Impingement is not impingement: the case for calling it "Rotator Cuff Disease"

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Summary

Historically, many causes have been proposed for rotator cuff conditions. The most prevalent theory is that the rotator cuff tendons, especially the supraspinatus, make contact with the acromion and coracoacromial ligament, resulting in pain and eventual tearing of the tendon. However, more recent evidence suggests that this concept does not explain the changes in rotator cuff tendons with age. The role of acromioplasty and coracoacromial ligament release in the treatment of rotator cuff disease has become questioned. Evidence now suggests that tendinopathy associated with aging may be a predominant factor in the development of rotator cuff degeneration. We propose that the overwhelming evidence favors factors other than "impingement" as the major cause of rotator cuff disease and that a paradigm shift in the way the development of rotator cuff pathology is conceptualized allows for a more comprehensive approach to the care of the patient with rotator cuff disease.

KEY WORDS: acromioplasty, impingement, rotator cuff, shoulder, tendinopathy surgery, treatment.

Introduction

The cause of rotator cuff conditions has been debated for more than 100 years. Theories include intrinsic tendon degeneration, vascular factors, tension overload, differential stress in layers of the tendon, and impingement syndromes. The latter has become synonymous with all rotator cuff conditions and rotator cuff disease in general. As a result, anterior and lateral shoulder pain is commonly described by many providers as "impingement". However, rotator cuff disease is a condition with protean presentation and multifactorial intrinsic or extrinsic causes, and biologic, biomechanical, anatomical, and clinical information increasingly suggests that the theory of impingement often does not reflect the reality of the pathogenesis of rotator cuff disease. This commentary will make the arguments that: 1) the term "impingement" for the symptoms of rotator cuff abnormalities does not reflect modern knowledge and promulgates a viewpoint that adversely affects the science and the evolution of patient treatment, 2) the pathogenesis of rotator cuff disorders results from a variety of factors and not just "impingement" and that, therefore, the constellation of symptoms attributed to rotator cuff abnormalities should be called "rotator cuff disease."

History of terminology and current findings

It was only in 1972 that Dr. Charles Neer[1] fully elucidated the idea that rotator cuff problems resulted from contact or "impingement" of the rotator cuff tendons to the acromion, to the coracoacromial ligament, or to the undersurface of the acromioclavicular joint. His revolutionary idea was that the condition could be successfully treated by partial anterolateral acromioplasty and coracoacromial ligament release as opposed to total acromionectomy. For the last 40 years, the Neer concept of "impingement" has been the overwhelming theory on the cause of rotator cuff disease and the basis for clinical tests for deciphering its symptoms, for describing radiographic or magnetic resonance imaging changes, and for the rationale for non-operative and surgical treatment. Over

time, the concept of impingement has been altered to include other potential causes of rotator cuff abnormality, such as contact with the superior glenoid contact (arm in flexion)[2,3], with the posterior and superior labrum (arm in abduction and external rotation)[4,5], and with the coracoid (arm in flexion and internal rotation)[6].

Increasingly, rotator cuff disease has been appreciated as a form of the tendinopathy seen in other tendons in the body. It is an overuse tendinopathy that includes a spectrum of clinical features and pathologic characteristics[7]. Despite the frequent use of the term "tendinitis", rotator cuff tendinopathy is characterized histologically by little evidence of inflammation. Instead, histologically, the findings are more typical of a "failed healing response", with a haphazard proliferation of tenocytes, intracellular abnormalities in tenocytes, disruption of collagen fibers, and subsequent increase in non-collagenous matrix[8-10]. Some of these intratendinous changes may be related to the normal ageing process of the tendon and soft tissues[11], but the exact pathophysiologic mechanism is still unclear. However, heavy physical loading, injury, vibration, infection, smoking, genetic factors, and fluoroquinolone antibiotics can produce such histologic features[11].

The exact cause of tendinopathy in any tendon remains controversial and may involve a combination of factors intrinsic to the tendon and of extrinsic factors[12,13]. Excessive load, repetitive load, or loads applied from different directions have been implicated in the process of tendinopathy. Other theories include localized hypoxia produced by tensile load[14], hyperthermic injury as the tendon heats up with exercise[15], tenocyte apoptosis[16], and cytokines or proteolytic enzymes released as a result of applied stress[17]. The release of nitrous oxide has also been implicated in the tendinopathy process[18].

