Subacromial impingement syndrome: The role of posture and muscle imbalance

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Changes in upper body posture, colloquially termed forward head posture (FHP), are considered to be an etiologic factor in the pathogenesis of subacromial impingement syndrome (SIS). The literature suggests that postural deviations associated with FHP follow distinct patterns involving an increase in the thoracic kyphosis angle and a downwardly rotated, anteriorly tilted, and protracted scapula, which in turn leads to increased compression in the subacromial space. These postural changes are thought to occur concurrently with an imbalance of the musculature, and conservative rehabilitation commonly involves addressing both posture and muscle imbalance. There is a paucity of evidence supporting the hypothesis that posture and muscle imbalance are involved in the etiology of SIS. The purpose of this study was to investigate whether FHP was associated with an increased thoracic kyphosis, an altered position of the scapula; and a reduction in glenohumeral elevation range. Selected sagittal and frontal plane postural measurements were made in 60 asymptomatic subjects and 60 subjects with SIS. The findings suggested that upper body posture does not follow the set patterns described in the literature, and further research is required to determine whether upper body and scapular posture and muscle imbalance are involved in the pathogenesis of SIS. (J Shoulder Elbow Surg 2005;14:385-392.)

Subacromial impingement syndrome (SIS) is a commonly diagnosed condition involving the shoulder and is associated with pain and a loss of function.[9] The etiology of SIS is not understood, and a number of hypotheses have been suggested. Structures and contributing factors have included the acromion,[35] the acromial shape,[3] the os acromiale,[35] the coracoacromial ligament,[49] the superior aspect of the glenoid fossa,[14,21] hypermobility and instability of the glenohumeral joint,[31,54] glenohumeral capsular contraction,[30] rotator cuff tendinitis,[35,36] and intrinsic rotator cuff tendinosis.[5,40,52] It has also been suggested that functional limitations caused by evolutionary changes that have occurred within the human shoulder girdle may also contribute to SIS.[27] Poor upper body posture, colloquially referred to as forward head posture (FHP), is considered to be present when the head is held forward of the shoulders (Figure 1) and has been cited as a potential etiologic factor in the pathogenesis of SIS.[18,47] This is because FHP has been associated with an increase in the thoracic kyphosis angle, a forward shoulder posture (FSP), and a scapula that is positioned in relatively more elevation, protraction, downward rotation, and anterior tilt.[6,18,24] The effect of these changes leads to a loss of glenohumeral flexion and abduction range,[6,18,26] compression and irritation of the uppermost (bursal) surface of the supraspinatus tendon, and a reduction in the range of glenohumeral elevation.[1,6,26,47] Grimsby and Gray[18] have stated the following:

> In a person with good postural alignment, elevation of the arm is free to proceed through a full 160° to 180° of motion without impingement of soft tissues in the subacromial space. In the patient with the classic forward head, rounded shoulders, and increased thoracic kyphosis, the scapula rotates forward and downward, depressing the acromial process and changing the direction of the glenoid fossa. Now as the patient attempts to elevate the arm, the supraspinatus tendon and/or the subdeltoid bursa may become impinged against the anterior portion of the acromion process.

These beliefs concerning posture and muscle imbalance have permeated into medical and physiotherapy clinical practice, are used to explain to patients the basis for pathology and the rationale for rehabilitation, and underpin the importance of the postural and muscle examination for subjects with SIS.[6,24,26,47] However, the evidence to support these theories is limited, with research studies reporting

