

General

Postoperative Stiffness After Upper Limb Surgery: Prevention and Management

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The upper limb experiences postoperative stiffness as a frequent disabling complication that restricts range of motion, extends recovery time for work and sports activities and diminishes patient contentment. The condition develops from an intensified wound-healing response which primarily involves inflammation and fibrosis. The clinical presentation of joint stiffness manifests as frozen shoulder (adhesive capsulitis) with capsular thickening and rotator-interval/coracohumeral ligament contracture but elbow contracture includes both capsular fibrosis and heterotopic ossification (HO). The prevention of stiffness requires atraumatic surgical methods combined with precise hemostasis, selective capsular release for high-risk patients, optimized pain management and early structured rehabilitation.

The diagnosis process requires serial goniometry measurements, pain scales, joint-specific functional scores and imaging tests to rule out structural causes and detect HO.

The management sequence starts with education, analgesia and physical therapy before progressing to hydrodilatation, corticosteroid injections, and the moving to open surgical management. The success of patient outcomes depends on early identification of patients, proper risk assessment and coordinated care during the perioperative period. Future research should focus on conducting adequately powered randomized trials, developing biomarker-based risk prediction systems, testing new antifibrotic/biologic treatments and creating personalized sensor-based rehabilitation plans.

1. INTRODUCTION

Postoperative stiffness is defined as a persistent limitation in joint motion that exceeds the expected course of surgical recovery, leading to pain, functional impairment, and a diminished quality of life.¹ In the shoulder, this complication typically presents as adhesive capsulitis, characterized by capsular thickening and contracture resulting in marked loss of both active and passive motion.² In the elbow, postoperative contracture arises from capsular fibrosis and periarticular soft-tissue tightening, with heterotopic ossifi-

cation (HO) occasionally adding further mechanical restriction and pain.³ Such stiffness can develop after a range of procedures, including rotator cuff repair, shoulder arthroplasty, elbow fracture fixation, and elbow arthroplasty or arthrolysis.⁴ Its onset is influenced by patient-related factors, as well as surgical factors including prolonged operative time, hematoma formation, and extensive soft-tissue dissection.^{5,6}

This review examines the underlying mechanisms, risk factors, and preventive strategies associated with postoperative stiffness, summarizes current diagnostic and man-

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agement approaches, and identifies emerging directions in biomarkers, antifibrotic therapies, advanced imaging, and personalized rehabilitation.

2. PATHOPHYSIOLOGY OF POSTOPERATIVE STIFFNESS

Upper limb postoperative stiffness is a complication caused by a multitude of factors ranging from patient susceptibility, surgical trauma, and others.⁷ Despite their differences, both shoulder and elbow stiffness share same biological mechanisms.⁸

2.1. BIOLOGICAL MECHANISMS

Following upper limb surgery, the body initiates a normal wound-healing cascade. In some patients, this healing response can become excessive and maladaptive, leading to capsular fibrosis and joint contracture.⁹

The pathogenesis of postoperative stiffness involves aberrant activation of fibroblasts and excessive deposition of extracellular matrix (ECM) components. TGF- β 1 drives fibroblast-to-myofibroblast differentiation and upregulates the synthesis of collagen types I and III.^{10,11}

Fibronectin (FN) and tenascin C (TNC), ECM proteins regulated by TGF- β , are involved in fibrosis.^{12,13} Their increased expression, along with upregulation of TGF- β 1 receptor I, is linked to capsular injury and contributes to the inflammation and fibrosis seen in frozen shoulder.^{12,14} Vimentin, a cytoskeletal protein and marker of fibroblasts and myofibroblasts, is significantly expressed in the anterior capsular regions such as the rotator interval, coracohumeral ligament, and axillary pouch, while absent in the posterior capsule.^{15,16}

In addition to fibrosis, neovascularization and neoinnervation play crucial roles in FS pathogenesis. Increased vascular endothelial growth factor (VEGF) and cluster of differentiation 34 (CD34) drive angiogenesis, which likely contributes to the intense pain experienced by patients.^{17,18}

3. DIFFERENCES BETWEEN FROZEN SHOULDER AND ELBOW CONTRACTURE

Despite similar pathophysiological processes, FS and elbow contracture (EC) have distinct features.

