EVALUATION AND TREATMENT OF SHOULDER INJURIES

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INTRODUCTION

Shoulder injuries are quite common in tennis players. These injuries can occur as a consequence of a trauma, but most of the injuries in tennis can be defined as overuse injuries (chronic injuries) coming from the repetitive micro-trauma inherent in the sport[1]. Since they are overuse in causation, preventative strategies can be considered to improve the athlete's abilities to withstand the micotrauma. Shoulder pain or shoulder injury is associated with kinetic chain dysfunction, scapular dyskinesis and glenohumeral internal rotation deficit (GIRD)[2]. The repetitive stressors and loading sequences in tennis create muscular imbalances specific to the sport that requires preventative interventions believed to lower injury risk. This article will discuss the mechanism these injuries in tennis players, will discuss the treatment of these injuries and will highlight the prevention strategies that can be applied for players.

EPIDEMIOLOGY AND MECHANISM OF TENNIS SHOULDER INJURIES

The upper extremity is one of the most frequently injured region in tennis players[3]. In the 2013 ATP World Tour shoulder injuries (10% of all injuries) were in 4th position after spine (26%), thigh muscles (13%) and foot/ankle (11%) injuries. Sallis et al found that there is no significant difference in injury rate between men and women. The most common specific diagnoses in tennis players with shoulder pain are 'impingement', 'rotator cuff tendonitis' and labral injuries. All of these injuries are thought to be related to alterations in kinetic chain function. Specific alterations in the kinetic chain create the potential for injuries in the shoulder. The most common alterations are kinetic chain dysfunction, scapular dyskinesis and GIRD.

Kinetic chain function and dysfunction

The overhead throwing motion is developed and regulated through a sequential co-ordinated and task-specific kinetic chain of force development and a kinematic chain of sequential body positions and motions. The kinetic chain has several functions:

1. using integrated programmes of muscle activation to temporarily link multiple body segments into one functional segment (e.g. the back leg in cocking stance and push-off, the arm in long axis rotation prior to ball release or ball impact) to decrease the degrees of freedom in the entire motion,
2. providing a stable proximal base for distal arm mobility,
3. maximising force development in the large muscles of the core and transferring it to the hand,
4. producing interactive moments at distal joints that develop more force and energy than the joint itself could develop and decrease the magnitude of the applied loads at the distal joint and
5. producing torques that decrease deceleration forces.

The clinical implications of the use of the kinetic chain for performance have been demonstrated in several studies. The tennis serve motion can be evaluated by analysing a set of six 'nodes' or positions and motions that are correlated with optimum biomechanics. These key positions have been correlated with optimum force development and minimal applied loads, and can be considered the most efficient methods of co-ordinating kinetic chain activation. In tennis, maximum speed of the racquet at ball impact is correlated most highly with the velocity of the back hip going from its lowest position in cocking to its highest position in follow through. The fastest tennis serves are associated with a muscle activation pattern that utilises the back leg muscles pushing up and through ball impact.

Clinical implications for injury risk have also been demonstrated. A mathematical model showed that a 20% reduction in trunk kinetic energy development resulted in a requirement of 33% more velocity or 70% more mass in the distal segments to maintain the same energy at ball impact. A tennis study showed that not adequately flexing the knees in the cocking phase while serving resulted in a 17% increase in shoulder load and a 23% increase in elbow valgus load if resultant ball velocity was maintained at speeds comparable to players flexing the knees. Decreased hip range of motion is associated with shoulder injury and poor mechanics. Scapular dyskinesis is associated with rotator cuff disease, impingement and internal impingement. These findings highlight the fact that the shoulder joint frequently is the 'victim' with the kinetic chain dysfunction becoming the 'culprit' so that the evaluation of the anatomical injury with clinical findings affecting the 'shoulder girdle' must include kinetic chain alterations.

