

# CONTRACTURE

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## CONTRACTURE

**Primary Disciplinary Field(s):** Medicine, Physical Therapy, Rehabilitation Science

### 1. Core Definition and Mechanism

A contracture is defined clinically and pathologically as the permanent shortening of a muscle, tendon, ligament, or joint capsule, resulting in severe restriction or complete loss of passive and active range of motion (ROM) in a joint. This irregular reduction or shrinking of soft tissue structures often leads to irreversible impairment because the affected tissues lose their inherent elasticity and ability to expand or stretch fully. Unlike temporary stiffness, a true contracture represents a fixed deformity resistant to passive stretching, establishing a state where the joint is held in a specific position, typically flexion, significantly impacting mobility and functional independence.

The core mechanism involves a transformation of the normal tissue architecture. In muscle tissue, this frequently involves a reduction in the number of functional sarcomeres (the contractile units), which are essential for muscle lengthening. Concurrently, the non-contractile connective tissues surrounding and within the muscle--the fascia and perimysium--undergo pathological changes characterized by the proliferation of collagen fibers and increased cross-linking. This fibrotic change replaces normal, pliable tissue with dense, inelastic scar tissue, effectively setting a physical limit to joint excursion. This transformation is typically progressive, meaning that the longer the condition goes untreated or unmanaged, the more severe and fixed the impairment becomes.

The development of a contracture frequently arises following conditions that promote extended inactivity or that cause chronic discomfort upon movement. When movement is uncomfortable or mechanically inhibited, the body naturally minimizes motion. Extended, implemented inaction, often necessitated by severe illness, immobilization (such as prolonged casting), or profound weakness, allows the tissues to adapt to the shortened position. This adaptation is reinforced by neurological factors, suchological, and biomechanical forces, culminating in a functionally debilitating structural deformity.

### 2. Classification of Contractures

Contractures are broadly classified based on the primary tissue or system involved in their genesis, crucial information for determining appropriate therapeutic intervention. The four principal categories include myogenic, neurogenic, dermatogenic, and tendinous contractures, though mixed types are common. Myogenic contractures, perhaps the most common, originate directly within the muscle tissue itself, often due to muscle shortening following trauma, disuse atrophy, or specific localized conditions like ischemic injury, famously exemplified by **Volkman's ischemic**

**contracture.**

**Neurogenic contractures** stem from underlying central or peripheral nervous system pathology that results in continuous muscle imbalance or spasticity. Conditions such as cerebral palsy (CP), stroke (CVA), spinal cord injury, or traumatic brain injury lead to abnormal muscle tone (hypertonicity) which pulls joints into fixed positions. Over time, the sustained high tone structurally shortens the muscles, causing a true fixed deformity. This category highlights the critical interplay between neurological signaling and musculoskeletal structure.

**Dermatogenic contractures** occur when the skin and underlying subcutaneous tissues are damaged, resulting in scar formation that restricts mobility. Severe burns are the prototypical cause, as the healing process involves massive collagen deposition and wound contraction, which pulls adjacent joints into flexion, particularly common at the elbow, knee, and neck. Finally, **\*\*tendinous or arthrogenic contractures\*\*** involve the shortening of tendons or the thickening and tightening of the joint capsule and ligaments, often secondary to chronic inflammatory arthritis or prolonged joint effusion, where the joint is maintained in a position of maximum comfort (usually a slightly flexed position) to minimize pain.

### 3. Etiology: Primary Causes and Risk Factors

The development of contractures is multifactorial, usually stemming from a combination of mechanical, neurological, and inflammatory triggers. The most straightforward cause is prolonged **immobilization**, where a limb is held in a static, shortened position for weeks or months, such as following fracture fixation or in the care of critically ill patients who are heavily sedated. In these scenarios, the lack of mechanical stretch signal to the muscle fibers and connective tissue triggers the replacement of functional sarcomeres with non-extensible fibrous material.

A significant contributing factor is the presence of underlying **neurological disorders** that disrupt the normal balance of agonist and antagonist muscle groups. Conditions characterized by spasticity or hypertonicity, such as multiple sclerosis, Parkinson's disease, or post-stroke hemiplegia, generate excessive, constant tension on certain muscle groups. This chronic high tone gradually leads to the structural shortening that defines a contracture. Furthermore, genetic disorders, particularly various forms of **muscular dystrophy** (e.g., Duchenne), predispose individuals to contractures as the healthy muscle tissue degenerates and is replaced by fibrotic and fatty tissue, causing progressive joint limitation even with active movement.

Other major risk factors include extensive trauma, particularly deep thermal **burns**, which necessitate extensive skin grafting and generate severe scarring. Inflammatory joint diseases, such as rheumatoid arthritis, also increase risk, as the painful and swollen joint naturally adopts a position of least pain, leading to protective, fixed positioning. The elderly population is particularly vulnerable due to generalized sarcopenia, reduced mobility, and increased incidence of conditions

requiring extended bed rest or immobilization.

#### 4. Pathophysiology: Cellular Mechanisms

Understanding the cellular changes underlying contracture formation is essential for targeted intervention. The process involves complex shifts in both muscle fiber biology and the organization of the extracellular matrix (ECM). In disuse-related contractures, the primary change within the muscle fiber itself is the net loss of sarcomeres in series. This anatomical shortening means that even if the muscle were relaxed, it physically lacks the necessary length to allow full joint extension.