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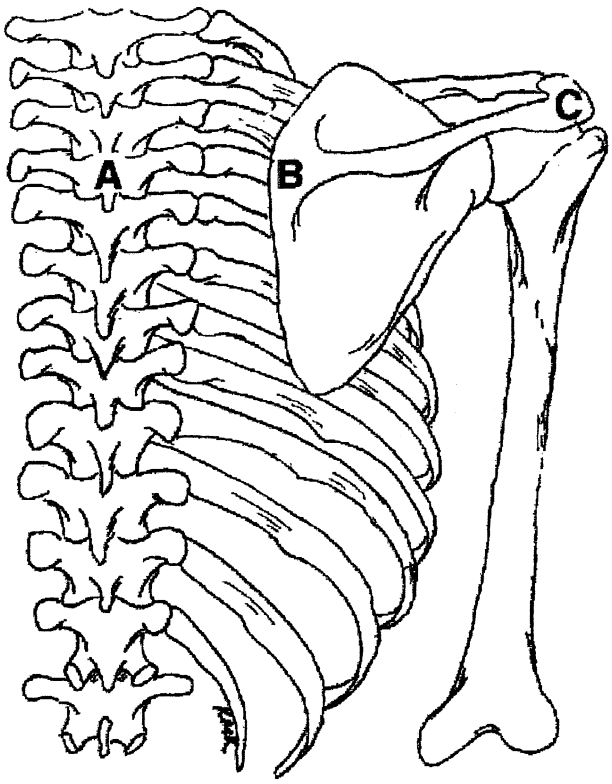

Figure 1 Bony landmarks identified by palpation used to investigate resting position of the scapula. (Reprinted from Lewis J, Green A, Reichard Z, Wright C. Scapular position: the validity of skin surface palpation. Man Ther 2002;7:26-30, with permission from Elsevier.)

equivocal findings.[12,13,15,16,23,27,28,43,44] The purpose of this study was to investigate the relationship between FHP and other selected upper body postural variables, as well as the relationship between posture and glenohumeral joint range of movement, in asymptomatic subjects and subjects with SIS.

The null hypothesis (H_0) for this investigation was that no correlation between FHP and FSP, thoracic kyphosis, scapular protraction, glenohumeral flexion and abduction range, and pain in the symptomatic subjects existed.

MATERIALS AND METHODS

Ethical approval for this study was granted by the Riverside Research Ethics Committee. Subjects were provided with information booklets explaining the purpose of the study and signed informed consent documents before participation.

Power analysis

This study formed part of a randomized, placebo-controlled, same-subject, crossover design investigating the effect of changing posture on selected variables in patients with SIS. The sample size required for the investigation into the effect of postural change in asymptomatic subjects and subjects with SIS for a significance level of .05 and a power of .8 to detect a moderate effect size was calculated to be 60 subjects in each group. The pre-intervention baseline postural measurements provided the data to investigate the postural relationships of interest for this study. The sample size required to detect a moderate correlation (0.3) with a power of .8 and significance level of .05 was calculated to be 85 subjects in each group.

Table I details the inclusion and exclusion criteria for the asymptomatic subjects and for the subjects with SIS.

Table I Inclusion and Exclusion Criteria

The inclusion criteria for asymptomatic subjects were as follows:
- Male and female subjects aged between 18 and 75 y

The inclusion criteria for symptomatic subjects were as follows:
- Male and female subjects aged between 18 and 75 y
- Unilateral shoulder pain for more than 1 wk localized (anterior and/or anterolateral) to the acromion
- Pain produced or increased during flexion and/or abduction of the symptomatic shoulder

Additional criteria for symptomatic subjects included having ≥4 of the following:
- Positive Neer impingement sign[36]
- Positive Hawkins sign[19]
- Pain reproduced during supraspinatus empty-can test[22]
- Painful arc of movement between 60° and 120°[25]
- Pain with palpation on the greater tuberosity of the humerus