Adhesive capsulitis (AC), or FS, is characterized by progressive loss of shoulder range of motion (ROM), both active and passive, accompanied by pain, due to fibrosis and contracture of the joint capsule.¹⁹ FS involves reduced joint capsule volume from loss of the synovial lining, adhesion of the axillary recess to itself and the humeral neck, tightening the glenohumeral capsule.¹⁹ Thickening and fibrosis of the rotator interval, particularly of the coracohumeral ligament, play a key role in restricting ROM.^{20,21}

EC refers to stiffness of the elbow caused by thickening and fibrosis of the capsule, ligaments, and tendons.²² Prolonged elbow immobilization leads to fibrotic remodeling

of both anterior and posterior capsule and periarticular soft tissues.²³

A major contributor to decreased ROM in EC is heterotopic ossification (HO), the formation of bone in periarticular soft tissues from abnormal differentiation of mesenchymal stem cells into osteoblasts. HO is driven by factors such as bone morphogenetic proteins, prostaglandin-E2, and inflammatory cytokines. Ectopic bone lacks a periosteum and features distinct histological zones with elevated osteoblast and osteoclast activity.²⁴ The risk of clinically significant HO depends on injury severity.²⁵

3.1. INCIDENCE AND RISK FACTORS

Several factors, including patient comorbidities, type of injury, surgical procedure, and the quality and timing of postoperative rehabilitation, influence its development. AC affects approximately 2%–5% of the general population,²⁶ while post-traumatic elbow stiffness occurs in up to 5% of patients following elbow trauma.²²

3.1.1. PATIENT COMORBIDITIES

Many risk factors have been identified for the development of post-operative upper limb stiffness. Women have been shown to be at a higher risk of developing adhesive capsulitis than men.²⁷ Lower body weight, lower body mass index (BMI), and a positive family history of idiopathic adhesive capsulitis also increase the risk for shoulder stiffness.²⁸ Age is another significant factor, as frozen shoulder most commonly presents in individuals aged around 55–60 years.²⁹ In contrast, younger age has been correlated with a higher incidence of elbow contracture.³⁰

Endocrine conditions contribute substantially to stiffness risk. Patients with diabetes mellitus (DM) are more likely to develop both idiopathic and postoperative adhesive capsulitis compared to healthy individuals.³¹ A meta-analysis by Zreik et al. reported that individuals with DM are five times more likely to develop FS. Moreover, 13.4% of patients with AC had DM, and up to 30% of people with DM were found to have AC.³² Retrospective analyses have also shown that diabetic patients have a higher risk of poor outcomes following open arthrolysis for post-traumatic elbow stiffness.³³

Thyroid disorders further increase the risk of post-operative stiffness.³¹ A meta-analysis of 10 case-control studies involving over 127,000 patients found a higher prevalence of thyroid disease among AC patients (OR = 1.87), particularly hypothyroidism (OR = 1.92) and subclinical hypothyroidism (OR = 2.56), while hyperthyroidism showed no significant association.³⁴

3.1.2. SURGICAL APPROACH

A prospective study by Ghoraishian et al. showed that in patients undergoing reverse total shoulder arthroplasty (RTSA), stiffness was observed in 47% of patients at 3 months, 31% at 6 months, and 25% at one year.³⁵ A retrospective study by Robinson et al. found that out of 55 total elbow arthroplasties, HO was seen in 84%, particularly high

in trauma cases (96%), significantly higher than in elective surgeries (72%).³⁶ Following arthroscopic rotator cuff repair (ARCR), shoulder stiffness occurs in approximately 4% to 15% of patients.⁵ Another prospective study by Salas et al. observed shoulder stiffness in 31% of patients at 3 months post-op, decreasing to 20% by 6 months.⁷ Elbow procedures also carry a risk, with a systematic review of over 14,000 elbow arthroscopies reporting an overall stiffness rate of 4.5%.³⁷

4. PREVENTION STRATEGIES

4.1. SURGICAL TECHNIQUES

It has been observed that minimally invasive arthroscopic techniques may lead to better outcomes compared to open techniques in terms of post-operative stiffness. A systematic review by Denard et al. showed that an average of 150° in forward flexion after open rotator cuff repair, while arthroscopic techniques averaged between 161° and 166°, an average of 14° difference.³⁸ This difference might be due that the arthroscopic techniques have a smaller incision, leading to less soft tissue damage, therefore a faster recovery and potentially less fibrosis compared with open techniques.³⁹

As for rotator cuff tears, the prevention of postoperative stiffness can be achieved by a cuff repair with concomitant capsular release. In high-risk patients, such as those with comorbidities or a history of preoperative stiffness, arthroscopic capsular release (ACR) has proven effective both preventively and therapeutically.⁴⁰

