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The Effect of Posterior Versus Anterior Glide Joint Mobilization on External Rotation Range of Motion of Patients with Shoulder Adhesive Capsulitis

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THE EFFECT OF POSTERIOR VERSUS ANTERIOR GLIDE JOINT MOBILIZATION ON EXTERNAL ROTATION RANGE OF MOTION OF PATIENTS WITH SHOULDER ADHESIVE CAPSULITIS.

By

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Each person whose signature appears below certifies that this publishable paper, in his or her opinion, is adequate in scope and quality as a publishable paper in lieu of a thesis for the degree Doctor of Physical Therapy Science.

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ABSTRACT

THE EFFECT OF POSTERIOR VERSUS ANTERIOR GLIDE JOINT MOBILIZATION ON EXTERNAL ROTATION RANGE OF MOTION OF PATIENTS WITH SHOULDER ADHESIVE CAPSULITIS

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Study Design: Repeated measures, randomized comparison of two treatment groups.

Objective: To compare effectiveness of two manual therapy procedures, posterior and anterior glide joint mobilization, for improving external rotation in patients with adhesive capsulitis of the shoulder.

Background: Joint mobilization procedures are often used by physical therapists to treat the accessory motion impairments associated with shoulder adhesive capsulitis. However opinions differ regarding the value of posterior versus anterior glide mobilization, especially for improving external rotation.

Methods and Measures: Subjects with idiopathic adhesive capsulitis exhibiting a specific external rotation range deficit, were randomly assigned to one of two treatment groups. For six sessions, all subjects received ultrasound, joint mobilization, and upper body ergometer exercise. Subjects in Group 1 were treated with posterior glide mobilizations and subjects in Group 2 were treated with anterior glide mobilizations. External rotation range was measured initially and after each treatment session and compared within and between groups. Data were analyzed using repeated measures factorial ANOVA and independent t-tests.

Results: Following six treatment sessions, subjects who received posterior glide mobilizations showed a statistically significant improvement ($p < .001$) in external rotation range of 31.2° whereas subjects receiving anterior glide mobilizations showed only a mean improvement of 3.0° ($p = .68$).

Conclusions: Posterior glide joint mobilization was significantly better than anterior glide joint mobilization for increasing external rotation range in just six treatment sessions for subjects with idiopathic adhesive capsulitis with a specific ROM deficit.

Primary adhesive capsulitis and frozen shoulder are current terms used to describe an insidious onset of painful stiffness of the glenohumeral joint.[1] This condition affects from 2% - 3% of the general population and is the main cause of shoulder pain and dysfunction in individuals aged 40 - 70 years.[2] The range of motion (ROM) impairments associated with this condition can impact a patient's function and the ability to participate in self-care, occupational, or leisure activities.[3] Even though this condition is considered self-limiting, with most patients having spontaneous resolution within three years,[4,5,6] some patients can suffer long-term pain and restricted shoulder motion beyond three years.[7,8]

Most authorities agree that adhesive capsulitis is caused by inflammation of the joint capsule and synovium that eventually results in the formation of capsular contractures.[1,9] Observations during arthroscopic surgery show that the main sites of the contractures are in the anterior capsule, rotator interval, and coracohumeral ligament,[10,11,12,13,14,16] however, any part of the capsule and its supporting ligaments can be affected.[9,17] The capsule does not become adhered to the humerus, as the term "adhesive" implies, but the contracted capsule holds the humeral head tightly against the glenoid fossa.[14,16] Clinically, there is global loss of both active and passive range of motion of the glenohumeral joint with external rotation usually being the most restricted physiologic motion.[2,4,8]

Currently, no standard medical, surgical, or therapy regimen is universally accepted as the most efficacious treatment for restoring motion in patients with shoulder adhesive capsulitis.[18] Manual therapy is commonly prescribed for this condition and there is evidence that joint mobilization can lessen the associated glenohumeral mobility restrictions.[2,16,19] The most efficacious type of joint mobilization procedure to restore external rotation however, is not clear in the literature. Physical therapy textbooks recommend the use of anterior glide joint mobilization to improve external rotation ROM, based on the concave-convex theory.[20,21] In contrast, Roubal et al and Placzek et al used posterior glide manipulation, based on the capsular constraint mechanism,[22] to restore external rotation ROM.[23,24] The purpose of this study was to compare the effects of posterior versus anterior glide joint mobilization for improving external rotation ROM of patients with idiopathic adhesive capsulitis of the shoulder with a specific ROM deficit.[25]

METHODS

Subjects

Patients who were referred by their physician for evaluation and treatment to an outpatient physical therapy clinic were invited to participate in the study if they met the inclusion / exclusion criteria below. All subjects signed the California Experimental Subject's Bill of Rights, the Institutional Review Board Authorization for Use of Protected Health Information (PHI) Form and a consent form, which were approved by the Institutional Review Boards of Loma Linda University and Beaver Medical Group.

Inclusion criteria: 1) diagnosis of idiopathic or primary adhesive capsulitis (i.e. insidious onset) in one shoulder, 2) age between 25-80 years, 3) normal findings on radiographs within the previous 12 months, 4) no previous shoulder surgeries to the affected shoulder, 5) no previous manipulations under anaesthesia of the affected shoulder, 6) glenohumeral external rotation ROM deficits that could primarily be attributed to glenohumeral capsular restrictions. Patients were excluded from the study if they had: 1) a history of significant trauma to the affected shoulder, 2) glenohumeral external rotation ROM that could primarily be attributed to muscle flexibility deficits, 3) shoulder girdle motor control deficits associated with neurological disorders (e.g. stroke, Parkinson's disease).

In this study, a patient's glenohumeral external rotation ROM deficit was primarily attributed to a glenohumeral capsular restriction if the range deficit of glenohumeral external rotation was greater as the shoulder was abducted.[25] Glenohumeral external rotation ROM deficit was attributed to muscle flexibility deficit (e.g.

subscapularis flexibility deficit) if the range deficit of glenohumeral external rotation was lessened as the shoulder was abducted.[25]

Subjects were randomly assigned to either treatment Group 1 (posterior glide group) or Group 2 (anterior glide group) using a using a random numbers table. Folders labeled with the group number and subject number were made up ahead of time and used sequentially as the subjects joined the study. Upon entering the study, each subject was given a handout with instruction to use their involved arm in pain-free activities of daily living. Activities that involved resisted motions (e.g. pushing, pulling, closing the car door, opening stiff doors, gardening, vacuuming, sawing) or lifting objects that weighed more than five pounds were discouraged. No home exercise program was given.[15]

Tests and Measures

Before the first treatment, each subject marked a visual analogue scale for pain[26] and completed five questions regarding how his/her shoulder pain and impairment affected sleep, general daily activities, and specific tasks (dressing, grooming and reach) that usually require shoulder external rotation range.[27] These measures were used to determine how similar the groups were at the beginning of the study.

Glenohumeral active external rotation ROM was chosen for the outcome measure and was measured at baseline and after each of six treatment sessions. The primary researcher (AJ), a physical therapist with 29 years of clinical experience, took all the measurements. Figure 1 demonstrates the external rotation measurement procedure. During the first measurement for each subject, the glenohumeral joint was passively taken into end range of abduction in the coronal plane before externally rotating the humerus.[19,28] This abduction angle measured was recorded in order to place the glenohumeral joint in the same amount of abduction prior to the external rotation ROM measurement in each subsequent measurement.

To obtain a consistent abduction range, an adjustable wall goniometer (Sammons Preston Rolyan, P.O. Box 5071, Bolingbrook, IL 60440-5071) was adapted by cutting it in half, then mounting it, with clear tape, to ¼ inch thick high-density pressboard. One half of the board goniometer was used for measuring right shoulders, and the other half was used for measuring left shoulders. The board goniometer was placed on a moveable table and was carefully positioned parallel to the sternum with its axis under the glenohumeral joint.

