Prevention and Treatment of Elbow Stiffness: A 5-Year Update

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Continuing Medical Education

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Disclosures for this Article

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Learning Objectives

- Describe the various causes of elbow stiffness.
- Discern the molecular changes that take place in posttraumatic capsular contracture.
- Elaborate on the nonsurgical treatment modalities for elbow stiffness.
- Summarize the role of surgical treatment options for different types of elbow stiffness.
- Define the roles of open and arthroscopic elbow contracture release.

Deadline: Each examination purchased in 2013 must be completed by January 31, 2014, to be eligible for CME. A certificate will be issued upon completion of the activity. Estimated time to complete each month's JHS CME activity is up to 2 hours.

Elbow stiffness is a challenging and common problem faced by upper extremity surgeons. Although functional improvements can be made with both nonsurgical and surgical management strategies, physicians must remain vigilant with efforts to prevent stiffness before it starts. Recent advancements in the biology and pathology of elbow contracture have led to improved understanding of this difficult problem, and they may lead to future breakthroughs in the prevention and treatment of elbow stiffness. This article serves as an update to our previous review of elbow stiffness, focusing on recent advancements in the past 5 years, as well as updating our current algorithm for treatment. *(J Hand Surg Am. 2013;38(12):2496–2507. Copyright © 2013 by the American Society for Surgery of the Hand. All rights reserved.)*

Key words Contracture, elbow, heterotopic ossification, stiffness, ulnar neuropathy.

When stiffness occurs, a multidisciplinary and highly coordinated approach using nonsurgical modalities followed by surgical interventions can result in functional improvements. Elbow stiffness results from abnormalities of bone, soft tissue, or a combination of both; these causative factors guide the classification, diagnosis, prevention, and treatment. Management should focus on improving motion, with the goal of regaining a functional arc of motion. The functional arc of elbow motion has been defined as 100° for both flexion-extension and pronation-supination; most studies use these values as benchmarks for evaluating and treating elbow stiffness. These values have been recently challenged[2] while evaluating contemporary tasks, demonstrating an average flexion-extension arc of 130° and pronation-supination arc of 103°. Regardless, functional limitations can be seen with less severe loss of motion, and treatment should focus on the functional needs and desires of each patient.

Currently, no ideal management solution has been defined, and room for improvement exists, as evidenced in the consistent flow of literature over the past several decades. Due to the nature of the problem, most reports are small case series, but some attempts have been made to attain higher levels of evidence. Outcomes of both nonsurgical and surgical modalities continue to show consistent functional improvements. Despite this, after elbow stiffness develops, a "normal" elbow is rarely achieved. This review serves as an update to our previous review of elbow stiffness,[1] focusing on recent advancements in the past 5 years and summarizing our current algorithm for treatment.

ETIOLOGIES

Elbow stiffness can result from traumatic and atraumatic causes. Traumatic causes include fractures, dislocations, crush injuries, burns, and head injury. Atraumatic causes of elbow stiffness include rheumatoid arthritis, osteoarthritis, postseptic arthritis, hemarthroses associated with hemophilia, congenital contractures, and congenital radial head dislocation. The surgeon must be mindful that elective surgery results in tissue trauma that may be complicated by elbow stiffness, particularly when motion is restricted after surgery.

Mechanical blocks to motion, generally due to bony and/or soft tissue abnormalities, ultimately result in elbow stiffness. These mechanical blocks can be defined as *intrinsic* or *extrinsic*, depending on their proximity to the joint. Intrinsic contractures result from intra-articular pathology, whereas extrinsic contractures result from extra-articular pathology and secondary joint contracture. The majority of contractures are of mixed etiology. Intra-articular fractures, malunions, osteochondral defects, and arthritic changes alter the geometry of the elbow joint leading to stiffness. Heterotopic ossification (HO) occurs in response to tissue trauma, blocking elbow motion (Figs. 1A, 1B). The incidence of HO of the elbow varies in the literature from approximately 3% following simple elbow dislocations to as much as 89% in patients with a combination of elbow trauma and head injury.[1,3,4] Soft tissue changes typically occur due to bony pathology leading to contracture of the capsule, collateral ligaments, and muscles. Burns can also cause skin contractures, leading to loss of motion.

PATHOLOGY

Recently, posttraumatic capsular contracture has been the focus of basic science research. Contracted elbow capsules have been shown to be substantially thicker than normal with associated collagen disorganization and fibroblast infiltration (Fig. 2). In addition, altered levels of cytokines were thought to play a role in this process as elevated levels of matrix metalloproteinases (MMP-1, MMP-2, and MMP-3) and abnormal distribution tissue inhibitors of MMPs (TIMP-2) were demonstrated in contracted capsules.[5]

Myofibroblasts, and their expression of the contractile protein alpha-smooth muscle actin (α-SMA), have been identified as key contributors in the development of posttraumatic capsular contractures.[6,7] Over the past decade, Hildebrand et al have studied capsular changes in posttraumatic elbow contractures, as well as a correlative rabbit knee model of posttraumatic capsular contractures.[8–16] They have demonstrated that myofibroblast numbers and the expression of α-SMA are elevated in contracted elbow capsules,[9] as are collagen types I, III, and V and various MMPs, indicating increased matrix turnover.[10] Moreover, myofibroblast numbers were inversely related to elbow range of motion.[11] Upregulators of myofibroblast and collagen synthesis, transforming growth factor-beta 1 (TGF-β_1), and connective tissue growth factor (CTGF) were also

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