BULLETIN OF
THE NEW YORK ACADEMY
OF MEDICINE

VOL. 36, NO. 8 AUGUST 1960

THE COMPONENTS OF THE FROZEN SHOULDER*

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THE term "frozen shoulder", though in common use by orthopedic surgeons, is not often well defined. The clinical findings which delineate this condition are as follows[1]:

Pain is an outstanding complaint. It is usually the first noticed element, present at rest and becoming severe during the night.

Initially, restriction of motion seems voluntary on the basis of pain; subsequently it is noted that there is restriction of both active and passive motion.

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* Presented in part by Dr. Kopell at The New York Academy of Medicine before a combined meeting of the Section on Orthopedic Surgery and the Philadelphia Orthopaedic Club, March 9, 1959.
From the Neuro-Muscular Study Group, New York University-Bellevue Medical Center. Supported by the Charles A. Dana Foundation.

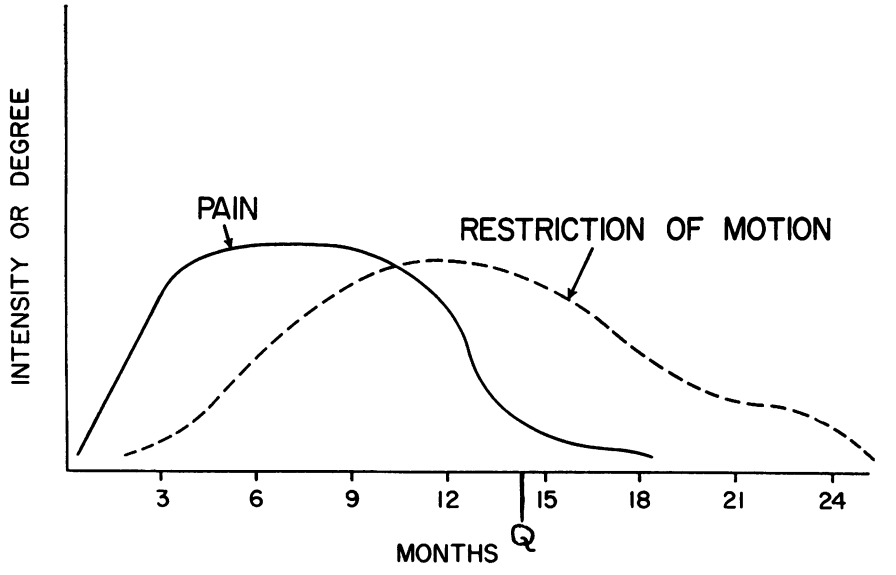

Fig. 1—Relationships of motion restriction and pain generalized for a frozen shoulder.

Significant shoulder trauma may not be recalled, but this entity may follow other upper extremity injury such as a Colles' fracture.

In some cases there may be a history of a previous acute or subacute inflammatory episode which had been ascribed to a bursitis. The acute episode was then gradually replaced by the clinical picture of a frozen shoulder.

The condition is usually two or three months old before the patient is seen by an orthopedic surgeon.

The patient is almost always more than 30 years of age.

There is some atrophy about the shoulder. Early in the course of the condition discrete points of tenderness are usually not elicited.

X-rays of the shoulder are often negative, though in some cases calcification at the insertion of the rotator cuff may be present. The laboratory findings are within normal limits.

This particular type of joint involvement is peculiar to the shoulder.

The general course of the disease follows the pattern shown in Figure 1.

Before going further, some terms require definition. The first is shoulder motion. Shoulder motion occurs at two major sites — the

glenohumeral joint and between the scapula and thorax. The acromioclavicular and sternoclavicular joints permit scapulothoracic motion. When an arm is abducted, motion takes place at the glenohumeral joint and then in the scapulothoracic area. The joint participation in movement can be simplified by stating that abduction, up to 60 degrees, is a glenohumeral function. Past this point the movement is increasingly scapulothoracic. When a frozen shoulder is at its worst there is restriction of both motions.

The next terms are rest pain and motion pain. We believe rest pain in this condition indicates an inflamed nerve segment, the result of an entrapment neuropathy. Although motion will cause mechanical irritation of the nerve segment and produce pain, immobilization does not relieve this pain as it will for the pain initiated by inflamed ligament, tendon or joint structures. These can cause motion pain. In the active phase of a frozen shoulder, both types of pain are present.