External rotation measurement was taken with a standard 12" goniometer.[30] The goniometer was adapted by cutting off the range from 180° to 360° so that the stabilizing arm lay flat against the surface of the board goniometer as shown in Figure 1. To prevent measurement bias, the back of the goniometer scale was covered with white adhesive paper before the study.[29] Two goniometers were made – one for measuring right shoulders and one for measuring left shoulders.

With the stationary arm of the adapted goniometer flat against the board goniometer, the moving arm was lined up with the shaft of the ulna. During the measurement, motion in the trunk was prevented and the humerus was not allowed to lift off the table. There was no external stretching force applied to facilitate humeral external rotation. The end position was determined by the subject as the researcher gave a verbal cue to actively take the humerus backwards as far as he/she could, then relax.

Figure 1. External rotation measurement technique.

Before the study commenced, the researcher underwent training for competency for the external rotation measurement technique. This was done by measuring subjects with glenohumeral restrictions. Competency was presumed when repeated measures were within three degrees.[30] After this, a reliability study commenced for both the abduction measurement (16 shoulders) and the external rotation measurement (20 shoulders) in shoulders with adhesive capsulitis or other shoulder diagnoses with joint restrictions similar to adhesive capsulitis. The ICC for the abduction measurement and the external rotation measurement were .989 and .998 respectively.

Intervention

Each subject had six treatment sessions that consisted of ultrasound at 1.5 W/cm^2, continuous for 10 minutes,[31,32,33,34] joint mobilization (either posterior glide or anterior glide procedures) for 15 minutes, and exercise on an upper body ergometer (Tru-kinetics Upper Cycle from Tru-Trac Therapy Products) for three minutes.

The joint mobilization procedures utilized in this study were "stretch mobilizations" which are similar to those described by Kaltenborn.[21] Each mobilization was held for at least one minute at the limit of the available abduction and external rotation range.[35] Each subject received a total of 15 minutes of stretch during each treatment session.[33] During the performance of the joint mobilization techniques, the subject was expected to feel a sensation of moderate stretch. Subjects were instructed to describe his/her sensation during the performance of the procedures, so that the researcher could modify the force or position as necessary.

Group 1 received lateral glide with posterior glide stretch mobilizations and Group 2 received lateral glide with anterior glide stretch mobilizations. Figure 2a shows the position for the initial technique for the posterior glide mobilizations. Figure 2b shows the position used when the subject progressed in range and tolerance to the technique. The researcher first performed a lateral glide, to reduce the joint compressive forces of the mobilization,[24] then a posterior glide stretch mobilization was performed.

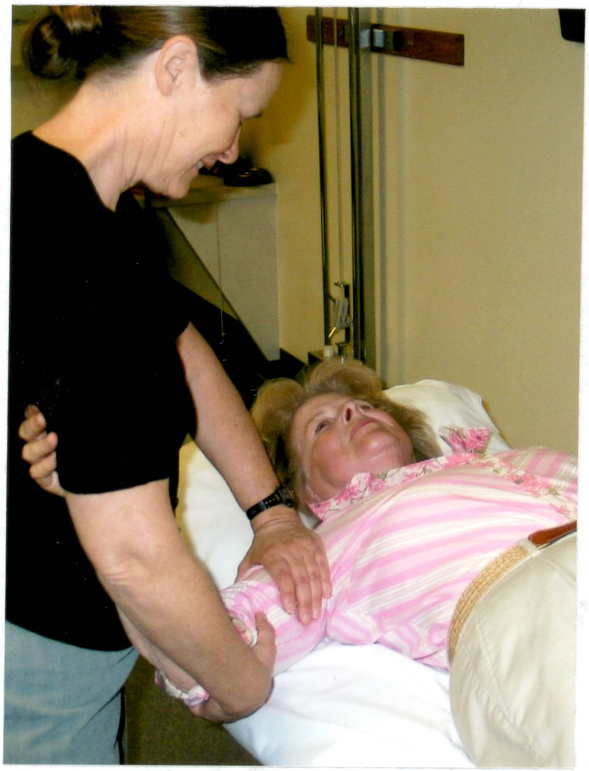

Figure 2a. Initial posterior glide mobilization technique.

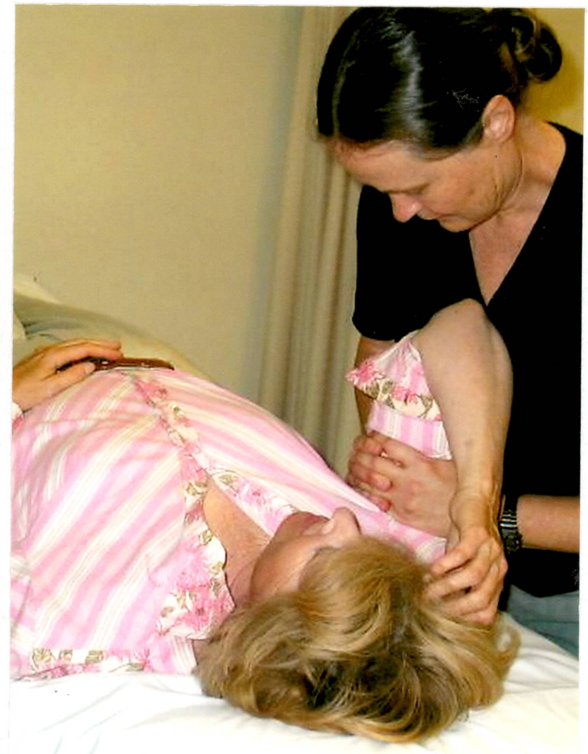

Figure 2b. Progression of the posterior glide mobilization technique.

Figure 3a shows the initial position for the anterior glide mobilization. Figure 3b shows the position used for the anterior glide mobilization when the patient progressed in range and tolerance to the technique. Again the researcher first performed a lateral glide then an anterior glide stretch mobilization.

Upon completion of the joint mobilization procedures at each of the six treatment sessions, external rotation measurements were taken. The subjects then exercised by using the upper body ergometer in the forward direction only.

The frequency of the treatment sessions was two to three times a week for a total of six sessions. The short duration of the study was designed to optimally assess the changes attributable to the mobilization techniques and to minimize the changes due to the natural history of adhesive capsulitis.

Figure 3a. Initial anterior glide mobilization technique.

Figure 3b. Progression of the anterior glide mobilization technique.

Data Analysis

To determine how similar the groups were at the beginning of the study, subject gender, occupation, dominant arm, affected arm, and joint end-feel at baseline were compared between groups using frequencies and Chi-square tests whereas age, height, weight, duration of symptoms, passive abduction range, external rotation range and VAS pain scores were analysed with independent t-tests for equality of means between groups. The function questions one and two were scored from 0 to 4 (very severe to no difficulty) and analysed and compared between groups using Chi-square tests and questions three through five used combined scores and were analysed and compared between groups using the Mann-Whitney U test.

External rotation range was measured initially and at the end of each of the six treatment sessions and compared by group using a repeated measures factorial ANOVA. Because there was an interaction between group and time, an independent t-test was performed between groups for each time interval and a repeated measures ANOVA was performed in order to assess the difference in the measured variable over the six treatment sessions for each group separately. Alpha was set at 0.01. Intra-class correlation coefficients were calculated to describe the degree of intra-rater reliability of the abduction and external rotation measurements.

RESULTS

Subjects

From October 2003 to January 2005, 58 patients with the diagnosis of adhesive capsulitis or frozen shoulder, were evaluated by the primary researcher. Thirty-eight were eliminated as they did not meet inclusion / exclusion criteria. Subjects were excluded for the following reasons: 13 had abnormal radiographs, one presented with proximal humerus fracture, two had rotator cuff tears as diagnosed by arthrograms, two had neurological conditions i.e. cerebral palsy and stroke, one had a diagnosis of tendonitis, three presented with having a manipulation under anaesthetic and two were planning on manipulation under anaesthetic before six visits, 14 had an external rotation range deficit primarily associated with muscular shortening, and two did not speak English well enough to understand the consent forms.

