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Review

Kinematic patterns in normal and degenerative shoulders. Part II: Review of 3-D scapular kinematic patterns in patients with shoulder pain, and clinical implications

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ABSTRACT

Background: The global range of motion of the arm is the result of a coordinated motion of the shoulder complex including glenohumeral (GH), scapulothoracic, sternoclavicular and acromioclavicular joints.
Methods: This study is a non-systematic review of kinematic patterns in degenerated shoulders. It is a based on our own research on the kinematics of the shoulder complex and clinical experience.
Results: For patients with subacromial impingement syndrome without rotator-cuff tears, most kinematic studies showed a small superior humeral translation relative to the glenoid and decreased scapular lateral rotation and posterior tilt. These scapular kinematic modifications could decrease the subacromial space and favor rotator-cuff tendon injury. For patients with shoulder pain and restricted mobility, the studies showed a significant increase in scapular lateral rotation generally seen as a compensation mechanism of GH decreased range of motion. For patients with multidirectional GH instability, the studies found an antero-inferior decentering of the humeral head, decreased scapular lateral rotation and increased scapular internal rotation.
Conclusion: The clinical or instrumented assessment of the shoulder complex with a degenerative pathology must include the analysis of scapula-clavicle and trunk movements complementing the GH assessment. Depending on the individual clinical case, scapular dyskinesis could be the cause or the consequence of the shoulder degenerative pathology. For most degenerative shoulder pathologies, the rehabilitation program should take into account the whole shoulder complex and include first a scapular and trunk postural-correcting strategy, then scapulothoracic muscle rehabilitation (especially serratus anterior and trapezius inferior and medium parts) and finally neuromotor techniques to recover appropriate upper-limb kinematic schemas for daily and/or sports activities.

1. Introduction

In the companion article, Part I, we described the kinematic patterns of the normal shoulder complex explaining that the global elevation range of motion (RoM) of the arm was mainly the result of a coordinated motion between glenohumeral (GH) and scapulothoracic (ST) movements [1]. Therefore, the major mobility of the shoulder complex is allowed by a low congruity between the GH joint surfaces and also the associated movement in rotation and sliding of the scapula on the thorax. The scapular movements involve clavicle movements serving as a "setting wand". Clavicular motion occurs at the sternoclavicular (SC) and acromioclavicular (AC) joints contributing to the mechanical coupling between GH and ST movements. Thus, the efficiency of the shoulder kinematics is based on the coordinated and combined movements of the GH and the ST "functional" joint including SC and AC joints [1]. Therefore, any modification of one element of the shoulder complex will have an impact on this "kinematic chain". In particular, abnormal ST motion may be due to abnormal SC and AC motion.

Most shoulder pathologies have a dynamic component, which can be highlighted by the clinical examination, rather than by usual imagery techniques that remain essentially static. The standard clinical examination allows for assessing movements

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(not just static postures) but it is centered on the GH joint and does not sufficiently take into account the other complex joints, in particular the ST.

A better knowledge of the role of the ST joint in the shoulder kinematics has modified the shoulder clinical examination and resulted in the concept of "scapular dyskinesis" [2] defined as any "abnormality of the scapula posture or movement" whatever its etiology. It is characterized by a premature or excessive rotation of the scapula during elevating or lowering the arm with a posterior displacement of its medial border and/or inferior angle from the thoracic wall that appears as a scapula winging from the thorax [2].

Dyskinesis of the scapula is a clinical diagnosis and includes all disorders affecting the scapular posture. Two causes of scapular dyskinesis must be distinguished: (1) primary dyskinesis due to the injury of one of the ST anatomic elements (scapula fracture, clavicle, muscular rupture, bursitis, endochondromosis and/or neuromuscular injury) [3] (Videos 1 and 2; supplementary appendix) and (2) secondary dyskinesis due to a trunk static pathology (scoliosis or cyphosis) or a GH degenerative pathology [2]. However, modified scapular schemes may be secondary to numerous shoulder chronic pathologies such as rotator-cuff pathologies, GH osteoarthritis, adhesive capsulitis, instability, and shoulder arthroplasty [2,4–7].

The article is based on a non-systematic search in MEDLINE via PubMed for studies published in English from January 1994 to July 2016 with the keywords shoulder OR scapula AND kinematics AND degenerative pathologies AND rehabilitation, our own research and clinical experience, and relates the measurement techniques and the consensus used to describe the 3-D kinematics movements of the normal shoulder. In the first part, we describe the modifications of the 3-D scapular movements linked to the main shoulder degenerative pathologies: subacromial impingement syndrome (SIS) or rotator-cuff tendinosis, shoulder stiffness and shoulder instability. In the second part, we describe the clinical consequences of the concept of scapula dyskinesis in the rehabilitation program of the shoulder complex. In combination with the companion article, Part I, this description aims at a comprehensive investigation into scapular kinematics associated with shoulder pain due to degenerative pathologies.