The difficulty in defining and treating the condition is a reflection of the lack of appreciation that two entities are present, one concerned with rest pain associated with alteration in scapulothoracic motion, and the other concerned with motion pain and associated with restriction of glenohumeral movement. Either entity may vary in intensity or be in a different position with relation to its time curve. The two elements that compose a frozen shoulder are presented below.

Scapulothoracic Restriction—Rest Pain

It has been demonstrated that rest pain in this condition is associated with an entrapment neuropathy of the suprascapular nerve as it passes through the suprascapular foramen[1, 2]. The suprascapular nerve is derived from the upper trunk of the brachial plexus. It runs downward and backward to reach the posterior aspect of the scapula by passing through the suprascapular notch. The notch is converted into a foramen by the superior transverse scapular ligament. The suprascapular nerve is a motor nerve, that is, it has no skin representation and carries only deep tissue sensation. It innervates the supraspinatus and infraspinatus muscles and portions of the shoulder and acromioclavicular joints. Pain resulting from irritation of this nerve is deep, and poorly localized. It is greatest at the posterolateral aspect of the shoulder.

The edges of the foramen are sharp and unyielding, compared to the nerve trunk. The margin for passage of the nerve is not large.

Motion of the scapula, carrying the foramen, may be likened to moving a ring along a slack string which represents the nerve. At the extremes of motion the nerve is taut. One particular motion renders the nerve taut and kinks it over a foramen edge—that is, cross-body adduction of the humerus with forward and medial protusion of the scapula. If the nerve is inflamed, this motion will increase the process by mechanical irritation.

If there is restriction of glenohumeral motion, forcing the desired range of arm motion will demand a greater excursion of the scapula. This increases the possibility of mechanical irritation of the suprascapular nerve by the foramen edges.

Once a neuropathy has occurred, either by a single severe trauma or repeated small injuries, there will be restriction of scapulothoracic motion on a voluntary basis. This restriction will disappear under anaesthesia, general or local. A block with a local anaesthetic will abolish pain arising from the neuropathy area and from the more distal areas of the nerve supply. Infiltration of the nerve segment with hydrocortisone will relieve only the pain caused by nerve inflammation, as hydrocortisone has no appreciable local anaesthetic property. These two substances offer a means of elucidation of the origin of pain.

Figure 2 shows the influence of scapular motion, in this case cross-body adduction, on the tension and course of the suprascapular nerve.

Glenohumeral Restriction—Motion Pain

Normal motion at joints requires two things—a stable hinge mechanism at which the motion occurs, and motor forces. The greater majority of joints require more than the joint capsule and associated ligaments for the hinge stability or fulcrum establishment. In the shoulder, as in most large joints, stability is also insured by musculature. Most of the common painful shoulder difficulties are caused by derangement of the muscular and ligamentous apparatus responsible for the retention of the humeral head against the glenoid. Any portion of the rotator cuff insertion or coracoacromial ligament may be the site of an inflammatory process. A non-specific connective tissue inflammatory process occurs in the shoulder which may or may not be associated with ectopic calcification. The same process occurs elsewhere in the musculoskeletal system at various tendon or ligament insertions with much less frequency. In addition, an almost asymptomatic calcification can occur

