

Conservative treatment preferences and the plausible mechanism of Neer's stage 1 of shoulder impingement in younger people

Keramat Ullah Keramat

Abstract

The interaction of various factors in the vicious cycle (VC) of subacromial impingement syndrome (SIS) is complex and there are conservative treatment preferences for speedy rehabilitation. The mechanism of SIS is not fully understood and the inappropriate treatment priorities cause delay in rehabilitation. SIS is related to the reduction in the subacromial space (SS). Posterior capsular tightness (PCT) and rotator cuff (RC) dysfunction are the two basic mechanisms in this regard. PCT may cause anterosuperior translation of humeral head (HH) and anterior acromion tipping through scapular dyskinesia, thereby reducing the subacromial space. Alteration in the force couple of muscle secondary to scapular dyskinesia eventually causes injury to subacromial structures. The rotator cuff is important in centring the humeral head in the glenoid cavity and superior translation of humeral head occurs if their function is compromised. Posterior capsular tightness may affect the function of rotator cuff action which leads to early fatigue, dysfunction of these muscles and eventually impingement. Adhesive changes take place in various structures around the shoulder secondary to impingement pain and relative immobility, which further aggravates the problem. To reverse the vicious cycle, conservative intervention should therefore be directed to loosen posterior capsular tightness, restore rotator cuff function through appropriate exercise in a pain-free range through appropriate exercise, mobilisation of adhesion through taser-assisted friction or pain-free mobilisation or grade I and grade II manipulation. Depending on the level of irritability, pain control intervention could be considered alongside.

Keywords: Vicious cycle, Subacromial impingement syndrome, Posterior capsular tightness.

Introduction

Shoulder complaints rank third after back and knee problems in outpatient appointments and reports of its

.....
Department of Physiotherapy, St. Patrick's Hospital, Carrick-on-Shannon, Co. Leitrim, Ireland.

Correspondence: Email: karamatjee@yahoo.com

prevalence range from one-in-three people at some stage of their lives to more or less half the population experiencing at least one episode of shoulder pain in a year.^{1,2} The subacromial impingement syndrome (SIS) refers to a condition in which the tendons of the rotator cuff muscles or the subacromial bursa or long head of biceps are entrapped between the humeral head (HH) inferiorly and anterior acromion superiorly.^{2,3} The pathology of rotator cuff (RC) failure is multifactorial and results from a combination of intrinsic, extrinsic and environmental factors.⁴ In recent literature, SIS has been described as a group of symptoms rather than a specific diagnosis⁵ and the causative factors are identified as trauma, daily wear and tear, repetitive use, age-related degeneration, RC dysfunction, scapular muscle weakness and dysfunction, imbalance between the concentrically activated agonist and eccentrically activated antagonist, posterior capsular tightness (PCT),^{6,7} glenohumeral instability, postural abnormalities and scapular dyskinesia.⁷⁻¹¹

The current review considers PCT of the glenohumeral joint as a major factor along with RC dysfunction which triggers the vicious cycle (VC) of events in SIS. The purpose of this review was to present an evidence-based review of the key elements of vicious circle of SIS and to identify interventional strategies to rehabilitate and restore the function of the shoulder with SIS.

Mechanism of SIS

General consideration

It is a common clinical experience that immobilisation causes stiffness and ligament contracture in a normal healthy joint. It is frequently observed that immobilisation after wrist fracture or elbow injury frequently causes shoulder joint capsular and ligament contracture. These ligament contractures are clearer in the region of less stretch e.g. flexed joint remains flexed after immobilisation in flexion over period of time.^{10,11} It appears that ligament shortness on one side of the joint may affect the articular movement mechanism and may cause abnormal translation of the articular surfaces. Repeated overload induces changes in a ligament and it gets stronger and hypertrophied.¹²⁻¹⁵ A stronger and hypertrophied

ligament may contract more after immobility and can cause articular translation which results in undue stress on the soft tissue, disturb the force couple of multiple prime movers, imbalance, early fatigue and loss of stabilising action of the designated stabilising muscle of the joints and may lead to injury eventually. In the author's clinical experience, the translation is often trivial and cannot be observed during clinical examination but repeated movement might produce symptoms.