2. Scapular kinematics modifications and degenerative shoulder pathologies

2.1. Scapular kinematics modifications and SIS

In total, 44% to 75% of patients who consult for a shoulder pain have a diagnosis of SIS [8]. SIS is defined as the repeated compression of the rotator-cuff in the subacromial space (SAS), the interval between the coracoid-acromial arch, the antero-inferior part of the acromion and the humeral head. Thus, SIS can be associated or not with rotator-cuff disease: SA bursitis, tendinopathy, and partial-thickness or full-thickness rotator-cuff tears [9]. SIS is characterized by its "dynamic" component: the symptoms appear in active movements, are relieved at rest and are reproducible by a clinical active test [8]. Therefore, SIS is associated with the biomechanical concept of the "dynamic narrowing" of the SAS [10].

The SIS aetiology is multifactorial and can be attributed to 2 main factors: "extrinsic" and "intrinsic". The SIS intrinsic origin is attributed to a degenerative rotator-cuff disease due to an inevitable age-related degenerative process of the tendons or to excessive or repeated solicitations, constraints or micro-injuries [9]. The SIS extrinsic origin corresponds to a mechanical compression of the tendon by external structures related to the acromion anatomical shape [11] or altered biomechanics of the shoulder complex during movements (humeral-head superior translation or lateral rotation and/or modification of the scapular movements causing an anterior and inferior positioning of the acromion [5,12–14].

Several studies proposed that SIS was due to the superior translation of the humeral head relative to the glenoid during arm elevation, which is greater in patients with SIS versus healthy people [9,10,15]. However, a detailed analysis of these studies showed SAS reduction only in cases of full-thickness rotator-cuff tear, which suggests that the superior translation is a result and not a cause of the SIS [13,16–18]. The increase in humeral-head superior translation in patients with a rotator-cuff tear injury could rather be linked to the decrease in rotator-cuff muscle performance, especially the infraspinatus [19]. In any case, biomechanical studies measuring only small superior humeral-head translation in patients with SIS often found a correlation with a change in the glenoid position [14]. Several studies described modifications of the humeral-head anterior-posterior translation in SIS without rotator-cuff tear [15]. The lack of GH lateral axial rotation has also been described as likely causing SIS by bringing the great tuberosity closer to the acromion. However, only a few studies have directly measured this conflict, probably because of the difficulty in precisely measuring the GH lateral rotation simultaneously with the humeral-head elevation in 3-D [11,20].

The other extrinsic cause of dynamic SIS could be the modification of the scapular posture during arm elevation, such as decreased scapular posterior tilt and lateral rotation [12] (Video 3, supplementary appendix). Most studies of symptomatic SIS without rotator-cuff tear that used radiography [22], inclinometers [23], and 3-D kinematic measures [4,21,23–25], found decreased scapular lateral rotation during arm elevation. In contrast, studies including only patients with rotator-cuff tear showed an increase in scapular lateral rotation [13,16,26]. In addition, most studies found decreased scapular posterior tilt and increased clavicle elevation in symptomatic shoulders with SIS without rotator-cuff tear [4,6,21,24,27]. The link between the scapular kinematic modifications and the decrease in SAS has been little studied in vivo and in 3-D [6,9,10,14,28–30]. A recent CT-scan study showed that, when the arm was actively elevated to 90° in the scapular plane, passive immobilization of the scapula induced a reduction in the SAS [30]. The Bdaiwi et al. ultrasound study showed an increase in the SAS during contraction of the muscles producing the posterior tilt (trapezius inferior part and serratus anterior) [31].

The kinematic study from Lawrence et al., using cortical pins, summarises well the current state of knowledge: with arm elevation between 30° and 60°, GH elevation increased about 6° simultaneously with decreased scapular lateral rotation in 10 patients with SIS as compared with healthy volunteers [15,32]. This zone of 30° to 60° arm elevation corresponds to that described by Giphart et al., whereby the footprint of the supraspinatus on the greater tuberosity is most in contact with the acromial inferior shape [15,33] (Fig. 1).

Whether dyskinesis is the cause or the consequence of the shoulder degenerative pathology is unknown. In the absence of trauma or systemic pathologies, the dynamic scapular factors could favor the rotator-cuff tendon injury by increasing shearing and compression constraints. An early correction of these scapular abnormalities should prevent the evolution of the rotator-cuff tear. Once the rotator-cuff tear is established, there could be a compensatory phenomenon such as an increase in the scapular lateral rotation [2,9,14,23,28]. This theory is supported by McCully et al., who showed that a suprascapular engine bloc (supra- and infraspinatus paralysis) increased lateral rotation and decreased internal rotation during active upper-limb elevation [34].

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