June 22, 2015

Dear Editor,

Please find enclosed the edited manuscript in Word format (file name: 17041-review.doc).

**Title: SLAP lesions of the shoulder - current diagnostic and therapeutic standards**

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**Name of Journal:** *World Journal of Orthopedics*

**ESPS Manuscript NO:** 17041

The manuscript has been improved according to the suggestions of reviewers:

1 Format has been updated.

2 Revision has been made according to the suggestions of the reviewer (modifications in red letters).

(1) page 5:
description of the different biceps tendon attachments has been removed

(2) page 5:
*The Buford complex….*
An appropriate citation has been added

(3) page 6:
*Chronic/degenerative SLAP lesions*
Reference to detailed description within the following paragraphs added.
"…which will be described more detailed in the following."

**"Internal impingement**
Some clinical and cadaveric studies [26, 27] demonstrated that contact occurs between the undersurface of the postersuperior rotator cuff and the posterosuperior part of the glenoid in abduction and external rotation both in symptomatic and asymptomatic shoulders. In overhead athletes internal posterosuperior impingement sometimes gets pathologic and has been associated with partial-thickness articular surface tears of the deep side of the rotator cuff and lesions of the labro-bicipital complex resulting from repetitive microtrauma due to recurrent overhead motion under extreme loading conditions [28]. This leads to anterior microinstability caused by gradual stretching of anterior capsulolabral structures and consecutive aggravation of internal impingement [27].

**Peel-back mechanism**

*Burkhart et al.* [24] disagreed with the hypothesis of internal impingement provoking shoulder pathologies in the overhead athlete and presented a pathologic cascade leading to SLAP lesions with peel-back mechanism as an important factor.

The posteroinferior capsular contraction is assumed to be the point of origin over the course of the above-mentioned cascade because glenohumeral rotation center migrates to a posterosuperior position with consecutive relaxation of the anteroinferior capsule. Now hyperexternal rotation is possible by reason of minimized cam effect of the proximal humerus and greater tuberosity clearence off the glenoid rim resulting in a magnified arc of external rotation before internal impingement appears.

The peel-back phenomenon [29] has its origin in a biceps vector change in the position of abduction and external rotation resulting in torsional forces to the labrobicipital complex. Once fatigue failure of the posterior superior labrum is nascent, it will start to progressively rotate medially over the upper rim of the glenoid. In cocking position, peel-back forces are at a maximum and additional shearing forces arise during throwing cycle from core energy which is transmitted to the shoulder [24]."

(4) page 6

*GIRD*

Detailed description added.

**"Glenohumeral rotation deficit**

Alteration in rotational magnitude has been identified as a potential risk factor in developing shoulder injuries by changing normal glenohumeral kinematics [12].

Overhead athletes frequently present asymmetrically decreased glenohumeral internal rotation on the dominant side which is considered to be part of positive adaptation to improved force development in conjunction with increased external rotation. The total rotational range of motion usually remains symmetric. Bony changes, capsuloligamentous factors and muscular components have been related to affect glenohumeral range of motion [13].

When exceeding beyond certain dimensions, alterations in glenohumeral internal rotation and total rotational range of motion can obtain clinical relevance. Side-to-side asymmetries greater than 5° in total rotational range of motion are denoted TROMD (total rotational range of motion deficit). GIRD (glenohumeral internal rotation deficit) is defined as a side-to-side asymmetry in glenohumeral internal rotation greater than 18° [14]. Both findings are implicated in increased risk of shoulder injuries by modifying normal glenohumeral kinematics [15].

(5) page 6:

"switches" has been replaced by "migrates".

(6) page 7:

*kinetic chain – scapular dyskinesia*

Definition of kinetic chain has been added. Dyskinesia has been replaced by dyskinesis.

**"Kinetic chain**

Overhead action consists of a complex series of sequential coordinated motions to achieve appropriate body position and motion, and to develop required muscle activity. According to *Kibler et al.* [14], the kinetic chain meets the following requirements: linking multiple body segments into one functional segment [16], providing a stable base for distal arm mobility, maximizing force development of the core and transferring it to the hand, interaction of distal joints to increase force and energy capability and decrease distal joint load [17], and reduction of deceleration forces by producing torques [18].