A- Posterior glenohumeral capsular tightness

During arm elevation in the healthy shoulder, the HH remains centred and rotates downward in the glenoid cavity while the scapula rotates superiorly (around horizontal axis perpendicular to it), internally (around a vertical axis through its plane) and tilt posteriorly (around horizontal axis in the plane of it).¹⁵ Healthy RC muscles and lax posterior and inferior capsule¹⁶ are prerequisites for the glenoid centred rotation of HH during elevation. Glenoid-centred rotation of HH is hindered by the failure of each of them. On the contrary, anterosuperior translation may occur if the RC is dysfunctional or posterior and inferior parts of shoulder capsule is contracted.¹⁷ Simultaneous limitation of internal rotation is the well-known feature of posterior capsule where HH translates anteriorly and produces painful symptoms.¹⁷ A variety of other alterations occur (Figure-1) that are discussed below.

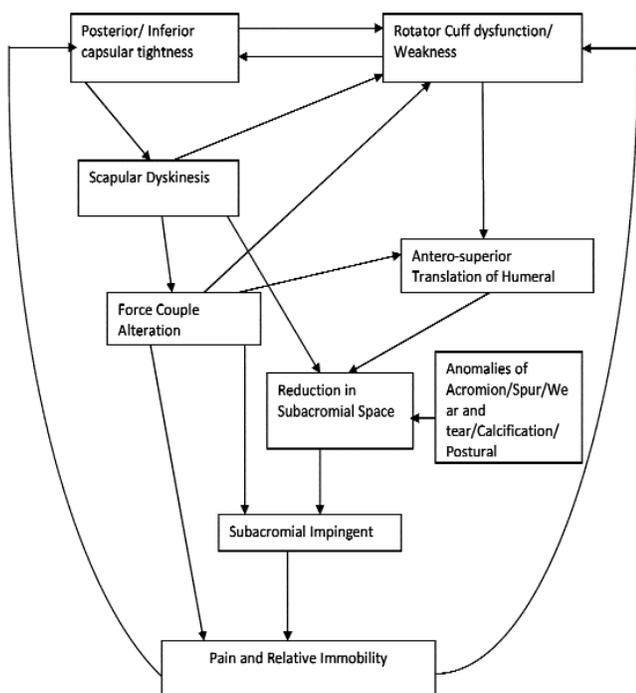


Figure-1: Complex interaction of key components in the vicious cycle of subacromial impingement syndrome.

1- Internal Rotation and Reaching Up Behind The Back Movement

Glenohumeral internal rotation deficits (GIRDs) are consistent problems seen during the examination of overhead athletes.¹⁸ The deficits in internal rotation are attributed to the tight posterior capsule.^{18,19} Reaching up behind the back (RUBTB) is an important functional movement and difficulty in this movement is the frequent complaint in patients with SIS. It is a composite of three movements; extension and internal rotation at the shoulder and flexion of the elbow. Range of this movement varies from person to person and side to side; usually limited internal rotation and more external rotation on the throwing side in overhead athletes. During the RUBTB movement in a healthy shoulder, the HH rotates internally in the glenoid cavity and the scapula reaches to its end limits of available ranges, manifest variable features from person to person and shoulder to shoulder.

RUBTB is frequently assessed in clinical examination against the anatomical landmarks. Although various researchers do not consider it a true measure of the internal rotation of glenohumeral joint,²⁰ yet it is an important functional movement that sum up the contribution of glenohumeral and scapulothoracic articulation in internal rotation and may reflect the true end range of internal rotation. The relative contribution of glenohumeral and scapulothoracic articulation is approximately a 2:1 ratio²⁰ in this movement and maximum internal rotation (66%) occurs in bringing the arm up to the sacrum from neutral hanging position.²¹ The rest of the internal rotation (34%) hasn't been fully explored, but the scapular movement seemingly plays an important role as it is most often very prominent from sacrum to T12 level.

Isolated active internal rotation or extension of shoulder is often pain-free in most of the impingement cases, but combination of these movements in RUBTB nearly always produces anterior shoulder pain. The tightness in the capsule and its adhesion with the fascia is probably allowing the uni-planar movement, but opposes the torsional stress combined in RUBTB movement. Furthermore, it appears that the HH occupies a relative superior position in the glenoid cavity in neutral side hanging position in chronic SIS and additional upward translation takes place during the elevation, reaching to the maximum at around 90 degrees and subsequently irritates the tendon of long head (LH) of biceps and the injured RC.