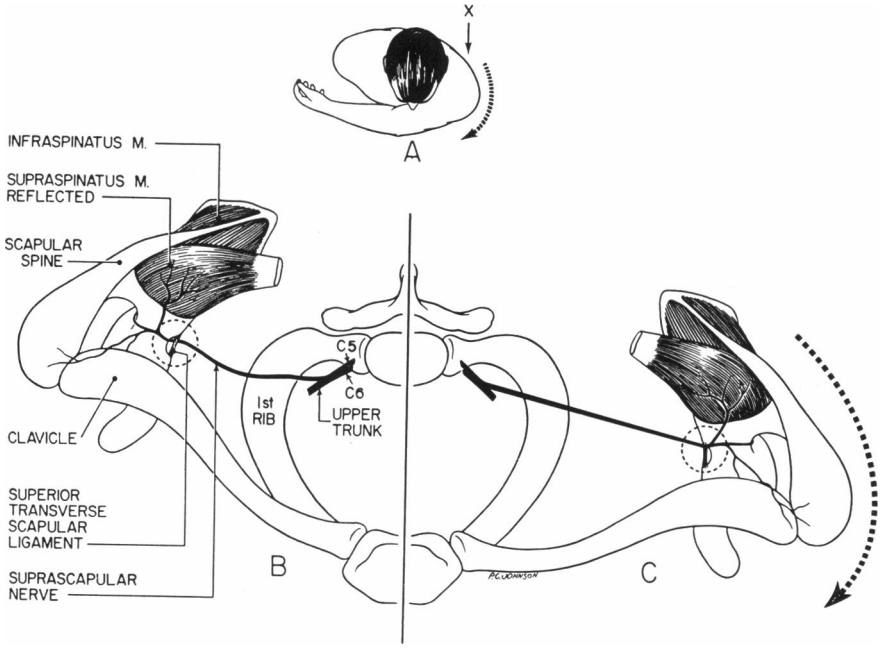

Fig. 2—Coronal view of relationship of suprascapular nerve. B is normal anatomical position; C depicts nerve tension produced by arm posture as shown by arrow in A. (Figures 1 and 2 reproduced by permission of *Surg. Gynec. Obstet. 109*:92-96, 1959.)

concomitantly with degeneration of the intrinsic tendon structure, brought about by aging and wear.

The acute phase of this inflammatory condition, generally called a bursitis, which responds well to anti-inflammatory medication and immobilization, is of no concern here. It is the possible residuals of the acute inflammatory reaction, or of trauma both direct and indirect, that are important in causing limitation of glenohumeral motion.

We have been impressed by the importance of the coracoacromial ligament[3] in causing restriction of glenohumeral motion in the majority of the disabled shoulders. A point of tenderness immediately lateral to the coracoid process is a consistent finding in most frozen shoulders which still show impaired function. This is the area of attachment of the coracoacromial ligament. The shape and character of this ligament is usually not well depicted in texts. It is a tough band which stretches from the coracoid process to the tip of the acromion. In life and in cadaver specimens, the band is bulged upward by the humeral head

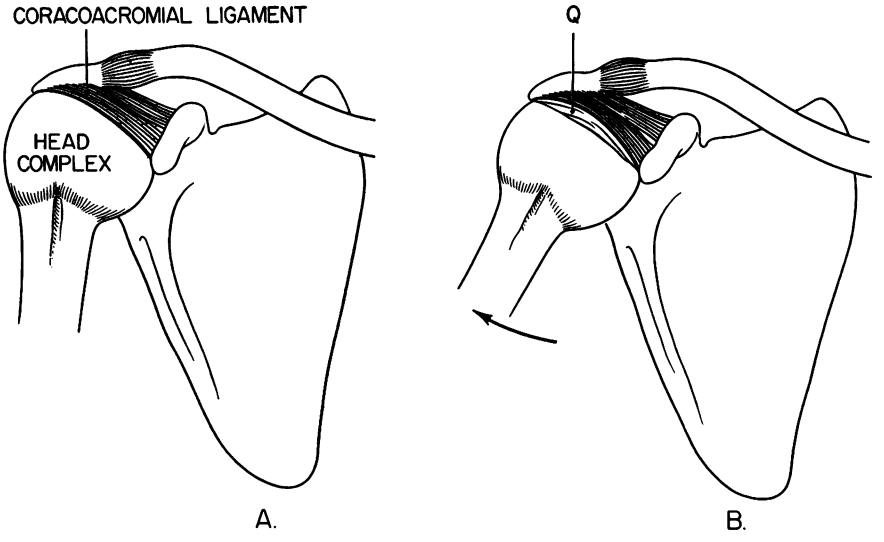

Fig. 3—Diagram of coracoacromial ligament and head complex (head complex—tendons of supraspinatus, infraspinatus, and teres minor covering and blending into capsule).

A. Normal anatomical position.

B. Impingement of head complex against ligament (Q) on motion.

with its covering of capsule and rotator cuff insertions (supraspinatus, infraspinatus and teres minor). One gets the impression that if the humeral head were removed, the ligament would subtend the acromial arch in a straight line, the chord of a circle. The compound curve, convex superiorly and anteriorly, which it assumes *in situ*, is not inherent in the ligament but is, as noted above, a function of head pressure. The head complex must slip against this ligament. Any loss of the ability of the head complex to slip by the ligament will lead to wrinkling of the underlying soft tissues.