4.2. PHARMACOLOGIC INTERVENTIONS

The use of non-steroidal anti-inflammatory drugs (NSAIDs) for the prevention of HO following elbow surgery is still in debate.⁴¹ Although NSAIDs have been shown to significantly reduce HO, especially post-hip surgeries,⁴²⁻⁴⁴ their role in elbow trauma remains less clear.⁴⁵ Based on Atwan et al. there was no significant difference in the formation of HO between patients taking indomethacin and the control group.⁴⁶ Similar findings were observed by Bochat et al. between patients who received a 3-7 day course of NSAIDs and control group.⁴⁷

As for Radiotherapy (RT), it has shown promising results in preventing HO following elbow surgery. In a study by Robinson et al., 36 patients with significant postoperative elbow stiffness were treated with a single-fraction dose of radiotherapy (600–700 cGy) between 1 and 4 days post-operatively. Of these, 33 patients demonstrated an improvement in range of motion compared to baseline measurements.⁴⁸ Similar findings were reported by Maender et al.,⁴⁹ and Geller et al. found that administering RT either preoperatively or postoperatively in elbow surgery significantly reduced the incidence of HO, with no significant difference observed between the two timing strategies.⁵⁰

When discussing the prevention of FS, many other pharmaceutical agents have been shown to reduce pain, but limited evidence was found in the conservation of the ROM.⁵¹

Berner et al. found that intra-articular corticosteroids (IA CS) and platelet-rich plasma (PRP) were more effective than physical therapy alone for pain relief, with PRP also having a positive effect on the range of motion in the shoulder. However, oral NSAIDs and hyaluronic acid injections did not show any significant benefit.⁵²

4.3. REHABILITATION PROTOCOLS

Early mobilization is crucial following upper limb surgery, as most range-of-motion gains occur within the first few months postoperatively.⁵³ When early continuous passive motion (eCPM) is paired with standard physiotherapy, it accelerates recovery and increases the likelihood of regaining functional motion within one year after elbow contracture release.⁵⁴ Huang et al. reported that patients receiving physical therapy (PT) plus eCPM achieved higher functional ROM at 6 months (70.4% vs 44.8%) and functional flexion $\geq 130^\circ$ (85.2% vs 58.6%) compared with PT alone.⁵⁴ Wang et al. demonstrated that three months of CPM is optimal; one month was insufficient, while five months offered no added benefit.⁵⁵

Effective pain control also facilitates early mobilization and helps prevent postoperative stiffness. A recent RCT by Babasiz et al. investigated the use of peripheral nerve block following arthroscopic elbow arthrolysis and showed significant ROM improvement in flexion-extension ($95^\circ \rightarrow 124.4^\circ$) and pronation-supination ($150^\circ \rightarrow 170.6^\circ$) at 6 months.⁵⁶

Su et al. compared different nerve block techniques for shoulder surgery; it showed that interscalene block provided better pain relief than suprascapular block in the first 12 hours, and combining suprascapular with axillary block further enhanced analgesia.⁵⁷

Other important factors include patient education and the adoption of individualized rehabilitation strategies rather than a one-size-fits-all approach.^{58,59}

5. DIAGNOSIS AND ASSESSMENT OF POSTOPERATIVE STIFFNESS

5.1. CLINICAL ASSESSMENT

Postoperative stiffness should be suspected in any patient who continues to have limited ROM beyond the expected recovery period. Diagnosis relies primarily on clinical evaluation. Assessment begins by identifying which movement planes are most restricted, determining whether the limitation affects active, passive, or both ROMs, and noting whether end-range resistance is due to pain or mechanical constraint. This distinction is essential to differentiate muscular guarding from capsular contracture or bony obstruction. For example, shoulder stiffness is characterized by a global loss of motion, with both active and passive deficits. Importantly, a loss of external rotation is often the earliest and most specific indicator of adhesive capsulitis.⁶⁰ Elbow stiffness typically manifests as impaired extension, flexion, or both, and a functional arc of motion below 30° – 130° can severely impair daily activities.⁶¹

To quantify these limitations, goniometry remains the clinical gold standard for measuring joint ROM. It allows objective documentation of motion in different planes and is crucial for monitoring progression and treatment response over time,⁶² and it's essential to incorporate it in routine assessment.⁶³

