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Active Stiffness and Strength in People With Unilateral Anterior Shoulder Instability: A Bilateral Comparison

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Context: Active muscle stiffness might protect the unstable shoulder from recurrent dislocation.

Objective: To compare strength and active stiffness in participants with unilateral anterior shoulder instability and to examine the relationship between active stiffness and functional ability.

Design: Cross-sectional study.

Setting: University research laboratory.

Patients or Other Participants: Participants included 16 males (age range, 16–40 years; height = 179.4 ± 6.1 cm; mass = 79.1 ± 6.8 kg) with 2 or more episodes of unilateral traumatic anterior shoulder instability.

Main Outcome Measure(s): Active stiffness and maximal voluntary strength were measured bilaterally in participants. In addition, quality of life, function, and perceived instability were measured using the Western Ontario Stability Index, American Shoulder and Elbow Surgeons Standardized Shoulder Assessment Form, and Single Alpha Numeric Evaluation, respectively.

Results: We found less horizontal adduction strength ($t_{15} = -4.092$, $P = .001$) and less stiffness at 30% ($t_{14} = -3.796$, $P = .002$) and 50% ($t_{12} = -2.341$, $P = .04$) maximal voluntary strength in the unstable than stable shoulder. Active stiffness was not correlated with quality of life, function, or perceived instability (r range, 0.0–0.25; $P > .05$).

Conclusions: The observed reduction in stiffness in the unstable shoulder warrants inclusion of exercises in the rehabilitation program to protect the joint from perturbations that might lead to dislocation. The lack of association between active stiffness and quality of life, function, or perceived instability might indicate that stiffness plays a less direct role in shoulder stability.

Key Words: recurrent instability, recurrent dislocations, glenohumeral instability, recurrent glenohumeral dislocations

Key Points

- Shoulder stiffness was less in people with recurrent shoulder instability.
- Differences in stiffness across shoulder joints were not related to quality of life, function, or perceived instability.
- Exercises should be included in the rehabilitation program to protect the joint from perturbations that might lead to dislocation.

Rates of recurrent shoulder instability as high as 94% have been documented in athletes less than 40 years of age.[1,2] Nonoperative treatment remains unsuccessful despite attempts to correct deficits in strength[3–5] and proprioception.[6–8] Indeed, Jakobsen et al[9] reported that 75% of nonoperatively treated patients were dissatisfied with their function because of pain, instability, or recurrence of their injuries.

Some authors[10,11] have suggested that active stiffness might play a role in improving the outcomes of nonoperative treatment. The active stiffness of the shoulder musculature resisting horizontal extension has not been investigated, and this property of muscle might have particular relevance to the person with an unstable shoulder. The rationale behind this belief is that when the unstable shoulder is in an abducted and externally rotated position, in which most subluxations and dislocations occur,[12] the movement of horizontal extension of the humerus is likely to stretch the active anterior shoulder muscles. As an active muscle is lengthened by a transient stretch, the muscle fibers and tendons resist that stretch with a change in force. The ratio of change in force to the change in muscle length is called *muscle stiffness*, and the reciprocal is called *compliance*. A muscle that has less compliance (greater stiffness) will resist a perturbation more effectively. This concept might be important for the functional prospects of people with unstable shoulders because stiffer anterior shoulder muscles would resist more effectively a sudden posterior displacement of the humerus, which occurs during episodes of subluxation when the arm is positioned in an externally rotated position above the shoulder.

Stiffness can be measured with the muscle in a passive or active state; the latter describes the contribution of contractile elements to the stiffness of the muscle. Depending on the level of muscle activation, different components of the muscle will provide greater resistance to stretch.[13] Therefore, active stiffness is not just a function of muscle strength. Testing stiffness with

muscles in an active state provides a measure that is more related to how the shoulder complex might respond if it were subjected to a perturbation that might sublux or dislocate the joint.