RUBTB is a useful movement to assess the true status of posterior capsular tightness. It simultaneously affects

glenohumeral internal rotation¹⁷ and scapular kinematics,²² to a variable degree and often cannot be appreciated during clinical observation despite its presence. The scapula moves abnormally to compensate for the deficit in glenohumeral internal rotation. When both the scapula and glenohumeral movement reaches their available limit, further attempts at reaching up behind the back put undue stress on the anterior structures, causing anterior shoulder pain and discomfort.

2- Scapular Dyskinesis

The synchronous movement of the scapula with the arm is necessary for the execution of normal humeral movements. The dysfunction of the synchronous movement of the scapula is termed scapular dyskinesia and is described as a non-specific response to a painful condition in a shoulder.²²⁻²⁵ However, in the author's clinical experience, the alteration in scapular movement is specific to posterior capsular tightness. This dyskinesia is always present in a typical pattern during arm elevation and manifested as delayed upward rotation (around horizontal axis perpendicular to the plane of scapula) and posterior tilt (around a horizontal axis in the plane of the scapula) while early internal rotation (around a vertical axis through the plane of the scapula)^{24,25} (Figure-2). Reversal of this position of scapula significantly increases



Figure-2: Reduced subacromial space due to fixed pattern of scapular dyskinesia in subacromial impingement syndrome i.e. delayed upward rotation (around horizontal axis perpendicular to the plane of scapula) and posterior tilt (around a horizontal axis in the plane of the scapula) and early internal rotation (around a vertical axis through the plane of the scapula).

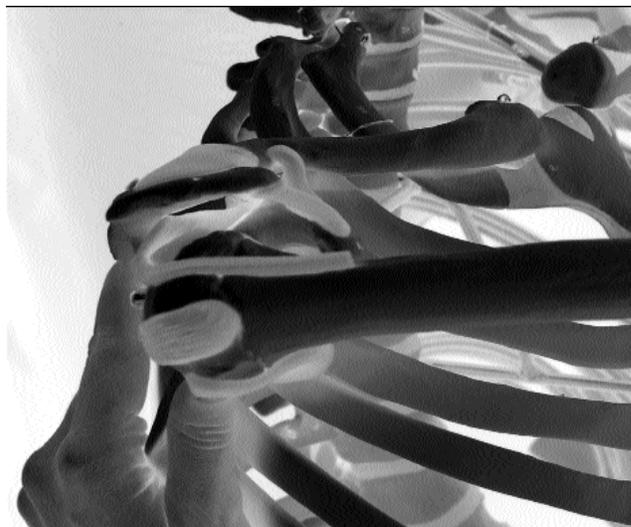


Figure-3: Increased subacromial space by the correction of scapular dyskinesia.

the subacromial space (SS) (Figure-3). Person-to-person variations of the shape of ribcage, bulk of soft tissues around the scapula and side-to-side variation of soft tissue make the clinical appreciation of this movement pattern difficult to observe in the acute stage, but is marked during chronic stage. Though trivial in nature, this position of the scapula brings the anterior acromion very close to the HH at 60-90 degree of elevation/abduction in which arc pain is present and probably is the most likely cause of impingement. The upward translation of HH during elevation/abduction is present in healthy shoulders and may not be the only cause of impingement.

3- Force couple in elevation

The altered position of the scapula alters the force couple of muscle groups, both at glenohumeral and scapulothoracic articulation. In deltoid/rotator cuff force couple, deltoid is overactive and the rotator cuff is underactive in elevation of the arm. Similar alterations in force couple of upper and lower trapezius and dysfunction of serratus anterior muscles occur which decreases the Subacromial space.²⁵ This causes excessive superior translation of the HH²⁶⁻²⁸ and repeated movement leads to the injury of longhead of biceps and the rotator cuff tendon itself. Serratus anterior is underactive and upper trapezius is overactive in upward scapular movement (upward, internal and posterior tilt) during the elevation.²⁹ The resultant motion of the scapula is desynchronised with HH motion and the acromion process does not clear the HH underneath. Force couple alteration may also be present in anterior, middle and posterior fibres. The altered resting position of spinous and lateral acromion may produce trivial length

difference with the anterior, middle and posterior deltoid fibres and it will produce a desynchronised pull during elevation. Repeated movement may produce pain in the deltoid muscle itself.