Twenty patients (four men and 16 women), between the ages of 37 and 66 years, met the inclusion/exclusion criteria and joined this study. Two subjects had had open-heart surgery within the previous six months. No subjects had diabetes, thyroid, or cervical problems. None of the subjects had received previous physical therapy for their shoulder. All subjects were still under medical care for their shoulder condition, which usually included anti-inflammatory medication. No subject had steroid injections, while participating in the study. One subject left the study because she received arthroscopic surgery with manipulation under anaesthetic. Another subject left the study because of a fall. Both the subjects that left the study were in Group 1. Data from these subjects were omitted from the analyses. Demographic data for the remaining 18 subjects is presented in Tables 1 and 2. The groups were statistically similar initially, except for the side of the affected arm.

TABLE 1. Comparison of subject characteristics at baseline by group.

Subject Characteristics	**Group 1** (n=8) Mean ± SD	**Group 2** (n=10) Mean ± SD	*p* value
Age (years)	50.4 ± 6.9	54.7 ± 8.0	.40*
Height (cnetimeters)	668.7 ± 4.0	166.9 ± 4.0	.74*
Weight (kilograms)	388.5 ± 119.2	348.5 ± 93.7	.44*
Symptom Duration (months)	13.5 ± 19.0	5.8 ± 3.3	.22*
Gender	6 females, 2 males	8 females, 2 males	.80 †
Dominant arm	8 right	10 right	a. †
Affected arm	3 right, 5 left	9 right,1 left	.006†
Occupation	6 sedentary, 2 manual	6 sedentary, 4 manual	.64 †
Injury recalled	3 yes, 5 no	3 yes, 7 no	.64 †
Joint end-feel	2 soft, 6 firm	4 soft, 6 firm	.80 †

SD = Standard Deviation
* Independent t test
† Chi square test
a. no statistics computed because dominant is a constant

TABLE 2. Disease specific characteristics of subjects by group.

Condition Characteristics	Group 1 (n = 8) Mean ± SD	Group 2 (n =10) Mean ± SD	*p* value
Passive abduction range (degrees)	51.3 ± 10.8	55.0 ± 9.4	.63*
External Rotation (degrees)	1.3 ± 16.8	11.1 ± 15.5	.21*
VAS pain scale (centimeters)	7.4 ± 2.3	8.3 ± 2.9	.48*
Function Q1 §	3.9 ± 0.3	3.7 ± 0.7	.65†
Function Q2 §	2.7 ± 0.9	2.3 ± 0.7	.70†
Function Q3 - 5‖	8.9 ± 1.9	8.7 ± 2.1	.90‡

SD = Standard Deviation
*Independent t test
†Chi-square test
‡Mann-Whitney U
§ Q1 and Q2 (scores 0 - 4, where 0 = no limitation/never, 4 = severe/unable/daily)
‖ Q 3-5 (scores as above are summed, 0 - 12)

External rotation range of motion

The comparison of external rotation ROM at baseline and after each treatment, by group, is presented in Table 3 and Figure 4. At the end of the six treatment sessions, Group 1 showed a statistically significant ($p < .001$) mean improvement of 31.2°, whereas, Group 2 showed a mean improvement of only 3.0° ($p = .68$). These results indicate that posterior glide mobilizations of the humeral head on the glenoid result in a significant improvement in external rotation range as compared with anterior glide mobilizations, over the span of six treatment sessions.

TABLE 3. Comparison of external rotation of motion (degrees) at baseline and after each treatment by group.

Treatment Sessions	**Group 1** (n = 8) Mean ± SD	**Group 2** (n = 10) Mean ± SD	*p* value*
0	1.3 ± 16.8	11.1 ± 15.5	.21
1	10.1 ± 18.7	16.2 ± 20.5	.53
2	16.1 ± 21.3	16.4 ± 14.6	.98
3	22.5 ± 22.0	18.7 ± 17.2	.69
4	24.9 ± 20.1	13.2 ± 17.8	.21
5	26.6 ± 22.9	14.8 ± 14.1	.23
6	32.5 ± 20.7	14.1 ± 13.0	.04
p value†	<.001	.68	

SD = Standard Deviation
* Independent t-test
† Repeated measures ANOVA

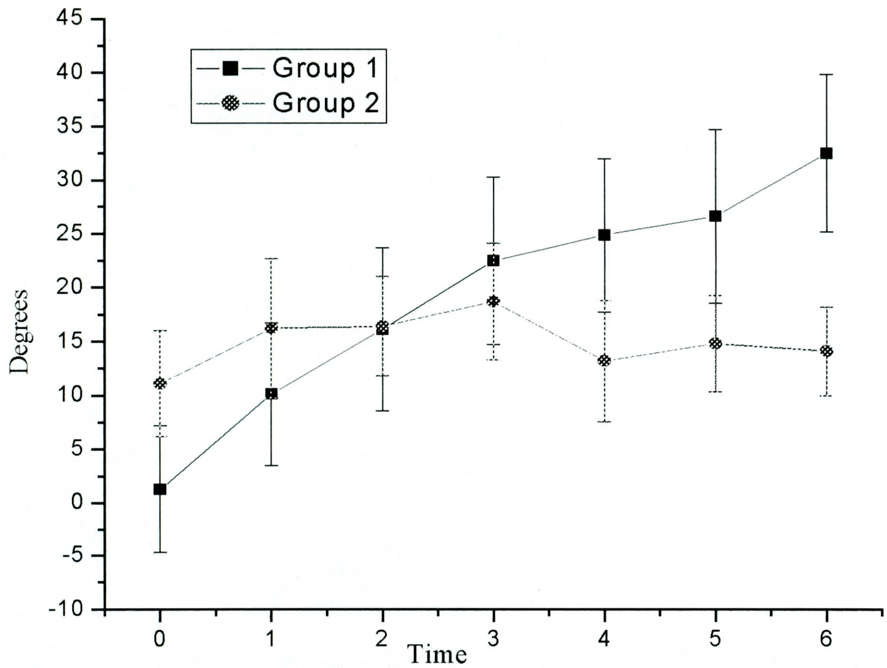

FIGURE 4. Comparison of external rotation at baseline and after each treatment by group (Mean +/-SEM).

DISCUSSION

Full elevation of the normal shoulder requires external rotation of the humeral head to clear its tuberosity from abutting beneath the acromion and thus impinging the tissues in the subacromial space.[36] This external rotation of the humeral head has been associated with a posterior instantaneous center of rotation and posterior translation with reference to the glenoid cavity.[22,,37,38,39] In adhesive capsulitis, contractures develop which limit this posterior translational motion and may produce abnormal obligatory translations in the opposite (i.e. anterior) direction.[40] It is also common with these patients to palpate the humeral head displaced anteriorly.[24,38] Posterior glide mobilization may both, center the head of the humerus in the glenoid cavity as well as restore the normal posterior translational motion associated with normal external rotation, which in turn also restores external rotation range of motion.

The results of this study indicate that posterior glide mobilizations are effective in treating the external rotation range deficit commonly found in patients with adhesive capsulitis and support the findings of Roubal et al and Placzek et el who report marked increases in external rotation with a posterior gliding manipulation.[23,24]

Other studies[4,41,42] however, have found that intensive joint mobilization techniques have been ineffective for treating patients with adhesive capsulitis. It may be that the subjects in these studies were treated with the traditional anterior glide techniques, which this study also found to be ineffective for improving external rotation range for patients with adhesive capsulitis.