The exclusion criteria for both groups of subjects were as follows:
- Health professionals
- Staff members of the institutions where data were collected
- Systemic illnesses
- Pregnancy
- Cervical pain at rest or cervical pain during active cervical movements
- Reproduction of shoulder symptoms during active cervical movements (flexion, extension, left rotation, right rotation, left-side flexion, right-side flexion)
- Reproduction of shoulder symptoms with the addition of over-pressure at the end of range of left and right cervical rotation, left rotation combined with left-side flexion, and right rotation combined with right-side flexion
- History of cervical pain or treatment to this region over the past 12 mo
- History of spinal or upper limb surgery
- History of spinal or upper limb fractures
- Posttraumatic onset of symptoms
- Radiographic evidence of shoulder instability (if available)
- Presence of a positive sulcus sign
- Presence of a positive load-and-shift test[20]
- Presence of a positive active compression labral test[38]
- Presence of clinical signs of acromioclavicular pathology[38]
- Known allergies to taping
- Subjects involved in elite levels of sport

Procedure

Postural measurements were made on the dominant side of the asymptomatic subjects and on the painful side of the subjects with SIS. Male subjects were asked to remove their shirts, and female subjects were asked to wear an open-backed bathing suit. Subjects stood 30 cm in front of a plumb line hanging from the ceiling, with the nondominant

shoulder 20 cm from a plain white wall. Tape was marked on the floor to identify these distances. After palpation, non-allergenic adhesive markers of 6 mm in diameter were attached to the following anatomic points, which were designated with an alphabetic reference (Figure 1):

- the root of the spine of the scapula (point B)
- the posterior aspect of the acromion (point C)
- the thoracic spinous process (SP) corresponding with the root of the spine (point A)
- the lateral midpoint of the humeral head
- the tragus of the ear
- the seventh cervical (C7) SP (to which a 3-cm straw marker was attached through a hole in adhesive felt padding of 3.2 mm in width)

Subjects were then asked to adopt a comfortable and natural standing position. To facilitate this, subjects were informed that during the investigation, it was important that a natural posture would be adopted and that they should pretend that nobody was observing them. The following instructions were given:

> Please stand in a comfortable position that feels natural for you. Now gently and slowly bend your head and neck backwards and forwards three times. Stop in a position that feels natural for you. Now gently swing your arms backwards and forwards by your side, three times. Let them hang comfortably by your side in a position that feels natural for you. Slowly take 3 deep breaths and stand in a posture that feels natural and comfortable for you.

To generate FHP and FSP angles, a lateral photograph was then taken of the cervicothoracic region with an Olympus OM2 SLR camera (Olympus Optical Company, London, England), set at 100 ASA by use of a 28- to 50-mm adjustable lens. The lens aperture was set at F-stop 8. The camera was placed 2 m from the subject and mounted on a tripod. The tripod was leveled with a bubble spirit level to control frontal and sagittal angles. The C7 marker was placed approximately in the center of the lens so as to reduce lens error. The base of the camera was parallel to the ground, and the front of the camera was parallel to the facing wall to minimize parallax error. We used 100-ASA color photographic film (Eastman Kodak Company, Rochester, NY). This procedure has been used in previous published studies.[43,44] Figure 2 depicts how the FHP and FSP angles were determined from the lateral photograph.

After the photograph was taken, the following measurements were each made 3 times with the same standard, non-stretch measuring tape and recorded to the nearest millimeter on the data collection form. The measurements (AC and BC) were the lengths between the landmarks identified in Figure 1. These measurements were used to generate the normalized protraction ratio for the scapula, by dividing the length AC by the length BC. This method of deriving the normalized scapular protraction value has been described in previous studies.[13,15,37] The protraction distance AC is divided by the width of the scapula BC to compare scapular protraction between individuals. If only the AC measurement was used, a larger value in a larger individual would be interpreted as indicating a more protracted scapula than a smaller value in a smaller individual. By use of the normalized protraction ratio, the potential for confounding the findings as a result of the relative size of an individual is reduced. A larger value for the normalized protraction ratio would indicate that the scapula is relatively more protracted.

