INTRODUCTION:
Superior labrum anterior-posterior (SLAP) lesion is a common injury for throwers as well as increased anterior shoulder laxity. Andrews et al.1 reported that in baseball throwers SLAP lesions were often associated with shoulder ligamentous laxity and that thermal capsulorraphy following SLAP repair improved the long term result of SLAP repair. However there is no biomechanical study of effects of SLAP lesion with increased shoulder ligamentous laxity followed by SLAP repair.

The relationship between SLAP lesion and increased shoulder joint laxity or instability is unclear. Previous cadaveric studies by Pagnani et al.2 and Burkart et al.3 showed a significant increase in shoulder joint laxity after creation of a type II SLAP lesion. In these studies the creation of a type II SLAP lesion was performed by cutting the superior labrum at the glenoid insertion. The effect of a surgically created type II SLAP lesion may be different from that of an actual type II SLAP lesion. The purpose of this study was to biomechanically assess the effect of pathologically created type II SLAP lesion and arthroscopic repair. The pathologic type II SLAP lesion was created by incremental nondestructive stretching of the glenohumeral joint capsule in humeral external rotation (Figure 1).

RESULTS:
External rotation was significantly increased by 20.8 ± 2.1º after excessive external rotation. Following SLAP repair external rotation decreased by only 4.0 ± 0.7º. Anterior translation was significantly increased at 60º/90, 60º/0, and 30º/0 (2.4 ± 0.7, 1.5 ± 1.0 and 1.2 ± 0.4 mm) after excessive external rotation and decreased only at 30º/0 (1.0 ± 0.2 mm) after repair (Figure 3, 4). Inferior translation was significantly increased at 60º/90, 60º/0, and 0º/0 (1.5 ± 0.5, 0.7 ± 0.3, and 1.3 ± 0.3 mm, respectively) after excessive external rotation and decreased at 60º/0 and 0º/0 position (0.8 ± 0.2 and 1.3 ± 0.3 mm) after repair.

DISCUSSION:
All pathologically created SLAP lesions were associated with elongation of the anterior capsular ligaments (anterior shoulder ligamentous laxity). Pathologically created SLAP lesion with elongated anterior capsular ligaments resulted in a significant increase in external rotation, and anterior and inferior glenohumeral translations at both lower (30º for anterior and 0º for inferior) and higher (60º) glenohumeral abduction. However SLAP repair only partially restored external rotation and restored anterior and inferior glenohumeral translations only at lower abduction. Therefore for throwers, the increased external rotation and increased anterior and inferior shoulder joint laxity at 90º shoulder abduction (60º glenohumeral abduction) may be due primarily to elongation of the anterior capsular ligaments.

 Clinically SLAP lesions produced by excessive external rotation, as seen in throwers, may be associated with anterior shoulder ligamentous laxity. Therefore some SLAP lesion patients may have symptoms of instability at 90º shoulder abduction (positive apprehension test). However this instability may result not from a SLAP lesion but from elongated anterior capsular ligaments. Also for SLAP lesion with shoulder instability or pathologic laxity at 90º of shoulder abduction, anterior shoulder ligamentous laxity should be addressed in addition to SLAP repair.

METHODS:
Six cadaveric shoulders were tested using a custom shoulder testing system and Microscribe (Immersion Corp, San Jose, CA) (Figure 2). To create excessive humeral external rotation the shoulders were nondestructively stretched 20% beyond maximum external rotation in 60º of glenohumeral abduction. After the excessive external rotational stretching, the size of the superior labral sulcus was increased (more than 2mm in depth), creating pathologic type II SLAP lesion for all specimens. The anterior glenohumeral capsular ligaments were also elongated. The pathologically created type II SLAP lesions were then repaired arthroscopically using two suture anchors (Arthrex, Naples, FL) placed anterior and posterior to the biceps tendon. Rotational ROM was measured at 60º of glenohumeral abduction while anterior and posterior translations were measured at 60º abduction and 90º external rotation (60º/90), 60º abduction and neutral rotation (60º/0), and 30º abduction and neutral rotation (30º/0). Inferior and superior translations were measured at 60º/0 and 60º/0 positions as well as at 0º abduction and neutral rotation (0º/0). Neutral rotation was defined as the center point of total (External + Internal) rotational ROM. Anterior–posterior loading at 60º/0 and 30º/0 position simulated a load and shift test and inferior loading at 0º/0 position simulated the sulcus test. Data were analyzed using repeated measures analysis of variance followed by Tukey’s post hoc test (p < 0.05).

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