Differences in muscle length due to the typical form of scapular dyskinesis are likely to be present and seem to be the exact cause of the dysfunctional force couple action. The force couple acting at the scapulothoracic and glenohumeral joint is disturbed and abnormal HH translation is facilitated. This mechanism can be readily observed in chronic shoulder impingement pathologies during arm elevation. The HH appears to be locking against the acromion at 60-90 degrees during elevation due to the excessive translation/acromion tipping, although magnetic resonance imaging (MRI) may report intact RC. Passive elevation in such a scenario is often possible beyond 90 degrees without significant pain.

4- Muscle flexibility

Chronic pathology of the shoulder affects the length of surrounding muscles. Coracoid-based pectoralis minor and levator scapulae muscles are usually tight. Their tightness keeps the scapula in a protracted position³⁰ and contributes to the VC of events in SIS. A degree of shortness in the full muscle or part of the muscle may be present in serratus anterior. Other muscles such as trapezius (upper and lower), rhomboids, pectoralis minor and major, subclavius and latissimus dorsi muscles may be tight and needs to be addressed in the rehabilitation process.

B- Rotator cuff failure

The function of the RC is complex. The major function of the RC is to rotate the HH within the glenoid cavity of the scapula. Deltoid is the major abductor of the arm and causes the superior translation of the HH from neutral hanging position to 90-degree abduction due to its angle of pull. The superior translation is opposed by the synchronised contraction of RC muscles²⁶ and, to some extent, by the LH of biceps^{31,32} so that the HH rotates in the opposite direction in the glenoid cavity during elevation/abduction. RC muscle contribution in abduction is much essential in the range of 60-90 degrees of abduction³² and their dysfunction in this range predisposes to impingement^{34,35} of these structures themselves.

A stable base is necessary for the best performance of RC muscles. All the RC muscles arise from the scapula and the dyskinesis of the scapula alters the length of these muscles. As a result, their combined action is desynchronised and optimum performance at 60-90 degrees of elevation/abduction is compromised and impingement may occur. Once injury to the RC tendon or

LH of biceps occurs, a VC of events sets in.³⁶ The longer the history of the symptoms, the more pronounced are the changes at glenohumeral and scapulothoracic articulation. Uninterrupted events in the VC usually lead to a partial or full thickness tear of RC tendon or LH of biceps and becomes a challenge for the rehabilitation specialist.

C- Anatomic anomalies and degenerative changes

The height of normal subacromial space, from the coracoacromial arch to the HH, is only 1-1.5cm as seen on radiograph³⁶ and is occupied by the supraspinatus tendon, subacromial bursa, LH of the biceps tendon, and the capsule of the shoulder joint. The amounts of HH anterior and superior translations, ranging from 1mm to 5mm, seem insignificant when compared to normal available space. However, because of the typical pattern of scapular dyskinesis^{22,23} which causes the anterior acromion tipping and subsequent reduction in space combined with the presence of the subacromial structures, there is little room for "error".¹

Anatomic anomalies of acromion, presence of subacromial spurs, degenerative changes at acromio-clavicular joint, calcification and postural anomalies contribute to the reduction in SS and are the secondary factors which put the individual at greater risk of impingement.

D- Sources of pain in impingement

Chronic impingement is associated with multiple sources of pain. The primary sources of pain in SIS are the tendon of RC, LH of biceps and subacromial bursa. In the acute stage, pain is localised to the involved structure and is aggravated by elevation/abduction of the arm. Pain at rest in the acute stage might indicate inflammation of the structure involved.²

The relative immobility or malfunction of the shoulder over a period of time produces changes in muscles and ligaments at glenohumeral, acromioclavicular and scapulothoracic joint. Ligaments and muscles get shorter and produce pain on movement. Adhesion occurs at various levels. Adhesive changes affect the mobility of the bicep tendon within the bicipital groove and its synovial sheath becomes the secondary source of pain. Adhesion also takes place between the posterior capsule and the fascia of external rotators³⁸ and presents a challenge in diagnosis and the rehabilitation process. Muscle spasm take place due to primary pain and prolonged inactivity in upper and middle trapezius, Rhomboids, belly of subscapularis and serratus anterior, pectoralis major and minor, and these muscles become tender. The excessive tension in these muscles often affects the vascular and nerve components in

chronic cases and imitates radicular pain.

