

ARM MOBILITY VERSUS GLENOHUMERAL STABILITY

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INTRODUCTION

Subacromial pathologies ranging from impingement to major cuff tearing are characterized clinically by shoulder pain and reduced arm mobility. The pathology is associated with a reduction of the subacromial space due to glenohumeral (GH) instability [1,2]. Arm mobility and GH-stability are both determined by shoulder muscle forces. Arm mobility requires a GH torque; stability requires GH force equilibrium [3,4].

We determined arm mobility, pain, external force and isometric muscle coordination before and after subacromial injection of lidocain in 6 patients with major rotator cuff tears. We will discuss our results in the scope of a torque versus force muscle function hypothesis.

METHODS

Six patients (4♂, 2♀, 61y SD=8) with irreparable rotator cuff tears, pain and reduced arm mobility were included prior to Teres Major tendon transfer surgery¹. Before and 10 min. after sub-acromial lidocain injection (5ml, 1%) we quantified the Principal Action (PA) of 6 muscle(part)s, i.e. the direction of maximum muscle activation provoked by a force of constant magnitude rotated in a plane perpendicular to the humerus [5,6]. For this purpose, the injured arm was fixed into a splint and connected at the elbow onto a force transducer constraining only two translation degrees of freedom (dof's) perpendicular to the humerus. The arm was to be held still for the four remaining (dofs) in about 60° elevation in the 45° plane of abduction; the elbow was 90° flexed, the forearm positioned at 45° above the horizontal plane. The magnitude of the external force equaled the minimum of 12 maximum voluntary forces (MVF) generated in equidistant directions of 30° onto the force transducer. Shoulder pain was quantified using a Visual Analog Scale (VAS) and range of arm abduction (RoM) was determined by means of electromagnetic motion tracking [7].

RESULTS AND DISCUSSION

The VAS decreased significantly after lidocain injection both in rest and during the experiment resulting in an increase of the minimum MVF with a factor 2.0 (SD 0.6). This was consistent with earlier reports [8]. The observed increase of ROM was not expected from model simulation, were transposition of the Teres Major muscle was found to be required for restoration of the insufficient GH abduction moment [4].

This study shows that the potential external muscle torque was not the limiting factor for ROM reduction. The possible explanation was found in the altered coordination of the shoulder muscles, illustrated here by the Principal Action (Figure 1). The ●-marks indicate the average normal function of the Deltoid muscle parts (Dlt), generating an abduction moment and activated around PA=0°, and the Latisimus Dorsi

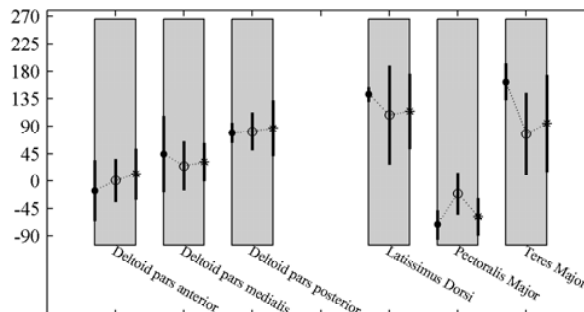


Figure 1: Principal action (degrees) of six shoulder muscles recorded by means of surface EMG for normal subjects (●) ± 95% C.I. [6] and patients with cuff tear prior to (○) and after subacromial lidocain injection (*). PA=0°: vertical abduction; PA=90°: horizontal abduction; PA=180°: vertical adduction; PA=270°: horizontal abduction.

(LD), Pectoralis Major (PM) and Teres Major muscles (TM), generating abduction and endoflexion moments and activated around PA=150° – 270° (i.e. equal to -90°)

Patients exhibit a relatively normal Deltoid PA. On average and prior to lidocain injection, the LD, PM and TM were co-activated during external abduction forces (change towards PA=0°). Lidocain injection reduced this effect and the external abduction moment and ROM were partly restored.

The underlying hypothesis is that the patients exhibit a GH instability. Superior translation of the humerus during abduction results in a reduction of the sub-acromial space and a pain sensation. Downward and inward directed muscle forces on the humerus stabilize the humeral position into the glenoid cavity. The LD, PM and TM muscles generate these downward and inward forces, which consequently results in the reduction of the abduction moment generated by the deltoid muscle and a reduction of RoM. Lidocain reduces the GH 'pain-feedback' activation of the adducting LD and TM, thus increasing the external moment and ROM.

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