Deficits in kinetic chain components have been shown to be associated with shoulder injuries in baseball players and tennis players [19]. To maintain the same energy at ball impact in case of a 20% reduced provision of trunk kinetic energy, 33% more velocity or 70% more mass in distal segments was necessary in mathematical calculations [20].

Kinetic chain alterations become clinically significant by identifying components of non-shoulder deficits in shoulder injury pathogenesis, even though occurrence and mechanisms in the course of injury sequence remain unclear [14].

**Scapula dyskinesis**

The scapula occupies a central position in basic movement patterns of the shoulder and there is strong evidence of scapular kinematic alteration contributing to a variety of shoulder pathologies [21]. Scapula performance is essential to maintain functional interaction with the humerus for efficent motion, joint stabilisation, muscular capability and control [22].

By definition, scapular dyskinesis characterizes the alteration of normal kinematics and reflects the loss of normal control of scapular motion [23].

Subsequent internal rotation and anterior tilt can lead to increased tensile strain on the anterior ligaments, enhance the peel-back mechanism of the labro-bicipital complex and give rise to a pathologic internal impingement [24].

The acronym SICK (Scapular malposition/inferior medial border prominence/coracoid pain/dyskinesis of scapular movement) was used by *Burkhart et al.* [25] to characterize a pattern of scapular abnormality in the disabled overhead athlete shoulder. The excessive scapular protraction leads to glenohumeral hyperangulation in external rotation increasing strain to the undersuface of the posterior rotator cuff area and the anterior-inferior capsular structures, which can intensify the peel-back mechanism [25]."

(7) page 10:

*clinical tests*

Utility of clinical tests has been demonstrated on the basis of current literature. Table 2 has been added.

"In order to determine utility of clinical tests in physical examination of the shoulder, *Hegedus* [37] presented a systematic review with meta-analysis. Among the traditional SLAP tests, relocation test showed best sensitivity; best specificity was found in Yergason´s test. The compression-rotation test presented the best positive predictive value. (Table 2).

More recent tests seem to be encouraging but warrant further investigation. The passive compression test [38] showed a sensitivity of 81.8% and a specificity of 85.7%. The positive predictive value was 87.1%, and the negative predictive value was 80.0%.
The modified dynamic labral shear demonstrated sensitivity of 72%, specificity of 98%, accuracy of 0.84, and a positive likelihood ratio of 31.57 [39]. Combinations of clinical tests provide higher accuracy, in case of labral lesions, the best combination was identified to be the modified dynamic labral shear test and O'Brien's maneuver.
As clinical tests are a key element in diagnosing SLAP lesions, there is still a great need for further studies to improve the diagnostic conclusion and allow the shoulder surgeon to be more efficient in making a firm diagnosis."

(8) page 10:
reference added

(9) page 16:
*SLAP and repair*
Remark concerning the flaws of cited articles has been added.
„Overall, reported results after SLAP repair are non-homogeneous and uniform recommendations can not be imposed as a general rule for specific surgical treatment, resulting in variable rates of return to preinjury level. This might be based on multiple confounding variables not consistently accounted for including differences between studies in population demographics, surgical details related to surgical technique, surgeon experience, hardware used, and post-surgical rehabilitation parameters."

(10) page 17:
*SLAP and tenodesis*
conclusions have been softened
"Study validity is lessened by a relatively small sample size and the nonrandomized design."
"tenodesis of the biceps tendon may be an appropriate alternative with proven benefit to the patient"
"mini-open tenodesis above the upper border of the great pectoral tendon is an alternative in cases with absent signs of fresh injury [77]."

3 References and typesetting were corrected. PubMed citation numbers and DOI citation have been added so lang as available.

Thank you again for publishing our manuscript in the *World Journal of Orthopedics*.

Sincerely yours,

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