An Original Study

Quantification of Two-Dimensional Glenohumeral Rhythm in Persons With and Without Symptoms of Shoulder Impingement

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Abstract

A repeated-measures design was used to assess glenohumeral rhythm in 10 patients with shoulder impingement and 10 pain-free persons and to assess the effects of subacromial injection on glenohumeral rhythm within the impingement group. Scapular-plane anterior-to-posterior x-rays of the scapula and humerus were obtained at 5 angles of arm elevation (resting, 30°, 60°, 90°, 120°). For the impingement group, x-rays were repeated after subacromial injection (10 mL of 1% lidocaine). No significant differences in glenohumeral rhythm were found between the impingement and control groups across all arm-elevation angles.

Subacromial impingement is the most common cause of shoulder pain.[1] Clinically, patients present with a painful arc of motion during active arm elevation, typically 70° to 120°.[2] Shoulder crepitus, weakness, and tenderness are also commonly reported.[3] Pain associated with subacromial impingement also can be reproduced passively by stabilizing the scapula and elevating the patient's arm with the glenohumeral joint internally rotated. Neer[2] called this maneuver the *impingement sign*. Subacromial impingement can be clinically confirmed when pain decreases after an injection of 10 mL of 1% lidocaine beneath the acromion.[2]

Although subacromial impingement is a common orthopedic problem, the etiology of this disorder has not been clearly documented. Neer[2] described subacromial impingement as the result of a mechanical process in which there is compression of the supraspinatus tendon and subacromial bursa between the humeral head and the acromion/coracoacromial ligamentous complex. This repetitive compression has been reported to lead to inflammation, fibrosis, and, ultimately, rupture of the rotator cuff.[3]

Although the mechanism of subacromial impingement is commonly accepted as being mechanical, what causes this mechanical compression has not been clearly elucidated. There are several hypothesized factors, including rotator cuff weakness, glenohumeral capsular tightness, muscle imbalance, shape of the acromion, and altered glenohumeral rhythm.[1,4-20] Given the large variety of factors implicated in subacromial impingement, proposed treatment approaches have been many and varied.[1,4,7,10,16,21-25]

One component commonly addressed in the conservative treatment of subacromial impingement is restoration of glenohumeral rhythm.[4,16,21,23-30] *Glenohumeral rhythm* is defined as the ratio of humeral motion to motion of the scapula on the thorax.[31] Although abnormal glenohumeral rhythm could be the result of excessive motion of the scapula, limited scapular mobility has been postulated as being more closely associated with subacromial impingement.[32] This premise is based in part on the belief that, in persons with subacromial impingement, the scapula lacks normal upward rotation, which could potentially limit clearance for the greater tuberosity of the humerus during active elevation and increase compression under the acromion.[2] As the treatment of subacromial impingement is often directed toward improving scapulothoracic mobility as well as strengthening the scapular stabilizers and upward rotators, the role of altered glenohumeral rhythm in this symptomatic population needs to be adequately documented. In addition, it is not known whether altered glenohumeral rhythm is a cause or effect of subacromial impingement syndrome.

Using static radiographic techniques, Poppen and Walker[31] quantified glenohumeral and scapulothoracic motion in 15 patients with various shoulder pathologies. They found glenohumeral rhythm to be altered in patients with shoulder pathology but did not report the specific glenohumeral rhythm abnormalities in relation to the different diagnoses. Paletta and colleagues,[33] using similar radiographic techniques to assess glenohumeral rhythm in patients diagnosed with shoulder instability and with rotator cuff tears, reported a significant difference in glenohumeral rhythm for the instability group versus individuals without shoulder pathology.

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Evidence of altered scapular rotation in persons with symptoms of subacromial impingement was provided by Ludewig and Cook,[32] who reported decreased upward scapular rotation at 60° of humeral elevation in the scapular plane compared with individuals with non-painful shoulder motion. In contrast, Lukasiewicz and colleagues[34] quantified 3-dimensional (3-D) scapular kinematics and did not find any differences in upward scapular rotation between impingement and control groups. However, it should be noted that the study conducted by Lukasiewicz and colleagues[34] evaluated only 3 angles of arm elevation (resting, 90°, maximum elevation). Although the studies by Ludewig and Cook[32] and Lukasiewicz and colleagues[34] provide some insight into the role of the scapula in this syndrome, the role of scapular rotation and, perhaps more important, the relationship of scapular motion with respect to the humeral motion (ie, glenohumeral rhythm) needs to be further defined. To date, no study has quantified glenohumeral rhythm in an impingement population.