The pain related to rotator cuff disease has been reported to be associated with stimulation of the free nerve endings in the bursa. One report indicated that the stimulation was provided by one or more of the substances mentioned above[19]. A histologic study has shown that the largest number of free nerve endings around the should be in the subacromial bursa, followed by the biceps tendon and the capsule[20]. The rotator cuff tendons have few free nerve fibers, so although the tendons play a role in generating pain, it is most likely through some indirect mechanism whereby some peptides or transmitters initiate a pain response from the pain fibers in the bursa, biceps tendon, or the joint lining[21].

There are several other concerns with the theory of impingement as a cause of rotator cuff disease. Although rotator cuff tears have been shown to be associated with spurs on the acromion and the shape of the acromion, these two observations have not been proven to be causally related. Morrison and Bigliani[22] described three acromial shapes that increased with age and were associated with rotator cuff tears, but this relationship was not causally established in that work. Unfortunately, studies have shown that the radiograph to determine the acromial shape does not have high intra- or interobserver reliability[23-25]. The shape of the acromion is also affected by the angle of the radiograph, so subtle changes in the radiological beam can change the perceived shape of the acromion.

The traditional surgical treatment for rotator cuff symptoms has been a partial anterolateral acromioplasty and a release of the coracoacromial ligament. To our knowledge, no clinical study has correlated surgical results with converting the acromion shape to a type 1. One study evaluated acromial shape in patients with preoperative small to medium rotator cuff tears and prospectively randomized the patients to cuff repair with or without acromioplasty[26]. They found that both groups had the same tendon re-tear rate and that the failure had no correlation to acromial shape[26].

The success of rotator cuff surgery has been largely reported to be independent of acromioplasty and coracoacromial ligament release[1,27-29]. Holt and Allibone[30] found that one of the major functions of the coracoacromial ligament was to serve as a major restraint to superior migration of the humeral head, especially in the presence of large to massive rotator cuff tears. Several studies have reported no difference in clinical results for patients who had rotator cuff surgery with or without acromioplasty and coracoacromial ligament release[26,31-33]. In a systematic review, Chahalet al.[34] reported, at intermediate follow-up, no difference in subjective results for arthroscopic rotator cuff repair with or without partial acromioplasty.

There also is a large disconnect between the presence of a rotator cuff tear and the presence of pain. Some patients with large rotator cuff tears have no pain whatsoever, whereas some with small tears have substantial pain. Numerous clinical studies[35-37] have shown that an overwhelming percentage of patients who have a failed rotator cuff repair obtain pain relief from surgery despite the fact that one or more rotator cuff tendons may be torn. These studies suggest that rotator cuff tendon healing to bone is not necessary for a good surgical result and that some mechanism other than impingement is responsible for the pain.

Other investigators have questioned the role of acromioplasty in preventing the progression of rotator cuff disease. In a nine-year follow-up of 96 patients who had partial anterolateral acromioplasty, Hyvönen et al.[38] reported that, in 20% of their patients, the rotator cuff disease progressed, suggesting other etiologic factors as causative in the disease. Kartus et al.[39] found that, in a long-term (9 years) follow-up of a cohort of patients with partial anterolateral acromioplasty, more than one third of the patients had rotator cuff disease progression so that the failure rate of the repairs increased over time. They concluded that rotator cuff surgery consisting anterolateral acromioplasty did not prevent progression of the disease.

Rotator cuff disease has been the subject of several extensive analyses. A recent systematic review by Papadonikolakis et al.[40] examined five commonly held assumptions about rotator cuff disease. They re-

ported little support in the literature for acromioplasty and its place in the treatment of rotator cuff disease. In a consensus statement on the management of rotator cuff disease, the American Academy of Orthopedic Surgeons suggested that few treatments of rotator cuff disease can be supported by high level of evidence studies[41]. Therefore, it is logical to conclude that rotator cuff disease is the result of a combination of intrinsic tendon factors and extrinsic factors[42]. Intrinsic factors would include the poorly understood process of progressive tendinopathy, the poor blood supply near the insertion of the tendons, and the inability of the tendons to heal intrinsically[43]. Extrinsic factors would include the amount of stress applied to the tendon by tension or muscle activity and also potentially the impingement of the tendons on other structures.