Disturbances in force coupling action of deltoid/RC, within deltoid (anterior, middle and posterior fibres) or serratus anterior/trapezius may be the cause of pain active movement.

Treatment priorities

Posterior and inferior capsular tightness, RC dysfunction, pain and adhesion are the four key elements in the VC of impingement and should be addressed during the rehabilitation of chronic SIS. Posterior capsular tightness is one of the prime reasons of shoulder impingement and should be addressed during rehabilitation and prehabilitation. Poor techniques are commonly used to stretch the posterior capsule. To perform the manoeuvre, the patient can be positioned either in sitting or side lying and the clinician can then stabilise the scapula in maximum protraction and medial rotation through one hand or with the assistance of an assistant. The affected arm is then brought in slight abduction and extension, followed by stretch along the axis of humerus and passive medial rotation in small amplitude. Immediate reassessment of the patient active movement is mandatory. This manoeuvre frequently produces dramatic outcomes. It is advisable to educate the patient regarding the problem.

Strengthening exercises programme should be started simultaneously but in a pain-free range. Isometric resisted in neutral position of the arm or isometric co-contraction exercises (simultaneous contraction of all muscles) in overhead (>120 degree of elevation/abduction) for a short time (10-15 seconds) position are effective in the early phase of rehabilitation, followed by Kibler's⁹ exercise regime to gain neuromuscular control. Gradual progression to advance close chain or open chain exercises are added if the symptoms are improving.

Adhesions are present at various sites as describe above and should be mobilised accordingly. Transverse friction and manipulation (grades I & II) in this regard are the treatments of choice.

Occupational interventions and life style modification may be desirable in some chronic and resistant to treatment conditions while strict immobilisation or aggressive mobilisation are not recommended.³⁹

A successful management of SIS can only be accomplished through a better understanding of the most common biomechanical deficits and effective treatment techniques which requires thorough understanding of the mechanism of SIS. For the treatment to be effective, the entire kinematic chain needs to be

address rather than a specific aspect of the SIS.⁴⁰

Limitations

The main focus of this article is the secondary intrinsic factors involved in the VC of events in SIS as a result of faulty kinetics and kinematics. These secondary events are commonly ignored or poorly addressed during the rehabilitation of SIS. The review does not thoroughly address the primary extrinsic factors, older age-related shoulder pathologies, suprascapular nerve lesion, inflammatory, septic conditions and the main focus is on Neer's stage 1 of SIS while steering clear of Neer's stage 2 and 3. Shoulder instability is also out of the scope and the clinicians are advised to rule out the red-flag conditions during the assessment of shoulder.

Conclusion

Posterior and inferior capsular tightness is one of the prime causes of SIS. It is the likely cause of scapular dyskinesia and subsequent acromion tipping, and simultaneous anterosuperior translation of the HH. The altered position of the scapula alters the force couple action of deltoid/RC muscles and serratus anterior/trapezius. Repeated elevation/abduction movement in 60-90 degree range in the presence of reduced SS aggravates/causes injury to subacromial structures. Acromion anomalies, subacromial spur, calcification and degenerative changes contribute to the reduction of SS. Presence of pain sets up a VC of events. The relative immobility of the shoulder over a period of time causes various changes. These changes are dysfunction of RC muscles, adhesion between the joint capsule and muscle fascia, adhesive changes around the LH of biceps, contraction of the shoulder joint capsule, shortening in certain muscles and generalised muscle weakness. The rehabilitation programme should therefore include loosening of the posterior and inferior capsule, mobilisation of adhesion, scapular stability and appropriate analgesia if the pain is constant, followed by restoration of the strength of RC muscles in pain-free range. Stretching of posterior capsule or RC strengthening alone may not be appropriate. An RC strengthening programme alone may be hazardous in chronic impingement. Mobilisation of adhesions at posterior shoulder and LH of biceps in the bicipital groove should be included in the rehabilitation process.

Acknowledgement

Thanks are due to Ailing Goughran, Senior physiotherapist, for editorial assistance.