Pain assessment is another key component of evaluation and should be integrated with ROM findings. It is measured using the Visual Analog Scale (VAS) or Numeric Rating Scale (NRS) to help distinguish mechanical stiffness from pain-limited motion caused by inflammation, edema, or guarding.⁶²

Clinical provocation tests, such as the Hawkins-Kennedy, Neer, and Speed's tests, may be useful in ruling out coexisting pathology (e.g., impingement, biceps tendinopathy).⁵⁸ For the elbow, valgus stress testing and palpation can differentiate intrinsic joint restriction from soft-tissue contractures or nerve entrapments. A firm end-feel or mechanical block typically indicates intra-articular pathology such as capsular fibrosis or osteophyte formation, while tenderness over the medial epicondyle or signs of ulnar nerve irritation may suggest soft-tissue involvement or cubital tunnel syndrome.⁶⁴

5.2. IMAGING MODALITIES

Magnetic resonance imaging (MRI) and ultrasound are non-invasive tools for evaluating soft tissue and joint capsule abnormalities. MRI details capsular thickening, synovitis, tendon integrity, and muscle atrophy, relevant after rotator cuff repair or capsular release.^{65,66} Ultrasound increasingly allows dynamic assessment of postoperative shoulder stiffness and aids procedures such as aspiration or corticosteroid injections, while evaluating rotator cuff integrity and subacromial bursitis.⁶⁷

For the elbow, ultrasound identifies joint effusion, synovial hypertrophy, and heterotopic ossification. Plain radiographs detect joint incongruity, osteophytes, or calcifications, and CT provides high-resolution imaging for surgical planning, subtle hardware malposition, or post-traumatic deformities.⁶⁸⁻⁷¹

Dynamic fluoroscopy helps distinguish intrinsic from extrinsic stiffness by visualizing real-time joint movement and mechanical blocks, particularly in post-traumatic elbow contracture involving both intra- and periarticular structures.⁷²

5.3. CLASSIFICATION AND TIMING OF INTERVENTION

Postoperative stiffness can be categorized by time since surgery to guide therapy:

- **Acute phase (<6 weeks):** Inflammatory stage; characterized by pain, swelling, and restricted movement from soft tissue irritation and edema. Conservative strategies—including cryotherapy, anti-inflammatory medications, and supervised pain-free passive range-of-motion exercises—are most effective in this window.⁷³
- **Subacute phase (6–12 weeks):** Inflammation begins to resolve, but fibrous tissue deposition and capsular

thickening dominate. Interventions may escalate to corticosteroid injections or hydrodilatation to preserve motion.⁷⁴ Persistent restriction may signal the need for procedural consideration.

- **Chronic phase (>12 weeks):** Characterized by established capsular fibrosis, muscle atrophy, and often heterotopic ossification, this stage is less responsive to noninvasive treatments. Surgical intervention—either arthroscopic or open capsular release—is often necessary to restore range of motion.^{75,76}

5.4. IMPORTANCE OF EARLY RECOGNITION AND RISK STRATIFICATION

Early diagnosis and intervention are essential to prevent chronic, debilitating stiffness. Evidence shows that initiating therapy during the acute phase significantly reduces the risk of long-term functional impairment.⁶⁰ Several factors exist to assess whether escalated treatment is needed.

5.4.1. INDICATORS FAVORING CONTINUED CONSERVATIVE MANAGEMENT⁶⁰

- Gradual improvement in ROM with therapy
- No mechanical block on imaging
- Mild to moderate pain responsive to medication
- Clinical signs of muscle guarding rather than structural contracture

5.4.2. INDICATIONS FOR TRANSITIONING TO INVASIVE MANAGEMENT^{74,76}

- Plateaued progress despite 6–12 weeks of therapy
- Mechanical end-range resistance suggesting fibrosis or intra-articular obstruction
- Imaging-confirmed heterotopic ossification or severe capsular thickening
- Pain that impedes rehabilitation
- Progressive functional limitation despite adherence

5.5. CLASSIFICATION SYSTEMS TO GUIDE MANAGEMENT

Several joint-specific classification systems provide important guidance for identifying the etiology of stiffness and selecting appropriate treatments.

In adhesive capsulitis, the Zuckerman and Rokito classification distinguishes between:

- **Primary (idiopathic):** often associated with systemic conditions such as diabetes or thyroid dysfunction. It typically responds to conservative management and follow a self-limiting course.⁶⁰
- **Secondary:** postoperative, post-traumatic, or systemic in origin⁷⁷; which is more resistant to therapy and often require procedural intervention.⁶⁰

For the elbow, Morrey's classification divides stiffness into:

- **Intrinsic:** intra-articular causes such as osteophytes, capsular fibrosis, or adhesions.