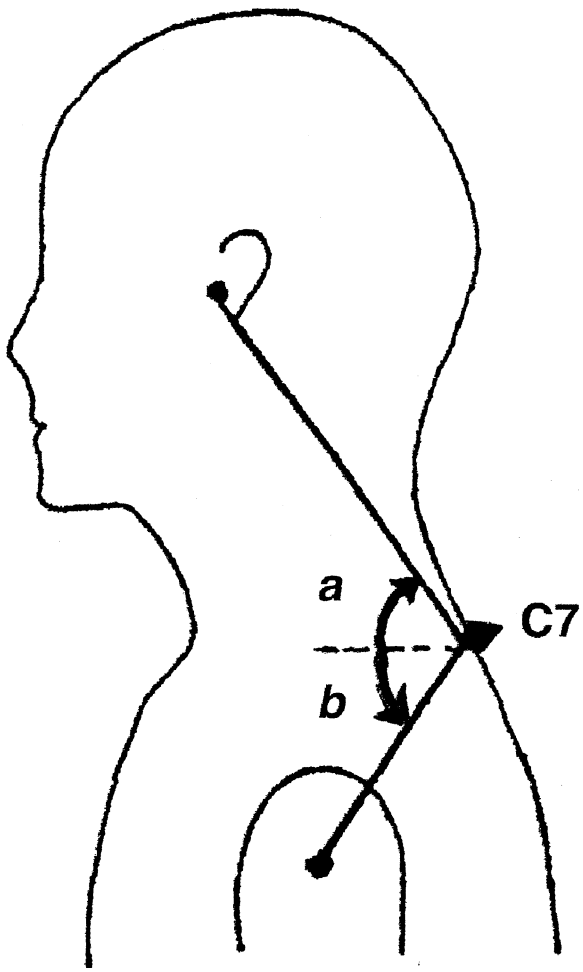

Figure 2 Forward head posture was determined by calculating the angle *a* made between the horizontal and the tragus of the ear from the C7 SP. FSP was determined by calculating the angle *b* made between the horizontal and the midpoint of the shoulder.

Inclinometers (Isomed Inc, Portland, OR) were then placed with their feet over the T1 and T2 SP and the T12 and L1 SP, and the angles were recorded. Each measurement was made 3 times. This method of generating a clinical measurement for thoracic kyphosis has been described previously.[11,39] Figure 3 details how the kyphosis angle was measured.

Two lines, marked with tape on the floor, served as guides for the direction of flexion and abduction of the glenohumeral joint. Flexion was performed in the sagittal plane, and abduction was performed in a plane defined as being 30° anterior of the frontal plane. Each movement was performed 3 times; subjects were asked to elevate their arms to the end of their available range (asymptomatic subjects) or the first point of pain or the first increase in their resting pain (symptomatic subjects). Glenohumeral flexion

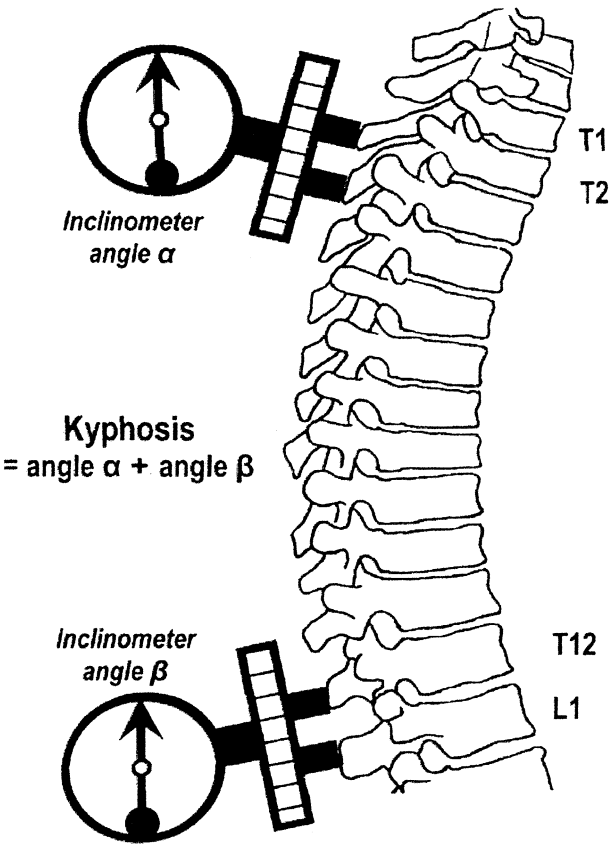

Figure 3 Thoracic kyphosis angle was calculated by the summation of the inclinometer placed over T1 and T2 (angle α) and the inclinometer placed over T12 and L1 (angle β).

