A. Mechanisms of Injury

Many factors produce mechanical injuries or trauma in sports. Soft tissue damage occurs through direct or indirect trauma to muscles, ligaments, and joint capsules. Usually, direct trauma refers to an injury occurring from blunt trauma or a sudden overload, and is known as macrotrauma, i.e., true muscle tear or ligament sprain. In contrast, indirect trauma results from repeated submaximal loading, leading to clinical signs and symptoms. Injury presents itself in three stages: acute, subacute/overuse, and acute/chronic.

The first, or acute, stage of direct trauma stems from sudden overloading, or macrotrauma (e.g. a 100 meter runner exploding out of the starting blocks). The subacute/overuse stage occurs when increased loads degenerate body tissues due to excessive cumulative loading, leading to microtrauma and an accompanying inflammatory response (e.g. achilles tendinitis in the endurance athlete or runner, Figure 9-1). The last type, acute/chronic stage, integrates both cumulative loading and sudden overloading (e.g. chronic achilles tendinosis that ruptures in a long jumper). Chronic tendinosis is a degenerative condition without inflammation.

Figure 9-1. Schema demonstrating theoretical pathways of sports-induced tissue damage.
Whether muscle injury is caused by direct or indirect trauma, the end result is tissue dysfunction characterised by pain, inflammation, and altered internal tissue stress. The injury often results in functional disability, whereby an athlete may be able to carry on daily living routines, but is limited in his or her capacity to train and compete.

Any activity loads and deforms tissue, an effect known as a stress/strain, and described through a load and tissue elongation curve. As connective tissue is deformed it either stretches or tears, depending on the magnitude, rate, and intensity at which the loading occurs. Collagen deforms under low loading and fails at high loads. When the load is removed from normal tissue during the elastic phase, the material returns to its pre-stretch length. Injury occurs when the tissue is stretched into the plastic phase, causing tissue failure.

Of all the tissues involved, tendon is the least elastic. The most frequent site of injury in muscle strains is the myotendinal junction, because of increased collagen content at the transition zone of muscle sheath to tendon. This area has decreased local extensibility, as does scar tissue, and is frequently termed a stress riser. This transition in biologic tissues, which also appears at the tendoperiostial junction, is a point that is more susceptible to stress and injury.

The relatively new term “tendinopathy” has been adopted as a general clinical descriptor of tendon injuries in sports. In overuse clinical conditions in and around tendons, frank inflammation is infrequent and if seen, is associated mostly with tendon ruptures. Tendinosis implies tendon degeneration without clinical or histological signs of intratendinous inflammation, and is not necessarily symptomatic. The term “tendonitis” is used in a clinical context and does not refer to specific histopathological entity. Tendonitis is commonly used for conditions that are truly tendinosis, however, and leads athletes and coaches to underestimate the proven chronicity of the condition.

Paratendonitis is characterised by acute oedema and hyperemia of the paratendon with infiltration and inflammatory cells, and possibly the production of a fibrinous exsudate with the tendon sheath causing a typical crepitus, which can be felt on clinical examination.

The term “partial tear of the tendon” should be used to describe the macroscopically evident partial tear of a tendon. This is an uncommon acute lesion. Most articles describing the surgical management of partial tears of a given tendon in reality deal with degenerative tendinopathies. The combination of pain, swelling, and impaired performance should be labeled tendinopathy. According to the tissues affected, the terms tendinopathy, paratendinopathy, or pantendinopathy (from both the tendon and the surrounding tissues involved) should be used.

B. Examining Soft Tissue Injuries

Examination for injury in soft tissues such as muscle involves initial palpation with minimal force or compression (in the case of acute injuries), and progresses to firmer compression or higher loads if increased density has not been distinguished
or pain has not been provoked at the site of the suspected lesion (see Table 9-1 for examination steps). One can also have the athlete contract the muscle to increase the tension or passively stretch the myotendinal unit while palpating the area. The pain associated with palpation is secondary to the stimulation of free nerve endings with inflammation, decreased extensibility of tissue, or tissue insufficiency.