One of the main reasons to desist calling rotator cuff disease "impingement" is that it limits the thinking of practitioners and researchers who address this condition. Rotator cuff disease is a complex disease entity, as are painful tendinopathies in other parts of the body. The framework of "impingement" defies the complexity of the process, and researchers need to be able to study this condition without this restrictive concept. Braman et al.[44] suggested that the term "impingement" should be replaced with either anterior or posterior "shoulder pain" and that the term "impingement" is too broad and includes too many possible pathomechanical entities.

There are other medical practitioners whose interpretation of this disease influences how orthopaedic surgeons manage patients. For example, radiologists often describe "impingement" of the acromion on the rotator cuff or suggest that the acromial morphology is consistent with "impingement". It is also not uncommon to have radiologists comment in reports on "impingement" of spurs at the acromioclavicular joint on the muscular portion of the supraspinatus muscle. This information confuses patients, and radiologists not infrequently suggest that surgery to remove the spurs is recommended. Physical therapists, too, often express concerns that patients under their treatment have "impingement" when, in reality, they have anterior and lateral shoulder pain from a variety of causes. The causes of anterior and lateral shoulder pain include arthritis, stiffness, biceps tendon tenosynovitis or tears, rotator cuff abnormalities, instability, and superior labrum anterior and posterior tears. In fact, the examination for this constellation of symptoms is inexact, and to conclude that it is "impingement pain" limits one's ability to reach a proper diagnosis and deliver proper treatment to the patient. In our opinion, pain in the anterior and lateral shoulder should be called "anterolateral shoulder pain syndrome" and not "impingement" pain.

Another pervasive theory is that rotator cuff impingement is associated with a protracted scapula, such as is seen with kyphosis of the spine or in some athletes[45]. The theory is that, in athletes, the protracted scapula is accompanied by a relative anterior tilting of the scapula, which has been reported to decrease the subacromial space[46], causing the rotator cuff to impinge on the acromion[47]. Physical therapy is directed at correcting the protracted scapula. However, if impingement is not impingement, meaning that the pathophysiology is not the rotator cuff hitting the acromion, then it is likely that other mechanisms, and not acromial contract, result in pain in patients with a protracted scapula. The cause-and-effect relationship between a protracted scapula and pain is currently not known. It is entirely possible that the shoulder reacts to pain of any cause by becoming protracted to decrease stress on the tendons or other structures. If rotator cuff disease is not primarily an impingement issue, then this theory and the observations of scapular motion may need to be revised to include other possible mechanisms of the pain.

Similarly, increasing kyphosis, which is seen with increasing age, has been postulated as a cause of rotator cuff disease by creating a protracted scapula. The theory is that the protracted scapula closes down the subacromial space and leads to "impingement pain"[48]. However, causality between the two observations has not been proved, and if rotator cuff disease is primarily a degenerative phenomenon and not the result of impingement, then it is possible that kyphosis has nothing to do with the development of rotator cuff disease and that the two phenomena are purely age-related changes. This change in conceptualizing impingement as the cause of rotator cuff disease would drastically change the approach to patients previously thought to have rotator cuff symptoms from structural kyphosis or from kyphosis associated with poor posture.

Conclusions

Rotator cuff disease is a multifactorial condition, the origin of which is unclear, but the failed healing response typically seen in other tendinopathies is the end result. The predominant theory of causality in which the rotator cuff wears down after contact with one structure or another has not been proven and does not explain the clinical manifestations of the condition. As a result, we recommend that the spectrum of rotator cuff abnormalities no longer be called "impingement disease" but rather "rotator cuff disease". Similarly, pain in the anterior and lateral shoulder should not be presumed to arise from rotator cuff contact with structures and should no longer be called "impingement pain" but rather "anterolateral shoulder pain". Evidence for continuing to focus on acromial morphology and acromial shape as a major contributor to rotator cuff disease is inadequate. Imaging studies reporting that there is "impingement" should be modified to note contact between structures but should no longer be interpreted as "impingement" because causality cannot be established on a static radiographic study. Lastly, clinical and experimental energy should be directed toward establishing the pathophysiology of rotator cuff disease, its natural history, the source of pain in rotator cuff disease, and

its effective treatments. The term "rotator cuff disease" will free the scientific community from the restraints of the limitations of the concept of "impingement" and will allow exploration of other causes and treatments.

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