range and abduction range were recorded with the inclinometer facing toward the ceiling and with its proximal foot at the insertion of the deltoid.

Measurement reliability

The reliability of the investigator, using the measuring devices to produce the measurements of interest, was investigated in a separate preparatory study of 15 asymptomatic subjects and 15 symptomatic subjects. The same methods and procedures used in the main investigation were tested in this pilot study. The intratester reliability measurements are detailed in Table II.

The results for the intratester reliability study suggested that the measurement reliability for the outcome measurements of interest were acceptable for clinical investigations.[8,41]

Data analysis

Six postural variables (FHP, FSP, thoracic kyphosis angle, normalized scapular protraction, and the ranges of sagittal-plane glenohumeral flexion and abduction in the plane of the scapula) were considered for analysis. The Kendall τ was selected to assess the degree of concordance between the sets of postural variables.[4,7,10,48] Measurements were taken on 3 occasions, and the mean value was used in the data analysis.

Table II Intratester reliability study for measurements of interest used in this investigation

Measurement	95% CI for ICC (2,1)	Single-measure ICC (2,1)	SEM
Asymptomatic subjects (n = 15)			
AC	0.97-0.99	0.98	0.3 cm
BC	0.77-0.97	0.92	0.3 cm
AC/BC	0.75-0.97	0.91	—
Glenohumeral flexion	0.95-0.99	0.98	1.1°
Glenohumeral abduction	0.95-0.99	0.98	1.1°
FHP	0.77-0.98	0.93	1.1°
FSP	0.78-0.99	0.96	1.4°
Thoracic kyphosis			
T1/2	0.91-0.99	0.97	0.9°
T12/L1	0.87-0.98	0.95	1.9°
T1/2 + T12/L1	0.91-0.98	0.96	1.5°
Symptomatic subjects (n = 15)			
AC	0.81-0.98	0.93	0.5 cm
BC	0.85-0.98	0.95	0.3 cm
AC/BC	0.78-0.97	0.92	—
Glenohumeral flexion	0.97-0.99	0.99	2.9°
Glenohumeral abduction	0.97-0.99	0.99	3.3°
FHP	0.77-0.98	0.93	1.1°
FSP	0.78-0.99	0.96	1.4°
Thoracic kyphosis			
T1/2	0.86-0.98	0.95	0.9°
T12/L1	0.87-0.98	0.95	1.9°
T1/2 + T12/L1	0.83-0.98	0.94	2.5°

ICC, Intraclass correlation coefficient; *SEM*, standard error of measurement.

RESULTS

For this investigation, 31 asymptomatic female subjects and 29 asymptomatic male subjects were recruited. There were 2 left hand–dominant and 58 right hand–dominant individuals. The symptomatic group consisted of 25 female subjects and 35 male subjects with SIS. In this group there were 8 left hand–dominant and 52 right hand–dominant individuals. The left shoulder was painful in 25 subjects and the right in 35. The mean duration of symptoms was 1.1 years (SD, 2.5 years) with a range of 2 weeks to 22 years. Further data are detailed in Table III.

Table IV details the results of the statistical analysis of the relationships between the postural variables of interest by use of the Kendall correlation coefficient.