Core stability

The dynamic scapulo-thoracic stability and the importance of the 'core stability' give the ideal hint to understand those mechanisms that when altered can lead to 'shoulder dysfunction'. The shoulder is a complex mechanical structure containing several joints connecting the humerus, the scapula, the clavicle and the sternum. The relation between the rotations of humerus and scapula is commonly referred to as the 'scapulo-humeral rhythm'. The scapular motion strongly affects the 'mechanical energy' delivered by muscles and the 'metabolic cost' required to obtain the desired force. At the same time the scapula has different roles being a functional part of the glenohumeral joint, retracting and protracting along the thoracic wall and elevating the acromion. It is a site for muscle

attachments and a link in the proximal to distal sequencing of velocity, energy and force that allows the most appropriate shoulder function[4].

The 'core' is where the centre of gravity is located and where movement begins. An efficient core allows for maintenance of the physiological length-tension relationship of functional agonists and antagonists, and for normal force-couple in the lumbo-pelvic hip complex. The musculoskeletal core of the body includes the spine, the hips, the pelvis, the proximal lower limb and abdominal structures; muscles of the trunk and pelvis are responsible for the maintenance of stability of the spine and are critical for the transfer of energy from large to small body parts during many work/sports activities. The thoraco-lumbar fascia is an important structure that connects the lower limbs (via the gluteus maximus) to the upper limbs (via the latissimus dorsi). Core stability is essential for the maximum efficiency of the shoulder function. Core muscle activation is used to generate rotational torques around the spine and provides stiffness to the entire central mass, making a rigid cylinder that confers a long lever arm around which rotation can occur and against which muscles can be stabilised as they contract[9].

Scapular dyskinesis

Scapular dyskinesis, one of the most important abnormalities in scapular biomechanics, is actually the loss of the 'link function' in the kinetic chain. If the scapula does become deficient in motion or position, transmission of the large generated forces from the lower extremity to the upper extremity is impaired. This creates a deficiency in resultant maximum force that can be delivered to the hand or creates a situation of 'catch up' in which the more distal links have to work more actively to compensate for the loss of the proximally generated force. This can impair the function of the distal links because they do not have the size, the muscle cross section area or the time to efficiently develop these larger forces. Calculations have shown that a 20% decrease in kinetic energy delivered from the hip and trunk to the arm necessitates an 80% increase in mass or a 34% increase in rotational velocity at the shoulder to deliver the same amount of resultant force to the hand[7]. This required adaptation can cause overload problems with repeated use. In condition of sport-related stress, regulatory imbalance might result both in typical reaction patterns and individual response specificity; this can explain the anatomo-pathological difference of the several lesions (extension, site, degrees of retraction etc), and it justifies those clinical pictures that, even if triggered by similar lesions, appear at different times and with different clinical features.

GIRD

Repetitive concentric and eccentric demands on the rotator cuff and hypermobility and excessive laxity of the glenohumeral joint could lead to scapulo-thoracic muscular fatigue altering the normal shoulder biomechanics. Fatigue affects sensation of joint movement, decreases athletic performance and increases fatigue-related shoulder dysfunction. The muscular imbalance during the deceleration phase transfers distraction forces to the posterior capsule that becomes tight and leads to an internal rotation reduction. Although the factors contributing to secondary shoulder impingement are multiple, the posterior capsule tightness is thought to alter shoulder kinematics, with superior translation of the humeral head during flexion such that the rotator cuff is compromised by the overlying coracoacromial arch and posterior translation with external rotation (from Grossman). Glenohumeral joint tightness can also create abnormal biomechanics of the scapula. Posterior shoulder inflexibility, due to capsular or muscular tightness (infraspinatus thixotropy), affects the smooth motion of the glenohumeral joint and creates a 'wind up' effect so that the glenoid and scapula actually get pulled in a forward and inferior direction by the moving and rotating arm[10]. This can create an excessive amount of protraction of the scapula on the thorax as the arm continues into the horizontally adducted position in follow-through. Because of the geometry of the upper aspect of the thorax, the more the scapula is protracted in follow-through, the further it and its acromion move anteriorly and inferiorly around the thorax.