The present study was conducted to determine whether patients with symptoms of shoulder impingement demonstrated altered 2-dimensional (2-D) glenohumeral rhythm compared with asymptomatic controls and to assess the effects of subacromial injection of 10 mL of 1% lidocaine on 2-D glenohumeral rhythm within the impingement group. We hypothesized that patients with symptoms of shoulder impingement would demonstrate altered glenohumeral rhythm (decreased scapular upward rotation relative to humeral elevation) and that glenohumeral rhythm would be restored by the pain-relieving subacromial injection.

Materials and Methods

Subjects

The study included 5 men and 5 women (mean age, 40.3 years; SD, 8.5 years) with symptoms of shoulder impingement and 5 men and 5 women (mean age, 40.4 years; SD, 8.7 years) who were asymptomatic. The 10 persons with shoulder pain were seeking medical care for their symptoms and were recruited from the Healthcare Consultation Center at the University of Southern California. Only patients with a history related to overuse or insidious onset and diagnosed with a positive subacromial impingement test by an orthopedic surgeon were considered. Minimizing the possibility that symptoms were related to tensile failure of the supraspinatus tendon (ie, rotator cuff tear) involved excluding from the study patients who were older than 45 and patients whose symptoms resulted from trauma or acute dislocation.[2,3,15,28] Mean time from onset of symptoms was 5.9 months (range, 2 weeks to 2 years). Eighty percent of the patients with symptoms of shoulder impingement reported an activity-related mechanism of injury (reaching, pushing, pulling, lifting), and 90% reported symptoms in the dominant arm.

The diagnosis of subacromial impingement syndrome was based on 4 clinical criteria: painful arc on active elevation of the arm in the scapular plane, pain on palpation of the subacromial space, decreased active range of motion (ROM) in elevation compared with the contralateral side (≥ 20° side-to-side difference as determined by goniometer), and positive Neer impingement test.[2,3,9] Subjects were excluded from the study if there was evidence of glenohumeral joint instability, as indicated by a positive relocation test or sulcus sign; rotator cuff tear, as indicated by a positive drop arm test; humeral head, glenoid, or acromial fractures; previous shoulder surgery on the involved side; moderate to severe scoliosis; neuromuscular disorders affecting the shoulder or trunk muscles; tumors or masses in the shoulder; or congenital anomalies affecting the shoulder.

The asymptomatic controls were recruited from the staff and employees of the University of Southern California Health Sciences campus and were matched with the patients on age, sex, and arm dominance. Several criteria were used to select controls: no history of shoulder pain or pathology, no current shoulder pain, full active and passive range of glenohumeral joint motion, normal strength of the shoulder musculature, no neurologic disorders, no tumors or masses in the shoulder, and no congenital anomalies affecting the shoulder.

The number of subjects recruited for this study was based on a sample size calculation. Ten subjects would be required per group to detect a 30% difference in kinematic variables (significant group effect) with a power level of .80 ($\alpha = .05$ for a 1-tailed test).

Procedure

Before participation, the procedures and risks of the study were explained to all subjects, and informed consent was obtained. For the impingement group, active range of arm elevation in the scapular plane was recorded with a standard goniometer.[35] Magnitude of pain associated with arm elevation was assessed with a visual analog scale and was at least 50% reduced in order to be included.

Five anterior-to-posterior x-rays of the scapula and humerus were obtained at resting and at 30°, 60°, 90°, and 120° of arm elevation in the scapular plane (Figure 1). To

Figure 1. Standard x-ray taken in scapular plane (60°) for measurement of glenohumeral and scapulothoracic angles.