- **Extrinsic:** extra-articular sources like muscular contracture, skin scarring, HO, or deformity.
- **Mixed:** involving both intrinsic and extrinsic components.⁷⁸

This classification guides surgical decision-making: intrinsic contractures may benefit from arthroscopic or open capsular release, while extrinsic stiffness may require soft tissue lengthening, HO excision, or deformity correction.

For post-traumatic elbow stiffness, Kay's classification evaluates structural involvement, including:

- Soft tissue contractures
- Joint incongruity
- Malalignment
- Instability

Collectively, these systems support clinical decision-making since prompt recognition of stiffness unresponsive to initial treatment is essential to prevent permanent contractures and optimize surgical outcomes.^{60,76,78}

6. MANAGEMENT OF ESTABLISHED STIFFNESS

6.1. NONOPERATIVE TREATMENTS

6.1.1. PHYSICAL THERAPY

Nonoperative management is the first-line approach for most cases of postoperative stiffness in the early and subacute phases. Structured physical therapy (PT) should begin early to prevent fibrous adhesions and loss of motion. PT should be progressive and individualized, starting with passive stretching to maintain joint capsule length, followed by active-assisted and active ROM exercises as tolerated. For shoulder stiffness, core exercises include pendulum movements, table slides, wall climbs, and capsular stretches. Elbow stiffness typically involves sustained flexion and extension stretching, often using gravity-assisted positioning or pulley systems.^{62,79}

Active stretching, introduced once pain and inflammation are controlled, improves dynamic control, neuromuscular re-education, and functional mobility. Repetitive, high-frequency active movements—such as wall push-ups, pulley exercises, or towel-assisted stretches—performed 3–5 times daily have been effective in improving shoulder and elbow ROM and reducing long-term stiffness.^{62,79} Low-load, long-duration static stretching (e.g., dynamic splints or manual hold-relax techniques) has shown superior gains in capsular compliance compared to high-load, short-duration stretches.⁸⁰

Manual therapy—including joint mobilization, end-range mobilization, proprioceptive neuromuscular facilitation (PNF), and scapular stabilization—is often incorporated to enhance outcomes. End-range mobilizations and oscillatory movements help break adhesions, PNF techniques improve muscle activation and joint control, and scapular-focused rehabilitation prevents compensatory movements in shoulder dysfunction.⁷⁹

Adjunctive modalities may further enhance outcomes:

- **Superficial heat therapy:** Moist hot packs, paraffin baths, or infrared radiation applied 15–20 minutes before stretching increases tissue extensibility and collagen elasticity.^{79,81}
- **Cryotherapy:** Ice packs or cold compression for 15–30 minutes post-exercise reduces inflammation, edema, and muscle spasm, improving therapy tolerance.⁸¹
- **Transcutaneous Electrical Nerve Stimulation (TENS):** Modulates pain via A β fiber stimulation and spinal nociceptive inhibition, aiding compliance during ROM exercises.^{81,82}
- **Therapeutic ultrasound (US):** Elevates deep tissue temperature to promote capsular and muscular relaxation; pulsed US shows mild benefit in adhesive capsulitis when combined with exercise and manual therapy.^{62,81}
- **Phonophoresis and iontophoresis:** Transdermal delivery of corticosteroids or NSAIDs, mainly for pain relief in tendinopathy or bursitis; evidence for post-operative stiffness is limited.⁸¹
- **Neuromuscular electrical stimulation (NMES):** Promotes muscle recruitment, prevents atrophy, and improves dynamic stabilization in shoulder and elbow muscles after immobilization.⁸¹
- **Laser therapy (low-level laser therapy, LLLT):** May have anti-inflammatory effects and promote tissue healing, though evidence in capsular contracture is preliminary.⁸¹

Combining these modalities enhances outcomes by reducing pain, improving compliance, and optimizing tissue responsiveness. Clinicians should also individualize treatment based on recovery phase, tissue irritability, and patient tolerance.