In those patients with limited internal rotation and flexion, a therapy programme should be directed at improving these motion planes

Table 1

Task	Node
Baseball pitch	*Weight on back leg with trunk straight*
	Hip and trunk synchrony
	Elbow at or above 90° abduction with scapular retraction and hand on top of ball
	Front foot directly towards home plate
	Long axis rotation: shoulder internal rotation and forearm pronation
Tennis serve	*Use of back foot to push off*
	Knee flexion greater than 10°
	Back hip counter rotation away from the net and downward tilt
	Trunk rotation away from the net, hip/trunk separation angle of 30°
	Arm cocking in the scapular plane
	Long axis rotation

Table 1: Key positions and motions for baseball pitch and tennis serve.

In all cases with suspected impingement, a careful examination of both passive and active motion of the shoulders in all planes is needed. In those patients with limited internal rotation and flexion, a therapy programme should be directed at improving these motion planes.

PREVENTION OF SHOULDER INJURIES

Shoulder injuries resulting from overuse may involve rotator cuff, biceps tendon and labral pathology. These are often secondary to not only the repetitive concentric and eccentric demands on the rotator cuff but also the underlying hypermobility and excessive laxity of the glenohumeral joint[11]. The high levels of muscular control required to maintain stability of the shoulder joint during tennis strokes have been reported by Ryu et al[5]. This study reported high levels of normalised concentric and eccentric muscular activity using electromyography for the rotator cuff and scapular stabilisers during virtually all strokes. For example, during the cocking phase of the tennis serve, muscular activities of the supraspinatus (53%), infraspinatus (41%) and serratus anterior (70%) function to position the scapula and stabilise the glenohumeral joint, while during the follow-through phase, eccentric activation of the rotator cuff (40%) and serratus anterior (53%) assists with further stabilisation and deceleration of the shoulder[5]. The fact that the modern game of tennis is characterised by over 75% forehands and serves, which inherently require powerful concentric internal shoulder rotation for power generation, is consistent with the common finding of muscular imbalance between the posterior rotator cuff (external rotators) when compared with the internal rotators[5]. Additionally, isokinetic testing of the shoulder has repeatedly shown either equal or decreased dominant arm external rotation strength and 15 to 30% increases in dominant arm internal rotation strength compared with the non-dominant arm in elite-level players[6,7]. This finding, coupled with reports of scapular dysfunction and muscular weakness in the upper back and thorax among experts who routinely evaluate elite tennis players, has led to the recommendation of preventative exercises to increase posterior rotator cuff and scapular stabilisation[8]. These exercises should be performed using a multiple-set paradigm (2 to 3 sets) and high repetition base (15 to 20 repetitions per set) to promote local muscular endurance. Exercise programmes that contain multiple exercises using the low resistance, high repetition format have been used in both tennis players and overhead athletes, resulting in modification of the external to internal rotation ratio, improved strength and endurance of the rotator cuff and performance enhancement[9]. The exercises should be performed using moderate exercise intensities (approximately 40% of maximum voluntary isometric contraction levels) during rotator cuff exercise to enhance and facilitate the contribution from the rotator cuff and minimise deltoid activation and compensatory shearing during external rotation strengthening exercises.

TREATMENT OF SHOULDER LESIONS

The abnormal scapular biomechanics, occurring as a result of dysfunction, create an abnormal scapular position that decreases normal shoulder function and exposes the shoulder to injury if prevention strategies are not applied. The most common shoulder lesions in tennis players requiring surgical treatment are 'superior labral tears' (SLAP) and 'rotator cuff tears'. Posterior or posterior superior shoulder pain is felt without mechanical symptoms usually described as occurring during the late cocking and early acceleration phases of the throwing cycle. This is due to posterior superior glenohumeral instability directed by the posterior inferior capsular contracture as the shoulder abducts and rotates. The posterior superior shift of the glenohumeral contact and rotational point creates strain on the posterior superior labral glenoid interface as well as allows for increased external humeral rotation which brings the undersurface of the posterior superior rotator cuff in contact with the posterior superior glenoid margin resulting in the early symptoms of 'internal impingement'. The SLAP event occurs when the posterior superior labrum and biceps

anchor fail in tension from their glenoid attachments secondary to the capsular contracture mediated by posterior superior glenohumeral instability. Once the SLAP event has occurred, the thrower promptly develops mechanical symptoms in late cocking and early acceleration phases.