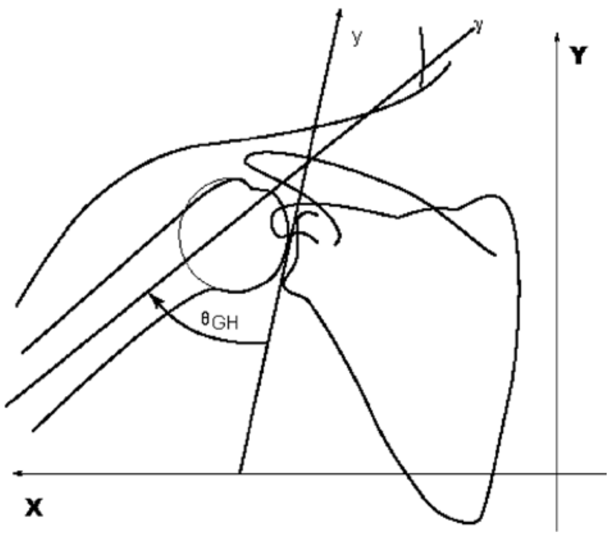

Figure 2. Glenohumeral angle (θ_{GH}) was defined as the angle formed by a line bisecting the longitudinal axis of the humerus (γ) and a line connecting the superior and inferior aspects of the glenoid fossa (y).

ensure a perpendicular view of the scapula, subjects stood at a 30° to 45° angle with respect to the x-ray source, and the x-ray tube was adjusted for each subject such that the scapula was parallel to the x-ray plane.[31] A metal chain (visible in the x-ray field) was hung as a plumb line and served as a vertical axis for measurements (Figure 1).

All x-rays (with the exception of the resting view) were taken with the subject performing a static isometric contraction. Each arm-elevation angle was determined with a goniometer, and a mark on a stationary pole provided feedback regarding arm position during the radiographic procedure. When the estimated degree of arm elevation (using the goniometer) was compared with the actual elevation angle based on x-ray measurements, the difference between the 2 measures (when averaged across all angles of arm elevation) was 7.9°, with the largest variability between the 2 measurements within the resting to 30° range.

Subjects were required to hold each position for approximately 10 seconds. To prevent a lateral trunk lean toward the noninvolved side, a crutch was placed upright into the axilla opposite the arm being x-rayed. A 1-minute rest was given between each of the x-rays obtained. None of the subjects reported fatigue during the radiographic evaluation.

After the initial radiographic evaluation, each patient in the impingement group received a subacromial injection of 10 mL of 1% lidocaine administered by an orthopedic surgeon. Five minutes after receiving the injection, the patient was instructed to repeat active arm elevation in the scapular plane, and pain was reassessed. The injection was considered positive if there was pain reduction of at least 50% with active elevation. If the pain criterion was met, the same radiographic procedures were repeated. If pain was not reduced by 50%, the patient did not continue in the study. All subjects evaluated met the pain criterion.

Data Analysis

Each x-ray was evaluated to determine the glenohumeral angle and the scapulothoracic angle as reported by Poppen and Walker.[31] The glenohumeral angle was defined as the angle formed by a line defining the longitudinal axis of the humerus and a line defining the orientation of the glenoid fossa (Figure 2). The longitudinal axis of the humerus bisected the proximal third of the bone, whereas the line defining the orientation of the glenoid connected the superior and inferior aspects of the fossa.[31] Scapulothoracic angle was defined as the angle formed by a line defining the orientation of the glenoid fossa and a vertical line perpendicular to the horizontal plane (Figure 3). As with the glenohumeral angle, the line defining the orientation of the glenoid connected the superior and inferior aspects of the fossa. The vertical line in the x-ray field was defined by the plumb line chain and represented the y-axis of the trunk.[31]

Glenohumeral rhythm was defined as the ratio of the glenohumeral angle to the scapulothoracic angle. To determine the ratio of glenohumeral motion to scapulothoracic motion for a given subject, the glenohumeral angle from a given position was subtracted from the glenohumeral angle of the next consecutive position. The same was done for the scapulothoracic angles, and a ratio was then determined. Glenohumeral rhythm for a given ROM was defined as the ratio of the glenohumeral angle to the scapulothoracic angle between consecutive arm-elevation angles (ie, resting to 30°, 30° to 60°, 60° to 90°, 90° to 120°).[23,31,36] For example, a value of 2.0 was indicative of 2° of glenohumeral joint motion for every 1° of scapulothoracic motion for a given arc of motion. This method of determining glenohumeral rhythm has previously been shown to be capable of assessing differences between various clinical populations.[31,33] All radiographic measurements were made by the same investigator using a standard straight edge and a goniometer.

Measurement Reliability

To assess the intrarater reliability of the measures used to calculate glenohumeral rhythm, x-rays from 4 asymptomatic subjects were evaluated on 2 separate occasions, 2 weeks apart. The same procedures were used for both measurement sessions, as described above. The investigator was blinded to the results of previous measurements (all lines drawn on film were erased). New x-rays were not taken as part of the reliability assessment.

