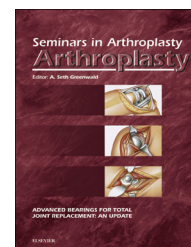


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Stiffness following shoulder arthroplasty: To manipulate or not

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ABSTRACT

Shoulder arthroplasty is considered the gold standard for treatment of end stage glenohumeral arthritis in select patients. This procedure routinely achieves excellent clinical outcomes for most patients, and fortunately complications are rare. Post-surgical stiffness is one such complication, which can be difficult to appropriately manage clinically. Causes include a preoperative diagnosis of proximal humerus fracture, infection, inadequate postoperative rehabilitation, and implant-related factors such as malpositioning. When nonoperative modalities such as physical therapy and corticosteroid injections fail, surgical options include glenohumeral debridement, subacromial decompression, and capsular release with or without manipulation.

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1. Introduction

Total shoulder arthroplasty (TSA) is considered the gold standard for management of symptomatic glenohumeral arthritis in patients with intact rotator cuff function, and this procedure has demonstrated good medium and long-term clinical outcomes as well as patient satisfaction scores [1–5]. Fortunately, complications following TSA are rare with the most common including glenoid component loosening, glenohumeral instability, and periprosthetic fracture [5,6]. While not always defined as a major complication following TSA, postoperative stiffness is an important clinical outcome that can lead to significant functional impairment and patient dissatisfaction [7,8]. Along with recalcitrant pain, stiffness is the most common symptom present in patients with failed TSA [8].

When managing the patient with a stiff TSA, the surgeon must remain vigilant with regard to diagnosing the underlying etiology of the stiffness and treating it appropriately.

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When a clear underlying etiology is discovered, such as periprosthetic infection or component malposition, revision arthroplasty is the optimal surgical management strategy. However, when the etiology of the stiffness is less transparent, the surgeon must either elect for nonoperative treatment with physical therapy or in more refractory cases, capsular release with or without manipulation. The objective of the present review is to highlight the underlying etiologies of postoperative stiffness following TSA, and to evaluate both the nonoperative and operative treatment strategies to manage this difficult problem, ultimately determining whether a manipulation under anesthesia is of benefit or not.

2. Etiology of stiffness following TSA

Common causes of stiffness following TSA include preoperative diagnosis of proximal humerus fracture, inadequate postoperative rehabilitation, incomplete capsular release,

heterotopic ossification, and implant-related factors including component malposition and inappropriately sized implants [7–9]. An important cause of stiffness that warrants special attention is periprosthetic infection, especially those culture-positive for *Propionibacterium acnes* [10].

Component malposition whether inappropriate version, height, or component size can all predispose to postoperative stiffness [8]. While malposition can be an important contributor to failure following TSA, few studies have specifically addressed this complication with regard to TSA failure [8,11,12]. Rather, they emphasize that reconstruction of the proximal humeral anatomy is paramount to success [13], and that component malposition can predispose to glenohumeral instability following TSA [5]. A common cause of stiffness is “overstuffing” the glenohumeral joint by using implants that are too large for the available volume, often in attempts to prevent glenohumeral instability. This over-tensions the soft tissue envelope, predisposing to stiffness [14]. Cadaveric studies have demonstrated that each millimeter of overstuffing the glenohumeral joint reduces glenohumeral motion by approximately 3°–4° [14].

Heterotopic ossification (HO) is a well-documented complication following joint arthroplasty. The most recent study documenting HO following shoulder arthroplasty reported a rate of 15%, all grade 1 or 2 [9]. While HO may predispose to postoperative stiffness, there is a paucity of data regarding management of HO following shoulder arthroplasty. Further, in the limited available literature, non-steroidal anti-inflammatory medications (NSAIDs) do not appear to have a protective benefit to HO formation in the shoulder, as they do in the hip [9,15].

Infection following TSA is a devastating complication, with published rates in the literature exceeding 1% [16–20]. More than half of these infections are culture-positive for *P. acnes*, which unfortunately does not typically present with the obvious clinical signs of infection or confirmatory laboratory values. Moreover, the incidence of *P. acnes* periprosthetic infection is likely under-recognized, given the failure of laboratories to utilize optimal methods of culturing this bacterium [10]. While the diagnosis and management of periprosthetic infection following TSA is beyond the scope of this review, it is imperative for surgeons to understand that infection with *P. acnes* following TSA may present with isolated stiffness or instability, and surgeons must maintain a high index of suspicion for infection when a patient presents with this clinical picture.

3. How to treat stiffness after TSA?

There is a significant paucity of data regarding both non-operative and operative treatment of postoperative stiffness following TSA. While watchful waiting is a reasonable option, there is no data to recommend for or against it. Furthermore, while some surgeons many advocate manipulation of the stiff shoulder under anesthesia, there is no literature to guide surgeons on this treatment. This leaves surgical options, which would include revision arthroplasty in the setting of infection or component malposition, if these are the underlying cause of stiffness. If infection or malposition is not

suspected, however, surgical options include open or arthroscopic glenohumeral debridement with subacromial lysis of adhesions and capsular release.

3.1. Nonoperative treatment

Unfortunately, there is minimal data in the literature to guide the nonoperative treatment of stiffness following TSA. Thus, most of the recommendations herein are level V evidence, and represent the treatment strategies of the senior author (AAR). If a patient presents postoperatively after completing an extensive standardized rehabilitation program with forward elevation greater than 90° and external rotation greater than 20°, we advocate for conservative, nonoperative treatment (Fig. 1). This includes a scheduled dosage of NSAIDs with or without oral or an intraarticular corticosteroid injection to help control inflammation, which could preclude successful rehabilitation. The patient is prescribed a guided physical therapy regimen to focus specifically on stretching in multiple planes, and the patient is advanced to a home program to include daily stretching exercises. In our experience, these patients do improve with these conservative efforts.

3.2. Operative treatment

There is limited data in the literature documenting the efficacy of arthroscopy following TSA [21,22]. In a case series of arthroscopic management of failed TSA, Hersch et al. reviewed 13 procedures for various pathology after TSA



Figure 1 – In a patient with forward flexion of at least 90°, external rotation of at least 20°, and no evidence of infection or component malposition, we elect nonoperative treatment with physical therapy, NSAIDs, and corticosteroids.

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