Only Myers[14] has investigated active stiffness in people with shoulder instability. He found no difference in active stiffness of internal rotators of the shoulder between groups with and without shoulder instability. One reason for this finding might be that shoulder dislocation commonly occurs because of a combination of external rotation and horizontal abduction.[12] The muscles resisting horizontal abduction (eg, pectoralis major) might be more important than the internal rotators for maintaining stability at the shoulder joint. Myers et al[15] provided evidence that pectoralis major activity is less in unstable shoulders than in stable shoulders during perturbations into external rotation. We wanted to build on the work of Myers.[14] Therefore, the primary purpose of our study was to examine active stiffness of unstable shoulders during a perturbation into horizontal abduction. We hypothesized that active stiffness would be less in shoulders with a history of recurrent instability than in stable shoulders. Our secondary purpose was to investigate the relationship between levels of shoulder stiffness and quality of life, function, and perceived instability. We hypothesized that people with lower levels of stiffness in the shoulder would have lower levels of quality of life and function and higher levels of perceived instability.

METHODS

Experimental Design

We conducted a cross-sectional study to investigate participants with unilateral recurrent shoulder instability and to compare unstable and stable shoulders.

Participants

We posted advertisements in gymnasiums and clinics throughout Auckland and the surrounding area and used word of mouth to recruit participants. People were included if they were male, were aged 16 to 40 years, had a self-reported history of 2 or more episodes of instability (recurrent instability), and had positive apprehension and subluxation/relocation signs, indicating anterior or anteroinferior instability. An *instability episode* was defined as a dislocation necessitating assistance to relocate the shoulder or as a subluxation in which the patient perceived that the humerus moved away from the glenoid fossa with or without associated neural symptoms that prevented movement of the arm after the incident.[16] Exclusion criteria included previous shoulder or cervical spine surgery, bilateral symptoms, pain in the shoulder or cervical spine region at the time of the study, posterior or multidirectional shoulder instability, or other conditions that might alter sensory or motor function (eg, diabetes, rheumatologic disorders, or peripheral nerve disorders). Females were excluded from the study because of the possible influence of sex on muscle stiffness.[17]

Twenty of the 36 people who responded to our advertisements were excluded because they had bilateral symptoms, previous surgery, pathologic elbow conditions, or shoulder pain. The remaining 16 people (age = 21.6 ± 4.6 years, height = 179.4 ± 6.1 cm, mass = 79.1 ± 6.8 kg) met the inclusion criteria and were enrolled in the study. The mean time since injury was 2.67 months (range, 0.5–10 months). All 16 participants were right-limb dominant, and the dominant limb was affected in 9 participants. The *dominant limb* was defined as the arm with which a participant threw. Radiographs and magnetic resonance imaging scans were available for 6 of the 16 participants. The mean number of instability episodes experienced after the initial injury was 7 ± 6 (range, 2–20). The mean level of upper limb activity was 13.8 ± 3.4 as measured by the Brophy Upper Limb Activity score, which has a maximal score of 20 that indicates a high level of activity.[18] Ten participants were involved in organized, officiated contact sports; 2 participants were involved in informal, nonofficiated contact sports; and 4 participants were not involved in contact sports. Four participants were involved in organized, officiated overhead sports; 5 participants were involved in informal, nonofficiated overhead sports; and 7 participants were not involved in overhead sports. With respect to further intervention, 10 participants were awaiting surgery to treat recurrent anterior instability, whereas 6 participants were satisfied with their nonoperative management and were not seeking further treatment.

Written and oral explanations of all experimental procedures were provided. All participants provided informed consent, and the Auckland University of Technology Ethics Committee approved the study.

Shoulder Outcome Scores

The Western Ontario Shoulder Instability Index (WOSI) has been shown to be a valid and reliable measure of quality of life in people with shoulder instability.[19] It has a maximal score of 2100, and a high WOSI score indicates decreased quality of life. Valid and reliable measures of function also have been reported using the patient self-report section of the American Shoulder and Elbow Surgeons Standardized Shoulder Assessment Form (ASES), which has a maximal score of 30.[20] A low ASES score indicates decreased levels of function. A global measure of perceived instability also was recorded using the Single Alpha Numeric Evaluation (SANE), which has a maximal score of 10. For this question, the participants were instructed to indicate the level of instability in their shoulders, and their responses were recorded on a 10-point scale with the anchors 0, *very stable;* and 10, *very unstable*.