Diagnosis of a SLAP tear can be difficult. The best method is to combine elements of the history and physical exam, use imaging as needed, and arthroscopically treat what patho-anatomy is found. Helpful history findings include posterior joint pain with abduction/external rotation, pop or click, feeling of sliding and feeling of stiffness and decreased ball velocity. There are many sensitive clinical tests for SLAP tear diagnosis (Yergason's, Speed's, bear hug, belly press, O'Brien's, anterior slide, upper cut and modified dynamic labral shear) but none of them is specific[10]. The modified dynamic labral shear has high sensitivity, positive predictive value and positive likelihood ratio when the test is administered correctly. MRI can be sensitive and helpful in confirming a clinically derived diagnosis, but is not specific, with a high percentage of false positives, so it should not be used as the sole or major criterion to make the diagnosis. There is good evidence to show that making the SLAP diagnosis from the arthroscopic evaluation is inconsistent with both false negatives and many false positives. Arthroscopy is very helpful in determining all the aspects of the SLAP lesion to fix. The symptomatic throwing shoulder can frequently be successfully treated by a series of focused posterior inferior capsular stretches to eliminate the contracture and strength exercises to rehabilitate any concomitant rotator cuff and scapular stabiliser deconditioned musculature. However, if rehabilitation fails, surgical treatment is necessary. Arthroscopic treatment for SLAP lesions is different depending on the case. For a Type I lesion arthroscopic debridement is indicated. For unstable SLAP lesions (Type II, III, IV), arthroscopic fixation is recommended.

Arthroscopic evaluation of the suspected labral injury must be specific in order to understand and treat the labral injury properly. The arthroscopic findings most frequently associated with a clinically significant labral injury include:

1. a type II or higher lesion denoting loss of attachment from the glenoid,
2. a peel-back phenomenon indicating labral detachment, increased compliance, loss of washer effect and loss of bumper effect,
3. glenoid articular cartilage damage or chondromalacia indicating increased translation,
4. loss of capsular tension indicated by a drive-through sign or loss of tension in the posterior band of the inferior glenohumeral ligament,
5. increased posterior labral thickness, indicating increased translation and shear with compression on the labrum and/or
6. excessive posterior inferior capsular thickness and scar indicating end stage capsular damage that helps create GIRD.

Care must be taken to differentiate labral detachment from anatomic variants such as sublabral foramina, a Buford complex attachment of the middle glenohumeral ligament or a meniscoid-like labral attachment that does not peel back.

Based on these principles, arthroscopic treatment guidelines for labral injury include:

1. evaluation of the peel-back, labral injury and mobility, glenoid surface and capsular tension by direct visualisation
2. preparation of the glenoid to maximise bone-to-labrum healing,
3. multiple anchor placement to secure at least two-point fixation of the labrum on the posterior superior glenoid (10:30 and 11:30 on the right shoulder) (a double loaded single anchor is still only one point fixation),
4. placement of enough posterior superior anchors to eliminate the peel-back,
5. evaluation of biceps mobility after anchor and suture placement to make sure there is adequate motion of the biceps in shoulder external rotation,
6. rare placement of anchors and sutures in the anterior superior glenoid (12:00 to 2:30 on the right shoulder) to reduce the chance of biceps tethering,
7. evaluate the effect of the labral repair on capsular tension by evaluation in the posterior band of the inferior glenohumeral ligament tautness and elimination of the drive through,

8. assess total glenohumeral rotation to ensure no external rotation has been lost and
9. treatment of the associated pathology in the joint.

