Dynamic Chiropractic

SOFT TISSUE / TRIGGER POINTS

Shoulder: Primary or Secondary Impingement?

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In 1972 a surgeon by the name of Neer developed the theory of shoulder impingement caused by hereditary anterior acromial enlargement resulting in a stenosis of the coracoacromial arch.[1] This theory greatly influenced the surgical community and acromioplasty became the operation of choice for shoulder impingement problems. This operation "often failed to return the athletically active patient to a prior level of athletic activity."[2] Neer also developed an hypothesis describing three progressive stages of impingement[3] which has appeared in many articles over the years. Like much information available to us these stages have been accepted as gospel:

- Stage I referred to patients under 25 years old with a reversible condition due to edema, inflammation and hemorrhage.

- Stage II involved patients between 25 and 40 years of age with a thickened bursa, fibrosis and scarring of cuff tendons and biceps which was considered irreversible.

- Stage III referred to patients over 40 years of age with partial or full thickness rupture and bony changes.

Radiographic signs appeared which were not present in the first two stages. According to Uhthoff and Sarkar[4] "there has been no adequate pathologic studies to establish the characteristic features of the first two stages." They feel that at present science is unable to classify impingement syndrome on the basis of pathologic changes. It is better to think of the syndrome as either uncomplicated (early and late stages usually responding to conservative treatment) or complicated (cuff ruptures and bony spurring which may require surgery). Ruptures can be due to chronic degeneration and be associated with impingement, but not necessarily a consequence of impingement.[4]

Tendon overuse that would be found aggravating younger patients under 25 rarely shows evidence of inflammatory cells. Nirschl[2] has coined the term angiofibroblastic tendinosis (disorganized collagen, fibroblasts and vascular elements without inflammation) to describe the pathology in Stage I and into the succeeding stages.

Neer was describing a primary impingement due to pressure of the anterior acromion. Therefore, according to his theory most of the pathology leading to eventual rupture should occur on the bursal or superior portion of the tendon. Based on the experience of Uhthoff and Sarkar[4] most of the tears begin at the articular side (i.e., below the tendon) close to the insertion of the tendon. Nirschl[2] states that "The theory of primary impingement therefore comes under major challenge in that no clinical

series reports clear, observable evidence of subacromial changes in over 25 percent of the cases." The supraspinatus first, then the infraspinatus and rarely the teres minor bare the brunt of most of the rotator cuff pathology.

Especially in the younger age group below 40 the etiology of most shoulder impingement problems are due to secondary factors rather than hereditary anomalies (anterior acromial beaking). Some secondary causes are:

- eccentric muscular overload causing cuff fatigue and tendon swelling;

- muscle imbalances (such as tight posterior shoulder muscles and overdeveloped internal over external rotators) causing excessive anterior-superior humeral translation, stressing the cuff and capsuloligamentous tissue, or glenohumeral instability (anterior subluxation) resulting in: upward humeral migration and stress of the articular portion of the cuff tendons; fibrosis of the subacromial bursa; hypertrophy of the cuff (weightlifters); lagging of scapular motion, a result of weakened scapular rotators (especially the serratus anterior) failing to provide a stable base for the humerus and allowing excessive anterior superior humeral head translation.

Rehabilitation should be directed to strengthening all of the glenohumeral and scapular muscles that are weak. Soft tissue methods such as friction massage and Leahy's active release technique are excellent methods of eliminating adhesions and restoring normal mechanical motion. While except for gout, rheumatoid arthritis, pyogenic infections or tuberculosis, most bursitis conditions are secondary pre-tendinitis problems.[5] The chronic fibrous bursa responds extremely well to deep friction over the involved bursa,[6] although the patient should be warned that the first three visits may temporarily aggravate the condition.

References

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the hip or shoulder. JMPT 1993;16,2:107-111.

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Editor's Note: Dr. Hammer will be conducting his next Subluxation Complex Myopathology (SM) seminars April 29-30 in Toronto, Canada, and May 20-21 in Seattle, Washington. To register, call 1-800-359-2289.

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