Procedures

Participants were tested during a single session in a temperature-controlled laboratory. Initially, participants warmed up on a rowing ergometer for 5 minutes. To assess active stiffness at different percentages of maximal voluntary strength (MVS), a measure of peak torque was needed. Peak torque during horizontal adduction was determined using a dynamometer (Biodex System 3; Biodex Medical Systems, Inc., Shirley, NY). Participants were positioned supine, and their upper arms were strapped to a custom-designed attachment on the arm of the dynamometer at 90° of shoulder flexion and 0° of external rotation (perpendicular to the floor). Straps were placed over the anterior aspect of the clavicle and attached under the axilla to stabilize the scapula and limit movement at the scapulothoracic joint. Additional straps were placed across the trunk and pelvis area to stabilize the pelvis and prevent movement in the trunk (Figure 1). For the strength test, participants were instructed to exert force and attempt to move the elbow to the opposite shoulder. They were taught to limit internal rotation during this movement by focusing on horizontal flexion of the elbow. Next, they performed 2 submaximal isometric contractions at approximately 80% of maximal effort.

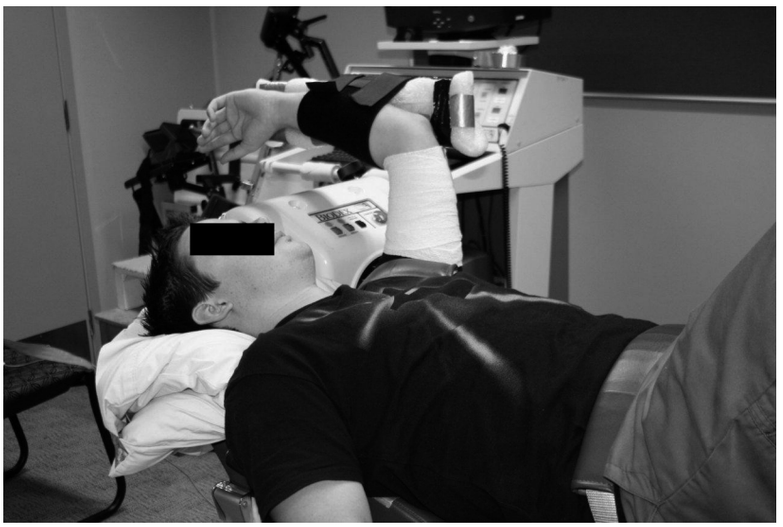

Figure 1. Participant positioned in the dynamometer for strength testing and perturbation testing.

Thereafter, participants performed 3 maximal-effort contractions and rested for 2 minutes between repetitions. The duration of each MVS trial was 6 seconds. Standardized oral encouragement was provided throughout the maximal effort. The highest torque of the 3 trials provided the MVS for later calculations.

Stiffness Testing

Participants were positioned supine with their arms strapped to the arm of the dynamometer as described for strength testing. The unstable limb was tested first. Using the Researcher's Toolkit Software (Biodex Medical Systems, Inc), we programmed the dynamometer to accelerate the limb to a constant angular velocity of 250°/s in 60 milliseconds. This duration was chosen to limit the potential neural responses that can occur with a longer-duration stretch.[21] The angular displacement over the duration was 24°. Torque, angle, and velocity data were recorded simultaneously from the dynamometer at a sampling frequency of 1000 Hz and relayed to a computerized data acquisition system (SuperScope II version 3.0; GW Instruments, Inc, Somerville, MA) for storage and subsequent processing. The torque recorded during this motion was corrected for the effects of gravity and inertia.[14] Thereafter, the difference in torque values at 60 and 0 milliseconds was divided by the change in angle over the same period to provide a value of active stiffness (Nm/°). Participants undertook 2 trials at 3 submaximal levels of MVS (30%, 50%, and 70%). The target torque was displayed on a screen to the experimenter (M.O.) and the participant, and the perturbation trial did not commence until the participant was steady at the required torque level. Participants were instructed to hold the required torque level until the perturbation had finished. Trials were conducted in a random order, and the mean of the 2 stiffness values was used in subsequent analyses.

