fluid within the capsule. It is interesting to note that in a frozen shoulder this may reduce to as little as 5ml. Numerous theories have been posited regarding the mode of joint lubrication; it being latterly suggested that the fluid there present is trapped within irregularities in the surface of articular cartilage – the boosted lubrication theory of synovial lubrication (Longfield et al. 1969). The synovial fluid is squeezed out of the cartilage with joint movement.

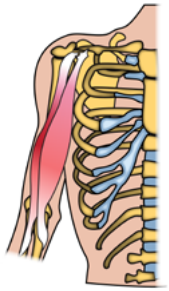
Glenohumeral Ligaments

The three glenohumeral ligaments reinforce the capsule. The superior glenohumeral ligament passes from the base of the coracoid process and upper part of the glenoid labrum to the upper part of the neck of the humerus, between the lesser tubercle and the articular margin. The middle glenohumeral ligament arises from a wide attachment along the anterior glenoid margin and passes inferolaterally to attach to the lesser tuberosity, deep to the tendon of subscapularis, with which it blends. The thicker and longer inferior glenohumeral ligament arises from the anterior, middle and posterior margins of the glenoid labrum and passes anteroinferiorly to attach to the inferior and medial aspects of the neck of the humerus. The inferior ligament is thickened anterosuperiorly to form the superior band, which attaches to the anterior aspect of the capsule at the axillary pouch.

The Upper Arm

The long head of biceps tendon passes in the intertubercular sulcus between the insertions of teres major and pectoralis major, held in place by the transverse humeral ligament, and becoming intracapsular with its investing synovial sheath to attach to the supraglenoid tubercle at the root of the coracoid process. The short head remains extra-capsular inserting into the coracoid process, the tip of which is palpable through the anterior fibers of deltoid about 2.5cm below the junction of the lateral fourth and rest of the clavicle. The narrow, elongated coracobrachialis lies medial to the short head of biceps. The axillary artery and vein, along with the lateral and medial cords of the brachial plexus, lie medial to coracobrachialis and biceps in their proximal part.





Rotator Cuff Muscles

The rotator cuff muscles (supraspinatus, infraspinatus, teres minor and subscapularis) act as the fixators of the shoulder, acting to stabilize the head of the humerus in the glenoid cavity. As fixators they are deeply placed, blending with the capsule and forming a musculotendinous cuff that reinforces the capsule.

Researchers have shown (Clark & Harryman 1992) that the four tendons of the rotator cuff fuse to form a common insertion on the humeral tuberosities. Deep fibers from subscapularis and infraspinatus interdigitate with those of supraspinatus. The tendon of infraspinatus and teres minor are difficult to separate from each other in vivo, functioning more as a conjoined rotator cuff tendon, with the supraspinatus and infraspinatus showing the greatest interdigitation (Curtis et al. 2006).

The tendinous portion of the cuff is also confluent with the shoulder capsule and the glenohumeral and coracohumeral ligaments forming a five-layered cuff-capsule complex

Ligamentous Anatomy

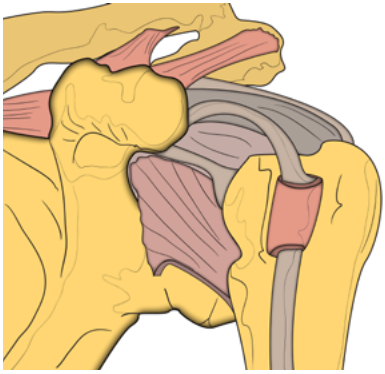
Coracoclavicular Ligament
- Trapezoid
- Concoid

Acromioclavicular Ligament

Transverse Humeral Ligament

Glenohumeral Ligaments
- Superior Glenohumeral Ligament
- Middle Glenohumeral Ligament
- Inferior Glenohumeral Ligament

Coracohumeral Ligament

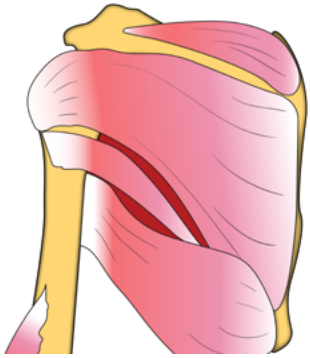


Rotator Cuff Muscles

Muculotendinous Cuff

- Supraspinatus
- Infraspinatus
- Teres Minor
- Subscapularis

Deltoid



Movement and Stability of the Shoulder

Movements of the shoulder girdle and pectoral girdle are described separately in anatomical texts, but this division is entirely arbitrary, as movements such as raising the arm above the head into flexion or abduction are brought about by a combined action of the both the glenohumeral joint and the pectoral girdle. Limitations of any part of this complex impair movements of the whole.

The stability of the shoulder is largely dependent on the integrity of the surrounding musculature, the capsule and ligaments of the joint being slack in most of the joint's positions until end range is reached. Various mechanisms contribute to mid-range stability of the glenohumeral joint including limited joint volume and adhesion- cohesion (Goldstein 2004). Dynamic stability of the glenohumeral joint is conferred by means of concavity compression (Matsen et al. 1994), also called coaption of the glenohumeral joint. Concavity compression, first described by Lippitt et al. (1993), refers to the compressive forces placed on the glenohumeral joint during rotator cuff muscle co-contractions. These forces press the