A limitation of this study was small group sizes (Group 1 = 8, Group 2 = 10). The groups, however, were found initially to be similar, due to randomization. Because of this, the statistically significant difference found in external rotation range between groups at the end of the study could be reasonably attributable to the difference in the treatment received. A larger sample, however, would have improved this study's ability to generalize the results to all patients with primary adhesive capsulitis. Although this study used a convenience sample from one outpatient physical therapy clinic, this setting is usually where these patients seek treatment, so it is reasonable to presume that this sample was typical of the population of patients with adhesive capsulitis.

Another limitation of this study was not being able to blind the measurer to the treatment group, which could have potentially introduced bias in the range of motion measurements. To counter this, the researcher took steps beforehand and during the study to minimize this potential bias, such as covering the back of goniometer with white adhesive paper so that the numbers could not be seen by the researcher when measurements were taken, and also taking the goniometer to full 180° before each measurement. The subjects, however, were blind to the treatment group.

In this study, the mobilizations were conducted for a total of 15 minutes during each treatment session, with the intention of elongating the glenohumeral capsular contracture in patients with adhesive capsulitis.[15] As substantial improvements were made in just six visits, over a two to three week period, a component of the improvement in external rotation ROM may be associated with centering the humeral head in the glenoid fossa and restoring the normal posterior translational motion that was described by Harryman et al.[22] If this is the case, then posterior glide stretch-mobilizations of shorter duration may be sufficient. Further studies could be designed to find the optimal duration of the stretch-mobilizations for improving glenohumeral range of motion.

The ROM improvement associated with the posterior glide mobilization may indicate that the subjects selected for this study may represent a subgroup of adhesive capsulitis.[39,40] It is not known if posterior glide mobilization would be effective with other subgroups, e.g. stroke, fractured humerus, acromial clavicular problems, rotator cuff tears as well as secondary adhesive capsulitis caused by immobility after trauma or surgery. Future studies focused on effectiveness of posterior glide mobilization for these other subgroups of adhesive capsulitis are recommended.

During the long course of shoulder pain and stiffness associated with the diagnosis of adhesive capsulitis, muscle flexibility deficits may also contribute to the restricted range. It is noteworthy that 14 of the patients who were referred for physical therapy with a diagnosis of adhesive capsulitis actually had limitations that the authors believe fit the primary diagnosis of subscapularis muscle flexibility deficit deficit. Including these 14 patients in the study may have resulted in less dramatic improvements in shoulder ROM with the posteriorly directed mobilization. The investigators of this study suggest that patients with adhesive capsulitis be carefully assessed for the source of the external rotation range deficit and that treatment be selected to address the specific deficit.

For example, if glenohumeral external rotation ROM becomes greater as the shoulder is abducted, impairments in muscle flexibility are likely and interventions, such as soft tissue mobilization and muscle stretching procedures should be selected to normalize the muscle flexibility deficits.[25] On the other hand, if glenohumeral external rotation ROM becomes less as the shoulder is abducted – indicating capsular involvement – then treatment should be focused on the capsular associated mobility impairments with the use of posterior glide mobilization. A multi-group, clinical trial where subjects with presumed muscle flexibility and capsular impairments are randomly placed in intervention groups that receive either soft tissue mobilization/muscle stretching procedures or posterior glide mobilization procedures would verify whether it is beneficial for interventions to be selected to address the primary impairment found during the physical examination.

CONCLUSION

Posteriorly directed mobilization procedures are substantially more effective than anteriorly directed mobilization procedures for treating glenohumeral external rotation ROM impairments in patients with idiopathic or primary adhesive capsulitis.

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APPENDIX 1

LITERATURE REVIEW

ADHESIVE CAPSULITIS: CONSIDERATIONS FOR JOINT MOBILIZATION

The normal shoulder has anatomic pecularities that makes its mechanics more complicated than other joints. For full elevation of the shoulder, external rotation of the humerus is necessary to prevent impinging the tissue between the humeral tuberosity and the acromion.[1] Rotational movement of the humeral head has been found to be associated with translations[2,3] due to the marked discrepancy between the shallow glenoid fossa and the large hemispherical humeral head, which provides little bony stability.[4]

Translational movements also occur because the normal capsule, which surrounds the glenohumeral joint, is a loose, thin-walled structure with a surface area nearly twice as large as that of the humeral head and allows about 2 centimeters of joint distraction.[5] The glenohumeral ligaments that reinforce and strengthen the capsule, which are actually thickenings in the capsule,[6,7,8] are elastic and also allow about 2 centimeters of separation between humeral head and glenoid fossa[8] and thus also permit translational movement.[9] The role of the ligaments therefore, is to limit excessive amounts of movement.[10] The anterior capsuloligamentous component limits external rotation and the posterior component tends to limit internal rotation.[11] In glenohumeral joints with an intact capsule, during external rotation, the instantaneous center of rotation becomes positioned more toward the posterior component[12] and the humeral head translates posteriorly.[2,13]

So as the arm moves, the humeral head initially translates across the glenoid surface in the direction opposite to the motion of the humerus, due to the joint's geometry.[3] When the capsule begins to tighten, however, it becomes a constraint to glenohumeral motion, causing the humeral head motion to reverse.[3] This capsular restraining force acts to re-center the humeral head on the glenoid surface.[3] Thus the tension in capsular tissues,[13] rather than joint surface geometry, controls the translatory movements of the humeral head.[3,14] So, due to this capsular constraint mechanism, external rotation is coupled with posterior translation, especially towards the end of the range.[2]

Capsular tightness has the most potential to impact humeral motion, especially when tension in the capsule increases as the arm is taken further into elevation.[15] It has been found that asymmetrically tightening the capsule caused excessive translation of the humeral head in the opposite direction of the tightness.[2] A tightened posterior capsule caused increases in anterior and superior translation with flexion and the translations occurred earlier in the range of motion than normal.[2] However, a tightened rotator interval caused significant reduction in posterior and inferior translation of the humeral head.[10] Thus, there is a complex interaction among various regions of the capsule, which affect passive motion and translation[16] and is also highly dependent on arm position[17,18]

Muscular contraction introduces yet another effect on humeral head translations. Rotator cuff muscle contraction compresses the humeral head against the glenoid.[8,17] The subscapularis and infraspinatus/teres minor force couple provides stability in the anterior/posterior plane throughout the range of abduction.[19] So muscle contraction has a centering effect on the humeral head[20] and reduces the translational motion during arm elevation,[7,21] but specifically with rotational ranges.[22]

Thus normal shoulder joint motion requires adequate coordination of all passive and active stabilizers to maintain shoulder stability and pathologic changes in any of these can lead to unphysiologic translations of the humeral head relative to the glenoid cavity.[7,17,23] This delicate balance between mobility and stability makes the shoulder vulnerable to injury. Fatigue or imbalance of the rotator cuff muscles has been linked to abnormal translations of the humeral head.[21] Abnormal mechanics caused by muscle weakness, tightness, fatigue, microtraumas or pain[21,24] which may lead to abnormal translations that then may result in inflammation in the capsule. The most vulnerable part of the capsule is the anterior portion.[21] If the muscles supporting the anterior capsule become weak or fatigued, then the anterior capsule becomes stretched during activity. This displaces the humeral head anteriorly and the posterior capsule becomes relatively tight which also displaces the humeral head anteriorly.[24]

In adhesive capsulitis the capsule and its ligaments become inflamed and eventually contract thus severely limiting the normal humeral head translational motion and producing abnormal obligate translations.[10] The biceps long head tendon becomes stenosed in its groove.[25] Other muscles develop atrophy and tightness[26] and also affect the humeral head's ability to translate normally. Furthermore, on examination, adhesive capsulitis usually exhibits an anteriorly displaced humeral head.[27,28] With the humeral head translated and displaced in its anterior-most excursion, posterior glide would potentially be limited, thus may limit external rotation.[28]