To ensure that intrinsic stiffness was measured before the onset of muscle reflex activity, surface electromyography (EMG) signals were recorded from the pectoralis major using active electrodes (DE-2.3; Delsys Inc, Boston, MA) with an interelectrode distance of 10 mm, placed on the skin per guidelines of the Surface Electromyography for the Non-Invasive Assessment of Muscles project[22] and other studies.[23,24] The EMG signals were amplified by 1000, band-pass filtered at 3 Hz and 1 kHz, and sampled at 1000 Hz. Root mean square values were calculated over 5-millisecond epochs with a 1-millisecond overlap. The criterion for observing increased electromyographic activity was a rise of 2 standard deviations for more than 5 milliseconds above the level recorded just before the perturbation.[25,26]

The reliability of these procedures was established during pilot testing of 15 healthy participants. Intraclass correlation coefficients (ICCs) were calculated. A 2-way mixed model was used with the mode of assessment (days) as the fixed variable and the participants as the random variable.[27] The ICCs for 30%, 50%, and 70% MVS ranged from 0.91 to 0.96. Based on a 15% difference in stiffness across shoulders for a power of 0.8 and with the α level set at .05, a sample size of 15 participants was needed.

Statistical Analysis

Statistical analysis was undertaken using GraphPad Prism (GraphPad Software, Inc, La Jolla, CA) and SPSS (version 15.0; SPSS Inc, Chicago, IL). The dependent variables of interest were MVS (Nm), active stiffness (Nm/°), quality of life (measured with the WOSI), function (measured with the patient self-report section of the ASES), and perceived instability (measured with the SANE). The appropriateness of using parametric analysis was determined by analyzing descriptive statistics. Data were checked with the Grubbs test to identify outliers and with the Kolmogorov-Smirnov test to identify abnormalities in the distribution (ie, kurtosis and skewness). We found no differences. Two-tailed dependent *t* tests were used to examine differences across shoulders for each percentage of MVS. Effect sizes were calculated using the equation presented by Kazis et al[28] and classified according to Cohen.[29] Pearson product moment correlations were used to examine the associations between strength deficits, active stiffness (normalized to maximal strength), quality of life, function, and perceived instability. For all statistical analyses, the α level was set a priori at .05.

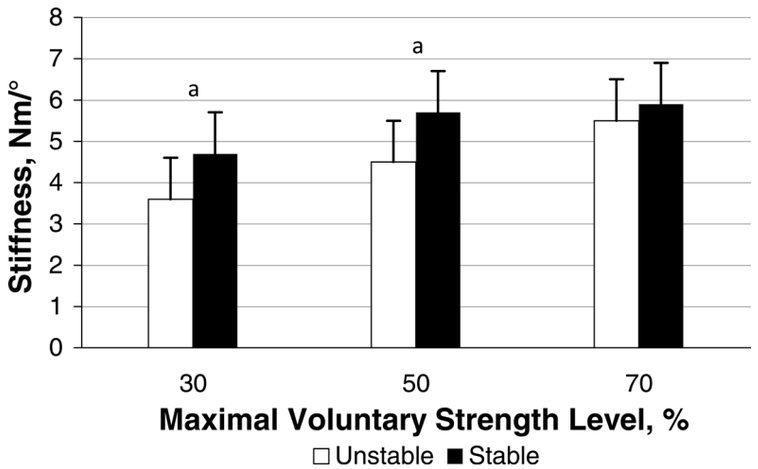

Figure 2. Active stiffness in the stable and unstable shoulders at 30%, 50%, and 70% maximal voluntary strength. [a] Indicates difference between limbs ($P < .05$).