References

1. Reilingh ML, Kuijpers T, Tanja-Harfterkamp AM, van der Windt DA. Course and prognosis of shoulder symptoms in general practice. *Rheumatology (Oxford)*. 2008;47: 724-30.

2. Greving K, Dorrestijn O, Winters JC, Groenhof F, van der Meer K, Stevens M, et al. Incidence, prevalence, and consultation rates of shoulder complaints in general practice. *Scand J Rheumatol*. 2012;41:150-5.
3. Neer CS, 2nd. Impingement lesions. *Clin Orthop Relat Res*. 1983 ;173:70-7.
4. Lewis JS. Rotator cuff tendinopathy. *Br J Sports Med*. 2009;43:236-41.
5. Cools AM, Declercq G, Cagnie B, Cambier D, Witvrouw E. Internal impingement in the tennis player: rehabilitation guidelines. *Br J Sports Med*. 2008 ;42:165-71.
6. Silva RT, Hartmann LG, Laurino CF, Biló JP. Clinical and ultrasonographic correlation between scapular dyskinesia and subacromial space measurement among junior elite tennis players. *Br J Sports Med*. 2010;44:407-10.
7. Atalar H, Yilmaz C, Polat O, Selek H, Ura? I, Yanik B. Restricted scapular mobility during arm abduction: implications for impingement syndrome. *Acta Orthop Belg*. 2009;75:19-24.
8. Wilk KE, Macrina LC, Reinold MM. Non-Operative Rehabilitation for Traumatic and Atraumatic Glenohumeral Instability. *N Am J Sports Phys Ther* 2006;1:16-31.
9. Kibler WB. Rehabilitation of rotator cuff tendinopathy. *Clin Sports Med*. 2003;22:837-47.
10. Andersen LL, Holtermann A, JÃrgensen MB, SjÃgaard G. Rapid muscle activation and force capacity in conditions of chronic musculoskeletal pain. *Clin Biomech (Bristol, Avon)*. 2008;23:1237-42..
11. Reiman MP, Lorenz DS. INTEGRATION OF STRENGTH AND CONDITIONING PRINCIPLES INTO A REHABILITATION PROGRAM. *Int J Sports Phys Ther* 2011;6:241-53.
12. Pastor MF, Smith T, Struck M, Wellmann M. [Stability versus mobility of the shoulder. Biomechanical aspects in athletes]. *Orthopade*. 2014;43:209-14.
13. Nakagawa S, Yoneda M, Mizuno N, Hayashida K, Yamada S, Sahara W. Influence of posterior capsular tightness on throwing shoulder injury. *Knee Surg Sports Traumatol Arthrosc*. 2013;21:1598-602..
14. Gaber S, Zdravkovic V, Jost B. [The throwing shoulder]. *Orthopade*. 2014 ;43:223-9.
15. Kirshhoff C, Imhoff AB. Posterosuperior and anterosuperior impingement of the shoulder in overhead athletes-evolving concepts. *Int Orthop*. 2010; 34: 1049-58.
16. McClure PW, Michener LA, Sennett BJ, Karduna AR. Direct 3-dimensional measurement of scapular kinematics during dynamic movements in vivo. *J Shoulder Elbow Surg*. 2001;10:269-77.
17. San Juan JG, Kosek P, Karduna AR. Humeral head translation after a suprascapular nerve block. *J Appl Biomech*. 2013; 29: 371-9.
18. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology Part I: pathoanatomy and biomechanics. *Arthroscopy*. 2003; 19: 404-20.
19. Gerber C, Werner CM, Macy JC, Jacob HA, Nyffeler RW. Effect of selective capsulorrhaphy on the passive range of motion of the glenohumeral joint. *J Bone Joint Surg Am* 2003; 85-A: 48-55.
20. Bourne D, Choo A, Regan W, MacIntyre D, Oxland T. Accuracy of digitization of bony landmarks for measuring change in scapular attitude. *Proc Inst Mech Eng H*. 2009; 223: 349-61.
21. Wakabayashi I, Itoi E, Minagawa H, Kobayashi M, Seki N, Shimada Y, et al. Does reaching the back reflect the actual internal rotation of the shoulder? *J Shoulder Elbow Surg* 2006; 15: 306-10.
22. Kibler WB, Sciascia A. Current concepts: scapular dyskinesia. *Br J Sports Med* 2010; 44: 300-5.