Simultaneously, the connective tissue component undergoes a highly significant fibrotic cascade. Fibroblasts, the primary cells responsible for ECM maintenance, become hyperactive, transitioning into myofibroblasts which produce excessive amounts of rigid, type I collagen. This collagen is laid down in a disorganized manner and develops abnormal cross-links, stiffening the tissues that normally provide elasticity, such as the perimysium, epimysium, and fascia. The resulting ECM is dense, less hydrated, and highly resistant to deformation, mechanically restricting the muscle's ability to lengthen and the joint's ability to move.

In ischemic contractures, such as Volkmann's, the pathology is even more aggressive, involving cell death (necrosis) due to lack of blood supply. The resulting dead muscle tissue is rapidly replaced by large, dense sheets of non-contractile fibrous scar tissue. Furthermore, chronic inflammation, often present in neurogenic and arthritic conditions, releases cytokines and growth factors that actively stimulate this fibrotic process, accelerating the conversion of pliable soft tissue into rigid, permanent restrictive material.

#### 5. Clinical Manifestations and Diagnosis

The most salient clinical manifestation of a contracture is the significant reduction in passive ROM, meaning the joint cannot be moved fully by an external force, and active ROM, meaning the individual cannot move the joint themselves. The affected joint is often visibly held in a fixed, characteristic posture, such as a flexed wrist or elbow, or a foot held in equinus (pointed down). Patients often report feelings of tightness, stiffness, and potentially localized pain upon attempting to stretch the restricted limb.

Diagnosis relies primarily on meticulous physical examination using **goniometry** to accurately measure the joint angles and the degree of ROM restriction. The key diagnostic differentiator is the "fixed" nature of the deformity; unlike spasticity, which may temporarily yield to slow stretching, a contracture cannot be overcome by manual force without causing pain or tissue damage. Clinicians assess whether the restriction affects both active and passive movement equally, which helps distinguish a fixed contracture from simple muscle weakness or pain-related guarding.

In complex cases, imaging modalities are used to assess the underlying structural integrity. Musculoskeletal ultrasound can visualize the organization and texture of the muscle and tendon, revealing fibrotic changes and tendon thickening. Magnetic resonance imaging (MRI) is often employed to differentiate between muscle atrophy, fatty infiltration, and true dense scar tissue replacement, particularly in diagnosing conditions like fibrosis or extensive muscle damage. These diagnostic tools confirm the degree of structural change and guide the choice between conservative and surgical management.

## 6. Management and Treatment Modalities

The management of contractures is highly dependent on their severity, chronicity, and underlying cause, necessitating a multidisciplinary approach involving physical therapists, occupational therapists, and orthopedic specialists. Conservative treatment focuses on reversing or mitigating the structural changes through sustained mechanical load. This includes aggressive, prolonged stretching protocols, often combined with heat application to increase tissue extensibility.

A cornerstone of non-surgical management is the use of orthotic devices, specifically splinting or serial casting. **Serial casting** involves applying a series of casts, each holding the joint in a slightly increased range of extension, maintaining the stretch for several days before a new cast is applied. This method applies a low-load, long-duration stretch proven effective in promoting the addition of new sarcomeres in series and potentially remodeling the connective tissue matrix. Static or dynamic splinting is used post-casting or in milder cases to maintain the achieved ROM.

When conservative methods fail to achieve functional ROM, surgical intervention becomes necessary. Surgical procedures aim to release the restricting tissues, a process often termed **contracture release**. This may involve tenotomy (cutting the tendon), fasciotomy (releasing the fascia), or lengthening procedures (Z-plasty techniques) on the shortened muscle-tendon unit. In cases involving severe joint capsule restriction, arthrolysis (surgical release of the joint capsule) may be required. Post-surgical rehabilitation is critical and intensive, as immediate and aggressive mobilization is necessary to prevent the surgically released tissues from scarring down and reforming the contracture.

## 7. Prevention and Prognosis

Given the difficulty in fully reversing established contractures, prevention is paramount, particularly for high-risk patient populations such as those with neurological deficits or prolonged immobilization. The primary preventative measure involves maintaining full passive range of motion (PROM) through regular, consistent stretching exercises performed multiple times per day. For bedridden patients, proper positioning, ensuring that joints are kept in neutral or slightly extended positions rather than the typical position of comfort (flexion), is essential.

The prognosis for contractures varies significantly based on etiology. Contractures caused by disuse or temporary muscle imbalance often have a good prognosis if managed early and aggressively with physical therapy and casting. Conversely, contractures secondary to severe neurological damage (e.g., severe spastic cerebral palsy) or extensive ischemic necrosis often carry a guarded prognosis, as the underlying pathology is ongoing or the structural damage is irreversible, necessitating lifelong management to maintain functional status. The key factor influencing long-term outcome is patient compliance and adherence to prescribed stretching and splinting protocols.

## 8. Further Reading

[Contracture - Wikipedia](#)

[Range of Motion \(ROM\)](#)

[Volkmann's Ischemic Contracture](#)

[Cerebral Palsy and Spasticity](#)

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