Table III Descriptive statistics for the subjects

	Mean	SD	Minimum	Maximum
Asymptomatic subjects (n = 60)				
Age (y)	34.1	9.9	19	65
Height (cm)	170.9	10.4	150.0	188.0
Weight (kg)	67.8	13.4	43.0	100.0
FHP (°)	50.5	5.2	39.9	66.6
FSP (°)	61.9	10.4	38.4	83.7
Thoracic kyphosis (°)	35.7	8.2	10.0	53.0
Scapular protraction (normalized)	1.53	0.13	1.28	1.83
Glenohumeral flexion (°)	157.3	11.9	133.0	180.0
Glenohumeral abduction (°)	156.1	12.1	131.0	183.0
Symptomatic subjects (n = 60)				
Age (y)	48.9	15.2	19.0	75
Height (cm)	171.2	9.7	149.0	189.0
Weight (kg)	74.5	12.7	52.0	108.0
FHP (°)	47.2	8.4	17.5	59.6
FSP (°)	57.4	9.6	28.1	74.1
Thoracic kyphosis (°)	37.1	7.1	21.0	59.0
Scapular protraction (normalized)	1.53	0.13	1.22	1.93
Asymptomatic-side glenohumeral flexion (°)	155.9	14.7	119.0	185.0
Asymptomatic-side glenohumeral abduction (°)	153.2	14.2	113.0	185.0
Symptomatic-side glenohumeral flexion (°)	120.5	30.9	51.0	172.0
Symptomatic-side glenohumeral abduction (°)	111.3	31.8	39.0	169.0
VAS (pain) glenohumeral flexion	3.1	2.4	0.0	7.9
VAS (pain) glenohumeral abduction	3.3	2.5	0.0	8.9

VAS, Visual analog scale.

Table IV Summary of Kendall correlation coefficient results for relationships between components of posture

	Asymptomatic subjects (*n* = 60)		Symptomatic subjects (*n* = 60)	
Variables (components of posture)	**Kendall correlation**	***P* value**	**Kendall correlation**	***P* value**
FHP/FSP	–0.109	.221	–0.177	.046*
FHP/kyphosis	–0.143	.113	–0.175	.053
FHP/protraction (normalized)	0.172	.054	0.081	.362
FHP/GH flexion	0.043	.632	0.041	.646
FHP/GH abduction	0.077	.392	0.058	.515
Kyphosis/protraction (normalized)	0.125	.164	–0.056	.535
Kyphosis/GH flexion	–0.173	.057	–0.016	.858
Kyphosis/GH abduction	–0.146	.110	0.005	.959

GH, Glenohumeral.
*Significant at .05 level.

DISCUSSION

The key to the clinical examination of posture is commonly described as the assessment of FHP.[24] If FHP is evident, it has been proposed that the clinician may assume that the thoracic kyphosis angle will have increased and the position of the scapula will have been altered, leading to a reduction in glenohumeral range and the potential for pathology.[6,26] The results of this investigation suggest that the null hypothesis cannot be rejected, as the postural patterns described in the literature associated with FHP were not observed. The findings challenge the belief that posture and its concomitant muscle imbalance comprise an etiologic factor in SIS, given the lack of concordance in the postural measurements investigated. The results suggest that neither asymptomatic individuals nor subjects with SIS conform to set deviations of posture led by FHP but that they demonstrate unique and individual patterns. These findings are supported by the conclusions of Raine and Twomey,[44] who examined sagittal and coronal plane postural variables in 160 asymptomatic subjects and reported that no relationship was found to exist between FHP, FSP, and the curve of the thoracic kyphosis. They are also supported by results reported by Greenfield et al,[15]

who found no association between FHP, thoracic kyphosis, and components of scapular position in both asymptomatic and symptomatic subjects. A central tenet of the postural and muscular imbalance model is that there is an ideal posture from which deviations are associated with abnormal joint stress and an imbalance of the surrounding musculature. This is yet to be confirmed in vivo. Grimmer[17] examined FHP in 427 randomly selected asymptomatic subjects. Subjects were examined during unconstrained sitting by use of a custom-built linear excursion–measuring device. The plumb-line measurement described by Kendall et al[24] was defined as the baseline for ideal posture. No subject demonstrated a resting FHP perfectly aligned with the ideal norm (vertical reference line).