While palpating muscle tissue, one should search carefully through various layers of tissue to find remnants of injuries and healing. Subtle tissue texture abnormalities may exist, and might be missed if the tissue were examined erratically. These abnormalities must be considered in formulating an assessment. However, the clinician must avoid going too deep or hard with palpation, using pain as a guideline.

The clinician needs to apply pressure and to sense the reactivity of the tissue. Since scar tissue heals three dimensionally, it does not fall into place like a brick. Instead, scar tissue reaches in the direction of the fascia and the neighbouring muscle sheaths, binding these tissues together. For example, when a runner strains a hamstring, the sheath tear heals and binds to neighbouring muscle sheath. The hamstring muscle group still functions to flex the knee, yet the athlete complains of dull ache or pain in the posterior thigh. The reason may be that independent movement has been lost and the area of scar tissue has limited the extensibility of the myotendinal unit. Muscles do function and limbs do move, but the normal gliding that occurs between neighbouring tissues is lost. As a result, there is a constant low-grade inflammatory process at the site of the decreased mobility. Scar tissue has a poor blood supply and is not as strong or resilient as the primary tissue it replaces. This area will likely be a site of re-injury secondary to the transition zone of normal tissue to scar tissue.

Table 9-1. Examination of soft tissue injury.

1. History
   - onset
   - pain location
   - mechanism of injury
   - prior treatment and rehabilitation
   - goals of athlete

2. Physical exam
   - inspection
   - AROM/PROM
   - palpation
   - neurological; myotome, dermatome, peripheral nerve tests, deep tendon reflexes
   - strength and motor control
   - special tests
   - functional exam
   - gait analysis

3. Assessment

4. Treatment goals

5. Treatment plan

6. Treatment procedures
C. The Wound Healing Process

1. Reaction: The Inflammatory Phase

This first phase can last up to 72 hours, and involves a number of inflammatory responses, manifested by pain, swelling, redness, and increased local temperature. Accumulation of exudate and oedema begins the process of tissue repair following injury when a blood clot forms and seals the area. In musculotendinous injuries, there is myofilament reaction and peripheral muscle fiber contraction within the first two hours. Oedema and anoxia result in cell damage and death within the first 24 hours, and the release of protein breakdown products from damaged cells leads to further oedema, tissue hypoxia, and cell death. Oedema and joint swelling, with or without pain, is associated with a reflex inhibition of spinal activation of skeletal muscle. Phagocytosis then begins to rid the area of cell debris and oedema.

2. Regeneration and Repair: The Fibro-elastic/Collagen-forming Phase

This phase lasts from 48 hours up to 6 weeks. During this time structures are rebuilt and regeneration occurs. Fibroblasts begin to synthesise scar tissue. These cells produce Type III collagen, which appears in about four days, and is random and immature in its fiber organisation. Capillary budding occurs, bringing nutrition to the area, and collagen cross-linking begins. As the process proceeds, the number of fibroblasts decreases as more collagen is laid down. This phase ends with the beginning of wound contracture and shortening of the margins of the injured area.

3. Remodelling Phase

This phase lasts from 3 weeks to 12 months. Gradually, cross-linking and shortening of the collagen fibers promote formation of a tight, strong scar. It is characterised by remodelling of collagen so as to increase the functional capabilities of the muscle, tendon, or other tissues. Final aggregation, orientation, and arrangement of collagen fibers occur during this phase.

Regeneration of the injured muscle does not fully restore muscle tissue to its prior levels, as fibrous scar tissue slows muscle healing. The two processes of healing and fibrosis compete with each other and impair complete regeneration. Transforming Growth Factor–Beta 1 (TGF-β1) is an ubiquitous substance that initiates a cascade of events that activate both myogenesis and fibrosis.

Measures that may block fibrosis have been shown experimentally to alter the effects of TGF-β1 on the fibrotic process. Decorin is a proteo-glycan that impedes fibrosis by combining with TGF-β1. Suramin is an anti-parasitic drug that competes with TGF-β1 for its binding sites to the growth factor receptor. Interferon gamma disrupts the pathways involved in TGF-β1 signal transduction, and when given i.m 1–2 weeks after an injury improved muscle function in animal models. All of these agents are under active study, and have undergone clinical trials.
References