RESULTS

Shoulder Outcome Scores

The mean WOSI score was 923 ± 456.34 (range, 296–1642; 44%). The mean ASES score for the unstable shoulders was 22.7 ± 4.8 (range, 12–28; 76%) and for the stable shoulders was 30 ± 0.0 ($t_{15} = –6.057$, $P = .001$). The mean score for the SANE was 4.5 ± 2.9 (range, 2–9.5). Limb dominance was not related to any of the outcome scores ($P > .05$).

Strength Test

We found a difference between isometric horizontal adduction strength in the stable and unstable shoulders ($t_{15} = –4.092$, $P = .001$). The mean was 39.8 ± 14.1 Nm in the unstable shoulders and 45.1 ± 14.1 Nm in the stable shoulders. We found no association between strength deficits in the unstable shoulders and the WOSI ($r = 0.191$, $P = .48$), ASES ($r = –0.326$, $P = .22$), or SANE ($r = 0.567$, $P = .22$). In addition, because all participants were right-limb dominant and we did not find a difference between percentage deficit in those affected on the right or left side ($t_{14} = 0.857$, $P = .41$), no relationship existed between limb dominance and strength.

Active Stiffness Test

We found a difference between stable and unstable shoulders at the 30% ($t_{14} = –3.796$, $P = .002$) and the 50% ($t_{12} = –2.341$, $P = .04$) MVS levels (Figure 2). No effect was found at the 70% MVS level ($t_{11} = –0.90$, $P = .39$). The mean values of stiffness in the stable and unstable shoulders at 30% MVS were 4.7 ± 1.1 Nm/° and 3.6 ± 1.7 Nm/°, respectively (effect size = 0.79). At the 50% MVS level, the mean values were 5.7 ± 1.7 Nm/° and 4.5 ± 1.2 Nm/° for the stable and unstable shoulders, respectively (effect size = 0.82). Under the Cohen[29] classification system, these can be regarded as moderate to large effects. The mean values of stiffness for stable and unstable shoulders at 70% MVS were 5.9 ± 0.17 Nm/° and 5.5 ± 0.1 Nm/°, respectively. We found no associations between normalized stiffness values and the WOSI, ASES, or SANE (R range, 0.0–0.25; $P > .05$).

Up to 58 milliseconds after the perturbation, EMG levels recorded from the pectoralis major were not different from those recorded in the 100 milliseconds before the perturbation. At 58 milliseconds, increased EMG activity of 2 standard deviations was noted. Because stiffness measurements were calculated over a 60-millisecond epoch, the increased activity observed at 58 milliseconds was not thought to be influential, particularly when consideration was given to electromechanical delay.

DISCUSSION

Some authors[10,11] have suggested that active stiffness might be a factor influencing the presence of recurrent shoulder instability. However, Myers[14] found no difference in active stiffness of the internal rotators of stable and unstable shoulders. Given that horizontal abduction also is implicated in occurrences of dislocation, we examined active stiffness of the horizontal adductors. No previous investigators have examined active stiffness of these muscles.

Our participants represented the typical male with shoulder instability. Quality-of-life scores as measured by the WOSI score (mean = 923 ± 456.34; 44%) were comparable with those from other studies.[12,30] Limitations in function as measured by the ASES score (mean = 22.7 ± 4.8; 76%) were also similar to those reported by other authors.[20] The stiffness values we reported are similar to those reported by Zhang et al,[31] who perturbated the shoulders of young males into horizontal abduction. However, our stiffness values differed from those of Myers,[14] who examined the active stiffness of the internal rotator muscles in participants with stable and unstable shoulders. We believe the larger muscle size of the pectoralis major relative to the subscapularis was the key component responsible for the greater stiffness values we observed.

We observed a lower level of stiffness in unstable shoulders at 30% and 50% MVS but no difference at 70% MVS. These findings suggest that less protection from such perturbations is provided in the unstable joint at lower levels of muscle activa-

tion. Although the mechanisms behind the loss of stiffness cannot be determined directly from our methods, several factors might be responsible or might contribute.