Statistical Analysis

Intrarater reliability of the 2 measures used to calculate glenohumeral rhythm was assessed with the intraclass correlation coefficient (ICC: 3,1).[37] ICCs were generated with the equation BMS – EMS / BMS + (k – 1) EMS, in which BMS = between-subjects mean square, EMS = error mean square, and k = number of raters (k = 1 in this case, as only 1 rater measured angles). A univariate, repeated-measures analysis of variance (ANOVA) using the reliability data was performed to obtain the necessary values for the ICC equa-

Table I. Glenohumeral Angles in Impingement and Control Groups, Mean Degrees (SD)

Group	Resting	Arm Elevation 30°	60°	90°	120°
Impingement	13.9 (6.1)	46.9 (10.6)	59.5 (9.0)	71.8 (8.2)	83.9 (8.4)
Control	9.2 (4.9)	42.6 (15.3)	56.7 (11.7)	67.9 (7.7)	76.6 (5.8)

Table II. Scapulothoracic Angles in Impingement and Control Groups, Mean Degrees (SD)

Group	Resting	Arm Elevation 30°	60°	90°	120°
Impingement	–3.3 (4.6)	–2.6 (9.0)	2.7 (8.7)	12.5 (9.4)	27.6 (11.2)
Control	–4.2 (7.5)	1.0 (8.7)	5.3 (10.4)	16.2 (6.7)	33.9 (6.2)

Table III. Glenohumeral Rhythms[a] in Impingement and Control Groups, Mean Ratio (SD)

Group	Arm Elevation Range Resting–30°	30°-60°	60°-90°	90°-120°
Impingement	14.1 (13.4)	4.9 (4.9)	1.0 (0.3)	0.6 (0.3)
Control	12.5 (15.2)	5.4 (4.8)	1.6 (1.5)	0.6 (0.4)

[a]Defined as ratio of glenohumeral motion to scapulothoracic motion.

tion. For both the glenohumeral and scapulothoracic angle measurements, factors used in the ANOVA were subjects (4) and trial (2). Reliability of measurements was assessed at each degree of arm elevation.

To determine whether glenohumeral angle and scapulothoracic angle varied between the impingement and control groups, separate 2x5 (group x angle) ANOVAs with repeated measures on 1 factor (angle) were used. To determine whether glenohumeral rhythm varied between groups, a 2x4 (group x range) ANOVA with repeated measures on 1 factor (range) was used.

To determine whether glenohumeral angle and scapulothoracic angle varied within the impingement group before and after injection, separate 2x5 (group x angle) ANOVAs with repeated measures on 2 factors (injection, angle) were used. To determine whether glenohumeral rhythm varied between groups, a 2x4 (group x range) ANOVA with repeated measures on 2 factors (injection, range) was used.

For each repeated-measures ANOVA, significant main effects were reported if there was no interaction. If a significant interaction was found, the individual main effects were analyzed separately. Statistical software (BMDP Statistical Software Inc, Los Angeles, CA) was used for all analyses. Level of significance was set at $P<.05$.

Results

For the reliability portion of this study, the ICC for the glenohumeral angle (averaged across all angles of arm elevation) was .95. Mean ICC for the scapulothoracic angle was .94.

There was no significant difference in the glenohumeral angle or scapulothoracic angle between the impingement and control groups when averaged across all ranges of arm elevation (no group effect, no interaction; Tables I, II). Similarly, there was no difference in glenohumeral rhythm between the impingement and control groups when averaged across all ranges of arm elevation (no group effect, no interaction; Table III). The largest difference in glenohumeral ratios between the 2 groups was observed from resting to 30° (impingement group mean, 14.1, and SD, 13.4; control group mean, 12.5, and SD, 15.2). However, this was not statistically significant.