23. Kibler WB, Ludewig PM, McClure PW, Michener LA, Bak K, Sciascia AD. Clinical implications of scapular dyskinesia in shoulder injury: the 2013 consensus statement from the 'Scapular Summit'. *Br J Sports Med* 2013; 47: 877-85.
24. Shinohara H, Urabe Y, Maeda N, Xie D, Sasadai J, Fujii E. Does shoulder impingement syndrome affect the shoulder kinematics and associated muscle activity in archers? *J Sports Med Phys Fitness*. 2014; 54: 772-9.
25. Lopes AD, Timmons MK, Grover M, Ciconelli RM, Michener LA. Visual Scapular Dyskinesia: Kinematics and Muscle Activity Alterations in Patients With Subacromial Impingement Syndrome. *Arch Phys Med Rehabil* 2015; 96: 298-306.
26. Sharkey NA, Marder RA. The rotator cuff opposes superior translation of the humeral head. *Am J Sports Med* 1995; 23: 270-5.
27. Chen SK, Simonian PT, Wickiewicz TL, Otis JC, Warren RF. Radiographic evaluation of glenohumeral kinematics: a muscle fatigue model. *J Shoulder Elbow Surg* 1999; 8: 49-52.
28. Paletta GA Jr, Warner JJ, Warren RF, Deutsch A, Altchek DW. Shoulder kinematics with two-plane x-ray evaluation in patients with anterior instability or rotator cuff tearing. *J Shoulder Elbow Surg* 1997; 6: 516-27.
29. Ludewig PM, Cook TM. Alterations in shoulder kinematics and associated muscle activity in people with symptoms of shoulder impingement. *Phys Ther* 2000; 80: 276-91.
30. Tate A, Turner GN, Knab SE, Jorgensen C, Strittmatter A, Michener LA. Risk factors associated with shoulder pain and disability across the lifespan of competitive swimmers. *J Athl Train* 2012; 47: 149-58.
31. Inman VT, Saunders JB, Abbott LC. Observations of the function of the shoulder joint. 1944. *Clin Orthop Relat Res*. 1996; 330: 3-12.
32. Peat M. Functional anatomy of the shoulder complex. *Phys Ther*. 1986; 66: 1855-65.
33. Bjelle A, Hagberg M, Michaelson G. Occupational and individual factors in acute shoulder-neck disorders among industrial workers. *Br J Ind Med*. 1981; 38: 356-63.
34. Chopp JN, O'Neill JM, Hurley K, Dickerson CR. Superior humeral head migration occurs after a protocol designed to fatigue the rotator cuff: a radiographic analysis. *J Shoulder Elbow Surg* 2010; 19: 1137-44.
35. Myers JB, Hwang JH, Pasquale MR, Blackburn JT, Lephart SM. Rotator cuff coactivation ratios in participants with subacromial impingement syndrome. *J Sci Med Sport*. 2009; 12: 603-8.
36. Fyhr C, Gustavsson L, Wassinger C, Sole G. The effects of shoulder injury on kinaesthesia: A systematic review and meta-analysis. *Man Ther* 2015; 20: 28-37.
37. Papadonikolakis A, McKenna M, Warme W, Martin BI, Matsen FA 3rd. Published evidence relevant to the diagnosis of impingement syndrome of the shoulder. *J Bone Joint Surg Am*. 2011; 93: 1827-32.
38. Yoneda M, Nakagawa S, Mizuno N, Fukushima S, Hayashida K, Mae T, et al. Arthroscopic capsular release for painful throwing shoulder with posterior capsular tightness. *Arthroscopy*. 2006; 22: 801 e1-5.
39. Diercks R, Bron C, Dorrestijn O, Meskers C, Naber R, de Ruitter T, et al Dutch Orthopaedic Association. Guideline for diagnosis and treatment of subacromial pain syndrome: a multidisciplinary review by the Dutch Orthopaedic Association. *Acta Orthop*. 2014; 85: 314-22.
40. Kennedy DJ, Visco CJ, Press J. Current concepts for shoulder training in the overhead athlete. *Curr Sports Med Rep*. 2009; 8: 154-60.