Clinical Biomechanics 30 (2015) 641–648

Contents lists available at ScienceDirect

Clinical Biomechanics

journal homepage: www.elsevier.com/locate/clinbiomech

Review

An evidence-based review of current perceptions with regard to the subacromial space in shoulder impingement syndromes: Is it important and what influences it?

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ARTICLE INFO

Article history:
Received 19 March 2015
Accepted 1 June 2015

Keywords:
Ubacromial space
Shoulder impingement
Acromiohumeral distance
Rotator cuff impingement
Biomechanical mechanisms

ABSTRACT

Background: Reduction of the subacromial space as a mechanism in the etiology of shoulder impingement syndromes is debated. Although a reduction in this space is associated with shoulder impingement syndromes, it is unclear if this observation is cause or consequence.
Method: The purposes of this descriptive review are to provide a broad perspective on the current perceptions with regard to the pathology and pathomechanics of subacromial and internal impingement syndromes, consider the role of the subacromial space in impingement syndromes, describe the intrinsic and extrinsic mechanisms considered to influence the subacromial space, and critique the level of evidence supporting these concepts.
Finding: Based on the current evidence, the hypothesis that a reduction in subacromial space is an extrinsic cause of impingement syndromes is not conclusively established and the evidence permits no conclusion.
Interpretation: If maintenance of the subacromial space is important in impingement syndromes regardless of whether it is a cause or consequence, research exploring the correlation between biomechanical factors and the subacromial space, using the later as the outcome measure, would be beneficial.

1. Anatomy of the subacromial space and pathogenesis of impingement syndrome

One of the most common musculoskeletal complaints of patients seeking medical advice is shoulder pain, with shoulder impingement syndrome being the most commonly diagnosed shoulder disorder (de Witte, 2011; Michener et al., 2003; Parsons et al., 2007; Seitz et al., 2011). Despite the commonality of SIS, etiology is still unclear and much debated. Modern advances in anatomy, biomechanics, and research have gone some way in improving the understanding of impingement syndrome (Ellenbecker and Cools, 2010), but despite this, it is still a debated topic. Typically, patients present with rotator cuff tendinopathy. This is the term used broadly to cover pathology in the tendon without assuming specific knowledge of the underlying mechanism causing the condition (Seitz et al., 2011).

The superior boundary of the subacromial space is formed by the acromion and the coracoacromial ligament (Fig. 1). The acromion, the coracoacromial ligament, and the coracoid together form the coracoacromial arch (Fig. 1). The anterior acromion and superior boundary of the subacromial space have to move superiorly for the humeral head to elevate during arm elevation. Should this not occur, it is the anterior acromion that has been identified as the site at which compression on the bursal side of the rotator cuff tendon occurs (Brossmann et al., 1996; Flatow et al., 1994; Lee et al., 2001). The inferior subacromial space is defined by the humeral head, superior glenohumeral joint, and the coracohumeral ligament (Fig. 1). Only 25%–30% of the surface of the head of humerus is said to be in contact with the glenoid at one time (Hurov, 2009). The instant center of rotation of the humeral head, although movable, has to be controlled with in this limited surface contact. Failure to control the instant center of rotation in the glenohumeral joint compromises the integrity of the inferior surface of the subacromial space. Impingement syndrome, involving tendinopathy of

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http://dx.doi.org/10.1016/j.clinbiomech.2015.06.001
0268-0033/

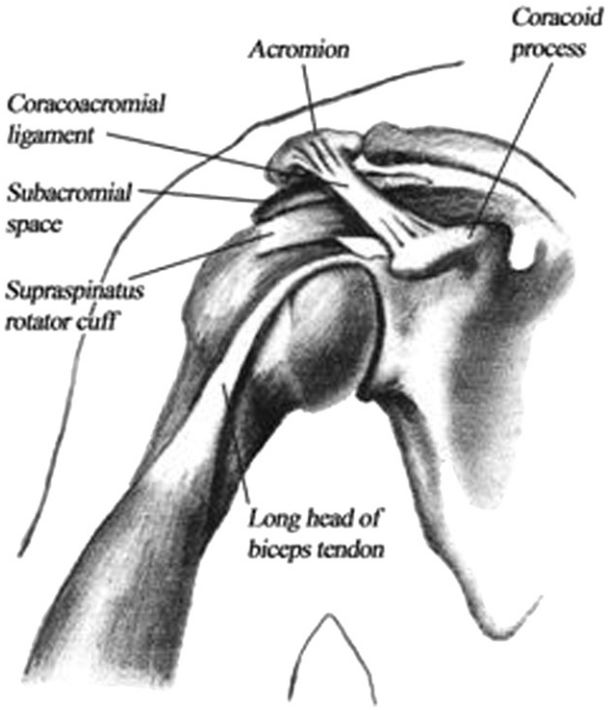

Fig. 1. Coronal cross-section of the subacromial space.