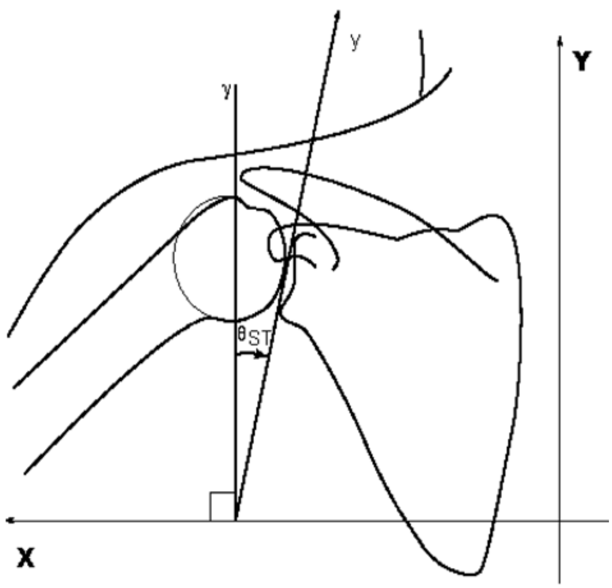

Figure 3. Scapulothoracic angle (θ_{ST}) was defined as the angle formed by a line defining the orientation of the glenoid fossa (y) and a vertical line perpendicular to the horizontal (γ).

Table IV. Impingement Group: Glenohumeral Angles Before and After Subacromial Injection, Mean Degrees (SD)

Group	Resting	Arm Elevation 30°	60°[a]	90°[a]	120°
Preinjection	13.9 (6.1)	46.9 (10.6)	59.5 (9.0)	71.9 (8.4)	83.9 (8.4)
Postinjection	15.3 (6.8)	44.2 (9.1)	53.6 (9.5)	65.5 (5.6)	78.6 (10.2)

[a]Postinjection value significantly less than preinjection value.

Table V. Impingement Group: Scapulothoracic Angles Before and After Subacromial Injection, Mean Degrees (SD)

Group	Resting	Arm Elevation 30°	60°	90°	120°
Preinjection	–3.3 (4.6)	–2.6 (9.1)	2.7 (8.7)	12.5 (9.4)	27.6 (11.2)
Postinjection	–3.8 (5.7)	0.1 (6.0)	6.8 (7.3)	18.0 (5.7)	31.3 (8.5)

Table VI. Impingement Group: Glenohumeral Rhythms* Before and After Subacromial Injection, Mean Ratio (SD)

Group	Arm Elevation Range Resting–30°	30°-60°	60°-90°	90°-120°
Preinjection	14.1 (13.4)	4.9 (4.9)	1.0 (0.3)	0.6 (0.3)
Postinjection	11.6 (10.2)	4.0 (4.9)	1.0 (0.6)	1.0 (0.6)

[a]Defined as ratio of glenohumeral motion to scapulothoracic motion.

After the subacromial injection in the impingement group, a significant group effect (no interaction) was found for both the glenohumeral angle and the scapulothoracic angle. Post hoc analysis revealed that, after the subacromial injection, there were mean decreases of 5.9° and 6.4° in the glenohumeral angle at 60° and 90°, respectively, and mean increases of 4.1° and 5.5° in the scapulothoracic angle at 60° and 90°, respectively (Tables IV, V). However, there was no difference in glenohumeral rhythm within the impingement group before and after the subacromial injection when averaged across all ranges of arm elevation (no injection effect, no interaction; Table VI). The largest difference in glenohumeral ratios before and after injection was found from resting to 30° (preinjection mean, 14.1, and SD, 13.4; postinjection mean, 11.6, and SD, 10.2). However, this was not statistically significant.

Discussion

This study found no differences in glenohumeral angle, scapulothoracic angle, or 2-D glenohumeral rhythm between patients with symptoms of shoulder impingement and asymptomatic controls. More specifically, our data do not support the premise that patients with symptoms of shoulder impingement demonstrate decreased scapular upward rotation relative to humeral elevation in the scapular plane.

As no group differences were evident with respect to glenohumeral rhythm, it is not entirely surprising that a pain-relieving injection did not change this parameter in the impingement group. Nonetheless, small but significant differences in the glenohumeral angle and the scapulothoracic angle were observed after injection. On average, the glenohumeral angle was significantly smaller at 60° and 90° after injection, and the scapulothoracic angle was significantly larger at 60° and 90° after injection. However, these differences did not translate into a change in glenohumeral rhythm in this range.

That these differences were evident at the ROM points that commonly reproduce impingement symptoms suggests that the pain-relieving injection affected scapular mechanics. For example, at 60° and 90°, a larger amount of scapular upward rotation was found. Thus, to achieve the desired angle for testing, less glenohumeral motion was required. For the most part, the postinjection values for both angles resulted in mean values being closer to those of the control group. However, that the 2 groups were not significantly different at the start makes interpretation of this finding difficult.