Clinically, there is global loss of both active and passive motion of the glenohumeral joint with compensation of early scapular motion.[29] There is no characteristic motion restriction or "capsular pattern" as once thought,[18] but external rotation is usually the most restricted motion.[26,30,31,32] In the acute phase, when the shoulder is passively moved, pain limits motion before reaching the end of the joint range. The clinician feels no resistance to movement, so there is an "empty" end-feel to the motion.[33] In the chronic phase, the patient reports pain as the end of joint range is reached. The clinician feels a leathery or firm block to passive end range.[5,26,28,33,34] Accessory joint mobility testing usually reveals globally limited glenohumeral joint translations.[28]

This loss of glenohumeral joint mobility restricts the ability of the humeral head to externally rotate and glide underneath the acromion when the humerus is elevated. The pain and restricted motion causes the patient to develop protective behaviors and to stop using the joint normally. As the glenohumeral joint remains severely restricted, muscle atrophy develops.[26,35]

Most authorities agree that the glenohumeral joint capsule is the main site of the lesion.[32,36,37] Observations at arthroscopic surgery have found that, in the acute phase of adhesive capsulitis, there is a red and thickened synovium.[25,32] The synovitis has been more frequently observed in the anterior capsule at the site of the rotator interval (See section on Rotator Interval) and the coracohumeral ligament, which spans the rotator interval.[10,25,39] However, any part of the capsule and its supporting ligaments can be affected.[34,40] Other affected structures include subscapular bursa, middle glenohumeral ligament, inferior capsule, axillary pouch, the rotator cuff, the biceps tendon, posterior capsule wall, superior glenohumeral ligaments, subacromial space, and subacromial bursa.[16,21,31,32,34,37,40] In the chronic phase of the disease, the synovitis resolves with subsequent reactive capsular fibrosis,[41] leaving the affected tissue with a contracture. This contracture consists of a dense matrix of Type III collagen with fibroblasts and myofibrobalsts[38] and resembles fibroplasia, similar to that found in Dupuytren's contracture.[31] Arthography indicates that there is loss of capsular extensibility, with reduced capsular capacity to hold synovial fluid,[25,26] loss of the dependent fold, and restricted capsular expansion for shoulder motion.[42]

The capsule does not become adhered to the humerus, as the term "adhesive" implies, but it and the affected ligaments become thick and shortened in the resulting contracture.[37,40] This contracted tissue holds the humeral head tightly against the glenoid fossa,[10,25,32] reducing the space between the humeral head and the acromion,[43] between the long head of the biceps tendon and the humeral head, and between the rotator cuff and the humeral head.[40]

Primary or idiopathic adhesive capsulitis and frozen shoulder is a painful stiffness of the glenohumeral joint without known cause.[27,35,43,44,45] This condition is the main cause of shoulder pain and dysfunction usually affecting individuals aged 40-70 years.[26] The pain is often generalized and hard to localized,[36,45] may radiate both proximally and distally, is aggravated by movement, and alleviated with inactivity.[45] As the joint stiffens, the patient even loses the natural arm swing in gait.[45] The loss of motion can severely impact independence (reaching to scratch one's back, stretching to relax), self care (grooming, toileting, hygiene, dressing), activities of daily living(driving, fastening a seat belt, opening a door, lifting, pushing, hand shaking, hugging, reaching for one's back pocket), and employment (especially with manual laborers, waitresses, store workers, factory workers, and jobs that require repetitive motion or the operation of machinery that vibrates).[45] Patients learn to compensate for lost movement, however this causes overwork and fatigue to other muscles and joints.[45] Shaffer et al found that neither pain or motion restriction was related to age, menopause, dominance, affected side, nature of onset, duration of symptoms, or associated medical conditions.[30] There was a strong association, however, between functional limitations and symptoms.[30] Binder found that dominant arm involvement and manual labor were associated with less satisfactory results with conservative treatment.[43]

This condition is considered self-limiting, because by about three years, most patients will have spontaneous resolution of symptoms and joint restrictions.[31,44,46] Some patients, however, can be symptomatic with long-term pain and restricted shoulder motion for more than three years after onset.[30] A disability of this duration places severe emotional and economic hardship on the afflicted person.[27,46] Most patients are unwilling to suffer this pain, prolonged disability, muscle wasting, sleep deprivation and often depression without seeking treatment.[25,46]

Adhesive capsulitis can be divided into two groups: primary or idiopathic and secondary. Patients with primary or idiopathic adhesive capsulitis have no significant findings in their history (insidious onset), clinical examination, or radiographic evaluation that explains their loss of motion and severe pain. Those with secondary adhesive capsulitis describe an event that preceded shoulder symptoms, such as trauma or surgery to the affected upper extremity.[41]

Classically, symptoms of adhesive capsulitis have been divided into three phases:[41] (1) the painful stage (acute phase), (2) the stiffening phase (chronic), (3) the thawing phase (resolution). In the initial phase, there is a gradual onset of diffuse shoulder pain, usually near the deltoid insertion. The stiffening phase is characterized by a progressive loss of motion. Most patients lose glenohumeral external rotation, internal rotation and abduction range during this phase. The final thawing phase constitutes a period of gradual motion improvement and return of function.[36,41,45]

The etiology of adhesive capsulitis has not been definitely established.[37] It is thought that any condition that contributes to dependency of the arm for an extended period of time can lead to capsular contracture (e.g tendonitis, coronary artery disease, mastectomy, other chest or shoulder surgeries, distal injuries of the extremities, cervical spine radiculopathy).[36] This compares to findings in a dog model study by Schollmeier et al. After immobilizing the dogs' forelimbs, changes occurred that resembled those of may result in the development of subacromial impingement.[47] Everything, however, from autoimmune disease, a virus, and hormones have been suggested as causes of adhesive capsulitis.[41]

Currently no standard conservative or surgical treatment is universally accepted.[48] Most physicians initially recommend conservative treatment of non-steroidal anti-inflammatory medication, oral corticosteroids, or corticosteroid injections into the subacromial space, and physical therapy for 3-6 months.[45,49] Conservative management usually results in most patients gaining satisfactory range of motion and reduction in pain.[26] Some patients, however, are refractory to conservative treatment and are usually considered for other treatment options.

The most common treatment option is manipulation under anaesthetic which achieves its effects by complete rupture of the contracted joint capsule.[37] It has been theorized that the rupture of these structures depends on the relative strengths of the affected tissues and occurs at the weakest point of the joint capsule and not necessarily in the pathological areas of fibrosis that limit the motion.[50] Other surgical options available include arthroscopy as an adjunct to manipulation,[37] arthroscopic release[32,34], or joint capsule distension.[48]

Although physical therapy is usually considered a component of conservative treatment,[37] there is still no consensus on the best protocol for this patient population.[51] Different treatment protocols have been investigated and recommended.[30,46,52] These recommendations have ranged from benign neglect,[44] a supervised self stretching program,[35] a combination of exercise therapy and modalities,[30,53] gentle passive joint mobilizations, followed by stretching and strengthening exercises as pain subsides,[26] and intensive end-range mobilization techniques.[33,42,54] Most of these regimens report reasonable success in 3-6 months as evidenced by increased range of motion, reduced pain, patient satisfaction, and increase in joint capsule capacity as seen by arthrography.[26,42] Many researchers, however, have found that local steroid injections were as effective as physical therapy,[55] Griggs et al reported acceptable results with a simple self-stretching program[35] and Bulgen et al found little long-term advantage in any of the treatment programs that he studied.[46] Carette et al found that adding physical therapy to a program of corticosteroid injections and a simple home exercise program, improved range of motion faster, but when used alone, physical therapy was of limited value compared to placebos.[55]

Most studies, have been hampered by no established standardized definition of adhesive capsulitis, no uniform means of selecting or grouping patients, small sample sizes, or no control / comparison groups. Thus they have suffered from reduced validity, reliability and generalizability of their results.[35]