CONCLUSION

Shoulder injuries can occur in two main modes: acute or chronic injuries. The **acute mode** is the result of an acute macro-trauma. The **chronic mode** is the result of a micro-trauma with a gradual onset of symptoms. Injury may lead to mild subclinical tensile overload in specific tissues (pre-SLAP lesion, partial rotator cuff tear). It is noteworthy how a seemingly localised shoulder microtrauma (such the peel-back in SLAP lesion during the cocking phase of throwing or the posterior capsular overload in follow through) generally does not result only from a local cause. For example, a scapular dyskinesis causes more protracted scapula and therefore a higher bicipital anchor stress during late cocking or early acceleration phase of the throwing sequence and reduces the eccentric posterior cuff contraction causing a posterior capsular overload during

the follow-through (localised postero/inferior capsular contracture). The functional shoulder overload, often resulting from a compensation mechanism due to the lack of force and energy delivered through the more proximal links within the kinetic chain, tends to appear in structures (rotator cuff tendons, subacromial bursa, the capsular labral system) susceptible to microtraumas (overload), this 'catch-up phenomenon' is dangerous and inefficient for the shoulder structures which have to absorb more stress and load. When more load is introduced to the shoulder, anatomical-biomechanical deficits ensue in these tissues with the relevant increased lesion risk and decreased performance. A tennis-specific strength and conditioning programme can play a key role in preventing common injuries in tennis players. Specific exercises are suggested in this review based on these sport-specific muscular imbalances, which are designed to attempt to prevent injuries and enhance a player's performance. On the other hand, when a lesion is already present (SLAP lesion, rotator cuff lesion), arthroscopic fixation is recommended.

References

1. *Pluim BM and Staal JB. Tennis injuries in Olympic Sports. In: Encyclopedia of Sports Medicine. Caine D, Harmer P, Schiff M, eds. Hoboken, NJ: Wiley-Blackwell 2009.*
2. *Van der Hoeven H, Kibler WB. Shoulder injuries in tennis players. Br J Sports Med 2006; 40:435-440.*
3. *Sell K, Hainline B, Yorio M, Kovacs M. Injury trend analysis from the US Open Tennis Championships between 1994 and 2009. Br J Sports Med 2014; 48:546-551.*
4. *Kibler WB. Biomechanical analysis of the shoulder during tennis activities. Clin Sports Med 1995; 14:79-85.*
5. *Ryu KN, McCormick J, Jobe FW, Moynes DR, Antonelli DJ. An electromyographic analysis of shoulder function in tennis players. Am J Sports Med 1988; 16:481-485.*
6. *Ellenbecker TS. Shoulder internal and external rotation strength and range of motion in highly skilled tennis players. Isokinet Exerc Sci 1992; 2:65-72.*
7. *Ellenbecker TS, Roetert EP. Testing isokinetic muscular fatigue of shoulder internal and external rotation in elite junior tennis players. J Orthop Sports Phys Ther 1999; 29:275-281.*
8. *Kibler WB. The role of the scapula in athletic shoulder function. Am J Sports Med 1998; 26:325-337.*
9. *Niederbracht Y, Shim AL, Sloniger MA, Paternostro-Bayles M, Short TH. Effects of a shoulder injury prevention strength training program on eccentric external rotation muscle strength and glenohumeral joint imbalance in female overhead activity athletes. J Strength Cond Res 2008; 22:140-145.*
10. *Kibler WB, Sciascia AD, Hester P, Dome D, Jacobs C. Clinical utility of traditional and new tests in the diagnosis of biceps tendon injuries and superior labrum anterior and posterior lesions in the shoulder . Am J Sports Med 2009; 37:1840-1847.*
11. *Kibler WB, Kuhn JE, Wilk KE, Sciascia AD, Moore SD, Laudner KG et al. The disabled throwing shoulder – Spectrum of pathology: 10 year update. Arthroscopy 2013; 29:141-161.*

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