The scapulothoracic angle data obtained from the controls compare favorably with the healthy subjects' 3-D scapular upward rotation data reported by Ludewig and colleagues.[38] In addition, our group comparison results for the scapulothoracic angle agree with the data of Lukasiewicz and colleagies,[34] who reported no significant differences in upward scapular rotation between patients with impingement syndrome and persons without shoulder pathology. Similarly, Ludewig and Cook[32] reported no differences in scapular upward rotation in patients with shoulder impingement symptoms at humeral angles of 90° and 120°. They did report a small but significant difference (4.1°) at 60°, but data were collapsed across various loading conditions—making comparisons with our results difficult.

Taken together, the findings of these studies suggest that limited scapular upward rotation may not play as large as role as previously thought with regard to etiology of subacromial impingement syndrome. Although mechanical compression of the supraspinatus between the humeral head and the acromion (a result of decreased scapular upward rotation) is commonly accepted as a potential cause of impingement symptoms, other factors, such as superior migration of the humeral head relative to the glenoid fossa, should be evaluated. Furthermore, the data of Lukasiewicz and colleagues[34] and Ludewig and Cook[32] suggest that scapular motion in different planes (ie, anteroposterior tilting in the sagittal plane; superoinferior motion in the frontal plane) may be important factors in the etiology of subacromial impingement. Continued research is needed to clearly define the role of the scapula in this population.

The results obtained for 2-D glenohumeral rhythm in our controls compare favorably with the normative data of Poppen and Walker.[31] For 30° to 120° of elevation, Poppen and Walker reported mean glenohumeral rhythm to be 1.25, versus our 1.1. In contrast, Inman and colleagues[23] reported glenohumeral rhythm to be 2.0 throughout the same range of elevation. One possible explanation for this discrepancy may be related to Inman and colleagues' measuring glenohumeral rhythm in the frontal plane. Their method would lead to errors in quantifying scapular rotation, as the scapula rests on the thorax approximately 37° anterior to the frontal plane.[39]

Glenohumeral rhythm is often reported for the overall range of arm elevation. However, our study results show that the ratio of humeral elevation to scapular rotation was not consistent throughout the entire range. For example, during arm elevation from resting to 30°, the glenohumeral ratio of our control group was 12.5, indicating that most of the motion was obtained from the humerus moving away from the body, while the scapula contributed very little to overall motion. Although the larger contribution of humeral motion in relation to scapular motion has previously been observed in this range, our glenohumeral ratio was higher than that reported by other investigators. For example, Freedman and Munro[36] reported a value of 1.35, and Poppen and Walker[31] and McQuade and Smidt[39] reported values of 4.3 and 7.5, respectively. The discrepancy in glenohumeral ratios from 0° to 30° is consistent with the conclusions of Inman and colleagues[23] in that, during the initial stages of elevation, the scapula tends to "seek stability," resulting in large variability in healthy subjects.

The glenohumeral ratio from 30° to 60° was 5.4 for the control group, indicating that the scapula contributed a relatively small amount of upward rotation to achieve 60° of arm elevation. This ratio is comparable to the 6.7 reported by McQuade and Smidt,[39] who used a 3-D tracking system. From 60° to 90°, however, the contribution of the scapula increased substantially (ratio, 1.6), while the final degrees of arm elevation were dominated by upward scapular rotation as evidenced by a ratio of .6.

Caution must be taken in generalizing the results of this study to the entire subacromial impingement population, as only 10 patients were evaluated. Although significant differences were observed in kinematic variables within the impingement group (preinjection vs postinjection), the a priori power analysis revealed we had enough subjects to detect a 30% difference in kinematic variables between the impingement and control groups. However, data variability was higher than expected, and smaller group differences were observed. Post hoc analyses revealed power values of .73, .43, and .05 for the between-group comparison (significant main effect) of glenohumeral angle, scapulothoracic angle, and glenohumeral rhythm, respectively. Although a modest increase in sample size may have resulted in significant group differences for the glenohumeral angle and the scapulothoracic angle, a very large sample size would have been needed to detect such a small group difference in glenohumeral rhythm. Whether the difference detected in such an analysis would be considered clinically relevant remains to be seen.