A recent study using a combination of supervised exercises, pool therapy, daily massages improved flexion, extension, abduction, adduction, and strength, but did not improve external or internal rotation.[53] Thus, joint mobilization, not exercise, seems to be a required component as it attempts to restore the normal translational motions (arthrokinematic motion) needed for the restricted external rotation range of motion (osteokinematic motion) and when external rotation range was improved, it has been found that there was also an improvement in joint space capacity in patients with adhesive capsulitis.[26]

Joint mobilization has become widely used as a manual therapy procedure to increase range of motion in restricted synovial joints.[5,51] It is a form of passive movement used to elongate connective tissue structures that restrict joint motion.[27] Several forms of joint mobilization exist and terminology varies among authorities. Joint mobilization uses a small amplitude passive movement[54,56] to restore translational movement to improve physiological movement.[1] The direction of the mobilizing force is usually toward the connective tissue that is limiting the translational motion, as established in accessory motion testing.[51]

Although joint mobilization is commonly used as a physical therapy treatment for adhesive capsulitis, there are few controlled clinical trials to establish protocols, especially for the direction of the mobilizing force. Mao et al gave no specifics on their mobilization technique except for describing gentle, passive joint mobilization.[26] Vermenlen et al, used intensive end-range mobilization techniques in the inferior, anterior and lateral direction.[42] Nicholson chose lateral glide instead of anterior glide, to restore external rotation with resultant improvement in abduction range.[27] Hsu et al studied normal cadaver shoulders and found no difference between anterior and posterior glide mobilizations for improving abduction range.[9,57] Most current manual therapy regimens are aimed at stretching the anterior capsule contracture using anterior glide techniques, and stretching the inferior capsule and decompressing the subacromial space by using inferior glide techniques.[5,51] Roubal et al and Placzek et al, however, manipulated the joint in an inferior and posterior direction, gaining immediate external rotation and internal rotation range.[28,58]

Most clinicians agree that lateral glide stretches the whole capsule and separates the joint surfaces[51] and that inferior glide is used for subacromial decompression.[59] There seems to be disagreement, however, regarding the best direction of joint mobilization for improving external rotation in patients with adhesive capsulitis.

Joint mobilization is a passive maneuver, so shoulder mechanics under passive conditions may be need to be considered to determine what joint mobilization protocol would be most appropriate. Karduna et al found that joint conformity had a significant influence on translations during active positioning but not during passive positioning and glenohumeral ligament wrap lengths correlated with translations when joints were positioned passively but not actively.[22] So joint conformity appears to play a role in controlling translations during active motions whereas capsular constraints become more important during passive motions.[22] This indicates that the concave-convex rule may only apply during active motion or when the capsule has been excised, and the capsular constrain mechanism seems to be applicable during passive motions and should be applied when using joint mobilization techniques.

The Rotator Interval

The rotator cuff is perforated anterosuperiorly by the coracoid process which separates the supraspinatus tendon from the subscapularis tendon, creating a triangular rotator interval (RI), which is bridged by capsule and the coracohumeral ligament (CHL).[10,60] The CHL originates from the coracoid process. Its fibers interlace with the fibrous capsule and insert with the capsule into both tuberosities of the humerus.[25]

This triangular space contains the biceps tendon and the superior glenohumeral ligament (SGHL). The capsular tissue is relatively weak and normally elastic membranous tissue, which enhances the range of motion of the glenohumeral joint.[6] This elastic quality varies in individuals. Even though the CHL strengthens this part of the capsule, its ability to function in stability is questionable.[6] Inferior translation of the adducted arm is restricted by SGHL and CHL and negative intra-articular pressure. A sealed glenohumeral capsule is necessary to maintain this negative pressure, and a relatively thin capsular covering of RI provides that seal.[6]

Nobuhara classified 2 types of abnormal rotator interval, Type I which was contracted and Type II which demonstrated abnormally laxity.[39] In Type I the RI becomes tight. Mild cases of tightness cause impingement of the rotator cuff. In severe cases of adhesive capsulitis, the RI becomes a thick, fibrotic and contracted cord that holds the humeral head tightly against the glenoid fossa and restricts glenohumeral movement in all directions.[25,39] Even though the RI is not the only area of capsule that is affected in adhesive capsulitis, some believe that RI is of center importance in development of adhesive capsulitis.[6]

APPENDIX 2

MEASUREMENT OF EXTERNAL ROTATION

In adhesive capsulitis of the shoulder there is usually severe restriction of external rotation range. Limitation of this humeral rotation helps to explain the limited elevation seen in adhesive capsulitis. In patients with adhesive capsulitis, it has also been found that there is a high correlation between improvement in external rotation range and improvement in abduction range,[52] increase in joint capacity, reappearance of the axillary recess and smoother capsular margins.[26] Thus external rotation was chosen as the outcome measure for this study.

To measure external rotation, it is important to take the humerus into full available abduction range before externally rotating the humerus as passive external rotation performed prior to placing the humerus at its full available abduction range results in a greater external rotation measurement.[59] The frontal plane was selected for the measurement for a more reliable measurement technique.

To obtain a reliable abduction range for each measurement, a Board Goniometer was designed as shown in Figure 1. A Wall Goniometer was adapted for this purpose. It was purchased from Sammons Preston Rolyan, P.O. Box 5071, Bolingbrook, IL 60440-5071. It comes as a laminated, printed, white cardboard protractor. The sides of the wall goniometer were long enough to support a human humerus. As only 90° of abduction was needed, the wall goniometer was cut in half – one for measuring right shoulders and one for measuring left shoulders – then each piece was mounted with clear tape onto ¼" thick high density pressboard (commonly called masonite). The board goniometer was placed on a movable bolster, that sat atop a table with wheels, which held the Board Goniometer essentially parallel to the ground as it was moved to the subjects side.

Figure 2 demonstrates the external rotation measurement technique. The subject lay supine on a treatment table, with a pillow under the head. The treatment table was hydraulically raised or lowered as needed to keep the board goniometer parallel to the floor as it spanned the space between the treatment table and the bolster. The board goniometer was carefully positioned under the shoulder, parallel to the sternum. The glenohumeral joint was palpated posteriorly by locating the posterior/inferior aspect of the acromion. The axis of the Board Goniometer was positioned under this joint reference point. To check the accuracy of this position, the humerus was gently abducted with elbow bent at 90°. With the board positioned correctly, with reference to the glenohumeral joint, the long axis of the humerus would line up with each of the 10° lines on the board goniometer. The scapular was stabilized with the abduction board's positioning and the researcher was in a position to see if any scapular movement occurred during the measurement. With the elbow bent at 90°, the humerus was taken into abduction by sliding it across the board goniometer. Care was taken that the humerus did not lift off the board goniometer, as pure frontal plane abduction for measurement reliability was needed for the superimposed external rotation measurement.

The humerus was taken into glenohumeral abduction until the first-resistance was felt by the researcher in chronic phase, or pain limited motion in acute phase. The measurement was maximum, pain-free abduction range or 90° abduction, whichever came first. This then became the abduction angle from which all the following external rotation measurements were taken for the rest of the study. None of the subjects were able to attain 90° of abduction at the beginning of the study. There was no attempt to stretch the arm into abduction or external rotation.

The external rotation measurement was taken with a standard 12" goniometer. It had 360°, and was constructed of clear, plastic and was tight, to prevent slippage. The goniometer was adapted for the above measurement technique by cutting off the range from 180° to 360°. This was done to give the goniometer a flat surface to rest the stabilizing arm against the board goniometer as shown in Figure 2. Before the study commenced, the accuracy of the goniometer was examined by measuring 10 randomly chosen reference angles between 0° and 180°. The goniometer was also examined before each measurement session to ensure proper functioning and between each measurement, it was taken to 180° degrees so that the previous position would not influence the new measurement.