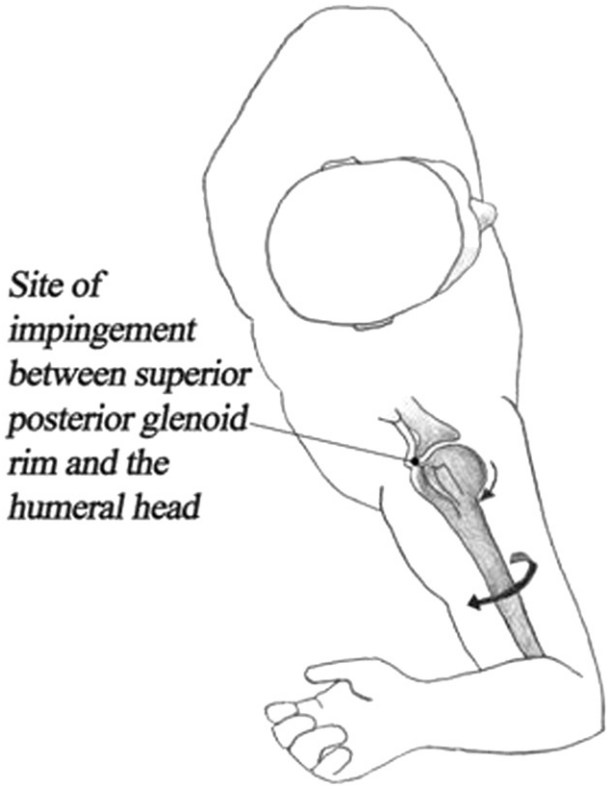

Fig. 2. Internal impingement: the tendon of the rotator cuff becomes compressed between the superior posterior glenoid rim and the humeral head.

the rotator cuff tendons, can be divided into two broad groups defined according to anatomical site (bursal or articular) of the tendon being impinged upon, and by the pathomechanics involved. These two broad groups are referred to as subacromial impingement syndrome and internal impingement syndrome.

1.1. Pathogenesis of subacromial impingement syndrome

In 1972, Neer coined the term subacromial impingement and proposed a pathomechanical process in which mechanical compression of the soft tissues in the subacromial space occurred due to a narrowing of the subacromial space (Neer and Welsh, 1977). He asserted that the soft tissues most commonly involved was the bursal side of the supraspinatus and long head of biceps tendons which compress against the anterior and lateral edge of the acromion and coracoacromial ligament. Neer proposed that any reduction of the subacromial space would lead to IS. Contact between the supraspinatus tendon and the biceps tendon with the coracoacromial ligament has been confirmed in cadaveric studies to occur between 45 and 60 degrees of abduction (Burns and Whipple, 2013). Converging evidence from radiographs and MRI determined that the distal supraspinatus tendon was engaged between the greater tuberosity and the acromion as early as 30 degrees of flexion and abduction (Brossmann et al., 1996). It has been suggested via x-ray determination that at rest, the distance between the acromion and humerus is on average 11 mm, and at 90 degrees abduction, this distance is reduced to 5.7 mm on average (Flatow et al., 1994). A reduction in the subacromial space correlated to the incidence of IS in subjects (Burkhart, 1995; Werner et al., 2008). These observations do not define whether reduction in subacromial space is cause or consequence.

1.2. Pathogenesis of internal impingement syndrome

An impingement syndrome, commonly considered to be prevalent in overhead sportsman, has been identified and named "internal impingement syndrome" (Jobe and Pink, 1996; Kibler and Sciascia, 2009). This impingement syndrome occurs when the arm is in the abducted, extended, and eternally rotated position. The area of compression on the rotator cuff tendon is the articular side as oppose to the bursal side of the tendon as in subacromial impingement syndrome (Seitz et al., 2011). The tendon becomes compressed between the superior posterior glenoid rim and the humeral head (Ellenbecker and Cools, 2010) (Fig. 2). Increased capsule laxity or instability of the glenohumeral joint (Brukner and Khan, 2010) is considered a mechanism in internal impingement syndrome. Capsule laxity or instability of the glenohumeral joint results in an altered instantaneous axis of rotation of the humeral head in the glenoid, which can impose on the subacromial space and lead to a decrease in (Azzoni et al., 2004) the acromiohumeral space, and subsequently to compromise of this space.

2. Biomechanical influences on the subacromial space

There is controversy with regard to the exact pathomechanics and biomechanical causes of shoulder impingement syndrome. Possibly, factors are multifactorial (Wilk et al., 2009). Pathological factors that are considered to contribute to impingement syndrome can be divided into extrinsic and intrinsic categories. Extrinsic factors are considered to be those that compress the structures within the subacromial space (extra-tendinous), and intrinsic factors are those associated with degeneration within the rotator cuff tendons themselves (intra-tendinous) (Seitz et al., 2011). Extrinsic factors that encroach upon the subacromial space and contribute to compression of the rotator cuff tendons have been broadly grouped by the authors into alignment factors, anatomical/osseous factors, glenohumeral or scapular kinematic factors, muscular extensibility and performance factors, as well as ergonomic and sport-specific factors. Intrinsic factors that contribute to rotator cuff tendon degeneration due to tensile/shear overload include alterations in biology, mechanical properties, morphology, and vascularity within the tendon (Seitz et al., 2011).

The diverse nature of these speculated mechanisms indicates that impingement syndrome is not a homogenous entity. Treatment aimed at addressing mechanical factors appears to be beneficial for patients with impingement syndrome but not for all patients (Seitz et al.,