It is easy to traumatize the tissues intervening between the humeral head and the ligament by an upward, medial or forward directed force. Any irregularities or thickening of the immediately underlying rotator cuff will impinge on the ligament. The resulting ligament irritation can lead to inflammation which will have a marked binding effect on motions of the humeral head, with subsequent secondary contracture. Early in the course of our investigation, we thought that the point of tenderness might be attributable to an inflammation of the coracohumeral ligament or a tenosynovitis of the biceps tendon. However,

subsequent dissections failed to substantiate a significant motion-blocking role to either of these two structures. Nor can we ascribe significance to the terms often used to describe this condition, i.e., pericapsulitis, or adhesive peri-arthritis, as primary effects.

Mechanical irritation of the ligament by movement of the humeral head with its overlying structures will cause motion pain. The innervation of the ligament is by the medial anterior thoracic and musculocutaneous nerves. This accounts for some of the referred pain pattern found in a frozen shoulder.

Figure 3 shows the relationship of the coracoacromial ligament to the humeral head complex.

Discussion

The basis for the earlier statement that this type of joint involvement is peculiar to the shoulder resides in the mechanical fallibility of two extra-articular factors. One factor is the derangement of glenohumeral motion, most commonly brought about by mechanical interference of the gliding of the humeral head complex under the coracoacromial ligament. The other factor is the vulnerability to trauma of the suprascapular nerve by the edges of its foramen, because of abnormal motion of the scapula.

While some cases of frozen shoulder arrive full-blown, without a good clue as to their origin or development, in most of them a careful history discloses either the initiation or the relationship of the factors. These follow several patterns:

A. Trauma to the shoulder which causes the scapula and humeral head to be driven forward, upward and medially. This force direction can cause trauma to both the suprascapular nerve and the ligament by jamming the rotator cuff between the humeral head and coracoacromial ligament. The injury may result from force applied directly to the shoulder, or from a distance. An example of the latter is the shoulder strain that results from the follow-through of the upper arm at the time a Colles' fracture occurs.

In this group there is the concurrent nerve and ligament lesion which can progress further through the entire course of a frozen shoulder.

B. Residuals of an inflammatory process, with or without calcification, or residuals of trauma to tendons or bone which cause restriction of glenohumeral motion, chiefly through impingement of the distorted structures on the coracoacromial ligament. As mentioned earlier, though other structural causes may be at fault, we believe this mechanism, implicating the coracoacromial ligament, is responsible in the majority of cases. The restricted glenohumeral motion forces a wider range of scapulothoracic motion to achieve desired arm movement. The increased scapular motion can bring about mechanical irritation of the suprascapular nerve, which will result in an entrapment neuropathy. The compensatory scapular excursion may come about through the individual's voluntary actions or even through external manipulation for therapeutic reasons.

C. There is a group that have sustained an entrapment neuropathy of the suprascapular nerve with little if any restriction of glenohumeral motion. This may come about through occult trauma. In this latter group are the effects of abnormal posture, as for example, the position assumed by the shoulder girdle in a hemiplegia. One shoulder girdle motion has particular significance for trauma to the nerve by the edges of the suprascapular foramen. The motion is lateral and forward displacement of the scapula. This occurs in cross-body adduction of the arm. A good example of this is the position of the left arm at the beginning of a golf drive.

Having seen the relationship that exists between the factors and the curve in Figure 1, we can account for the difficulty of evaluating previous treatment programs. Most previous treatments have had as a basis the notion that an increase in glenohumeral motion will decrease pain, and they have attempted to accomplish this by some type of manipulation, surgical lysis, or local applications of energy forms. It will be seen that in form A a lysing procedure will produce relief at point Q (Figure 1) when the entrapment neuropathy has run its course. A great many entrapment neuropathies become relatively asymptomatic in about twelve months.

In form B, lysing procedures (glenohumeral) will be effective if done before compensatory scapular motion has caused an entrapment

phenomenon incapable of spontaneous resolution. If glenohumeral lysis is successful, but the entrapment neuropathy is still active, rest pain and protective inhibition of scapular motion will continue.

In form C, which is basically a pure entrapment neuropathy, treatment, to be of value, must relieve the neuropathy.

Conclusion

The frozen shoulder is a syndrome which can be defined by the clinical features noted at the beginning of this paper. The chief elements are pain, both at rest and on movement, and restriction of motion. Both glenohumeral and scapulothoracic motions are limited. Rest pain is associated with alteration of scapulothoracic motion; and movement pain with restriction of glenohumeral motion. These two factors, which are combined in the frozen shoulder syndome, may exist in differing proportions and in varying degrees because of time relationships. This fact, of two separate variables composing one condition, each having its own rate or progress, has led to difficulties in defining the condition and evaluation of the various forms of treatment.

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