To prevent measurement bias, the back of the goniometer scale was covered with white adhesive paper before the study. This prevented the researcher from seeing the values when taking the measurements, but allowed the measurement on the front side to be seen for recording the measurement. Because of this adaptation, two goniometers were made - one for measuring the left shoulder and one for measuring the right shoulder.

External rotation was measured with the stationary arm of the adapted goniometer lying flat against the board goniometer. The moving arm was lined up along the shaft of the ulnar. The forearm moved backwards as the arm (humerus) rotated outward. If the subject was not able to get to perpendicular, then the measurement was recorded as a negative value. During the measurement, it was important to prevent any motion in the trunk or scapula and to not allow the humerus to lift off the table.

APPENDIX 3

ULTRASOUND PROTOCOLS

All subjects were given a thermal dose of ultrasound treatment over the portion of the capsule that was to be targeted for mobilization (posterior capsule for Group 1 and anterior capsule for Group 2). This was intended to preheat the targeted joint capsule and ligaments and thus alter the viscoelastic properties of the connective tissue and maximize the effectiveness of the stretch mobilizations to follow.[61]

A thermal dose of ultrasound was calculated to provide the target tissue with a moderate to vigorous temperature rise of 3°- 4° centigrade which is deemed enough to alter the viscoelastic properties of connective tissue.[61] Ultrasound doses were determined individually for each subject in order to provide the appropriate dose, as the capsule is at different depths on different individuals, due to different body sizes and types. In general, most posterior capsules were treated with I MHz because it was determined that the capsule was 2 – 5 cm deep and most anterior capsules were treated with 3 MHz used as the targeted anterior capsule was determined to be .5 – 2 cm deep.[61] If a subject was a large man, a 1 MHz may have been needed to target the anterior capsule, and if a subject was a small thin female, a 3 MHz may have been needed for the posterior capsule. The specific parameters chosen were set at the discretion of the researcher and were recorded at the first visit and repeated for each of the following treatments. All ultrasound treatments were at 1.5 W/cm2, continuous, for 10 minutes, using a Sonicator® Ultrasound Generator, Model ME 730, purchased from Mettler Electronics® Corporation, Anaheim, California, 92805, USA. Calibration and electrical safety inspection performed yearly, prior to testing (August) and 1 year later. Coupling gel used was Kendall Life Trace Ultrasound Gel, from Tyco Health Care Group LP, Mansfield, MA, 02048, U.S.A.

The sound head was moved in a circular pattern at the rate of approximately 4cm/second. The area covered by the ultrasound head was about twice the size of the sound head. Subjects were instructed to report any discomfort. If the subject reported discomfort, the sound head was moved more rapidly and if this was not adequate, the intensity of the dose was reduced. Two subjects needed the intensity reduced for a couple of treatments, until they were more comfortable with the higher dose. Because the effect of ultrasound in connective tissue lasts for just a few minutes, joint mobilization followed as soon as the subject could be positioned appropriately.

APPENDIX 4

JOINT MOBILIZATION TECHNIQUES

Joint mobilization is a specialized form of passive movement to produce movement between joint surfaces,[61,62] to elongate tight connective tissues (ligaments, joint capsule, periarticular fascia)[63] that are restraining joint motion.[5,54,55,64] Because the capsule of the glenohumeral joint is normally loose, this joint is particularly suited for restoring motion with joint mobilization.[65] It is believed that joint mobilization promoting normal joint translations may be critical in the safe, effective treatment of adhesive capsulitis, because it may restore the flexibility of the joint capsule necessary for the translational motions associated with normal glenohumeral joint movements.[28]

In this study, mobilizations were performed with the capsule in a lengthened range (greater abduction) for a more effective stretch.[56] Gliding mobilizations produce some intra-articular compression, and more so with stiffer joints.[13,21] Thus before and during the glide mobilization was performed, care was taken to gain and maintain lateral distraction to reduce these compressive forces and thus facilitate the glide motion.[28] To achieve this, a lateral glide mobilization was performed in the resting position with both hands for at least a minute or more as tolerated by the patient. Then a posterior or anterior glide was superimposed onto the lateral distraction, as described below.

A stretch-mobilization technique was chosen for this study. Group 1 received lateral glide with a superimposed posterior glide. Group 2 received lateral glide with a superimposed anterior glide. Each of the stretch mobilizations was held for a total of 15 minutes at each treatment session, with only short breaks for researcher/subject comfort. The force applied was expected to elongate the connective tissue at the targeted site of the capsule.[64] A sensation of moderate stretching was expected in the area of the posterior capsule with posterior glide techniques for Group 1 and in the area of the anterior capsule for Group 2. Subjects were instructed to report symptoms during the technique, so that the researcher could modify the force and direction as necessary. If the procedure produced protective muscle spasm, significant pain or other symptoms, the force was reduced, more lateral distraction given or a different angle tried. If not enough stretch was reported, then more force was applied, or an alternative position (see Figure 2b) was chosen for moderate stretch to be felt during the whole 15 minutes of the procedure in order to effect permanent elongation or plastic deformation of the connective tissue.[64,65]

Posterior glide mobilization (Figures 2a and 2b)

The subject lay supine and the researcher stood at the head of the treatment table, facing across the subject's shoulders. The researcher placed both hands on the shaft of the humerus. A dorsally directed force was applied to the ventral aspect of the proximal humerus by the reseracher's proximal hand, while the distal hand maintained lateral distraction and held the humerus parallel to the floor.[66] A posterior glide of the humeral head was superimposed with the flat part of the proximal hand over the anterior aspect of the humeral head. During the procedure, as more abduction range was gained, then the arm was taken further into abduction so that the mobilizations could be performed in the lengthened range.[57,67]

As the subject progressed with more flexion range and greater tolerance to the stretch-mobilization, he/she would be positioned supine, with the humerus was flexed to 90°. The elbow was bent allowing whatever degree of internal or external rotation was comfortable for the subject. The researcher stood at the subjects shoulder level, facing the subject, looking across both shoulders. The proximal hand held the humerus, close to the joint, maintaining the lateral glide. The distal hand controlling the direction of the mobilization was placed over the olecranon process.[56] Pressure was directed through the long axis of the humerus, into a straight posterior direction[28] with the researcher's distal hand, or sternum, if necessary, while maintaining lateral glide. See Figure 2b. After each stretch-manipulation, the humerus was passively and gently taken into more abduction and external rotation, as the joint would allow. This took up the gain in range during the technique and kept the stretch near the point of restriction as further posterior glide mobilizations were performed.

Anterior glide mobilizations (Figures 3a and 3b)

The subject lay supine and the researcher stood at the head of the treatment table, facing across the subject's shoulders. The researcher placed both hands on the shaft of the humerus. A ventrally directed force was applied to the dorsal aspect of the proximal humerus by the researcher's proximal hand, while the distal hand maintained lateral distraction and held the humerus parallel to the floor.[56] An anterior glide of the humeral head was superimposed with the flat part of the proximal hand over the posterior aspect of the humeral head. During the procedure, as more abduction range was gained, then the arm was taken further into abduction so that the mobilizations could be performed in the lengthened range.[57] See Figure 2a.

As the subject progressed with more flexion range and greater tolerance to the stretch-manipulation, he/she would be positioned prone near the edge of the bed. The researcher stood at the head of the subject. The researcher's proximal hand grasped the humerus close to the humeral head. The distal hand grasped the humerus just above the elbow. The proximal hand of the researcher then directed anterior pressure to the head of the humerus, while the distal hand held the humerus in lateral distraction and parallel to the ground.[56] See Figure 2b. With each repetition of mobilization, the humerus was taken into more abduction and external rotation, as tolerated by the subject, to take up the gain in range during the technique.