Other limitations of this study were that glenohumeral rhythm was assessed during an isometric contraction rather than during dynamic movement and that glenohumeral rhythm was quantified with 2-D measurements. Although comparisons of glenohumeral rhythm during active and static conditions have not been made, with current advances in kinematic magnetic resonance imaging techniques and "open" imaging systems, new studies should be able to address this limitation. With regard to use of the 2-D measuring technique to quantify glenohumeral rhythm, McQuade and Smidt[39] reported that quantification of upward scapular rotation using 2-D radiographic measurement of in vivo glenohumeral elevation showed excellent agreement when compared with scapular rotation data obtained from electromagnetic sensors placed on the scapula and trunk (r^2 = .94). This suggests that the 2-D technique can provide data comparable to those of current 3-D techniques using external markers. However, the 2-D x-ray technique is subject to projection errors with any rotations out of the plane of analysis, which may contribute to impingement symptoms.

Conclusions

Patients with symptoms of shoulder impingement did not demonstrate differences in 2-D glenohumeral rhythm when compared with pain-free controls. Although a pain-relieving injection led to small but significant increases in upward scapular rotation at 60° and 90° in the impingement group, no differences in glenohumeral rhythm were observed.

These results do not support the clinical hypothesis that altered glenohumeral rhythm may be a contributory factor in the etiology of subacromial impingement syndrome. Care must be taken in generalizing these findings given the relatively small sample size and the limitations associated with our 2-D analysis of glenohumeral rhythm. More work is needed to elucidate the mechanical causes of subacromial impingement to assist in the development of specific and improved treatment programs.

Authors' Disclosure Statement and Acknowledgments

The authors report no actual or potential conflict of interest in relation to this article.