6.1.2. CORTICOSTEROID INJECTIONS

Intra-articular corticosteroid injections, like triamcinolone acetate (20–40 mg) or methylprednisolone acetate (40–80 mg), administered under ultrasound guidance, are valuable in reducing synovial inflammation and facilitating PT. Effectivity is highest when administered during the painful (freezing) phase of adhesive capsulitis, typically within the first 3–6 months of symptom onset. Pain relief generally occurs within days, allowing earlier initiation or intensification of ROM exercises.^{83,84}

Optimal effect is achieved with a single injection, but up to 3 injections spaced at least 4–6 weeks apart may be considered in refractory cases.⁸⁵

6.1.3. HYDRODILATATION

Hydrodilatation involves injecting a combination of saline (10–40 mL), corticosteroids (e.g., 40 mg triamcinolone), and local anesthetics (e.g., lidocaine) into the joint capsule under ultrasound or fluoroscopic guidance. The goal is to mechanically distend the capsule, rupture adhesions, and reduce pain, often producing a palpable “pop” during injection. The procedure is typically done once but may be

repeated after 2–4 weeks if stiffness persists.^{61,86} It was found to significantly improve shoulder function and ROM at both 6-week and 3-month follow-up, when combined with corticosteroids and PT.⁸⁶

6.1.4. ELBOW-SPECIFIC INTERVENTIONS

Nonoperative strategies are key for managing elbow extension contractures and capsular tightness. Dynamic splints applying low-load prolonged stretch (LLPS) are most effective when worn 6–8 hours daily, often at night, producing meaningful ROM gains over weeks to months.⁸⁰ Static progressive splints, which allow incremental tension adjustments, are also effective in chronic contractures.

Adjunctive PT for the elbow should include:

- Active-assisted ROM exercises using pulley systems or gravity.
- Joint mobilizations (e.g., humeroulnar distraction, posterior glide).
- Soft tissue techniques for periarticular fibrosis and myofascial restrictions.

In post-traumatic, post-arthroplasty, or post-burn cases, the risk of HO is higher. Pharmacologic prophylaxis is indicated, with indomethacin (75–150 mg/day for 1–3 weeks) being the most common. Bisphosphonates such as alendronate (35–70 mg/week) also inhibit osteoblastic activity and reduce HO incidence and severity when administered early postoperatively.^{87,88}

6.2. MINIMALLY INVASIVE INTERVENTIONS

Usually used when conservative management fails after 8–12 weeks. It is most effective in patients with primary (idiopathic) adhesive capsulitis in the freezing or frozen phase, with no significant osteoarthritis, instability, rotator cuff tears, or previous shoulder surgery.^{89,90} However, it is less effective in patients with osteoporosis, post-traumatic stiffness, and diabetes due to the high risk of complications.⁶²

6.2.1. MANIPULATION UNDER ANESTHESIA (MUA)

MUA is performed in a controlled sequence under general anesthesia, starting with forward elevation, followed by external rotation at the side, abduction with external rotation, and finally internal rotation behind the back. Each motion is applied with steady force and end-range oscillation until a capsular release is felt, taking care to avoid excessive leverage.⁹⁰

Risks include humeral fractures, rotator cuff tears, labral injuries, and brachial plexus stretch injuries, with higher incidence in older, osteoporotic, or diabetic patients. Fracture rates may reach 1.6–2.6%, and cuff injury rates up to 7% in some cohorts.^{79,91}

Despite risks, MUA offers rapid early gains in ROM, and when followed by intensive therapy, may restore function in appropriately selected cases.

6.2.2. ARTHROSCOPIC CAPSULAR RELEASE

Arthroscopic capsular release (ACR) is a precise, minimally invasive procedure indicated for patients with recalcitrant adhesive capsulitis unresponsive to ≥3 months of therapy, particularly diabetics or those with secondary frozen shoulder.⁷¹

Intraoperatively, release typically begins at the rotator interval and proceeds to the coracohumeral ligament, anterior capsule, and inferior capsule, with the posterior capsule addressed in severe cases.⁹² Outcomes are favorable, with ROM gains of +40° in forward flexion, +30–50° in external rotation, and sustained pain relief, especially when combined with rehabilitation.⁹³

For elbow stiffness, ACR allows direct resection of the anterior and posterior capsule, release of tethering bands, excision of heterotopic ossification, and removal of loose bodies or synovitis. ACR is particularly beneficial in post-traumatic or post-surgical elbow stiffness where fibrosis predominates without fixed bony blocks.⁹⁴ Complication rates remain low (~2–3%) in experienced hands, with improvements of 30–50° in arc of motion depending on preoperative stiffness severity.