Authors[17,32] have documented damage and subsequent increased length in the capsule and ligaments of the glenohumeral joint after dislocation. The muscles involved in providing stability around the shoulder also have attachments to the capsule and ligaments.[33] At low levels of activation, resistance to horizontal abduction might be reduced and lead to decreased stiffness. At higher levels of muscle activation, these muscles might be able to increase stiffness in the system.[34] Alternatively, more muscles might be involved in resisting motion at higher levels of contraction. At low levels of muscle activation, stiffness might be provided predominantly by the rotator cuff musculature. Stronger muscles, such as the pectoralis major, might be activated at the higher level of muscle contraction and might provide increased stiffness.

Another reason for the absence of a difference in stiffness at the 70% MVS level might be the effect of cocontraction about the joint. Akazawa et al[35] examined stiffness in the human thumb and found that stiffness increased with an increase in cocontraction of the flexor and extensor muscles. Similarly, Louie and Mote[36] showed that quadriceps-hamstring cocontraction can reduce knee laxity and increase stiffness at the joint. At the low levels of muscle activation in our study, cocontraction might have been limited; however, at 70% MVS of muscle activation, increased cocontraction might have led to similar amounts of stiffness across shoulders. In the future, researchers might use EMG to measure levels of activation.

The technique we used to measure stiffness does not enable delineation of the particular components of the muscle that might contribute to active stiffness. However, based on the Hill[37] model of muscle contractile elements, the series elastic components and the parallel elements probably are implicated. *Series elastic components* refer to the tendon and aponeurosis that lie in series with contractile tissue, whereas *parallel elastic tissue* denotes connective tissue around the muscle, epimysium, perimysium, and endomysium. Parallel tissue forms the basis of tension resistance in a noncontractile state,[38,39] whereas the actin-myosin filaments form the basis of tension resistance in a contractile state. In addition, the role of the joint receptors to mediate muscle stiffness at a joint via the gamma muscle spindle system can affect the level of stiffness about the joint.

Therefore, although differences in stiffness were apparent across shoulder joints, these were not related to quality of life, function, or perceived instability. This was an unexpected finding. We hypothesized that the relationship between active stiffness and these outcomes would be positive. This hypothesis was based on previous work in the unstable knee,[40] in which investigators examined stiffness in the sagittal plane and found a positive association with function. However, movement is much more restricted at the knee than at the shoulder, with knee movement occurring primarily in 1 plane and most shoulder movement occurring across 3 planes during work and recreation. Therefore, active stiffness measures in 1 plane of motion might be too limited to reflect the complex nature of the musculotendinous system providing stability at the shoulder joint during physical tasks. Although no relationship between stiffness and function was noted, the observed loss of stiffness to the horizontal adductors means that less protection is present when the joint is perturbated unexpectedly in this plane of motion. Therefore, we recommend exercises that would remedy this deficit and reduce the chance of a dislocation.

Maximal isometric peak torques of the horizontal adductors (mean = 45.1 ± 14.1 Nm) were similar to those reported by other researchers investigating these muscles in stable shoulders.[41,42] We observed a deficit in horizontal adduction strength. Many researchers have detailed decreased[3,4,43] or altered[5] strength in external and internal rotation, but no other researchers have examined horizontal adduction strength in people with recurrent shoulder instability. Although increased strength in the dominant limb of athletes has been documented,[44,45] all participants were right-hand dominant, and the dominant arm was affected in 56% of participants. Because we did not detect an association between strength and limb dominance, our findings are unlikely to be confounded by the effect of limb dominance. Determining whether a specific muscle is more affected than others by the contribution of numerous muscles to strength in this plane of motion is difficult. However, because many shoulder tasks in sport and work require motion combined with large of amounts of force in this plane, recommending strengthening exercises to remedy the observed deficits and thereby to improve performance in such tasks seems logical.

CONCLUSIONS

Although we noted differences across strength and stiffness, the relevance is questionable given the lack of a relationship between active stiffness and quality of life, function, and perceived instability. The lack of a relationship between active stiffness and these factors suggests that other factors, to which stiffness might contribute, play a greater role in allowing people with instability to return to their work and sporting activities. The multidimensional nature of shoulder movement might require a limit to the amount of stiffness allowed if shoulder movements are to be undertaken efficiently and effectively.

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