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# Which shoulder motions cause subacromial impingement? Evaluating the vertical displacement and peak strain of the coracoacromial ligament by ultrasound speckle tracking imaging

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**Background:** Subacromial impingement is a common cause of shoulder pain and one cause of rotator cuff disease. We aimed to identify which shoulder motions cause subacromial impingement by measuring the vertical displacement and peak strain of the coracoacromial ligament using ultrasound speckle tracking imaging.

**Methods:** Sixteen shoulders without shoulder disability were enrolled. All subjects were men, and the average age was 28.6 years. The vertical displacement and peak strain of the coracoacromial ligament were analyzed by the motion tracing program during the following active assisted motions (active motion controlled by the examiner): (1) forward flexion in the scapular plane, (2) horizontal abduction in the axial plane, (3) external rotation with the arm at 0° abduction (ER0), (4) internal rotation with the arm at 0° abduction (IR0), (5) internal rotation with the arm at 90° abduction (IR90), and (6) internal rotation at the back (IRB).

**Results:** The mean vertical displacement of the coracoacromial ligament during forward flexion (2.2 mm), horizontal abduction (2.2 mm), and IR90 (2.4 mm) was significantly greater than that during the other motions (ER0, −0.7 mm; IR0, 0.5 mm; IRB, 1.0 mm; $P < .003$). The mean peak strain was significantly higher in forward flexion (6.88%), horizontal abduction (6.58%), and IR90 (4.88%) than with the other motions (ER0, 1.42%; IR0, 1.78%; IRB, 2.61%; $P < .003$).

**Conclusions:** Forward flexion, horizontal abduction, and IR90 showed higher vertical displacement and peak strain of the coracoacromial ligament, causing subacromial impingement. It is recommended that patients with impingement syndrome or a repaired rotator cuff avoid these shoulder motions.

Institutional Review Board approval for the study protocol was provided by Seoul St. Mary's Hospital, The Catholic University of Korea: No. KC13OISI0679. Written informed consent was obtained from every subject.

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**Keywords:** Coracoacromial ligament; impingement; in vivo; displacement; strain; 2D speckle tracking echocardiography

Subacromial impingement syndrome is a common cause of shoulder pain and disability. In 1972, Neer first reported subacromial impingement syndrome.[16] He indicated that the coracoacromial ligament and the anterior third of the acromion are responsible for subacromial impingement of the rotator cuff. After this description, many investigators examined the clinical importance of impingement syndrome. Bigliani et al described the association of bone morphology of the acromion with rotator cuff disease, and Nyffeler et al emphasized that large lateral extension of the acromion is related to rotator cuff tear.[4,18] It is well known that subacromial impingement is a major extrinsic factor in rotator cuff diseases, although intrinsic factors, such as degeneration, microtrauma, oxidative stress, and tendon apoptosis, might be initiating factors.[4,8,14,17] Several recent studies have suggested that subacromial bursal cells may be activated to secrete proinflammatory cytokines, such as substance P, stromal cell–derived factor 1, and vascular endothelial growth factor, by various stimuli, such as subacromial impingement, and that highly concentrated proinflammatory cytokines might result in subacromial bursitis in rotator cuff disease.[5,10,11,28] Therefore, acromioplasty has traditionally been performed as a routine part of rotator cuff repair.[6,21,23] MacDonald et al designed multicenter randomized controlled trials to compare clinical outcomes of rotator cuff repair with and without acromioplasty.[15] They concluded that there was no significant difference in functional scores between the 2 groups; however, they did find a strong trend toward a greater number of reoperations in the nonacromioplasty group. Furthermore, Bigliani et al demonstrated that insufficient subacromial decompression might be a cause of failed rotator cuff repair.[3]

Many modalities are used to evaluate shoulder motions that reproduce subacromial impingement. Wu et al calculated the displacement of the coracoacromial ligament using dynamic ultrasonography in shoulders with rotator cuff tears during passive and active shoulder abduction and internal rotation.[25] They observed greater displacement of the coracoacromial ligament in torn rotator cuffs than in intact ones during passive shoulder abduction and internal rotation. Yamamoto et al designed a cadaveric study to evaluate the contact pressure between the coracoacromial arch and rotator cuff tendons during several shoulder motions.[27] Specific shoulder motions, such as flexion, horizontal abduction, and extension, resulted in higher contact pressure than other motions did. These previous studies show that specific shoulder motions are likely to cause subacromial impingement. However, few studies have explored the in vivo conditions of the coracoacromial ligament during these shoulder motions. Some studies were performed to evaluate the in vivo properties of the coracoacromial ligament, but only during limited shoulder motions.[24-26]

The purpose of this study was to analyze the in vivo vertical displacement and intraligamentous strain of the coracoacromial ligament, which might reflect the presence of subacromial impingement during various shoulder motions. We hypothesized that subacromial impingement occurs in the normal shoulder and that specific shoulder motions are more closely related to subacromial impingement.

## Materials and methods

This was an ultrasonographic observational study to analyze the in vivo vertical displacement and intraligamentous strain of the coracoacromial ligament during various shoulder motions. Eight healthy volunteers with no history of shoulder pain were enrolled for a total of 16 shoulders. All were men, and the average age was 28.6 years. All enrolled volunteers were Korean; the height ranged from 170 to 180 cm, and all were right handed. The exclusion criteria were as follows: patients younger than 18 years, a history of shoulder pain or trauma, and any pathologic findings during ultrasonographic examination. A single orthopedic surgeon (Y.-S.K. with 10 years of experience in ultrasonographic examination) examined all shoulders using 2-dimensional speckle tracking echocardiography (2D STE). During static ultrasonography, the examiner assessed structures including the biceps tendon, rotator cuff, and subacromial bursa with routine ultrasonographic examination maneuvers. Dynamic ultrasonography with the motion tracking program was used to evaluate the vertical displacement and peak strain of the coracoacromial ligament during the following motions: (1) forward flexion in the scapular plane, (2) horizontal abduction in the axial plane, (3) external rotation with the arm at 0° abduction (ER0), (4) internal rotation with the arm at 0° abduction (IR0), (5) internal rotation with the arm at 90° abduction (IR90), and (6) internal rotation at the back (IRB). All shoulder motions were evaluated during active assisted motion with an average velocity of 22.5°/s. During ultrasonographic examination, the examiner assisted the subjects in actively moving their arms at a constant velocity and precise direction that means active assisted shoulder motions. Forward flexion was performed with the arm in the neutral position and flexed in the scapular plane from 0° to 90° because ultrasonographic tracing of the coracoacromial ligament during forward flexion above 90° was technically difficult. Horizontal abduction was defined as backward movement of the arm from 0° to 45° with 90° of