Strength of stretch mobilization

Before the study commenced, the amount of force that this researcher could produce, in these mobilization positions, was measured by using an upright scale. The mobilization position was assumed on a normal male shoulder with the researcher standing on the scales. The scales were preset for the researcher's body weight and as the researcher performed the mobilization technique, there was an unweighting on the scales. The amount of unweighting was measured. It was demonstrated that a force of 60 kilograms was comfortable for this researcher to apply to a shoulder joint for up to 15 minutes. This was comparable to forces calculated by other therapists doing manual therapy techniques.[66] Most subjects, however, could not tolerate this amount of pressure.

APPENDIX 5

COMPARISON OF INITIAL AND FINAL PAIN SCORES

Visual Analogue Scale (VAS) pain scores were analysed at two times, initial and final. Table 5 compares the pain scores in centimeters of the initial and final VAS per group. Initial and final VAS pain scores were compared within each group using paired t-tests. An independent t-test was used to compare VAS scores between groups' initial and final scores. Alpha was set at .05.

There was no significant difference, (p = .48) between the two groups initially, however, both groups received a reduction of pain levels by the end of six treatments. Group 1 had a significant reduction of pain (p = .02) compared with Group 2 where pain reduction was not as significant (p = .07). See Figure 5.

TABLE 4. Comparison of initial and final VAS pain scores (centimeters) by group.

Time	**Group 1** (n = 8) Mean± SD	**Group 2** (n = 10) Mean ± SD	*p* value*
0	7.4 ± 2.3	8.3 ± 2.9	.48
6	4.9 ± 2.5	6.6 ± 3.8	.29
p value†	.02	.07	

* Independent t-test
† Paired t-test

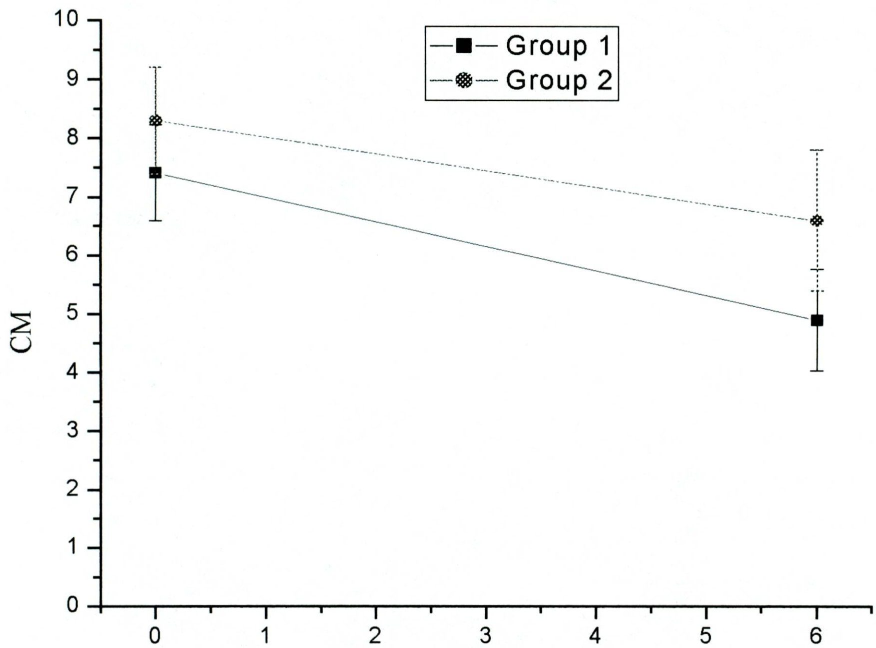

FIGURE 5. Comparison of initial and final mean VAS pain score by group (Mean +/-SEM).

APPENDIX 6

COMPARISON OF FUNCTIONAL IMPROVEMENT

Impairment and function are found not to be correlated in patients with adhesive capsulitis.[30] This finding seems to be due to the ability to compensate with the unaffected arm for most activities. A self-assessment questionnaire was developed to assess functional status of the subjects in this study.

This self-assessment, five functional questions, was completed by each subject initially, and at the final treatment session. See Figure 6. Questions 1 and 2 were scored separately. The scores of Questions 3, 4 and 5 were combined as they had similar content. The initial and final scores were compared using median, minimum and maximum scores. Within group scores were analysed using the Wilcoxon signed-rank test and the two groups were compared using a Mann-Whitney U test. Alpha level was set at .05.

Table 5 shows initial and final scores for the functional questions. Within Group 1 there was a significant improvement ($p = .05$), with Questions 3-5 combined over time. In Group 2 there was a significant improvement ($p = .02$) with Question 1 over time. There was no significant different between the groups for any of the functional questions.

TABLE 5. Comparison of functional improvement by group.

Function Questions		**Group 1** (n = 8) Median(min,max)	**Group 2** (n = 10) Median(min,max)	*p* value*
Question 1: (scores, 0-4)‡	Initial	4.0 (3,4)	4.0 (2,4)	.76
How often does the pain in your shoulder make it difficult for you to sleep at night?	Final	4.0 (1,4)	3.0 (1,4)	.70
	p value†	.14	.02	
Question 2: (scores, 0-4)‡	Initial	2.5 (1,4)	2.0 (1,3)	.70
Considering all the ways you use your shoulder during daily personal and household activities (i.e. dressing, washing, driving, household chores, etc.), how would you describe your ability to use your shoulder?	Final	2.0 (0,3)	2.5 (1,3)	.24
	p value†	.06	1.00	
Question 3-5: (scores, 0-12)§	Initial	9.0 (6,11)	8.5 (6,12)	.90
3. How much difficulty have you had putting on or removing a pullover, sweater or shirt due to your shoulder problem? **4.** How much difficulty have you had combing or brushing your hair due to your shoulder problem? **5.** How much difficulty have you had reaching shelves that are above your head due to your shoulder problem?	Final	6.0 (1,9)	7.0 (2,10)	.36
	p value†	.05	.10	

* Mann-Whitney U
† Wilcoxon signed ranks test
‡ (0 = no difficulty, 4 = unable / severe)
§ combined scores (0-12)

APPENDIX 7

CORRELATIONS

Considering all the subjects together, Spearman's rho was used to find if there were any significant correlations between the change in external rotation range, the change in the functional questions or the change in VAS pain scores over time, independent of group. Alpha was set at .05.

Significant correlations were found with the change in VAS pain scores and a change in Question 2 (r_s = .56, p = .02) and combined Questions 3 - 5 (r_s = .61, p = .01) which indicated that there was a correlation between pain and function. This agreed with the findings of Shaffer et al in which as subjects experienced less pain, then their function would improve.[30]

A change in external rotation range was negatively correlated with a change in Question 2 (r_s = -.57, p = .01) and combined Questions 3 – 5 (r_s = -.47, p = .05). This indicated that as external rotation improved, the subject had an improvement in overall daily function as well as specific functional tasks that involved reach and external rotation range. Other variables had no significant correlations. This result has not been reported by other researchers. It is not known, however if the modified function questions were a factor or whether the amount of improvement in the posterior translation manipulation group was significant enough to change this correlation. Further studies are needed to find the factors that help function and impairment become correlated.

For the total of six acute subjects (soft joint end-feel)[33] from both groups, there was no correlation between VAS pain scores, function questions or external rotation range. The small sample size of this group may have influenced this lack of correlation. For the other 12 subjects that were in the chronic phase of the condition (firm joint end-feel)[33] from both groups, the VAS pain score did correlated with Question 2 (r_s = .63, p = .03) and with Questions 3-5 (r_s = .72, p = .008). This shows a strong correlation between function and pain in the chronic phase but there was no statistical correlation between external rotation measures and pain. These results were similar to those found by Shaffer et al.[30]

APPENDIX 8

APPENDIX REFERENCES

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