Comparative studies indicate that ACR provides superior long-term outcomes and lower complication rates than MUA, although initial ROM gains may be faster with MUA.⁹³ Diabetic patients appear to benefit more from ACR than MUA due to dense fibrosis that may not yield with manipulation alone.^{62,71}

6.3. OPEN SURGICAL PROCEDURES

Open capsular release is reserved for patients with dense fibrosis, failed arthroscopic treatment, or extensive HO. It provides direct access to the joint and allows complete excision of fibrotic tissue, ossified masses, and osteophytes. It is indicated in post-traumatic elbow stiffness and in cases with extra-articular tethering.^{78,95}

Excision of HO is often performed concomitantly with open capsular release when HO significantly limits joint motion or causes pain and neurovascular compromise. Indications for HO excision include mature, well-demarcated ossifications confirmed by imaging (typically CT with 3D reconstructions), failure of nonoperative management, and progressive functional impairment.^{68,70}

Infection remains a notable risk, with rates between 1.55%–6.7%, rendering prophylactic antibiotics and strict intraoperative sterility essential, especially in cases with extensive dissection or previous implants.³⁷

Neurovascular injury to the ulnar (the most common), median, and radial nerves in the elbow, is a serious complication. Preoperative nerve mapping, intraoperative nerve identification and protection, and, in some cases, prophylactic anterior transposition of the ulnar nerve are strongly recommended.^{88,96,97}

Postoperative rehabilitation is critical after open or arthroscopic interventions. Protocols begin immediately or within 24–48 hours depending on wound integrity, with Continuous passive motion (CPM) devices often used in the first 1–2 weeks to maintain intraoperative ROM gains.⁹⁸

Supervised physical therapy typically follows a phased approach:

- Phase I (0–2 weeks): Pain control, edema reduction, gentle passive ROM, and early neurovascular monitoring.
- Phase II (2–6 weeks): Progression to active-assisted and active ROM, scapular and core stabilization, and early strengthening.
- Phase III (6+ weeks): Full strengthening, proprioceptive training, and gradual return to functional tasks.^{78,99}

When heterotopic ossification (HO) or prior trauma is present, therapy may need to continue for 3–6 months.

7. OUTCOMES AND PROGNOSIS

Frozen shoulder requires a longer recovery period when treated non-operatively, often taking six months to over a year. Nonoperative methods result in slow but steady improvements in range of motion (ROM), which are generally slower than those achieved with surgical interventions.¹⁰⁰ Procedures such as MUA or arthroscopic capsular release accelerate recovery,¹⁰¹ with marked improvements in both active and passive ROM noted within two to three months post-procedure.¹⁰² Early surgical treatment also allows patients to regain function faster than conservative management.¹⁰³

Patients with long-standing adhesive capsulitis frequently develop pronounced joint stiffness and respond less effectively to rehabilitation. Comorbidities, particularly diabetes mellitus, further complicate recovery, increasing the risk of delayed ROM improvement and recurrence.¹⁰⁴ For example, 72% of patients undergoing MUA regained pre-injury function within six months, and 92% were satisfied with the outcome; early timing of the procedure was associated with better results.¹⁰⁵

In contrast, arthroscopic capsular release often provides more sustainable outcomes. In one study, 98.7% of patients reported high satisfaction, returning to work by approximately two months and to sports such as swimming or tennis by around 2.5 months.¹⁰¹ Early improvements in ROM and pain relief were observed within one week to one month, with complete pain resolution at a mean of 3.7–3.8 weeks.¹⁰²

In elbow contracture, the extent of capsular fibrosis and the presence of heterotopic ossification (HO) largely determine recovery. Surgical procedures such as capsular release or HO excision are highly effective, particularly in severe cases, with early intervention often improving the flexion-extension arc from 57° to 116°.¹⁰⁶

A systematic review found that patients treated within 6–10 months post-injury achieved the greatest ROM gains and lowest complication rates. Patients operated on after one year also improved, but recovery generally required a longer rehabilitation period.¹⁰⁷ Structured rehab programs are essential for restoring joint function, with many patients returning to everyday activities like writing or lifting within six months.¹⁰⁸ Eighty percent of individuals un-

dergoing HO-related surgery reported favorable long-term outcomes when early surgery was combined with disciplined rehabilitation.¹⁰⁹

8. FUTURE DIRECTIONS AND RESEARCH GAPS

8.1. NEED FOR HIGH-QUALITY RANDOMIZED TRIALS

Research into treatment options for postoperative stiffness faces challenges due to a lack of high-quality randomized controlled trials. Most elbow contracture studies rely on small case series and retrospective analyses. Multicenter RCTs are needed to compare surgical versus nonsurgical interventions and to determine optimal timing for procedures such as MUA and ACR. Studies should include standardized outcome measures (e.g., ASES, MEPS) and extended follow-up periods, as patients often experience prolonged symptoms.¹¹⁰ Robust RCTs will be essential for guiding clinical decisions, particularly in patients with diabetes.