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References

1. Green A. Arthroscopic treatment of impingement syndrome. *Orthop Clin North Am*. 1995;26:631-641.
2. Neer CS. Impingement lesions. *Clin Orthop*. 1983;(173):70-77.
3. Matsen FA III, Arntz CT. Subacromial impingement. In: Rockwood CA, ed. *The Shoulder*. Philadelphia, PA: Saunders; 1990:623-646.
4. Allegrucci M, Whitney SL, Irrgang JJ. Clinical implications of secondary impingement of the shoulder in freestyle swimmers. *J Orthop Sports Phys Ther*. 1994;20:307-318.
5. Allegrucci M, Whitney SL, Lephart SM, Irrgang JJ, Fu FH. Shoulder kinesthesia in healthy unilateral athletes participating in upper extremity sports. *J Orthop Sports Phys Ther*. 1995;21:220-226.
6. Armstrong RJ, Buckle P, Fine LJ, et al. A conceptual model for work-related neck and upper-limb musculoskeletal disorders. *Scand J Work Environ Health*. 1993;19:73-84.
7. Babyar S. Excessive scapular motion in individuals recovering from painful and stiff shoulders: causes and treatment strategies. *Phys Ther*. 1996;76:226-259.
8. Burns WC, Whipple TL. Anatomic relationships in the shoulder impingement syndrome. *Clin Orthop*. 1993;(294):96-102.
9. Corso G. Impingement relief test: an adjunctive procedure to traditional assessment of shoulder impingement syndrome. *J Orthop Sports Phys Ther*. 1995;22:183-192.
10. DiVeta J, Walder ML, Skibinski B. Relationship between performance of selected scapular muscles and scapular abduction in standing subjects. *Phys Ther*. 1990;70:470-476.
11. Gibson MH, Goebel GV, Jordan TN, Kegerreis S, Worrel TW. A reliability study of measurement techniques to determine static scapular position. *J Orthop Sports Phys Ther*. 1995;1:100-106.
12. Glousman RE. Instability versus impingement syndrome in the throwing athlete. *Orthop Clin North Am*. 1993;24:89-99.
13. Gold RH, Seeger LL, Yao L. Imaging shoulder impingement. *Skeletal Radiol*. 1993;22:555-561.
14. Greenfield B, Catlin PA, Peyton WC, Green E, McDonald JJ, North C. Posture in patients with shoulder overuse injuries and healthy individuals. *J Orthop Sports Phys Ther*. 1995;21:287-295.
15. Jobe CM. Superior glenoid impingement—current concepts. *Clin Orthop*. 1996;(330):98-107.
16. Kamkar A, Irragang JJ, Whitney SL. Nonoperative management of secondary shoulder impingement syndrome. *J Orthop Sports Phys Ther*. 1993;17:212-224.
17. Kelley MJ, Clark WA. *Orthopedic Therapy of the Shoulder.* Philadelphia, PA: Lippincott; 1994.
18. Leroux JL, Hebert P, Mouilleron P, Thomas E, Bonnel F, Blotman F. Postoperative shoulder rotators strength in stages II and III impingement syndrome. *Clin Orthop*. 1995;(320):46-54.
19. Paine RM, Voight M. The role of the scapula. *J Orthop Sports Phys Ther*. 1993;18:386-391.
20. Rahme H, Nordgren H, Hamberg H, Westerberg CE. The subacromial bursa and the impingement syndrome. A clinical and histological study of 30 cases. *ACTA Orthop Scand*. 1993;64:485-488.
21. Brewster C, Moynes Schwab DR. Rehabilitation of the shoulder following rotator cuff injury or surgery. *J Orthop Sports Phys Ther*. 1993;18:422-426.
22. Host HH. Scapular taping in the treatment of anterior shoulder impingement. *Phys Ther*. 1995;75:803-812.
23. Inman VT, Saunders JB, Abbott LC. Observations on the function of the shoulder joint. *J Bone Joint Surg Am*. 1944;26:1-30.
24. Jobe FW, Tibone JE, Perry J, Moynes S. An EMG analysis of the shoulder in throwing and pitching. A preliminary report. *Am J Sports Med*. 1983;11:3-5.
25. Kvitne RS, Jobe FW, Jobe CM. Shoulder instability in the overhand or throwing athlete. *Clin Sports Med*. 1995;14:917-935.
26. Kibler WB. Role of the scapula in the overhead throwing motion. *Contemp Orthop*. 1991;22:525-532.
27. Litchfield R, Hawkins R, Dillman CJ, Atkins J, Hagerman G. Rehabilitation for the overhead athlete. *J Orthop Sports Phys Ther*. 1993;18:433-441.
28. Moseley JB, Jobe FW, Pink M, Perry J, Tibone J. EMG analysis of the scapular muscles during a shoulder rehabilitation program. *Am J Sports Med*. 1992;20:128-134.
29. Pink M, Jobe FW. Shoulder injuries in athletes. *Orthop Clin Manage*. 1991;11:39-47.
30. Townsend H, Jobe FW, Pink M, Perry J. Electromyographic analysis of the glenohumeral muscles during a baseball rehabilitation program. *Am J Sports Med*. 1991;19:264-272.
31. Poppen NK, Walker PS. Normal and abnormal motion of the shoulder. *J Bone Joint Surg Am*. 1976;58:195-201.
32. Ludewig PM, Cook TM. Alterations in shoulder kinematics and associated muscle activity in people with symptoms of shoulder impingement. *Phys Ther.* 2000;80:276-289.
33. Paletta GA, Warner JJP, Warren RF, Deutsch A, Altchek DW. Shoulder kinematics with two-plane x-ray evaluation in patients with anterior instability or rotator cuff tearing. *J Shoulder Elbow Surg*. 1997;6:516-527.
34. Lukasiewicz AC, McClure P, Michener L, Pratt N, Sennett B. Comparison of 3-dimensional scapular position and orientation between subjects with and without shoulder impingement. *J Orthop Sports Phys Ther.* 1999;29:574-586.
35. Norkin C, White DJ. *Measurement of Joint Motion: A Guide to Goniometry.* Philadelphia, PA: Davis; 1995.
36. Freedman L, Munro RR. Abduction of the arm in the scapular plane: scapular and glenohumeral movements. *J Bone Joint Surg Am*. 1986;48(8):1503-1510.
37. Portney LG, Watkins MP. *Foundations of Clinical Research: Applications to Practice*. Norwalk, CT: Appleton & Lange; 1993.
38. Ludewig PM, Cook TM, Nawoczenski DA. Three-dimensional scapular orientation and muscle activity at selected positions of humeral elevation. *J Orthop Sports Phys Ther*. 1996;24:57-65.
39. McQuade KJ, Smidt GL. Dynamic scapulohumeral rhythm: the effects of external resistance during elevation of the arm in the scapular plane. *J Orthop Sports Phys Ther.* 1998;27(2):125-133.