In our study the amount of scapular protraction was not found to influence, or be influenced by, other postural or range-of-movement variables. This finding was supported by that of Lukasiewicz et al,[29] who did not find any statistically significant difference in the medial-lateral position of the scapula in asymptomatic subjects and those with SIS. This study did not investigate whether scapular rotation and anterior tilt are postural patterns associated with SIS, and this is acknowledged as a limitation. With respect to other studies that have reported differences in scapular position between asymptomatic and symptomatic subjects,[28,29] it is not known whether these changes have contributed to, or have occurred as a consequence of, the underlying pathology. Static downward scapular rotation has been considered to lead to shoulder pathology.[33] However, after a shoulder girdle strengthening and stretching exercise program, increased glenohumeral abduction was associated with a number of changes, including a more downwardly rotated scapula when the arm was horizontal.[53] Further research is required to determine whether downward rotation is a pathologic position or whether it is possible to function, and not have pathology develop, from a range of static positions of the scapula.

Ideal and symmetrical posture, together with balanced muscles, may not be possible in reality, as large variations in the osseous anatomy of the clavicle, acromion, and humerus have been reported.[2,32,42,45,46,50,51] Postural deviations observed during the clinical examination may, in fact, reflect normal shoulder girdle developmental and osseous asymmetries and may not necessarily indicate postural deviations. The osseous asymmetries would not be correctable by conservative means. Furthermore, postural theorists have described how alterations to the normal position of the scapula will lead to tendinitis[18] or compressive tendinopathy[47] as a result of acromial irritation. This argument is further challenged in cadaveric studies[21] and by studies that have suggested that the primary pathology in SIS is a degenerative, noninflammatory tendinosis of the rotator cuff[5,40,52] and that the articular (non-acromial) side of the tendon is more vulnerable than the bursal (acromial)–side fibers.[34] This finding questions the involvement of the acromion in the pathogenesis of the condition, and hence the relevance of treating posture and muscle imbalance as currently described, to halt the compressive impingement or tendinopathy[1,18,47] as acromial irritation may not be involved in the pathogenesis of the condition. An acknowledged limitation of this study is that the sample size calculation suggested that up to 85 subjects were required for a significance level of .05 and a power of .8 in each group. Large sample size estimates have been reported in previous studies of posture,[44] and future studies investigating postural correlations in larger populations are required.

Conclusion

It has been proposed that postural changes associated with forward-head, kyphotic, or slouched upper body posture may be a cause of SIS or a mechanism capable of perpetuating symptoms. In the absence of trauma or a specific identifiable cause, the relevance of posture is considered to be of greater importance. This is because deviations in posture are thought to coexist with imbalance of the muscle and articular systems, resulting in altered joint position leading to movement dysfunction and pain. Importance is placed on the postural assessment to identify deviations from a normal ideal posture. Once identified, rehabilitation techniques are implemented in an attempt to improve posture and restore normal muscle control. The aim is to reverse the pathologic process by improving posture. Much of the basis for the involvement of posture in SIS is hypothetical and lacks scientific evidence.

The findings of this investigation suggest that static posture in asymptomatic subjects and subjects with SIS does not follow the set pattern referred to extensively in medical, physiotherapy, and osteopathy textbooks and articles. The results suggest that posture may appear to be faulty, yet the individual may be flexible and capable of large ranges of movement. With the drive to provide scientific evidence to underpin the basis of clinical practice, the findings of this study suggest that static sagittal-plane postural assessment has a very limited role in the clinical decision-making process in subjects with SIS. There is a need for further research to advance the understanding of the relevance and involvement of static and dynamic posture and muscle control in the pathogenesis of SIS in larger numbers of subjects.

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