8.2. BIOMARKERS FOR RISK PREDICTION AND THERAPEUTIC TARGETING

Cytokines and fibrotic mediators, including TGF- β 1, IL-6, TNF- α , and fibronectin, are present in adhesive capsulitis and elbow contracture tissues and may serve as predictive markers and therapeutic targets.^{14,111,112} The “fibrotic signature” concept—identifying patients with elevated TGF- β 1 or pro-fibrotic gene polymorphisms—could inform preventive interventions.¹¹³ Future studies should implement synovial fluid or serum assessments to establish predictive models and guide individualized treatment approaches.

8.3. EMERGING ANTIFIBROTIC AND BIOLOGIC TREATMENTS

Innovative pharmacologic and biologic therapies targeting fibrotic processes may serve as adjuncts to mechanical interventions. Pirfenidone, an oral antifibrotic, blocks TGF- β 1 and other mediators and shows promise based on pre-clinical studies.¹¹⁴ PRP injections have demonstrated superior pain and ROM improvement in early-stage adhesive capsulitis compared with corticosteroids and physical therapy.¹¹⁵ MSCs possess immunomodulatory properties but require further preclinical and safety evaluation due to potential pro-fibrotic effects.¹¹⁶ Timing and patient selection remain critical for all antifibrotic therapies, including ketotifen and metformin.¹¹⁷

8.4. INNOVATIONS IN IMAGING AND MOTION TRACKING

Early detection of stiffness before contracture becomes permanent is crucial. Shear wave elastography (SWE) enables assessment of capsular and ligamentous stiffness and has demonstrated sensitivity in detecting progressive stiffness in coracohumeral ligaments and rotator cuff tendons.²¹ Wearable motion sensors and inertial measurement units (IMUs) offer continuous, objective ROM monitoring to identify plateaus and guide therapy adjustments in real-

time.¹¹⁸ 3D CT reconstructions improve surgical planning for elbow stiffness by mapping HO and capsular anatomy.⁷⁸ Integrating elastography with motion tracking may provide a comprehensive, patient-specific diagnostic approach.

8.5. PERSONALIZED REHABILITATION PROTOCOLS

Although rehabilitation is the cornerstone of stiffness management, most protocols remain standardized rather than individualized.^{119,120} Sensor-based feedback systems and app-guided exercise platforms facilitate personalized rehabilitation, enhancing compliance and accelerating recovery.⁹⁸ Future studies should explore adaptive protocols that adjust exercise intensity, frequency, and modality based on real-time ROM, pain scores, and functional objectives.

8.6. PSYCHOSOCIAL AND EDUCATIONAL INTERVENTIONS

Patient beliefs, expectations, and emotional states strongly impact adherence and outcomes. Individualized educational programs tailored to personal objectives and cultural characteristics improve compliance and reduce therapy discontinuation.^{121,122} Integrating cognitive-behavioral therapy or motivational interviewing into multidisciplinary care enhances outcomes in refractory cases. Digital platforms using videos and interactive apps offer cost-effective opportunities for patient education and improved satisfaction.^{121,122}

9. CONCLUSION

Effective management of frozen shoulder and elbow contracture requires a proactive, multidisciplinary approach. Whenever feasible, surgical treatment should remain minimally invasive with careful neurovascular protection. Postoperative care should include multimodal analgesia, patient education, supervised therapy, corticosteroid injections or hydrodilatation for shoulder treatment, and HO prophylaxis for the elbow. The combination of optimized conservative care and manipulation under anesthesia, along with arthroscopic capsular release provides reliable motion restoration when stiffness persists. Open procedures should be reserved for dense fibrosis, bony blocks, or mature HO, alongside extensive rehabilitation. Future advancements will rely on well-powered randomized trials, validated biomarkers for risk stratification, and evaluation of emerging antifibrotic and biologic therapies. Incorporating advanced imaging techniques—including elastography—and wearable motion-tracking technologies may enable earlier diagnosis and more individualized rehabilitation. Long-term registry data will be essential for optimizing treatment protocols and enhancing patient-centered outcomes.

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